Significance of Low Perfusion With Increased Oxygen Extraction Fraction in a Case of Internal Carotid Artery Stenosis

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Background and Purpose: Decreased cerebral blood flow with an increased oxygen extraction fraction, the so-called misery perfusion syndrome, suggests a vulnerability to reduction in cerebral perfusion pressure and a tendency to develop cerebral infarction. It is uncertain, however, whether the infarct would occur in the brain region specifically exhibiting this condition.

Case Description: We report the case of a patient with right intracranial internal carotid artery stenosis who presented with mild left hemiparesis resulting from a right frontal watershed infarct. Positron emission tomography 2 months after the stroke showed decreased cerebral blood flow with an increased oxygen extraction fraction in noninfarcted areas of the affected hemisphere. Maximal changes were detected in the watershed area between the middle cerebral artery and the posterior cerebral artery. Three months later, while on antiplatelet therapy, he suffered a new infarct in the right temporo-occipital watershed area that had shown the highest oxygen extraction fraction value on the first positron emission tomographic study. One month after the recurrence of stroke, a second study showed that low perfusion with increased oxygen extraction fraction persisted in the affected hemisphere to a lesser degree than in the first study.

Conclusions: This observation suggests that the area of low perfusion exhibiting the highest oxygen extraction fraction has the highest risk for infarction. Increased oxygen extraction fraction may be an important factor in the development of hemodynamic infarction. (Stroke 1992;23:431-432)

KEY WORDS • carotid artery diseases • perfusion • hemodynamics

Decreased cerebral blood flow (CBF) with an increased oxygen extraction fraction (OEF) is considered to imply a vulnerability to reduction in cerebral perfusion pressure and a tendency to develop cerebral infarction due to hemodynamic factors. No report has, however, documented progression to cerebral infarction in the brain area specifically exhibiting this condition as a result of chronic occlusive cerebrovascular disease.

This report concerns a patient with a watershed infarct and chronic low perfusion who suffered a recurrent episode during the follow-up period. The new infarct was located in the region that had shown the highest OEF value on the positron emission tomography (PET) study before the second attack.

Case Report

We studied a 47-year-old man with mild left hemiparesis, followed by complete recovery after 1 month. The patient had experienced two previous attacks of transient left hemiparesis. He had a history of hypertension and had smoked one pack of cigarettes a day for many years. Computed tomography showed a corticocortical watershed infarct in the right superior frontal region. Cerebral angiography revealed a severe stenosis of the right intracranial internal carotid artery with collateral circulation through a leptomeningeal anastomosis from the right posterior cerebral artery (PCA) to the right anterior cerebral artery. Although he was treated with ticlopidine HCl 0.2 g/day, he continued to occasionally experience dizziness and transient paraesthesia of the left upper and lower extremities with abrupt standing.

Two months after the onset of symptoms, a PET oxygen-15 steady-state study was performed using a Positologica III PET scanner that had an intrinsic spatial resolution of 7.6x7.6x12.0 mm in full width half maximum. We analyzed three tomographic planes parallel to the orbitomeatal line, corresponding to the levels of the basal ganglia and thalamus, body of the lateral ventricle, and centrum semiovale. We examined each image by placing a total of 18–20 circular regions of interest over the cerebral cortex. The regions of interest in all three images were included in the distribution of the anterior cerebral artery, middle cerebral artery (MCA), and PCA, as well as the watershed areas between the anterior cerebral artery and MCA and the MCA and PCA.
Positron emission tomography showed decreased CBF with an increased OEF and relative preservation of cerebral metabolic rate of oxygen (CMRO₂) in noninfarcted areas of the affected hemisphere (hemispheric CBF 27.0 ml/100 g/min, CMRO₂ 2.23 ml/100 g/min, OEF 0.54 [excluding the infarcted area]); PaCO₂ 43.1 mm Hg, PaO₂ 93.1 mm Hg, hemoglobin 11.7 g/dl, and hematocrit 34.6%, as compared with eight normal controls 39 ± 14 (mean ± SD) years of age (mean ± SD hemispheric CBF 39.5 ± 5.7 ml/100 g/min, CMRO₂ 3.18 ± 0.60 ml/100 g/min, OEF 0.41 ± 0.05; PaCO₂ 41.1 ± 2.4 mm Hg, PaO₂ 96.7 ± 6.5 mm Hg, hemoglobin 14.0 ± 1.4 g/dl, and hematocrit 41.0 ± 4.4%). The highest changes were detected in the watershed area between the MCA and PCA (CBF 23.3 ml/100 g/min, CMRO₂ 2.22 ml/100 g/min, and OEF 0.61).

Four months after the first event, he experienced left homonymous hemianopsia. Computed tomography disclosed a new infarct in the right temporo-occipital watershed area that had shown the maximum OEF value on the first PET study (CBF 22.6 ml/100 g/min, CMRO₂ 2.21 ml/100 g/min, OEF 0.64), and cerebral angiography revealed no change. One month after the recurrence of stroke, a second PET study demonstrated that low perfusion with increased OEF persisted in the affected hemisphere (hemispheric CBF 26.2 ml/100 g/min, CMRO₂ 2.37 ml/100 g/min, OEF 0.47 [excluding the infarcted area]; PaCO₂ 44.0 mm Hg, PaO₂ 94.1 mm Hg, hemoglobin 14.8 g/dl, and hematocrit 42.9%) to a lesser degree than in the first study.

Discussion

Because the most distal brain region perfused by the collateral circulation is most susceptible to a reduction in perfusion pressure in large-vessel occlusive diseases, low perfusion and, probably, cerebral infarction owing to hemodynamic factors occurs easily in this area. Although the first PET study showed decreased CBF and increased OEF in all noninfarcted areas of the affected hemisphere, the watershed area between the MCA and the PCA exhibited the maximal changes. This region is the most distal area perfused by the diseased carotid artery through the MCA, which was considered hemodynamically vulnerable based on this patient's angiographic findings. It was clear that the recurrent infarct occurred in this region. Areas with increased OEF exhibited decreased PaCO₂ responsiveness, indicating a poor autoregulatory response that causes prompt CBF decrease with perfusion pressure reduction. Therefore, these areas have a greater risk for hemodynamic infarction than do the other areas of the brain.

The natural course of low perfusion with increased OEF is probably variable; it would persist in some patients and others would escape from it, complicating the prognosis of the patients showing it. Only serial study in each patient would disclose this problem. Contributing factors might include the development of collateral circulation, progression of atherosclerosis or arteriolosclerosis of cerebral arteries, and variations in general circulatory perfusion pressure and cardiac output. In any case, a small subgroup of patients with increased OEF might be at increased risk for stroke, implying that identification of these patients is essential for the prevention of stroke.

In the patient described here, the infarction actually occurred in the region with the highest OEF, indicating that this region is at higher risk for stroke. Our patient suffered recurrent stroke despite antplatelet therapy, which probably has little benefit for the prevention of hemodynamic infarction. We must seek other specific medical therapy for patients exhibiting low perfusion with increased OEF.

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