

Other full case

Bilateral posterior cerebral artery infarction

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We report the case of a 70-year-old man who presented with short-term memory impairment and a homonymous left inferior quadrantanopia secondary to simultaneous bilateral posterior cerebral artery (PCA) territory infarction. As in more than a quarter of cases of PCA infarction, no aetiological cause was identified. Unlike the transient nature of symptoms in some cases following unilateral infarction, his deficits persisted on 2-month follow-up.

BACKGROUND

As shown by the following case, severe and disabling disorders of spatial orientation and memory can be observed in limited bilateral posterior cerebral artery (PCA) occlusion.

CASE PRESENTATION

A 70-year-old Caucasian man presented to the emergency department with acute disturbance of vision and confusion. He had been working up to the time of presentation. He

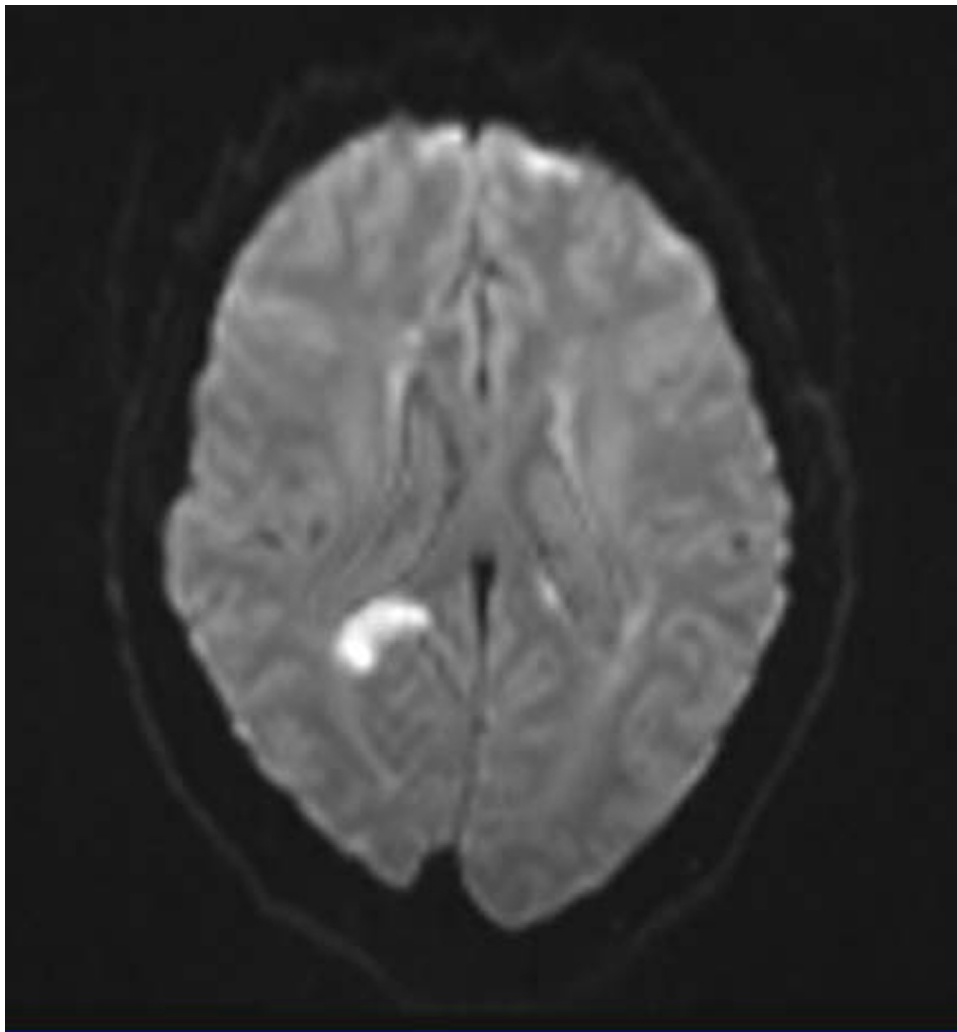


Figure 1 MRI FLAIR (fluid attenuated inversion recovery) showing area of right PCA territory infarction.

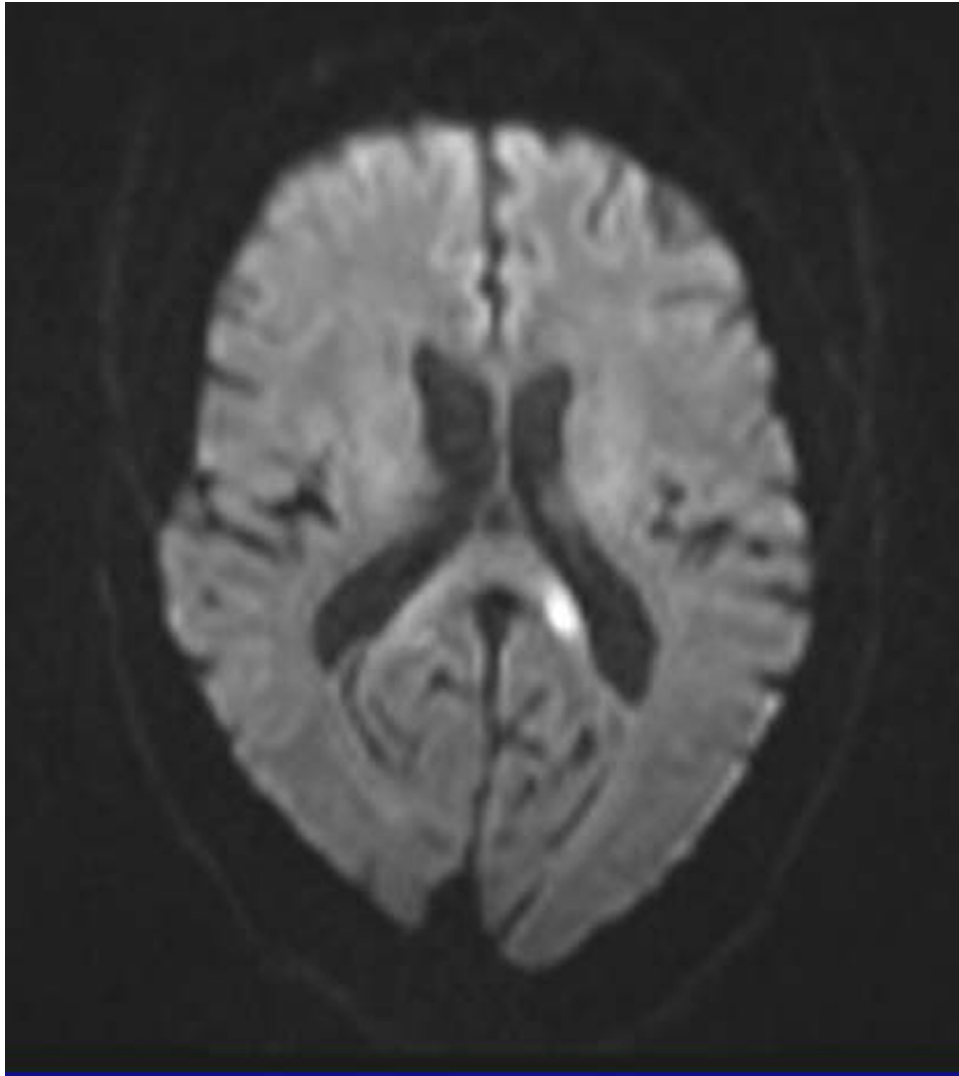


Figure 2 MRI FLAIR showing area of left PCA territory infarction.

was an ex-smoker who had quit 32 years previously and seldom consumed alcohol. He was not on any medications. There was no reported family history of strokes.

A collateral history was provided by his wife as the patient was unable to give a detailed history. The patient had complained of a head cold earlier in the week and his general practitioner had commenced him on co-amoxiclav on the evening prior to presentation. The following morning he complained of being unable to read the newspaper. He could not remember having seen the doctor the previous evening and was repeating questions. The patient himself was not conscious of being confused.

In the emergency department his blood pressure was 140/90 mm Hg. He had a homonymous left inferior quadrantanopia. There were no other focal neurological signs. He was orientated in time and person but not in place. There was evidence of a profound amnesic syndrome with impaired delayed recall (0/3 on Mini-Mental State Examination recall). He could not remember why he had been brought to the hospital.

INVESTIGATIONS

Blood tests including a full blood count, urea and electrolytes, liver function tests, thyroid function tests, C reactive protein, lipids and fasting glucose were all normal. CT of the brain showed an area of low attenuation in the medial aspect of the right occipital lobe. MRI and magnetic resonance angiography of the brain demonstrated acute infarction in the distribution of bilateral PCA territories with no intravascular abnormalities identified (figures 1, 2). Carotid duplex ultrasound demonstrated no significant stenosis. Echocardiogram revealed left ventricular hypertrophy, mild aortic stenosis and mild mitral regurgitation. Transoesophageal echocardiogram did not provide any additional information. 24-h Holter monitoring showed normal sinus rhythm.

TREATMENT

The patient was treated with aspirin, atorvastatin and perindopril.

OUTCOME AND FOLLOW-UP

On review 2 months later he had a persistent short-term memory deficit and left homonymous inferior quadrantanopia and was unable to return to work.

DISCUSSION

Unilateral PCA is common with much known about its associated signs and symptoms.¹ In a study by Yamamoto *et al*, aetiological mechanisms leading to PCA infarction included cardiac embolism, cryptogenic embolism, intrinsic PCA disease, vasoconstriction and coagulopathy.² The aetiology of PCA territory infarction is unknown in more than a quarter of patients³ and no cause was identified in our case. The PCA supplies parts of the midbrain, the hippocampus, the thalamus, the mesial inferior temporal lobe, the occipital and the occipitoparietal cortices. It is also a source of collateral supply for the middle cerebral artery territory. Therefore features of PCA infarction may include visual, memory, sensory, psychological and motor deficits which may be transient or persistent in nature.³ Neau and Bogousslavsky have identified a syndrome of posterior choroidal artery territory infarction (PChA). The PChA originates from the second segment of the PCA.⁴ There are similarities between our case and Neau and Bogousslavsky's study in which one of 10 patients studied experienced short-term memory dysfunction and five of those 10 patients with

PChA infarction had visual field deficits.⁵ Our case shows similarities to this syndrome.

Learning points

- ▶ Posterior cerebral artery infarction may present with memory and visual field disturbances.
- ▶ These disturbances may be persistent in nature.
- ▶ The aetiology remains unknown in more than a quarter of cases.

Competing interests None.

Patient consent Obtained.

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