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Bilateral Posterior Cerebral Artery Infarction

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Abstract **Case Report**

We report the case of 58-year-old women who presented sudden Right-sided weakness ans dysarthria 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. MRI is the main imaging modality for establishing a diagnosis of posterior cerebral artery Stroke.

Keywords: Right-sided weakness, Dysarthria, Stroke, Hypertension.

INTRODUCTION

Stroke is the main cause of long-term disability in adults and the second leading cause of death in the world. Posterior circulation cerebral infarction is a kind of cerebral infarction involving the vertebrobasilar system. About 20–25% of ischemic stroke occurs in the posterior circulation system [1–3]. Prognosis of acute posterior circulation cerebral infarction (PCCI) is poor, and the morbidity and mortality are high [4].

CASE REPORT

A 58-year-old women presented to the emergency department with sudden Right-sided weakness ans dysarthria. Past medical history reported hypertension for 10 years, there was no diabetes. On admission, blood pressure was (TA= 18/12). All blood tests were normal. The patient underwent a brain CT scan and showed a bilateral occipital hypodensity, with hyperdense left PCA sign **(Fig. 1).**

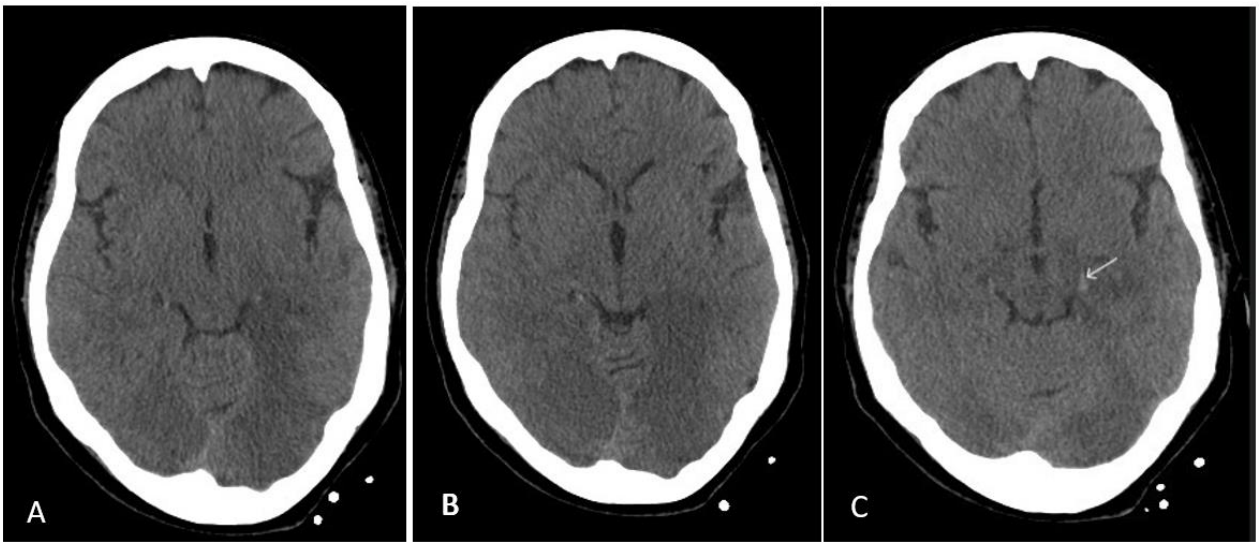

Fig 1: CT scan which objectified the presence of 2 hypodense lesions in spontaneous contrast at the level thalamic bilaterally (A to B), with hyperdense left P2 PCA sign (arrow) (C)

The Magnetic Resonance Imaging (MRI) performed 12 hours after clinical onset showed bilateral and asymmetric acute occipital infarction, extending to both thalami (Figs. 2 and 3).

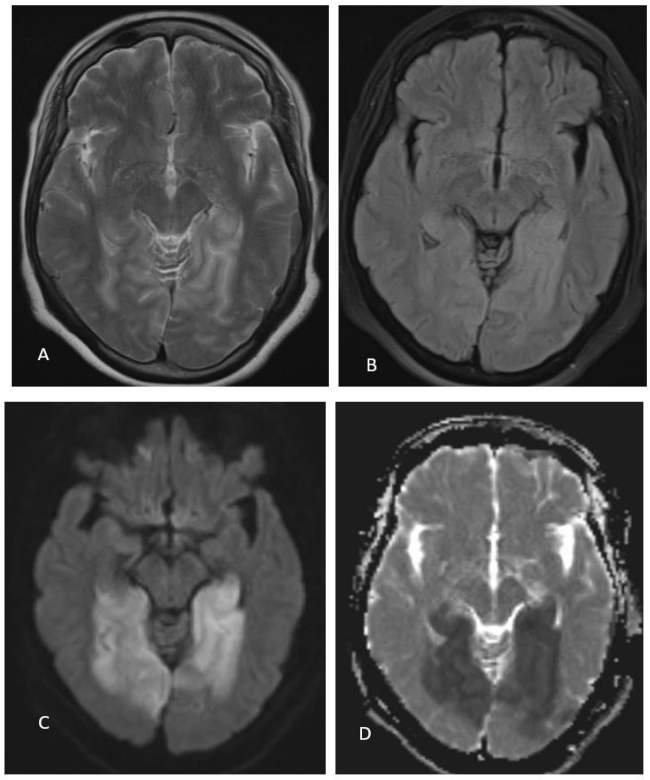

Fig 2: MRI in axial sections, T2 (A), flair (B), and diffusion (C) sequences showing the presence of a high signal with low Apparent Diffusion Coefficient (ADC) (D) in the occipital lobes

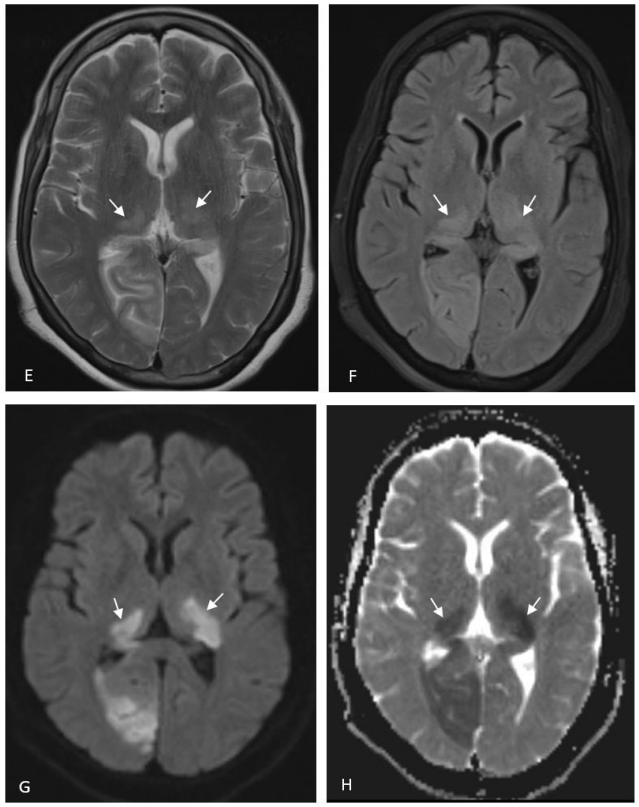

Fig 3: MRI *T2* (E), flair (F), and diffusion-ADC (G-H) showing area of bilateral thalamic territory infarction (white arrows)

Time of Flight (3DTOF) shows P2 segments of posterior cerebral arteries (PCA) bilateral occlusion (Fig 4).

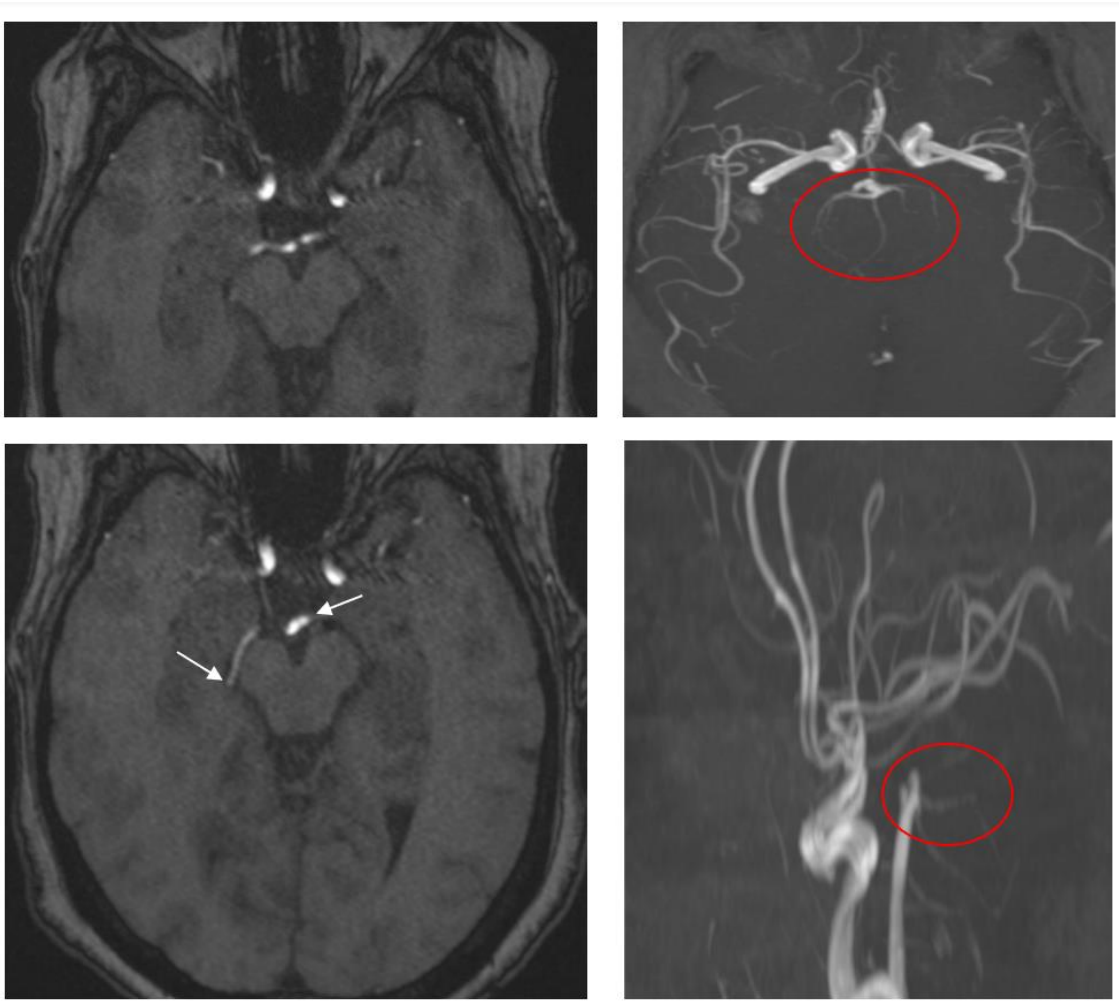

Fig 4: MRI-angiography (A to D) showing occlusion of posterior cerebral arteries (arrows and circle)

DISCUSSION

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 patients. [3] 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. [4]

There are similarities between our case and occurrence of motor deficits with PCA territory ischemia is considered unusual [5]. However, in the series of Brandt *et al*. [6] in 28% of patients a mostly transient and slight hemiparesis was seen similar to the series of Johansson [7], who reported this finding in 17/71 (24%) of patients, with occipital infarcts and no brainstem symptoms.

Milandre *et al*. [8] described a motor deficit in 28/82 (34%) patients with PCA infarcts, involving, however, an exceptionally high proportion of deep PCA territory infarcts.

CONCLUSION

Posterior circulation poses a significant clinical challenge because of its variable and non-specific symptoms and the potentially grave consequences of a delayed diagnosis. There has been significant progress in using advanced imaging for posterior cerebral stroke.

The aetiology remains unknown in more than a quarter of cases.

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