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Case Report

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A Rare Presentation of a Patient with Limb-Shaking TIA due to Severe Carotid Artery Stenosis

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ABSTRACT

Background: A limb-shaking Transient Ischemic Attack (TIA) is a rare neurological symptom of hemodynamic origin caused by severe carotid artery disease. Physicians should be aware of its presence because it has a non-typical presentation and is cured by endarterectomy or stenting of the stenosed carotid artery. The aim of the present study was to describe a rare case of limb-shaking TIA.
Description of case: A 76 year-old man with a 80% right internal carotid artery stenosis presented with numerous episodes of involuntary jerky movements of his left arm and leg, associated with episodes of brief weakness of the left leg, caused by preoperative optimization of his hypertension. No stroke ensued despite the daily appearance of symptoms for several months. Limb-shaking TIA was diagnosed and all symptoms disappeared immediately after an uncomplicated carotid endarterectomy was performed.
Conclusion: The clinical presentation of this limb-shaking TIA case, the first to our knowledge to appear during treatment of hypertension, supports further the hemodynamic theory of limb-shaking TIAs.

KEYWORDS: Brain TIA; Arterial disease, carotid; Carotid endarterectomy.

INTRODUCTION

Limb-Shaking Transient Ischemic Attack (LS-TIA) is a rare form of a TIA characterized by involuntary limb movements or "shaking", caused by severe stenosis or occlusion of the carotid artery ipsilateral to the appropriate brain hemisphere. LS-TIAs were first described by C. Miller Fisher of Massachusetts General Hospital some 50 years ago.[1] Contrary to the vast majority of TIAs, hemodynamic mechanisms are thought to be involved in the pathogenesis of LS-TIAs.[2-4]

Because LS-TIA may mimic focal motor seizures, clinicians should be aware of its existence and make the correct diagnosis.[2] Timely carotid endarterectomy can not only cure LS-TIA, but also prevent carotid occlusion and/or stroke. Herein we present a rare case of LS-TIA caused by preoperative management of hypertension leading to deterioration of brain hypoperfusion.

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DESCRIPTION OF CASE

A 76 year-old male patient with a 80% asymptomatic right carotid stenosis and a negative stress echo was on the waiting list for endarterectomy pending control of his otherwise poorly controlled long-standing hypertension (systolic blood pressure values around 170 mmHg). The initial diagnosis was made by Duplex ultrasound and confirmed by Computed Tomography (CT) angiography. The latter showed a mixed plaque (type II) at the right carotid bulb obstructing the lumen by 80%, according to the North American Symptomatic Carotid Endarterectomy Trial (NASCET) methodology (Figure 1a). The contralateral carotid had a 30% stenosis, while the aortic arch and supra-aortic arteries were all patent. There was no evidence of anatomic variations of the circle of Willis (Figure 1b). Both vertebral arteries were patent with antegrade flow on Duplex. The patient was referred back to his primary care physician for optimization of his blood pressure before carotic endarterectomy was performed, while he was instructed to immediately report symptoms of amaurosis fugax, TIA or stroke. Although such symptoms did not occur, he started getting involuntary jerky movements five months after he was originally seen. He failed to report these symptoms for five months up to the point he was called in order to re-examine his situation and schedule an endarterectomy. These movements affected his left arm and leg, occurred several times every day each one lasting for a few seconds, and were associated with episodes of weakness of his left leg, short in duration; a positioning effect was not reported. Based on the clinical characteristics of his symptoms and the history of severe carotid stenosis, the diagnosis of LS-TIA was made.

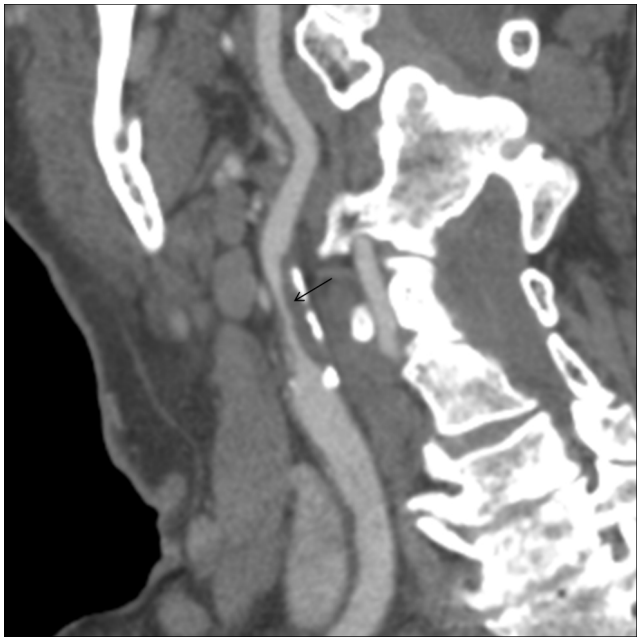

Figure 1a: Sagittal MPR image of the right carotid, shows mixed atherosclerotic plaque (type II) at the level of carotid bulb (black arrow).

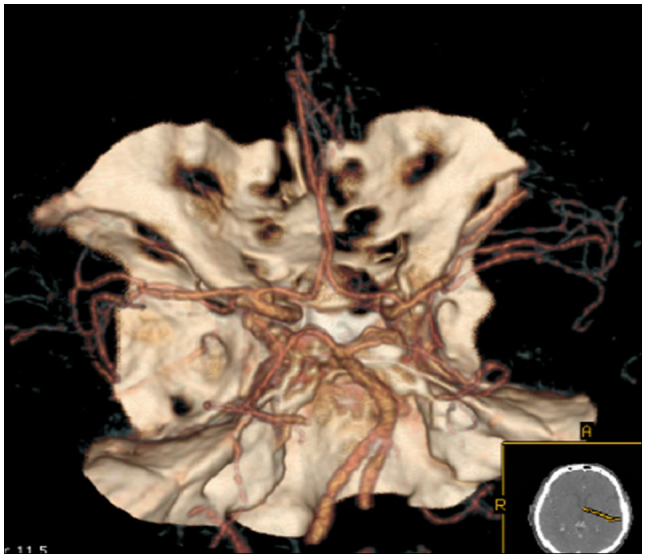

Figure 1b: VRT (Volume Rendering Technique) reconstruction of the Circle of Willis (CoW), shows no anatomic variations of CoW.

Past medical history included hypertension, diabetes mellitus, hyperlipidemia and excision of bladder papillomas. Medications taken were clopidogrel 75 mg OD, losartan/hydrochlorothiazide 50 mg/12.5 mg BID, lasipidin 4 mg BID, atenolol/chlorothalidone 50 mg/12.5 mg OD, glimepiride 4 mg OD, metformin 850 mg BID, simvastatin 40 mg OD and alfuzosin 10 mg OD.

Upon admission to the hospital he was normotensive (with the exception of one abnormal blood pressure reading), a right carotid bruit was noted and he had a normal neurological examination. CT scanning excluded brain infarction or a tumor and a repeat duplex scanning excluded interval carotid occlusion not amenable to endarterectomy. He underwent carotid endarterectomy under general anesthesia with cerebral oximetry monitoring; hypoperfusion during carotid clamping was not detected. Immediately after the operation all neurological symptoms resolved completely. Postoperative course was uneventful and he was discharged home on the second postoperative day on an enhanced antihypertensive regimen (losartan tb 50 mg OD in the afternoon); three months later he had a twenty minute Electroencephalogram (EEG) that was normal with no signs of any epileptiform activity. Our patient remains asymptomatic 16 months postoperatively, with no evidence of restenosis on carotid Duplex and his hypertension being fully controlled.

DISCUSSION

This is the first case of a LS-TIA caused by management of hypertension in a patient with a tight carotid stenosis, leading to hypoperfusion of a critical watershed brain territory, and to the best of our knowledge the first LS-TIA to be managed with endarterectomy in Greece.

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Our patient reported short in duration episodes of left leg weakness, in addition to the involuntary limb movements. Frequently other typical TIA manifestations co-exist in patients with LS-TIA,[5] these can be missed if careful history is not taken. LS-TIA may mimic a focal motor seizure, and thus should be meticulously differentiated. Neurological deficits in TIAs are maximal at onset whereas symptoms in a focal seizure tend to evolve over seconds. The lack of a Jacksonian march,[2] together with the sparing of the facial muscles characterizes the LS-TIA. The presence of other vascular paroxysmal dyskinesias, such as ataxia, myoclonic jerks, dystonic limb posturing and parkinsonism, may aid the clinical diagnosis of LS-TIA, which is further supported by a normal EEG, which rules out epileptic seizures.

It is interesting that despite the repetitive long-standing TIAs, our patient did not develop stroke, perhaps because of the transient hemodynamic nature of LS-TIA,[2] whereas the classical TIAs are caused by embolism from an unstable carotid lesion, not present in this patient. The hemodynamic mechanism of LS-TIAs is further supported not only by the observation that in our patient all symptoms started after the attempts to manage his poorly-controlled hypertension, but also from other reports where symptoms were elicited by orthostatic hypotension,[6,7] postprandial hypotension,[8] hypertension control in association with complete carotid occlusion successfully managed with antihypertensive dose reduction,[9] external compression,[10] or balloon occlusion,[11] of the carotid artery. During evolvement of symptoms in a patient in one study, a dramatic decrease of flow velocities in the left middle cerebral artery was observed on transcranial Doppler, a finding that further support the hemodynamic theory.[12]

Carotid endarterectomy led to immediate disappearance of limb shaking of our patient, similarly to a previous report on six patients.[5] No complications occurred, including the hyperperfusion syndrome and hemorrhage into the revascularized brain territory, which might be seen more often in patients with LS-TIAs because of loss of cerebrovascular vasomotor reactivity due to the high grade carotid stenosis. Although suboptimal hypertension control may prevent LS-TIA symptoms, on the other hand it may increase the frequency of the hyperperfusion syndrome of the brain.[13] Carotid revascularization (endarterectomy or stenting) is considered the treatment of choice for patients with LS-TIA due to severe stenosis because of better patient prognosis compared to medical treatment.[14] Indeed our patient remains asymptomatic more than a year after his carotid endarterectomy was performed. On the other hand LS-TIA in patients with complete carotid artery occlusion can respond to blood pressure optimization.[2,15]

In conclusion, a rare case of LS-TIA is described. Clinicians should be aware of its existence to make the correct diagnosis, while its clinical presentation-association with attempts to manage hypertension-supports further the hemodynamic nature of this entity.

CONFLICTS OF INTEREST: None

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