

INTRODUCTION

Pure sensory stroke (PSS) is a lacunar syndrome affecting various areas of the somatosensory system. PSS is defined as a specific type of stroke displaying prominent hemisensory symptoms without other major neurological deficits. [1] While thalamic stroke remains the most common cause of PSS, it can also manifest secondary to small non-thalamic lesions involving the cerebral cortex, internal capsule or brainstem. [2] Unfortunately, brainstem lesions remain difficult to identify due to their relatively small size and may take days to weeks before changes are evident on imaging studies. [2] Brainstem pure or predominant sensory strokes can present with mild transient non-sensory symptoms, most commonly dizziness and gait ataxia. [3] Acute ocular pain has also been implicated in impending brainstem ischemia. [4, 5, 6] This case report highlights a pure sensory brainstem stroke with subtle clinical features that help to localize its origin within the brain. It stresses the importance of performing an accurate history and thorough clinical exam while maintaining a high index of suspicion for brainstem lesions.

CASE DESCRIPTION

A 40-year-old male presented to the emergency department with a one-day history of acute onset right upper and lower extremity numbness/tingling. Prior to the paresthesia onset, the patient experienced transient mild gait ataxia and left ocular burning pain sensation with complete resolution at the time of presentation. The patient denied any trauma or previous episodes of similar symptoms.

- **Past Medical Hx:** denied
- **Family Hx:** father CVA x 2 (between 40-50 years-old)
- **Social Hx:** denied EtOH, tobacco, illicit drug use

Vitals: BP 165/111, HR 89, RR 16, SaO2 98% RA, Temp 98.6F, BMI 34.6

Neurologic Exam

- Persistent light touch (brush) impairment localized to the right fingertips extending to the elbow and right toes extending to the knee (Figure 1).
- Minimal transient proprioception and vibration impairments on the distal right fingertips and toes.
- Both pain (pin prick) and temperature sensations remained intact.
- Muscle strength and tone were grossly normal.
- No other focal neurological deficits.

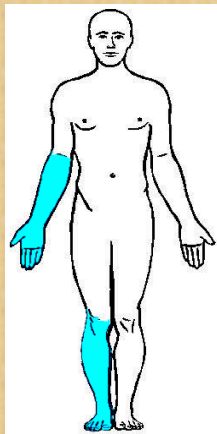


Figure 1. Illustration depicting the localized distribution of right-sided light touch impairment (bright turquoise).

INVESTIGATIONS

Routine admission blood tests revealed an elevated glucose of 255 mg/dL. All other initial laboratory tests were within normal limits. Electrocardiogram revealed normal sinus rhythm. Urgent non-contrast CT brain and contrast-enhanced CTA head/neck were both unremarkable. On the following day, MRI brain with and without contrast revealed a small left posterior infarct within the brainstem at the junction between the pons and midbrain (Figure 2). MRI of the cervical spine with and without contrast showed multilevel degenerative changes with moderate-severe foraminal stenosis on the right at C5-C6 and on the left at C6-C7 with exiting nerve root impingement. In light of the MRI cervical spine findings, neurosurgery was consulted and recommended medical management without any need for surgical intervention.

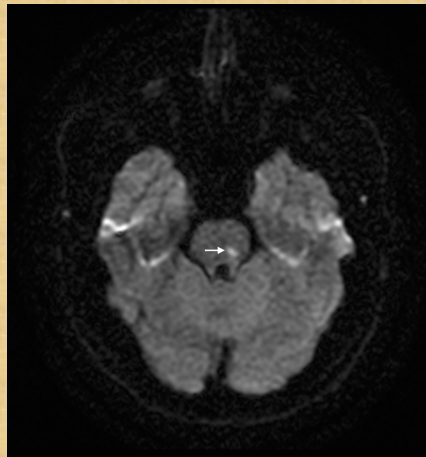


Figure 2. MRI (Axial DWI) of brain with and without contrast demonstrating a small left posterior infarct within the brainstem at the junction between the pons and midbrain (white arrow).

Further testing with echocardiogram and hypercoagulability studies were unremarkable. Dual-antiplatelet therapy was initiated and the patient experienced minimal paresthesia improvement proximally over his five-day hospital course. Risk factors for stroke were addressed and the patient was found to have multiple risk factors:

- Positive family history of father with two strokes at a young age
- Newly diagnosed hypertension
- Newly diagnosed hyperlipidemia (TG: 283 mg/dL, HDL: 24 mg/dL)
- Newly diagnosed diabetes mellitus (HgbA1C: 11.0%)

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DISCUSSION

In general, brainstem strokes will have more sensory deficits if the lesion is localized posteriorly and more motor deficits if the lesion is localized anteriorly. [7] On review of the literature, the main mechanism of brainstem infarcts presenting with predominant early-onset paresthesia strongly suggests involvement of the medial lemniscus tract in the paramedian posterior pontine region. [3] While thalamic lesions are the most common cause of PSS, pure lemniscal sensory deficits associated with the thalamus are uncommon. [2] Thalamic lesions typically have additional involvement of the spinothalamic tract due to the wide projection of spinothalamic fibers to numerous thalamic nuclei. [8] However, the medial lemniscus and spinothalamic tracts within the brainstem remain separate with their proximity varying from level to level. In the medulla, the medial lemniscus and the spinothalamic tracts are well separated from each other. As these tracts continue to ascend to the pons and inferior midbrain, they get much closer and more likely to become affected simultaneously. [7, 9] Small lacunar infarcts still have the ability to affect very localized regions within a single tract. Given the minimal hemisensory neurological deficit and small posterior brainstem infarct on MRI, it is reasonable to conclude that the lesion affected the medial lemniscus tract without spinothalamic tract involvement.

In addition, of particular interest was the transient mild gait ataxia and ocular eye pain that preceded the paresthesia onset. Previous studies have reported gait ataxia and dizziness as the most frequent minor non-sensory signs/symptoms involved with PSS of the brainstem. [3] Researchers have postulated that the cause of gait ataxia may be secondary to the proprioceptive sensory deficit or potential pontocerebellar fiber involvement. [7] Regardless, these non-sensory symptoms are often cited to be mild and short-lived compared to the predominant hemisensory symptom. In addition, acute ocular pain has been implicated as a rare feature of impending brainstem ischemia. Previous investigations have described this sensation as sharp, burning or "salt and pepper" in or around the eyes. Researchers suggest involvement of the quintothalamic tract in the pons as the primary cause. [4, 5, 6]

CONCLUSION

This case report highlights an acute brainstem stroke presenting with predominant hemisensory symptoms. Presentations of brainstem lesions can range from subtle, non-specific features to profound deficits. It can present with associated transient non-sensory symptoms, most notably gait ataxia and acute ocular pain in this particular case. Clinical recognition and localization of the lesion can help expedite an accurate diagnosis with early treatment initiation and prevention of devastating neurological deficits. This case serves to emphasize the importance of performing a thorough clinical exam while maintaining a high index of suspicion for brainstem lesions.