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Artery of Percheron Infarction, a Rare Presentation; a Case Report

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Abstract

Posterior fossa strokes, namely "Strokes in the territory of the posterior cerebral artery," remain a challenging diagnosis. Its variable stuttering symptoms and non-focal manifestations result in a delay of its diagnosis and poor outcome. Artery of Percheron (AOP) is an anatomical vasculature variant arising from the P1 segment of a Posterior Cerebral Artery (PCA) as a single dominant thalamic perforating artery that supplies bilateral paramedian thalami and the rostral mesencephalon. Bilateral thalamic infarcts can present with altered mental status, hypersomnolence up to coma, aphasia, memory impairment, and vertical gaze palsy. Given the lack of classic stroke signs, the majority of AOP infarcts are not diagnosed in the emergency setting. Diagnosis is usually made following an MRI brain scan, which is usually obtained outside the therapeutic window for IV tissue plasminogen activator (tPA) administration. Timely vessel imaging is critical for diagnosis and initiation of treatment in those patients. This case highlights the rare presentation and diagnostic difficulty of a patient with an AOP infarction and reminds us to include thalamic pathology in patients presenting with nonspecific neurological symptoms and no obvious signs of stroke.

Introduction

There are three anatomic variations of artery of Percheron, a single dominant thalamic perforating artery to the paramedian thalamic and mesencephalic region [1]. The prevalence of AOP is still unknown; 0.6% estimation percentage of ischemic strokes cases in an ischemic stroke registry of 2,750. The presentation of AOP infarction varies widely due to the complex functions of the human thalamus and midbrain structures and it is characterized by non-focal neurological deficits, altered mental status, memory impairment, and decreased level of consciousness being among the most common [2]. in this case report we presented a rare form of stroke whose presentation can mimic many other conditions, including seizures. on initial computed tomography (CT) scan AOP infarction is usually missed, and further imaging isn't usually done due to low suspicion for stroke. A large number of reports of AOP infarction illustrated a diversity of its clinical presentations. Diagnosis is usually made following an MRI brain scan, which is usually done outside the window period for IV tissue plasminogen activator (tPA) administration. the time of diagnosis of an acute bilateral thalamic infarct can be challenging, and this case report draw attention to the non-common neurological presentation of AOP infarction. We propose an approach with a high suspicion of an AOP infarction, including an extensive diagnostic radiological approach for patients to be conducted in the emergency department in a time-sensitive manner. A large number of the underlying the cause of posterior circulation strokes remain cryptogenic. Traumatic vertebral artery dissection is one of the most common causes and should be suspected in patients presenting with cervical pain neurological deterioration. Also, other causes include cervical spine or skull base fracture, cervical instability [3] arteritis, meningitis, aneurysms, and neurosyphilis [4]. Behcet vasculitis more commonly involves the posterior circulation [5].

Case Presentation

A 37-year-old Saudi female had no significant past medical history apart from gestational diabetes and history for recurrent painful oral ulcers, red-eye, and severe abdominal pain. Few weeks prior to her presentation to our emergency department in Aseer central hospital (ACH) patient had prodromal symptoms with a stuttering course of intermittent headaches, vomiting, and vertiginous episodes upon which admitted in her nearby hospital and underwent full investigation for five days that came up to be unremarkable. The patient presented as a transfer from another hospital; she was brought to our emergency department with the same episode but in a hypersomnolence state, poorly responsive with a non-focal neurological examination. She was last seen normal 5 hours prior to her arrival to ER. Her husband reported that She developed at home a self-terminating episode of loss of consciousness associated with deviation of angle of mouth to left side and drooping of both eyelid associated with double vision with generalized weakness.

Her vitals on arrival were; blood pressure was 164/90 mmHg, heart rate was regular with an average of 88 per minute, respiratory rate was 22 breaths per minute, and she was afebrile. On exam, she appeared drowsy, nonverbal, and intermittently following one-step commands. As regard cranial nerves examination, ptosis was noted bilaterally but more on the left eye with dilated fixed-left pupil and normal-sized right pupil. Pupillary light reflex in the left eye was non-reactive while sluggish reactivity in the right eye. Ocular movements examination was normal, apart from complete upward gaze palsy and nystagmus. The findings are consistent with bilateral partial third cranial nerve palsy. On motor exam, she had mild generalized weakness but was able to move all extremities against gravity. The sensory exam was confounded by her decreased mental status. Bilateral plantar reflexes were equivocal. National Institute of Health Stroke Scale (NIHSS) was 10. She was out of the 4.5-hour time window to consider IV thrombolysis therapy, and on the exam, her presenting symptoms did not localize to one cerebral vascular territory. Initial diagnostic work-up: serum white blood cell count 5000/uL, hemoglobin 13.3g/dL, platelets 221/uL, sodium 138mmol/L, potassium 4.1mmol/, blood urea nitrogen 14.6mg/dL, creatinine 0.6 mg/dL, glucose 93mg/dL, troponins <7ng/L, aspartate aminotransferase 13.4 U/L, and alanine aminotransferase14U/L, Erythrocyte sedimentation rate 50 mm/hr and positive C-reactive protein, as regard lipid profile total cholesterol level 121mg/dl, cholesterol VLDL 21 mg/dl triglycerides 105 mg/dl.

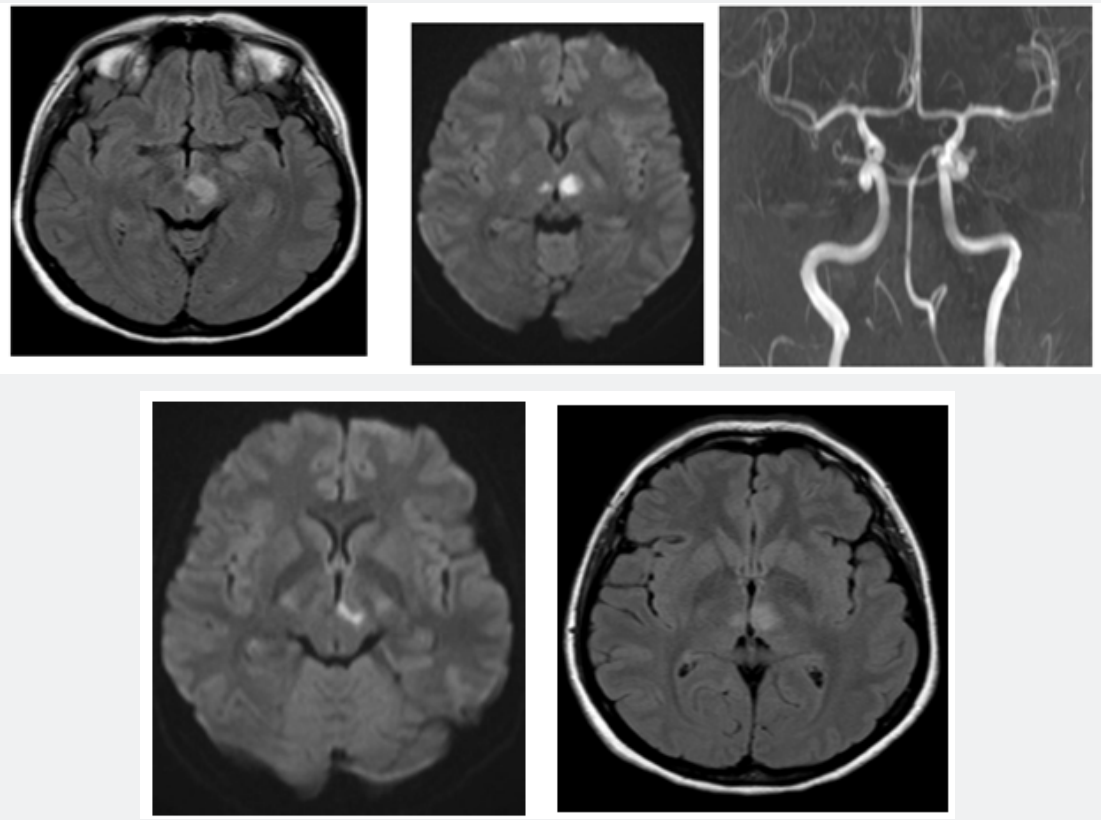

Figure 1: MRI brain showed bilateral paramedian thalamic infarcts extending into the midbrain with a reduced signal on the apparent diffusion coefficient images indicating restricted diffusion in keeping with acute bilateral medial thalamic infarcts and petechial micro-hemorrhagic in the midbrain.

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The non-contrasted CT brain was unremarkable. A CT angiogram (CTA) demonstrated a hypoplastic right vertebral artery with non-flow segment, an Artery of Percheron (AOP) couldn't be properly visualized (Figure 1) with no large vessel occlusions. An electroencephalogram (EEG) was requested early in the workup to support the initial diagnosis of seizure but was unfortunately not immediately available. MRI brain done on an urgent basis demonstrated bilateral paramedian thalamic infarcts extending into the midbrain with a reduced signal on the apparent diffusion coefficient images, indicating restricted diffusion in keeping with acute bilateral medial thalamic infarcts and petechial micro-hemorrhagic in the midbrain. Once this finding had been made EEG was no longer considered necessary for diagnostic purposes. A thorough cardiac investigation revealed her ejection fraction was 65% with no atrial septum shunt on a transthoracic echocardiogram. Twenty-four-hour ECG showed normal sinus rhythm with no evidence of arrhythmias. Autoimmune profile and thrombophilia screen were done and came up negative. Her stroke etiology was thought to be secondary to the artery to artery embolus. MRI brain performed later on the same day of admission showed increased signal on the B1000 diffusion images within the medial thalami bilaterally

Discussion

This case of AOP infarction is highly unusual because its clinical presentation mimicked seizure activity, the cognitive impairment overshadowed, and was the predominant clinical manifestation, with minimal motor impairment. It was later shown to be transient disturbances of consciousness due to arterial infarction. Clinicians should be aware that seizures as a presenting feature of acute stroke are not uncommon, the frequency ranging from 1.5% to 5.7% [1]. However, these tend to occur in younger patients and those with hemorrhagic rather than ischemic strokes. There was no evidence on her initial CT scan of acute hemorrhage or early signs of ischemia. She took no regular medication, and there was no history of alcohol use. Serum glucose, urea, sodium, and calcium were also normal, and there was no evidence of infection. An unprovoked seizure was also considered a possibility in this case. In particular, complex partial seizures may present with atypical features such as episodes of confusion or apparent syncope [3]. With the absence of positive phenomena such as aura or postictal confusion and normal serum lactate, the seizure did not seem likely. Her ability to localize to painful stimuli indicated reduced awareness rather than seizures. MRI brain revealed bi-lateral thalamic involvement consistent with AOP infarction. However, bilateral abnormalities of the thalamus with basal ganglia involvement on MRI can be due to a variety of systemic and focal etiologies. These include toxic poisoning by methanol, carbon monoxide, and cyanide.

In this case, there was no history of accidental exposure to these toxins or suicidal attempts. Arterial and venous blood gas was normal, making toxic poisoning an unlikely cause of the patient's symptoms. Systemic metabolic disorders such as hypoglycemia and hyperglycemia, chronic liver disease, Wernicke's encephalopathy, and Wilson's disease can also be differential diagnoses [4]. These were unlikely due to the sudden onset of symptoms and normal liver function tests in an otherwise healthy individual [2,6-10]. There can be other types of infarctions that may result in bilateral thalamic lesions apart from the AOP infarcts; these include Top of basilar artery syndrome that was area supplied by posterior cerebral, pontine arteries and superior cerebellar are also involved when the basilar artery is occluded in addition to bilateral paramedian thalami. Another differential is cerebral venous sinus thrombosis (CVST), as a result of occlusion of internal cerebral veins. It may be present with headache, vomiting, and papilledema and will result in infarction that can present with seizures, focal neurological deficits, and aphasia. Wernicke's encephalopathy is a differential diagnosis because it results in lesions in bilateral thalami, periaqueductal gray tectal plate, dorsal medulla, and mammillary bodies. Infections such as meningitis and encephalitis also osmotic myelinosis, vasculitis, and cerebritis. A diagnosis of AOP infarction was made based on the correlation between MRI findings and the patient's clinical presentation.

AOP infarct is uncommon causing 0.6% of all strokes. The resulting strokes can result in severe disability.6 Timely diagnosis allows the use of acute interventions to recanalize the vessel, which can include intravenous thrombolysis, intra-arterial thrombo- lysis, and mechanical endovascular procedure which improve their functional outcomes [6,7]. The time window for intervention has not been established for posterior fossa strokes, but endovascular interventions in published series have often been longer than the typical windows used in the anterior circulation. Time to intervention is likely less important than the degree of baseline ischemia, which can be assessed on plain CT using posterior circulation Acute Stroke Prognosis Early CT score.6The diagnosis of AOP is complicated not only by its relative infrequency and limited exposure among clinicians but also by its variability in clinical presentation. In another case series of 85 patients with basilar artery occlusion, 53 patients were later documented to have had prodromal symptoms ultimately attributed to the basilar artery occlusion [10]. The most common prodromal symptoms were vertigo and nausea followed by neck pain and headache. A notable portion of their patients also developed early dysarthria, diplopia and hemiparesis. Most commonly, these patients had symptom onset about 2 weeks before a formal diagnosis. Prodromes, minor strokes, or fluctuating symptoms described in 40% to 50% of patients in other series [8,9].

Conclusion

The clinical setup in our patient was altered mental status, memory impairment and hypersomnolence without any focal deficits. Such symptoms pointed more towards a non-focal neurological cause. This case drow attention to the importance

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of thorough evaluation to rule out systemic causes while at the same time keep in mind that it could be an atypical presentation of stroke, especially involving the midbrain and thalami. The investigations should be directed towards evaluating the etiology stepwise to optimize patient care and outcome.

Summary

The morbidity and mortality of posterior fossa stroke remain high, despite advances in stroke prevention and treatment in ischemic stroke. There should be a high suspicion for a patient with dizziness plus any other neurological deficit, particularly fluctuating level of consciousness, diplopia, or unsteady gait. The best treatment and optimal treatment time are unknown but reperfusion approaches involving IV t-PA and/or endovascular therapy are appropriate within 4.5 hours.

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