Orthostatic Hypotension Improved After Bilateral Carotid Endarterectomy

—Case Report—

Yoshinori AKIYAMA, Nobuo HASHIMOTO, and Masafumi MORIMOTO

Department of Cerebrovascular Surgery, National Cardiovascular Center, Suita, Osaka

Abstract

A 60-year-old male with recurrent syncopal attacks presented with orthostatic hypotension on the head-up tilt test. Angiography also showed severe stenosis of the bilateral extracranial carotid arteries. He underwent two-staged bilateral carotid endarterectomy. After the operations, the orthostatic hypotension resolved and the syncopal attacks have disappeared completely. Orthostatic hypotension in this patient was due to vasodepressor-type carotid sinus syndrome caused by compression of the carotid baroreceptors by atherosclerotic plaques.

Key words: bilateral internal carotid artery stenoses, carotid endarterectomy, carotid sinus syndrome, orthostatic hypotension

Introduction

Orthostatic hypotension is characterized by low upright blood pressure levels. There are many etiologies causing orthostatic hypotension, but vasodepressor-type carotid sinus syndrome is one of the less common. The basis for this syndrome is excessive vasodepressor response of baroreceptors in the carotid sinus, leading to hypotension. The etiology of this syndrome is little known, but includes local pathology in the neck region such as neoplastic tumor and history of radiation. We treated a patient who presented with both orthostatic hypotension and bilateral carotid artery stenosis. After removal of atherosclerotic plaques by bilateral carotid endarterectomy, the orthostatic hypotension completely disappeared.

Case Report

A 60-year-old male was referred to our clinic in June 1996, complaining of recurrent syncopal attacks without warning symptoms. He had experienced nine syncopal attacks after standing up and/or hyperextending his neck. He had always recovered from the loss of consciousness within 5 to 20 seconds. Neither neurological deficits nor convulsions occurred during or after the syncopal attacks. He had no history of major disease, nor was he taking any medicine.

Neurological examination, laboratory screening, and electroencephalographic and electrocardiographic findings were normal. The 60-degree head-up tilt test found that the systolic blood pressure (SBP) decreased by approximately 40 mmHg during head-up. The SBP was 138.8 ± 4.2 mmHg, 97.6 ± 8.5 mmHg, and 135.8 ± 7.5 mmHg, in the supine position before head-up, in the head-up position, and in the supine position after head-up, respectively, without marked change in heart rate (Fig. 1 upper). However, syncope was not induced during the orthostatic blood pressure measurements. Digital subtraction angiography demonstrated severe stenosis of the bilateral extracranial internal carotid arteries (Fig. 2A, B), but no steno-occlusive lesion was demonstrated in the vertebrobasilar system. Single photon emission computed tomography using N-isopropyl-p-[123I]iodoamphetamine in the supine position showed that the cerebral blood flow was decreased in both hemispheres (Fig. 3 upper row). Computed tomography demonstrated multiple lacunar infarctions in both cerebral hemispheres (Fig. 4 left).

Carotid endarterectomy was performed under general anesthesia with transverse skin incision.
without the use of an internal shunt on the right carotid artery on July 17, and on the left carotid artery on August 14. There was no complication after these operations.

Digital subtraction angiography one month after the second operation demonstrated good patency of both carotid arteries (Fig. 2C, D). The cerebral blood flow in both cerebral hemispheres was normal (Fig. 3 lower row). Computed tomography demonstrated no development of a new lesion (Fig. 4 right). The head-up tilt test found no orthostatic hypotension. The SBP was 141.7 ± 5.2 mmHg, 137.6 ± 5.0 mmHg, and 145.8 ± 4.5 mmHg, in the supine position before head-up, in the head-up position, and in the supine position after head-up, respectively, on September 3, 1996, and 145.7 ± 7.9 mmHg, 141.8 ± 5.6 mmHg, and 142.8 ± 4.3 mmHg, respectively, on August 3, 1997 (Fig. 1 middle, lower). Since the surgery, the patient has had no syncopal attack and has been free from other complaints.

**Discussion**

Orthostatic hypotension is widely recognized as a potential cause of syncope resulting from reduction of cerebral blood flow due to hypotension during
postural stress. However, orthostatic hypotension alone may not be a single cause of syncope, and multiple problems may act combine to cause syncope.\textsuperscript{1,5} In our patient, low cerebral perfusion caused by bilateral carotid artery stenosis may have been involved in association with the underlying orthostatic hypotension.

Many etiologies for orthostatic hypotension have been identified, but can be divided into three general mechanisms. The first mechanism is processes interfering with the normal compensatory responses to upright posture, for example, peripheral neuropathy, such as is found in diabetes, and diseases affecting the central nervous system such as Shy-Drager syndrome. The second mechanism involves the overwhelming of otherwise normal reflexes by an intense orthostatic stimulus, for example, those associated with dehydration, deconditioning, and/or malnutrition. The final mechanism relates to interference with reflex responses by, for example, drugs or baroreceptor disturbance.

Vasodepressive-type carotid sinus syndrome, precipitated by hyperactive carotid sinus reflex, is considered to be a cause of orthostatic hypotension, and is characterized by a reduction in SBP without heart rate slowing, leading to syncope. The afferent limb participating in the carotid sinus reflex is the carotid sinus branch of the glossopharyngeal nerve, and the efferent limb is the cardiovascular autonomic nerves. In contrast to the established concept of the peripheral limb of the carotid sinus reflex, the evidence for a central regulatory mechanism of the reflex arc is still not conclusive.

The causative site in carotid sinus syndrome can be situated anywhere along the course of the reflex arc, but the pathophysiological process has not been fully elucidated. A lesion at the level of the carotid baroreceptors may be a major cause,\textsuperscript{10,11} supported by observation that syncope can occur in patients with neoplastic neck tumors, which may directly compress the carotid baroreceptors, and may disappear after removal of the lesions.\textsuperscript{3,9}

Atherosclerotic plaque frequently develops at this region, so its involvement in dysfunction of the carotid sinus can be assumed, although there is no direct evidence. This speculation has received indirect support from the demonstration that the receptor sensitivity to pressure changes is disturbed in patients with atherosclerotic plaques.

The diagnosis of orthostatic hypotension in our case was based on the 60-degree head-up tilt test, which showed the SBP decreased by approximately 40 mmHg without marked change in heart rate. The orthostatic hypotension completely resolved after removal of atherosclerotic plaques by carotid en-
darterectomy. These observations suggest that the orthostatic hypotension in the present patient was due to vasodepressor-type carotid sinus syndrome caused by dysfunction of carotid baroreceptors resulting from compression by the atherosclerotic plaques. This speculation is also supported by the observations that the patient's syncopal attacks were induced by both standing (postural stress) and hyperextension of the neck, which would probably stimulate the carotid sinus.

Therefore, extracranial carotid artery stenosis should be considered in the evaluation of syncope of unknown etiology.

References

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Address reprint requests to: Y. Akiyama, M.D., Department of Cerebrovascular Surgery, National Cardiovascular Center, 5–7–1 Fujishirodai, Suita, Osaka 565–8765, Japan.