

Clinical Case Report

Peduncular hallucinosis in 'top of the basilar syndrome': An unusual complication following coronary angiography

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ABSTRACT

Cerebral thromboembolism is a potential, although rare, complication of coronary angiography. An elderly woman presented with visual hallucinations, features of bilateral third nerve palsy, impaired vertical and horizontal gaze and mild motor weakness of the left upper limb, following diagnostic coronary catheterization. These findings suggested the anatomical location of the lesion to lie in the caudal midbrain, which was confirmed on computed tomography of the brain. Peduncular hallucinosis following cardiac catheterization, to the best of our knowledge, has only been described once in the literature. Awareness of this entity and its clinical presentation is essential for appropriate investigation and management.

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THE CASE

A 72-year-old woman presented with effort angina and dyspnoea on exertion of 6 years' duration. She was a known hypertensive on regular antihypertensive medication. She had no past history of an acute coronary event or a cerebrovascular event. She underwent a coronary angiogram under fluoroscopic guidance, through the femoral route. In the ward she was started on aspirin (75 mg once daily) and clopidogrel (75 mg once daily) and at the start of the procedure a single dose of heparin 5000 i.u. was administered intravenously. Coronary angiogram revealed single-vessel disease involving the left anterior descending coronary artery (LAD). Abdominal angiogram revealed significant bilateral renal artery stenosis. Following the procedure, the patient was noticed to be drowsy. However, she recovered and was discharged. She was advised a percutaneous transluminal angioplasty (PTA) for the renal artery stenosis with a re-look at the LAD at that time. She was brought to the emergency department within 24 hours of the procedure with complaints of inability to open both her eyes. The patient related that she was able to see, talk to and interact with a relative. She also saw vivid, colourful imagery of her home.

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On examination she was found to have an elevated blood pressure. All peripheral pulses were palpable and there was no carotid bruit. General physical examination was normal. She was oriented to time, place and person. Her speech was normal. Neurological examination revealed bilateral ptosis with a complete third nerve palsy on the right side. She also had impaired vertical and horizontal gaze. Visual acuity was preserved. Examination of the other cranial nerves was normal. She had minimal motor weakness in the left upper limb. Motor and sensory examination was otherwise normal. She had no features to suggest cerebellar involvement.

The features on neurological examination suggested bilateral involvement of the third nerve, the medial longitudinal fasciculus and the horizontal gaze fibres. The well-formed, vivid visual hallucinations were in keeping with a diagnosis of 'peduncular hallucinosis'. These findings imply anatomical involvement of the caudal midbrain with the horizontal extent of the lesion lying in the paramedian and posterior part of the midbrain. Considering the temporal relation of the cerebrovascular event to the preceding angiogram, the likely aetiology was considered to be ischaemia secondary to embolism of an atheromatous plaque. This clinical picture is suggestive of a 'top of the basilar artery syndrome' with occlusion of the penetrating branches of the basilar artery, which may result in infarction of the caudal midbrain, thalamus and portions of the temporal and occipital lobes. A computed tomography scan of the brain revealed a hypodense lesion in the midbrain, in the right paramedian location, involving the tegmentum and part of the tectal plate (Fig. 1). The lesion extended superiorly into the right thalamus along the medial aspect of the right cerebral peduncle. These findings were consistent with acute infarction in the given clinical setting. The patient, however, was discharged at request and was lost to follow up.



FIG 1. Hypodense lesion in the midbrain, in the right paramedian location, involving the tegmentum and part of the tectal plate

DISCUSSION

Cerebral thromboembolism is a rare, but well recognized complication of angiographic procedures (incidence 0.07%–0.38%).^{1,2} However, MRI diffusion weighted studies have shown a much higher incidence of asymptomatic thromboembolic events.³ An increased risk of post-procedural stroke has been found in the elderly; those with diabetes, hypertension; those with past strokes, renal impairment; patients with severe coronary artery disease and lower left ventricular ejection fraction.^{4,5} The length of fluoroscopy time has been associated with an increased risk of post-procedural strokes.³ Stroke after cardiac catheterization usually results from embolization because of the catheter tip dislodging an atheromatous plaque or calcium from aortic cusps or thrombus formation over the catheter or guidewire. Rarer causes include air embolization, aortic dissection during guidewire manipulation, metallic embolus from a fractured guidewire and post-procedural hypotension.⁶ It has been hypothesized that there is a preponderance of left cerebral hemisphere strokes due to manipulation of the catheter tip at the distal bend of the aortic arch, which is closer to the origin of the left common carotid artery. This in turn may cause dislodgement of atheromatous material preferentially into that artery.

Vertebrobasilar strokes are much less common and two studies have shown that there is an increased incidence of these strokes through the antecubital and brachial approach probably due to atheromatous material being dislodged into the subclavian artery and then preferentially into the vertebrobasilar system.² Embolism into the vertebrobasilar territory may result during femoral route angiograms due to inadvertent entry of the guidewire into the subclavian artery during catheter insertion (usually when this part of the procedure is done without fluoroscopic guidance). Early diagnosis and treatment through reperfusion techniques, namely thrombolysis, have been shown to prevent long term neurological morbidity.¹

Jean Lhermitte, a French neurologist, first wrote about a patient with visual hallucinations due to damage of the midbrain and the pons, as early as 1922. Subsequently, this rare and interesting syndrome was described by other authors and the term 'peduncular hallucinosis' (PH) was coined by Van Bogaert. PH consists of vivid, coloured and well formed visual images of animate (including people) and inanimate objects.⁷ The

hallucinations are of brief duration and in some cases the patient may even enjoy the experience! The visual images are not stereotyped and vary from time to time. The visual hallucinations may be of two different varieties. In the first type the hallucinations are not mistaken for reality and are detached from the patient's own thoughts and actions. In the second type the patient considers the hallucinations as part of reality and hence is often agitated and caregivers may consider the patient to be in delirium or even have psychosis. Classically PH is associated with nocturnal insomnia, since the hallucinations are more pronounced at night, and subsequent day-time somnolence.

PH has been described with intrinsic lesions of the midbrain though it can occur with extrinsic lesions. The aetiology includes vascular, infective and compressive lesions involving the midbrain, pons, pars reticularis of the substantia nigra and the diencephalon.^{8,9} The aetiology of hallucinations in PH is not clear; however, it has been suggested that they may be due to damage of the ascending reticular activating system. Hence, PH may be due to the release of dream activity that is normally suppressed during wakefulness.⁹ PH is an entity that must be recognized as a symptom of 'top of the basilar artery syndrome' particularly in patients at high risk for embolic strokes.

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