

Case Report

Artery of percheron infarct: a case report

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ABSTRACT

Artery of Percheron is a part of the posterior circulation occlusion of which is relatively uncommon. It is classically characterised by bilateral infarcts in areas involving the rostral midbrain and/or ventromedial thalamus best seen by a diffusion-weighted imaging (DWI) sequence using MRI. Clinical presentations are variable and include, amnesic impairment, aphasia, dysarthria, ocular movement disorders, motor deficit and cerebellar signs. Our case was a 60-year-old hypertensive and diabetic male with history of alcohol abuse who presented with sudden derangement of sensorium along with restriction of ocular movements and marked cerebellar signs. The diagnosis of werniche encephalopathy suggested initially by the radiologist was rejected because of the acute onset, history of hypertension and marked cerebellar signs which suggested a cerebrovascular accident. Bilateral infarcts with the occlusion of a single artery i.e. artery of percheron which supplies structures bilaterally can easily be confused with werniche encephalopathy which has similar clinical and radiological picture but are managed on different lines. This diagnosis should be kept in mind in drowsy patients with restricted ocular movements and bilateral thalamic and midbrain hyperintensities.

Keywords: Artery of percheron, Bilateral thalamic and midbrain infarcts, Drowzy patient, Werniche encephalopathy

INTRODUCTION

The artery of Percheron is a rare variant of the posterior cerebral circulation. It is named after the French neurologist Gérard Percheron, who described it in 1973.¹ According to Percheron there are four normal variants of the neurovascular anatomy of the thalami and midbrain. Variant I is most common, where each perforating artery arises from each left and right PCA. Variant IIa is a less common, asymmetrical variant, where perforating arteries arise directly from the proximal segment of one of the PCAs. However, in some people, a single arterial trunk stems off the P1 segment of one of the posterior cerebral arteries and this trunk then divides to supply both thalami and the upper midbrain (type IIb); this is the artery of Percheron (Figure 1). It supplies the paramedian thalami and the rostral midbrain bilaterally (but variably).

In addition to the medial thalamic nuclei, these paramedian thalamic arteries supply the interpeduncular nucleus, the decussation of the superior cerebellar peduncles, the medial part of the red nucleus, the third and fourth cranial nerve nuclei and the anterior portion of the periaqueductal grey matter.² Consequently, occlusion of the artery of Percheron causes bilateral paramedian thalamic infarctions which can be considered pathognomonic of occlusion of the artery of Percheron with or without midbrain infarction. Therefore, Percheron artery exemplifies the presence of a small single artery that supplies bilateral vital structures. The complex anatomy and function of the human thalamus and its variable vascular supply are responsible for the variable clinical features when this structure is damaged by an ischemic infarction; in addition, the vascular overlap with the underlying midbrain will extend the spectrum of these

clinical features to include midbrain signs. The characteristic Percheron artery infarct pattern was estimated to occur in 1% and 3% of all stroke patients.³ Other small series have demonstrated Percheron artery infarct in 2% of ischemic strokes (of all stroke patients) and in the range of 4%-18% of thalamic strokes (of all stroke patients).⁴ Not many cases have been reported in literature.⁵⁻⁸

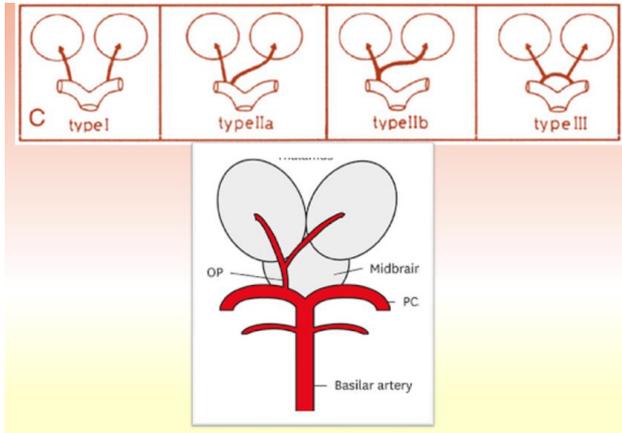


Figure 1: Paramedian artery: artery of percheron (AOP).

CASE REPORT

The patient was a 60-year-old male with history of hypertension and diabetes for the past 11 years and also was a chronic alcoholic who suddenly felt giddy and became unconscious. He was found to have a BP of 210/110mmHg and was taken to a local hospital and treated for 2 days but did not improve. CT scan was normal. The MRI showed linear hyperintensity extending from medial thalami and medial aspect of bilateral cerebral peduncle extending upto midbrain in periaqueductal region with suggested possibility of werniche encephalopathy/acute infarcts by the radiologist. After 2 days he was brought to SGRD medical college in a deeply comatose state with a BP of 180/100. On physical examination, Glasgow coma scale score was 3/15. Plantar reflexes were down going. Respiratory, cardiovascular, abdominal examination, blood picture and routine biochemistry were unremarkable. He was treated on the lines of acute infarct due to the typical clinical presentation. His sensorium improved after 3 days and he showed motor movements after 3 days when it was found that the patient had ptosis and could not lift both eyelids.

Also, the ocular movements were restricted on both sides. A diagnosis of bilateral 3rd N palsy was made. The motor power was grade 5 in all limbs. Patient had prominent bilateral cerebellar features with slurring of speech and titubation. His higher functions were normal and there was no memory disturbance. MRI was repeated with the same report of werniche encephalopathy by the radiologist. MRI showed areas of hyperintense signal in

dorsomedial thalamus, periaqueductal grey matter, in the paramedian location bilaterally more on the left side along with diffuse white matter ischemic changes and mild diffuse cerebral atrophy (Figure 2, 3). Patient remained drowsy during his stay in the hospital and was discharged after 15 days after he showed some improvement. On follow up after another 15 days he could stand with support, but the cerebellar signs were persisting bilaterally. The ptosis and oculomotor movements of right eye recovered by then but left sided oculomotor palsy persisted (Figure 4). The patient is on regular follow up.

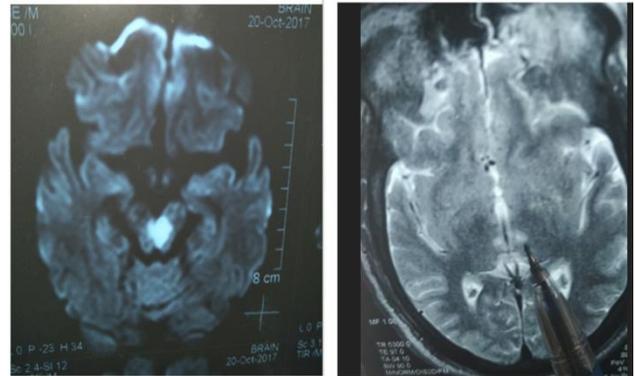


Figure 2: Infarcts seen as hyperintensities in midbrain and thalamus.

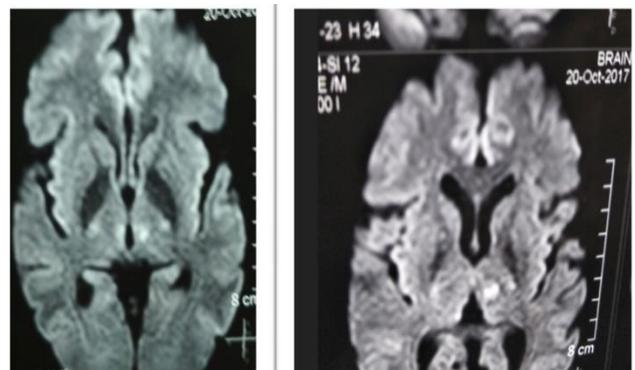


Figure 3: MRI showing thalamic infarcts.



Figure 4: Ptosis on left side due to 3rd n palsy.

DISCUSSION

Occlusion of the artery of percheron presents with a variety of signs and symptoms collectively termed the paramedian thalamic syndrome. Occlusion of this artery is rare and results in a multitude of neurological signs and which might prompt the physician to think of an inflammatory, infectious or malignant cause of this cerebral dysfunction. The functions of the thalamus and midbrain include the regulation of consciousness, sleep and alertness therefore thalamic involvement is usually characterized by a triad of vertical gaze palsy, memory impairment and coma. An ischemic stroke in the territory of an artery of Percheron usually presents with confusion, somnolence and loss of consciousness. Patients are often obtunded or agitated.

It can also produce a bizarre disturbance such as sleep from which the patient cannot be awakened. The altered mental status is explained by involvement of reticular activating system and the disrupted connections between the thalamus and the anterior, orbitofrontal and medial prefrontal cortices. The patients recover partially with variable degree of remaining neurological deficits. Eye movement disturbances such as vertical gaze palsy is due to disruption of the cortical input that traverses the thalamus to reach the rostral interstitial medial longitudinal fasciculus.⁶ When also accompanied by rostral midbrain lesions, a mesencephalothalamic or thalamo-peduncular syndrome occurs, which includes oculomotor disturbances, hemiplegia, hemisensory loss, cerebellar ataxia and movement disorders, along with the triad mentioned above. If the nucleus of the third cranial nerve is involved patients present with oculomotor nerve palsy.⁶

Most prevalent long-term consequences are orientation and concentration deficits, behavioral disturbances and memory impairment which is present when lesions affect the mammillothalamic tract, anterior nucleus and dorsomedial nucleus.⁹ The clinical spectrum was studied in detail by Arauz et al.¹⁰ According to him on clinical analysis there were 7 main clinical patterns

- Mental status disturbance (MSD),
- Behavioral amnesic impairment (BAI),
- Aphasia or dysarthria, which includes any alteration of the language components or slurred speech,
- Ocular movement disorders (OMDs),
- Motor deficit,
- Cerebellar signs, and
- Others.

An MSD was considered present if somnolence, stupor, or coma was observed. A BAI included confusion, agitation, apathy, disinhibition, hyperphagia, pseudobulbar affect and any type of amnesia. Any combination of ophthalmoplegia, that is, vertical or horizontal gaze paresis that was with or without pupil involvement, was consistent with the OMDs. A motor

deficit was defined as the presence of any type of paresis or paralysis of the facial, upper or lower limbs. Ataxia or dysmetria were the cerebellar signs. Hypersomnia, tremor, asterixis, seizure, and hyperthermia were considered “others” signs.

The following risk factors were noted: age, sex, hypertension, diabetes mellitus, current smoking, hypercholesterolemia, and heart disease, including atrial fibrillation, patent foramen ovale, interatrial communication, recent myocardial infarction, and previous stroke. The following were considered as the potential causes of Percheron artery infarct

- Vertebrobasilar large-artery diseases were presumed in patients with a stenosis of at least 50% of the lumen diameter
- Small-vessel disease was presumed in patients with longstanding hypertension or diabetes mellitus, in absence of other etiologies;
- Cardioembolic mainly included nonvalvular atrial fibrillation, left ventricular dyskinetic segment, intracardiac thrombus, or other less common sources; and
- Other etiologies and undetermined causes. The main cause of bilateral thalamic infarction was small artery-disease, followed by cardioembolism.¹⁰

The artery of Percheron is rarely visualized with conventional angiography. On imaging, it is classically characterized by bilateral infarcts in areas involving the rostral midbrain and/or ventromedial thalamus. Ordinary brain MRI T2/FLAIR sequences could fail to properly detect brain small lesions determined by the AOP occlusion. Only the application of the DWI/ADC sequences may allow identification of the ischemic lesions.¹¹ Imaging studies performed within the first week after the stroke, showed 4 different clinic-anatomical patterns

- Bilateral paramedian thalamic with rostral midbrain infarction (BPTRMI),
- Bilateral paramedian thalamic without midbrain infarction (BPTWMI),
- Bilateral paramedian and anterior thalamic with midbrain infarction, and
- Bilateral paramedian and anterior thalamic without midbrain infarction.¹⁰

Wernicke's encephalopathy can also be a differential diagnosis of bilateral thalamic lesions, in which T2-weighted MRI findings include symmetric hyperintensity in the medial thalami but also in the tectal plate, periaqueductal gray, mamillary bodies and dorsa medulla. Wernicke's encephalopathy results from a dietary vitamin B1 deficiency in patients with malnutrition or malabsorption and is frequently associated with chronic alcohol abuse. This neurologic disorder is classically characterized by ataxia, altered consciousness, anterograde amnesia and ocular dysfunction. In our

patient the MRI diagnosis of werniche encephalopathy did not correlate clinically. The onset was very sudden. Though the patient was an alcoholic he had been abstaining many days before the attack. The thiamine-deficient Korsakoff amnestic syndrome was not present. Sudden appearance of marked cerebellar signs are unusual in werniche encephalopathy. At a given time usually only one artery gets occluded in a cerebrovascular accident but the lesions in our case were bilateral. Keeping that in mind the case was discussed with the radiologist and the revised diagnosis of artery of percheron infarct was made. It could also be a variant of the paramedian arterial supply as the lesions were asymmetrical and more on the left side. The cerebellar signs were bilateral. Both eyes were involved initially but later the right eye recovered. Incidentally clinical picture of Werniche syndrome has described after AOP infarct.¹²

A thorough review of the patient's history, physical examination and MRI of the brain is crucial in excluding Wernicke's encephalopathy from bilateral thalamic infarctions. In a patient with an acute onset of a neurological deficit and changes in the described locations, a diagnosis of a stroke of an artery of Percheron must be considered.¹³ The diagnosis was based on symptoms of AOP infarction as described in the literature, radiological signs of AOP infarction, as well as exclusion of differential diagnoses. In our case there was no vertical gaze palsy but a 3rd N palsy. A right-sided pyramidal weakness and left oculomotor nerve palsy, has been described in a case report.¹⁴ The variable clinical picture in AOP infarct is due to the variants in the paramedian arteries of thalamus. Cerebellar signs have been less frequently described in case reports. In a case series 33% had cerebellar signs. Our patient had hypertension which was present also in 33% cases in this case series.¹⁰

AOP infarction is not the only condition that can result in bilateral thalamic lesions. Other vascular etiologies of bilateral thalamic lesions include top of the basilar syndrome and deep cerebral venous thrombosis. Top of the basilar syndrome can present with bilateral thalamic infarcts but there are usually also infarcts present within the vascular territories of the superior cerebellar artery and posterior cerebral artery. Deep cerebral venous thrombosis can in rare instances result in bilateral symmetric involvement of the thalamus and basal ganglia. Differential diagnoses also include neoplasms, infections, Wilson's disease, and osmotic myelinolysis.¹⁵

CONCLUSION

Knowledge of this peculiar variant of the posterior circulation and the typical MRI findings of bilateral thalamic and midbrain hyperintensities is important for reaching the correct diagnosis so that appropriate management can be done. A thorough history and examination helps to exclude etiologies like werniche

encephalopathy with similar MRI and clinical features in a drowsy patient with restricted eye movements.

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