

CASE REPORT

Fluctuating drowsiness following cardiac catheterisation: artery of Percheron ischaemic stroke causing bilateral thalamic infarcts

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SUMMARY

An 81-year-old man underwent cardiac catheterisation to investigate breathlessness and left ventricular impairment of unknown cause. He had unobstructed coronary arteries. Immediately following the procedure, he became suddenly unresponsive with vertical gaze palsy, anisocoria and bilateral upgoing plantar responses. He made a rapid recovery to his premonitory state 25 min later with no residual focal neurological signs. He then had multiple unresponsive episodes, interspaced with complete resolution of symptoms and neurological signs. MRI of the brain identified bilateral medial thalamic infarcts and midbrain infarcts, consistent with an artery of Percheron territory infarction. By the time the diagnosis was reached, the thrombolysis window had elapsed. The unresponsive episodes diminished with time and the patient was discharged to inpatient rehabilitation. At 6-month review after the episode, the patient has a degree of progressive cognitive impairment.

BACKGROUND

Acute stroke is a rare complication of cardiac catheterisation. Artery of Percheron territory infarction is even rarer, with an unknown incidence in the general population. It is dependent on a specific anatomical variant of the posterior cerebral artery (PCA) supply of the thalami. The presentation of this condition is highly atypical for that of a stroke, yet it is important to be aware of to aid prompt diagnosis and facilitate timely treatment.

CASE PRESENTATION

An 81-year-old man was admitted to hospital with breathlessness and a non-productive cough. His medical history included mild left ventricular (LV) systolic dysfunction of unknown aetiology diagnosed on transthoracic echocardiogram 1 year previously, paroxysmal atrial fibrillation for which he was appropriately anticoagulated with rivaroxaban, polymyalgia rheumatica, rheumatoid arthritis, adrenal insufficiency and iron deficiency anaemia.

Clinical examination on admission identified signs consistent with pulmonary oedema. ECG demonstrated atrial fibrillation (AF) with a rapid ventricular response and a pre-existing left bundle branch block. Chest radiograph demonstrated cardiomegaly with some congestive features.

A diagnosis of mild left ventricular failure in the context of uncontrolled AF and known LV systolic impairment was made. The AF rate control was

optimised with bisoprolol. He was offloaded with furosemide. Transthoracic echocardiogram demonstrated a deterioration in LV systolic function (estimated ejection fraction of 20–25%), with a hypokinetic basal-mid interventricular septum and a hypokinetic basal-mid anterior wall.

Following successful diuresis, a coronary angiogram was arranged to assess for underlying obstructive coronary artery disease as a cause for the patient's symptoms and LV systolic impairment with regional wall motion abnormalities. Rivaroxaban was suspended 24 hours prior to the procedure. The coronary angiogram was performed from the right femoral artery, using a 5 French sheath and 10mL of 1% lignocaine into the subcutaneous tissue. The angiogram demonstrated normal unobstructed coronary arteries. No sedating medications or opiates were used during the procedure. No heparin or anticoagulant therapy was administered during, or prior to the procedure. There were no hypotensive episodes during the procedure.

Immediately following the coronary angiogram, the patient became suddenly unresponsive. He had not reported any specific preceding symptoms. There was no witnessed seizure activity. Neurological examination identified bilateral vertical gaze palsy with anisocoria (right pupil smaller than left), both pupils remained reactive to light. Bilateral upgoing plantar responses were identified. The Glasgow Coma Scale (GCS) score was recorded 5/15 (breakdown: eyes 1, verbal 1, motor 3).

Twenty-five minutes after the onset of symptoms, the patient awoke with an apparent complete resolution of the drowsiness and neurological signs, returning to his baseline condition. He was fully conversant and had no recollection of the preceding period of reduced consciousness.

After a further 30 min had elapsed, the patient again became acutely unresponsive, again dropping GCS to 5/15 (eyes 1, verbal 1, motor 3). This was not accompanied by a gaze palsy or upgoing plantars on this occasion. The patient awoke again within 15 min, seemingly making a full recovery once more back to baseline condition and conscious level.

DIFFERENTIAL DIAGNOSIS

1. Atypical presentation of a posterior circulation stroke.
2. Multiple transient ischaemic attacks.
3. Atypical seizure activity.
4. Contrast-induced encephalopathy.



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INVESTIGATIONS

Urgent CT of the brain and CT intracranial angiogram performed immediately following the first episode of reduced consciousness demonstrated no evidence of intracerebral bleed, infarction or any other significant abnormality to account for the clinical picture.

MRI of the brain and MR carotid angiogram performed 3 days later showed features of subacute ischaemic insult confined to the territory of the artery of Percheron; there was T2-fluid-attenuated inversion recovery (FLAIR) hyperintensity and diffusion-weighted imaging (DWI) abnormality involving the bilateral medial thalami as well as the midbrain, especially the right cerebral peduncle (figure 1). MR angiogram demonstrated mild non-occlusive atheromatous disease of the common carotid bulbs but no flow limiting stenosis.

TREATMENT

At the time of the initial presentation, given the fluctuating symptoms and signs and repeated rapid recovery, a decision was taken not to thrombolysise the patient. The consensus opinion among the stroke physicians at this point was that a stroke was unlikely given the fluctuating features of the presentation. The diagnosis was ultimately reached following the MRI brain 3 days later, by which point clearly the thrombolysis window had elapsed.

The patient was subsequently re-established on anticoagulation therapy using apixaban. He was referred to stroke occupational therapy and physiotherapy teams for rehabilitation.

OUTCOME AND FOLLOW-UP

The patient continued to have intermittent drowsy episodes; these became progressively less frequent and shorter in their duration. He continued to slowly improve with ongoing rehabilitation and was discharged home.

Three months on from the admission, he was making slow progress. He has some progressive cognitive impairment since his stroke. He still has episodes of drowsiness and hypersomnolence. He had a period of inpatient rehabilitation and has been referred for neurorehabilitation.

DISCUSSION

Stroke is a known rare complication of cardiac catheterisation. The vast majority of strokes following cardiac catheterisation are ischaemic in their aetiology; haemorrhagic strokes are much less frequent. The incidence of stroke following cardiac catheterisation varies between patients undergoing diagnostic coronary angiograms and those undergoing percutaneous coronary intervention (PCI). The most up to date large-cohort study estimates a stroke incidence of 0.09% following diagnostic coronary angiography and 0.23% following PCI.¹ Specific patient risk factors for stroke following cardiac catheterisation include advanced age, diabetes mellitus, hypertension, previous stroke, heart failure and severe coronary artery disease. Specific procedural risk factors include long procedural times, emergency procedures, high contrast use, retrograde catheterisation of the left ventricle in patients with aortic stenosis, bypass graft procedures, presence of coronary thrombus and use of an intra-aortic balloon pump.¹⁻³ There is no significant difference in the incidence of stroke between transradial or transfemoral cardiac catheterisation.⁴ There have been some reports that there is a disproportionately high incidence of posterior circulation strokes in cardiac catheterisation-related stroke,^{5 6} however the most recent large-scale study does not support this observation indicating an incidence of posterior circulation stroke of 23.8% in cardiac catheterisation-related stroke,⁷ which is comparable with their incidence among the general population.⁸

It is therefore clear that stroke is, in its own right, an infrequent complication of cardiac catheterisation. Bilateral thalamic stroke in this context is exceptionally rare and has only been reported once previously in the medical literature to the best of our knowledge.⁹

To understand the pathophysiology of this condition, we must consider the complex anatomy of the thalamic arterial supply. This is based on a number of perforating branches from both the posterior cerebral artery and the posterior communicating artery. The paramedian thalamic territory is supplied by perforating thalamic arteries called the paramedian arteries, which generally arise from separate origins within the P1 segment of both posterior cerebral arteries. These sometimes share a common origin with superior paramedian mesencephalic arteries, which supply medial areas of the upper brainstem.¹⁰ There are a number of rare anatomical variants of this cerebrovascular arterial blood supply. In one such variant, all the paramedian perforating thalamic arteries arise from a single arterial trunk called the artery of Percheron, arising from the P1 segment of one PCA and supplying both the paramedian thalami and rostral midbrain bilaterally¹⁰ (figure 2). Therefore, when the artery of Percheron anatomical variant is present, occlusion of this artery can result in bilateral thalamic stroke with or without additional midbrain stroke. The distribution of the infarction on the MRI brain in this case indicate the presence of this anatomical variant in this patient and suggests occlusion of this as the cause for the bilateral thalamic strokes with a right midbrain stroke.

Arterial occlusion causing bilateral thalamic strokes can either be a result of small vessel disease or of cardioembolism. Aside from an artery of Percheron occlusion, a ‘top of basilar syndrome’ whereby there is a thromboembolic occlusion of the top

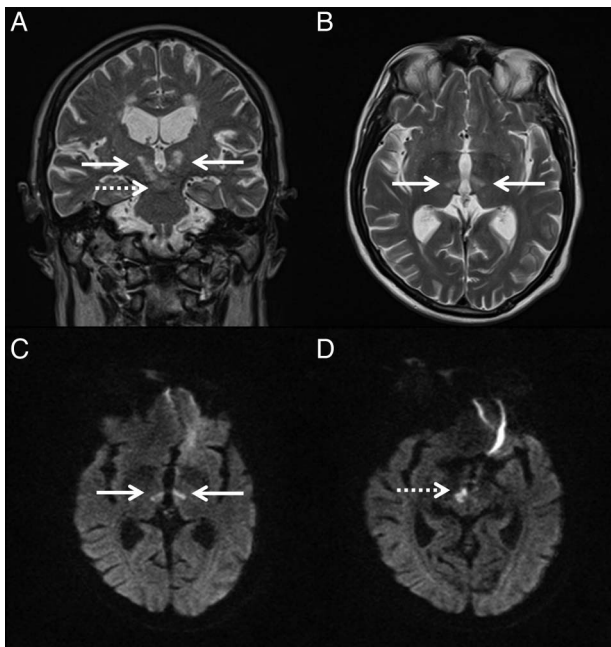


Figure 1 (A and B) Coronal and axial T2-weighted MRI demonstrating ill-defined hyperintensity involving the bilateral thalami (solid arrows) which contiguously involves the brainstem (dotted arrow). (C and D) Axial diffusion-weighted imaging (DWI) showing diffusion restriction in the distribution of artery of Percheron that is, the medial thalami (solid arrows) and the midbrain (dotted arrow).

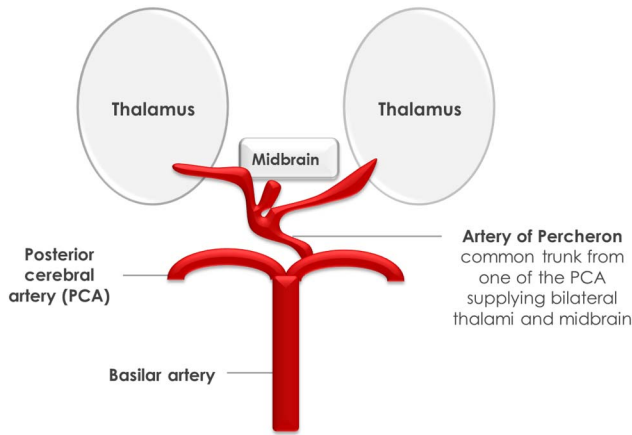


Figure 2 Pictorial illustration of artery of Percheron.

of the basilar artery, can be an alternative cause of bilateral thalamic strokes, however in this case this was excluded by CT intracranial angiogram. Where basilar thrombus is identified on CT angiogram, thrombectomy is a possible treatment strategy. Beyond arterial occlusion, it is possible that bilateral thalamic strokes can be caused by thrombosis of the venous drainage system of the thalamus although this condition is typically associated with a different clinical profile characterised by headache, nausea and vomiting and was not in keeping with the presentation of this patient.^{11 12}

The incidence of artery of Percheron variant anatomy in the general population is not known. The artery itself can rarely be reliably visualised on conventional brain imaging modalities including those that include angiography.¹³ In this case, the artery of Percheron was not identified on the MRI. One retrospective study of 1253 consecutive patients with ischaemic stroke within an 8-year period found that 0.7% of patients had bilateral paramedian thalamic lesions on MRI.¹⁴

The clinical syndrome associated with artery of Percheron infarction is variable and differs significantly from the classical presentation of a stroke. The most commonly reported clinical findings are vertical gaze palsy, memory impairment, confusion and disturbances of consciousness or coma.¹⁵ Long-term neurophysiological sequelae include amnesia, memory impairment and impairment of executive function.¹⁶ Long-term outcomes are generally favourable at follow-up, but are less favourable when there is midbrain involvement.¹⁷

There is a paucity of evidence to guide optimal treatment of artery of Percheron infarction. In general, thrombolysis should be considered if within the appropriate timeframe where there are no contraindications, with the aim of achieving recanalisation of the occluded vessel. Case reports also describe a different approach, which aims to prevent further occlusion by giving anticoagulants to the patient. Both full and partial recovery has been reported with both treatment strategies.¹⁸ The low number of reported cases means there is a limited evidence base to compare outcomes. In the case described in this paper, thrombolysis would have in fact been against current American Heart Association/American Stroke Association international guidance as the patient had taken rivaroxaban 24 hours prior to the stroke; the current guidance advises that thrombolysis may be harmful in patients taking Factor Xa inhibitors unless they have not received a dose for more than 48 hours prior to the stroke.¹⁹

The fluctuating nature of this clinical syndrome is well described^{20 21} and may lead to the diagnosis of stroke being

overlooked in favour of other differential diagnoses.²² This may ultimately delay diagnosis and treatment. CT brain, especially in the acute phase, will often not identify the lesion; therefore MRI should be considered at the earliest opportunity to expedite a diagnosis, as this has a higher diagnostic yield than CT.^{20 23 24}

The exact mechanism of stroke in this case is uncertain. The onset directly after cardiac catheterisation would favour thromboembolic aetiology. The patient did have evidence of atherosclerosis of his carotid arteries and therefore is likely to have atherosclerosis affecting other major blood vessels. It is therefore feasible that during the coronary angiogram there may have been disruption of atherosclerotic plaque in the aorta, resulting in a substrate for embolisation. An alternative mechanism would be a separate source of cardioembolism, either from an undiagnosed LV thrombus or coincidentally from an atrial thrombus given the history of paroxysmal AF. These would seem less likely however given the long-term anticoagulation he had received up until 24 hours prior to the procedure. Indeed a repeat transthoracic echocardiogram subsequently performed prior to his discharge did not identify an LV thrombus. Further imaging to look in more detail for a thrombus with a contrast echo, transoesophageal echocardiogram or cardiac MRI was not pursued in view of the fact the patient required long-term anticoagulation anyway for AF.

Learning points

- ▶ Stroke is a known but rare complication of cardiac catheterisation.
- ▶ A posterior circulation anatomical variant called the artery of Percheron results in a single unilateral arterial trunk supplying both the paramedian thalami and rostral midbrain areas; occlusion of this produces bilateral thalamic strokes with or without additional midbrain stroke.
- ▶ Patients displaying a constellation of signs including vertical gaze palsy, memory impairment, confusion and disturbances of consciousness or coma, should prompt immediate consideration of whether the patient has had a posterior circulation stroke, immediate referral to a hyperacute stroke unit is recommended. These signs may have occurred as a result of an artery of Percheron occlusion.
- ▶ MRI brain has a higher diagnostic yield than CT for detecting this infarction and if should be sought as a diagnostic tool at the earliest opportunity if CT is non-diagnostic. There is a relative paucity of evidence regarding the optimal treatment strategy in this condition and the effect of this on clinical outcomes.

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