Case Reports

Volume Therapy in Orthostatic Transient Ischemic Attacks

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We report the case of an 83-year-old man with recurrent orthostatic transient ischemic attacks despite anticoagulation and crystalloid therapy. An initial cerebral angiogram revealed a nearly occluded right carotid artery with a string sign. Following aggressive volume expansion with albumin, the patient became asymptomatic. A second angiogram demonstrated the resolution of the carotid string sign and unmasked a high-grade, very tight, surgically approachable stenosis. The role of a high intravascular volume status is discussed. (Stroke 1989;20:1267–1270)

Transient ischemic attacks (TIAs) are pathophysiologically heterogeneous although most are thought to occur because of embolization from the heart or more proximal vessels; hemodynamic TIAs are thought to be relatively uncommon.1,2 The aim of treatment of TIAs is to prevent stroke. With this objective in mind, experimental studies and clinical trials have evaluated platelet antiaggregants, anticoagulants, and surgery.3 Empirical acute management of TIAs has emphasized antithrombotic therapy despite a lack of supporting data from the literature.4,5 The following case points out that, in a few patients, the combination of marginal hypovolemia and large-vessel disease may be better treated with vigorous volume expansion.

Case Report

An 83-year-old man was admitted following the acute onset of left-sided weakness as he was getting up from bed one morning. He described an episode of similar symptoms a month earlier when standing up after dinner. On that occasion, the deficits spontaneously resolved within 1 hour. Medical history was otherwise noncontributory, and he was taking no medications.

On initial examination, when lying flat, his blood pressure was 130/70 mm Hg and his pulse rate was 80 beats/min and regular; on standing, his blood pressure dropped to 112/68 mm Hg and his pulse rate increased to 108 beats/min. A right carotid bruit was audible. The neurologic examination was significant for mild left central facial nerve weakness, left upper extremity plegia, mild proximal left lower extremity paresis, and hyporeactive left-sided reflexes with a Babinski’s sign. In addition, hypesthesia to light touch and pinprick, graphesthesia, stereognosis, and extinction to double simultaneous tactile stimulation were present on the left side.

A computed tomogram of his head showed only mild diffuse enlargement of the lateral ventricles and sulci and a small, well-defined area of focal low density, most consistent with old infarction in the right parietal region.

The patient was treated with heparin and intravenous fluids. One hour later, approximately 2.5 hours after the acute onset of his symptoms, the neurologic deficits resolved. Symptoms recurred acutely 16 hours later when he again developed acute left upper extremity plegia and left lower extremity weakness while sitting up in bed. An orthostatic systolic blood pressure drop of 24 mm Hg was demonstrated. The deficits again cleared within 1 hour.

Despite increasing intravenous fluids and anticoagulation with heparin, the patient experienced seven transient episodes of left hemiparesis, each lasting approximately 1 hour, over the next 72 hours (Figure 1, white field). All TIAs appeared to be related to orthostatic changes although his blood pressure had increased to 140–160/70–85 mm Hg and no longer changed upon sitting up. Even so, an orthostatic pulse rate increase of 15–20 beats/min was still present.

At this point, a cerebral angiogram (Figure 2, left) showed irregularity of the proximal right internal carotid artery, with no contrast seen distal to approximately 1 cm above the bifurcation. On delayed films, a thin “string” of contrast medium was visualized for 6 cm beyond the point of apparent obstruction. Injection of contrast medium into the
left carotid artery demonstrated a smooth plaque at the bifurcation with 15–20% stenosis, normal filling of the intracranial branches, and no cross-filling to the right hemisphere. The patient’s angiographic study was complicated by the development of paralysis of his left upper and lower extremities approximately 10 minutes after injection of contrast medium into the right carotid artery. The deficits resolved after approximately 30 minutes.

Volume expansion with 5% albumin was started (Figure 1, dotted field). Over 28 hours, the patient’s weight increased by 6.2 kg, central venous pressure increased from 5–6 to 13–14 cm H₂O, and his hematocrit dropped by 9%. Mean arterial blood pressure drop); days of hospitalization.

In summary, this case graphically demonstrates the critical role of total body volume depletion, a frequent but rare cause of TIAs. The patient’s response to volume expansion during the early days of hospitalization graphically demonstrated the dramatic impact of aggressive volume restoration on orthostatic TIAs.

**Discussion**

This case is noteworthy because of the orthostatic nature of his focal deficits, stabilization only after aggressive volume expansion, and the radiographic demonstration of resolution of a carotid string sign.

The patient’s symptoms were consistently triggered by orthostatic changes. Though rare, orthostatic TIAs are a good indicator of large-vessel disease. The symptoms are probably caused by the combination of extracranial obstruction and an additional drop in cerebral perfusion due to orthostasis, for which the collateral circulation can no longer compensate. Sudden hypotension due to a variety of other mechanisms in patients with hemodynamically significant extracranial artery stenosis has been reported as a cause of TIAs.  

Initial evidence for hypovolemia was unambiguous but not dramatic (systolic blood pressure decrease 13–20 mm Hg and a pulse rate increase of 30–22 beats/min following orthostatic changes). With intravenous crystalloids, blood pressure changes ceased. However, the patient’s pulse rate continued to rise in response to orthostatic changes, and neurologic deficits were precipitated. We speculate that peripheral vascular sympathetic activity was high to prevent systemic blood pressure drops. Sympathetic hyperactivity constricts the large cerebral vessels, shortening the autoregulatory plateau in situations of hypovolemia.  

More vigorous volume expansion with colloids may have reduced systemic and cerebral vascular sympathetic tone, reestablishing autoregulation at lower blood pressures.

Volume expansion is also associated with reduction of the hematocrit, a major factor influencing blood viscosity, especially in low-flow states. The role of hemodilution in managing TIAs, however, has not been established. Even in completed ischemic infarction, the benefit of hemodilution is controversial.

After intravascular volume expansion, the poststenotic carotid artery filled, with resolution of the string sign, which probably represented poststenotic collapse secondary to decreased blood flow. The poststenotic filling may have occurred via collaterals filling the vessel from above. In any instance, the reversal of the string sign was somewhat unexpected since the extracranial cerebral arteries are not thought to play a capacitance role with marked volume dependence. Perhaps this is altered in the situation of high-grade stenosis.

In summary, this case graphically demonstrates that TIAs may occasionally be provoked by orthostatic changes signaling large-vessel disease and that high volume status may then be beneficial. In
this context, it is important to remember that many patients with acute cerebral ischemia are hypovolemic upon presentation. Vigorous volume expansion with colloids may be necessary when symptoms continue despite treatment with crystalloids in patients with signs of tight extracranial stenosis.

References

KEY WORDS • cerebral ischemia, transient • hypotension, orthostatic
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Stroke. 1989;20:1267-1270
doi: 10.1161/01.STR.20.9.1267

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