Spontaneous Cervical Internal Carotid Dissection Presenting With Intracerebral Hematoma

Cervical internal carotid artery dissection is recognized as an uncommon cause of stroke.1–3 We describe a rare case of spontaneous cervical internal carotid dissection presenting with an intracerebral hematoma (ICH).

A 49-year-old right-handed man was referred to our hospital because of right parietal ICH (Fig 1). On admission, he was confused and had left hemiparesis and hemisensory disturbance. Mild dyscalculia, dressing apraxia, and left hemianopsia were present. Horner’s sign was not present, and carotid bruit was not audible on either side. He had asthmatic diaphoresis but had no history of hypertension or cervicocephalic trauma. He was not a drug abuser. Routine laboratory blood analysis revealed no particular abnormality. Both bleeding time and coagulation time were within normal limits, although prothrombin time was 64% of the control. Admission computed tomography (CT) also revealed a small low-density area at the tip of the right temporal lobe (Fig 2A). This low density appeared definitely hyperintense on both T1- and T2-weighted images on magnetic resonance imaging examination (Fig 2B and 2C). A right carotid angiography revealed an aneurysmal outpouching and distal stenosis of the cervical internal carotid artery at the level of C1-2 (Fig 3). There was no evidence of intracranial arterial occlusion or vasculitis. The patient was treated conservatively, without anticoagulants. Four serial angiograms performed from onset to the fourth month showed progressive improvement, with resolution of the hematoma. Antiplatelet drug was started for prevention of embolism from the dissection 2 months after the onset. The patient was discharged for outpatient follow-up with minimal neurological deficits.

According to the literature, common clinical manifestations of spontaneous cervical carotid dissection are focal cerebral ischemia, headache, oculosympathetic palsy, bruit (tinnitus), amaurosis fugax, and neck pain.1–4 None of these symptoms are associated with intracranial hemorrhage, and to our knowledge there have been no reports of a spontaneous cervical internal carotid dissection presenting with an ICH. We believe the right parietal ICH was caused by cerebral embolism from the dissection for the following


FIG 1. Axial computed tomographic scan on admission shows a right parietal subcortical hematoma with mild to moderate surrounding edema.

FIG 2. Axial computed tomographic (CT) scan (A) and T1-weighted (B) and T2-weighted (C) magnetic resonance images taken on admission. The hyperintensity at the tip of the right temporal lobe (arrow) on T1- and T2-weighted images corresponds to the low-density area (arrowhead) on CT scan. This lesion is considered to represent an infarcted area with slight hemorrhagic transformation.
reasons: (1) Hemorrhagic infarctions may sometimes form a massive hematoma; (2) The patient had no other apparent predisposing factors responsible for the lobar ICH; (3) The ICH in the parietal lobe was within the vascular territory of the affected artery; and (4) The coexisting right temporal lesion was also a hemorrhagic infarction within the same vascular territory. We suggest that cervical internal carotid dissection be recognized as a rare cause of lobar ICH.

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References

Red Blood Cell Deformability Related to Perfusion Pressure in Cerebral Infarction With and Without Hypertension

Patients with acute stroke are often hypertensive; however, some authorities do not recommend lowering blood pressure because of the risk of cerebral ischemia. The lowering of blood pressure leads to a decrease in perfusion pressure in the cerebral microvascular bed, with disturbance of the passage of red blood cells (RBCs) through capillaries. It has been suggested that RBC passage may be disturbed more when there is a decrease in RBC deformability. We have studied RBC deformability modeling different levels of perfusion pressure in patients with acute brain infarction.

Forty-eight patients (27 men aged 45 to 60 years) with hemispheric infarction were observed during 3 days after the onset of symptoms. Patients were excluded if there was a major life-threatening illness that might interfere with survival (eg, diabetes, myocardial infarction, renal failure, or malignant hypertension) or evidence of cerebral hemorrhage or brain tumor. All patients had atherosclerosis, and 30 had had arterial hypertension for 5 to 15 years. Systolic arterial pressure ranged from 160 to 180 mm Hg and diastolic pressure from 90 to 115 mm Hg.

Red cell deformability was determined by the method of Reid et al. Washed red cells suspended in phosphate buffer saline (hematocrit, 0.08) were passed through a 5-μm filter under a perfusion pressure of 1 to 60 cm H2O at a temperature of 37°C. The results were expressed as a filtration rate (FR), calculated as the volume (in milliliters) of RBCs passing through the filter in 1 minute. Normal FR levels were measured in a control group of 30 healthy volunteers (17 men aged 42 to 60 years) without a history of diseases associated with increased risk for cerebrovascular events.

In healthy subjects, the FR measurement at different pressures showed that above 8 cm H2O, FR remained practically independent of the pressure. Below this value, FR decreased sharply (Figure). In hypertensive patients with brain infarction, FR decreased below 20 cm H2O; in nonhypertensive patients, the decrease in FR appeared below 12 cm H2O (Figure). These changes resulted in differences of RBC filterability at low and high levels of the perfusion pressure between healthy subjects and patients with brain infarction. At 40 cm H2O, FR in the hypertensive and nonhypertensive patients was less than that in control subjects by 18.1±1.2% and 11.7±1.1%, respectively (P<.05).

Filtration rate (FR, ml/min) of red blood cells depending on perfusion pressure (cm H2O) in healthy subjects (1) and patients with brain infarction without (2) and with (3) hypertension.
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