

EM Resident

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Acute Abdomen in a Pediatric Patient

The Business of Care
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TOGETHER
WE HEAL

Are You OK?

Priyanka Lauber, DO

Editor-in-Chief, *EM Resident*

@PriyankaLauber

Hello EMRA Family,

How are you all doing? We have all gone through some life changes lately, haven't we?

Residents have transitioned from one level to another. Medical students have become interns. Senior residents have graduated and entered this new and challenging workforce. It's been an exhilarating time for many, but also a difficult time for most. I have spent the past few months taking the time to invest in others. I talked to people who are graduating, who are experiencing significant life transitions, who are making huge life decisions... and **one thing has been clear — many people are struggling**. As we near the end of the COVID era of closed restaurants and social distancing, I think many people are retaking inventory of their lives.

Many have made changes, including leaving marriages and relationships, moving to different states or even

countries, and many people have straight-up quit their jobs or pursued other passions. It has been a fascinating time to see people bloom, but it's not all just roses. There are bound to be some thorns along the way. We *must* grant ourselves grace and patience as we evolve from our comfort zone to pursue things that often make us uncomfortable.

I wanted to write this editorial forum for anyone struggling: You are worthy and what you're going through matters. And **there are resources to help** you.

One of the biggest things I have noticed, especially in high-functioning individuals, is too much compartmentalizing. We don't often take the time to feel and experience our emotions. We shove them deep within us in a box. It's what we are hard-wired to do.

Think about it — we work in an environment where we can potentially see a very sick child or diagnose a pleasant individual with cancer and then literally walk into another room to diagnose an ear infection. And we do this day-in and

day-out for years, even decades. This is not how humans are supposed to work, mentally and emotionally. We are empathic and social creatures. We yearn to bond and care and are often not built for trauma as we experience repeatedly in EM. So over the years, our brains get re-wired so we can function at our jobs. Unfortunately, we also let this emotional and mental aspect bleed into other parts of our lives.

We think we can “fix” ourselves like we fix others, sometimes without investing in ourselves. We believe if we can get through one hurdle and onto another, we can be “happy.” But that is not how happiness works. I read this fantastic quote that **“happiness isn't a goal, but rather the side effect of intentional living.”** That quote changed my life and has been helpful to many. We are happy when we are in charge of our own life. We are delighted when we make plans and goals and follow through. So if you're going through a difficult time in life, make sure you start with a checklist. Here's one I put together for myself and others.

- **Check-in with family and friends.** Frequently during residency/medical school, we tend to ignore our friends and families inadvertently. This often also happens when a romantic relationship goes south. Often it's not intentional, but instead, we do not have the emotional or time bandwidth. Now is the time to reach out. With residency ending, I reconnected with old friends. I went on trips to see them. Reinvest your time into people who matter to you and made you who you are.
- **List the activities you *used* to enjoy.** Sit down and intentionally think about the activities that brought you joy and enjoyment in the “beforetime” — before COVID, before a relationship, before medical school. Medicine gives us a lot of meaning and purpose, but, unfortunately, it can also take a lot away from us. Now is the time to take it back. For me, I realized I used to love working out, reading books for fun, hiking, and traveling. I enjoyed playing board games and bar games. Those are things that brought me immense happiness that I have been trying to reprioritize.
- **Journal.** Often we don't pause to acknowledge our thoughts and feelings. We just jam it down and move on. “If we don't handle the past, it will handle us.” Stop and write down thoughts and feelings, especially when it feels like it's just a big swirling mess in there. It'll help us to respond rather than react. And it can reveal the real underlying issues. The writing-down process doesn't have to be perfect; you can even use your phone. The important thing is to be consistent and do it.
- **Reach out to get help.** Often, most workplaces offer access to therapists who provide individual or even couples counseling for reduced or no cost. Therapy works best when one's matched with the ideal therapist and you feel comfortable and honest. If your work doesn't offer therapy, check out apps and online resources that are manageable even without insurance that covers mental health. Before each session, reflect on your week and write down your expectations for that session. Don't be afraid to be honest about your expectations during the beginning of each session, but it's also equally important to set very reasonable goals and expectations.

And at the end of the day, **give yourself time**. It's OK to have setbacks, and it's OK not to feel good all the time. Remember, you can't have happiness without sadness. And sometimes we hold onto suffering, but suffering doesn't have to hold on to us. Let it go. ★

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EDITORIAL STAFF

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Priyanka Lauber, DO
Lehigh Valley Health Network

EDITORS

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Greenville Health System

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Shiprock, NM

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The Power of Connection

RJ Sontag, MD

EMRA President
Mid Ohio Emergency Services
@RJSontagMD

She stood alone on the stage, looking at the crowd. She took a deep breath, closed her eyes, and opened her soul.

We leaned forward as she began telling the story of a trauma patient encounter where everything seemed to go wrong. From conflicting details about the mechanism to frustrations with consultants to lamenting a broken health care system, her story illuminated the struggles we all endure everyday.

She was honest and introspective in a way that only happens when you are looking people in the eyes — when you know that the whole truth must be shared. I have never seen anyone be so vulnerable in front of so many strangers. It was cathartic and important and necessary — and unlike anything I had ever seen before.

The moment was part of EMRA's Airway Stories, an intimate evening where dozens of us gather annually to share a drink and a story. Unscripted and spontaneous, it serves as a catharsis for those who take the stage and those who sit and listen. It is one of the things I have missed most about canceled conferences, and I cannot wait to gather again to support other young physicians as we share our stories.

It is one of many in-person events EMRA is planning for October's ACEP Scientific Assembly in Boston. **Make plans to join us as we create moments that can only happen face-to-face.**

HIGHLIGHTS

20 in 6: Get ready to watch residents and fellows shine as they compete to deliver the best fast-paced, informative six-minute lectures using only 20 slides.

Case-Con: From classic presentations of zebra cases to rare presentations of common complaints, Case-Con is chock full of unique moments for students and residents alike.

EMRA Committees: 6,000+ EMRA members work together in 20 committees to help you find your niche in EM. Whether your passion is ultrasound or prehospital or social EM, EMRA committees have an event for you.

Job & Fellowship Fair: As the pandemic evolves, the job market is opening up for graduating residents, and this is your opportunity to meet in person with your future employer.

RepCo & Town Hall: Join representatives from residencies across the country as we create the policy that shapes the specialty we love.

SimWars & MedWAR: No one does competition like EMRA! Cheer on your co-residents as they compete in innovative scenarios to challenge their medical knowledge and physical abilities in these entertaining classic EMRA events. ★



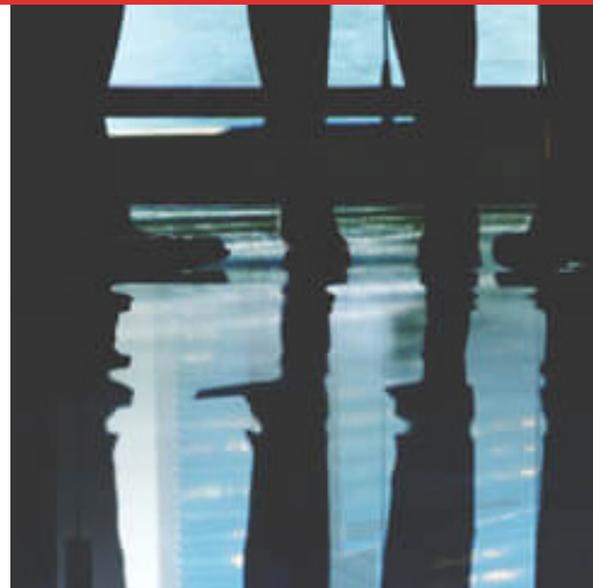
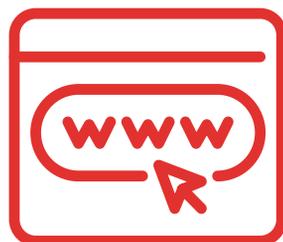
SAVE THE DATE!

EMRA's Job & Fellowship Fair will be held in-person at ACEP21 on Monday, Oct. 25. Meet with recruiters and programs from across the country, and get ready to network with leading emergency medicine employers.



EMRA JOB & FELLOWSHIP FAIR

Monday, Oct. 25 at ACEP21 | 5 – 7 pm Eastern
Boston Convention & Exhibition Center



<https://www.emra.org/be-involved/events--activities/acep/>

Business and Administrative Curriculum for Emergency Medicine Residents

A National Needs Assessment

Author contributions: All authors were involved with developing the concept and design for the study. Dr. Cozzi and Dr. Jarou were involved with data acquisition. Data analysis was performed by Dr. Jarou. All authors were involved with interpretation of the data. Dr. Cozzi was primarily responsible for drafting the manuscript, along with Dr. Jarou and Dr. Messman. All authors were involved with manuscript revision.

Nicholas P. Cozzi, MD, MBA

EMRA Representative to ACEP Board
Spectrum Health/Michigan State University
FDNY

Zachary J. Jarou, MD, MBA

EMRA Past President
University of Chicago

Adam J. Rodos, MD

University of Illinois-Chicago

Nicholas R. Stark, MD, MBA

EMRA Administration & Operations
Committee Chair
University of California-San Francisco

Arnab Sarker, MD, MBA

New York University

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Baylor College of Medicine

Philip Tseng, MD, MBA, MEd

The Ohio State University

Jason P. Seamon, DO

Emergency Medicine Residency
Program Director
Spectrum Health/Michigan State University

Jeffrey S. Jones, MD

Spectrum Health/Michigan State University

Anne M. Messman, MD, MHPE

Wayne State University

Emergency medicine (EM) residency exposes trainees to the informal curriculum of practice management. Through the Kern model of curriculum development, we present a national targeted needs assessment of EM residents and attendings to guide the development of future business of EM curricula.

INTRODUCTION

EM residency training requires the acquisition of vast amounts of medical knowledge and procedural skills necessary to skillfully diagnose and treat acute, undifferentiated patients of all ages and walks of life. However, beyond becoming clinically excellent, EM residents are also expected to learn about practice management topics such as billing and coding, resource utilization, reimbursement, risk management, operations management, patient flow, patient experience, quality and safety. Awareness of and competency in non-clinical, practice management issues is essential for career success.¹ Both

the Accreditation Council for Graduate Medical Education (ACGME) and the American Board of EM (ABEM) codified these components of the curriculum through the ACGME's mandated integration of systems-based practice competencies into resident milestones^{2,3} and inclusion in ABEM's *Model of the Clinical Practice of EM*, a framework for the core content of the specialty of EM.⁴

Prior studies of young physicians have shown that while the majority felt residency prepared them to practice medicine, very few felt prepared to manage business aspects related to practicing medicine.⁵ Many attending physicians believe that inclusion of practice management skills during residency would lead to increased initial success post-residency.⁶ While the amount of time EM residencies dedicate to education on documentation, coding, and reimbursement has slowly increased over recent decades, many residents still spend less time learning these topics than the length of a single clinical shift during their entirety of their residency training.⁷ Inadequate knowledge of EM practice management issues may place residents at a competitive disadvantage in the workplace and when searching for post-

graduation employment opportunities given the increased competition.

Curricula for teaching the business of EM have previously been created and implemented at some EM residency programs.^{8,9} The widely-known Kern model of curriculum development emphasizes that planning educational experiences is a multi-step, iterative process involving: (1) problem identification and general needs assessment, (2) targeted needs assessment, (3) goals and objectives, (4) educational strategies, (5) implementation, and (6) evaluation and feedback.¹⁰

The objective of this study is to perform a national, targeted needs assessment of EM residents and attendings to guide the development of future business of EM curricula. We will explore the current state of EM practice management education during residency training, perceived barriers to implementing this curriculum, and preferred modalities for learning this type of content moving forward. More specifically, for multiple administrative topics, we will describe current self-reported knowledge, perceived importance, and interest in learning more.

METHODS

Study Design

This study was an observational, cross-sectional needs assessment deemed Institutional Review Board (IRB)-exempt by the <<blinded for submission>> IRB (2019-641). The survey was first piloted with members of the Administration/Operations and Research Committees of the Emergency Medicine Residents' Association (EMRA), as well as with two individual residency programs with subsequent minor revisions in the language and format of the survey. Survey design was optimized using expert insight from national emergency medicine research fellowship directors. Survey responses were collected using SurveyMonkey (San Mateo, CA). Respondents who submitted incomplete surveys were excluded. Results are reported in accordance with STROBE Guidelines.¹¹

Study Setting and Population

Unique survey links were sent to current resident members of EMRA (6537) in December 2019 with no financial or any other kind of incentives for completion. One reminder email was sent via these unique links. A faculty-specific needs assessment was also created and distributed on engagED (the online community of the American College of Emergency Physicians) and the listservs of 40 departments of EM, the Council of Residency Directors in EM (CORD), and three state chapters of the American College of Emergency Physicians (Michigan, Texas, and Illinois) due to ease of access.

Measurements and Key Outcomes Measures

Demographic information collected for resident respondents included their EM residency program, graduation year, age, gender and ethnicity. Demographic information about faculty respondents included the name of the residency program they attended as well as current residency program affiliation (if applicable), job title, and year of residency graduation. Both groups were questioned as to whether they possessed additional business or administrative training such as an advanced degree, completing a short-course, or being involved with local or national committees.

All respondents were asked to rate their general knowledge of business in EM concepts, what importance they place on learning these concepts during residency, perceived barriers to providing business/administrative education to EM residents, as well as details about any current administrative education currently in-place at their residency programs. Additionally, residents were asked to rate how interested they would be to learn more about 20 proposed administrative and business topics, while faculty were asked to rate how essential they considered each of these topics to be, both using a 5-category Likert scale. Topics were generated from pilot study at one author's residency program. Lastly, respondents were asked to rate the learning modalities that they would consider most effective to learn these concepts. Copies of both the resident and faculty needs assessment can be found as supplemental files.

Data Analysis

For each topic, we calculated the proportions of residents who self-identified as having below-average knowledge and above-average resident interest in learning more, as well as the proportion of attendings that rated each topic as essential. Summary statistics describing the marginal distribution of responses for all survey questions were computed. Comparisons between categorical variables were evaluated using the Chi-square test. Comparisons between resident and attending perceptions regarding topic importance were evaluated using Pearson's correlation coefficients. P-values of <0.05 were considered statistically significant. All data analysis was performed using RStudio version 1.2.5001 running R version 3.5.1.

RESULTS

Characteristics of Survey Respondents

The survey invitation email was opened by 3158 residents, 291 of whom clicked through to the survey, and 207 of whom responded (6.6% response rate). There were 254 faculty respondents. Fourteen resident and 22 faculty surveys had to be excluded due to incomplete responses, leaving 193 and 232 resident and attending responses, respectively. Resident respondents were 64% male and 32% female. Two-thirds of residents (67.5%) were at 3-year residency programs, while 32.5% were at 4-year residency programs. Residents were 31% PGY1, 30% PGY2, 30% PGY3, and 6% PGY4 and represented 132 unique residency programs. The majority of attending respondents worked primarily in academic settings (55.6%), followed by 26.3% primary community and 18.1% mixed settings. The median residency graduation year of attending respondents was 2007 (IQR 2002-2014). Approximately half of respondents reported training or working at a program with an EM administrative residency elective. Many respondents reported exposure to administrative topics outside of residency training (see Table 1 for further details).

Main Results

Overall, 89.5% of respondents indicated that learning about business and administrative topics during EM residency is 'important' or 'very important' with no significant difference between residents and attendings ($p=0.601$). 49.8% of residents rated their knowledge of these topics as below average, compared with only 21.5% of attendings ($p<0.001$).

TABLE 1. Exposure to Additional Training

	Resident (N=197)	Attending (N=232)	Total (N=429)
Exposure to Additional Training			
Advanced degree	17 (8.6%)	29 (12.5%)	46 (10.7%)
ACEP ED Directors Academy (EDDA)	2 (1.0%)	24 (10.3%)	26 (6.1%)
ACEP Committee/Section	7 (3.6%)	22 (9.5%)	29 (6.8%)
Administrative fellowship	1 (0.5%)	7 (3.0%)	8 (1.9%)
Current/former administrative position	8 (4.1%)	81 (34.9%)	89 (20.7%)
Program Has EM Admin Elective			
Yes	95 (48.2%)	128 (55.2%)	223 (52.0%)
No	102 (51.8%)	104 (44.8%)	206 (48.0%)

A majority of residents (61%) stated their residency program does not prepare graduates for EM administrative challenges, though residents at programs with administrative electives feel more prepared than those at programs without electives, 47% vs 19% respectively ($p < 0.001$).

“Lack of time” was the top barrier identified to the administration of a business of EM curriculum during residency by both residents and attendings at 58% and 40%, respectively. The second largest barrier identified by attendings was “lack of faculty expertise” (33%), however residents were significantly less likely to cite this as a barrier (10%, $p < 0.01$). The most preferred learning method for residents was “self-paced online learning” (25%), followed by “administrative case discussions” (17%) whereas the most preferred from the attending perspective was “administrative case discussions” (35%), followed by “attending hospital committee meetings” (17%).

Residents were interested in learning more about all topics (range 75-95%), whereas attendings had a much broader range of what they considered essential (range 25-81%). Attending physicians found “documentation” and “patient flow/ throughput” as the two most essential topics (81% and 81%, respectively) while residents stated their desire to more fully learn these topics as: 87% vs. 89%, respectively. Sixty-eight percent of residents reported below average knowledge of “quality and value based payments (QVBP)” with an associated 81% of residents desiring more education in this topic, and 62% of attendings regarded this topic as essential. Sixty-six percent of residents stated their knowledge of “reimbursement, compensation, and finance (RCF)” was below average, with 95% of residents desiring to learn more and 77% of attendings finding this topic essential. Finally, the largest percentage of residents stated “contracts and practice models” as an area of below average knowledge (72%) with 91% of residents desiring more education and 75% of attendings finding this topic essential. The topic of highest weighted average of that which attendings find essential and residents desire knowledge was

“risk management of litigation (RML), with the lowest being “EM Informatics (Informatics)” at 53%.

Using an equally weighted composite of desire to learn and essential nature of topics, the top 10 topics included: risk management & litigation; reimbursement; operations; personal finance; documentation; contracts and practice models; complaints, crisis, conflict, and negotiation; physician leadership; billing and coding; and oversight of nurse practitioners (NPs) and physician assistants (PAs) (range 74-86%).

Correlation between below-average knowledge and the desire to learn more was not significant ($r = 0.37$; 95%CI -0.07 to 0.71). There was a statistically significant, moderate correlation between the essential nature of a topic as rated by attendings and resident desire to learn more ($r = 0.55$; 95%CI 0.17 to 0.81). The two most significant outliers between resident interest and attending physicians’ rating as essential

included: entrepreneurship and innovation (91% vs 25%, respectively) and basics of informatics (75% vs 31%, respectively). See table 3.

DISCUSSION

Both residents and attendings find learning EM admin topics during residency to be important, yet they continue to be insufficiently taught. This is consistent with findings from multiple other residency specialties, including family medicine,^{12,13} internal medicine,¹⁴ anesthesia,¹⁵ obstetrics/gynecology,¹⁶ otolaryngology,¹⁷ psychiatry,¹⁸ pathology,¹⁹ and general surgery^{20,21} which have all indicated a need for, and current deficiencies in, practice management education during residency training.²² It has been further proposed that management skills should be made a core component of medical education prior to residency to improve healthcare delivery systems and to develop a pipeline of physician leaders.²³ While the percentage of medical school graduates completing

TABLE 2. Importance, Current Knowledge, Biggest Barriers, and Preferred Modality to Learn EM Admin Topics During Residency

	Resident (N=197)	Attending (N=232)	Total (N=429)	p-value
Importance of Learning EM Admin Topics in Residency				0.601
1: Not at all important	1 (0.5%)	2 (0.9%)	3 (0.7%)	
2: Less important	9 (4.6%)	7 (3.0%)	16 (3.7%)	
3: Neutral	11 (5.6%)	15 (6.5%)	26 (6.1%)	
4: Important	99 (50.3%)	103 (44.4%)	202 (47.1%)	
5: Very important	77 (39.1%)	105 (45.3%)	182 (42.4%)	
Overall Knowledge of EM Admin Topics				< 0.001
1: Low	35 (17.8%)	13 (5.6%)	48 (11.2%)	
2: Less than average	63 (32.0%)	37 (15.9%)	100 (23.3%)	
3: Average	68 (34.5%)	74 (31.9%)	142 (33.1%)	
4: Above average	27 (13.7%)	76 (32.8%)	103 (24.0%)	
5: High	4 (2.0%)	32 (13.8%)	36 (8.4%)	
Biggest Barrier				
Lack of time	108 (54.8%)	108 (54.8%)	202 (47.1%)	0.003
Lack of faculty expertise	20 (10.2%)	75 (32.3%)	95 (22.1%)	< 0.001
Clinical obligations	44 (22.3%)	23 (9.9%)	67 (15.6%)	< 0.001
Lack of resident interest	18 (9.1%)	24 (10.3%)	42 (9.8%)	0.675
In-Service Training Exam prep	1 (0.5%)	3 (1.3%)	4 (0.9%)	0.399
Most Preferred/Effective Learning Modality				
Administrative Case Discussions	30 (15.3%)	80 (34.6%)	110 (25.8%)	< 0.001
Self-paced online (for Asynchronous Credit)	47 (24.0%)	30 (13.0%)	77 (18.0%)	0.003
Lectures during weekly residency conference	21 (10.8%)	27 (11.7%)	48 (11.3%)	0.765
Attending hospital committee meetings	17 (8.7%)	40 (17.2%)	57 (13.3%)	0.009
Lectures at national EM conferences	9 (4.6%)	8 (3.5%)	17 (4.0%)	0.552

TABLE 3. Proportion of Survey Respondent Perceptions on Proposed Topics for EM Admin Curriculum

	Residents with below-average knowledge	Attendings find essential	Residents want to learn more	Average of “Attending Essential” and “Resident Learn More”
Risk management and litigation	58.6%	80.6%	91.7%	86.2%
Reimbursement, compensation, and finance	65.8%	77.2%	94.8%	86.0%
Patient flow and throughput	34.7%	81.0%	88.6%	84.8%
Personal finance and investment	34.7%	78.0%	90.7%	84.3%
Documentation	16.1%	81.0%	86.5%	83.8%
Contracts and practice models	72.0%	74.6%	91.2%	82.9%
Complaints, conflicts, crises, and negotiation	45.1%	76.3%	89.1%	82.7%
Physician leadership	30.6%	74.6%	89.1%	81.8%
Billing and coding	58.0%	75.9%	85.5%	80.7%
Supervision of PAs and NPs	63.7%	63.4%	85.0%	74.2%
Quality and value-based payments	67.9%	62.1%	81.4%	71.7%
Well-being and burnout	8.8%	65.1%	78.2%	71.7%
Team dynamics	16.1%	55.6%	77.2%	66.4%
Regulatory and legal	64.8%	49.6%	82.4%	66.0%
People management and human resources issues	54.4%	48.3%	82.4%	65.3%
Patient satisfaction and experience	17.6%	54.3%	74.6%	64.5%
Medical staff relations	40.4%	50.4%	76.7%	63.6%
Licensing, credentialing, board certification	46.6%	45.3%	78.2%	61.8%
Entrepreneurship and innovation	65.8%	24.6%	90.7%	57.6%
Basics of EM informatics	54.9%	31.5%	74.6%	53.0%

dual Medical Doctor/Master of Business Administration (MD/MBA) degree programs increased by 50% between 2015 and 2019, still only 0.9% of medical school graduates pursue this route.²⁴

Both residents and attendings cite lack of time as the most common barrier to teaching administrative topics, as has been previously described.²⁵ Surprisingly, a significant proportion of EM attendings cite lack of faculty expertise as a barrier, suggesting that there may be a role of a national curriculum to deliver this content that may not be able to be delivered locally.

Attending physicians most preferred learning EM administrative topics

through administrative case discussions and by attending hospital committee meetings, while residents most preferred learning through self-paced online modules that could be completed for asynchronous conference credit. Given the time constraints and clinical obligations of completing residency, it is not surprising that residents are in favor of an asynchronous learning option. This provides both short- and long-term flexibility that allows residents to learn at their own pace, avoid missing content that is only delivered once during their training due to a scheduling conflict, and also allows residents who are earlier in residency to focus on becoming clinically excellent, while

allowing residents later in training to tackle this non-clinical curriculum once they feel comfortable with their clinical knowledge. However, online based modules would have to be high-quality and engaging to prevent residents from simply clicking through to completion. The advent of free, open-access medical education (FOAMed) has provided physicians-in-training new avenues of learning that were not previously available prior to the widespread adoption of social media.²⁶ Online modules, such as the Approved Instructional Resources (AIR) Series created by Academic Life in Emergency Medicine (ALiEM), which provide opportunities for residents to asynchronously earn conference credit while learning about clinical topics, have been shown to be widely adopted and valuable tools for enhancing resident education.²⁷ We anticipate that an asynchronous curriculum teaching EM administrative topics would be equally successful.

As expected, there was moderate correlation between topics that attendings found essential and topics that residents wanted to learn more about. It is unclear why there were such large discrepancies between resident and attending viewpoints related to the topics of informatics and entrepreneurship. As a technical, subspecialty area, it may have been

FIGURE 1. Weak Positive Correlation Between Current Resident Knowledge of EM Admin Topics and Interest in Learning More (r=0.37; 95%CI -0.07 to 0.71)

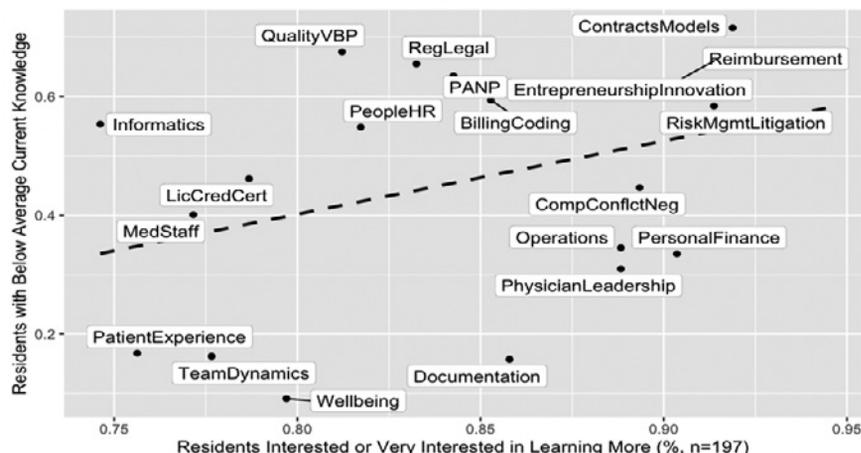
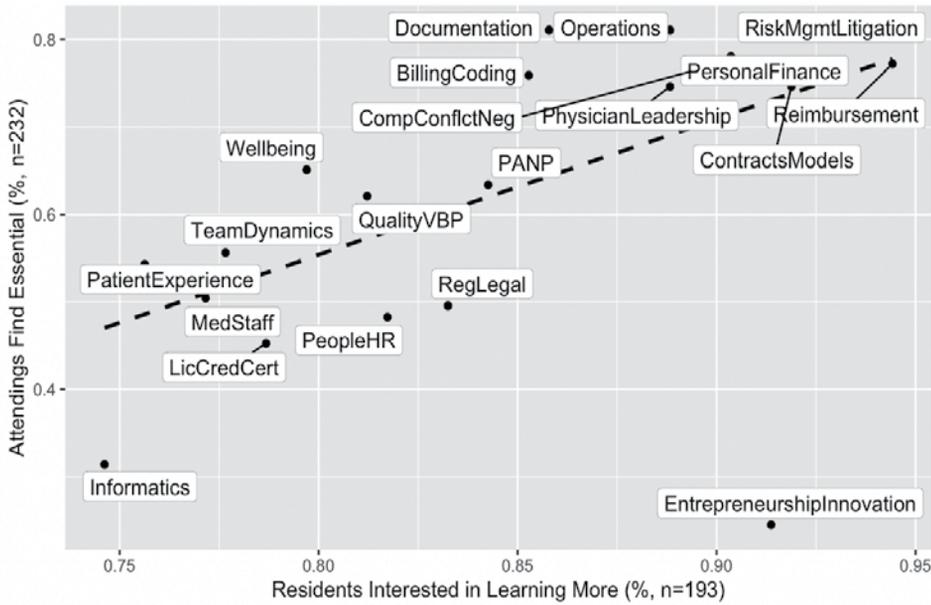


FIGURE 2. Moderate Positive Correlation Between Resident Interest in Learning More About EM Admin Topics and Attendings Finding Topic Essential ($r=0.55$; 95%CI 0.17 to 0.81)



possible that survey respondents did not fully understand what the field of clinical informatics entails. Additionally, while many resident respondents may be interested in learning more about entrepreneurship during residency, this interest may fade over time if they do not embark upon entrepreneurial activities early in their careers, leading to a lower proportion of attending respondents considering the topic essential. Surprisingly, there was not a strong correlation between topics that residents had below average current knowledge and those that they were most interested in learning more about. This may be because residents are not interested in learning in certain topics that they don't currently know about, such as informatics and quality/value-based payments, or because they want to continue to learn more about topics that they believe they have above average knowledge of already such as documentation and well-being.

Throughout the COVID-19 pandemic, the issue of workforce preparedness and equipping emergency medicine graduates to be competition applicants has been paramount. It can be hypothesized that a resident taught the practice essentials of emergency medicine may be at a competitive advantage in the job market given the value-added skills this training brings to the department and

physician group. In addition, training emergency medicine resident physicians value-based payments and quality is essential as we transition to a value-based reimbursement system.

LIMITATIONS

Our national survey has inherent limitations including surveying residents about the quality of their administrative education before completing the totality of their residency training. Determination of the response rate for attendings was not possible as the survey was distributed on various department and organizational listservs, making a true denominator

difficult to determine. Furthermore, it is difficult to account for potential overlap from individuals present in multiple listservs. Amongst residents invited to complete the survey, many were unwilling or unable to complete the optional survey leading to non-response bias. The sample of attending respondents is affected by voluntary response bias as likely those who are involved in resident education or those with interests in EM business and administration were more likely to complete the survey than attendings with different backgrounds. Attending responses related to their own residency training are subject to recall bias since the median time from residency graduation was 12 years.

CONCLUSION

Learning business and administrative topics is regarded as very important during residency, yet the majority of residents report low levels of knowledge and preparation and many faculty do not feel like they have content expertise. Based upon this targeted needs assessment, there is an opportunity to create a national, self-paced, online, case-based curriculum that is engaging and high value. Overall, residents and attendings agree about most, but not all, topics that should be prioritized in an EM business and administration curriculum. This is the first targeted needs assessment to identify and stratify topics of importance to both resident and attending physicians regarding the business of EM, allowing educators to confidently progress to subsequent stages of Kern's model of curriculum development. ★

The Future of Telehealth Panel Reflection

Christina Brown

Rush Medical College

Nicolas Kahl, MD

University of California-San Diego

Michael Dorritie, DO

John H. Stroger Hospital of Cook County

Nicholas Stark, MD, MBA

University of California San Francisco –
San Francisco General Hospital

A decade ago, telehealth seemed like a complicated, inefficient way to deliver care. With advancements in technology and the COVID-19 pandemic, telehealth has grown into a vital tool for emergency medicine physicians. At CORD Academic Assembly 2021, EMRA's Administration and Operations Committee partnered with the Informatics Committee to host "The Future of Telehealth in Emergency Medicine" panel discussion to gain an in-depth look at the implementation opportunities and barriers facing telehealth and what this could mean for its future within the field of emergency medicine.

The telehealth experts who joined for the panel included:

- **Aditi Joshi MD, MSc**, Thomas Jefferson University Hospital
- **Emily Hayden, MD, MHPE**, Massachusetts General Hospital
- **Sari Lahham, MD, MBA**, Kaiser Permanente

Throughout the COVID-19 pandemic, telehealth has emerged as an effective venue to deliver high-quality patient care. While several institutions implemented telehealth initiatives prior to the pandemic and were well-positioned to expand their efforts, many others started from scratch at the onset of COVID-19. Their ability to adapt quickly to the demands forced upon them by the pandemic affirmed the exciting opportunity for growth in telehealth. However, it also exposed some of the challenges that telehealth continues to pose to patients and physicians.

5 IMPORTANT CONSIDERATIONS FOR TELEHEALTH

1 Workflow can make or break telehealth.

Dr. Hayden discussed how it is important to acknowledge the barriers to adopting telehealth for both patients and physicians, and how to address these barriers. Some patients may not have the access to the hardware, software, or internet services that are required for a virtual visit. Additionally, Dr. Joshi noted that the telehealth interface for the physician must be user-friendly in order to reduce resistance to this new form of patient encounters.

2 Balance patient satisfaction and patient care.

Dr. Lahham emphasized how telehealth visits should mirror normal in-person visits as much as possible. It is important to educate the patient on their visit and train physicians on how to address patient concerns virtually.

3 Integration of virtual care and electronic health records.

Dr. Hayden shared how most video systems for virtual visits are separate applications from electronic health records, which ultimately leads to the need for double-documentation by physicians and staff. For telehealth visits, documentation remains an important part of the visit and should be consistent and detailed. Physicians must always include their recommendations during a virtual visit.

4 Consider patient and physician privacy.

Dr. Joshi explained how, even during virtual visits, HIPAA privacy laws still apply. It is necessary to ensure that both the patient and physician are in a location that maximizes privacy. The platform on which the virtual visit occurs also needs to be securely encrypted.

5 Be transparent about billing.

Dr. Lahham discussed how, with changing laws surrounding telehealth, billing has become complicated, as virtual visits require different codes than in-person visits. Importantly, Dr. Hayden noted that it is critical to inform patients that virtual visits are billable so they are not surprised upon receiving an invoice.

In addition to existing considerations, the panelists shared up-and-coming telehealth projects. Dr. Hayden discussed utilizing specially trained paramedics to help with managing patient care in the prehospital field through telehealth platforms. Dr. Joshi shared a new system at her hospital that allows the emergency department to virtually triage patients from their home. Lastly, Dr. Lahham explained how utilizing telehealth can help emergency departments across the country manage high patient volumes by virtually seeing patients in the waiting room.

Even with the exciting possibilities that telehealth offers, it is important to remember that it is a relatively new addition to the field of emergency medicine. As such, there will likely be increasing needs to improve medical education and training for medical students and residents in telehealth. It also requires improved access to technology and data management, both for patients and physicians. These challenges are multifactorial, and their solutions will depend on institutional commitment, community requirements, and financial investment in telehealth as a critical driver for quality patient care. While there may not be a one-size-fits-all approach for each organization, flexibility will allow institutions to remain creative and nimble in providing additional telehealth services to their patients.

Moreover, given the explosive growth and opportunity to shape the field, all the panelists advised that the current moment is a great chance for medical students and residents to get involved. Dr. Joshi recommends joining institutional or national committees devoted to telehealth projects. For students and residents who have an idea for a project, reach out to an attending faculty member who may be interested in driving growth in the telehealth space for your healthcare system. Additionally, Dr. Hayden suggested advocating on a national level — whether through EMRA, ACEP, or another organization — to help improve telehealth access and security for patients. Telehealth is a rapidly growing field of medicine, making this an exciting opportunity to get involved at all levels of training. ★

**This paper is paraphrased based on the panel discussion at CORD Academic Assembly 2021 and does not contain direct quotations.*

BRASH Syndrome

A VICIOUS CYCLE

Emily Newton

Eastern Virginia Medical School

Gabrielle Ransford, MD

Eastern Virginia Medical School

Anjeza Cipi, MD

Assistant Professor

Eastern Virginia Medical School

BRASH syndrome is an uncommon but often fatal condition in which an acute renal injury initiates a vicious cycle of bradycardia, hyperkalemia, hypotension and worsening renal failure in patients who take AV-nodal blocking medications. We report a case of an 84-year-old male taking an AV-nodal blocker (amlodipine) who presented in acute renal failure with profound bradycardia and slight hyperkalemia. He had minimal symptoms and was well-appearing at presentation; however, his condition rapidly deteriorated and he died the next day. The final diagnosis was BRASH syndrome.

Case

An 84-year-old male presented to the emergency department via EMS from his long-term care facility after staff became concerned that he was "lethargic." EMS noted a heart rate of 23 bpm and administered a 1L NS bolus without improvement in heart rate. Upon arrival to the ED, the patient was sitting up in a position of comfort, interacting with staff, answering questions appropriately, moving all extremities equally and appeared well and nontoxic. Vital signs were remarkable for HR 31 bpm, BP 118/50, RR 18 rpm, O₂ saturation of 89% on room air. He was afebrile. There were no remarkable physical exam findings aside from bradycardia with a systolic murmur. The only symptoms the patient reported were an episode of thoracic back and bilateral shoulder pain the night before presentation and one episode of dark stools the week prior. His past medical history was significant for hypertension, congestive heart

BRASH syndrome consists of a vicious cycle involving a combination of bradycardia, renal failure, AV-nodal blocking medication, shock, and hyperkalemia. The pathophysiology underlying BRASH syndrome has been well established for decades.

failure, chronic kidney disease, and former tobacco use. His medications included amlodipine 10 mg daily, hydralazine 25 mg three times daily, atorvastatin 20 mg daily, furosemide 20 mg daily, and aspirin 81 mg daily.

EKG obtained on arrival demonstrated severe junctional bradycardia with a heart rate of 27 bpm (Figure 1). A broad workup included: CBC, BMP, BNP, high-sensitivity troponin, PT/PTT/INR, TSH, magnesium level, ionized calcium level, and CXR. The patient was found to be in acute renal failure, with a creatinine of 7.9 mg/dL. Potassium was slightly elevated to 5.8 mmol/L. Troponin and BNP were within range of the patient's baseline. Hemoglobin was low at 7.6 g/dL, but was within the patient's baseline. Chest x-ray demonstrated pulmonary vascular congestion and slight interstitial edema, which was thought to be the

etiology of his mild hypoxia in the setting of known CHF history.

Cardiology was consulted early in the ED course and the patient was given atropine, which brought his heart rate up to 46 bpm from 31 bpm (Figure 2). He was admitted to the ICU with the goal of improving renal function prior to placing a pacemaker. His ICU stay was notable for persistent bradycardia as low as 28 bpm, which was not responsive to continuous dopamine infusion. As his condition deteriorated, he was not deemed to be a candidate for pacemaker placement or hemodialysis and the family ultimately decided to transition to comfort care. An inciting event for the acute renal injury was not clearly identified but was thought to be cardio-renal syndrome.

Discussion

BRASH Syndrome is a fairly new entity that was formally recognized

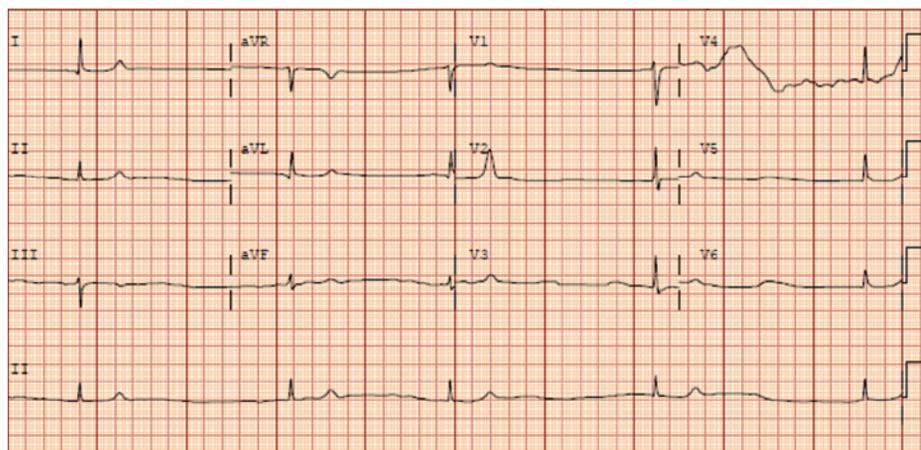


FIGURE 1. Initial EKG demonstrates junctional rhythm with severe bradycardia

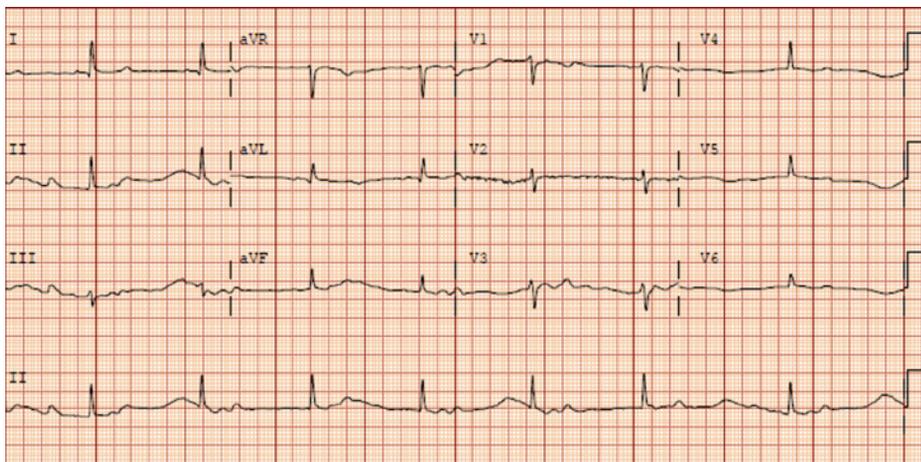


FIGURE 2. Repeat EKG after atropine; increased HR (though still bradycardic) with morphology unchanged

as a distinct disease process in 2016.¹ The acronym stands for: **B**radycardia, **R**enal failure, **AV**-nodal blockade, **S**hock, and **H**yperkalemia. The proposed pathophysiology centers on a cycle of hypoperfusion and bradycardia instigated by AV-nodal blockade and hyperkalemia.

BRASH Syndrome occurs in patients taking an AV-nodal blocking agent (commonly beta-blockers or calcium-channel blockers) who subsequently develop acute renal injury.¹ As the renal injury progresses, patients become hyperkalemic and the AV-nodal blocking agent begins to accumulate.¹ These two factors, hyperkalemia and AV-nodal blockade, act synergistically to cause bradycardia and hypotension.^{1,2} This leads to end organ hypoperfusion, which further worsens renal function.¹ And the vicious cycle continues. The presence of an angiotensin converting enzyme inhibitor or angiotensin II receptor blocker medication, which can also cause potassium retention and decrease GFR, potentiates the cycle.¹

The typical patient will be an elderly individual who clinically appears much better than expected for the degree of bradycardia.¹ However, patient presentations do range from asymptomatic to obtunded with multi-system organ failure.¹ The inciting event causing poor renal function may be dehydration from poor oral intake, increased output from illness such as enteritis, or any other cause of renal injury.

The diagnosis of BRASH

syndrome is complicated by the fact that patients often appear well with minimal symptoms and labs are often not particularly striking. BRASH syndrome patients often have only mild hyperkalemia (5.5-6.0 mmol/L) as opposed to the very high levels needed in pure hyperkalemia required to cause the same degree of bradycardia seen in BRASH syndrome.¹ This is due to the synergistic effects of mild hyperkalemia and AV-nodal blockade.¹ Similarly, EKG findings in BRASH syndrome typically show significant bradycardia without the classic stigmata of hyperkalemia (eg, peaked T waves, sine waves, etc.).^{1,3} Lastly, patients with BRASH syndrome are typically adherent to proper medication dosing and rarely have supra-therapeutic blood levels of AV-nodal blocking agents.^{1,4}

Treatment in the ED

The treatment of BRASH Syndrome prioritizes reduction of hyperkalemia, increasing heart rate, and addressing the underlying cause of acute kidney

injury.^{4,5,6} Standard treatment for hyperkalemia should be employed quickly, including IV calcium to stabilize the cardiac membrane, insulin with dextrose drips, high-dose albuterol, and bicarbonate to drive potassium into cells, as well as diuretics to expel potassium from the body.^{1,4,5,6,7} Calcium and albuterol have the added benefit of improving heart rate in these bradycardic patients. For those with profound bradycardia, epinephrine (first-line) or isoproterenol (second-line) can be used to increase heart rate if the patient is also hypotensive, otherwise dobutamine can be used if normotensive.^{1,4,5,6,7} Of note, atropine is often ineffective as it works at the AV node, which is blocked by the beta-blockers and calcium-channel blockers that instigate BRASH syndrome.^{4,5} Transcutaneous or transvenous pacing may be necessary, and at least two case reports document pacemaker placement.¹ Reestablishing adequate kidney perfusion is paramount, as this helps eliminate potassium from the body through kaliuresis and breaks the cycle of BRASH syndrome.^{1,4,5,6} This can be achieved by fluid resuscitation, especially given patients are often dehydrated, and pressor support if profoundly ill.^{4,6}

The precise prevalence of BRASH Syndrome remains unknown.⁵ However, if a physician encounters a patient with bradycardia on an AV-nodal blocking agent who does not respond to the standard ACLS guidelines for treatment of bradycardia, BRASH Syndrome should be considered.⁶ Although a rare condition, BRASH Syndrome can be lethal when not properly addressed. Therefore, it is essential to keep BRASH Syndrome in the differential diagnosis. ★

TAKE-HOME POINTS

- BRASH Syndrome is a constellation of **B**radycardia, **R**enal failure, **AV**-nodal blockade, **S**hock, and **H**yperkalemia.
- BRASH Syndrome is commonly triggered by an acute event (ie, dehydration) that results in kidney injury and hyperkalemia.
- Potassium levels in BRASH Syndrome are often only mildly elevated but act synergistically with AV-nodal blockade agents to cause hypotension and bradycardia.
- Treatment involves lowering potassium levels, improving renal function by treating the underlying cause, and increasing heart rate; however, atropine is often ineffective.

Kawasaki Disease vs. Multisystem Inflammatory Syndrome in Children (MIS-C)

Richmond M. Castillo, MD, MA, MS
Northwestern Memorial Hospital
@doctorbunso

Brad Goldberg, MD, FAA
Assistant Professor of Pediatrics
Children's Hospital Los Angeles
USC Keck School of Medicine
@BradGoldbergMD

Case 1. A 10-month old female with no past medical history presents to the Emergency Department (ED) with fever and rash. Her father notes fever for the past 5 days. Yesterday evening she developed a rash. Today she has decreased fluid and solid intake. Her father brought her to see the pediatrician who recommended an evaluation in the ED. Her vitals reveal a temperature of 38.0°C, a heart rate of 120 bpm, a blood pressure of 90/60 mmHg, a respiratory rate of 24, and a SPO₂ of 97% on room air. On exam the patient has conjunctival injection, red cracked lips, swollen hands/feet, and an erythematous papular rash to her back.

Case 2. A 9-year-old male presents to the ED with fever and abdominal pain. His mother reports the patient has had fever for 2 days and increasing abdominal pain. Today, the patient had three episodes of non-bloody, non-bilious emesis. The mother reports that he tested positive for SARS-CoV-2 three weeks ago. His vitals reveal a temperature of 38.7°C, a heart rate of 149 bpm, a blood pressure of 92/50 mmHg, a respiratory rate of 34, and a SPO₂ of 91% on room air. On exam the patient is very uncomfortable-appearing. The abdomen is noted to be soft but with guarding and tenderness to palpation in all quadrants. There was no rash present.

Epidemiology/Pathophysiology

Kawasaki Disease (KD) is a systemic vasculitis of unknown etiology. Approximately 80% of cases are in children less than 5 years old with peak incidence at 18-24 months of age. KD is rare in children less than 6 months of age.¹ Incidence is highest among Asians and Pacific Islanders.² Medium-sized

arteries are most affected, particularly the coronary arteries. Blood vessel damage appears to result from inflammatory cell infiltration into vascular tissue of unknown stimulus. This leads to disruption in the structural integrity of the arterial wall due to the loss of elastin and collagen fibers.³

Multisystem inflammatory syndrome in children (MIS-C) is a hyperinflammatory syndrome associated with SARS-CoV-2; 70% of MIS-C cases occur in patients greater than 5 years old with a peak incidence between 5 and 9 years of age. Hispanic/Latino and non-Hispanic Black patients account for approximately 70% of cases.⁴ Pathophysiology of MIS-C is not well understood and likely results from an abnormal immune response to a SARS-CoV-2 infection in the preceding weeks.⁵ The diagnosis may be challenging as some patients may have been asymptomatic during their primary SARS-CoV-2 infection.⁶ MIS-C shares clinical similarities with KD. However, based on literature review, MIS-C appears to have a different immunophenotype than KD.⁷

Signs and Symptoms

KD has a well described classic set of criteria used in establishing the diagnosis. In order to meet diagnostic criteria, patients must have a fever for 5 days or greater. Additionally, patients must meet at least four of the signs and symptoms listed in Table 1. These signs and symptoms usually do not present in a particular order. Clinicians should be aware and vigilant for the possibility of incomplete KD (iKD) which may be present despite patients not meeting at least 4 of the necessary signs and symptoms. The diagnosis of iKD requires laboratory data and often includes echocardiogram and expert consultation in aiding in the diagnosis. The most common cardiac manifestation is tachycardia out of proportion to the degree of fever.⁸ Blood pressures are mostly within normal limits. Rarely, patients may present in or develop

Kawasaki Disease Shock Syndrome in which hypotension is present.⁹

TABLE 1. Diagnostic Criteria for KD

The diagnosis of Kawasaki Disease requires the presence of fever for lasting at least 5 days with no other explanation, combined with at least 4 of the 5 following criteria:

- Bilateral bulbar conjunctival injection**
- Oral mucous membrane changes (injected or fissured lips, injected pharynx, strawberry tongue)**
- Peripheral extremity changes (erythema of palms or soles, edema of hands or feet, periungual desquamation)**
- Polymorphous rash**
- Cervical lymphadenopathy at least 1 lymph node > 1.5 cm in diameter**

The presentation of MIS-C is less straightforward as it may mimic other emergent conditions. Fever is universal in MIS-C patients as described in a recent systematic review of a cohort of 662 patients, however there is no defined fever length as is seen in KD at current.¹⁰ In the same systematic review, it was described that gastrointestinal symptoms are very common and seen in up to 73% of cases. Contrary to acute COVID-19, the majority of patients with MIS-C lack significant upper respiratory symptoms.¹¹ Other clinical signs and symptoms according to the recent systematic review can be found in Table 2. What is particularly notable in MIS-C is the high number of patients that present in shock or are initially stable only to rapidly decompensate in the ED or the inpatient setting.¹⁰

TABLE 2. MIS-C Clinical Signs/Symptoms
Adapted from Ahmed et al.¹⁰ (n=662)

SIGN/SYMPTOM	n (%)
Fever	662 (100)
Abdominal pain, diarrhea	488 (73.7)
Vomiting	452 (68.3)
Rash	372 (56.2)
Conjunctivitis	343 (51.8)
Cheilitis	213 (32.6)
Dyspnea, SOB	121 (18.3)
Cough	86 (13.0)
Rhinorrhea, congestion	47 (7.1)

Initial Workup & Evaluation

KD is a clinical diagnosis, with labwork when the patient does not meet the initial diagnostic criteria. This workup includes complete blood count with differential, ESR, CRP, LFT, albumin and urinalysis with microscopy. iKD is associated with elevated ESR/CRP, leukocytosis, anemia, thrombocytosis, sterile pyuria, elevated aspartate transaminase (AST), and hypoalbuminemia.

Diagnosis of MIS-C is more difficult, and ED providers should have a high index of suspicion in the ongoing COVID-19 era. Initial workup of MIS-C should be similar to that of patients with sepsis. MIS-C and sepsis are often indistinguishable in the ED setting. This includes blood cultures, assessment for end-organ dysfunction, and labs to assess for a hyperinflammatory state. A suggested work-up can be seen in Table 3.

TABLE 3. MIS-C Suggested Work-up

CATEGORY	TEST
Point of care labs	VBG, lactate, glucose
Basics	CBC, BMP, LFT, PT/PTT
Inflammatory labs	LDH, ESR, CRP, Ferritin, D-dimer, fibrinogen, procalcitonin
Specimen	UA with microscopy
Cultures	Blood, urine
Cardiac labs	Troponin, BNP
Cardiac adjuncts	EKG, echo
Imaging adjuncts	CXR, POCUS

Additionally, patients should have a baseline ECG and echo; POCUS can be helpful in providing quick estimates of both cardiac function and responsiveness to fluid resuscitation. Of note, some MIS-C patients may be fluid non-responsive and serial POCUS exams may help anticipate the need for vasopressor support. Imaging studies including CXR in the presence of respiratory distress can help rule out other sources of infection. Given that a large number of patients present with severe gastrointestinal symptoms, a formal ultrasound and/or CT scan of the abdomen/pelvis may be necessary to rule out other common diagnoses such as appendicitis.

Treatment and Disposition

Treatment for patients with KD includes intravenous immune globulin

(IVIG) and aspirin (ASA). A commonly dosed regimen for IVIG is 2 g/kg.^{12,13} For ASA, initial and maintenance dosing regimens vary across institutions.¹⁴ It is important to note that treatment should align with established hospital protocols. Most KD patients are appropriate for admission to the pediatric floor. If there is evidence of shock, such as seen in Kawasaki Disease Shock Syndrome, the PICU may be more appropriate. There are institutional variations in the consultants involved in the care of KD patients. Usually at a minimum, cardiology is needed for echocardiography to evaluate the coronary arteries. Some institutions may involve ID and rheumatology.

For MIS-C, treatment in the ED should include judicious fluid administration while serially reevaluating for responsiveness or evidence of worsened cardiac function. Additionally, given sepsis cannot be excluded, empiric broad-spectrum antibiotics are recommended in ill-appearing patients. Many patients may require some oxygen supplementation and vasopressor support.¹⁰ It is important to note that management of MIS-C requires a multidisciplinary team.¹⁵ When MIS-C is suspected in the ED, PICU and cardiology consults are generally indicated. Approximately 60-70% of MIS-C patients require intensive care, so involve PICU early.^{10,16} Generally, the ID is consulted when MIS-C is suspected. Depending on the institution, hematology, rheumatology, and allergy/immunology may be a part of the care team. For patients with severe cardiac dysfunction, activation of an ECMO team may be necessary. Patient disposition should be considered carefully. Medical centers without the multidisciplinary services listed above should consider early transfer to a regional pediatric center with access to all these services. The decision to admit to the pediatric floor vs. PICU should be made using similar indications to other disease processes. However, consultation with the PICU is reasonable for all patients being admitted with suspected MIS-C, given the high rates of rapid decompensation.¹⁰ With early resuscitation and close monitoring, patients with MIS-C tend to do well. According to recent systematic reviews, mortality is estimated at 1.7-1.8%.^{10,16} Future research

and following patients prospectively will be necessary in determining any long-term sequelae of MIS-C.

Case 1 Conclusion

You are confident in the diagnosis of KD. Upon consult, ID recommends administration of IVIG. You begin an IVIG infusion and admit to the general pediatric floor service. High dose aspirin was started on admission. On the wards, an echocardiogram demonstrates normal coronary arteries and an EF of 66%. No other significant findings were noted. The patient was discharged on hospital day 3 on daily aspirin with plans to follow up the primary care pediatrician and cardiology.

Case 2 Conclusion

*Sepsis protocol was initiated. The patient was placed on 4 L oxygen via nasal cannula and saturations improved to 97%. IV access, labs, urine, and blood cultures were obtained. A fluid bolus was given. Ceftriaxone and vancomycin were given to cover for bacterial sepsis. Metronidazole was added as a perforated appendix was considered in the initial differential. A second bolus was given for ongoing tachycardia. A cardiac POCUS showed an IVC without collapsibility. A central line was placed to deliver a vasopressor. An ECG demonstrated sinus tachycardia. CBC noted leukocytosis; VBG was normal; however, lactate was elevated. CRP, Ferritin, D-dimer, fibrinogen were all abnormal. High-sensitivity troponin was normal with a mildly elevated BNP. Blood chemistries were significant for normal creatinine and GFR. Small elevations in AST and ALT were present. PT and PTT were normal. CT of the abdomen and pelvis revealed hepatomegaly and hepatic inflammation, plus multifocal bilateral renal lesions concerning for possible pyelonephritis. The patient was admitted to the PICU for IVIG, steroids, prophylactic anticoagulation, and continued vasopressor support. Formal echo showed an EF of 45% without abnormalities of coronary arteries. ID, hematology, rheumatology, allergy/immunology were consulted. After 6 days in PICU followed by floor, the patient was discharged with appropriate follow-up. **

An Unusual Cause of Tachycardia

Focal Nonconvulsive Status Epilepticus

Following Acute Head Trauma

Bruce J. Grattan Jr., DO, MS, RD

Department of Emergency Medicine
Bon Secours Mercy/
St. Vincent Medical Center

John Leskovan, DO, FACS, FACOS

Department of Trauma Surgery
Bon Secours Mercy/
St. Vincent Medical Center

Background

Nonconvulsive status epilepticus (NCSE) is defined as a period of ongoing seizure activity which may present as an alteration in behavior or mental status, albeit in the absence of motor activity.¹ There are two recognized classifications of NCSE including absence status epilepticus (primary generalized) and complex partial (secondary generalized). NCSE has, in years past, been considered a rare condition. This is in large part the result of the fact that there have been very limited data from which to accurately describe the incidence of NCSE. Historically, most data have been drawn from single center studies with small sample sizes.² Nevertheless, the incidence has been estimated to be 15-20/100,000 cases per year.³ These values may underestimate the true incidence. In one study of patients who presented to the ED with altered mental status (AMS) and without convulsions who received EEG studies, 37% were found to have NCSE.⁴ Notably, nearly half of patients with NCSE will not have any history of epilepsy.⁵ While in years past NCSE was thought to have an incidence of 3%, subsequent studies have shown it to be more common ranging from 16-43% of all status epilepticus cases.⁶⁻⁹ This is notable as the morbidity and mortality of NCSE is estimated to be 39 and 18% respectively.¹

Nonconvulsive status epilepticus has traditionally been thought of as a rare condition. There is little data regarding its true incidence, but evidence suggests it is far more common than once believed. Nonconvulsive status epilepticus can have an array of presentations and can be a diagnostic enigma for those unaware of its existence or those who believe, as it was once taught, that this condition is isolated to critically ill patients in the intensive care unit. Herein, we discuss the case of a patient with a focal traumatic brain injury who was found to be in nonconvulsive status epilepticus. This condition is one that physicians need to consider, especially those physicians caring for patients with traumatic brain injuries.

Case

A 49-year-old male was brought to the ED by emergency medical services (EMS) after being found down outside. The patient was intoxicated (ethanol level of 459 mg/dL) and was unable to recall the preceding events but alleges he was struck in the head with a bottle. The patient's chief complaint was rib pain and headache. GCS was 15 and vitals were blood pressures of 116/74, heart rate of 60/min, temperature 97.4°, respiratory rate 14/minute, SPO₂ 100% on room air. The patient had no significant medical history and specifically no history of seizures. He reported drinking alcohol and smoking tobacco and marijuana daily. On physical examination, there was tenderness to palpation of the left chest wall and generalized abdominal tenderness. The patient underwent computed tomography (CT) imaging of his head, cervical spine, chest, abdomen, and pelvis, with findings of a traumatic subarachnoid hemorrhage in the right frontotemporal region (Hess and Hunt 3, modified Fischer grade 1) with a hyperdensity along the temporal operculum in a gyral pattern.

Given these findings and concern for acute alcohol withdrawal, the patient was admitted to the intensive care unit (ICU) for continued monitoring. He was started on levetiracetam 500 mg po every 12

hrs for 7 days. A follow-up CT brain was stable in appearance, and on hospital day (HD) 2 the patient developed multiple intermittent and random episodes of sinus tachycardia with a rate of 130s-150s. These episodes lasted seconds to minutes, during which the patient was without any hemodynamic compromise and he remained asymptomatic. A 12-lead ECG was obtained just after an episode, which showed a sinus rhythm at a rate of 92 beats/min, with normal axis and without any acute changes. The QT-corrected was noted to be prolonged at 497 msec, and 2 g IV magnesium sulfate was given. After an unremarkable workup for causes of sinus tachycardia, Cardiology recommended metoprolol tartrate 12.5 mg PO every 12 hrs.

On the evening of HD 2, the patient had repeated episodes of sinus tachycardia now associated with upward gaze and tonic-clonic hand motions concerning for seizures. Surprisingly, throughout these episodes the patient was interactive and cooperative, without any confusion or postictal state. The patient was placed on long term monitoring for epilepsy (LTME). 21 EEG electrodes were placed according to the International 10/20 System. A single EKG electrode was also placed. Video recording was time-locked with EEG recording.

The EEG was interpreted by a board certified epileptologist. Upon EEG initiation, the background was noted to demonstrate a continuous slow rhythm largely in the delta frequency of 1-3Hz, ranging between 10-50uV. There was no posterior dominant rhythm and no eye opening/closing artifacts. Spontaneous variability was present. The interictal rhythm was continuous, slow, generalized and lateralized to right. At times there was evidence of asymmetry, there were faster and slower activities on the right, particularly in the right frontal/temporal region when compared to the left side. During the patient's continuous video EEG monitoring, EEG evidence of seizures in the right hemisphere were captured from the beginning of the recording without any clear clinical signs. There was also evidence of diffuse encephalopathy and structural abnormality of the right hemisphere, consistent with the patient's known right SAH and hemorrhagic contusion. The patient was in focal EEG status since the initiation of the recording while the patient exhibited no clinical signs of seizure. Hyperventilation and photic stimulation were not performed. Single lead EKG showed regular, heart rate at 60 per minute. As a result of these findings, 3 g levetiracetam and 1 mg lorazepam IV were given followed by 1 g levetiracetam PO every 12 hrs. Following the initial bolus, there was no recurrence of seizure.

Nonconvulsive status epilepticus can have an array of presentations and can be a diagnostic enigma for those unaware of its existence, that this condition is isolated to critically ill patients in the intensive care unit.

Discussion

There are a variety of known etiologies of NCSE including electrolyte abnormalities, hypoxic-ischemic encephalopathy, traumatic brain injury (TBI) and acute hormonal disturbances.¹⁰⁻¹¹ Nearly 75% of patients with NCSE have no identifiable changes on physical examination other than a decrease in responsiveness,¹²⁻¹³ which can often readily be attributed to another etiology. NCSE can present with negative symptoms including confusion and lethargy or subtle and often overlooked positive symptoms including blinking, nystagmus, facial twitching or tremulousness. In a small study of 48 patients, abnormal ocular movements were shown to be a specific clinical finding in NCSE as compared with patients without NCSE.¹⁴ While much of the data on NCSE is drawn from populations of patients who are acutely ill or comatose, these data suggest that patients with NCSE may have a worse prognosis as compared with convulsive status epilepticus.^{15,16} Similarly, it has been demonstrated that up to 14 percent of patients treated for convulsive status

epilepticus persist in NCSE upon initiation of EEG monitoring.¹⁵ In contrast to critically ill patients in the ICU setting, NCSE outside the ICU can present with a plethora of subtle clinical manifestations.¹⁷ NCSE can be difficult to distinguish from absence seizure,¹⁸ with EEG being critical to the diagnosis where 50% of nonconvulsive seizures are identified within the first hour of EEG initiation.¹⁹

The scarcity of data regarding NCSE extends to treatment with very little literature being available regarding the most efficacious treatment modalities and without any randomized trials providing evidence to support treatment recommendations.²⁰ NCSE management is not addressed in the status epilepticus guidelines published by the Neurocritical Care Society,²¹ resulting in much variation in care and even recognition of this condition amongst clinicians.

NCSE and Traumatic Brain Injury

Among TBI patients, seizures are not uncommon and are an important component of prognosis. Nearly 25% of patients with traumatic brain injuries who have a seizure in the first week after injury, will progress to have subsequent seizures.²² The development of epilepsy after TBI has a broad incidence ranging from 4.4-53% depending on the population studied.²³ In a retrospective analysis of 451 adult TBI patients 9.3% had EEG evidence of electrographic status epilepticus with 19% being associated with intracerebral hemorrhage including trauma.²⁴⁻²⁵ NCSE may be particularly harmful among patients with TBI. As animal data suggests, the acutely injured brain has greater sensitivity to damage posed by NCSE.²⁶⁻²⁷

Conclusion

This case exemplifies an anomalous cause of sinus tachycardia. As described, patients with TBI are at high risk for seizures, including those who may present without typical clinical motor findings. This unique case of focal status epilepticus related to acute head trauma highlights the need for physicians to be cognizant of this condition and to have a high clinical suspicion for NCSE in patients with TBI who are presenting with disparate signs and symptoms. ★



Initial CT revealed traumatic subarachnoid hemorrhage in the right frontotemporal region with a hyper-density along the temporal operculum in a gyral pattern

Acute Abdominal Pain in a Child

Carolina Vega, MD

University of Massachusetts
PEM Attending Physician

Jennifer Noble, MD

Children's Hospital of Michigan
Central Michigan University

Jayashree Rao, MD

Children's Hospital of Michigan
Central Michigan University

Nirupama Kannikeswaran, MD

Children's Hospital of Michigan
Central Michigan University

We present the case of a 2-year-old male who was evaluated in the pediatric emergency department for acute onset, severe abdominal pain. Point-of-care ultrasound demonstrated a large intraabdominal cystic mass at the level of the umbilicus, which was intraoperatively determined to be a fluid-filled Meckel's diverticulum, with an associated partial small bowel obstruction.

Case

A 2-year-old male presented to the pediatric emergency department (ED) for severe abdominal pain that began one hour prior to presentation. Onset was acute, with the patient beginning to complain of pain while eating dinner, with localization to the right side of his abdomen. Review of systems was positive for increased urinary frequency on the day of presentation, and lack of bowel movements for 3 days. The family reported that he had been otherwise well. There was no associated fever, cough, congestion, polydipsia, vomiting, diarrhea, dysuria, hematuria, or rash. The family denied any preceding falls or other trauma. There were no known sick contacts, and the patient was fully immunized.

Vital signs at presentation to the ED demonstrated a temperature of 36.4°C, HR 100/min, RR 32/min, and BP 123/73 mmHg. The patient was alert and reclining on the gurney. Abdominal exam was significant for generalized moderate tenderness. Severe tenderness was localized to the periumbilical abdomen and right lower quadrant, with associated involuntary guarding. Bowel sounds were normal, and there was no abdominal distention or palpable hepatosplenomegaly. Genitourinary

exam was unremarkable with no signs of inguinal hernia, testicular enlargement, or tenderness.

The initial differential diagnosis included intussusception, ileus, constipation, and appendicitis. An abdominal x-ray was obtained which showed a mildly prominent small bowel loop in the right mid abdomen, and no signs of free air or abnormal bowel gas pattern (Figure 1).

A radiology-performed ultrasound revealed a complex cystic structure with fluid levels and internal debris near the umbilicus, with a diameter of 4.6 cm. Point-of-care ultrasound by a third-year pediatric emergency fellow was performed to demonstrate the findings to the family and optimize understanding. A large, round, avascular mass was visualized in the medial abdomen, extending inferiorly from the level of the umbilicus. The contents of the mass were grossly anechoic, suggesting fluid, with hyperechoic elements on the internal walls, and there was posterior acoustic enhancement (Figure 2, 3). The liver, spleen, kidneys, and bladder were visualized and were normal.

Laboratory evaluation included a complete blood cell count, C-reactive



FIGURE 1. Small Bowel Loop
Abdominal radiograph: Mildly prominent small bowel loop in the right mid abdomen. No signs of free air or air-fluid levels.

protein, and comprehensive metabolic panel that were unremarkable. Rapid testing for COVID-19, influenza, and respiratory syncytial virus was negative.

The patient was admitted to Pediatric Surgery and underwent surgical resection of the mass. Intraoperatively, the mass was found to be a fluid-filled Meckel's diverticulum with a stalk to the mesentery of the terminal ileum. There was a partial small bowel obstruction proximal to the diverticulum, secondary to adhesions of the serosa to the small bowel. The affected bowel was resected from proximal to distal to the Meckel's diverticulum, and end-to-end anastomosis was completed.

Intravenous ceftriaxone and metronidazole were administered as surgical prophylaxis for 2 days. The patient was discharged on postoperative day 4, at which time his pain was significantly improved, he was ambulating independently, and he was tolerating an oral diet.

Ultrasound Findings

Point-of-care ultrasound revealed a large, round mass in the infraumbilical abdomen. In the transverse and sagittal views, an elliptical mass measuring 3.6 x 4.7 x 4.2 cm was visualized. The walls of the structure were thin and layered in appearance. The internal walls of the structure were hyperechoic with hyperechoic projections to the interior, and the surrounding wall was hypoechoic. The internal contents were grossly anechoic, suggestive of fluid, and the overall structure demonstrated posterior acoustic enhancement (Figure 2, 3). Using color Doppler, vascular flow was visualized to the surrounding tissue, and there was absent vascular flow within the mass.

Ultrasound Technique

Point-of-care ultrasound for pediatric abdominal evaluation is performed using the low frequency curvilinear and phased array transducers.^{1,2} The linear transducer includes higher frequencies and is preferred when evaluating superficial structures due to improved resolution.^{1,3,4} The curvilinear transducer is favored in patients of larger habitus and for structures at greater depths due to the transducer's larger footprint and optimal tissue penetration.¹⁻³ When

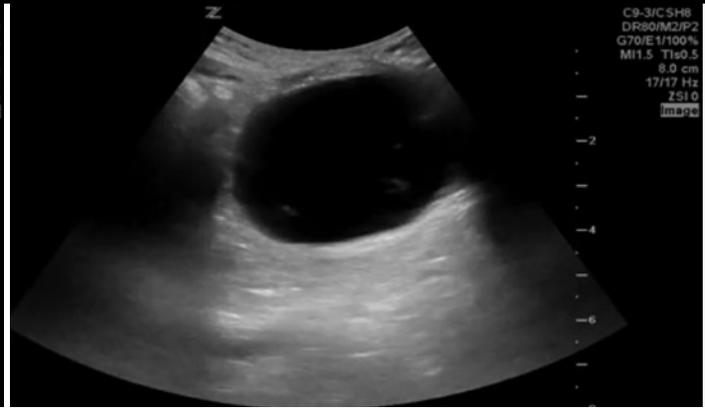
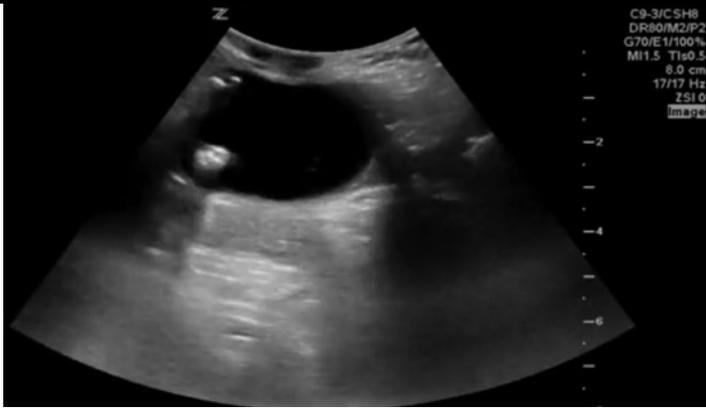


FIGURE 2. Abdominal Mass

Point-of-care ultrasound image: Transverse view demonstrating an elliptical structure with thin, hypoechoic, outer walls. There are hyperechoic projections from the internal wall, and the structure has posterior acoustic enhancement.

FIGURE 3. Abdominal Mass

Point-of-care ultrasound image: Sagittal view demonstrating the elliptical mass with hypoechoic outer walls. The hyperechoic internal projections are less visible in this view.

evaluating the abdomen for pathology, the exam should start at the site of greatest pain or tenderness.^{4,5} For inspection of the target area, water-based gel is applied and the minimum pressure needed for visualization is utilized to maximize patient comfort.^{1,3,4}

Graded compression is the recommended technique for evaluation of the bowels and lower abdomen for pathology. This technique displaces bowel gas and improves visualization of the evaluated intraabdominal structures.^{1,3-5} When an abnormality is visualized, the transducer should be rotated 90 degrees to evaluate the area in 2 planes.^{2,4,6} Any fluid-filled structures may demonstrate posterior acoustic enhancement.^{2,3}

Ultrasound can optimally be used to reveal the Meckel's diverticulum as a blind outpouching of bowel projecting from the small intestine.^{7,8} The appearance of Meckel's diverticulum varies based on whether the structure is inflamed. In both cases, the diverticulum may contain visible fluid and gas.^{7,9} A gut signature, which refers to the multilayer appearance of bowel, is often appreciated.^{5,7,9,10} Ultrasound can be used to visualize the bowel layers surrounding the fluid-filled contents: a hyperechoic inner layer that is often irregular and represents the mucosa and submucosa, a hypoechoic intermediate muscular layer, and a hyperechoic outer serosal layer.^{8,10} Color Doppler should be used during the evaluation, as when the diverticulum is inflamed, the structure's walls appear thickened and hyperemic in comparison to normal bowel wall.^{5,7,9,10}

Discussion

Meckel's diverticulum is a true diverticulum and is a congenital remnant of the omphalomesenteric duct that is present in 2% of the general population. The structure is present in the ileum, 40 to 100 cm proximal to the ileocecal valve. Its diameter is typically reported as approximately 2 cm, however cases have been described with diameters up to 10 cm.^{8,11} The majority of these diverticula are asymptomatic, with 4.2 to 9% of patients with Meckel's diverticulum developing symptoms in their lifetime.¹² 46.7% of children present with small bowel obstruction as our patient did, including cases secondary to volvulus or intussusception, compared to 25.3% who present with painless rectal bleeding, and 19.5% who present with diverticulitis.^{7,8,11,12} Complications of Meckel's diverticulum can present at any age, however they more commonly present in pediatric patients, with the median age ranging from 4-5 yrs.¹²

Point-of-care ultrasound has been described as a viable first line imaging modality used for detection of Meckel's diverticulum in symptomatic patients.^{5,9} The presentation of appendicitis and symptomatic Meckel's diverticulum can be similar, in particular with cases of diverticulum-associated obstruction and inflammation, which may present with fever, nausea, vomiting, and right lower quadrant pain.^{5,6,9} Like appendicitis, focal pain for a symptomatic Meckel's diverticulum may also be reported as periumbilical.^{5,13}

The sonographic commonalities of the appendix and Meckel's diverticula can further hinder differentiation, including the gut signature created by the multiple bowel layers, as well as the non-compressibility, thickened hyperemic walls, and lack of peristalsis appreciated in both appendicitis and inflamed Meckel's diverticula.^{5,6,8,9} However, visualization of the normal compressible appendix with point-of-care ultrasound assists with narrowing the differential, when considering small bowel abnormalities such as Meckel's diverticulum.⁵ Additionally, Meckel's diverticulum is typically larger than the non-compressible structure seen with appendicitis and is located in a different site. Meckel's diverticulum is approximately 2 cm in diameter by 5 cm in length and is located a greater distance from the cecum than the appendix. In comparison, the appendix is attached directly to the cecum, and the defining diameter of appendicitis is greater than 0.6 cm.^{5,8,13} As in our case, the Meckel's diverticulum can sometimes be visualized in the area of the umbilicus, or it may be visible in the lower abdomen or right lower quadrant.^{5,6,9,10}

Conclusion

Point-of-care ultrasound can be used to detect Meckel's diverticulum as the underlying etiology of lower abdominal pain in pediatric patients. In our case, bedside family-centered teaching and demonstration of the findings was a direct benefit of point-of-care ultrasound. Expedited management is an associated potential benefit of point-of-care ultrasound to evaluate abdominal pain by experienced providers. ★

In a Heartbeat Septic Cardiomyopathy



Lindsay Davis, DO, MPH, NRP

Central Michigan University

Bryce Taylor, DO, MS

Carilion Clinic – Virginia Tech Carilion
School of Medicine

Sepsis cardiomyopathy (colloquially known as septic heart) was first described by Parker et al in 1984. They described it as a rapid onset decreased left ventricular ejection fraction (LVEF) and an increased end diastolic volume (EDV) in patients with severe sepsis that lasted several days, followed by a full cardiac recovery in patients that survive.¹ Currently, there is a lack of formal criteria but there are clinical signs that are generally accepted to be a sign of a septic heart. Patients in septic shock have myriads of morbidity, and awareness of septic cardiomyopathy as a contributor to their hemodynamic stability may frequently be essential in improving outcomes in many critical cases.²

Epidemiology

Epidemiologic data is sparse. Two studies, one in Japan and one in Korea both reported similar risk factors for sepsis induced cardiomyopathy. The one in Japan, showed that sepsis induced cardiomyopathy is more common in males than females, younger age, higher lactate on admission, and in patients with a history of heart failure.³ The study by Jeong and colleagues also reported younger age and a history of heart failure, but reported additional risk factors of a history of diabetes melitus, elevated NT pro-BNP, positive blood culture.⁴ Incidence varies but studies report between 20-60% incidence in

the first few days of ICU admission.⁵ Further research is needed on more diverse populations and broader global scale to better understand the epidemiology and implications to patients.

Pathophysiology

The mechanism behind sepsis induced cardiomyopathy is still without a clear definition in the literature. It is thought to be a consequence of myocardial ischemia that results from inadequate blood flow and activation of the immune system via chemical mediators such as endotoxins, cytokines, and nitric oxide. Myocardial hypoperfusion was once thought to be a major cause but now it is thought that this plays a role more in the microvascular setting rather than macrovascular.⁶ The activation of the innate immune system probably is a result of many different mechanisms synergizing each other. Inflammatory mediators set off the coagulation cascade and mitochondrial dysfunction causes oxidative stress. An increase in endothelial permeability leads to edema (myocardial edema may cause contractility dysfunction), causing an increase in neutrophil trafficking into interstitium and fibrin distribution. Disruptions in calcium response cause increased troponin I due to decreased calcium sensitivity. Subsequent catecholamine toxicity causes increased sympathetic outflow. This systemic inflammation all leads to widespread vascular dilation, hypotension and tachycardia.⁷

The hallmark of septic cardiomyopathy is its apparent reversibility. Studies show patients recovering full cardiac function after resolve of sepsis within 7-10 days.³ The cardiac MRI shows patterns indicating altered metabolism and structural edema rather than ischemia and infarction. This includes a dilation of the left ventricle and global ventricular dysfunction without regional dysfunction. Thus sepsis-induced cardiomyopathy may be a protective mechanism leading to decreased mortality in those with hypokinetic heart than hyperkinetic heart. Septic cardiomyopathy is a global kinetic dysfunction as opposed to Takotsubo, which is a more regional kinetic dysfunction (leads to ballooning morphology on echo).²

Clinical Presentation and Diagnosis

The septic heart presents with a reversible cardiomyopathy in the setting of severe sepsis. While there are no standardized clinical criteria at this time, sepsis induced cardiomyopathy is generally defined as an ejection fraction less than 50% and with greater than or equal to a ten percent decrease in LVEF compared to the patient's baseline.^{3,4} This includes cardiogenic shock with a systolic blood pressure less than 90, urine output less than 30 mL/hr and normal or elevated left ventricular filling pressure.⁸ Patients will present with signs of severe sepsis such as hypotension, tachycardia, increased white blood cells and confirmed or suspected infection. Additionally, the cardiac presentation will show consistent

signs of decreased LVEF, left ventricular dilatation with normal or decreased filling pressure, depressed ejection fraction, and improvement within 7-10 days.² The onset of cardiomyopathy in the setting of sepsis is rapid but reversible following decompensation into septic shock. Future research for diagnosis should focus on creating standardized clinical criteria.

Standard critical care monitoring should be performed including cardiac monitor, vitals and central venous pressure (CVP). CVP monitoring with a goal pressure of < 8 mmHg. The lowest mortality was seen in patients with a pressure < 8 mmHg and the highest mortality with a CVP > 12 mmHg.⁹ It is important to differentiate between left ventricular diastolic and systolic dysfunction, diastolic dysfunction on echocardiogram poorly categorizes septic patients compared to systolic dysfunction.¹⁰

Echocardiogram is the most valuable diagnostic tool in identifying septic cardiomyopathy and has become widely available with the adoption of point of care ultrasound in emergency departments and intensive care units. Larger scale studies are needed on serial and longitudinal echocardiography for diagnosis and prognosis. It is important to exclude other causes of cardiomyopathy that can include ischemic cardiomyopathy and sepsis-induced Takotsubo cardiomyopathy.⁵ Worsened outcomes have been seen in septic patients with hyperkinetic cardiac activity. Patients believed to have septic cardiomyopathy tend to be hypokinetic or normokinetic with global dysfunction.² Echocardiographic speckle should be used to evaluate for decreased contractility regardless of LVEF. Increasing levels of strain found with echocardiographic speckle correlated with worsened outcomes even in patients with normal or pseudonormal LVEF.¹¹ ECG shows no diagnostic findings, most common rhythms include sinus tachycardia and atrial fibrillation. If you find abnormalities on ECG further cardiac evaluation should be performed.¹¹ Coronary angiography should be performed to rule out takotsubo cardiomyopathy.² No data was found on

presentation on chest x-ray in patients with a septic heart.

Important laboratory studies to obtain are brain natriuretic peptide (BNP), troponin, and coagulation studies. At this time, none of these are diagnostic of septic cardiomyopathy. BNP frequently rises but should not be used as a predictive value. Troponin frequently rises as well, and some studies showed that a rise in troponin had a positive correlation with mortality.¹² It should be emphasized that troponin levels are nonspecific in septic cardiomyopathy, as septic patients frequently have comorbidities that can lead to rise in troponin such as renal injury and acute coronary syndrome. Coagulation panels can indicate the presence of coagulopathies or disseminated intravascular coagulation, which may be significant as one possible contributor to septic cardiomyopathy is microvascular aberrancy.¹³ Adherence to your institution's evidence-based sepsis workup protocol regarding labs is of utmost importance in all septic patients.

Management

Overall treatment is similar to the treatment of sepsis alone. This includes early goal directed therapy with early and aggressive treatment for sepsis. Treat sepsis by chasing and eliminating the source while also managing the hemodynamics. Managing hemodynamics includes use of vasopressor therapy.² Address and attempt to prevent other comorbidities that may happen which may include shock liver, multiple organ failure and acute kidney injury. Intubation if indicated and antibiotics as per your facilities sepsis protocol. Patients will need fluids as with typical sepsis, but beware that there is worse mortality associated with positive fluid balance and elevated CVP. Approach fluid resuscitation in order to modulate afterload and optimize heart rate.¹⁰

Studies show varying suggestions and benefits of vasopressors. Nickson et al, suggest the benefits are uncertain.⁵ Dobutamine may have adverse effects in patients with sepsis such as arrhythmias, and is suggested to be avoided.² Noradrenaline is the first line, can add vasopressin but vasopressin is not recommended as a single agent. These vasopressor drugs are already a part of the guidelines for treating septic shock.¹⁴ Many of these vasopressors (dobutamine, dopamine, epinephrine, and norepinephrine) increase inotropy but are more likely to be arrhythmogenic.⁸ Depending on severity consider circulatory support such as balloon pump and ECMO. Balloon pump increases cardiac output and reduces the dosage of vasopressors needed, prolongs survival time. Internationally, Levosimendan is suggested due to its inotropic effects and lack of causing arrhythmias but this drug is not available in the U.S.²

Prognosis

Sepsis has a high mortality, but a septic heart is generally reversible and may be associated with a decrease in death as long as the patient is not hyperkinetic per Boyd et al.⁹ But an opposing study by Ehrman and colleagues' states that mortality may be two to three times greater.⁶ L'Heureax and colleagues agreed with Ehrman concluding that one year survival was decreased when isolated right ventricular dysfunction was found in sepsis. Additionally, L'Heureax concluded that echocardiographic assessment to guide hemodynamic management and to stop vasopressors earlier lead to decreased 28-day mortality.¹¹ Further studies to replicate and understand the outcome are needed. Early management of hemodynamics and source elimination is important to improve prognosis. Future studies into the rapidity of onset and how this relates to outcome are needed. ★

CLINICAL PEARLS

- ✓ Always perform an echocardiogram and look for the triad of decreased LVEF, increased EDV, and resolution within 7-10 days.
- ✓ Use Noradrenaline as the first line vasopressor if needed.
- ✓ Consider ECMO or balloon pump to reduce vasopressor use.
- ✓ Don't fluid overload your patient.

Euglycemic DKA and Hyponatremia Caused by a Commonly Prescribed Diabetic Medication

Aaron Tiffie, MD, FACEP

Our Lady of Lourdes

Daniel Stout, MD, FACEP

Taylor Curry

We present a case of a 49-year-old man with diabetes who had sudden onset confusion and profound hyponatremia with no obvious cause. Further investigation showed his diabetes medication as the culprit of his electrolyte imbalance and altered mental status.

According to the American Diabetes Association, about 1 in 10 Americans are affected by diabetes. Treatment has advanced significantly since the first case of the disease was noted in the 16th century. One of the most recent advancements in treatment is the use sodium glucose co-transporter 2 (SGLT-2) inhibitors, which have rapidly increased in popularity among prescribers since their FDA-approval in 2013. Canagliflozin, dapagliflozin, and empagliflozin have been approved for use in treating type 2 diabetes in the United States. The primary mechanism of these drugs is to promote glycosuria through the kidneys, effectively lowering the amount of glucose in the body. The drug target is SGLT-2 in the proximal tubule of the kidney. This transporter is responsible for up to 90% of the glucose uptake in the kidneys, therefore it is a reasonable and effective target in decreasing blood glucose. Although SGLT-2 inhibitors are known for cardioprotective effects, promotion of weight loss, reduction of blood pressure, and elevation of HDL, no drug is without adverse effects. Well known side effects of these drugs include acute kidney injury, genitourinary infections, and hypotension. One adverse effect of SGLT-2 inhibitors, though rare, is euglycemic diabetic ketoacidosis. An even less known complication is acute hyponatremia. The mechanism of the latter is not completely understood, but is believed to correlate

with the significant volume loss that occurs with these agents, resulting in production of antidiuretic hormone.

Case

A 49-year-old male with a history of type 2 diabetes mellitus treated with empagliflozin and sitagliptin-metformin was brought to the ED with the development of acute confusion noticed by his coworkers. According to his wife, he was in his usual state of health when he left for work that morning. The patient was unable to remember his wife's name, recall his home address, and was unable to state where he was. He seemed unbothered by this deficit. The patient was unable to contribute any additional information to his HPI due to his altered mental status. His wife and coworkers denied any recent head trauma, accidents, history of drug abuse, renal disease, liver disease, or shortness of breath. His medical history besides type 2 diabetes mellitus included COPD, hypertension, and hyperlipidemia.

CT of his head and neck revealed obstruction of the left carotid artery which was felt to be chronic and not causative of his current symptoms. He was found to have a severe hyponatremia of 115 and was noted to be in euglycemic ketoacidosis with a serum glucose of 100 and anion gap of 24. Urine ketones were positive. His home diabetes medications were held and the patient was started on sliding scale insulin for glycemic control. Urine toxicology was negative. Blood alcohol was noted to be 80 mg/dL. His wife stated he did not have anything to drink before he left for work that day, but has a history of consuming beer in social settings. Serum osmolar gap was 22, likely secondary to alcohol. Due to suspicion of ethylene glycol or methanol ingestion, the patient was treated empirically with fomepizole, although the levels of both later came back negative. Thyroid function and cortisol

levels were within normal limits. Urine osmolarity was 487 and urine sodium was 99, though this was measured after infusion of hypertonic saline.

On day 4 of his hospital stay, he was noted to have returned to his baseline mental status, confirmed by his wife. His sodium level at that time was 131. He stated that about two months prior, he had lab work done by his primary care physician, noting odium in the "high 120s." He was observed for an additional night, remained stable, and was discharged on day 5 of his stay. Over the course of his 5-day hospital stay, the cause of his hyponatremia was ultimately linked to one of his diabetes medications, empagliflozin, an SGLT-2 inhibitor with a rare side effect of euglycemic DKA and an even more rare side effect of hyponatremia, complicated by alcohol use. He was advised to stop taking the empagliflozin but to continue his sitagliptin-metformin for management of his diabetes.

Conclusion

SGLT-2 inhibitors can cause massive osmotic diuresis and intravascular volume depletion. The body's appropriate response to this is production of antidiuretic hormone, an excess of which is capable of causing hyponatremia, as demonstrated by our case. Over the past decade, the number of new agents for controlling glucose in patients with type 2 diabetes has increased dramatically. These agents have proven to be protective to the cardiovascular system and have helped with weight loss and control of blood pressure. It is important for physicians to recognize the rare but life-threatening complications of these agents as they are being increasingly prescribed. A literature search revealed only one other case of acute hyponatremia in the setting of euglycemic DKA, therefore we felt it was necessary to share this case. ★



James Nardini, MS, NRP

Chicago Medical School
at Rosalind Franklin University

STOP THE BLEED

Why It Matters Now

The other day, I woke up to start studying for my USMLE examination, only to find I had run out of coffee the day before. I let out a sigh of only mild inconvenience, and I got in my car and drove to the nearest grocery store — a place I have not previously known to be threatening.

Background

On the afternoon of March 22, 2021, 10 people were tragically killed at a King Soopers grocery store in the city of Boulder, Colorado. I have no personal details of what happened inside of the store, what their injuries were like, or what could have been done to help them in their crucial moments. My heart goes out to the families, friends, and acquaintances of all those affected. As a former paramedic in rural Kentucky, I once received a Stop The Bleed instructor training at a time when school shootings were on the rise. This initiative was started in 2017 by the American College of Surgeons and the ACS Committee on Trauma, in partnership with the Department of Homeland Security. As a second-year medical student who is greatly interested in pursuing emergency medicine, I circle back to my Stop The Bleed training every time I read the news about incidents like these. I think about the crucial minutes between injury sustained and the time first responders arrived. I have grown to understand that these situations can arise just about anywhere. I'm confident that I would know how to tamponade a bleeding wound in the field. And if I needed to, I would. But how can we as a whole community be more prepared? In this day and age, we need to treat it like CPR. The more bystanders who are ready and willing to work, the better.

What Does the Literature Say?

A randomized, controlled educational trial conducted by Goolsby et al. enrolled high school students from 39 states at a national conference and assessed the ability and willingness of high school students to apply a tourniquet prior to being educated on how to recognize the need for one and how to apply it correctly. Three educational modalities were utilized consisting of instructor-led, web-only, and blended education formats. This trial demonstrated that students who participated in hands-on interaction and learning were better able to apply a tourniquet correctly. However, all modalities saw nearly 100 percent success rate in teaching students how to recognize the need for a tourniquet as well as an improved willingness and comfort in applying one, should a situation arise where it would be appropriate to do so.¹

An additional pre-post evaluation conducted by Ali et al. through the NYU Winthrop Hospital assessed the effectiveness of the Stop The Bleed program for police officers and security guards of the surrounding areas who participated in the program. The pre- and post-evaluations demonstrated not only an improved comfort level in applying tourniquets, but also a significant improvement in correct tourniquet placement and decreased application time.² In a situation where someone has sustained a life-threatening injury, time certainly makes a difference.

How Can We Improve?

Not all training is necessarily created equal. There exist limitations to everything, and there are several factors that come into play. Another pre- and post-evaluation was conducted by Villegas et al. through the Weill Cornell Department of Surgery that reported significant participant improvements following their Stop The Bleed training for both hospital and community members. However, several

of the participants noted limitations in the program and believed the training would have been more effective if they were able to perform interventions on more realistic mannequins. It was noted that by properly simulating pulsatile blood flow in the practice care setting, the training would have had a more profound impact on their ability to apply these skills in the field.³ With that in mind, there are variables in the quality of training that individuals are receiving that may actually make a difference if they were to encounter a real-life situation where bleeding control is required.

Concluding Thoughts

So where does this leave us? Not only do we have the opportunity to educate students at various levels of education, but hemorrhage control programs such as Stop The Bleed remain a versatile option for responders and providers of all levels. You don't need to be working on an ambulance or in a hospital to make a difference in a moment where seconds matter. If we can implement a more widespread initiative to teach individuals how to control bleeding in various settings and provide them with the tools and the mindset to recognize the need for it, then we have the potential to make a substantial difference in the community-at-large. There is a tremendous amount of room for the Stop The Bleed initiative to be evaluated and improved upon, however I believe that access to training would improve bystander willingness to act. By correctly applying the skills they learn through hemorrhage control programs such as Stop The Bleed, they may be able save a life that could have otherwise not been saved.

More Information

ACEP has combined Stop the Bleed principles with CPR training and other basic bystander skills in an overall training called **Until Help Arrives**: <https://www.acep.org/uha/about-the-course>. ★

Ultrasound Guided Stellate Ganglion Block in Pulseless Refractory Ventricular Fibrillation

Chase Hamilton, DO

UNLV School of Medicine

Georgina Puentedurra, MD

UNLV School of Medicine

Garren Giles, DO

Attending Physician

University Medical Center

Stellate ganglion blockade has been used for over a century to decrease sympathetic tone to the heart. The left cardiac sympathetic denervation surgical procedure was first performed in 1916 to treat angina, severing the sympathetic innervation of the heart.¹ This surgical sympathetic denervation of the heart was later shown to reduce the incidence of ventricular fibrillation, specifically in animal studies.² A percutaneous procedure was developed in 1971 to interrupt sympathetic stimulation to the heart; the stellate ganglion block (SGB).³ The procedure involves injecting local anesthetic to the stellate ganglion. The SGB is most commonly used in the setting of electrical storm: 3 or more episodes of sustained ventricular arrhythmia over 24 hours.⁴ While the SGB has been documented as a treatment option for refractory ventricular arrhythmias, its use in the emergency management of pulseless arrest with shockable rhythm has not been well described.

Case Report

A 74-year-old man presented to the emergency department via Emergency Medical Services (EMS) in cardiac arrest. The patient had a reported history of hypertension and previous myocardial infarction with stenting in 2000. The patient had been playing blackjack at a casino and started having seizure-like activity. A bystander, who happened to be a nurse, was unable to find a pulse and initiated CPR reportedly

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within one minute of symptom onset. EMS arrived within 7 minutes. They placed a defibrillator and a mechanical compression device. EMS noticed him to be in ventricular fibrillation. He was defibrillated unsuccessfully with 200 joules. The patient was then intubated with a 7.5 endotracheal tube. Advanced Cardiac Life Support (ACLS) was continued and the patient was defibrillated three more times en route to the ED. With EMS, the patient was given epinephrine 3 times and amiodarone 300 mg once.

The patient arrived to the emergency department 25 minutes after his initial electrical event. Correct placement of the endotracheal tube was verified. The mechanical compression device was continued. The patient was placed on a Zoll defibrillator with anterior-posterior pad placement. He arrived with a right humeral intraosseous line. A second right tibial intraosseous line and right femoral central line were placed. Throughout the remainder of the patient's resuscitation the ACLS algorithm was followed with epinephrine every 3-5 minutes and pulse checks continued every 2 minutes.

After one initial 200 joule defibrillation was unsuccessful a second set of defibrillation pads were placed in the anterior-lateral position. The patient remained in refractory ventricular fibrillation and he was defibrillated using dual

sequential defibrillation with each machine delivering 200 joules, which was unsuccessful. The remaining defibrillations were dual sequential defibrillations. During the following rounds of ACLS the patient was given adjunct medications including calcium, bicarbonate, magnesium and a dose of 150 mg amiodarone.

At this point the patient had been defibrillated with 200 joules four times and had dual-sequential defibrillation with a total of 400 joules two times. The patient was then given lidocaine 100 mg IV, esmolol 4,535 mcg (500 mcg/kg) and an infusion of esmolol 100 mc/kg/minute was initiated afterwards. He remained in ventricular fibrillation.

ACLS was continued with defibrillation and epinephrine per ACLS guidelines. The patient remained in refractory ventricular fibrillation. At this point a stellate ganglion block was performed with ongoing CPR. The stellate ganglion was localized using ultrasound at the level of C4/C5. A total of 5 cc of 2% lidocaine was injected. At pulse check the patient remained in ventricular fibrillation. He was defibrillated with dual sequential defibrillation and at pulse check he had palpable radial pulses.

The patient had a total down time of approximately 45 minutes. Aggressive resuscitative measures were taken, as the patient had bystander CPR immediately initiated by a trained medical professional. The patient

also had a shockable rhythm during resuscitation, which encouraged the medical team to continue efforts.

Discussion

It is understood that ventricular arrhythmias are initiated by myocardial injury. Cardiac myocytes damaged from infarction become arrhythmogenic. This reduced threshold for arrhythmia and an increase in catecholamines with increased sympathetic nervous system activity creates ventricular arrhythmias that are difficult to treat.⁵ The surge of catecholamines caused by the increased sympathetic drive leads to release of endogenous epinephrine, which is inherently arrhythmogenic. This arrhythmogenicity is compounded by exogenous epinephrine given during ACLS resuscitative efforts. Esmolol has been described to help create a sympathetic blockade, terminating arrhythmias induced by hyper-sympathetic states.⁶ A fascial plane block of the stellate ganglion may also terminate otherwise intractable sympathetic activity. In this case report, we describe the use of the stellate ganglion block during cardiac arrest due to refractory ventricular fibrillation.

The sympathetic nervous fibers that innervate the head, neck, heart and upper limbs ascend from the first thoracic segments through the sympathetic chain to the superior, middle and inferior cervical ganglions.⁷ The stellate ganglion is composed of the fusion between the inferior cervical ganglion and the first thoracic ganglion. Its location is described as posterior to the carotid artery and between C5 and T1.^{8,9} Local anesthetic is instilled under the prevertebral fascia at that level allowing the anesthetic to be distributed to the stellate ganglion. There is evidence that the left stellate ganglion is preferential to the right stellate ganglion in providing sympathetic blockade to the heart, making the left SGB the ideal target for blockade.⁸

Procedural Tips

ED access to ultrasound with linear transducer capabilities is nearly

ubiquitous. The procedure for SGB has come to include ultrasound guided techniques, making the procedure safely accessible to most emergency departments.¹⁰

- A linear transducer is oriented transverse on the left anterior neck perpendicular to the trachea.
- The probe should be moved lateral for adequate visualization of the carotid artery, vertebral body of C6 or C7 and the longus colli muscle (Figure 1).

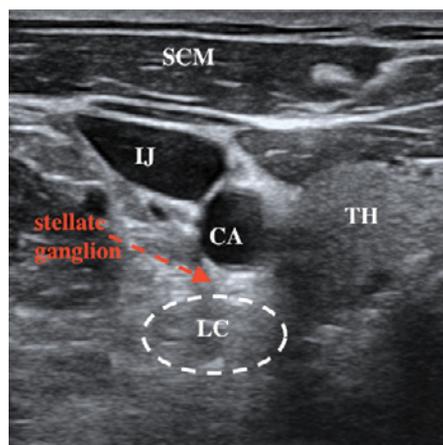


FIGURE 1. Nomenclature:

SCM—sternocleidomastoid; TH—thyroid; LC—longus coli; IJ—internal jugular; CA—carotid artery

- A needle is then inserted and directed posterior to the carotid artery and superficial to the longus coli just deep to the prevertebral fascia. A 2 cm needle is sufficient.
- Lidocaine should slowly be injected and observed on ultrasound to separate the muscle and fascial layers as confirmation of correct placement. In this case report a total of 10 mL of 1% lidocaine was instilled to create a temporary blockade of sympathetic activity. Bupivacaine is commonly used in the anesthesia literature at 0.25 to 0.5% at a volume up to 9 mL.¹¹ Lidocaine 2% of 8 mL has also been described and is effective, although the procedure can also be done successfully with similar volume doses of lidocaine 1%.¹²

In our case, the arrhythmia did not terminate immediately after the lidocaine

was instilled. The arrhythmia persisted for another 2 minutes, approximately, during which ACLS was continued. This delay in arrhythmia cessation suggests that the decreased sympathetic tone ultimately terminated the arrhythmia.

Further Study

The efficacy of the SGB in the setting of cardiac arrest due to ventricular arrhythmias is not well documented or understood. There have been case reports in cardiology, anesthesia, and only recently, emergency medicine.¹³⁻¹⁶ The emergent nature and rarity of cardiac arrest caused by ventricular arrhythmia makes it difficult to create effective studies about this subject. Margus et al. note in their case report that the American Heart Association and American College of Cardiology guidelines for the treatment of ventricular arrhythmias state that “the significant morbidity and limited options in these patients make cardiac sympathetic denervation a reasonable option”.¹³

The SGB is a relatively simple procedure that is minimally invasive. In cases of ventricular arrhythmias causing cardiac arrest this procedure can be utilized as an attempt at resuscitation.

Case Conclusion

EKG was obtained shortly after ROSC and showed a left bundle branch block meeting Sgarbossa criteria. Cardiology was called, and the catheterization laboratory was activated. The patient had a 100% right circumflex lesion, 70% left anterior descending stenosis, and 40% left circumflex lesion. Over the course of the next 24 hours the patient did not have meaningful recovery. Family decided to withdraw care. ★

TAKE-HOME POINTS

- The ultrasound guided stellate ganglion block may be used as an adjunct to established methods during pulseless ventricular arrhythmias.
- It is a potentially lifesaving procedure that is within the emergency physician’s scope of practice

Sudden-Onset Chest Pain and Subtle ECG Findings



Alexander Bracey, MD

Assistant Professor
Albany Medical Center,
Department of Emergency Medicine
@BraceyA

H. Pendell Meyers, MD

Assistant Professor
Carolinas Medical Center,
Department of Emergency Medicine
@PendellM

Case

A 52-year-old man presented to the ED with sudden onset sharp chest pain radiating down both arms with associated tingling. He reported the pain began suddenly while he was repairing his porch. He continued to report chest pain but was nontoxic appearing and had normal vital signs aside from bradycardia.

A prehospital ECG showed sinus bradycardia with poor-R wave progression; ≤ 1 mm ST elevation in V1-V3 with reciprocal ST depressions (STD) in II, III, and aVF; and large T-waves in V3-V4 suspicious for hyperacute T-waves.

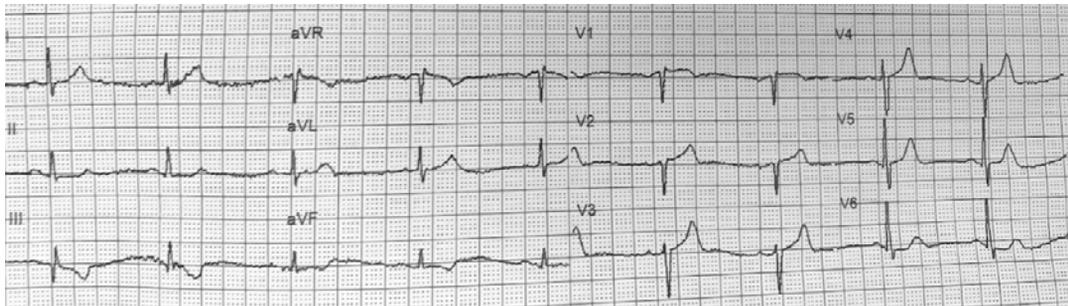


IMAGE 1. Prehospital EKG

A point-of-care ultrasound did not identify a pericardial effusion but did reveal anterior wall hypokinesia. Initial labs were normal except for a lactate of 2.66 mmol/L. His initial troponin T was < 0.01 ng/mL.

A second ECG performed 10 minutes after the initial study revealed sinus bradycardia with resolving hyper acute T-waves in the anterolateral leads and persistent STD in leads II, III, and aVF.

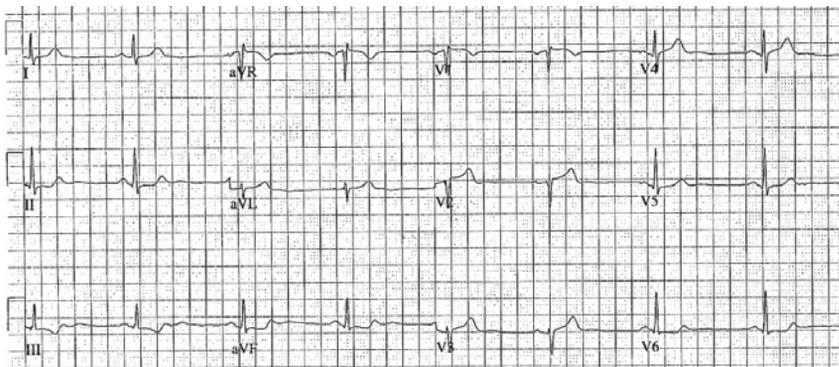


IMAGE 2. First In-Hospital EKG

Cardiology was consulted for emergent percutaneous intervention (PCI); however, the catheterization lab was not activated, as the ECG findings did not meet STEMI criteria (discussed below).

An additional ECG was performed after another 10 minutes. Findings included sinus bradycardia and further progression of STE in V1-V3, with persistent STD in II, III, aVF, V5-V6.

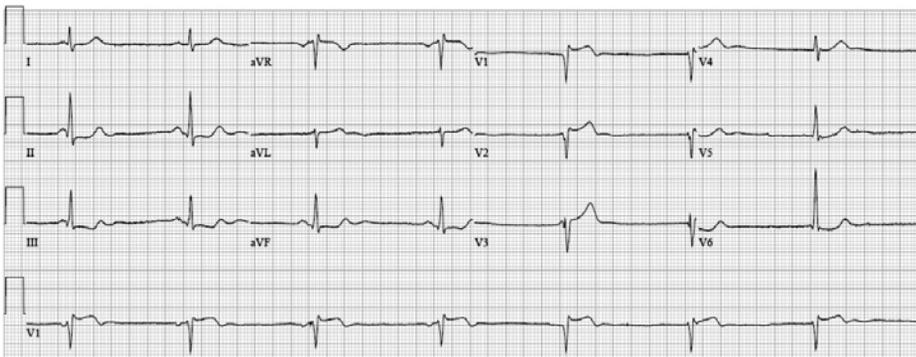


IMAGE 3. Repeat In-Hospital EKG

Careful measurement of V1 and V2 reveals 1.5 mm of elevation at the J-point, with V3 having 1.0 mm of elevation, which does not meet STEMI criteria.

While Cardiology discussed the case, the patient became unresponsive and was found to be in ventricular tachycardia. After one round of CPR and defibrillation, return of spontaneous circulation was achieved, and the rhythm returned to sinus bradycardia. The patient received heparin and amiodarone and was intubated for airway protection. Given findings suggestive of myocardial ischemia subsequent cardiac arrest due to ventricular tachycardia, Cardiology agreed to take him to the catheterization lab for coronary angiography. He was found to have a 100% proximal LAD lesion with acute thrombus. He successfully underwent PCI with a single drug-eluting stent placed, resulting in normal flow.

A repeat troponin obtained 2.5 hours after the initial undetectable measurement resulted in 3.65 ng/mL, which would ultimately peak 4.5 hours later at 8.47 ng/mL, representing a large myocardial infarction. Formal echocardiography performed 12 hours later revealed an EF of 45-50% with severe hypokinesis of the anterior wall, septum, and apex without LV aneurysm.

The patient was extubated 16 hours after presentation and was neurologically intact. He remained hospitalized for 6 days and was discharged after an uncomplicated course.

Discussion

This patient presented with sudden-onset chest pain while performing strenuous activity. The onset, timing,

and description of the pain warrants investigation of ACS. The pre-hospital ECG and initial ED ECG are highly suspicious for myocardial ischemia and injury; however, they do not meet STEMI criteria:

- Males age 40 or older: >2.0 mm of STE in leads V2 and V3 and >1.0 mm in all other leads
- Males < 40 years old: 2.5 mm of STE in leads V2-V3
- Women (any age): 1.5 mm STE in leads V2-V3

Classically, STEMI is considered to represent acute coronary occlusion syndromes. However, it has been demonstrated that at least 25% of patients with NSTEMI are found to have ACO on cardiac catheterization.

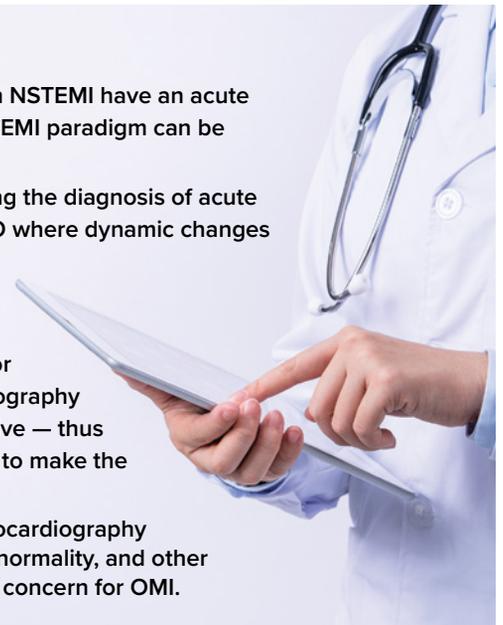
When compared to NSTEMI patients with ACO, NSTEMI patients with ACO demonstrate higher short-term and long-term mortality. Unfortunately, there is no perfect stratification tool or mechanism to identify these patients with ACO, which often delays cardiac catheterization and may adversely affect their prognosis.¹⁻⁶ This case report highlights this challenge by describing a patient with an LAD occlusion despite lack of STEMI criteria on initial ECGs and cardiac arrest in the setting of delay to cardiac catheterization.⁷ This case also highlights the current indications for emergent (<2 hr) angiography for NSTEMI patients with ischemia refractory to medical management (which, importantly, does not include opioids), ischemia with cardiogenic shock, or ischemia with electrical instability.⁷

In this case, serial ECGs helped to demonstrate an evolving pattern of ischemia. Had they not been performed, definitive management may have been delayed.

In addition to serial ECGs and the clinical features above, other adjuncts to the decision to perform emergent reperfusion therapy may include additional ECG leads (right sided, posterior, etc.), troponin, echocardiogram for the presence of wall motion abnormalities, and potentially even emergent CT coronary angiogram. ★

TEACHING POINTS

- Approximately 25% of patients with NSTEMI have an acute coronary occlusion; the STEMI/NSTEMI paradigm can be misleading.
- Serial ECGs are invaluable in making the diagnosis of acute coronary syndrome, especially ACO where dynamic changes may be prevalent.
- Conventional troponin tests take 4-6 hours to manifest in the serum; patients may require more urgent or emergent catheterization and angiography before the troponin becomes positive — thus troponin should not be relied upon to make the diagnosis of OMI.
- Consider serial ECGs, bedside echocardiography looking for regional wall motion abnormality, and other adjunctive tests when there is high concern for OMI.





Pushing Back Against the Stigma

Physostigmine for Severe Antimuscarinic Overdose

Sahar Rammaha

Michigan State University
College of Osteopathic Medicine

Larry B. Mellick, MD

Vice Chair and Professor of Emergency Medicine
Division Chief of Pediatric Emergency Medicine
University of South Alabama

Physostigmine has long been recognized as an antidote to reverse antimuscarinic delirium. However, its effectiveness, safety profile, and dosing have been disputed in recent years, leading to a decline in its use. Yet a growing amount of more current evidence suggests the toxicity profile associated with physostigmine is misunderstood and occurs when used inappropriately. Despite evidence that physostigmine is undeniably effective in treating the symptoms of confirmed anticholinergic poisoning, its use has plummeted since the early 1980s. Physostigmine became one of a handful of drugs that doesn't get the respect they deserve.

What is Physostigmine?

Physostigmine is a carbamate acetylcholinesterase inhibitor that is

readily capable of crossing the blood-brain barrier due to its tertiary amine properties. Consequently, physostigmine is effective in reversing the peripheral and central effects of anticholinergic toxicity.

Why the Stigma?

Physostigmine was used extensively in the 1960s and '70s in the treatment of undifferentiated delirium.¹ It was also during the 1970s that it was suggested as a potentially life-saving antidote in tricyclic antidepressant (TCA) toxicity.¹ However, following a report of two deaths during the treatment of tricyclic antidepressant overdoses with physostigmine, opinions rapidly changed.² The medical community's reaction to these two cases published in *Annals of Emergency Medicine* in 1980 was dramatic and global, resulting in an almost immediate rejection of physostigmine and movement towards seemingly safer delirium treatment options such as benzodiazepines. The most feared consequence of physostigmine administration includes cardiovascular

effects, especially sudden cardiac arrest. However, published reviews of these two cases subsequently suggested the associated fatalities were more likely due to the severe TCA toxicity rather than a consequence of physostigmine administration.^{3,4}

When is Physostigmine Effective?

In the setting of antimuscarinic toxicity, benzodiazepines have commonly been used to avoid the feared side effects of physostigmine. Despite physostigmine's effectiveness, it is underutilized due to its safety concerns and short duration of action.⁵ However, when presented with anticholinergic toxicity, in one small study physostigmine controlled agitation and reversed delirium in 97% and 87% of patients, respectively.⁶ This was in stark contrast to benzodiazepines controlling agitation in only 24% of patients while being relatively ineffective in the reversal of delirium.⁶

Why opt in for physostigmine over benzodiazepine?

Physostigmine administration is without significant complication when

given to patients in the ED suspected to have antimuscarinic delirium. Its antidotal effects have been reported to cover a range of drugs and different substance intoxications. The variety of drugs include antipsychotics, antihistamines (eg, diphenhydramine), muscle relaxants (eg, alprazolam, cyclobenzaprine, baclofen), antimuscarinic agents and plants including Jimson weed, belladonna, Hell's bells, trumpet flower, and Angels' trumpet.⁷

In addition to the wide coverage, the use of physostigmine has also been studied to reduce the rate of intubation in patients who present with delirium when compared to those treated by benzodiazepines. Endotracheal intubations were performed in patients who ultimately experienced neuromuscular paralysis due to benzodiazepines being ineffective in the control of agitation.⁶

Additionally, physostigmine administration has also been effective in avoiding unnecessary invasive testing and in reducing the length of stay when compared to benzodiazepines. In fact, patients treated initially or only with physostigmine had a lower complication rate and shorter time to recovery than those treated initially or only with benzodiazepines.⁶ The slower recovery rates seen in benzodiazepines could be contributed to the greater incidence of sedation, aspiration, and endotracheal intubations for patients initially treated with benzodiazepines.⁶ Neither of the complications were seen in patients initially treated with physostigmine, and prompt extubation was seen in some patients following physostigmine administration.⁶

How about pediatric populations?

Previous case reports have also shown promising safe and effective use of physostigmine infusions in control of pediatric patients experiencing antimuscarinic delirium and agitation.^{5,12} In one case of imipramine intoxication, a child presented with serious life-threatening coma, hypotension, and cardiac dysrhythmias that were reversed upon the administration of physostigmine.⁸ Physostigmine has

been highlighted to be the best agent for treating severe poisoning caused by imipramine and other drugs or plant poisoning that may induce anticholinergic-like toxicity.^{8,12}

Appropriate dosing

Dosing regimen of physostigmine remains uncertain, but employing a titration approach with low doses administered slowly and extending doses in intervals may minimize the risk of adverse effects. Initial doses can be lowered to 0.5 mg diluted in 10 mL of D₅W or normal saline, infused over the rate of 2-5 minutes while carefully observing the patient for any improvement in presenting symptoms or adverse side effects, as well as continuous cardiac monitoring.⁹ If the desired effect has not been obtained, then use a titration of additional 0.5 mg doses at 5- to 10- min intervals up to a maximum total dose of 2 mg over the first hour.⁹ This has been a consistent approach utilized and recommended by other authors, with delirium being found to reverse usually with an initial ≤ 2 mg.^{7,9} Pediatric IV dosing is 0.02 mg/kg with a maximum dose of 0.5 mg. This can be repeated every 5 to 10 minutes until a desired response occurs. Slow administration (≤ 0.5 mg/minute for pediatric patients) is required to prevent bradycardia, respiratory distress, and seizures. The maximum recommended total dose is 2 mg.¹²

How Do I Avoid the Misuse of Physostigmine?

Investigate the symptoms and patient history! Be sure to ask EMS or family members about any possible contact with the drugs in question. This will help narrow down the differential diagnosis and provide you with more confidence when selecting physostigmine as the antidote. If the patient history or drug in question remains unclear, check for the following signs and symptoms:

Signs and symptoms of cholinergic intoxication

Below are favorable conditions for using physostigmine

- Severe agitation and delirium are of most significance⁶
- Altered mental status, hallucinations⁷

- Drowsiness, obtundation⁷
- Restlessness, tachypnea, tachycardia
- Carphologia (lint-picking behavior), mumbled speech, and inability to follow commands¹⁰
- Dry skin and mucous membranes¹⁰
- Sweating, excessive salivation, bronchorrhea⁹

Be cautious in these cases

Below are unfavorable conditions for using physostigmine

- Very high risk of seizures¹¹
- Known underlying cardiac disease⁹
- Evidence of cardiac toxicity¹¹
- Prolonged QT interval, widened QRS complex¹¹ or QRS greater than 100ms¹¹
- Reactive airway disease
- Intestinal obstruction
- Known TCA poisoning!

If you decide to play it safe and are unsure about using physostigmine as your initial antidote, there is no contraindication in using a benzodiazepine. However, if all alternative methods fail, one should consider the use of cholinesterase inhibitors in anticholinergic delirium resistant to non-pharmacological delirium management.¹¹

Extra Resource

Watch a video of a pediatric patient being treated emergently with physostigmine: <https://youtu.be/6aj-Z8giv10>. ★



TAKE-HOME POINTS

- Physostigmine is useful in treatment of anticholinergic toxicity and appears far superior to benzodiazepines.¹¹
- Do not use physostigmine if TCA toxicity is suspected.
- The optimal risk/benefit in treatment with physostigmine would be to use a titrated dose of 0.5 to 1 mg physostigmine (0.01 – 0.02 mg/kg in children).^{11,12}

Bilateral Adrenal Hemorrhages

Due to Pheochromocytomas and MEN2A Syndrome

Kevin Vinokur, DO

New York Presbyterian-Brooklyn Methodist

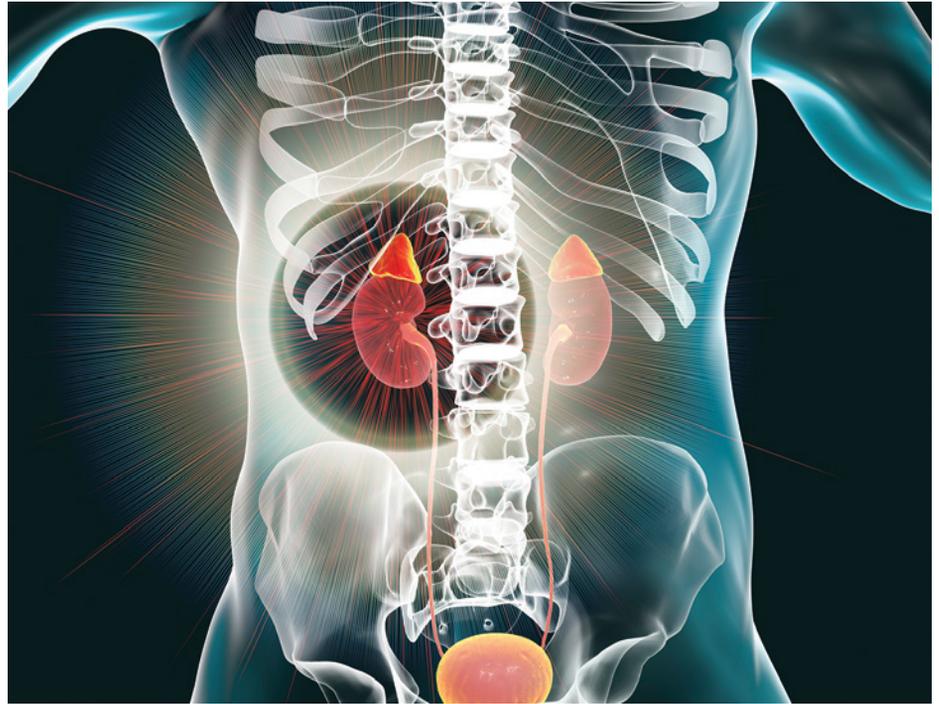
Christopher Mendoza, MD, MA, FACEP

New York Presbyterian-Brooklyn Methodist

Multiple endocrine neoplasia (MEN) syndromes are rare, inherited disorders in which several endocrine glands develop non cancerous (benign) or cancerous (malignant) tumors or grow excessively without forming tumors. There are multiple subtypes of MEN depending on the cluster of abnormal growths. Type 2A involves the thyroid, adrenal or parathyroid glands. Adrenal hemorrhage as the inciting cause is a very rare etiology of abdominal pain, accounting for 0.14%-1.8% in post-mortem analysis,¹ but in patients with hemorrhages underlying mass lesions are the most common cause, and pheochromocytoma is the most common identified mass.² Overall the chief complaint of abdominal pain accounts for 4-5% of emergency department visits per year, making adrenal hemorrhages a rare cause of a very common complaint.³ We present a case of bilateral adrenal hemorrhages due to pheochromocytomas and MEN2A syndrome.

Case

A 47-year-old female with a medical history significant for type 2 diabetes mellitus, hyperlipidemia, multinodular goiter, hypertension, and congenital left eye blindness presented to the ED because of significant abdominal pain worsening over the past several weeks. Her abdominal pain was poorly localized, and the patient could not identify where her pain began, but only that it is getting significantly worse. Her pain is associated with nausea, vomiting, subjective fevers, fatigue, and weight loss. Social history is significant for cannabinoid use. Past surgical history was significant for laminectomy and peroneal nerve decompression. Patient states that she



has not been able to take her blood pressure medications due to persistent vomiting and has been intermittently compliant with her home medications in the recent past.

In the emergency department, her vital signs were significant for tachycardia (122 bpm), and hypertension (176/137). Otherwise she was afebrile, with normal respiratory rate and normal oxygen saturation. Her physical exam was significant for an elevated heart rate with no audible murmurs or additional heart sounds. Lungs were clear to auscultation bilaterally. Her abdominal exam was significant for diffuse abdominal tenderness with both rebound and involuntary guarding. The rest of her physical exam was non-contributory.

Due to the lack of focality on physical exam in conjunction with no signs of impending decompensation, the decision was made to order a full complement of labs to help guide our choice of imaging modality. At this time due to the vague

abdominal exam and extended duration of symptoms, we were concerned for many different intra abdominal processes, including pyelonephritis, cystitis, biliary pathology, trauma, bowel obstructions, appendicitis, among others.

Initial laboratory findings were significant for a minimal white blood cell count elevation, 11.8k, with left shift, elevated blood glucose level (308), prerenal azotemia (BUN:Cr — 46:1.64) with an estimated GFR of 37, a lactate of 4.31 without acidosis, and a troponin elevation to 0.196 (negative <0.15). Due to the impaired renal function, the decision was made to get abdominal imaging without an intravenous contrast load. In the meantime the patient received two liters of IV fluids (Ringer's lactate), Ondansetron 8 mg IV for nausea, and Ketorolac 15 mg for pain. Previous labs were reviewed at the time of presentation and showed a normal GFR.

CT scan was significant for bilateral adrenal gland hemorrhages. Due to the

history of subjective fevers and sepsis as a potential cause of adrenal hemorrhage, blood cultures and broad spectrum IV antibiotics (vancomycin and cefepime) were ordered. Endocrinology was also consulted and they recommended IV hydrocortisone 100 mg. At this point the patient was admitted to the ICU for further management, close monitoring and to continue the search for an underlying cause of this presentation. During her admission, additional laboratory findings were diagnostic for pheochromocytoma, including a metanephrine level of >40,000, with a normal level less than 205. Additional findings showed an elevated calcitonin level. The constellation of findings and labs led to the diagnosis of Multiple Endocrine Neoplasia 2A (MEN 2A). Patient was discharged after being medically optimized, and was awaiting outpatient surgical intervention for bilateral hemorrhages/pheochromocytomas. She was admitted to the hospital for a total of 13 days, and spent all 13 of them in the ICU due to the requirement of intensive and difficult to control blood pressure. In the ED we did not treat her blood pressure due to concern for possible progression to hypotension from hemorrhage and possible septic shock. There is no consensus on a goal blood pressure for pheochromocytoma. She was initially treated with Hydralazine, but with minimal efficacy, the patient was transitioned to a titratable Cardene drip. Eventually discharged on a combination of calcium channel blockers, diuretics, and beta blockers. If pheochromocytoma is on the differential, it is important in the ED to remember not to give beta blockers due to the risk of worsening hypertension from unopposed alpha adrenergic stimulation.

Discussion

Adrenal hemorrhage is a rare and potentially life threatening cause of abdominal pain and shock.⁴ The etiology of adrenal hemorrhage is varied and includes trauma, adrenal lesions, anticoagulation therapy, congenital or acquired bleeding disorders, sepsis due to certain organisms, and pregnancy.⁴ In our patient due to the lack of trauma, pertinent history, medication uses, the two most likely candidates were an underlying lesion or sepsis. Sepsis due

to *Neisseria meningitidis* is the most common cause of adrenal hemorrhage, with less common organisms *H. influenzae*, *S. pneumoniae*, *S. aureus*, *N. gonorrhoeae*, *P. aeruginosa*, and *K. oxytoca*.⁵ During her hospital stay, blood cultures were drawn and never grew an identifiable organism. Her markedly elevated metanephrine levels indicated that the cause was more likely to be an adrenal mass.

Adrenal hemorrhage management is complicated as it must address two different problems — adrenal crisis often with accompanying shock, and hemorrhage from adrenal glands which can cause hypovolemic shock and may necessitate adrenal artery angioembolization.^{6,7} Hypovolemic shock is primarily managed by surgical or interventional radiology intervention. While no definite management guidelines exist, consensus states that early IV hydrocortisone 100 mg prevents deterioration and shock, and should be started if IV fluids resuscitation does not adequately support blood pressure.⁸

Pheochromocytomas are catecholamine secreting tumors that arise from the medulla of the adrenal glands.⁹ They are rare, occurring in less than 0.2 percent of patients with hypertension with hypertension being one of the most common presenting findings of pheochromocytomas.¹⁰ The classic presentation of pheochromocytoma is headache, hypertension, and sweating occurring paroxysmally over several months to years. Our patient on presentation denied having any headaches or sweats but was noted to be hypertensive. Given her history of hypertension and medication non-compliance we did not initially associate her elevated blood pressure with her acute complaints.

In patients in whom one suspects a pheochromocytoma, the most commonly used and accurate tests are both serum and urine metanephrine screens. Metanephrines are metabolites of catecholamines, which are produced by the rapidly reproducing chromaffin cells of pheochromocytoma tumors.¹¹ Management of pheochromocytomas includes a combination of surgical

intervention and chemotherapy — a combination therapy of vincristine, cyclophosphamide, and dacarbazine. For the emergency clinician, controlling blood pressure is of vital importance, especially before any surgical intervention can take place. Initial management involves alpha adrenoceptor blockade, before beginning conventional beta adrenergic blockade therapy.¹²

As part of syndromes, pheochromocytomas occur in Multiple Endocrine Neoplasia Type 2, both 2A and 2B. Unlike pheochromocytomas in regular patients, pheochromocytomas that arise as part of a genetic syndrome do not generate the same symptoms as in non-syndromic presentations. Half of patients with pheochromocytomas as part of MEN 2 do not have symptoms, and only one third of patients have hypertension.¹³ A similar finding is present in patients with Von Hippel Lindau disease, with 35% of VHL pheochromocytoma patients being asymptomatic.¹⁴

Multiple endocrine neoplasia type 2 (MEN2) is a rare disorder involving the development of medullary thyroid carcinoma, unilateral or bilateral pheochromocytomas, and proliferation of other endocrine tissues within the same individual.¹⁵

Abdominal pain, as a presenting complaint to the ED, will continue to be one of the more complicated syndromes we face, as it can be caused by multiple different disease processes involving multiple different organ systems. While rare, the presentation of an endocrine emergency, more specifically adrenal hemorrhage, as a consequence of MEN2 pheochromocytomas is potentially life threatening and requires a multidisciplinary approach to management. Her presentation of diffuse abdominal pain, in the setting of adrenal hemorrhage while hypertensive is not common, and should prompt more extensive evaluation. After admission, and medical optimization in the ICU patient was safely discharged to outpatient surgical follow up. Two weeks later she underwent surgical removal of the tumors and hematomas, and is currently doing well. ★

Intraabdominal Abscess Identified by Point-of-Care US in a Child

Carolina Vega, MD

University of Massachusetts
PEM Attending Physician

Roxanne Cheung, MD

Children's Hospital of Michigan
Central Michigan University

Dineece Barran, MD

Children's Hospital of Michigan
Central Michigan University

Jennifer Noble, MD

Children's Hospital of Michigan
Central Michigan University

Periappendiceal abscesses are a rare complication of pediatric appendicitis.¹ We present the case of an 11-year-old male who was evaluated for suprapubic pain, diarrhea, and fecal incontinence. Point-of-care ultrasound revealed a large lobular mass posterior and superior to the bladder, which was ultimately determined to be an abscess secondary to a perforated appendix.

Case

An 11-year-old male presented to the pediatric ED for severe lower abdominal pain and diarrhea. Onset of pain was 2 months earlier and was initially intermittent, transitioning to persistent lower abdominal pain 2 weeks prior to presentation. The pain became severe after meals, sometimes waking him from sleep. 1 week prior to presentation he developed diarrhea, with several non-bloody liquid stools per day, which progressed to fecal incontinence. He reported that hunching over relieved the pain partially, and he was urinating sitting down to avoid exacerbating the

pain. Review of systems was positive for weight loss, as well as 2 days of subjective fevers and non-bloody, non-bilious emesis that resolved a week prior. There was no report of cough, congestion, odynophagia, polydipsia, polyuria, hematuria, urinary incontinence, weakness, numbness, or rash. There were no known sick contacts, and the patient was fully vaccinated. The patient had 2 negative COVID-19 tests in the prior 3 weeks.

Vital signs at presentation to the ED were temperature 36.5°C, HR 90, RR 22, and BP 102/64. The patient was reclining on the bed. He had a thin habitus, and dry oral mucous membranes. Abdominal exam was significant for severe suprapubic tenderness with associated involuntary guarding, as well as moderate tenderness of the right and left lower quadrants. There was no costovertebral angle tenderness, hepatosplenomegaly, tenderness of the upper abdomen, inguinal lymphadenopathy, or signs of hernias or testicular torsion.

The differential diagnosis included urachal abscess, appendicitis, malignancy, and IBD. POCUS performed by a third-year PEM fellow using a phased array transducer revealed a large, round, avascular mass posterior to the bladder that connected to a similar structure superior to the bladder. A large proportion of the mass was hypochoic, with interspersed anechoic and hyperechoic elements (Figure 1, 2, 3). A radiology-performed abdominal

ultrasound confirmed a well-circumscribed lobular mass (12.3 x 5.0 x 7.0 cm), with heterogeneous echotexture and echogenic foci suggestive of air.

Serum testing was significant for an elevated WBC count 29.6 k/mm³ (range 4.1-10.1), 82% neutrophils, elevated immature granulocytes 2% (range 0-2), and an elevated CRP 95.4 mg/L (range <5). Urinalysis was significant for mild pyuria with 5-10 white blood cells/high power field (range <5) and elevated ketones.

An abdominopelvic CT with contrast confirmed a lower abdominal and pelvic lobular, thick walled, rim-enhancing, complex fluid collection that was most consistent with an abscess. The mass held air-fluid levels and layered debris, and measured 14.2 x 5.2 x 8.4 cm. The structure was in contact with multiple loops of bowel and was likely secondary to perforated appendicitis (Figure 4).

The patient was started on IV metronidazole and ceftriaxone, and he underwent CT fluoroscopic-guided drainage catheter placement with Interventional Radiology. He was transitioned to oral amoxicillin/clavulanic acid 2 days later to complete 2 weeks of antibiotics, was discharged on post-procedure day 3, and the drain was removed after 2 weeks.

Ultrasound Findings

POCUS demonstrated a large, lobular mass in the lower abdomen. In the transverse view, this mass was visible posterior to the bladder (Figure 1). In the sagittal view, the mass visibly extended

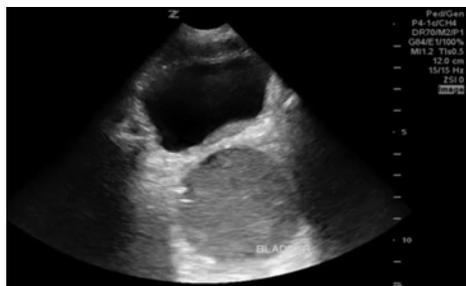


FIGURE 1. Transverse view demonstrating a round heterogeneous mass with posterior acoustic enhancement that is located posterior to the bladder.



FIGURE 2. Sagittal view demonstrating the lobular mass extending from posterior to superior to the bladder.

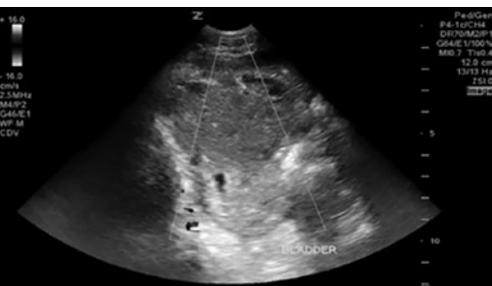


FIGURE 3. Transverse imaging with color-flow demonstrating lack of vascular flow to the mass.

from posterior to superior to the bladder and appeared structurally separate from the bladder (Figure 2). The structure was heterogeneous, with hyperechoic and anechoic foci, and demonstrated posterior enhancement (Figure 1, Figure 2). Using color Doppler, vascular flow was visible to the surrounding tissue and there was absent vascular flow to the mass (Figure 3).

Ultrasound Technique

POCUS for the evaluation of the pediatric abdomen uses low frequency transducers to evaluate deep structures. Both the curvilinear and phased array transducers can be utilized in the pediatric abdominal ultrasound examination, and these transducers commonly include the frequencies 2-5 MHz and 1-5 MHz respectively.^{2,3} The curvilinear transducer gives the advantage of a larger footprint and optimal penetration, and is preferred in patients of larger body habitus for both superficial and deeper structures.²⁻⁴ The higher frequency linear transducer is favored for evaluating superficial structures; at greater depths it has reduced resolution.^{3,4} Water-based gel should be applied to the target area and the minimum pressure needed for visualization should be applied to the transducer.^{3,5}

Graded compression is performed to systematically evaluate the lower abdomen for appendicitis.⁵⁻⁷ In visualizing an abnormality in the abdominal or pelvic area on ultrasound, it is important to rotate the transducer 90° and evaluate two perpendicular planes.^{2,5,7}

Abscesses are typically anechoic or hypoechoic in appearance, but the echotexture can also be heterogeneous. Abscesses can display septations, internal echoes, and posterior acoustic enhancement.^{3,4} Periappendiceal abscesses can additionally display hyperechoic foci due to air.⁷ The diagnosis of abscess is further supported by a swirl sign, characterized by swirling purulent material with compression. Abscesses often display hyperemic tissues surrounding the cavity, with absent vascular flow to the cavity itself. It is important to use color Doppler as it aids in distinguishing it from a vascular or tissue malformation.^{3,4}

Discussion

Abdominal pain is a common complaint with an extensive differential.⁸ Targeted imaging in conjunction with clinical exam findings can rapidly narrow the potential etiologies and determine the optimal intervention.^{8,9} Ultrasound is the first-line imaging for nontraumatic pediatric abdominal pain as part of stepwise clinical protocols designed to minimize radiation.¹⁰

While appendicitis is a relatively common pathology in the pediatric population, periappendiceal abscesses are rare.¹ The incidence of appendicitis in the U.S. is approximately 1 per 1000, and 15-50% of pediatric appendicitis cases are perforated.¹¹ However, the incidence of presentations of appendicitis complicated by periappendiceal abscess reached just 4.3% in one retrospective study.¹ These abscesses may be constituted by single or multiple fluid collections, and sizes range from 3 cm to 1047.2 cm³ in children.¹

Classic appendicitis presentation includes periumbilical pain that localizes to the RLQ, but diffuse abdominal tenderness may be present with appendiceal perforation.^{5,11} Exam may also demonstrate peritoneal signs such as guarding and rebound tenderness, and symptoms can include fever, anorexia, nausea, vomiting, and diarrhea.^{5,11}

In our case, POCUS facilitated the diagnosis of a large abdominopelvic abscess secondary to appendiceal perforation for a patient with an atypical presentation. POCUS identified abnormal findings 2 hours before the radiology-performed ultrasound. Timely diagnosis of appendicitis is a known advantage of POCUS, occurring an average of 2.5 hours earlier than radiology sonography in one prospective study.¹² Additionally, when evaluating for appendicitis, POCUS performed by PEM physicians with focused ultrasound training has a sensitivity and specificity of 85 and 93% respectively.¹²

When ultrasound is non-diagnostic in the evaluation of abdominal pain for appendicitis, consider abdominal CT or abdominal MRI. CT has reduced dependence on operator experience and elevated sensitivity and specificity of 90-97% and 91-99% respectively.^{8,13} MRI



FIGURE 4. CT Abdomen Pelvis with Contrast: Sagittal view demonstrating a rim-enhancing, thick walled, lobular pelvic mass holding debris and air-fluid levels that extends posterior to the bladder.

has no associated radiation and elevated sensitivity and specificity of 100 and 96% respectively.^{8,11,13}

Treatment of appendicitis complicated by large abscess formation requires immediate empiric broad spectrum IV antibiotics followed by IR percutaneous drainage. Elective appendectomy is performed several weeks after the acute episode to address the risk of recurrent appendiceal inflammation.^{1,11} In contrast, if the abscesses are small or are constituted by multiple discrete fluid collections that would necessitate multiple drainage sites, the first line of management is IV antibiotics, with interval imaging to evaluate for improvement.¹

Conclusion

Periappendiceal abscesses are a rare complication of appendicitis that can be detected early with targeted point-of-care ultrasound, which can enable prompt detection, diagnosis, and intervention for a large abdominopelvic abscess secondary to perforated appendicitis. ★

Compartment Syndrome

Secondary to Cocaine-Induced Rhabdomyolysis

Kyle Parker, MD

Emergency Medicine, PGY-2
Eastern Virginia Medical School

Leslie Rea, MD

Emergency Medicine, PGY-2
Eastern Virginia Medical School

Anjeza Cipi, MD

Assistant Professor of Emergency Medicine
Eastern Virginia Medical School

Acute non-traumatic rhabdomyolysis and compartment syndrome is a rare condition not frequently reported in the literature. We report on a 44-year-old male who presented to the ED with bilateral lower extremity compartment syndrome likely secondary to cocaine-induced rhabdomyolysis. The patient required bilateral four-compartment fasciotomy, ICU admission, and temporary hemodialysis. Ultimately, his cocaine usage was the only identifiable etiology of his presentation. Although substance-induced rhabdomyolysis has been widely reported, the progression to compartment syndrome is not frequently encountered and usually of multifactorial etiology. Rapid recognition of this uncommon condition is important as urgent fasciotomy is the mainstay treatment.

Introduction

We present a case of compartment syndrome likely due to substance-induced rhabdomyolysis. Rhabdomyolysis due to substance use, especially cocaine, is a well-documented occurrence.^{1,2} Additionally, non-traumatic rhabdomyolysis, usually due to strenuous exercise, has been reported as a rare cause of compartment syndrome without significant trauma.^{3,4} However, compartment syndrome due to cocaine-induced rhabdomyolysis is rare or underreported. Ikponmwosa, et al. described a case of substance abuse-associated compartment syndrome

Non-traumatic compartment syndrome is a rare diagnosis that requires a high index of suspicion on the part of the evaluating clinician.

secondary to intravenous injection of cocaine and heroin. However, this patient only developed isolated anterior upper extremity compartment syndrome near the injection site.⁵ Additionally, Huson and Fontenot reported a case of non-traumatic compartment syndrome associated with cocaine use, but they ultimately suspected multiple contributing factors including prolonged creatine usage and strenuous exercise.⁶ While there have been many reported cases of cocaine abuse resulting in rhabdomyolysis, our paper identifies a further complication of compartment syndrome that is not frequently encountered.

Case

A 44-year-old male with a past medical history of polysubstance abuse presented to the ED by EMS transport with bilateral lower extremity pain. He reported severe pain in both extremities that was worse in the lower legs. He had associated swelling of both legs and was unable to walk due to the pain. He denied any trauma. Reports waking up with these symptoms and denied any pain or symptoms before falling asleep. Denied tongue biting or bladder incontinence. He endorsed a history of IV drug use but states he had not used any in a few months. Of note, he was recently released from jail and had bilateral ankle bracelets — one for house arrest and one for continuous alcohol monitoring.

His triage vital signs were HR 89 bpm, RR 18 rpm, BP 130/100 mmHg and oxygen saturation 99% on room air. On initial evaluation, he was very

uncomfortable appearing. He had cold, blue, and mottled-appearing lower extremities with edema that extended to the mid-thigh bilaterally. He had diffuse tenderness of the lower leg, the skin was very tight, and he had worsening pain with passive flexion. His ankle bracelets were loose and not constricting his legs. Dorsalis pedis (DP) and posterior tibial (PT) pulses were not palpable nor able to be auscultated with portable doppler, however, both arteries were easily visualized with pulsatile flow on bedside ultrasound.

Initial differential diagnosis included arterial thrombus, congestive heart failure, deep venous thrombosis, proximal venous obstruction, and compartment syndrome. CBC was remarkable for an elevated white blood cell count of 17 K/dL. BMP was remarkable for an acute kidney injury with a BUN of 29 mg/dL and Cr of 1.9 mg/dL as well as hyperkalemia of 6.8 mmol/L. His CPK was 86,030 u/L and lactic acid was 1.9 mmol/L. Urinalysis showed large urine blood with 0-2 RBC/hpf and the urine drug screen resulted positive for benzodiazepines and cocaine. His EKG did not show any hyperkalemic changes. Fluid resuscitation was started, and his hyperkalemia was treated with calcium, insulin, and albuterol. Formal ultrasounds of the bilateral lower extremities were ordered with venous duplex and ankle-brachial index (ABI). This showed normal ABI bilaterally, no evidence of DVT, however, hemodynamics did suggest increased central venous pressure.



Right Lower Extremity Medial Fasciotomy



Right Lower Extremity Lateral Fasciotomy



Left Lower Extremity Medial Fasciotomy



Left Lower Extremity Lateral Fasciotomy

On re-evaluation, the overall discoloration of his legs had improved but he was still having significant pain despite multiple rounds of narcotic pain medications. Based on the increased central venous pressure noted on venous studies, a contrast CT of the abdomen/pelvis was obtained to rule out proximal obstruction which was unremarkable. At that time, the patient continued to have uncontrolled pain with edema below the knees bilaterally, and diffusely tight compartments. Orthopedic and vascular surgery were consulted due to concern for compartment syndrome. His ankle bracelets were removed prophylactically. Plain radiographs of bilateral tibia/fibula, as requested by orthopedics, did not show osseous abnormalities. Repeat BMP showed worsening renal function with BUN of 34 mg/dL and Cr of 2.6 mg/dL and increased CPK to 125,50 u/L. After evaluation by vascular surgery, he was taken urgently to the OR for a four-compartment fasciotomy. The intraoperative findings were notable for “tense right calf compartments with significant muscle swelling and edema in all compartments, without evidence of infection or muscle necrosis; left compartment tense with the majority

of muscle swelling and edema in the anterior compartment.” Photos were taken post-fasciotomy, see images 1, 2, 3 and 4.

Post-operatively, he was admitted to the ICU. His hospitalization was complicated by worsening renal failure and oliguria with a peak BUN of 42 mg/dL and Cr of 6.7 mg/dL ultimately requiring temporary hemodialysis. With continued supportive care, his CPK gradually improved and his renal function recovered. His leg wounds were ultimately closed 12 days after his initial procedure. He was discharged to a rehabilitation facility 19 days after admission with a final diagnosis of bilateral lower extremity compartment syndrome from cocaine-induced rhabdomyolysis.

Discussion

This was a case of suspected cocaine-induced non-traumatic rhabdomyolysis with resulting compartment syndrome requiring fasciotomy. The pathophysiology of rhabdomyolysis and compartment syndrome is interrelated and begins with an insult to the muscle cells. In rhabdomyolysis, the initial insult either directly damages the myocytes or reduces the availability of ATP or oxygen.

This leads to an alteration in ion influx that disrupts the intracellular space and sets off a cascade of events that result in muscle cell necrosis and the release of contents into the bloodstream. The myocytes release creatine kinase (CK) and myoglobin, the latter of which is directly nephrotoxic and is the primary cause of the acute renal failure that is seen in up to 33% of cases. The diagnosis is typically established by a marked elevation in CK (typically > 5x the upper limit of normal) or by the appearance of myoglobin in the urine.² Meanwhile, compartment syndrome is most commonly caused by direct trauma with 75% of cases associated with a long bone fracture.⁷ The resulting muscle damage and edema in the non-compliant muscle fascia leads to an increase in compartmental pressure that can collapse the arterioles and compromises perfusion. Muscle ischemia results which further compounds the damage and may lead to worsening CK elevation and rhabdomyolysis in the post-injury period.

The link between traumatic compartment syndrome and resulting rhabdomyolysis is well-established.^{2,7,8} However, as in this case, non-traumatic rhabdomyolysis can also result in

Rapid recognition of this uncommon condition is important as urgent fasciotomy is the mainstay treatment.

compartment syndrome through a cascade of muscle ischemia and fluid sequestration that causes an increase in compartment pressures. There is a wide range of causes leading to the development of atraumatic rhabdomyolysis including strenuous exercise, drugs, medications, malignant hyperthermia, genetic diseases, infections, and more. Additionally, non-traumatic compartment syndrome can occur in the absence of underlying rhabdomyolysis. This is a rare diagnosis with a wide variety of underlying etiologies including diabetes, hypothyroidism, autoimmune, DVT, and leukemia.^{9,10} It can even occur chronically with reversible ischemia usually caused by exertion in repetitive exercise.⁸ The anterior compartment of the lower leg is the most involved site,⁷ but it can occur anywhere even in the absence of direct trauma.

The development of rhabdomyolysis from cocaine is multifactorial due to increased sympathomimetic activity, muscle hyperactivity, hyperthermia, and arterial vasoconstriction that leads to muscle ischemia. It can develop with all routes of use but is more commonly seen with IV use or smoking crack cocaine due to higher blood levels of the drug.¹¹ There are a few case reports of compartment syndrome secondary to rhabdomyolysis with cocaine use, but these cases are frequently multifactorial in etiology. As mentioned previously, Ikponmwosa et al reported a case of compartment syndrome that developed near the site of injection with heroin and cocaine.⁵ The development of compartment syndrome was likely due to the direct trauma of needle injection in addition to polysubstance usage. In contrast, our patient only endorsed intranasal cocaine usage. Furthermore, the bilateral presentation makes any direct trauma less likely as an underlying factor. Riolo et al also identified a case of rhabdomyolysis associated with crack cocaine usage and subsequent compartment syndrome. This patient's rhabdomyolysis led to over resuscitation and four-limb

compartment syndrome.¹² However, given the timeline of events, it was likely not the crack cocaine abuse but rather the high volume of fluids given to treat rhabdomyolysis that led to compartment syndrome. Srivilasa, et al. report a similar case of cocaine-associated compartment syndrome requiring bilateral fasciotomies but their patient was suspected to have overdosed on olanzapine as well. Olanzapine is an atypical antipsychotic that has been reported as a rare cause of rhabdomyolysis.¹³ The combination of cocaine use with other potential muscle-damaging drugs should be considered when evaluating patients with non-traumatic muscle pain. Lastly, in a case series presented by Levine, et al., they identified three patients who presented with compartment syndrome after rhabdomyolysis associated with "bath salt" use.¹⁴ This included paraspinal and bilateral forearm compartment syndrome, of which two patients required fasciotomy. "Bath salts" are synthetic cathinones that also act as sympathomimetics and it is reasonable to assume that any substance with sympathomimetic activity could potentially present with these complications.

The diagnosis of compartment syndrome is generally considered clinical, with severe pain as the most consistent clinical sign. Additionally, pain with passive stretching is frequently mentioned in the literature as a classic feature.^{7,8} Symptoms such as paralysis and pulselessness are considered late findings and their absence should not make the diagnosis less likely. Compartment pressures can be measured with commercial products however their utility remains controversial and they are not frequently used at our institution.

The treatment of rhabdomyolysis is primarily supportive and includes identifying the cause, fluid resuscitation, correcting electrolyte abnormalities, and managing the downstream consequences such as renal failure.¹ In contrast, the treatment of compartment syndrome focuses on early identification, surgical consultation, and fasciotomy.^{3,6,8,13,15} As highlighted by Riolo, et al, aggressive fluid resuscitation in rhabdomyolysis can worsen compartment pressures and has been identified as an inciting factor in cases of nontraumatic compartment syndrome.¹² After six hours of ischemia, tissue necrosis starts to occur and can lead to permanent muscle and nerve damage. Meanwhile, if treated with fasciotomy within six hours, then complete recovery of limb function is anticipated.⁸ This highlights the necessity of vigilance on the part of the diagnosing provider as well as the prompt involvement of our specialist colleagues.

Conclusion

Non-traumatic compartment syndrome is a rare diagnosis that requires a high index of suspicion on the part of the evaluating clinician. Likewise, primary rhabdomyolysis has been identified as one cause of this atypical presentation. Meanwhile, cocaine usage is frequently reported as one of many etiologies causing rhabdomyolysis. To our knowledge, there are no reported cases where cocaine use was thought to be the only underlying etiology of rhabdomyolysis and subsequent compartment syndrome. This case helps to understand the underlying pathophysiology, raises awareness of the progression, and for similar causes of this rare presentation. Urgent recognition remains critical as the definitive treatment is surgical fasciotomy. ★

TAKE-HOME POINTS

- Compartment syndrome is a rare complication of substance-induced rhabdomyolysis.
- Treatment of rhabdomyolysis and related electrolyte abnormalities is supportive medical management
- In contrast, the treatment of compartment syndrome is emergent surgical fasciotomy therefore a high index of suspicion for this complication needs to be maintained.

Hydrofluoric Acid Related Injuries and Illness for First Responders

Jett MacPherson, MD
Wake Forest University

The 2020 siege on the U.S. Capitol shows that even peaceful protests can turn violent. Emergency physicians, tactical medical providers, and other first responders are tasked with taking care of those who become injured or ill in such gatherings. Hydrofluoric acid (HF) was likely used in incidents of vandalism during protests in Portland, Oregon this past year.¹ Accidental or intentional exposure of law enforcement officers (LEO), other first responders, protestors and bystanders to HF acid should therefore be planned for by emergency personnel especially given the complexity of treatment and the potentially fatal nature of HF toxicity. This article reviews the literature regarding presentation, pathophysiology and treatment of the chemical burns and systemic effects associated with HF.

Introduction

Hydrofluoric acid is an inorganic acid historically used for its corrosive properties in domestic and industrial settings. More recently, HF is being used at low levels in the manufacture of cleaning supplies, rust removers, fertilizer, pesticides, and some plastics.² Indeed, most of our knowledge about HF exposures comes from occupational exposures. HF is most commonly encountered as a colorless liquid similar to water in appearance or as a gas. It has a strong, irritating odor though may reach levels capable of causing harm to humans without a noticeable odor. Even small quantities of HF can cause life-threatening burns and systemic effects.³ Diagnosis of systemic toxicity may be delayed, as HF liquid often appears innocuous and symptoms of exposure may not present immediately. Delays in treatment may result in devastating consequences for the patient.

Mechanism of Injury

HF acid toxicity can occur via contact with skin or eyes, inhalation, or ingestion. All of these initial contacts can lead to

severe local and possibly systemic effects.

HF toxicity results from the weak HF acid penetrating deep into the skin and underlying tissue before dissociating into hydrogen and fluoride ions allowing fluoride anions into fascia, muscle, bone and the circulatory system.⁴ Fluoride anions sequester calcium, magnesium and manganese cations causing precipitous drops in these levels. Local and systemic hyperkalemia may ensue as cell membrane permeability to potassium is increased by local calcium depletion. Counterintuitively, hypokalemia may result as well, though the mechanism is not clearly understood. Hypocalcemia, hypomagnesemia and hyper and hypokalemia may lead to fatal dysrhythmias. Fluoride ions are believed to be directly toxic to myocardial cells by inhibiting adenylate cyclase. Locally, fluoride ions are also thought to directly inhibit Na⁺/K⁺ pumps, which adds to the local hyperkalemia with resultant neuronal depolarization and significant pain. HF is highly lipophilic causing liquefactive necrosis of deeper tissues resulting in the hallmark finding of HF burns: “pain out of proportion to exam.”

Clinical Manifestations

Clinically, the toxicity of HF exposure is directly proportional to:

1. Concentration of HF
2. Duration of exposure
3. Immediacy and adequacy of first aid measures, such as irrigation
4. Extent of body surface area exposed.

Unfortunately, outside of an occupational or household exposure, such as in an intentional attack, the concentration may not be known.

Cutaneous exposure: HF causes local injury via two primary mechanisms: as an immediate burn and as a delayed burn with skin penetration.⁵ At high HF concentrations (>50%), the hydrogen ion causes a corrosive burn similar to other acid burns, with immediate visible tissue damage,² immediate pain followed by the development of grey areas, necrosis or ulceration. Late manifestations may

include tenosynovitis and osteolysis.

At lower HF concentrations, which represent a majority of HF burns, immediate visible tissue destruction does not occur and there may be no initial evidence of chemical burn. Symptoms may be delayed up to 24 hours. Immediate burns and pain from high HF concentration exposures may actually have better prognosis as lower concentration asymptomatic burns may go undetected till severe local and systemic effects are already widespread.

Inhaled HF leads to symptoms related to local irritation, upper airway edema, non-cardiogenic pulmonary edema, reactive airway disease, and systemic absorption. Symptoms include mouth and throat pain, stridor, wheezing and dyspnea.

Ingested HF is less common and often the result of accidental or intentional ingestion of low HF concentration household products.⁶ Clinical manifestations related to local irritation include burning in the throat, esophagitis, gastritis, hemorrhagic pancreatitis, small bowel edema with resulting nausea abdominal pain, vomiting, GI bleeding, liver failure. Ingestion is often associated with rapid mortality but may result in systemic toxicity among those who don't die immediately.

Systemic HF toxicity can be difficult to manage. The systemic effects of HF are primarily related to potentially massive electrolyte disturbances, mainly hypocalcemia, hypomagnesemia, acidosis, fluorosis, and hyper or hypokalemia. Such disturbances can lead to severe alterations in renal, hepatic and cardiac function.⁷ Along with the symptoms associated with the mechanism of exposure, patients may present with systemic symptoms such as headaches, seizures, paresis, coma, hypotension, and cardiac arrhythmias. Signs of systemic HF toxicity include prolonged QT, cardiac failure, renal failure, coagulation disorders, rhabdomyolysis.⁸ These may be acute or late findings. Although toxicity is less likely to occur in

minor cutaneous exposures involving very low concentrations of HF⁹ the provider must remain vigilant for systemic effects even in this situation. For instance, consider the case of a patient with 3% BSA splash exposure to 20% HF who received immediate irrigation and calcium gluconate treatment and yet suffered cardiac arrest related to severe electrolyte imbalance.¹⁰

Management

Systemic toxicity is a feared complication of acute HF exposure, especially in the potential setting of attacks against first responders, terrorist attacks, accidental exposures, riots, and protests. In these settings, patients may not readily be aware of their contact and decontamination and treatment might be delayed. For this reason, first responders must be acutely aware of the possibility of exposure and all patients require rapid assessment.

As always, tactical assessment must precede treatment. Assessment of scene and tactical safety for the medical provider should occur, followed by ABCs. Patients should be triaged based on severity of injuries and if HF exposure is suspected, the provider must begin immediate decontamination with water or normal saline (NS).

Local pain can be used as a gauge of severity of exposure and HF concentration in unknown situations and helps guide triage and treatment. If the patient experiences severe, immediate pain, an exposure to $\geq 50\%$ HF should be suspected. If pain is delayed initially but develops within hours, HF concentration between 20-50% should be suspected. If local pain develops 12 to 24 hours after exposure, suspect HF concentrations $< 20\%$. Exposure to $> 50\%$ HF of any amount is of significant concern for life-threatening electrolyte abnormalities; $> 5\%$ TBSA with any concentration of HF is similarly higher risk for life threatening electrolyte abnormalities.

Prehospital Treatment Principles

1. **Skin irrigation with tap water should be initiated immediately** to dilute and remove HF from the skin. Lavage should be performed for 20-30 minutes. Delay of lavage until arrival to the hospital results in significantly more full-thickness injury, increased

likelihood of systemic effects as well as a hospital stay that is twice as long compared with those who received immediate first aid measures after HF chemical injury.¹¹ Make sure all jewelry is removed and skin underneath is irrigated.

2. Following irrigation, **calcium gluconate gel** should be applied to the affected skin in order to bind the cutaneous free fluoride ions and prevent penetration into the deeper tissues.¹¹ Because calcium ions have poor tissue penetration, the gel can only neutralize fluoride ions on the surface or the superficial skin layers. The gel can be purchased or made by mixing a water-soluble lubricant with a calcium gluconate solution or calcium gluconate powder (75 mL lubricant plus 25 mL of 10% calcium gluconate or 100 mL of lubricant plus 2.5 g of calcium gluconate powder).¹² This gel must be massaged into the affected area. Application of the gel should occur every 30 min initially and tapered down to every 4 hours until pain relief is achieved.¹³ Since there is such poor penetration of calcium into the deeper tissues, DMSO has been used as an agent to improve absorption. There is currently no consensus on use of DMSO, but this remains an option in contemporary literature.¹⁴ The person applying the gel must wear gloves to avoid self-contamination. One trick of the trade for treating hand exposures is to place the calcium gel in a large surgical glove then have the patient place their hand inside the glove.
3. Inhalational exposure can be treated with nebulized 2.5% calcium gluconate. (1.5 mL of 10% calcium gluconate in 4.5 mL of NS nebulized)
4. For ingested HF, the conscious patient should be encouraged to drink large amounts of water to dilute the HF followed by several glasses of milk or several ounces of Maalox, Mylanta or crushed Tums. Do NOT induce vomiting.
5. Institute immediate cardiac monitoring. A 12 lead EKG should be obtained to look for QT prolongation.
6. If severe systemic toxicity is suspected, immediately administer **IV calcium**

and magnesium. There is low risk for hypercalcemia or hypermagnesemia with this approach.

7. The eye is highly susceptible to HF exposure. The most important therapy is **immediate irrigation.** Contacts should be removed, and eyes should be irrigated with 1 liter of water or preferably normal saline. This can be administered using a nasal cannula setup or Morgan lenses for eye irrigation. A 1% calcium gluconate solution for follow up irrigation has been suggested in some articles, but this treatment is controversial and may cause worsening irritation. Prompt ophthalmological consultation should occur.

Field Considerations

In preparation for treating hydrofluoric acid exposures in the field, the tactical medical provider should remain alert for the possibility of HF exposure. Consider beforehand having access to a water supply for decontamination and carrying 10% calcium gluconate that can be readily mixed in the field. 10% calcium gluconate comes in 10 mL, 50 mL, and 100 mL plastic vials and does not require refrigeration. Normal saline and water-soluble lubricant should be carried so the provider can mix nebulized, and topical calcium treatments as needed.

If possible, mass decontamination with copious water should be performed on all victims as soon as possible. If there is a mass casualty situation, patients with immediate severe pain after exposure are likely to have had exposure to higher concentrations $> 50\%$ of HF and may be considered for more involved antidotes.

ED Treatment Principles

1. If not already performed in the field, decontaminate as above.
2. Other important considerations in HF burns include chemical injury to the nails. Fluoride ions readily passes through the nail plate and cause significant damage to the subungual tissues and could lead to systemic absorption, however finger and toenails block decontamination water and binding calcium gluconate from reaching the underlying tissue. Removal of the fingernail or drilling may be required to facilitate application of calcium gel to the underlying tissue.

Exposure	Treatment	Calcium gluconate 10%	Carrier
Cutaneous/skin	Topical gel	25 mL	75 mL water soluble lubricant
Inhaled	Nebulized	1.5 mL	4.5 mL NS

- Clinical evidence of hypocalcemia is often absent; therefore, patients who are high risk for HF exposure must be placed on cardiac monitoring and evaluated for prolonged QT interval and arrhythmias with electrocardiogram.¹²
- High-exposure groups and when severe systemic toxicity is suspected warrant immediate parenteral calcium and magnesium replacement even before the serum calcium and magnesium levels are determined. As mentioned above, it is rare to cause hypercalcemia or hypermagnesemia in this setting since total body stores of these ions are often severely depleted.
- Ionized calcium, magnesium and potassium must be emergently and frequently monitored in cases of suspected systemic toxicity. Other labs to consider obtaining include venous or arterial blood gas monitoring, kidney function and liver function tests.
- Proven hypocalcemia requires calcium gluconate infusion parenterally and frequent (hourly) serum calcium monitoring.¹⁵
- Treat hyperkalemia as per usual.
- In suspected systemic HF toxicity it is beneficial to maintain normal acid base status. In patients with systemic fluoride toxicity or metabolic acidosis, alkalization of urine by administering parenteral sodium bicarbonate improves excretion of fluoride and may help normalize acid-base status.
- Consider hemodialysis to reduce both fluoride and potassium levels, and to treat persistent hypocalcemia despite calcium infusion especially in patients with decreased renal function.
- If ingestion occurred recently, gastric lavage via NG tube may be of benefit. Risk of gastric or esophageal perforation must be balanced against the risk of death from systemic absorption. The provider placing the NG is at risk of dermal or inhalation exposure related to the procedure so proper PPE is a must.
- Calcium gluconate injections have been widely adopted for use on moderate to severe HF burns as it has been shown to reliably reduce pain.¹⁶ Indications include a central hard grey area with surrounding erythema and throbbing severe pain despite management with irrigation and gel. Infiltration is thought to be unnecessary for burns with HF concentrations $\leq 20\%$. A 27-gauge needle can be used to inject a 5% to 10% calcium gluconate solution into subcutaneous tissue beneath the burn. It is recommended to inject no more than 0.5 mL/cm² of burn surface area, as infiltration has been associated with increased compartment pressure and necrosis. Compartments should be monitored closely after injection. Edelman et al were first to describe this and set a limit of 0.5 mL per phalanx with repeated injections preferred.¹⁷
- For patients with severe HF burns with unrelenting pain despite aggressive calcium gel topical therapy, typically of the digits or other poorly distensible areas that will not tolerate intradermal injection, intra-arterial calcium infusion has been used; this management technique may be associated with complications, including artery spasm and bleeding, ulnar nerve palsy relating to position of immobilization, median nerve palsies secondary to hematomas, and carpal tunnel syndrome.² It is traditionally used for digital burns of the hand as the fingers are poorly distensible and may not tolerate local injection, but has also been described for the face and lower extremities. Intra-arterial calcium infusion was first described by Kohnlein and Achinger in 1978, in which they used angiogram to guide the route of infusion to the radial, ulnar, or brachial artery and then infused 50 mL of 4% calcium gluconate given over a 4-hour period.¹⁸ This cycle was repeated every 12 hours until the patient was free from pain. This protocol has since been adapted with decreased complication rates.

In this procedure, an artery such as the radial artery, proximal to the burns is cannulated. Placement of the cannula should be confirmed by angiography, although an arterial line with good wave form placed on first attempt without any difficulty could be assumed to be usable as well. **10 mL of 10% calcium gluconate is added to 40 mL of either D5W or NS (resulting in a 2% calcium gluconate solution) and infused interarterially over 4 hours.** This treatment may need repeated for recurrent pain.¹⁹

Conclusion

Hydrofluoric acid (HF) chemical burns are an important consideration in patients seeking medical treatment with signs of chemical injury following riots and violent protests. The hallmark physical exam finding in HF burns is “pain out of proportion to exam” though the exposed may be completely asymptomatic initially. Serious systemic toxicity can occur with even small cutaneous exposures. Alongside the normal field medical assessment and treatments, every known or suspected exposure should have immediate and thorough skin irrigation with water for 20-30 minutes followed by application of calcium gluconate topical gel. Providers should consider inhaled calcium gluconate for patients with signs of inhalational exposure. Ingestions should be treated with decontamination and dilution by drinking copious water and antacids. Eye exposure should be treated with immediate irrigation. Patients with signs of toxicity should be empirically treated with IV calcium gluconate and magnesium pending lab evaluation. Patients require prompt EKG and cardiac monitoring for dysrhythmias and prolonged QT interval. Once lab is available, serum electrolyte levels, liver and renal function, venous or arterial blood gas should be obtained urgently, and electrolytes must be repeated hourly at a minimum.

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The Road to PEM Fellowship During a Pandemic

A Conversation about Applying Virtually

Cindy Chang, MD
Cincinnati Children's Hospital

Michelle Davis, MD, MA
Maimonides Medical Center

Gwen Hooley, MD
Icahn School of Medicine
at Mount Sinai

While applying to fellowship in the best of times can be stressful, the 2020 application cycle was uniquely different in that all interviews were held virtually. Navigating this uncharted territory was stressful for both applicants and programs alike. At the moment, it appears likely the 2021 cycle will also be virtual for most programs. To help PEM fellowship applicants embarking on this journey, we sat down with recently matched PEM fellows from 2020 to discuss the process of applying for fellowship virtually. Dr. Cindy Chang (CC) completed her EM residency at Los Angeles County Harbor-UCLA and recently matched at Cincinnati Children's Hospital for PEM Fellowship. Dr. Michelle Davis (MSD) completed her EM residency at Rutgers New Jersey Medical and recently matched for PEM fellowship at Maimonides Medical Center. Both interviewed at programs throughout the country and were excited to share their experiences with this unprecedented application cycle.



Dr. Cindy Chang



Dr. Michelle Davis

Q: Did you end up applying to more places than you originally intended due to covid? Do you wish you had applied to more or less?

CC: I did apply to a few more programs than I originally planned to — part of it was the uncertainty with COVID, and unsure of how difficult it would be to match as an EM applicant. In retrospect, I wish I had applied to a few less. You do meet amazing people/future colleagues on the interview trail, but interviews are exhausting (or maybe I'm just getting old compared to when applying for residency!).

MSD: I applied to more programs than I originally intended, not because of the pandemic, but due to the competitive nature of matching into PEM. Originally, I intended to limit my application pool to certain geographic regions and ended up applying across the country to ensure that I would match into a fellowship program. I'm glad that I did because

the program that was my best fit, and ultimately where I matched, was not located back home.

Q: How did you get a feel for the programs since you couldn't go see them in person?

CC: I asked a lot of questions to EM/PEM and Peds/PEM mentors, and current fellows. There are a lot of EM-trained specific questions that you should ask and have answered at each program you interview at. Any program that is seriously considering training you will make the time to answer any questions you may have that are specific to you. Even though the interview was virtual this past season, you still get a good feel for the culture of the program through interacting with the faculty and fellows on interview day. I also connected with some current fellows and alumni outside of the interview day, everyone was very kind to phone/Zoom chat, answer questions, and offer advice!

MSD: Despite all the online research and reaching out to my PEM faculty, mentors, and networks, I found that the best way to get a feel for programs was meeting the PD, faculty, and fellows virtually. Getting to know the people and program's culture were the most important factors for me since each program's clinical experiences and academic expectations are universally the same thanks to ACGME standardization.

Q: What do you think is the best way for programs to get a sense of an applicant without meeting them in person?

CC: I think it's important to present the best paper application possible, it should convey your story, no one else's. Update your CV throughout residency so that it's easier to fill in ERAS when the time comes. Make things easy to read, I like using bullet points for each activity on ERAS. Write a thoughtful, strong personal statement — have many

people read it and give you feedback. Get strong letters of recommendations from PEM people, letters probably carry the strongest weight. And then in the interview, just be you :). It's very conversational, anticipate what you will be asked and have thoughtful questions. No one should know your application better than you.

MSD: Reflecting on my experience, I think my letters of recommendation, personal statement, and CV conveyed a unified story of who I am, why I want to pursue a career in PEM, and what I can contribute to the field. Many interviewers commented on how they appreciated the honesty, conciseness, and vision that came forth in my application, which helped them to get to know me. It gave my interviewers a baseline familiarity with me during our virtual conversations, which helped take the edge off the stress of interviewing on a novel platform.

Q: What are some tips for acing a virtual interview?

CC: Just be you. Make sure the lighting is good, otherwise I didn't do much else besides having a white background. Do wear a full suit. You can probably invest in having a nicer looking background to make it unique!

MSD: Spend the money on a good webcam, microphone, and light stand — interviewers notice those details! I also recommend doing some research on how to stage a professional setting either at home or at your residency's academic offices because the aesthetics of your webcam setting will definitely be a conversation starter during your interviews. Wear a full suit, including a belt and dress pants, just try to relax and be yourself as is appropriate. Interviewers want to get to know you and hear about your professional as well as personal activities, like that new bread baking hobby you developed during quarantine.

Q: Looking back, would you have done anything differently?

CC: No, I'm just sad you only get to match at one place — there are so many wonderful people at each program that would be lovely to work with and learn from. Honestly, no matter where

the algorithm would have thrown me, professionally I'm sure I would have been happy at any of the places I ranked.

MSD: No, I would not have done anything differently. I'm very happy that I matched at a place where I will fit in comfortably, and I look forward to professionally growing with them in the next few years. I'm especially grateful that the match worked out well for me considering how incredibly competitive and unusual this past application cycle was.

Q: With the match being more competitive this past year and Children's hospitals seeing lower volumes, would you consider delaying fellowship until after the pandemic?

CC: No, just try to match at a place that sees a large volume of children and variety in pathology.

MSD: I think the conventional wisdom of doing fellowship right after residency still stands. It becomes really difficult to secure letters of recommendations the farther away you are from training. I can also see how hard it must be to give up an attending salary to go back to a resident salary if you delay fellowship. In light of the pandemic, doing a fellowship sooner rather than later may be necessary to give job seekers the competitive edge they need now that the EP job market is going through some painful economic downsizing and reconfigurations across the country.

Q: What most surprised you about this past application cycle?

CC: Initially, I was concerned that the virtual interviews would make it difficult to truly grasp the culture of a program, but it actually wasn't. I think as emergency physicians, we are used to meeting people and forming relationships quickly, while building trust all at the same time. I was surprised that it wasn't that difficult figuring out which places would be a better fit for me.

MSD: I was most surprised by how flexible and transparent program leadership was during this past application cycle. They were literally

reinventing the wheel with the virtual-only format, which I think attributed to this sense of being more flexible and transparent than they may have been in a more traditional format. I also was very surprised by the great career advice and mentorship offered by my interviewers during this past cycle. Through that experience, I could tell how connected and supportive the PEM community was, which very much validated my decision to pursue fellowship training and join this community.

Q: Did you/were you able to do an away rotation? Would you recommend it?

CC: No. I think it depends on your situation. If you don't have a PEM program/faculty at your residency program, then you probably should do an away rotation.

MSD: I did not do an away rotation since my residency program has a dedicated pediatric ED and a robust NICU rotation in our curriculum. I would not recommend doing one since it does not add much to your application or chances of matching. Doing an away rotation is only necessary if your residency does not have a pediatric ED or PEM faculty to write you letters of recommendation.

Q: If you could pass on one piece of advice to a 2021 applicant what would it be?

CC: Just be you, share who you are, what drives you, why you love PEM and why you want it to be a significant part of your career. Remember too that the purpose of the interview is also for you to figure out if you like them, their culture, and if you will be supported and valued as an EM-trained fellow. It goes both ways and everyone wants a win-win situation in the end.

MSD: Take a deep breath, try to relax, and have fun on the interview trail. Be proud of all of your accomplishments and hard work, and let that shine in your application and interviews. You have made an excellent decision to join the kind, close-knit, and exciting world of pediatric emergency medicine so enjoy! ★



Knowing Our Patients' Rights

Laura R. Cuevas, MS4

UC Irvine School of Medicine

Carina Mireles-Romo, MPH, MS4

UC Irvine School of Medicine

Victor Cisneros, MD, MPH

UC Irvine Department of Emergency Medicine

@drvcisneros

It is no surprise that caring for patients in the ED requires collaboration between several key team players. Emergency physicians work with other healthcare providers, clinical social workers, case managers, and law enforcement officers (LEOs) to provide the best care for all patients. In the setting of emergency care and a criminal investigation, time motivates healthcare providers and LEOs, which can result in conflicts of interest when it comes to access to patients in the ED.¹ The Interdisciplinary Education Committee is a committee within the Emergency Medicine Interest Group (EMIG) at the University of California, Irvine School of Medicine (UCISOM). The goal of EMIG is to perpetuate interest in the field of EM through various educational meetings, workshops, and career advising. The Interdisciplinary Education Committee was created and is led by medical students in an effort to highlight some of the key team players in the ED. Additionally, this committee was created to share resources and best practices when caring for vulnerable populations, including uninsured and/or undocumented patients.

The group hosted a presentation titled *Knowing our Patients' Rights*, featuring 2 emergency physicians, a psychiatrist from Martin Luther King Jr. (MLK) Community Hospital in Los Angeles County, a UCI police officer, and an immigration attorney from the Public Law Center. The goal was for attendees to learn about legal and ethical information when caring for patients in the presence of LEOs, the role of LEOs in the ED, important information to know when faced with an Immigration and Customs Enforcement (ICE) agent, and local resources available for undocumented patients.

The discussion points and questions answered during the panel session were inspired from three controversial cases that either attracted attention at the regional or national level or were personally witnessed by the panelists. Here we also highlight relevant information that was shared during two episodes on *Emergency Medicine Reviews and Perspectives (EM:RAP)*. *Note that cases may vary; the information provided is not official legal advice but rather as a way to start and continue a discussion within medical education and residency training on these pressing topics. Please contact your hospital's risk management office or legal counsel for official advice.*

CASE 1. DRIVING UNDER THE INFLUENCE (DUI) AND BLOOD DRAWS

A patient is taken to the University of Utah Hospital in Salt Lake City in 2017 after being injured in a head-on collision leaving the other driver dead.² The ED nurse was confronted by LEOs seeking a blood sample. She explained she was unable to grant their request under their hospital policy. This case attracted national attention after release of the police body camera footage showing the interaction between the officer and nurse leading to the nurse's unlawful arrest.

1. Pre-hospital: How do LEOs assess intoxication?

In the context of DUI driving, officers will stop a driver for suspicion for something other than possible DUI, such as driving too fast, slow straddling or not reacting to a traffic light. Officers look for objective signs such as blood shot eyes, slurred speech, odor or staggering gait. A DUI sobriety test may follow and then the officer will determine if the individual will be placed under arrest or is safe to drive. The alcohol level can be assessed with a breath test or a blood draw. If the suspect does not consent to the test, then it gets complicated. The officer may explain that their license may be suspended given that in the state of California, when individuals

get a driver's license they give "implied consent" to a sobriety test. Officers can get a warrant within hours if the individual denies the test, however as we know, alcohol level diminishes as time progresses.

In Orange County, California, officers call a CFP (Certified Forensic Phlebotomist) who is responsible for drawing the blood to assess the blood alcohol level. This differs by county. Once a subject is a patient in the hospital, an officer calls upon a CFP and works with a nurse to facilitate the blood drawn. If an officer asks the nurse to draw blood (without the presence of a CFP) and the nurse collects the blood for them without knowing this policy, this blood can potentially be used in the future if there is a trial. In cases where a patient is unable to provide consent, such as during a trauma, and the blood has been drawn as part of the workup, the police department can subpoena later on to get hospital records, if needed, for a trial. Also, the concept of "chain of custody" must be taken into account. For example, if the nurse draws the blood but places it in her pocket or elsewhere for a few hours prior to giving it to police, this potentially breaks the chain of custody meaning that that can potentially be inadmissible in court because it is difficult to say if that blood is actually from the patient in question. Hence why it's important to have the official phlebotomists (to protect the chain of custody).

2. In hospital: LEOs are required to investigate criminal acts, however they are not required to abide by HIPAA (patient privacy law). Under what circumstances/policies should physicians share lab/exam findings with LEOs?

Under the Health Insurance Portability and Accountability Act (HIPAA), medical providers and facilities must follow strict rules about the health information that may be shared, how and with whom.¹ Sharing patient protected information with LEOs is a violation of HIPAA unless there is an exception, such as mandated reporting of domestic violence, providing information about a patient's status during a trauma activation when a crime is under investigation or when the officer provides a warrant or court order from

a judge. Other exceptions vary by state. As a reminder, a patient's name, address and date of birth are protected by HIPAA. Therefore, it is not okay for LEOs to take patient stickers that contain protected patient information. Sometimes avoiding a HIPAA violation may be difficult and unpreventable, such as when a LEO must remain present because of safety concerns for the physician or other patients in the department.³ Some health care institutions have guidelines in place outlining that outline how to care for patients in the presence of LEO in the ED.

3. Some patients may refuse certain tests even though LEOs may present search warrants or court orders to continue an investigation, what kind of questions should physicians ask LEOs to decide how to act in these situations? What kind of information are physicians legally obligated to share with LEOs?

LEOs need a warrant to draw blood if the patient refuses the request to draw blood. A warrant can be obtained fairly quickly over the phone.⁴ However, it is important to note that a healthcare provider is not required to perform any procedures they believe are not medically indicated or if the patient refuses, even if the LEO presents a warrant. Consider contacting the hospital legal representative if this situation occurs. Hospitals usually have protocols in place for these situations. Providers should have extreme caution in making these decisions without consulting its hospital legal representative. Note that providers have been sued for bad patient outcomes after performing a procedure requested by a warrant that was not medically indicated.³

4. Decision making capacity: Do intoxicated individuals have capacity?

The word *capacity* differs among law vs medical vs hospital policy. Medical capacity is based on four elements: 1) understanding of the risks, benefits and alternatives, 2) demonstrating appreciation of those benefits, risks and alternatives, 3) showing reasonings in making a decision and 4) communicating their choice. Any physician can determine if a patient has capacity.

A patient can be intoxicated and still

have capacity including the capacity to leave against medical advice (AMA) or to refuse certain procedures. Just because a patient's alcohol level is high does not mean that they do not have capacity. Some patients who have alcohol use disorder may "live" at a high number and may refuse tests or will leave AMA. It is important to assess these patients to make sure they are "clinically sober" so they can be discharged or leave AMA safely. Make sure that the patient can ambulate and tolerate oral intake and that they are not a danger to self or others at discharge. Lower alcohol levels may actually be dangerous for individuals who are chronic drinkers and consume large quantities on a daily basis, as they can withdraw and have dangerous outcomes.

In the context of law enforcement and driving under the influence, officers also determine capacity, for example, when asking for consent to draw blood or perform a breath test. According to the officer on this panel, when a person answers "yes", nods their head or says "mhm," it means the person can make that decision. However, when officers ask questions in other areas, this can be up for debate and can be questioned in court.

CASE 2. ATTEMPTED SUICIDE WHILE IN DETENTION

In the U.S. the number of individuals in ICE detention centers has increased dramatically from 7,474 in 1995 to 37,311 in 2020.⁵ Many detainees have histories of repeated physical and emotional trauma and the abuse experienced in detention can exacerbate their mental health illness. In fact, an analysis of detainee deaths discovered that suicide is one of the most common causes of death among detainees.⁶ Given this context, our panelists discussed treatment of a proposed detainee who is taken to the ED due to a suicide attempt while in detention.

1. Under what circumstances must doctors cooperate fully with ICE and when can doctors refuse to work with ICE?

A patient's rights depend on the hospital's internal procedures and even on how the physical space they are in is set up. For example, every commercial

space has an entryway open to the general public. ICE does not require a warrant to enter that space to approach or to question individuals. To the extent to which ICE is in a space that is considered a public space, there is really very little protection for individuals and ICE's ability to ask anybody questions is unfettered. It is up to the individual to exercise their right not to answer questions.

The question becomes more complicated in cases when an ICE officer wants to cross a physical barrier to go into a more private area. This is uncommon, but an officer usually asks whoever is guarding that private space for permission to enter. If a hospital does not have a policy to clarify what to do in this situation then it is possible for the "gatekeeper" to give permission for the ICE officer to enter and at least visualize the space and possibly also to question an individual.

From the medical standpoint, you are not morally or legally obliged to share any information directly with ICE. If an ICE officer or LEO presents a warrant, you can defer the request to the hospital's legal counsel and they can guide you and help you decide how to proceed or which questions to ask or answer.

2. Disposition planning: What type of healthcare is available for immigration detainees at detention centers?

Primary care in Adelanto, the detention center nearest Orange County, or any other ICE-contracted facility is primarily, but not exclusively provided by nurse practitioners (NPs). These facilities also have contracts with physicians who make infrequent visits. If a detainee needs higher level of care, they are transferred to specific hospitals that have contracts with GEO Group, a real estate investment trust that invests in private prisons, immigration detention centers, and mental health facilities.

3. What are the appropriate steps that physicians must take when discharging patients from the ED who need follow up care? Should physicians provide discharge instructions to the ICE officer or should materials be sealed?

Upon transfer or release from a detention center, ICE is required to

give the individual a summary of their medical records, any medically necessary medications and referrals to community-based providers.⁷ ICE is also required to provide follow up care, including specialty care, for individuals who remain in detention after their hospital visit. Therefore, when discharging patients from the ED who need follow up care, provide a copy of the written discharge instructions to the patient (print a copy in their native language too, if possible). Remember to use an interpreter to verbally communicate all discharge instructions directly to the patient if they speak a language other than English and/or they are unable to read. It is a federal legal requirement, protected by Title VI of the 1964 Civil Rights Act that requires medical providers to make interpretation services available to patients with limited English proficiency.⁸ Also, similar to cases when a LEO is present, you are required to abide by HIPAA when caring for patients who are accompanied by an ICE detention officer. Therefore, kindly ask the ICE officer to step out of the room while communicating with and evaluating the patient.

4. Do immigration detainees have decision making capacity?

The question on decision making capacity typically comes up in cases or immigration proceedings to decide who should be deported. Legally, not everyone has a right to an immigration hearing, and who does depends on a lot of complex factors including the patient's past immigration history and how they are currently in ICE custody. There is a threshold of decision making that an immigration judge must use to decide whether the patient has the mental capacity to understand what is happening during the proceedings. If the individual does not have an attorney and the judge decides that the patient does not have capacity to participate meaningfully in their proceedings, then the judge *may* appoint counsel at no cost to the individual. This is one of the rare situations in which a detained individual will be provided counsel at no cost, but not categorically the only situation. Rarely, deportation proceedings may even be terminated if the immigration judge cannot get enough information to decide whether the individual should be deported.

In southern California, because of

a lawsuit that was filed in the region, the Department of Homeland Security or ICE has an obligation to inform the immigration judge affirmatively if they have reason to believe that an individual suffers from a mental health illness. In the case of a patient who presents after a suicide attempt, for example, ICE is obliged to ask the hospital for medical records to present to the judge and this will actually help the detainee's case.

5. What is the policy for contacting family members of those who are in ICE custody?

According to the immigration attorney, she is not aware of any ICE policy on this topic, but can't see why they would prohibit the hospital or clinic from contacting the family. According to UCI Health policy, any individual in law enforcement custody is not permitted to have visitors without prior approval by the LEO supervising the patient in custody. Phone calls are permitted after authorization by LEO.⁹ It is important to contact the Risk Management department at your hospital to learn about policies in place at your facility.

CASE 3. DE-ESCALATING POTENTIALLY VIOLENT PATIENTS

On October 6, 2020, a Los Angeles County sheriff deputy shot a patient who was allegedly swinging a metal device while being treated at Harbor-UCLA Medical Center.¹⁰ According to the hospital report, the patient became aggressive and a deputy who was guarding another patient's room approached the situation to attempt to de-escalate the situation.

1. What emergency code is used at UCI to indicate that a potentially violent patient is in house?

Code gray is used to indicate that a combative or violent individual is in-house while code silver alerts an individual with a weapon or a hostage situation is in-house. Any individual can activate these codes by pressing the panic button which can be found at a nurse's station or by calling extension x6123. Simultaneously, if there is enough staff on hand, another health care provider on scene can call the dispatch line to talk to the public safety department directly and communicate details of the situation. It is important to

be informed about your institution's plan in place for whenever an individual with a weapon is present in-house.

2. What happens when the code is called and who is required to respond?

When these codes are activated, the call is directed to the operator and to the public safety supervisor, not to a police officer. The public safety supervisor will then send public safety officers and/or police officers to the scene who will collaborate with the clinicians to deal with the situation. The response is a team effort and if the situation allows, a discussion should take place among all of the team members to decide how to and who will respond. Typically, a public safety officer will respond first and the police officer will step in if deemed necessary, such as if the patient is violent. If the patient has a history of violence, the police officer may be the first one to step in. Immediate action to restrain the patient may be taken to keep everyone safe.

3. What are some strategies health care workers can use to de-escalate potentially violent patients without using physical force?

Several de-escalation models exist in the literature that can be used by healthcare providers including the Dix and Page model, the Turnbull et. al model and the Safewards Model.¹¹ Following are some recommendations shared by the panelists.

- Speak calmly to the patient and give them space. No one likes to be walled in or feel trapped. The WORST thing you can do is escalate yourself and expect a person who is agitated/violent to back down.
- Make sure that there are no objects that can be used as weapons in the room or on you.
- Ask all non-essential people present to leave the area. If a public safety officer or a police officer is present, ask them to stay back until needed.
- Consider the patient's mental status before entering the room/area. If the patient is experiencing psychosis, verbal de-escalation will be insufficient given the patient is incapable of linear thought and action. You can offer these patients

medications, however keep in mind that it may be difficult to get them to respond or calm down. Priority should be placed on their safety.

- If a patient is angry or upset about something and acting out, ask what happened and if there is anything you can do to help. Consider offering food, drinks, and oral medications as necessary. Sometimes if you tell a patient you can give them a tablet to help with their feelings, they may be more willing to take that instead of an injection.
- Remind the patient that your job as a physician is to be an advocate, to keep them safe and well taken care of. If they can help you understand their needs, you can work on their behalf to provide what they need.
- If the patient is upset about their wait time, validate their feelings and remind them that the workup can take time and that you will update them as frequently as possible.
- As the situation calms, consider setting boundaries for the patient. Explain that name calling and physical violence are not acceptable in the hospital. Tell them that you are happy to continue treating them as long as they abide by those rules, but if it continues, then you will be forced to end their care and will have to discharge them. The discharge will be reluctant, but will have to be done for the safety of the other patients and hospital staff.

4. De-escalation training of LEO's in Orange County

Both public safety officers and police officers receive specific training on de-escalation and training is often integrated. However, there are different levels of training and public safety officers receive more advanced specialized training on how to deal with patients since their primary duty is patient care and to guard health care workers. Overall, the use of force to de-escalate patients has decreased at UCI Health thanks to progressive and better training championed by Lieutenant Frisbee. According to the 2019 UCI Police Annual Review, "over 99% of responses by the public safety team to address aggressive and violent subjects were successfully de-escalated by the public safety and clinical

team through non-physical tactics".¹² A total of 15,002 Total Public Safety incidents were filed, 3,936 involved assisting clinical staff to de-escalate a situation, with 44 of those requiring physical tactics by a public safety officer (personal communication, Sgt. Chris Bolano and Lt. Anthony Frisbee, January 26, 2021).

5. What are some ways to build trust between LEO, the public and health care providers?

It is important that LEOs build trust with health care providers and patients to provide adequate patient-centered care. Positive nonenforcement contact with the public has shown to be an effective strategy to enhance police trust and legitimacy.¹³ In 2019, UCI Police and Public Safety officers hosted several community engagement events for patients and health care providers.¹² These events included inviting members of the healthcare community to share a cup of coffee and snacks with LEOs, continuing an annual toy drive with UCI athletes to pass out toys to pediatric patients, and conducting a Self Defense & Safety Awareness Course for healthcare employees to learn about personal safety.

SUMMARY

It is essential to know patients' rights and how to best advocate for them and yourself. Remember that LEOs are not obligated to comply with HIPAA, and it is the responsibility of the health care team to ensure HIPAA is not violated. Sometimes this may be unavoidable when there are safety concerns for the physician or other patients in the department that may require a LEO to remain present during the patient encounter. It is important to remember that healthcare workers are not obligated to perform any procedures they believe are not medically indicated or if the patient refuses, even if the LEO presents a warrant. If you are unfamiliar with specific policies at your institution regarding the presence of LEO, including ICE officers, contact the risk management office at your institution.

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EMRA AT ACEP21

EMRA programming will be held at the Omni Seaport, adjacent to the Boston Convention & Exhibition Center. Registration is now open; while registration for ACEP21 is not required to attend EMRA programming, it is encouraged.

Saturday, October 23

1:00 pm – 5:00 pm EMRA Medical Student Council Meeting

Sunday, October 24

4:00 pm – 6:30 pm EMRA/ACEP Leadership Academy (by invitation only)

Monday, October 25

12:30 pm – 5:30 pm Case-Con Residents

12:30 pm – 5:30 pm Case-Con Medical Students

12:30 pm – 2:00 pm EMRA Committee Programming

2:15 pm – 3:45 pm EMRA Committee Programming

4:00 pm – 5:30 pm EMRA Committee Programming

5:00 pm – 7:00 pm EMRA Job & Fellowship Fair (Boston Convention & Exhibition Center)



Visit <https://www.emra.org/be-involved/events--activities/acep/> for registration updates.
All times listed are Eastern.



Tuesday, October 26

- 7:00 am – 8:00 am EMRA Rep Council Registration
- 7:00 am – 8:00 am EMRA Rep Council Welcome Breakfast & Candidate's Forum
- 8:00 am – 1:30 pm EMRA Rep Council and Town Hall Meeting
- 9:00 am – 3:00 pm EMRA Resident SIMWars Competition
- 6:00 pm – 6:30 pm EMRA Awards, 25u45, VIP & Board Alumni Reception

Wednesday, October 27

- 1:00 pm – 3:00 pm EMRA 20 in 6 Resident Lecture Competition
- 5:00 pm – 7:00 pm EMRA Airway Stories

Thursday, October 28

- 7:00 am – 5:00 pm EMRA MedWAR (site to be announced)



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Who Are You Competing Against?



Meredith Busman, MD

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Over the past 2 years, the South Zone of my hospital's ED has become a sort of home. I know the workflows, key codes, and where to find the freshest graham crackers. Most importantly, I know the doctors and nurses, the clerks, pharmacists, and techs; coworkers who are really more like teammates. At its best, that familiarity is comforting. Other days, it is paralyzing.

I feel driven to impress. To wow. To razzle-dazzle 'em. Like a child seeking a parent's approval, I need to show my team that I am a confident, compassionate, decisive, and highly skilled emergency medicine superstar on the rise. At first, I thought this pressure to awe was the result of years of competition. After all, to get this far, I had to beat out thousands of other students for that college admission, that med school acceptance, that residency match. I told myself it shouldn't be a competition anymore; I had made it, hadn't I? But the shame I felt whenever I made a mistake was still there. I still beat myself up for not ordering the antibiotics soon enough, being slow to answer an attending's question, or not seeing the pathology on the CT scan that seemed clear to everyone else. I watched residents with the same amount of clinical experience lead resuscitations and wondered how they could seem so at ease. I tried to model the same confidence but always fell short.

I genuinely like my co-residents; I want them to do well and I cheer them on. Yet part of me couldn't stop seeing their successes as my failures. I championed

wellness initiatives, taught medical students to suture, ran marathons, and cultivated my personal interests. Still, what did that matter when my other residents were being inducted into national honor societies, or publishing papers, or landing jobs in hot markets? Moreover, what did it matter in the emergency department? All I wanted was for my ED team to see me as equally capable — instead I felt stuck at the bottom of the pile, the last choice when no one better was around.

I've come to realize the competition isn't my colleagues — it is myself I am trying to beat. As an intern, my program director told me I was "stuck in my head." As I began my third year of training, I was still leaving the hospital feeling like I let someone down. Some days it was myself, others it was my patients or my coworkers. And that feeling — of being a disappointment — was slowly turning into something worse: the feeling that I was a failure. Things might have been better if I could have truly believed the praise that I did receive. Instead, it was easier to let criticism sink into my bones and allow affirmation to run off like water. Stuck in my head, my own worst enemy.

I don't think I am alone in this sense.

We all carry a burden, invisible to some but crushing to us who can feel the weight. It can bring you low even on good days, and sink you further on the bad ones. Maybe it is a traumatic case that continues to haunt. Maybe it is feeling caught between personal and professional commitments. For me, it was the feeling of not being good enough to truly belong. Call it imposter syndrome or call it something else, but once you know the feeling, the name is irrelevant.

But recognizing that I struggle with insecurity allows me to face it. If this is a competition, that means I can win. When I try and fail, I can choose to pick myself up again, dust off the hurt, and get back to work — I can be better. I can refuse to let me beat me. I can continue to dwell on comparisons and ideals, or I can take pride in the skills I have cultivated. It is easy to lose perspective on the road to becoming a physician, but when I let myself look back, I am impressed, wowed, razzle-dazzled even by how much I have grown. I can look at my strengths, accept that they are different from the strengths of my colleagues, and see them as valuable. I have started to ask "What next?" instead of "What if?"

Emergency medicine is hard. As a mentor and colleague once told me, "We are normal people having normal reactions to abnormal situations." Look out for your colleagues, and look out for yourself. When you sense that you are struggling, I hope you can step back and ask why. I suspect, ultimately, the roadblock is not another person or a workplace. Feel the burden you are carrying and lift it higher. You are stronger for having carried it. Don't let you beat yourself.

Dr. Busman is a PGY3 EM Resident at Spectrum Health/MSU in Grand Rapids, Michigan. Her professional interests include advocating for resident and physician wellness. As a medical student she served as the president of her school's wellness committee, and she is currently a co-chair of the GRMEP Resident and Fellow Wellness committee. She is a Wellness Leadership Fellow through CORD and has been invited to speak as a panelist at Schwartz Grand Rounds. ★

ECG Challenge

Nathan Potter, MD
Christiana Care Health System

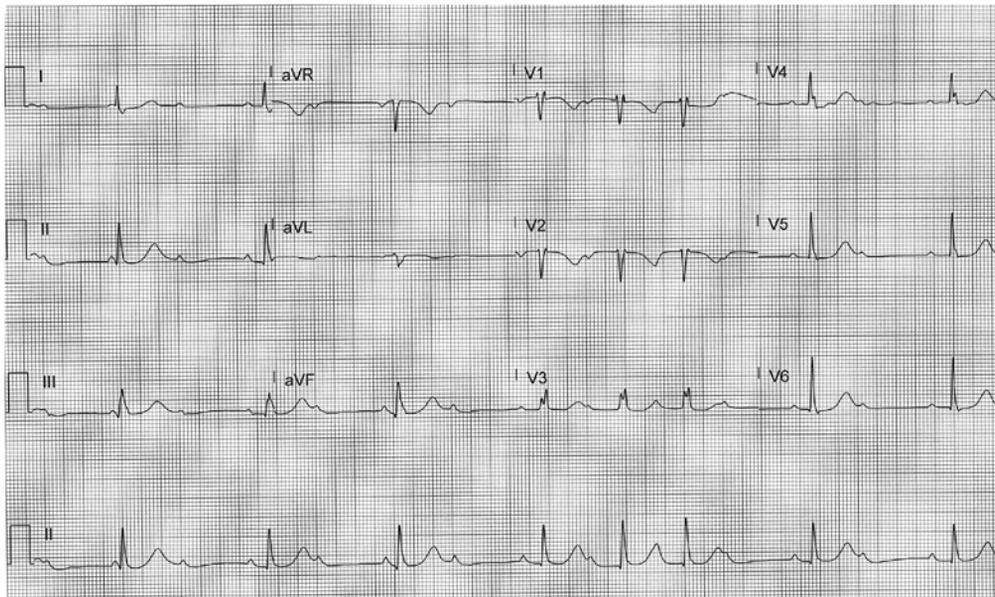
Jeremy Berberian, MD
Associate Director of Emergency Medicine Resident Education
Dept. of Emergency Medicine, Christiana Care Health System
@jgberberian

CASE.

A 65-year-old female with history of thoracic aortic aneurysm s/p aortic graft repair several years ago presented to the ED with positive blood cultures on outpatient labs. These had been ordered by her PCP secondary to 2 weeks of fevers and malaise. Other history is notable for a dental extraction 3 weeks ago.

What is your interpretation of her ECG?

See the **ANSWER** on page 50



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ECG Challenge

This ECG shows an irregular narrow-complex bradycardia with an average ventricular rate of 48 bpm, a regular atrial rate of 86 bpm, normal axis, variable PR intervals, and normal QTc interval.

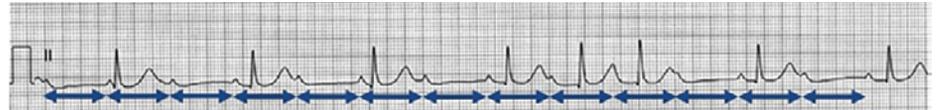
Discussion

The differential diagnosis for an irregular narrow complex rhythm with bradycardic ventricular rates includes:

- Atrial fibrillation (rate controlled)
- Atrial flutter with variable block (rate controlled)
- Atrial tachycardia with variable block (rate controlled)
- Wandering atrial pacemaker
- 2nd degree AV block Mobitz types I and II
- Variable high-grade AV block (e.g., 3:1, 4:1, etc.)
- 2nd degree sinoatrial block
- Sinus arrhythmia
- Sinus bradycardia, junctional rhythm, or junctional bradycardia with irregular pattern of PAC, PJC, and/or PVC
- Sinus bradycardia, junctional rhythm, or junctional bradycardia with regular patterns of PAC, PJC, and/or PVC (bigeminy, trigeminy, etc.)

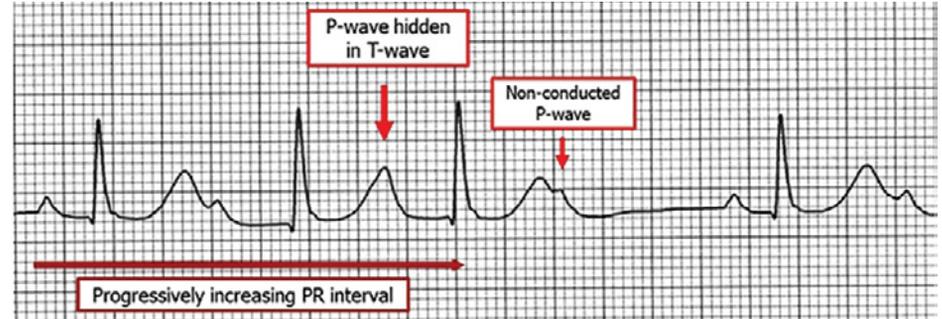
The key to interpreting this ECG is recognizing that the P-waves march out (i.e., there is a regular PP interval, see **Figure 1**). This narrows the differential to a 2nd degree AV block and/or variable high-grade AV block. Note that atrial tachycardia with variable block will show regular P-waves with an irregular ventricular rhythm but requires an atrial rate of 150-250 bpm, which is not seen in this ECG.

FIGURE 1. The P-waves march out at a regular rate of 86 bpm



This ECG is a hodgepodge of AV blocks. The 1st QRS complex is likely a junctional escape beat as the preceding PR interval is too short for there to be normal sinus conduction. This is followed by a 2nd degree AV block with 2:1 AV conduction for 2 beats. The 4th, 5th, and 6th QRS complexes have progressively lengthening PR intervals followed by a non-conducted P-wave (see **Figure 2**), consistent with a Mobitz type I 2nd degree AV block. The penultimate beat shows a 2nd degree AV block with 2:1 AV conduction. There is a P-wave that is partially hidden in the T-wave of the last beat, but the ECG cuts off before the type of block can be determined (e.g., 2-1 AV conduction, Mobitz type I, etc.).

FIGURE 2. The 4th, 5th, and 6th QRS complexes show progressively lengthening PR intervals followed by a non-conducted P-wave consistent with a Mobitz type I 2nd degree AV block



A repeat ECG showed a Mobitz type II 2nd degree AV block which then progressed to a 3rd degree AV block. This illustrates that while a Mobitz type I is generally considered benign, it can progress to more severe AV blocks if associated with a pathologic etiology. In other words, just because a Mobitz type I doesn't always require treatment, that doesn't preclude the presence of an underlying cause that does require treatment.

2nd Degree AV Block with 2:1 Conduction Learning Points

- 2nd degree AV block with 2:1 conduction will show blocked conduction of every other P-wave
- It is not always possible to determine if the underlying block is a Mobitz type I or type II, so assume the higher risk Mobitz type II conduction

Mobitz type I AV Block Learning Points

- Progressively increasing PR interval and decreasing RR interval until a non-conducted P-wave occurs (i.e., P-wave without accompanying QRS complex)
- PR interval immediately after non-conducted P-wave is shorter than PR interval preceding non-conducted P-wave
- RR interval that includes non-conducted P-wave < twice the PP interval
- Can be normal variant and usually does not produce hemodynamic compromise
- Typically associated with excess vagal tone and therefore usually responds to atropine when acute treatment is needed
- Can lead to a more advanced AV block if associated with a pathologic etiology
- Can be seen with inferior MI

Case Conclusion

This patient remained in 3rd degree AV block and became hypotensive, requiring transvenous pacing in the ED. She was admitted to the ICU, and blood cultures came back positive for *Streptococcus mitis*, a viridans group Streptococcus species commonly associated with endocarditis from a dental source (the bacteria was named for the mitral valve, off of which it was first isolated). A subsequent transesophageal echocardiogram was within normal limits, making endocarditis extremely unlikely. A cardiac PET scan was obtained due to her history of ascending aortic aneurysm repair and confirmed the diagnosis of aortic graft infection. Although infrequent, aortic graft infections can lead to 2nd or 3rd degree AV blocks due to the proximity of the AV node and bundle of His to the aortic valve and root. ★

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- 1. A 27-year-old man presents with dysuria and urethral discharge. What is the most appropriate test to determine the etiology of this patient's condition?**
 - A. Insert a swab into the urethra and send the sample for culture and blood agar plating
 - B. Obtain a pharyngeal swab and test for *Chlamydia trachomatis* and *Neisseria gonorrhoeae*
 - C. Send a urine sample for *Chlamydia trachomatis* and *Neisseria gonorrhoeae* PCR assay
 - D. Send a urine sample for urinalysis, microscopic examination, and bacterial culture
- 2. A 72-year-old woman presents with sudden-onset severe periumbilical pain, nausea, and vomiting; she is unable to get comfortable. Her medical history includes hypertension, diabetes, and a recent diagnosis of breast cancer. Her triage vital signs include BP 105/72, P 126, and T 38.3°C (100.9°F). Her abdomen is soft with no specific tenderness elicited, and bowel sounds are present. Which imaging modality is preferred to make the diagnosis?**
 - A. CT angiography
 - B. MRA
 - C. Plain x-rays
 - D. Ultrasonography
- 3. Which clinical presentation of herpes zoster most suggests an underlying immunocompromised state?**
 - A. Bacterial superinfection of the lesions
 - B. Lesions in three or more dermatomes
 - C. Vesicles in the auditory canal
 - D. Vesicles on the tip of the nose
- 4. A 29-year-old woman who is 34 weeks' pregnant presents after a motor vehicle crash with abdominal pain, cramping, and spotting. What is the best approach to assessing for signs of fetal distress?**
 - A. Cardiotocography
 - B. Kleihauer-Betke testing
 - C. Pelvic examination
 - D. Ultrasound examination
- 5. During the physical examination of a female sexual assault victim, the examiner is most likely to note injury to what part of the anatomy?**
 - A. Hymen
 - B. Labia
 - C. Posterior fourchette
 - D. Vaginal wall ★

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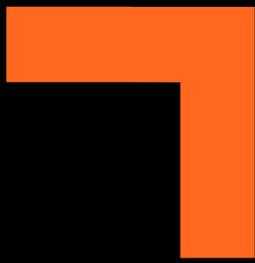
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