

CASE REPORT

Posterior Circulation Ischemic Stroke

¹J Vidyashree, ²Mamatha B Patil

ABSTRACT

Posterior circulation stroke syndrome has a lesser incidence compared with anterior circulation stroke with an incidence rate of 10 to 15%. Males are commonly affected than females. Patients most commonly present with symptoms of dizziness, diplopia, dysarthria, dysphagia, and dystaxia. The hallmark of posterior circulation stroke is crossed hemiplegia with cranial nerve involvement on the same side of the lesion and motor or sensory involvement on the opposite side. We report a case of a 60-year-old woman who presented with vertigo, nausea, vomiting, unsteady gait, and tendency to fall on either side.

Keywords: Ischemic stroke, Posterior cerebral circulation, Vertebra-basilar system.

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INTRODUCTION

The posterior cerebral circulation consists of the verte-brobasilar system. Any defect in this system can give rise to stroke. The incidence is 20 to 25% (17–40%) of all the strokes. Leading causes include basilar artery stenosis or occlusion (~40%), small artery diseases (15%), and cardiac embolism (13%). Other causes are arterial stenosis, occlusion, lacunar lesion, or potential cardiogenic source of embolism (13%) and unknown (10%). Early recognition of posterior circulation stroke can prevent morbidity and mortality. It is more difficult to recognize and treat than other types of stroke.

CASE REPORT

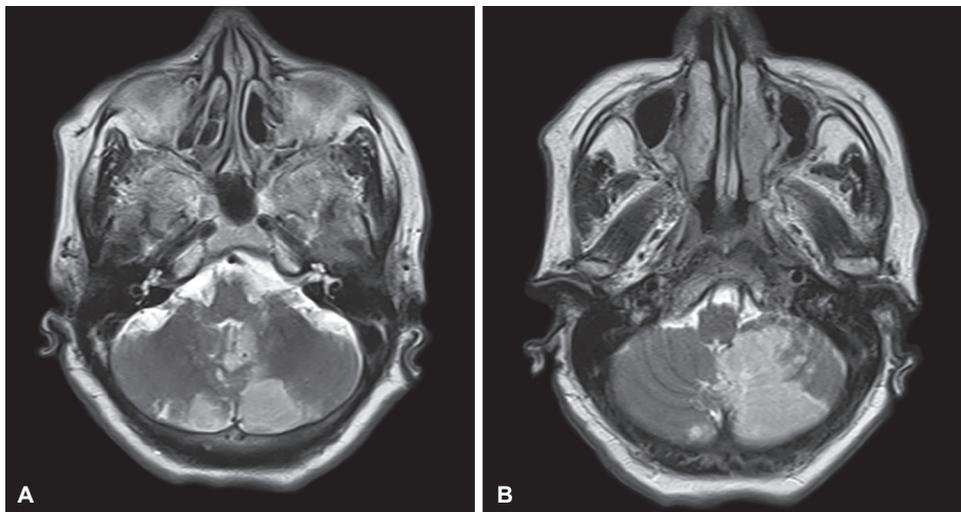
A 60-year-old woman, known diabetic and hypertensive since 5 years, presented with 3 days history of vertigo, nausea, vomiting, imbalance while walking and standing (tendency to fall on either side; left > right). There was no history of speech disturbances, weakness of limbs, no symptoms suggestive of cranial nerve involvement,

raised intracranial tension, ear discharge, hearing impairments, fever, headache, and trauma (head injury). She gave history of pulmonary tuberculosis 4 years back and had received complete course of treatment. There were no similar complaints in the past. On physical examination, patient was obese with body mass index of 40.2 kg/m², alert, and conscious. Her blood pressure, pulse, and respirations and other parameters were within normal limits. Central nervous system examination revealed no cranial nerve palsies. Motor system examination revealed left upper limb and lower limb hypotonia, bilateral pendular knee jerk, flexor plantar response, dysdiadokinesia. On examination, sensory system was normal. Cerebellar signs included positive past pointing, dysmetria, and negative Romberg sign (sways more to the left than right). Gait was wide-based stance with ataxia. There was impaired tandem walking. Fundus examination showed grade II hypertensive retinal changes. All other systems were within normal limits. Clinically, provisional diagnosis of acute bilateral cerebellar lesion of hemorrhagic or ischemic origin was made. Lab reports: Hemoglobin was 13.5 gm%, white blood cell count was 10,920 cells/cumm, platelets were 3 lakh/cumm, fasting blood sugar was 98 mg/dL, postprandial blood sugar level was 297 mg/dL, glycosylated hemoglobin was 8.2%, blood urea, serum creatinine, liver function tests, and serum electrolytes were within normal limits. Electrocardiogram showed left-axis deviation with left ventricular hypertrophy (LVH), QS complexes in leads 2, 3, and unipolar augmented limb lead suggesting old inferior wall myocardial infarction. Two-dimensional echocardiography showed LVH, left ventricular diastolic dysfunction grade I, ischemic heart disease with ejection fraction of 58%. Magnetic resonance imaging (MRI) brain (Fig. 1) showed infarction due to hemorrhage in bilateral cerebellar hemispheres (left > right), microvascular bleeds in basal ganglia and thalami. Small vessel ischemic changes–Fazekas grade II with cerebral atrophy. Magnetic resonance angiogram was normal. Carotid Doppler showed tiny calcific plaque in right carotid bulb and left proximal internal carotid artery with no significant stenosis. Initially, patient was treated with antihypertensives, antidiabetic drugs and symptomatic treatment was also initiated. Patient felt better with reduction of her symptoms. After 2 weeks, patient was advised antiplatelet drugs as a prophylaxis to prevent recurrence of stroke.

¹Postgraduate Student, ²Professor

^{1,2}Department of General Medicine, RajaRajeswari Medical College & Hospital, Bengaluru, Karnataka, India

Corresponding Author: Mamatha Patil, Professor, Department of General Medicine, RajaRajeswari Medical College & Hospital Bengaluru, Karnataka, India, e-mail: dr.mamatharamesh@yahoo.in



Figs 1A and B: Magnetic resonance imaging of the brain

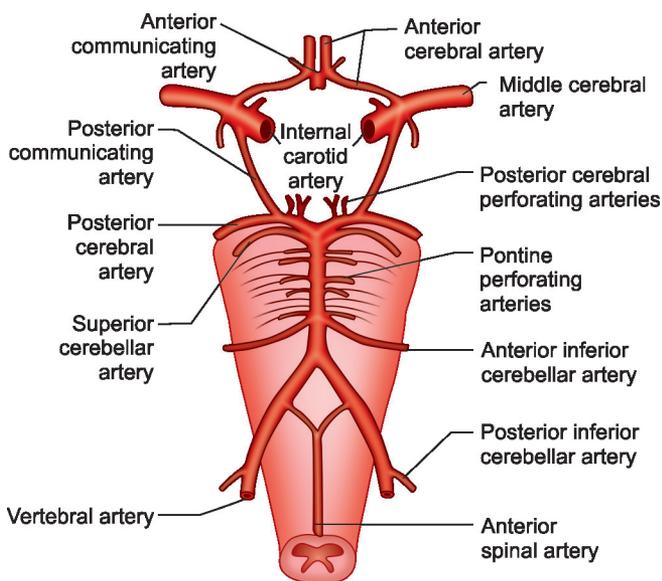


Fig. 2: Arterial blood supply of brain

DISCUSSION

Posterior circulation (Fig. 2) ischemic stroke is a clinical syndrome associated with ischemia related to stenosis, *in situ* thrombosis, or embolic occlusion of the posterior circulation arteries, and their branches. Most common causes of posterior circulation stroke are occlusion or embolism from large artery vertebrobasilar atherosclerosis or dissection, and embolism from the heart.^{1,2} Well-known risk factors include hypertension, smoking, hypercholesterolemia, atrial fibrillation, and coronary artery disease. As the blood supply (Fig. 2) to the brainstem, cerebellum, and occipital cortex decreases, patients complain of dizziness (47%), diplopia, dysarthria (31%), dysphagia, disequilibrium, ataxia (30%), and visual field defects (24%). Acute-onset “crossed” deficit-cranial nerve territory symptoms on one side and sensory or motor deficit of the opposite arm and leg are virtually diagnostic

of posterior circulation ischemia.³ Conditions like acute peripheral vestibular dysfunctions, basilar migraine can mimic posterior circulation stroke.

Early recognition of posterior circulation stroke is difficult compared with other types of strokes. If recognized early, morbidity and mortality can be significantly brought down.

Diagnosis of posterior circulation ischemic stroke is based on history and rapidly developing clinical signs of focal or occasionally general disturbances of cerebral function, with no definitive cause other than that of vascular origin. We should maintain an index of suspicion for posterior circulation stroke in patients presenting with acute neurological symptoms. It is important to establish the onset and tempo of symptoms, such as acute diplopia, visual field disturbance, or swallowing difficulties in the initial assessment phase.⁴ Recognition of stroke in the emergency room scale may be used in diagnosing the stroke in the emergency room. Doctors in the emergency department can rapidly recognize acute stroke using this tool as it includes assessment of visual fields.⁵ All cases of suspected stroke require urgent brain imaging with computed tomography or MRI to exclude hemorrhage. Magnetic resonance imaging has been recommended for assessing transient ischemic attack (TIA) and the posterior circulation defects as per the current international guidelines. Magnetic resonance imaging provides the greatest diagnostic yield when performed as soon as possible or within a few days of onset of symptoms, especially in minor stroke or TIA.⁶ In an emergency situation, airway, breathing, and circulation should be maintained. Intravenous tissue-type plasminogen activator may be used in patients with posterior circulation stroke who meet the eligibility criteria, within 4.5 hours of onset of symptoms.⁷ Acute endovascular therapy like intra-arterial clot removal or lysis has been used in acute

basilar occlusion because of the high likelihood of death or severe disability in the absence of recanalization.⁸⁻¹⁰ Neurosurgical intervention with or without external ventricular drainage or decompression may be lifesaving.¹¹

CONCLUSION

Delayed or incorrect diagnosis may have devastating consequences, including potentially preventable death or severe disability if acute treatment or secondary prevention is delayed. So early recognition and treatment is the crucial step in the case of posterior circulation stroke syndrome. Newer emergency treatment options and stroke prevention policies specific to the posterior circulation are areas of active research.

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