LETTER TO THE EDITOR



A case of thalamo-mesencephalon infarct presenting as transient global amnesia: do we overlook the diagnosis?

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Dear Editor.

Amnesia is the deficit in memory which is caused by brain damage. There are two main types of amnesia such as anterograde and retrograde amnesia. Anterograde amnesia is the inability to remember things for long periods of times due to the inability to transfer new information from the short-term store to the long-term store. Retrograde amnesia is the inability to retrieve information that was acquired before brain damage. Both can occur within a patient simultaneously.

Amnesia can be permanent or transient. Transient global amnesia (TGA) is a common clinical phenomenon in patients applying to emergency departments [1]. Etiology of TGA usually remains unknown and it is thought to be a benign issue; however, it is important to exclude the serious causes for ensuring proper treatment [1].

A 65-year-old female was admitted to neurology outpatient clinic in Bakirkoy Research and Training Hospital with headache, dizziness and amnesia.

Her complaints started 10 days ago with compressive headache, dizziness and mild hypertension which were followed by 5 h prolonged amnestic attack. Her family told that she wandered around in the house aimlessly, spoke and act perseveratively, and kissed people again and again. She went to an emergency clinic in where her symptoms were evaluated as psychiatric as she had antidepressant medicine usage history and there was no focal sign during the amnestic attack

period. Patient herself did not remember anything about the amnestic time period. While she was regaining consciousness, she started to remember the retrograde events firstly and her anterograde amnesia continued for a longer time. She did not remember any hallucinations before or during the amnestic period. She regained consciousness totally in 12 h period but her headache was still continuing during our examination in clinic.

She had fibromyalgia, hypertension, hypothyroidism which were under control with the medicines. There was no characteristic feature in family history.

Neurological examination was normal as she have applied to hospital 10 days after the amnestic attack. But she was still complaining about headache.

Laboratory examination was in normal ratio. Electroencephalography was normal. Normal sinus rhythm was seen in electrocardiography. Ejection fraction was 60%, and left ventricle was hypertrophic but systolic function was normal; grade 1 left ventricle diastolic dysfunction, mild mitral and tricuspid regurgitation were reported in echocardiography.

Ischemic infarcts were detected in left thalamus and left paramedian mesencephalon in magnetic resonance imaging (MRI) (Figs. 1, 2). Cavernous segment of right internal carotid artery (ICA) was thinner than the left one, distal part of the left vertebral artery was thinner in cervical magnetic resonance angiography (MRA) and there were no anterior cerebral artery (ACA) A1 segment, thinner right posterior cerebral artery (PCA) P1 segment, thinner right ICA cavernous segment in cranial MRA (Fig. 3).

She was diagnosed as ischemic left thalamo-mesencephalon ischemic infarct. She was treated by acetylsalicylic acid 300 mg/day.

TGA is a sudden, temporary episode of memory loss that can be attributed to a neurological condition which

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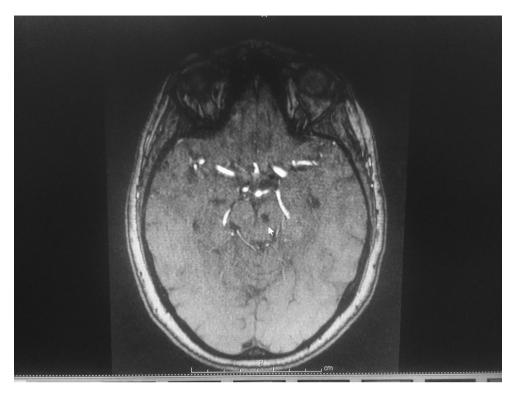


Fig. 1 Ischemic infarct in left paramedian mesencephalon in magnetic resonance imaging

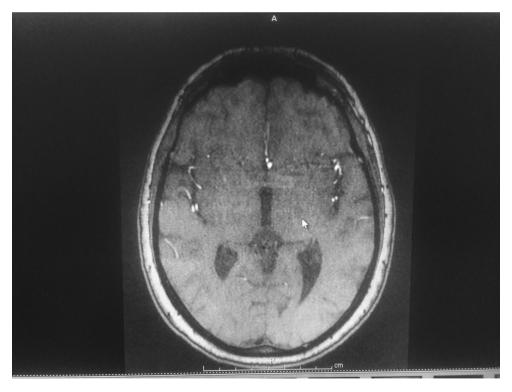
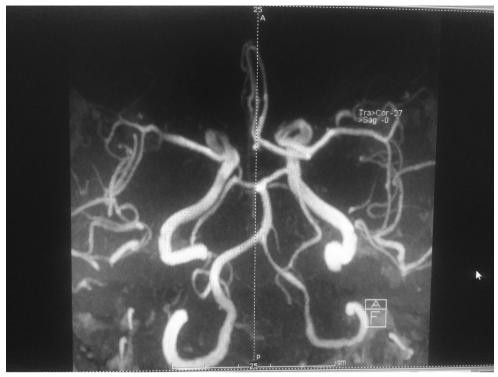


Fig. 2 Ischemic infarct in left thalamus in magnetic resonance imaging

damages cortical parts of the brain. There are several case reports which are about memory dysfunction due to ischemic lesions in brain parts such as diencephalon, mamillary body, and cerebellum [2, 3]. Memory dysfunction is a result of the damage in medial temporal lobe, hippocampus CA1 region, mamillary body and anterior



Fig. 3 Stenosis of left vertebral artery, right PCA-P1, ACA-A1 and ICA in cranial magnetic resonance angiography



thalamic nuclei which are known as the papez circuit [2]. The damage may occur due to cerebrovascular accidents, neurotoxic effects of the angiography agents such as Iomeprol and multiple sclerosis [4].

Especially, infarctions in the territory of PCA circulation can affect papez circuit. The PCA can be divided into 'deep' (P1 and P2 segments) and 'superficial' (P3 and P4) segments. Occlusion of paramedian perforating arteries arising from P1 causes rostral midbrain infarction with or without thalamic lesion [4]. Thalamo-mesencephalic infarcts can cause hyper somnolence, cognitive deficits and visual disturbances [4]. There are few atypical cases which mention pure amnesia without focal neurological signs in literature [5].

Our patient complained of amnesia for 5 h period. Her family told that she had perseverative speech and actions, such as kissing the people around again and again. They did not mention gaze palsy. Left thalamic and left mesencephalon paramedian ischemic infarcts were detected in cranial MRI. P1 segment of right PCA was stenotic in cranial MRA. Left vertebral artery was stenotic in cervical MRA.

Pure amnesia may be seen rarely in thalamo-mesencephalic infarcts; it occurs especially if the infarct is due to bilateral stenosis of posterior circulation. Our patient had left thalamo-mesencephalon infarct due to left vertebral artery stenosis but she had also severe stenosis in right PCA P1 segment. We thought that she had amnesia due to bilateral stenosis in posterior circulation but right-sided stenosis did not prolong for too long to develop an infarction.

TGA is an often seen syndrome applied in emergency clinics; it is usually underestimated and clinicians tend to diagnose it as psychological. It can occur due to posterior circulation infarcts, although it is pure and seen without visual disturbances or other focal neurological signs. We report this case to underline the importance of organicity in TGA because it may be overlooked easily and misdiagnosed as psychiatric disorder.

Compliance with ethical standards

Conflict of interest The authors state that they have no conflict of interest.

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