



Case report

Postprandial cerebral infarction resolved by extracranial-intracranial bypass surgery

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ABSTRACT

A 51-year-old man with type 2 diabetes mellitus was admitted with a 2-month history of repeated episodes of transient aphasia and right hemiparesis after food intake. His blood pressure (BP) fell when the neurological deficits developed. The fall in BP after each meal was confirmed by 24-h ambulatory blood pressure monitoring (ABPM), which established the diagnosis of postprandial hypotension (PPH). Diffusion-weighted magnetic resonance imaging of the brain showed multiple high-intensity lesions at the borderzone between the anterior and middle cerebral artery (MCA) territories in the left hemisphere. Digital subtraction angiography showed tapered occlusion at the origin of the left internal carotid artery (ICA). Despite sufficient antiplatelet therapy and medication for PPH, the transient symptoms remained. Positron emission tomography scanning using $H_2^{15}O$ showed decreased cerebral blood flow with increased oxygen extraction fraction in the left MCA territory. As the symptomatic left ICA occlusion was intractable, an extracranial-intracranial (EC-IC) bypass surgery was conducted without any perioperative complications. Although PPH remained, cerebrovascular ischemic events including repeated transient ischemic attack disappeared for 2 months after surgery. The coincidence of stroke with ABPM-proved transient hypotension suggested that the brain infarcts were caused by hemodynamic changes related to PPH co-existent with the chronic left ICA occlusion. ABPM is useful in evaluating hemodynamic infarcts associated with BP fluctuation, and should be considered for patients with chronic ICA occlusion. In addition, EC-IC bypass may be a treatment option for symptomatic chronic ICA occlusion due to PPH.

1. Introduction

Autonomic dysfunction is frequently present in patients with acute ischemic stroke and is associated with poor clinical outcome [1]. We here report a case of repeated cerebral infarcts caused by postprandial hypotension (PPH), which was successfully treated by extracranial-intracranial (EC-IC) bypass surgery.

1.1. Case report

A 51-year-old man was admitted with a 2-month history of repeated episodes of transient aphasia and right hemiparesis after food intake. He had been diagnosed with type 2 diabetes mellitus at the age of 30 years and suffered from simple diabetic retinopathy, stage 4 diabetic

nephropathy, and advanced neuropathy. Hemoglobin A1c was 10.1%, although insulin degludec and metformin had been prescribed. Hypertension and dyslipidemia were also noted.

He presented with transient aphasia and right hemiparesis again after a meal in the hospital. His blood pressure (BP) was 168/105 mmHg before the meal and dropped to 115/90 mmHg when the neurological deficits developed (Fig. 1). The fall in BP after each meal was confirmed by 24-h ambulatory blood pressure monitoring (ABPM), which established the diagnosis of PPH (Fig. 1).

Ambulatory blood pressure monitoring showing chronological changes in systolic blood pressure (BP) (black line), diastolic BP (black dashed line), and heart rate (HR) (blue line). Decreased BP and increased HR were recorded after each meal. Transient aphasia and right hemiparesis developed after supper (red arrow).

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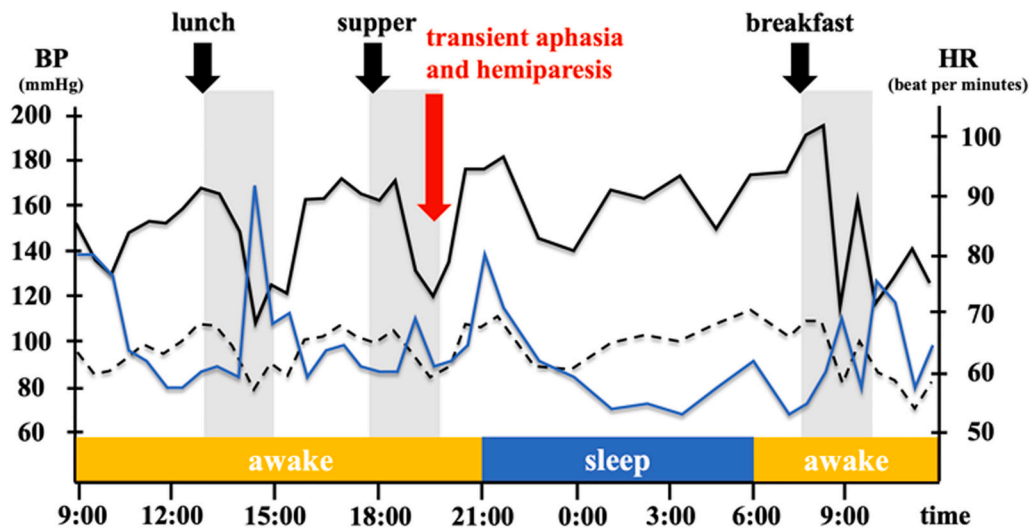


Fig. 1. Ambulatory blood pressure monitoring report on the day of the transient aphasia and hemiparesis.

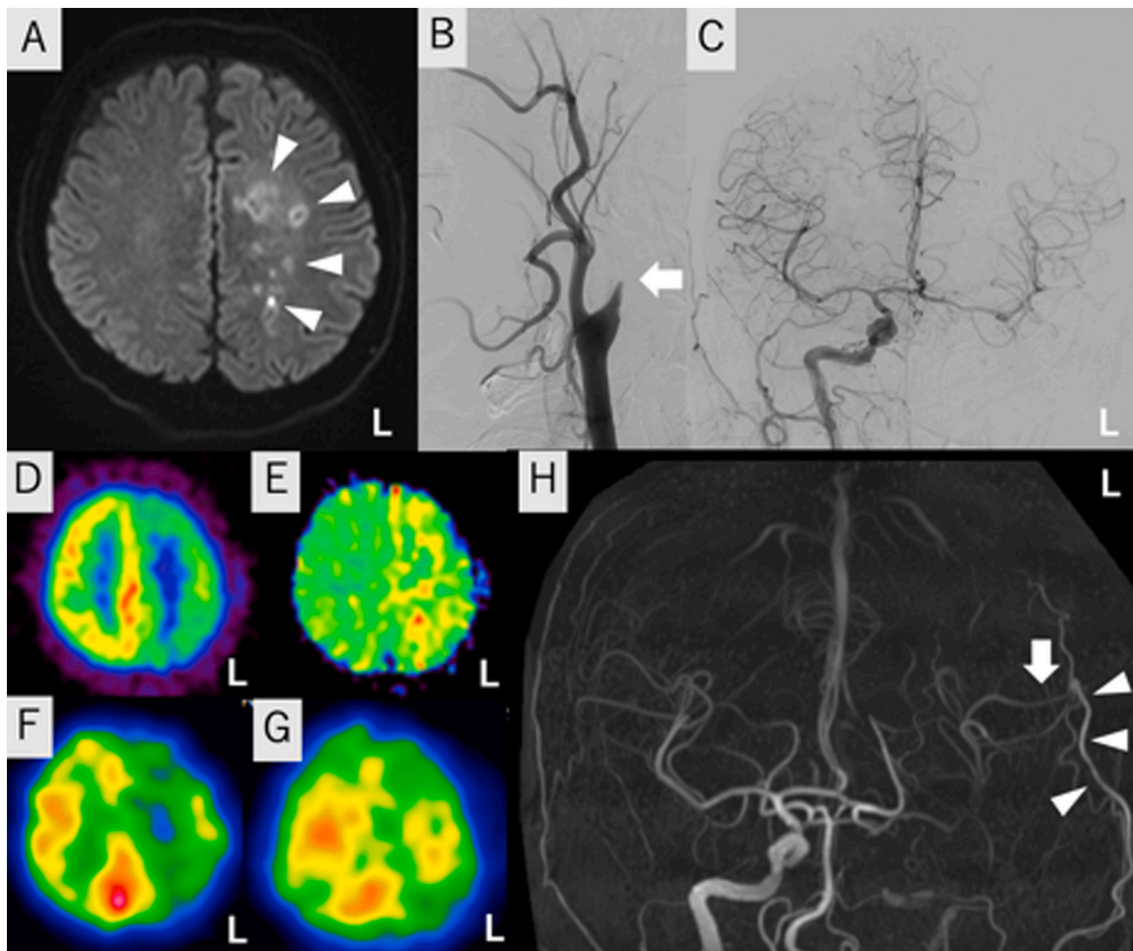


Fig. 2. Brain image findings. A: Multiple high-intensity lesions were illustrated at the borderzone between the anterior and middle cerebral artery territories in the left hemisphere on magnetic resonance diffusion-weighted image (arrowheads). B, C: Digital subtraction angiography showed tapered occlusion at the origin of the left internal carotid artery on lateral view (B, arrow) and perfused left middle cerebral artery (MCA) from the right internal carotid artery through the anterior communicating artery on frontal view (C). D, E: Positron emission tomography imaging with $H_2^{15}O$ showed decreased cerebral blood flow (CBF) (D) and increased oxygen extraction fraction (E) in the left MCA territory compared with the right MCA territory (left-to-right ratio of CBF: 78%; oxygen extraction fraction: 115%). F: Single photon emission computed tomography imaging with ^{123}I -iodoamphetamine showed decreased CBF in the left MCA territory compared with the right MCA territory (left-to-right ratio of CBF: 79%). G: Single photon emission computed tomography imaging with ^{123}I -iodoamphetamine 7 days after surgery showed improvement of CBF in the left MCA territory compared with the right MCA territory (left-to-right ratio of CBF: 90%). H: Magnetic resonance angiography 7 days after surgery confirmed superficial temporal artery-MCA (arrow) bypass patency.

Diffusion-weighted images of brain magnetic resonance imaging showed multiple high-intensity lesions at the borderzone between the anterior and middle cerebral artery (MCA) territories in the left hemisphere (Fig. 2A). Based on the diagnosis of acute cerebral infarction, aspirin and clopidogrel were promptly introduced. Despite sufficient antiplatelet therapy and medication for PPH as well as caffeine intake, meal splitting, and elastic stocking, PPH and transient symptoms remained. Digital subtraction angiography of the left common carotid artery illustrated tapered occlusion at the origin of the left internal carotid artery (ICA) (Fig. 2B). The left MCA was perfused from the right ICA through the anterior communicating artery (Fig. 2C). Positron emission tomography scanning using $H_2^{15}O$ showed decreased cerebral blood flow (CBF) (Fig. 2D) with increased oxygen extraction fraction in the left MCA territory (Fig. 2E). Single photon emission computed tomography with ^{123}I -iodoamphetamine showed reduced CBF in the left MCA territory (Fig. 2F). As the symptomatic left ICA occlusion was intractable, we performed an EC-IC bypass (superficial temporal artery-MCA bypass, single anastomosis) without any perioperative complications. Single photon emission computed tomography with ^{123}I -iodoamphetamine 7 days after EC-IC bypass also showed improvement of CBF in the left MCA territory (Fig. 2G), and magnetic resonance angiography confirmed bypass patency (Fig. 2H). Although PPH remained, ischemic stroke including repeated transient ischemic attack disappeared for 2 months after surgery. In contrast to his previous state, he was able to perform daily activities without assistance 2 months after surgery.

2. Discussion

The presented case was categorized into internal (subcortical) watershed infarcts [2]. The coincidence of stroke with ABPM-proved transient hypotension suggested that the infarcts were triggered by hemodynamic changes related to PPH in addition to the chronic left ICA occlusion. ABPM was useful in evaluating hemodynamic infarcts associated with BP fluctuation.

The common definition of PPH is a fall of at least 20 mmHg in systolic BP within 2 h after meal intake or < 90 mmHg systolic BP after meal intake and > 100 mmHg before meal intake [3]. PPH is occasionally overlooked because of its mild and non-specific symptoms, including dizziness, nausea, light-headedness, and visual disturbances. Nevertheless, PPH is associated with poor prognoses, as increased risks of syncope, falls, heart disease, and stroke have been reported [4]. PPH in the current case may have been induced by autonomic dysfunction related to severe diabetes mellitus [3], although the possibility of neurodegenerative disorders like multiple system atrophy was not totally excluded. Many reports have indicated the difficulty in differentially diagnosing between diabetic neuropathy and early-stage

neurodegenerative disorders, such as multiple system atrophy, Parkinson's disease, and dementia with Lewy bodies [5]. Furthermore, a previous report suggested that carotid plaque may interfere with the reflex responses to reduced blood flow [6], as the baroreceptors in the carotid sinus are involved in the maintenance of BP control. Therefore, the chronic ICA occlusion may also have affected the PPH in the presented case.

To our knowledge, this is the first report to have achieved symptom control and ischemic stroke recurrence prevention by EC-IC bypass for symptomatic ICA occlusion concomitant with PPH. Unfortunately, vasopressor agents that could improve hemodynamic cerebral ischemia were not recommended because of the patient's severe hypertension (Fig. 1). Although general evidence is lacking for EC-IC bypass in stroke prevention in patients with chronic ICA occlusion [7], it may substantially decrease ischemic events when accompanied by substantially decreased CBF and elevated oxygen extraction fraction [8]. The current case suggests that EC-IC bypass is an option for cases with symptomatic chronic ICA occlusion due to PPH; however, this still warrants further investigation.

In conclusion, although autonomic dysfunction is occasionally overlooked, it is an important condition, especially in patients with ICA occlusion. ABPM should be considered for the detection of hemodynamic fluctuations such as PPH.

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