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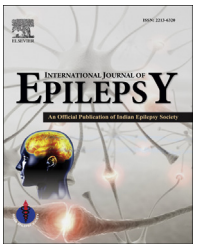

Case Report

Recurrent transient focal neurological deficits in convexity meningioma: TIA or negative motor seizures?

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ABSTRACT

Transient ischemic attack (TIA) is defined as a temporary focal neurological deficit of presumed vascular origin, lasting for less than 1 h, with no evidence of infarct on neuroimaging. If a patient is suffering from multiple cardiovascular risk factors and presents with transient and recurrent focal neurological deficits, the most likely diagnosis considered is TIA. As the possibility of stroke within first 24 h is high, such patients need aggressive investigation and management, and ideally should be hospitalized. TIA usually occurs due to major intracranial or extra cranial artery stenosis. However, if vascular imaging and cardiac workup is normal, possibility of small vessel disease is considered. Space occupying lesions usually presents with seizure, symptoms of raised intracranial pressure or progressive neurological deficits rather than TIA. There are a few case reports where meningioma presented as TIA, due to vascular compromise by encasing internal carotid artery. 1,2 transient focal neurological deficits can have other differentials like post ictal palsy and negative motor seizures. In a given patient with space occupying lesion like meningioma presenting with recurrent transient focal neurological deficits we need to consider all these differentials. We are hereby reporting a case of convexity meningioma, which presented as recurrent focal neurological deficit due to negative motor seizures, mimicking transient ischemic attack.

1. Introduction

Transient ischemic attack (TIA) is defined as a temporary focal neurological deficit which is of vascular origin, lasting for less than 1 h, with no evidence of infarction on neuroimaging. If a patient has multiple cardiovascular risk factors and presents with transient and recurrent focal neurological deficits which resolve spontaneously, the most likely diagnosis is TIA. In such a scenario the possibility of another stroke remains high, particularly within the first 24 h. Therefore, it becomes imperative to hospitalize such patients for urgent investigations and aggressive management. Generally TIAs occur due to major intra or extra cranial artery stenosis.

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However, if vascular imaging and cardiac workup is normal, the possibility of small vessel disease needs to be considered.

Space occupying lesions usually present with seizures, symptoms of raised intracranial pressure like headache or progressive neurological deficits rather than TIA. There are a few case reports where meningiomas have presented as TIAs. The most likely pathophysiological reason for such presentation is vascular compromise by encasing internal carotid artery.[1,2] Transient focal neurological deficits can have other differentials like post ictal palsy and negative motor seizures. In a particular patient with space occupying lesion like meningioma who presents with recurrent transient focal neurological deficits all these differential diagnosis need to be considered. We are, hereby, reporting a case of convexity meningioma, which presented as recurrent focal neurological deficit due to negative motor seizures, mimicking transient ischemic attack.

2. Case report

A middle aged person experienced sudden onset weakness and numbness of left half of body lasting for about 10 min. By the time the patient reached the hospital he was asymptomatic. On further questioning the patient gave history of 3–4 such similar episodes in past two weeks. Each episode lasted for 10–20 min. These episodes of motor deficits were preceded by a brief "funny" feeling in his left hand and arm before the onset of the motor deficit, although, there was no marching phenomenon.

His vital parameters were normal and random blood sugar was 105 mg%. He was morbidly obese and had diabetes mellitus, hypertension and obstructive sleep apnea for which he was on regular CPAP therapy. Keeping in view the clinical history and underlying risk factors, a provisional diagnosis of TIA was made. His complete blood count, liver and kidney tests, lipid profile and coagulation parameters were normal. Magnetic resonance imaging (MRI) of brain revealed a right-sided broad base mass involving fronto-parietal region suggestive of meningioma (Fig. 1). MR angiography (MRA) of brain and neck vessels was found to be normal. Detailed cardiac workup including a trans-thoracic echocardiogram and a 24 h Holter monitoring were also normal.

After reviewing these investigations, the possibilities considered were TIA and negative motor seizures. In view of significant risk factors and transient motor weakness, a diagnosis of transient ischemic attack, possibly due to small vessel disease, was made as major intra and extra cranial arteries were normal. Next day the patient was discharged on antiplatelets. He, however, was readmitted, two days later, with recurrence of symptoms. He had developed sudden onset left hemiparesis which lasted for 5 min only. By the time he reached the hospital, complete recovery had already occurred and examination was unremarkable.

This time the patient was re-evaluated and EEG was advised, keeping in view, the possibility of negative motor seizures. EEG revealed focal slowing over the right frontal region (Fig. 2). As the symptoms were corresponding to convexity meningioma, the possibility of seizure, presenting as negative phenomena, was strongly considered and patient was put on antiepileptic treatment. He responded very well with the optimal doses of antiepileptics and had no further events. Near total surgical excision of meningioma was done after one month. There was no encasement of any major arteries by the meningioma. A follow up MRI (Fig. 3) showed signal changes over the right frontal region mainly involving motor and premotor cortex which might be the region responsible for his negative motor seizures.

3. Discussion

Transient focal neurological dysfunction has been reported with extracerebral masses with possible mechanisms including compression of vessels subjacent to the mass, post-ictal suppression, and spreading cortical depression after mechanical stimulation of cortex.[3] A transient increase in regional swelling might also cause a critical degree of vascular

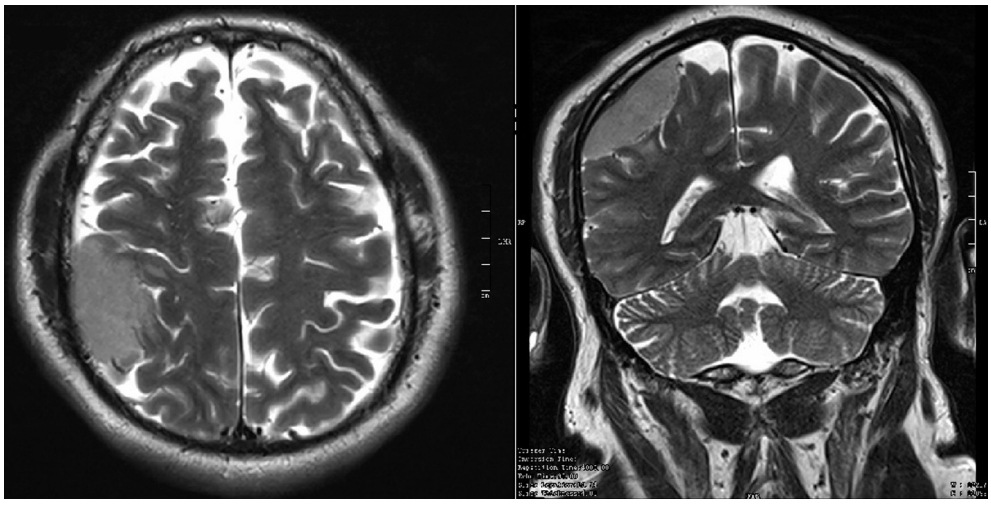

Fig. 1 – Right frontoparietal convexity meningioma lying over the parietal area and producing mass effect on underlying parietal cortex.

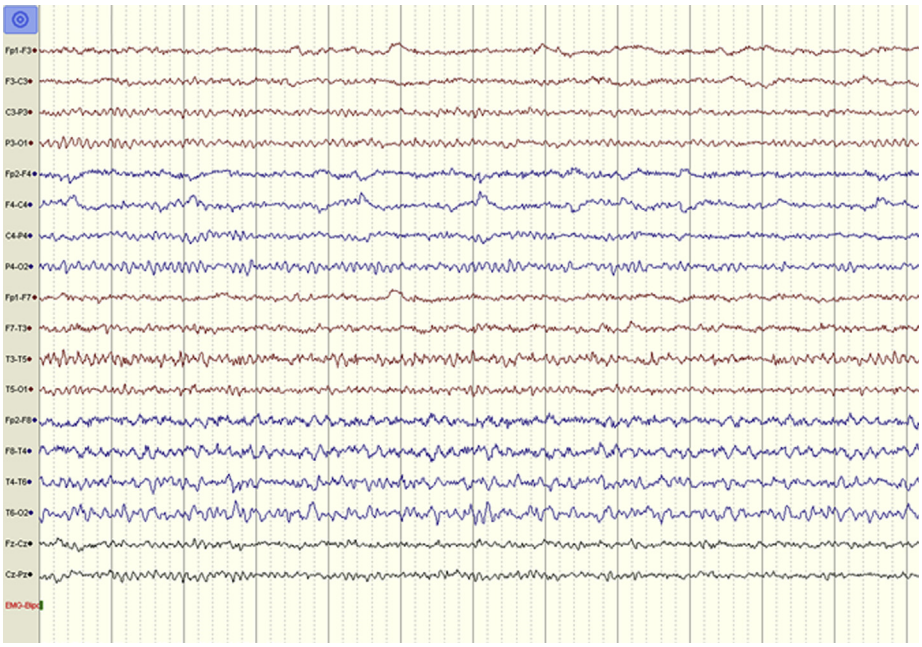

Fig. 2 – EEG showing slowing over the right hemisphere.

displacement and consequent ischemia.[4] However it has never been proven, that such a transient, but negative phenomenon is the result of vascular compromise and thus cannot be labeled as TIA.

Seizures typically consist of "positive" phenomena rather than the loss of neurologic function. A seizure manifesting solely as loss of motor function without loss of awareness is a rare epileptic condition and can be called as negative motor seizure (NMS).[5] Differential diagnosis in such cases are postictal paresis, transient ischemic attacks, migraine events, and psychogenic paralysis.[6] Postulated anatomical areas responsible for NMS includes primary sensori-motor area, the so-called primary negative motor area and the supplementary negative motor area.[7] Earlier Lüders also reported that a certain cortical area in the frontal cortices as negative motor area (NMA).[8]

Focal akinetic seizures belong to ''inhibitory motor seizures'' among focal motor seizures in the international classification of epilepsy and seizures in the report of the International League Against Epilepsy (ILAE) Classification Core Group.[9] Previously, three patients with focal ''ictal paresis'' were reported as having ''focal akinetic seizures''.[6]

Clinically there are few points which can help in differentiating TIA and NMS.[10] The usual age of onset in NMS is first or second decade while TIA starts in later age (4th or 5th decade onwards). In NMS there is spread of symptoms i.e. the symptoms will rapidly spread to involve different parts of the body while in TIA the symptoms are maximum at onset. The

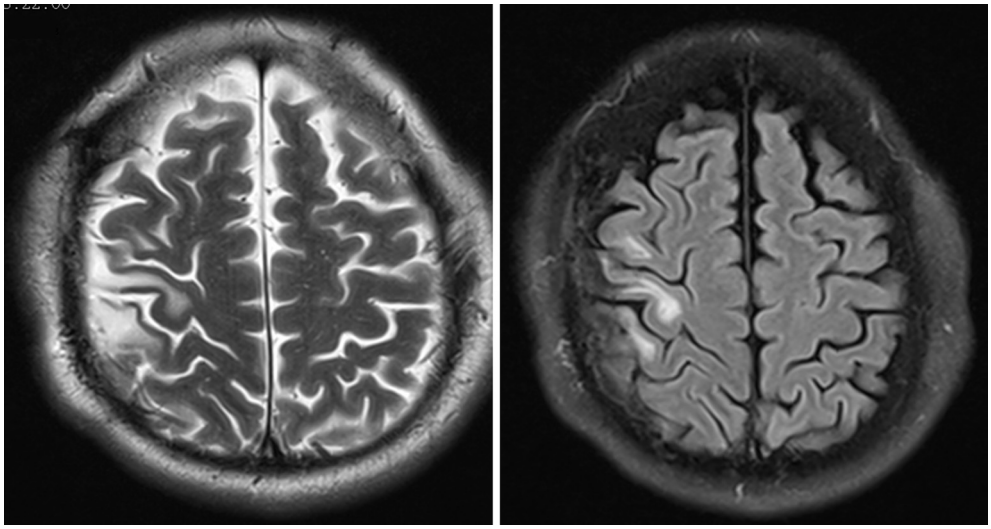

Fig. 3 – Post operative MRI showing T2 and FLAIR hyperintensity over the premotor and motor cortex.

duration of TIA is variable may last for 1 min to 1 h or some time many hours while NMS last only for 1–3 min.

Video-EEG is a very useful investigation in differentiating this rare epileptic phenomenon.[11] EEG during the event can show ictal changes over the frontal cortex confirming the diagnosis of NMS. However no episode could be recorded in our patient and interictal EEG showed mild slowing over the right frontal region.

In our case, an epileptic origin of the events was suspected clinically: a somato-sensory aura in the form of a "funny feeling" occasionally preceded the onset of the motor negative phenomena and all the events involved the side opposite to the convexity meningioma. Conversely, although, this is one of the clinically relevant criteria distinguishing epileptic and non-epileptic paresis, the atypical duration of the individual episodes was more similar to postictal paresis, a frequent and well-known phenomenon that lasts between a few minutes and 36 h and is most severe immediately after the seizure.[12]

To the best of our knowledge, convexity meningioma causing recurrent focal neurological deficits has never been reported. We cannot exclude the possibility that our patient had coincidental TIAs. However, the fact that initially the symptoms did not respond to antiplatelets and, later, symptoms did not recur after antiepileptic treatment and surgical removal of the tumor suggests a causal relationship. In addition there was an evidence of structural damage to the motor and premotor cortex in the post operative MRI which might have been responsible for these negative motor seizures.

Conflicts of interest

All authors have none to declare.

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