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Case Report

A rare variant of thalamic stroke- artery of Percheron infarct

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ABSTRACT

The thalamus is a walnut-sized structure that is located in the brain which receives rich blood supply from posterior cerebral artery and its communicating branches. One of the unusual presentations is the infarction of artery of percheron. Hereby reporting a case of 68-year-old female with no known co-morbidities who presented to the emergency department with complaints of sudden onset loss of consciousness in the morning. On further investigation, was found to have infarction of one of the variants of thalamic perforating arteries.

Keywords: Artery of percheron, Sudden loss of consciousness, Vertical gaze palsy, Memory impairment

INTRODUCTION

The thalamus is a walnut-sized structure that is located in the brain that receives blood supply from posterior cerebral artery and its communicating branches.[1] The paramedian territories are supplied by communicating arteries and hence referred as paramedian arteries.[2] There are 4 variants of this neurovascular supply to thalamus and midbrain. Variant I where perforating branches arise from right and left posterior cerebral arteries. Variant IIa is where the left P1 segment is the source of both paramedian arteries. In variant IIb, the perforating arteries arise from the artery of Percheron (AOP) which supplies the paramedian thalamus and rostral midbrain.[3] In these variants, the AOP comes from a part of the posterior cerebral artery, namely, the P1 segment. Variant III is the arcade variant which gives off small perforating branches from one arterial arc which bridges the P1 segments and the PCAs together. The incidence of infarct in artery of percheron is seen in 0.6-1.2% of the population, signifying its rarity.[4]

CASE REPORT

A 68-year-old female with no known comorbidities was brought to the casualty at around 12:30 pm with complaints of one episode of sudden loss of consciousness in the morning at around 7:30 am at her home while she was standing in the kitchen. She was found in her usual state of health by her son in the morning around 4 am. He witnessed her getting up for using the restroom and after which she complained of having headache. On arrival to the emergency room, her airway was patent and she was maintaining her saturation. On further neurological assessment, her GCS was found to be E1V1M3 (5/15). Bilateral pupils were 2 mm in size and sluggishly reacting to light. Gag reflex couldn't be elicited. She responded to deep pain by moving her right upper and lower limbs. Oculocephalic reflex was intact. Her blood pressure was 130/90 mmHg and capillary blood glucose was 158 mg/dl on arrival. Metabolic parameters were normal. She was obtunded and her bilateral plantar reflex was found to be mute. On arrival, her NIHSS score was 26 points. Electrocardiography (ECG) showed normal sinus rhythm. Computed tomography of brain was done at around 8:40 am in the nearby hospital and was found to be normal. In suspicion of vascular stroke, magnetic resonance imaging (MRI) brain was done which showed multifocal areas of diffusion restriction with corresponding ADC low signal noted in the bilateral paramedian thalamus and midbrain (cerebral peduncles) which shows no altered signal intensities on t2/flair and hypointense on T1 sequences with no evidence of GRE blooming and chronic infarct

noted involving right caudate nucleus and left thalamus and small vessel ischemic changes. These features were consistent with artery of Percheron infarct. In view of delayed presentation, lysis with tissue plasminogen activating factor (tPA) was deferred. Neurosurgery and neuromedicine consultation were obtained and was advised to manage conservatively. Two-dimensional echocardiography was done and was found to be normal.

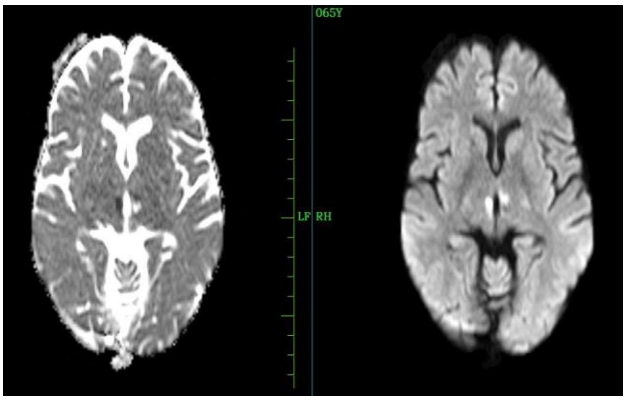

Figure 1: MRI brain showing bilateral low signal intensities over bilateral paramedian thalamus.

Figure 2: Image showing right sided complete drooping of eyelid.

Figure 3: Image showing upward vertical gaze palsy.

Management

After confirmation of the diagnosis, intravenous access was secured and ryles tube was inserted. Patient was catheterized. Inferior venacava collapsibility was seen and patient was adequately hydrated and started on dual antiplatelets (clopidogrel 75 mg+aspirin 150 mg) and high intensity statin (atorvastatin 40 mg) and other neuroprotective drugs like piracetam and multivitamins. Patient's GCS started to improve in less than 48 hours of admission. On examination of extra-ocular movements, there was restriction noted to upward gaze. Patient was started on oral feeds slowly. Speech and swallow assessment was done. Limb and speech physiotherapy were provided. Patient's gaze palsy improved slowly over 3-4 weeks. On day 3 of admission, patient's NIHSS score was 7 points. There was complete drooping of the right eyelid indicating palsy of third cranial nerve corresponding to the midbrain. Patient was started on oral feeds on day 10 of admission. On assessing the memory, there was found to be mild impairment of remote memory. Patient's aphasia improved gradually in the next 3 months of speech therapy.

Figure 4: Image showing right medial rectus palsy.

DISCUSSION

Artery of percheron is an anatomical variant which arise as a solitary arterial trunk from posterior cerebral artery which supplies rostral midbrain and bilateral paramedian thalamus. This patient's presentation is one of the rarest in the literature of AOP infarcts. Prompt suspicion of thalamic stroke is important for early diagnosis and treatment.

Infarct of artery of percheron can cause bilateral thalamic involvement with or without involvement of midbrain and oculomotor nerve.[5]

The classical triad of artery of percheron infarct is altered mental status or reduced level of consciousness, vertical gaze palsy and memory impairment due to involvement of the anterior thalamus. It can also present with severe cognitive impairment and apathy. Dysarthria and

psychosis are also commonly reported secondary to rostral midbrain involvement.[6] These manifestations can be due to involvement of mediodorsal and intralaminar nuclei. In previously reported cases, initial computed tomography has not shown any infarcts probably due to involvement of small vessels.[7] The common etiologies associated with infarction of AOP are smoking, alcoholism, systemic hypertension, diabetes mellitus, any hypercoagulable states, like any other stroke.

CONCLUSION

Prompt suspicion and early diagnosis of any kind of thalamic stroke is considered important due to the diversity of clinical presentations and lack of omnious signs of stroke.[8] Hence adequate knowledge is important in identifying these bizzare presentations and treating them if presented within the time window with tissue plasminogen activating factor (tpa). Previous studies have demonstrated 40% cases to have cardiac emboli as their source.[9] This case report is of importance due to the rarity of its presentations and to aid physicians in early diagnosis and treatment.

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