Orthostatic Transient Ischemic Attacks: A Symptom of Large Vessel Occlusion

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SUMMARY Orthostatic transient ischemic attacks (TIA) are very much rarer than orthostatic generalized cerebral ischemia (syncope). A case is described and previous reports reviewed. Orthostatic TIA appears to occur only with large vessel occlusion. In these patients, collateral blood supply is marginal and unable to support normal postural autoregulation.

TRANSIENT CEREBRAL ISCHEMIC ATTACKS may be focal or generalized (syncpe). The latter are usually caused by a fall in systemic blood pressure, often precipitated by standing. Focal transient ischemic attacks (TIA) however, are rarely associated with hypotensive episodes and even more rarely are precipitated by standing. A case of orthostatic TIA is presented and previous cases from the literature summarized.

Case Report

A 66 year-old left-handed female experienced sudden difficulty speaking, described as stuttering and knowing what she wanted to say, but being unable to say it. She then went to bed and awoke normal. One hour after rising, her symptoms returned for several minutes. Throughout the day, her symptoms recurred each time she stood.

Hypertension had been noted in her twenties, for which she took hydrochlorothiazide, hydralazine and guanethidine. Atrial fibrillation had been present for several years.

Neurological examination on admission revealed a single anomic error among 15 objects and reduced right arm swing. Pulse was 56 per minute and irregular. A pansystolic blowing murmur was heard over the cardiac apex. Blood pressure was 190/90 supine, without postural change. CT brain scan revealed a left internal capsular infarct.

On the second hospital day, prazosin 2 mg per day was begun. The next day, while having her standing blood pressure measured, she became aphasic. Spontaneous speech was unintelligible, with stuttering and paraphasias. Repetition and comprehension were impaired and she could not follow written commands. There was no pronator drift and visual fields remained full. Blood pressure fell from 128/72 supine to 110/60 standing. On return to the supine position, the aphasia resolved over several minutes. She was asked to stand two further times and on both occasions, aphasia appeared after 10 to 20 seconds and resolved on lying down. Antihypertensive medication was discontinued and heparin begun. Twenty-four hours later, she could stand without symptoms.

Carotid arteriography revealed mild stenosis and irregularity of both internal carotid origins and occlusion of several branches of the left middle cerebral artery at their common origin (fig. 1).

Coumadin was begun and hypertension controlled with propranolol.

Discussion

TIAs precipitated by standing have been rarely reported. In the only report devoted to this syndrome, Caplan and Sergay described four patients with the appearance or worsening of a focal deficit upon elevation from the supine position. One of these had occlusion of the left internal carotid artery, two had basilar artery occlusions and the fourth had severe stenosis of the left pericallosal artery. Barnett described a patient with basilar artery occlusion who developed orthostatic TIAs as a result of iatrogenic orthostatic hypotension. A patient with orthostatic TIAs reported by Ausman and colleagues had greater than 90 per cent stenosis of the basilar artery which progressed to complete occlusion within three weeks. Orthostatic TIAs occurred in a patient with bilateral vertebral artery occlusions reported by Hopkins and colleagues. Four patients with complete occlusions complicating carotid endarterectomy had a single posturally related TIA. Bogousslavsky and Regli recently described a patient with bilateral internal carotid occlusions who suffered orthostatic amaurosis fugax. Orthostatic TIAs occurred in a patient with complete occlusion of the right internal carotid artery due to cysticercosis. A syndrome termed "Primary orthostatic cerebral ischemia" by Sundt and colleagues refers to transient generalized cerebral ischemia on assumption of the erect posture without a fall in systemic blood pressure. Focal ischemia may also occur, but only one such case is described in detail. This patient had diffuse bilateral vertebral artery stenoses of such severity that the distal basilar artery was filled from carotid collaterals. The remaining 18 patients in whom the presence of focal
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Figure 1. Left carotid arteriogram showing occlusion of several branches of the left middle cerebral artery, including the operculofrontal complex.

symptoms was not stated. All had bilateral vertebral artery occlusions, unilateral vertebral artery occlusions with no communication between the other vertebral artery and the basilar artery or internal carotid occlusion.

Thus, all previously reported cases and the present case had complete occlusion or less frequently very severe stenosis of a major artery supplying the ischemic area. Orthostatic TIAs appear to be an indication of large vessel occlusion.

This syndrome illustrates the importance of collateral blood supply. When a large artery is occluded, if collateral supply is inadequate, infarction occurs. If it is sufficient, neurological dysfunction is temporary or absent. The vast majority of patients fall into these two groups. However, if collateral supply is marginal, recurrent orthostatic TIAs may occur. It is suspected that in these patients, collateral vessels are already maximally dilated in the supine position, leaving no autoregulatory reserve to prevent clinical ischemia when standing.

References

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