Case Studies

Postprandial Hypotension and Coma Following Subarachnoid Hemorrhage in a Patient with Parkinson's Disease

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A 79-year-old woman with a history of Parkinson's disease was admitted to our hospital because of a subarachnoid hemorrhage. She underwent clipping the next day. On postoperative days 7-9, she exhibited hypotension and disturbance of consciousness after each meal. The administration of midodrine relieved the hypotension, and postprandial coma was no longer observed. In this case, the autonomic dysfunction in Parkinson's disease and impairment of cerebral autoregulation during cerebral vasospasm may have been involved in the postprandial hypotension (PPH) and coma. PPH occurs not only in patients with Parkinson's disease but also in elderly patients, particularly those with diabetes or hypertension. Therefore, PPH must be considered in the management of cerebral vasospasm following subarachnoid hemorrhage. **Key Words:** Postprandial hypotension—subarachnoid hemorrhage—Parkinson's disease—cerebral vasospasm—midodrine.

Introduction

Postprandial hypotension (PPH) is defined as a decrease in systolic blood pressure (BP) of ≥20 mmHg within 2 hours after a meal.[1] Although there have been few previously reported cases of PPH associated with a cerebrovascular steno-occlusive disease, such as carotid stenosis/occlusion, to the best of our knowledge, this is the first case report of an unusual type of PPH and coma associated with symptomatic vasospasm following subarachnoid hemorrhage (SAH).

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Case Report

A 79-year-old female was admitted to the emergency department because of sudden headache. She had a history of Parkinson's disease (Hoehn and Yahr stage III) and was taking the following anti-Parkinson drugs: levodopa (300 mg/day), carbidopa (30 mg/day), and pramipexole dihydrochloride (.375 mg/day). Physical examination showed mild somnolence and disorientation (Hunt and Hess grade III). The brain computed tomography (CT) and CT angiography revealed a diffuse subarachnoid hemorrhage and an anterior communicating artery aneurysm (Figs 1, 2). Microsurgical clipping was performed the next day. After clipping, preventive treatment for cerebral vasospasm was started using fasudil (90 mg/day) as an inhibitor of Rho-kinase, cilostazol (200 mg/day), and pravastatin (10 mg/day). The anti-Parkinson's drugs were continued. The patient's BP was continuously monitored for 24 hours. Tube feeding was initiated because of insufficient oral intake on postoperative day 2. The amount of tube feeding was gradually increased, and PPH and coma became apparent on postoperative days 6 and 7, respectively (Fig 3A). Approximately 30 minutes after

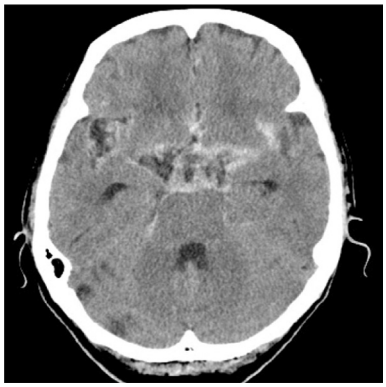

Figure 1. *Computed tomography revealing a diffuse subarachnoid hemorrhage (Fisher Group III) and an anterior communicating artery aneurysm.*

each meal, the patient's BP decreased to approximately 120/60 mmHg from 190/80 mmHg, and she could not open her eyes, did not respond to speech, and only showed withdrawal to pain in her limbs. These symptoms were transient, and after her BP recovered, she could open her eyes and talk. Severe decreases in BP were observed, particularly in the morning and evening. Midodrine

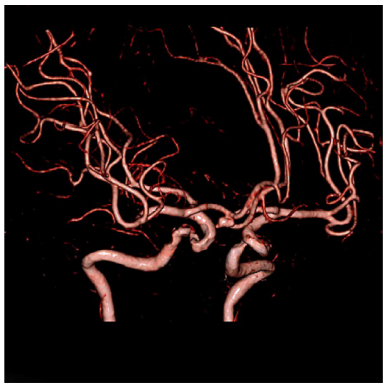

Figure 2. *Computed tomography angiography revealing an anterior communicating artery aneurysm.*

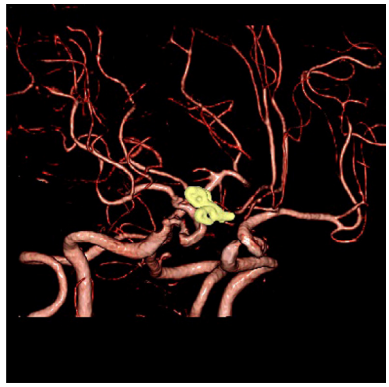

Figure 4. *Computed tomography angiography revealing a residual cerebral vasospasm in the distal anterior cerebral artery and middle cerebral artery on postoperative day 14. We also checked the clipped aneurysm.*

(4 mg/day) for PPH prevention was started on postoperative day 9, successfully relieving PPH (Fig 3B), and postprandial coma was no longer observed. Cerebral vasospasm became symptomatic but did not result in cerebral infarction. We confirmed the presence of a clipped aneurysm and residual vasospasm by CT angiography (Fig 4). Ventriculoperitoneal shunt was performed on day 40, after which the patient became better able to eat and walk. She remained in cognitive impairment and was transferred to a rehabilitation hospital.

Discussion

Autonomic dysfunction in patients with Parkinson's disease is well known, and PPH is often associated with the disease. However, PPH is also found in 25%-38% of elderly patients,[2] particularly those with diabetes or hypertension. Recognizing PPH is important for correct diagnosis and treatment. The pathophysiology of PPH appears to be secondary to a blunted sympathetic response to meal ingestion. The rapid decrease in BP is associated with a number of hypotensive phenomena, in-

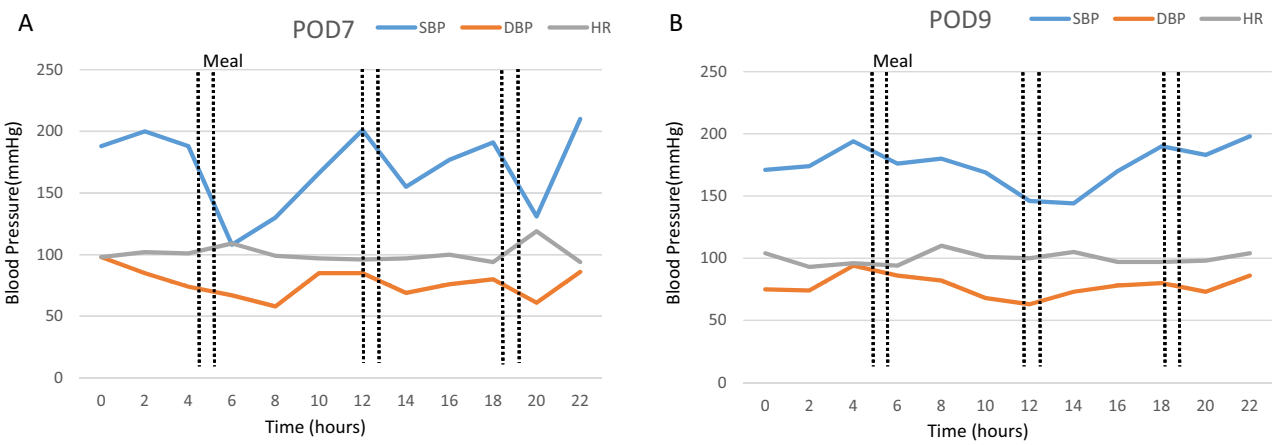

Figure 3. *(A) Graph showing the decrease in systolic blood pressure following meals on postoperative day 7. (B) Graph demonstrating that midodrine improved the decrease in systolic blood pressure on postoperative day 9.*