

## CASE REPORT

## Artery of Percheron: an unusual stroke presentation

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**SUMMARY**

An 86-year-old woman was admitted with multiple episodes of transient loss of consciousness. She was initially treated for seizures, and stroke was not considered likely. MRI on the same day of admission showed acute bilateral medial thalamic infarcts in keeping with the Artery of Percheron (AOP) territory infarcts. Investigation for polycythaemia and thrombocytosis showed JAK2 positive myeloproliferative neoplasm.

A diagnosis of AOP infarction is often missed or delayed because it is rare and presents with variable neurological symptoms. Initial imaging in the form of CT is often negative, and some report that initial MRI findings may also be normal. An awareness of a wide range of differential diagnoses alongside a multi-modality imaging approach is required to reach a diagnosis. Although there are several other case reports of AOP infarction in the literature, this is the first to present with transient symptoms initially mistaken for seizure activity.

**BACKGROUND**

The Artery of Percheron (AOP), first noted in 1973 by French neurologist Gérard Percheron, is a rare anatomical variant.<sup>1</sup> Characterised by a single trunk arising from a proximal segment of either of the two posterior cerebral arteries, it supplies the paramedian thalami and the rostral midbrain bilaterally. Clinical features of AOP ischaemic stroke vary hugely due to the complex functions of the human thalamus and midbrain structures, but there are three main features: vertical gaze palsy, memory impairment and coma.<sup>2</sup> This case report draws attention to a rare form of stroke whose presentation can mimic many other conditions, including seizures.

**CASE PRESENTATION**

An 86-year-old woman presented to the emergency department (ED) with recurrent episodes of unconsciousness, each reportedly lasting up to 5 min. Apart from hypertension, there was no other significant medical history. She had a further self-terminating episode of loss of consciousness while under observation in ED. During this episode, physical examination showed a Glasgow Coma Score of 7 (E1V1M5). Pupils were equal at 3 mm in diameter but minimally reactive. There was no nystagmus. Tone in all four limbs was initially flaccid but she developed bilateral lower limb hypertonia for a brief period. There were subtle jerking movements of the hands and feet which were more pronounced on the left, and she had bilateral extensor plantar

responses. There was no urinary incontinence or tongue biting.

Her unconscious state was accompanied by stertorous breathing and lasted 4 min. Thereafter she made a swift recovery and denied any pain, aura or presyncopal symptoms. She was alert and well orientated but had no recollection of the episode.

Her pulse during this episode was regular at 72 bpm and blood pressure 132/62 mm Hg. She was afebrile with a respiratory rate of 12 bpm and oxygen saturation of 97%. ECG showed normal sinus rhythm throughout. Serum lactate was normal.

She was treated with 300 mg intravenous sodium valproate, followed by 1 g intravenous phenytoin for presumed seizures. She continued to have brief unresponsive episodes similar to those described above despite this and therefore underwent further imaging by cranial MRI.

**INVESTIGATIONS**

Blood analysis showed a haemoglobin level of 163 g/L, platelet count of  $537 \times 10^9/L$  and haematocrit of 50%. All other blood investigations were normal.

Unenhanced cranial CT on admission showed widespread patchy low density within the white matter consistent with small vessel ischaemic change but no signs of acute infarction or haemorrhage and no other significant findings.

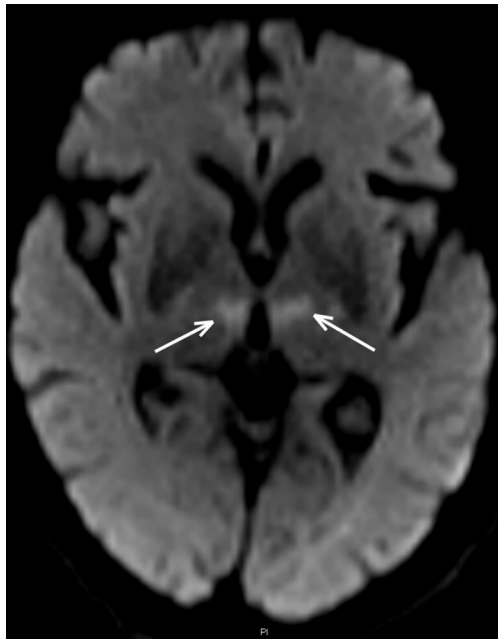
After several further brief self-terminating episodes of loss of consciousness that day, a repeat CT head showed no new changes. A CT cerebral angiogram showed normal equal calibre vertebral and carotid arteries and good calibre basilar and posterior cerebral arteries. The remainder of the study was normal. There were no signs of central venous sinus thrombosis.

Electroencephalogram (EEG) was requested early in the workup to support the initial diagnosis of seizure but was unfortunately not immediately available.

MRI brain performed later on the same day of admission showed increased signal on the B1000 diffusion images within the medial thalami bilaterally (see [figure 1](#)) with mild reduced signal on the apparent diffusion coefficient images (see [figure 2](#)) indicating restricted diffusion in keeping with acute bilateral medial thalamic infarcts. Once this finding had been made, the patient's transient symptoms had ceased, and EEG was no longer considered necessary for diagnostic purposes.



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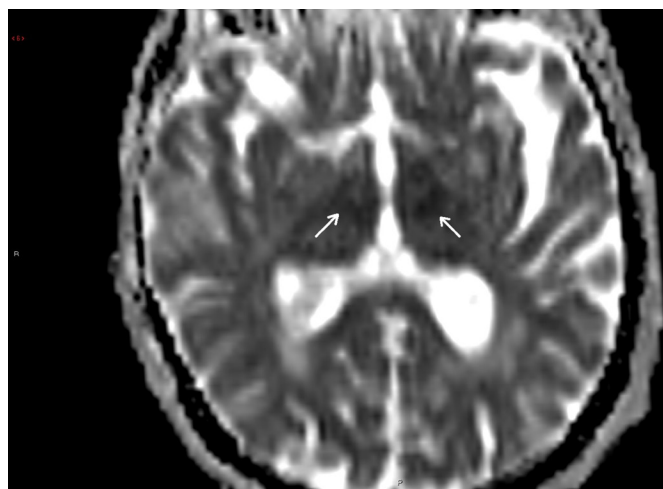


**Figure 1** Axial MRI B1000 diffusion-weighted image with arrows indicating increased signal affecting the medial thalami bilaterally.

### DIFFERENTIAL DIAGNOSIS

This patient's symptoms were initially mistaken for seizure in ED. Provoked seizures in the elderly in developed countries are commonly caused by acute stroke, medications and electrolyte disturbances. However, there was no evidence on her initial CT scan of acute haemorrhage or early signs of ischaemia. No other potential causes of seizure were identified on CT. She took no regular medication besides low-dose amlodipine, and there was no history of alcohol use. Serum glucose, urea, sodium and calcium were also normal, and there was no evidence of infection.

Unprovoked seizure was also considered a possibility in this case. Indeed, these are often underdiagnosed in the elderly. In particular, complex partial seizures may present with atypical features such as episodes of confusion or apparent syncope and are responsible for 70% of epileptic seizures in the elderly.<sup>3</sup> As



**Figure 2** Axial apparent diffusion coefficient image at approximately the same level as figure 1 showing mild reduced signal within the medial thalami indicating restricted diffusion typical of acute infarction.

the episodes of loss of consciousness continued and increased in frequency in the early hours of her admission, she was treated with anticonvulsants for presumed seizures. However, with a poor response to this initial treatment, an absence of positive phenomena such as aura or postictal confusion and a normal serum lactate, seizure did not seem likely. Her ability to localise to painful stimuli during these episodes indicated reduced awareness rather than seizures. Finally, the cessation of her symptoms after the withdrawal of anticonvulsant therapy also suggests that these were not seizures. It was not until her MRI brain that bilateral thalamic abnormalities were identified and AOP infarction diagnosed.

However, bilateral abnormalities of the thalamus and basal ganglia seen on MRI can be due to a variety of systemic and focal aetiologies. These include toxic poisoning by methanol, carbon monoxide and cyanide. In this case, there was no history of attempted suicide or accidental exposure to these toxins. Arterial and venous blood gas measurements were also normal, making toxic poisoning an unlikely cause of the patient's symptoms. Systemic metabolic disorders such as hypoglycaemia and hyperglycaemia, chronic liver disease, Wernicke's encephalopathy and Wilson's disease can also cause these MRI abnormalities.<sup>4</sup> These were not considered likely given the sudden onset of symptoms and normal liver function tests in an otherwise healthy individual. Furthermore, bilateral thalamic abnormalities on MRI with no involvement of the basal ganglia suggested focal rather than systemic aetiology.

Basilar artery occlusion, particularly of the tip, was considered but excluded by the normal CT angiogram. Central cerebral venous thrombosis was also a differential diagnosis, especially in the context of polycythaemia and thrombocytosis but was excluded by the CT angiographic study. A diagnosis of AOP infarction was made based on correlation between MRI findings and the patient's clinical presentation.

### TREATMENT

The patient was treated with 300 mg aspirin once daily and switched to 75 mg clopidogrel once daily after 2 weeks. She was also commenced on 20 mg atorvastatin for secondary prevention.

### OUTCOME AND FOLLOW-UP

The patient made a good recovery. She had a normal transthoracic echocardiogram and was discharged home within 48 hours. She was reviewed in clinic 10 weeks later. Twenty-four-hour ECG showed normal sinus rhythm with no evidence of arrhythmias. She had no focal neurological deficit at follow-up and reported no recurrence of her transient loss of consciousness.

Persistent polycythaemia and thrombocytosis were due to a JAK2 mutation myeloproliferative neoplasm. She was commenced on 500 mg hydroxycarbamide once daily and underwent regular venesection. Six months following her initial presentation, she was symptom free and living independently.

### DISCUSSION

This case of AOP infarction is highly unusual because its clinical presentation mimicked seizure activity but 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, with a frequency ranging from 1.5% to 5.7%.<sup>5</sup> However, these tend to occur in younger patients and those with haemorrhagic rather than ischaemic strokes. Poststroke seizures also tend to respond well to a single antiepileptic drug.<sup>6</sup> A recent case study reported seizure as an

initial presentation of bilateral thalamic infarctions. However, the patient in this study presented with unequivocal generalised seizure activity with convulsive movements in all four limbs, urinary incontinence and a bruised tongue before entering a sustained comatose state.<sup>7</sup>

Once identified by imaging, bilateral medial thalamic infarcts should raise suspicion of AOP involvement. They are usually secondary to small vessel disease or emboli rather than myeloproliferative neoplasm, as in this case.

Investigations to rule out basilar artery occlusion, central cerebral venous thrombosis, toxic poisoning and other metabolic causes are crucial in the workup for AOP infarction. There are several case reports of AOP infarction in the literature, and estimates of the prevalence of the AOP anatomical variant range between 4% and 12% of the population.<sup>8</sup>

Unless there is midbrain involvement, the prognosis is generally good, and if diagnosed within a narrow therapeutic window can be improved by thrombolytic therapy. MRI with diffusion-weighted

imaging usually shows acute infarction in the AOP territory and is the imaging modality of choice. Some case reports point to a normal initial MRI so a repeat study should be considered.<sup>9-11</sup>

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### Learning points

- ▶ Artery of Percheron (AOP) occlusion causing bilateral paramedian thalamic infarction presents with diverse and non-specific clinical symptoms including reduced level of consciousness which may mimic seizures.
- ▶ The diagnosis of acute thalamic infarction is best made by diffusion-weighted MRI imaging. The AOP variant is too small to be seen by CT angiography.
- ▶ A thorough knowledge of cerebral vascular anatomic variation and a high index of suspicion is required to accurately diagnose this life-threatening condition.
- ▶ Other potential causes of bilateral thalamic lesions should not be overlooked.

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