Dizziness When Eating: an Unusual Isolated Presentation of Cerebral Venous Thrombosis

Toshio Fukutake*,**, Yutaka Shimoe** and Takamichi Hattori*

Abstract

A previously healthy 60-year-old man had a two-year history of dizziness or faintness when eating but not when drinking. MRI of the brain detected deep venous dilatation, and digital subtraction cerebral angiography showed superior sagittal sinus thrombosis. These symptoms were completely resolved after the daily administration of 200 mg ticlopidine for four weeks. The pathomechanism of this unusual presentation is speculated episodic congestion of the jugular venous drainage during mealtime due to an increase in the circulatory volume of the external carotid-jugular system.

Key words: chewing, swallowing induced syncope, superior sagittal sinus thrombosis, MRI

Introduction

Eating consists chiefly of mastication (chewing) and deglutition (swallowing). Swallowing has been considered a rare but definite cause of syncope (1–4). Almost all patients with swallowing syncope have pathology which is esophageal, cardiac, or both (1–5). The mechanism of this syncope depends on the autonomic reflex of the glossopharyngeal or vagus nerves (5). In contrast, dizziness or syncope when chewing has seldom been reported. We report the isolated appearance of recurrent dizziness when eating (presumably chewing), not when drinking, in a patient with sagittal sinus thrombosis.

Case Report

A previously healthy 60-year-old man with a two-year history of dizziness or faintness when eating was admitted to our hospital. This unusual symptom occurred at meals, regardless of the menu, three to six times per month and disappeared completely afterwards. The duration of a typical episode was up to 10 seconds and there was no paresthesiae or facial flushing. He had never lost consciousness in association with the episode. The longer he chewed at meals, the more easily might develop dizziness. Drinking liquids did not cause this symptom. He had previously consulted two general physicians and an otolaryngologist, all of whom had detected no physical abnormalities and had prescribed minor tranquilizers without benefit. His general physical examination upon admission was unremarkable, except that his blood pressure was 140/98 mmHg and his pulse rate was 52 beats per minute. No orthostatic hypotension or carotid sinus hypersensitivity was observed. Twenty-four hours of continuous monitoring of ECG and blood pressure showed no significant cardiac arrhythmia or blood pressure changes, even during or after meal. Complete neurologic examinations detected no motor or sensory deficits. His cognitive, language and mental functions were normal. The ocular movements were normal in all directions. Other cranial nerve functions also were normal. Both EEGs during a meal and between meals showed no paroxysmal activity. MRI of the brain detected deep venous dilatation in the basement of the brain (Fig. 1), and digital subtraction cerebral angiography (DSA) showed superior sagittal sinus thrombosis (Fig. 2). MR angiography of the circle of Willis and carotid bifurcation showed no abnormality. Extensive examinations of his blood, urine and CSF failed to detect the causes of or predisposing factors for the thrombosis; the only abnormality being mild hyper-IgG-emia (2,372 mg/dl).

During the admission of 7 days, ordinary meals and experimental gum chewing in 10 to 20 minutes once per day failed to elicit an episode. Symptoms were completely resolved after the daily administration of 200 mg ticlopidine for four weeks. At that time, a follow-up MRI and DSA showed little changes in the above-mentioned abnormal findings.

Discussion

In the past, the diagnosis of cerebral venous thrombosis (CVT) was not possible without pathological verification or
selective angiographic confirmation. CVT therefore has been considered a rare disease with a severe clinical course often leading to death. With the advent of improved neuroimaging techniques, more favorable outcomes have been reported. CVT now is known to present with a remarkably wide variety of clinical symptoms and signs (6), and it may occasionally be so insidious that it is oligosymptomatic or asymptomatic (7). Recently Kuehnen et al (8) reported the first series of five patients with transverse/sigmoid sinus thrombosis who presented with single or multiple cranial nerve dysfunctions. Two of them developed vertigo and were initially diagnosed as vestibular neuritis and inflammation of cranial nerve. However, to our knowledge, dizziness that occurs only during eating has not been previously described with this condition.

Mastication has received modest attention in patients with temporal arteritis who occasionally show intermittent claudication of chewing due to narrowing of the external carotid artery or its branch (9). Recently a patient with occlusion of the unilateral common carotid artery was reported, who had episodic dizziness, visual disturbance, and facial and extremity weakness associated with eating (10). A vascular steal syndrome by mastication was speculated as its pathomechanism (10). Unlike these arterial narrowing mechanisms, the dizziness in the present patient when eating may have been caused by episodic congestion of the jugular venous drainage during mealtime due to an increase in the circulatory volume of the external carotid-jugular system, including the tongue and masticatory muscles. Actually our patient showed deep venous dilatation in association with sagittal sinus thrombosis, but no arterial narrowing. Furthermore, liquid swallowing, in which the tongue and masticatory muscles must play little role, elicited no episode. Ticlopidine treatment produced favorable outcome possibly due to improvement of venous congestion although its therapeutic effect for CVT has not been confirmed and there was no significant change between pre- and post-MRs and DSAs.

Swallow-induced or deglutition syncope is a rare disorder, characterized by loss of consciousness during or soon after a swallow. It is considered to be a form of neurally mediated syncope, with an increase in cardiac parasympathetic activity resulting in sinus bradycardia or atrioventricular block (1-4). Recently the first case of such syncope in which continuous hemodynamic monitoring showed a short-lived rise in blood pressure and heart rate, followed by severe hypotension and bradycardia, has been reported (4). This swallow-induced syncope/presyncope is unlikely in the present patient because no abnormal changes of heart rate or blood pressure were detected during and after eating meal or chewing gum. Eating epilepsy (5) is also unlikely because of normal EEGs during a meal and between meals.

In conclusion, dizziness when eating (chewing) should now be included as an unusual presentation of CVT although the mechanism remains unclear.

References

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