CASE REPORT

Internal carotid artery stenosis presenting as ipsilateral posterior cerebral artery ischaemic stroke: a lesson to be learnt

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SUMMARY

A 71-year-old lady presented with a symptomatic left cerebral occipital lobe infarct. With a history of paroxysmal atrial fibrillation a cardioembolic source was initially postulated. Prior significant bleeding while anticoagulated precluded warfarin therapy. Further investigations revealed a critical left internal carotid stenosis with a persistent fetal origin of the left posterior cerebral artery. She was successfully treated surgically and suffered no further ischaemic events. Physicians encountering posterior circulation stroke should be aware of this potentially treatable important diagnosis.

BACKGROUND

This is an important case highlighting the increasing diagnostic issue of identifying the causative mechanism of stroke, from multiple possible sources of pathology. This is especially important given an ageing population with a rising frequency of both atrial fibrillation and atherosclerosis.

A persisting foetal origin of the posterior cerebral circulation and carotid artery stenosis is a recognised cause of stroke; but published reports have previously been limited to neurology/stroke journals.

Given that in many hospitals general rather than stroke/neurology physicians manage stroke patients the authors believe that the lessons from this case should be shared through publication in a journal of wider interest.

CASE PRESENTATION

A 71-year-old woman presented to hospital complaining of bumping into objects on her right side with unsteadiness of a few days duration. She had also recently suffered a low-speed motor vehicle accident while driving.

Her vascular risk factors were dyslipidaemia, type 2 diabetes, obstructive sleep apnoea and a 20-pack-year cigarette smoking history. Eight years prior there were two documented episodes of transient atrial fibrillation related to exercise. She had been anticoagulated with warfarin, which had been discontinued following a gastrointestinal haemorrhage attributed to chronic peptic ulcer disease.

There was a history of depression. She was taking esomeprazole 40 mg twice daily and escitalopram 20 mg daily.

Neurological examination revealed a right homonymous haemianopia and mild right-sided

pyramidal weakness. Higher function testing was unremarkable.

She was clinically in sinus rhythm but hypertensive (160/70 mm Hg) with a loud left carotid bruit.

INVESTIGATIONS

Her full blood count, electrolytes, creatine and liver function tests were normal. An ECG confirmed sinus rhythm.

A CT scan of the brain (figure 1) showed a focal area of low attenuation in the left occipital lobe consistent with acute infarction in the posterior cerebral artery (PCA) territory.

Carotid duplex ultrasonography demonstrated marked stenosis of the proximal left internal carotid artery (ICA), which was difficult to quantify due to severely calcified atheroma. The reporting radiologist documented that this would not explain PCA territory infarction in the absence of a persistent foetal origin of the PCA.

Subsequent CT angiography confirmed critical stenosis involving the proximal left ICA with only pinpoint patency (figure 2). It also showed 50–75% stenosis of proximal right ICA and persistent foetal origins of both PCAs associated with developmental hypoplasia of the vertebrobasilar arterial system.

MRI confirmed an acute left occipital infarct demonstrated by an area of restricted diffusion involving the visual cortex. There were also several small foci of restricted diffusion involving the periventricular and subcortical white matter of the left frontal and parietal lobes consistent with acute infarction of the watershed territory between the anterior and middle cerebral arteries (figure 3). An MR angiogram confirmed persistent foetal origins of both PCAs (figure 4).

Figure 1 CT scan showing left occipital infarction (arrowed).

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Figure 2 CT angiography (left) and three-dimensional reconstruction (right) showing proximal left internal carotid artery stenosis (arrowed).

A transthoracic echocardiogram showed no overt underlying structural heart disease specifically any valvulopathy, intracardiac masses or obvious intracardiac thrombus. Owing to the cerebral MR angiographic findings, likely explaining the underlying source of the cerebral infarct, transoesophageal echocardiography was not undertaken.

DIFFERENTIAL DIAGNOSIS

1. Left internal carotid atherosclerotic stenosis embolising via persistent foetal origin of left cerebral artery.
2. Cardioembolic thrombi from paroxysmal atrial fibrillation.

TREATMENT

Three weeks after presentation a left internal carotid endarterectomy with venous patch was performed. This was complicated by an episode of postoperative atrial fibrillation, which resolved spontaneously.

OUTCOME AND FOLLOW-UP

She improved with rehabilitation with a residual right inferior homonymous quadrantanopia and returned to her usual domicile after 6 weeks hospitalisation.

DISCUSSION

In usual embryonic development, the PCA arises from the ICA and migrates caudally with the posterior communicating artery (PComA) to join the basilar artery via a dominant PCA proximal segment (P1) with subsequent atrophy of the ICA and PComA connections giving dominant supply from the vertebrobasilar (posterior cerebral) circulation. As in this case a foetal circulation may persist with hypoplastic development of the vertebrobasilar and/or P1 tributary circulation with a predominant supply of the PCA from the anterior cerebral circulation via the ICA and a dominant PComA (figure 5). This anomaly may be unilateral or bilateral and occurs with a prevalence of up to 30%.¹

Figure 3 Diffusion weighted MRI showing acute left occipital infarction (left) and patchy anterior circulation watershed infarction (right).

Figure 4 MR angiography confirming bilateral foetal posterior cerebral artery origins (arrowed).

PCA infarction accounts for a minority of ischaemic strokes² and is most commonly associated with a cardioembolic source, atheromatous disease or arterial dissection of the posterior circulation. Less common aetiologies include migrainous cerebral infarction (which preferentially affects the PCA distribution), hypercoagulable disorders, illicit substance use, vasculatures and other rare stroke disorders.

A persistent foetal circulation per se has not been associated with an increased risk of PCA territory stroke,³ but with this anatomical variant significant carotid disease may predispose to infarction.

Jongen *et al.*⁴ documented PCA infarction and persistent foetal circulation. Associated hemianopsia was described by Pessin *et al.*⁵ Both the groups recommended carotid imaging with coexistent carotid bruits or prior anterior circulation ischaemia and carotid endarterectomy if indicated.

Carotid bruits may indicate arterial disease but there is a weak correlation between the presence or absence of bruits and haemodynamically significant stenosis and importantly their absence does not exclude them.^{6,7}

Figure 5 Three-dimensional multidetector CT angiogram showing bilateral foetal posterior cerebral arteries (arrowed). These provide most of the blood supply to the posterior cerebral artery territory. Reproduced with kind permission from Dimmick and Faulder.¹

In this case, given paroxysmal atrial fibrillation, we cannot exclude a cardioembolic source of the cerebral ischaemic events but the medical imaging findings would suggest this as an unlikely aetiology.

Current stroke guidelines do not acknowledge the association of PCA infarction with this common anatomical variation and traditional teaching only recommends carotid imaging for anterior circulation stroke syndromes.

We believe documentation of PCA infarction or symptoms suggesting a PCA transient ischaemic attack should, even in the absence of clinical evidence of carotid disease, be investigated at least by carotid ultrasound. We do acknowledge, the known limitations of ultrasound when large calcified plaques are present,⁸ operator dependence as well as limited utility in diagnosing tandem lesions.⁹ It is also worth noting that MR capable centres have reported a diagnostic accuracy for MRA higher than that of sonography.¹⁰

Early endarterectomy in anterior circulation infarcts, to the corresponding relevant carotid artery, is now well validated.^{11,12} Therefore if a haemodynamically significant stenosis is revealed, further investigation to exclude a persistent foetal PCA origin with a view to carotid surgery should be undertaken. Furthermore, we feel stroke guidelines should highlight this issue.

Learning points

- ▶ A persistent foetal origin of the posterior cerebral artery (PCA) from the internal carotid artery (ICA) is a common anatomical variant.
- ▶ The correlation between the presence or absence of carotid bruits and haemodynamically significant stenosis is poor.
- ▶ A documented PCA ischaemic stroke even with concurrent atrial fibrillation should be further investigated to exclude a persistent foetal circulation and significant ipsilateral carotid stenosis.

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