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CASE REPORT

Posterior Cerebral Circulation Stroke Secondary to Foetal Origin of Posterior Communicating Artery: An Indication for Carotid Endarterectomy

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Introduction: Posterior cerebral circulation strokes are most commonly caused by posterior vasculature *in situ* thrombosis, cardiac emboli, or arterial dissection. However, the foetal origin of the posterior communicating artery is an anatomical variant of the cerebral circulation that results in communication between the internal carotid and posterior cerebral circulation. Therefore, rarely this can result in posterior cerebral territory infarction from internal carotid artery thrombo-embolism. This is the report of a case in which a patient suffered posterior circulation stroke secondary to this anatomical variation of the circle of Willis.

Report: A 71 year old male patient was admitted to the stroke team with seizures, headache, and confusion. Examination revealed a left sided homonymous hemianopia. Diffusion weighted magnetic resonance imaging (MRI) of the brain 36 hours into his admission revealed an acute right posterior circulation infarct with extensive haemorrhagic transformation. Duplex ultrasound three days later revealed a heavily calcified right internal carotid artery mixed echogenicity plaque with 80%–90% stenosis. Subsequent computed tomography angiography showed a large right foetal variant posterior communicating artery. Following improvement in functional status, the patient underwent uneventful carotid endarterectomy to reduce risk of future stroke.

Discussion: In patients presenting with posterior circulation infarction, clinicians should consider embolism from an atheromatous internal carotid artery via the variant foetal origin of posterior communicating artery. If detected, consideration should be given to undertaking carotid endarterectomy to reduce future stroke risk if no other source is detected.

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INTRODUCTION

Posterior cerebral circulation strokes account for approximately 20% of all ischaemic strokes and are commonly fatal or profoundly disabling.[1] The posterior cerebral circulation comprises the vertebral arteries, basilar artery, posterior cerebral arteries (PCA), and their associated branches.[1] Anastomosis between the posterior and anterior/middle cerebral circulation occurs via the Circle of Willis, but only 20% of patients have a complete Circle of Willis and several anatomical variants exist.[2]

The posterior communicating artery (PComA) is a small vessel connecting the internal carotid artery (ICA) and the ipsilateral distal PCA (Fig. 1A). The foetal origin anatomical variant is defined as the state whereby the PComA provides dominant perfusion to the distal PCA territory from the ICA. The prevalence of this variant is poorly described; however, previous reviews have estimated it to be approximately 4–29% in the general population.[2,3] Two types of this variant are thought to exist; the partial foetal type PComA where the initial proximal (P1) segment of the PCA is hypoplastic (Fig. 1B) and the true complete foetal PComA variant where the P1 segment is totally absent (Fig. 1C).

Typically, an ICA embolus affects the middle or anterior circulation cerebral territory, with sparing of the posterior circulation as a result of the small diameter of the PComA. However, the anatomical variant of PComA can lead to a thrombo-embolic posterior circulation stroke arising from the ICA.

In this study, a case of posterior cerebral circulation infarction secondary to internal carotid atheroma in the context of a partial foetal type origin of the PComA is reported, and the literature on this topic is reviewed.

CASE REPORT

A 71 year old male presented following new onset seizure activity at home, headache, and confusion. Examination revealed a left homonymous hemianopia and pronator drift. Past medical history included hypertension, paroxysmal atrial fibrillation (pAF), and mild chronic obstructive

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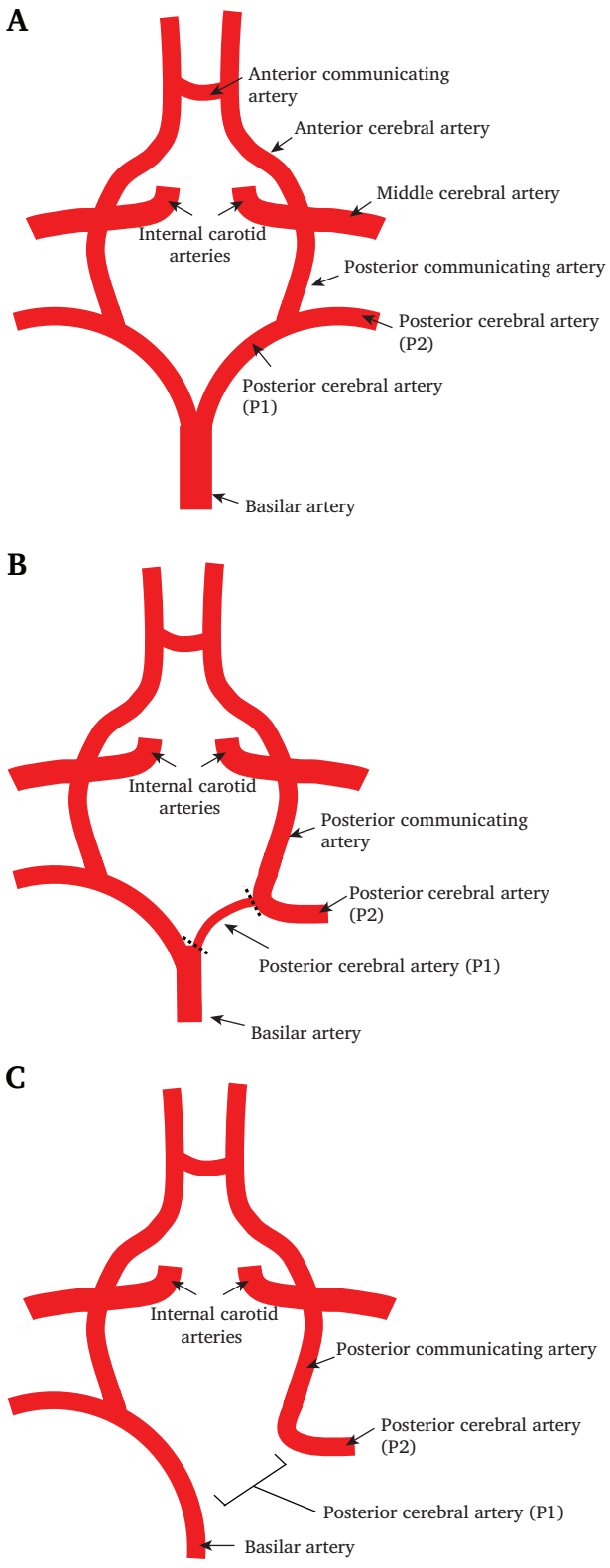

Figure 1. (A) Normal configuration of the complete Circle of Willis. (B) A partial foetal type posterior communicating artery (PComA) with a hypoplastic P1 posterior cerebral artery segment as seen in this patient. (C) The true foetal PComA with an absent P1 segment.

pulmonary disease. Significant medications upon admission included apixaban for pAF. He was an ex-smoker and independent in daily activities.

An initial computed tomography (CT) head scan was normal; however, brain magnetic resonance imaging (MRI) performed 36 hours into admission revealed a large right posterior cerebral circulation infarction affecting the occipital lobe and inferior right temporal lobe with extensive haemorrhagic transformation (Fig. 2A). While this stroke could have been cardio-embolic in aetiology secondary to pAF, the patient was compliant with his apixaban therapy prior to admission and was in rate controlled AF throughout his admission at the time of the stroke. A subsequent transthoracic echocardiogram also showed no mural thrombus. A carotid duplex ultrasound (Samsung RS85 model) was therefore performed three days later, which revealed a heavily calcified, mixed echogenic plaque at the origin of the right ICA, with a peak systolic velocity (PSV) of 404 cm/s. End diastolic velocity (EDV) was 128 cm/s and internal carotid to common carotid PSV ratio was 7.2. This was suggestive of an 80%—89% stenosis of the right ICA (Fig. 3). Minor calcified plaque was noted in the left ICA origin with a PSV of 126 cm/s, EDV of 35 cm/s and therefore a stenosis $<$ 50%. The vertebral arteries were of normal calibre and disease free.

Given this potentially clinically significant ipsilateral carotid stenosis, a CT angiogram of the carotid and cerebral circulation was performed. This confirmed a significant right ICA stenosis. It also demonstrated asymmetry at the Circle of Willis with a larger right PComA (Fig. 2B). Both vertebral arteries were patent and the PCAs were seen bilaterally. This is in keeping with a partial foetal type PComA anomaly with a hypoplastic P1 PCA segment. A transcranial Doppler and/or digital subtraction angiography could have been used to investigate this variant further. However, these tests are not routinely performed in the authors' hospital, and enough information had been gathered from CTA so that further tests were not considered necessary.

After initial recovery, the patient was discharged to stroke rehabilitation services and made a good functional recovery. His apixaban had been switched to clopidogrel during admission given the haemorrhagic transformation of the infarct. Outpatient review one month later revealed some residual homonymous hemianopia but no further seizures, motor deficit, or confusion. The patient had not been commenced on anticonvulsant medication. A repeat outpatient CT head showed maturation of the infarct with no further haemorrhagic change.

His case was discussed extensively at the vascular multidisciplinary team meeting and intervention was approved considering his good recovery and life expectancy of more than five years. Therefore, to reduce future stroke risk, he underwent a standard carotid endarterectomy under general anaesthetic using intra-operative Javid shunt placement and bovine pericardium patch repair. A calcified right ICA plaque with tight stenosis was noted intra-

operatively. The procedure was delayed for five months after initial presentation to reduce the risk of peri-operative haemorrhagic transformation of the large infarcted area. Clopidogrel had been discontinued and apixaban re-started approximately one month prior to carotid endarterectomy. A bridging protocol with low molecular weight heparin was used peri-operatively, with apixaban being re-commenced two days after the operation in combination with aspirin. The procedure was uncomplicated, and the patient remained well at outpatient follow up five weeks later with no evidence of peri- or post-operative stroke.

DISCUSSION

This case highlights the importance of detailed cerebral vascular assessment in individuals presenting with posterior circulation infarcts without an obvious cause (e.g., cardiac embolus) and significant ipsilateral ICA disease.

The foetal origin of the PComA is explained embryologically as a failure of the PComA to regress, which therefore results in PCA perfusion becoming reliant on the ICA. It can exist unilaterally or more rarely, bilaterally.[4] As a result, the severity of strokes in patients with this variant and ipsilateral ICA disease is potentially of greater consequence given the chances of concomitant infarction of both the posterior and anterior/middle cerebral circulations.[5] Furthermore, it is suspected that for patients with this variant, leptomeningeal vessels (which normally provide useful collateral supply between the anterior, middle, and posterior circulations) are poorly developed.[6,7]

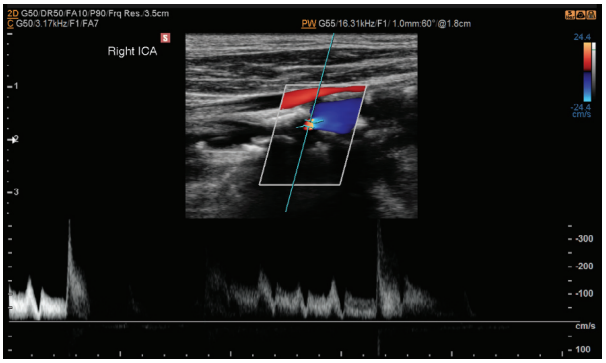

Figure 3. Carotid doppler of right internal carotid artery (ICA).

The patient described in this case report had a prior history of paroxysmal atrial fibrillation without any previous history of embolic events. However, he was anticoagulated with apixaban prior to admission and transthoracic echocardiography revealed no cardiac thrombus. Therefore, given the significant ipsilateral ICA plaque, thromboembolism via the foetal type posterior communicating artery was felt to be the most likely cause of infarction rather than cardiac emboli. Hunter et al. suggested that posterior territory infarcts should be investigated with carotid duplex imaging even when atrial fibrillation co-exists, to rule out a foetal PComA variant.[4]

The foetal origin of PComA as a cause of posterior circulation stroke is a rarely described entity within the

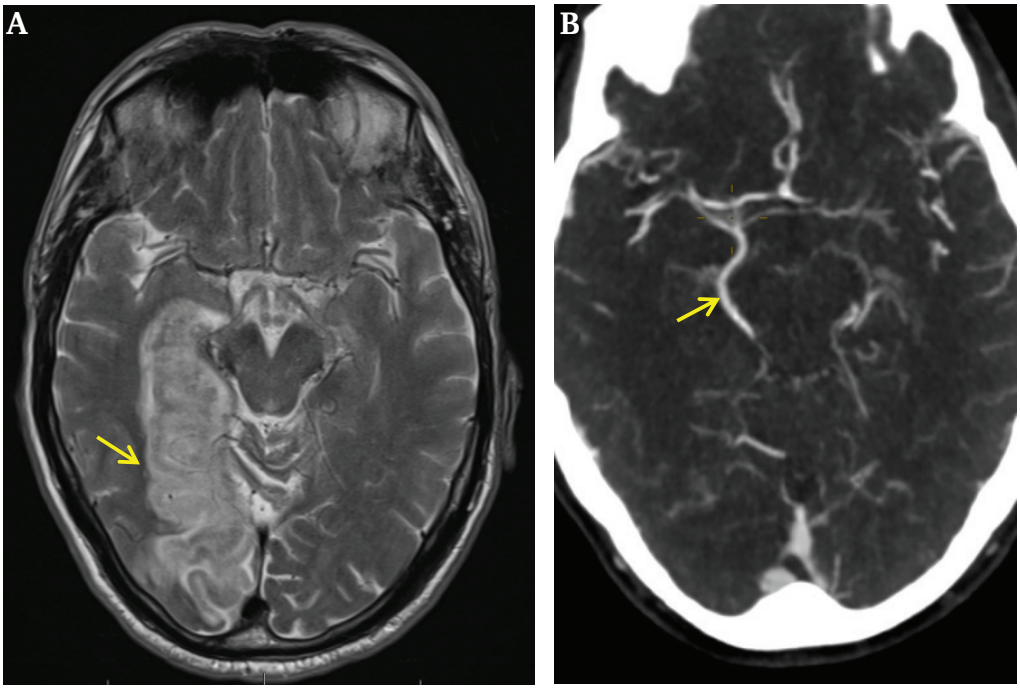

Figure 2. (A) Arrow showing magnetic resonance imaging of a brain with acute right posterior cerebral artery territory infarct. (B) Arrow showing computed tomography angiogram demonstrating the large right PComA.

Table 1. Case details of the reports.

Case reports	Clinical characteristics	Anatomical characteristics	Investigations	Treatment	Outcome
Mann et al. (this case report)	Seizure Headache Confusion Left homonymous hemianopia Pronator drift	Partial right foetal PComA (hypoplastic P1 segment)	MRI: acute right posterior circulation infarction Duplex: 80—89% R ICA stenosis	Right carotid endarterectomy Apixaban, aspirin, felodipine, atorvastatin	Good functional recovery
Hunter et al.[4]	Visual processing disturbance Right homonymous hemianopia	Foetal origins of both PCAs with vertebrobasilar hypoplasia	Left PCA territory infarction 50—75% stenosis of the contralateral ICA	Left carotid endarterectomy with venous patch	Improved with rehabilitation Residual right inferior homonymous quadrantanopia
Eswaradass et al.[7]	Sudden onset left hemiparesis Reduced GCS	True right foetal PCA (absent P1 segment)	Right ACA, MCA, and PCA territory infarction	No definitive treatment given poor outcome	Patient condition rapidly deteriorated, developing brain swelling and dying the same day
Kolukısa et al.[8]	Dysarthria Mild right hemiparesis and hemianopia	Left foetal PCA	Left MCA and PCA territory infarction	Early left carotid endarterectomy Patient on antiplatelet therapy prior to event	Discharged with mild hemiparesis and hemianopia
Ingram et al.[9]	Weakness Right hemianopia Right sided facial droop	Left foetal PComA	Left MCA and PCA territory infarction	Left carotid endarterectomy High dose statin and antiplatelet therapy	Excellent functional recovery

PComA = posterior communicating artery; MRI = magnetic resonance imaging; ICA = internal carotid artery; PCA = posterior cerebral artery; GCS = Glasgow Coma Scale; ACA = anterior cerebral artery; MCA = middle cerebral artery.

medical literature, although the prevalence of this condition may be as high as 29%. Furthermore, its significance and management is not discussed in international vascular surgical guidelines. To date only four case reports have been published.[4,7–9] The clinical features, anatomical considerations, investigations, and management plans for each case are detailed in Table 1. Three of the reports highlight that the potential for concurrent posterior and middle/anterior cerebral circulation infarction is greater in patients with the foetal PComA variant.[7–9] Two of these patients had infarction of both left PCA and middle cerebral artery (MCA) territories, and underwent successful left carotid endarterectomy.[8,9] The other patient, however, suffered infarction in all three cerebral territories and rapidly deteriorated, developing cerebral oedema and brainstem compression.[7] These reports highlight the importance of considering a foetal PComA in patients presenting with multiterritory strokes, with a low threshold for carotid artery imaging as part of initial workup.

In conclusion, consideration should be given to routine carotid duplex scanning in patients with posterior territory strokes given the high prevalence of the variant. Furthermore, alongside good secondary prevention with triple medical therapy (blood pressure control, intensive low density lipoprotein cholesterol lowering, and antiplatelet therapy) and lifestyle adjustments,[1] patients with foetal origin of PComA with significant ipsilateral ICA stenosis can be considered for carotid endarterectomy in the absence of other causes to reduce future stroke risk.

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CONFLICT OF INTEREST

None.

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