Cerebral Ischemic Attack Caused by Postprandial Hypotension

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Background Food ingestion sometimes induces systemic hypotension (postprandial hypotension). Although the possibility of stroke occurring postprandially has been suggested, no cases have been reported until now.

Case Description A 78-year-old man experienced repeated transient ischemic attacks after almost every ingestion of food and showed orthostatic and postprandial hypotension. An angiogram revealed occlusion of his left carotid artery and stenosis of his right middle cerebral artery.

Conclusions Postprandial as well as orthostatic hypotension can be a risk factor for stroke in patients with severe occlusive cerebrovascular disease. (Stroke. 1994;25:511-513.)

Key Words • cerebral infarction • hemodynamics • hypotension

Food ingestion may substantially lower blood pressure, producing such clinical symptoms as dizziness and syncope. Diseases that cause postprandial hypotension (PPH) are Parkinson's disease, multiple system atrophy, and pure autonomic failure. PPH also has been found in some normal elderly individuals. Thirteen of a group of 35 (37%) healthy Japanese subjects aged 41 to 60 years were reported to have PPH; therefore, it is not unusual even in normal individuals.

Appenzeller suggested that cerebrovascular accident may occur after a large meal, but no report of such a case has been described, nor has the mechanism been determined. We here report a case of transient ischemic attacks and cerebral infarction, the genesis of which is closely related to PPH.

Case Report

A 75-year-old, right-handed man was admitted to our hospital because of repetitive transient attacks of right-sided weakness that often occurred postprandially. Symptoms appeared in August 1987. He first noted transient dysarthria and weakness of the right upper limb, continuing for several minutes, a few times a month after having a meal. Approximately 4 months later he was diagnosed as having transient ischemic attacks and essential hypertension. He was treated with a diuretic agent (nicardipine HCL, 80 mg/d, orally). Neverthe less, he continued to experience occasional attacks of dysarthria, right hemiparesis, and drowsiness occurred more frequently, almost after every meal, and the duration also became longer, lasting more than 30 or 40 minutes. In August 1990 he again experienced the attack after a meal, and since then his right hemiparesis has persisted. Moreover, he developed pneumonia. He was admitted to the hospital on August 23, 1990.

On admission the patient was somnolent and had severe right hemiparesis and speech disturbance. His blood pressure fell from 124/68 to 101/59 mm Hg on postural change from recumbent to sitting. He then slavered from his right oral angle, and his right nasolabial fold became shallower. He became somnolent and seemed aphasic, but in a few minutes he recovered.

Magnetic resonance imaging showed multiple ischemic lesions in the bilateral coronal radiation, thalamus, basal ganglia, left ventromedial pons, and left cerebellar hemisphere with a hyperintense signal in the T2-weighted image. Moreover, the previously seen flow void of the left internal carotid artery had disappeared. An intra-arterial digital subtraction angiogram (Fig 1) showed occlusion of the left internal carotid artery, severe stenosis of the M1 segment of the right middle cerebral artery (MCA), and slow filling of both MCAs. Cerebral blood flow (CBF) was measured by xenon-133 inhalation single-photon emission computed tomography before and after intravenous injection of 10 mg/kg acetazolamide. CBF was severely decreased in the left MCA area, and the cerebral vasodilatory reaction to acetazolamide administration was impaired in that area.

An electrocardiogram was normal, and an echocardiogram showed no abnormality except mild mitral valve prolapse.
An electroencephalogram showed a diffuse alpha pattern while he was awake and diffuse 6- to 7-Hz theta waves during drowsiness after meals.

During and after food ingestion, blood pressure was monitored at 2-minute intervals with an automatic sphygmomanometer (Fig 2). The patient had been sitting up for more than 20 minutes, and he began to eat while in the sitting position. His blood pressure began to decrease during eating and reached a minimum approximately 10 minutes after he finished the meal. Soon he slavered from his right oral angle and his right nasolabial fold became shallower; he became somnolent and aphasic within several minutes. When he lay down, his blood pressure rose and those symptoms were relieved.

Upright tilt stimulation with blood sampling was done to evaluate his postural hypotension. Blood pressure and heart rate were monitored continuously with an automatic sphygmomanometer. An intravenous catheter was placed in his left femoral vein, and he was kept recumbent for approximately 30 minutes before tilting. Blood samples were collected just before tilting and at 5, 10, and 15 minutes while in the 60-degree upright position. Blood pressure and heart rate while recumbent were 174/81 mm Hg and 66/min but changed to 126/65 mm Hg and 71/min, without any clinical symptoms, 5 minutes after the upright tilt. Plasma norepinephrine increased from 151 to 213 pg/mL. A Valsalva maneuver for 10 seconds in the supine position failed to produce a hypertensive overshoot. The cold pressor test and the coefficient values of the RR intervals were normal. To evaluate the hypotensive effect of glucose, 75 g of oral glucose was given in the 30-degree head-up position. Blood samples were collected to measure levels of glucose, insulin, and norepinephrine. The patient's blood pressure before glucose loading (184/77 mm Hg) gradually decreased, reaching 124/61 mm Hg 105 minutes after loading. Serum glucose increased from 90 to 184 mg/dL after the intake of glucose, and plasma insulin increased slightly. There was no marked change in the plasma norepinephrine.

After his fixed stroke, the patient occasionally experienced postprandial symptoms such as deterioration of consciousness, right facial paresis, and aphasia. On the basis of the report of Onrot et al, he was treated for PPH with 250 mg caffeine orally per day, but with no relief of his PPH or symptoms. Simply lying down on the bed immediately after food intake had a more beneficial effect on his postprandial symptoms.

Discussion

This case was characterized by repetitive, stereotyped transient ischemic attacks after food ingestion. The
patient had left carotid occlusion and severe right MCA stenosis. Moreover, he experienced orthostatic hypotension, and examinations of autonomic function revealed impairment of the baroreflex. Impaired glucose tolerance might have been responsible in part for his PPH and orthostatic hypotension, which an antihypertensive agent could have exacerbated. However, we could not be certain of the cause of his PPH and orthostatic hypotension.

Stenosis or occlusion of one or more large cerebral arteries might induce "states of cerebral vascular insufficiency" with recurrent symptoms reflecting hemodynamic crises. The role of systemic hypotension in transient ischemic attack or stroke, however, is still a matter of controversy. The recent work of Dobkin suggests that orthostatic hypotension can be a risk factor for transient ischemic attacks if patients have severe occlusive cerebrovascular disease. Orthostatic hypotension in such patients is caused mainly by diabetes mellitus or the administration of antihypertensive agents. Ordinarily, meals are taken in a sitting position. In patients such as ours, who have both postural hypertension and PPH, the act of eating may result in severe systemic hypotension.

In normal subjects the autoregulation system works properly against hypertension so that CBF is maintained. In humans and experimental animals cerebral ischemia is known to impair autoregulation. These findings suggest that in patients with severe occlusive cerebrovascular disease autoregulation cannot function fully against the cerebral ischemia induced by a hypertensive episode such as PPH or orthostatic hypotension. In addition, chronic hypertension causes adaptive cerebral vascular changes that lead to a shift of the lower limit of autoregulation toward high pressure in young and middle-aged patients and in experimental animals. This functional adaptation is considered to be caused by structural hypertensive vascular change, which in elderly hypertensive patients might become irreversible. Our patient's hypertension and cerebral ischemia may have impaired his autoregulation.

In our patient's case it is clear that his stroke was closely related to PPH. PPH therefore must be taken into account in the management of patients with severe occlusive cerebrovascular disease.

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References

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