Listen to your Heart, or at Least theirs: Maintaining a high index of suspicion for infective endocarditis

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Introduction

After an endothelial injury, a fibrin and platelet cap forms. This cap is typically sterile but in times of infection can become colonized by microbes leading to a vegetation. When this occurs on the endothelium of the heart, it is termed infective endocarditis (IE). IE is a difficult diagnosis to make in the ED. The symptoms are vague. Classic exam findings are often absent, murmurs auscultation can be challenging and there is not a single diagnostic test. A delayed or missed diagnosis can be fatal; therefore, it is essential that ED physicians maintain a high index of suspicion in high risk patients (IVDA, valve disease, congenital heart disease, prosthetic valves and other cardiac devices) presenting with fever and/or general viral-like symptoms. Despite significant advances in medicine, the mortality and incidence of IE have not decreased over the last 30 years. This maybe in part due to diagnosing cases late in the disease course and IVDA associated with the ongoing opioid epidemic. Some of the sequelae include heart failure, septic emboli, intracerebral hemorrhage and coagulation cascade disturbances. Here we discuss a case of severe infective endocarditis and the unfortunate complications and outcome.

Case Description

44-year-old female with history of IVDA presented to ED with progressively worsening fever, myalgias, dyspnea and chest discomfort for 1-2 weeks. History limited 2/2 poor historian and no family or friends at bedside. On chart review, she was seen at a local free-standing ED 6 days prior with similar symptoms and discharged with a diagnosis of viral illness and PCP follow up. This chart played a vital role by aiding in the history and illness progression. Key comparisons outlined below.

<table>
<thead>
<tr>
<th>1st ED visit</th>
<th>2nd ED visit</th>
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<tbody>
<tr>
<td>38.4°, 80rpm, 120/68, 18RR, 100% on RA</td>
<td>38.2°, 122bp, 112/68, 20RR, 99% on RA</td>
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<tr>
<td>No acute distress</td>
<td>Uncomfortable, mild acute distress</td>
</tr>
<tr>
<td>RRR, no murmur</td>
<td>Tachycardic, harsh holosystolic heard throughout precordium</td>
</tr>
<tr>
<td>Reproducible chest wall tenderness</td>
<td>Diffuse musculoskeletal tenderness</td>
</tr>
<tr>
<td>No extremity cyanosis or edema</td>
<td>Bilateral hand erythema, 1+ pitting edema, feel cool to touch without cyanosis</td>
</tr>
<tr>
<td>No rashes</td>
<td>Petechial rash over RLO extending to right lateral hip</td>
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Neurosurgery placed external ventricular drain and transfused patient. The following morning both sets of blood cultures grew S. aureus and a TTE confirmed severe mitral regurgitation with vegetations on the anterior and posterior leaflets. Ultimately patient was declared brain dead and terminally exubated.

Hospital Course

Immediate concern for sepsis with IE source. Within the first ED hour, 2 sets of blood cultures were drawn and IV cefepime, IV vancomycin, and NS fluid boluses were administered. The diagram below shows the first 8 hours after ED arrival.

Troponin 0.475
EKG V3-V6 depressions, 324mg asa
NT-proBNP 18,533
Cr 1.38, proteinuria, hematuria
1st WBC 9.5, 92%, neutrophils, 44% bands, pts 20, PT 17.6, INR 1.8, fibrinogen 204
1B RLS, L5 tight on exam, MRI showed mass

D-dimer >30, R popliteal focal DVT, argatroban ggt, loss of distal RLE pulses, concern for acute limb ischemia (ALI) develops

Cath lab for ALI, 2k heparin bolus, intubated for discomfort & AMS, unsuccessful PTA & thrombectomy, IA IPA given

Stroke alerted, unequal pupils

Hospital Course

CT brain non contrast from admission compared to CT brain non contrast stroke alert IA filter

Discussion

The modified Duke criteria for diagnosing is not compatible with an ED diagnosis yet ED physicians are often the first involved in the care of IE patients. Delayed diagnosis and treatment leads to complications and poor clinical outcomes. While fever, sepsis and a new murmur are “textbook” for IE, studies estimate only 50% of patients have an elevated WBC and ~60% a new murmur. Understanding the pathophysiology (Figure 1) and disease process can aid in an ED clinical diagnosis.

The American Heart Association’s IE guidelines recommend an echo within the first 12hrs and at least 3 sets of blood cultures with the first and last set drawn 1 hour apart. In the critically ill, serial cultures should not delay antibiotics and the set of 3 can be obtained over a 5-20 minute duration. Stroke and thrombosis prophylactic anti-coagulation is not recommended in IE. Anti-coagulation for a pre or coexisting diagnosis is controversial and should be individualized to the case due to the risk of intracerebral hemorrhage.

Conclusion & Key Points

What is our role as EM physicians? Early clinical diagnosis and high index of suspicion are crucial. While majority of flu-like symptoms are from the flu, consider IE in patients with risk factors. Listen to your patients, even the poor historians, by auscultating. Prompt antibiotics after blood cultures(x3) can be lifesaving. Lastly, many potentially fatal complications arise from IE, some of which would warrant anticoagulation in a different setting; however, anticoagulation in IE should be approached very cautiously due to the high rate of intracerebral hemorrhage.

References

[1] North Florida Regional Medical Center, Gainesville, FL;    2 University of Central Florida College of Medicine, Orlando, FL


[5] Sayan E, Randhawa, M.S., Pile, J., Gomes, M. Can patients with infectious endocarditis be safely anti coagulated? Cleveland Clinic Jou

