Nonpulsatile Cerebral Perfusion in Patient With Acute Neurological Deficits

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Background and Purpose—An extremely low pulsatile cerebral perfusion can result in a massive cerebral infarction and poor outcome. We report a patient who had complete recovery from initial neurological deficits in spite of nonpulsatile perfusion in the middle cerebral artery.

Methods—We used carotid duplex and transcranial Doppler to evaluate cerebral hemodynamics and the National Institutes of Health Stroke Scale (NIHSS) to score the neurological deficits.

Results—A 62-year-old man had a sudden chest pain and right hemispheric symptoms with NIHSS score of 18 on arrival. Carotid duplex showed no blood flow in the right common carotid artery. Transcranial Doppler showed a nonpulsatile waveform with slow antegrade flow in right middle cerebral artery. Chest CT angiography revealed type A aortic dissection. After surgical repair for the aortic dissection with brain retroperfusion, the patient had dramatic recovery from the initial neurological deficit, and normal pulsatile cerebral perfusion in the right carotid territory.

Conclusions—Nonpulsatile cerebral perfusion points to a proximal source of arterial flow obstruction that may necessitate interventional treatment or surgery in order to restore brain perfusion and potentially reverse impending stroke. (Stroke. 2006;37:1562-1564.)

Key Words: acute stroke ▫ ischemia ▫ nonpulsatile

Case Reports

Cerebral arterial blood flow has pulsatile pattern except for during cardiopulmonary bypass when direct heart contractions are replaced with continuous pump-induced flow. The pulsatility of cerebral perfusion is decreased when an arterial occlusion is present in a proximal segment and compensatory dilatation is induced in the distal vascular bed. Extremely low pulsatile cerebral perfusion was noted in patients with acute proximal internal carotid artery occlusion and infarctions1,2 because it can lead to profound hypoperfusion, ischemia and poor clinical outcome.

We would like to present a patient who achieved complete recovery from a severe initial neurological deficit after surgical correction of the nonpulsatile cerebral perfusion in the middle cerebral artery (MCA).

Case Reports

A 60-year-old man was admitted after acute onset of the left-sided hemiparesis. He also had sudden chest pain and shortness of breath. He collapsed in his bathroom, and then was unable to move his left extremities. He had history of hypertension, myocardial infarction and cocaine abuse. He arrived at hospital within 1 hour after onset. His blood pressure was 110/80 mm Hg in the left arm. Arterial pulsation was good in the left radial artery, but it was not measured in the right side.

On examination, he had left hemiparesis, left facial palsy, dysarthria, gaze preference to the right side, and left homonymous hemianopsia. The initial National Institutes of Health Stroke Scale (NIHSS) score was 18 points.

Transcranial Doppler (TCD; PMD 100 Spencer Technologies) revealed a nonpulsatile flow in the right MCA at depth range 40 to 65 mm with the mean flow velocities of 25 cm/second. The right anterior cerebral artery (ACA) showed retrograde flow direction indicating the left-to-right anterior cross-filling (Figure 1). The left ACA showed a normal antegrade flow direction. Posterior communicating arteries were not identified on both sides. The right vertebral artery showed alternating flow with retrograde systolic flow and antegrade diastolic flow suggesting a severe occlusive disorder proximal to its origin (Figure 1). Carotid duplex (Sonosite, Bothell) with power mode did not show any flow in the right common carotid artery (CCA). B-mode showed echogenicity in the right CCA suggesting thrombus within the lumen (Figure 1). Spectral Doppler showed a mid-systolic velocity deceleration in the left CCA and left MCA. Such deceleration could be caused by a narrowing of the aortic lumen attributable to dissection creating transient suction (Ventury) effect at the origin of the left CCA, which in turn produces mid-systolic retraction in Doppler waveform of left
CCA and MCA. Based on ultrasound findings, CT angiography of the chest was performed. It showed type A aortic dissection involving the ascending aorta, the origins of the left CCA and right innominate arteries (Figure 2). The right CCA and proximal subclavian artery were not visualized, whereas the distal subclavian and right vertebral arteries were present (Figure 2). His noncontrast brain CT scan was normal.

Figure 1. Carotid duplex and TCD findings. TCD shows nonpulsatile flow in right MCA from depth of 60 mm to 40 mm (A and B) and retrograde flow in right ACA (arrow in B). Left ACA shows normal direction and pulsatility (C). Left MCA shows mid-systolic flow deceleration (D). Right vertebral artery shows alternating flow with retrograde systolic flow and antegrade diastolic flow (E). Proximal basilar artery shows antegrade flow (F). Duplex sonography shows echogenicity without flow signal in right CCA in power mode (H) and Doppler sonography (G). Left CCA shows mid-systolic flow deceleration in Doppler sonography (I and J).

Figure 2. Brain CT at 3 days after onset shows subcortical infarction in right frontal lobe (arrow in A). Chest CT angiography shows type A aortic dissection involving ascending aorta (arrow in E) and the origins of right innominate artery and left CCA (arrow in C and D). CT angiography shows distal segment of right subclavian artery, left subclavian artery (curved arrow in C and D), and right and left vertebral artery (arrow in B), whereas the right CCA and proximal subclavian arteries were not visualized.
The patient was transferred to cardiothoracic surgery, and at 3 hours after symptom onset an urgent surgical repair for the aortic dissection was initiated. Dissected ascending aorta and aortic arch were replaced with an artificial graft. Left CCA and right innominate artery were cleared of intimal flaps. Surgery was performed with extracorporeal perfusion pump, hypothermic isoelectric cardiac arrest, brain retroperfusion, and continuous TCD monitoring using previously published methods.4

Follow-up brain CT scan showed subcortical infarctions including right frontal lobe at 3 days after onset. It could be a watershed infarction between right MCA and ACA (Figure 2).

The patient’s neurological deficit improved markedly within 2 days after surgery by >12 NIHSS points, and he was subsequently sent home without any neurological deficit.

**Discussion**

The clinical significance of a nonpulsatile cerebral perfusion is not well understood yet. Our case illustrates that a nonpulsatile cerebral perfusion during acute cerebral dysfunction indicates a proximal arterial occlusion, should prompt a thorough vascular imaging, and may require an urgent intervention to open the proximal occlusion. Timely repair may lead to good clinical outcomes if cerebral perfusion is restored on time.

Pulsatility of cerebral perfusion is determined by the pulsatile energy of blood column delivered during the cardiac cycle to the cerebral vascular bed and by the degree of compensatory dilatation of distal vessels.2 With an internal carotid artery (ICA) occlusion, the MCA blood flow was usually maintained through long narrow collateral channels such as communicating arteries. These long narrow collateral channels may prevent the transmission of pulsatile energy to the MCA, leading to a delay in the systolic flow acceleration. This may appear as a blunted signal on TCD.5

A proximal arterial occlusion causes compensatory dilatation of the distal vascular bed because it decreases vascular resistance and creates a pressure gradient for collateral flow. Patients with an ICA occlusion may show exhausted vaso-motor reactivity, suggesting full dilatation of intracranial vessels. If the collateral circulation is insufficient and takes a long circuit to reach dilated MCA branches, the systolic acceleration and velocity decrease further, and systolic velocity becomes nearly equal to the diastolic flow. These mechanisms contribute to the appearance of a nonpulsatile MCA perfusion.

Nonpulsatile perfusion may be caused by several mechanisms, including prevention of transmission of pulsatile energy, compensatory arterial dilatation distal to the occlusion, and insufficient collateral circulation. Therefore, nonpulsatile cerebral perfusion may be a sign of an exhausted cerebral vascular reserve capacity.6

In a chronic ICA occlusion, nonpulsatile perfusion was not associated with any cerebral dysfunction and returned to normal pulsatile perfusion after removal of the proximal occlusion.6 However, extremely low pulsatile perfusion in acute ICA occlusion was associated with massive cerebral infarction and poor outcomes.2 So, nonpulsatile cerebral perfusion associated with acute cerebral dysfunction can be a sign of impending irreversible cerebral ischemic injury unless the offending lesion is corrected.

Our case illustrates that nonpulsatile cerebral perfusion coupled with bilateral abnormal flow findings in an acute stroke patient should make a clinician think about possible aortic dissection. It also suggests poor collateral circulation and may require an urgent intervention to repair the aortic dissection.

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**References**


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