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Case report

Pure sensory stroke due to brainstem lesion

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SUMMARY

A 40-year-old male patient presented to the emergency department with acute onset right-sided upper and lower extremity numbness/tingling over the past day. Prior to the paraesthesia onset, the patient experienced transient mild ataxia and left ocular pain with complete resolution at the time of presentation. Neurological exam revealed isolated right-sided sensory changes from his hand-to-elbow as well as foot-to-knee. No other focal neurological deficits were noted. MRI brain revealed a small left posterior infarct at the junction between the pons and midbrain. Dual-antiplatelet therapy was initiated and the patient experienced minimal paraesthesia improvement proximally over his 5-day hospital course. 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. This case serves to emphasise the importance of performing a thorough clinical exam while maintaining a high index of suspicion for brainstem lesions.

BACKGROUND

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 are usually caused by a paramedian dorsal pontine lesion involving the medial lemniscus tract and 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 ischaemia.[4–6] This case report highlights a pure sensory brainstem stroke with subtle clinical features that help to localise 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 PRESENTATION

A fit-and-well 40-year-old male patient (construction worker) presented to the emergency department with a 1-day history of acute onset right upper and lower extremity numbness/tingling. The patient reported associated transient symptoms of mild gait ataxia and left ocular burning pain sensation just prior to the paraesthesia onset. These associated symptoms were completely resolved at the time of presentation. The patient denied any trauma or previous episodes of similar symptoms. Although the patient denied any personal medical history or home medications, family history revealed that his father suffered from two strokes between the ages of 40 and 50 years old. The patient denied any alcohol, tobacco or illicit drug use.

On general examination, the patient was a muscular Caucasian male with a body mass index of 34.6. He was afebrile but noted to be hypertensive with a blood pressure of 165/111 mm Hg. Neurological exam revealed persistent light touch (brush) impairment localised to the right fingertips extending to the elbow and right toes extending to the knee. In addition, the patient displayed 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 were appreciated.

INVESTIGATIONS

Routine admission blood tests revealed an elevated glucose of 255 mg/dL. All other initial laboratory tests were within normal limits. ECG revealed normal sinus rhythm. Urgent non-contrast CT of the brain and contrast-enhanced CT angiogram of the head/neck were both unremarkable. On the following day, MRI of the brain with and without contrast revealed a small left posterior infarct within the brainstem at the junction between the pons and midbrain (figure 1). 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. Further investigations were performed to help delineate any underlying causes for stroke in such a young patient.

Subsequent laboratory testing revealed haemoglobin A1C at 11.0% and lipid profile with elevated triglyceride level at 283 mg/dL and decreased high-density lipoprotein level at 24 mg/dL. Urine toxicology was unremarkable. Transthoracic echocardiogram showed normal left ventricle size and systolic function without any regional wall motion abnormalities. Follow-up transoesophageal echocardiogram revealed normal left ventricular ejection fraction between 60% and 65% and left atrial appendage free of thrombus without any evidence

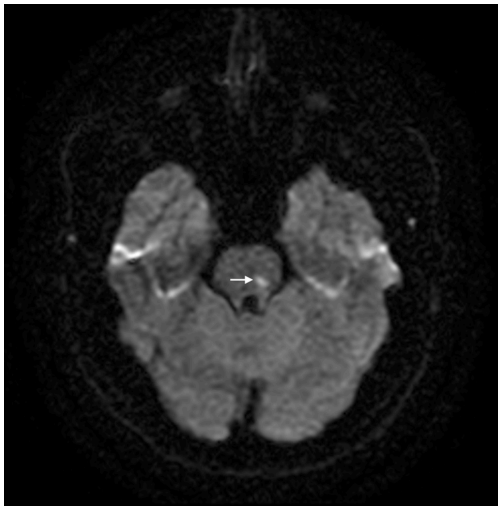

Figure 1 Contrast-enhanced MRI (axial diffusion-weighted imaging (DWI)) of brain demonstrating a small left posterior infarct within the brainstem at the junction between the pons and midbrain (white arrow).

of interatrial shunt. Additional hypercoagulability workup with genetic testing was negative for the following: prothrombin/factor II mutation, anti-thrombin III antigen/activity, cardiolipin antibody (IgA, IgG, IgM), lupus anticoagulant, protein C/S antigen/activity and antinuclear antibody (ANA).

DIFFERENTIAL DIAGNOSIS

On initial presentation to the hospital, the most likely diagnosis was a stroke versus unlikely radiculopathy. Although the patient was young and denied any comorbidities, his localised hemisensory symptoms were concerning for multiple reasons. For one, the patient reported that his symptoms started acutely and remained constant for greater than 24 hours. The patient also denied any trauma or previous episodes of similar symptoms. Examination revealed pure sensory deficits with distinct localisation to the distal portions of the right upper and lower extremities. While initial neuroimaging studies were unremarkable, the patient's history combined with his continued unilateral hemisensory deficits warranted further workup to rule out stroke.

Subsequent MRI of the brain confirmed the diagnosis of a small left-sided ischaemic brainstem stroke. Although MRI of the cervical spine did reveal multilevel degenerative changes, these cervical findings did not account for the full extent of the patient's hemisensory deficits. Risk factors for stroke were addressed and the patient was found to have multiple risk factors. The patient had a positive family history of a father with two strokes at a young age as well as newly diagnosed hypertension, hyperlipidaemia and diabetes mellitus. Further testing with echocardiogram and hypercoagulability studies were unremarkable. In conclusion, the ischaemic stroke was attributed to his extensive list of stroke risk factors, many of which were undiagnosed at the time of presentation.

TREATMENT

The patient was administered aspirin 325 mg in the emergency department. After acute brainstem stroke was identified on MRI brain, the patient was started on atorvastatin and clopidogrel for secondary prevention. The patient was also started on lisinopril and insulin for newly diagnosed hypertension and diabetes mellitus. In light of the MRI cervical spine findings, neurosurgery was consulted and recommended medical management without any need for surgical intervention.

OUTCOME AND FOLLOW-UP

The patient was discharged home after a 5-day hospital course with minimal paraesthesia improvement proximally over this time. Discharge prescriptions included aspirin 81 mg daily, clopidogrel 75 mg daily for 3 months, atorvastatin 80 mg daily, lisinopril 5 mg daily and insulin glargine 25 units nightly with insulin lispro 5 units three times per day before meals. The patient was instructed to follow-up with his primary care physician, cardiology and neurology within 1 week.

DISCUSSION

The brainstem is the posterior portion of the brain that remains continuous with the spinal cord. It is composed of the midbrain, pons and medulla. These subdivisions are responsible for cranial nerve III–XII activity, nerve conduction via ascending and descending pathways and integrative functions including vasomotor control, breathing, swallowing, sleeping and maintaining consciousness. Strokes within the brainstem can impair any of these functions. Depending on the size and location of the lesion, patients can present with mild symptoms to profound, life-threatening deficits. This underscores the importance of having a basic understanding of the brainstem anatomical structures along with the ability to perform a thorough history and neurological examination. Clinical recognition and localisation of the lesion can help expedite an accurate diagnosis with early treatment initiation.

In general, brainstem strokes will have more sensory deficits if the lesion is localised posteriorly and more motor deficits if the lesion is localised anteriorly.[7] On review of the literature, the main mechanism of brainstem infarcts presenting with predominant early-onset paraesthesia 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 fibres 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 localised regions within a single tract.

This case presentation has described a young man who developed acute onset right-sided hemisensory symptoms without any other major neurological deficits and was diagnosed promptly with a small left posterior brainstem stroke at the junction of the midbrain and pons. These sensory deficits were precisely localised to the right fingertips-to-elbow and right toes-to-knee. Patient displayed persistent light touch (brush) impairment and minimal transient proprioception/vibration impairments distally. Both pain (pin prick) and temperature sensations remained intact. 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.

Of particular interest in this case presentation was the transient mild gait ataxia and ocular eye pain that preceded the paraesthesia

Learning points

- Brainstem infarctions have diverse presentations ranging from subtle, non-specific features to profound deficits.
- Brainstem infarctions are often overlooked unless one maintains a high index of suspicion.
- Pure sensory stroke (PSS) from brainstem lesions most commonly involve the medial lemniscus tract in the paramedian dorsal pontine region.
- Gait ataxia and dizziness are common non-sensory features of PSS that are typically mild and short-lived.
- Acute ocular pain can be a rare presenting feature of impending brainstem ischaemia.

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 fibre involvement.[7] Regardless, these non-sensory symptoms are often cited to be mild and short-lived compared with the predominant hemisensory symptom. In addition, acute ocular pain has been implicated as a rare feature of impending brainstem ischaemia. 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.[4–6]

Risk factors for brainstem strokes are the same as risk factors for any type of stroke. These major risk factors include hypertension, hyperlipidaemia, diabetes mellitus, smoking, obesity, physical inactivity, atrial fibrillation, carotid artery stenosis, older age, African American race, family history and genetic disorders.[10] The patient in the case report had multiple stroke risk factors. These included a positive family history of father with multiple strokes at a young age, obesity and undiagnosed hypertension, hyperlipidaemia and diabetes mellitus. Although further hypercoagulability and genetic testing were unremarkable, these undiagnosed risk factors likely contributed to the patient developing a stroke at such a young age.

Antiplatelet therapy is recommended after an ischaemic stroke, including large artery atherosclerosis as well as small vessel disease. Current first-line antiplatelet options for secondary stroke prevention include either clopidogrel monotherapy, aspirin monotherapy or aspirin in combination with dipyridamole.[11 12] However, the combination of aspirin and clopidogrel has been shown to reduce the risk of stroke in the first 90 days without increased risk of haemorrhage.[12 13]

In conclusion, this case presentation highlights an acute, small brainstem stroke presenting with predominant hemisensory symptoms. These lesions most commonly affect the medial lemniscus tract in the paramedian dorsal pontine region. It can present with associated transient non-sensory symptoms, most notably gait ataxia and acute ocular pain in this particular case. Prompt neurovascular evaluation and treatment can help to prevent devastating neurological deficits. This case serves to emphasise the need to maintain a high index of suspicion for PSS involving the brainstem, as small lesions can present with minimal hemisensory neurological deficits.

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