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Transient crossed cerebellar diaschisis secondary to cerebral hyperperfusion following carotid endarterectomy

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We report a case of transient crossed cerebellar diaschisis secondary to cerebral hyperperfusion following carotid endarterectomy. The appearance of crossed cerebellar diaschisis under the presence of cerebral hyperperfusion may suggest the development of hyperperfusion syndrome.

Key words: carotid endarterectomy, cerebral hyperperfusion, crossed cerebellar diaschisis

INTRODUCTION

CROSSED CEREBELLAR DIASCHISIS (CCD) is defined as a reduction in metabolism and blood flow in the cerebellar hemisphere contralateral to a supratentorial lesion.[1] This phenomenon is often demonstrated in images obtained by single-photon emission computed tomography (SPECT) or by positron emission tomography.[1–5] The mechanism underlying CCD reportedly consists of interruption of the cerebropontocerebellar pathway that causes deafferentation and transneural metabolic depression of the contralateral cerebellar hemisphere.[3–5] When CCD is observed, regional CBF in the cerebral cortical area causing CCD is commonly decreased.[1]

Cerebral hyperperfusion after carotid endarterectomy (CEA) is defined as a major increase in ipsilateral cerebral blood flow (CBF) following surgical repair of carotid stenosis that is well above the metabolic demands of the brain tissue.[6,7] A rapid restoration of normal perfusion pressure following CEA may result in regional hyperperfusion secondary to impaired autoregulation that occurs in the context of chronic ischemia.[6] The clinical symptoms of cerebral hyperperfusion syndrome include unilateral headache, face and eye pain, seizure, and focal symptoms that occur secondary to cerebral edema or intracerebral hemorrhage.[6–8] Intracerebral hemorrhage due to cerebral hyperperfusion is uncommon, but the prognosis for patients with this condition is poor.[6–10]

In the present report, we describe a case of transient CCD secondary to cerebral hyperperfusion following CEA. The appearance of CCD may suggest the development of cerebral hyperperfusion syndrome.

CASE REPORT

A 72-year-old man with severe stenosis of the bilateral cervical internal carotid arteries (ICAs) was admitted for CEA after developing bilateral impairment of visual acuity and experiencing two episodes of transient ischemic attacks with right motor weakness. The patient's medical history was significant for hypertension, and his medication profile included aspirin and a calcium channel blocker. A magnetic resonance (MR) imaging scan revealed multiple small infarcts in the bilateral putamen and cerebral white matter. Cerebral angiography revealed 95% stenosis of the bilateral cervical ICAs. Regional CBF was quantitatively measured using the [^{123}I]*N*-isopropyl-*p*-iodoamphetamine autoradiographic method with SPECT scanning.[11] The SPECT studies were performed using a ring-type SPECT scanner, a Headtome-SET080 (Shimadzu Corp., Kyoto, Japan), which provides 31 tomographic images simultaneously. The spatial resolution of the scanner with a low-energy, all-purpose collimator is 13 mm FWHM at the center of the field of view, and the slice thickness was 25 mm FWHM at the field of view center. Image slices were taken at 5 mm center-to-center spacing parallel to the orbitomeatal line. The images were

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Table 1 Mean blood flow before and after surgery*

	Before surgery	Immediately after surgery	Three days after surgery	Nine days after surgery
Right cerebral hemisphere	24.1	29.8	30.3	26.7
Left cerebral hemisphere	21.4	47.4	53.9	26.9
Right cerebellar hemisphere	34.2	36.8	27.8	34.1
Left cerebellar hemisphere	35.7	35.9	35.1	33.5

*: ml/100 g/min

reconstructed using the weighted-filtered backprojection technique, in which the attenuation correction was made by detecting the edge of the object. An attenuation coefficient of 0.065 cm^{-1}, a Butterworth filter (cutoff = 0.45 cycle/cm; order = 3) and a ramp filter were used for image reconstruction. After a 1-min intravenous infusion of 222 MBq of [^{123}I]IMP (5-m*l* volume) at a constant rate of 5 m*l*/min and a 1-min infusion of physiological saline at the same rate, data acquisition was performed at a mid-scan time of 30 min after the IMP administration for a scan duration of 20 min. Preoperative SPECT showed decreased perfusion of the bilateral cerebral hemispheres (Fig. 1A, Table 1). Exercise electrocardiography, myocardial scintigraphy and coronary angiography were performed and were consistent with the absence of coronary artery disease.

The patient underwent a left CEA without the use of an intraluminal shunt. A continuous 16-channel electroencephalography tracing showed no abnormal findings including duration of ICA clamping. SPECT performed immediately after CEA revealed hyperperfusion in the left cerebral hemisphere and equal perfusion in the bilateral cerebellar hemispheres (Fig. 1B, Table 1). Following patient recovery from anesthesia, no new neurological deficits were noted, and intensive control of systolic blood pressure to a goal range between 100 and 140 mmHg was instituted using 0.45 mg/day of the oral centrally-acting alpha 2-agonist, clonidine (Catapres®), and 3 μg/kg/minute of the intravenous calcium antagonist, nicardipine (Perdipine®). SPECT performed three days after CEA revealed persistence of hyperperfusion in the left cerebral hemisphere and hypoperfusion in the right cerebellar hemisphere (Fig. 1C, Table 1). Subsequently, diffusion-weighted MR imaging was performed with a Signa VH/i 3.0 T (General Electric Systems, Milwaukee, WI) using the following parameters: TR 6000 ms, TE 80 ms, matrix 256 × 260, FOV 240 mm, 6 mm slice thickness, and b value 800 s/mm^2. The imaging demonstrated the absence of hyperintense lesions in the left cerebral hemisphere and the right cerebellar hemisphere (Fig. 2A, B, C). The systolic blood pressure goal was reduced to below 90 mm Hg, which was achieved by an increase in the intravenous nicardipine to 7–10 μg/kg/minute.

The patient had an uneventful course until the 4th postoperative day, when he experienced onset of confusion and right-sided motor weakness. Repeat diffusion-weighted MR imaging demonstrated an absence of hyperintense lesions in the left cerebral hemisphere and the right cerebellar hemisphere, and propofol (Diprivan®) coma was induced. SPECT performed on the 9th postoperative day showed resolution of the hyperperfusion in the left cerebral hemisphere and the hypoperfusion in the right cerebellar hemisphere (Fig. 1D, Table 1), and the propofol coma and intensive control of blood pressure were discontinued. The patient ultimately experienced full recovery following termination of the propofol coma.

DISCUSSION

In the present case, left cerebral hemispheric hyperperfusion was observed on SPECT imaging performed immediately and three days after CEA. In addition, diffusion-weighted MR imaging demonstrated an absence of abnormal lesions in the left cerebral hemisphere. Thus, confusion and right-sided motor weakness developing on the 4th postoperative day might indicate cerebral hyperperfusion syndrome. Blood flow in the right cerebellar hemisphere decreased after a three day period of left cerebral hemispheric hyperperfusion, and both the cerebral hyperperfusion and cerebellar hypoperfusion resolved with resolution of the hyperperfusion syndrome. Furthermore, diffusion-weighted MR imaging demonstrated an absence of abnormal lesions in the right cerebellar hemisphere. These findings suggested that hyperperfusion in the left cerebral hemisphere leads to subsequent hypoperfusion in the right cerebellar hemisphere, which is the definition of CCD.

When CCD is observed, regional CBF in the cerebral cortical area causing CCD is usually decreased.[1] While CCD also may occur in patients with cerebral hyperperfusion due to recanalization during the subacute stage of embolic stroke,[2,12] this form of CCD is not caused by cerebral hyperperfusion but, rather, by ischemic brain damage secondary to the cerebral embolism.[2] In addition, CCD in such patients persists for longer periods.[2,12] In contrast, while the epileptic focus ictally exhibits regional cerebral hyperperfusion in patients with seizure, blood flow in the contralateral cerebellum increases transiently.[13–15] These reversible phenomena of CCD have been called "crossed cerebellar hyperperfusion." Thus, the situation in which cerebral hyperperfusion directly leads to transient CCD without the presence of culprit

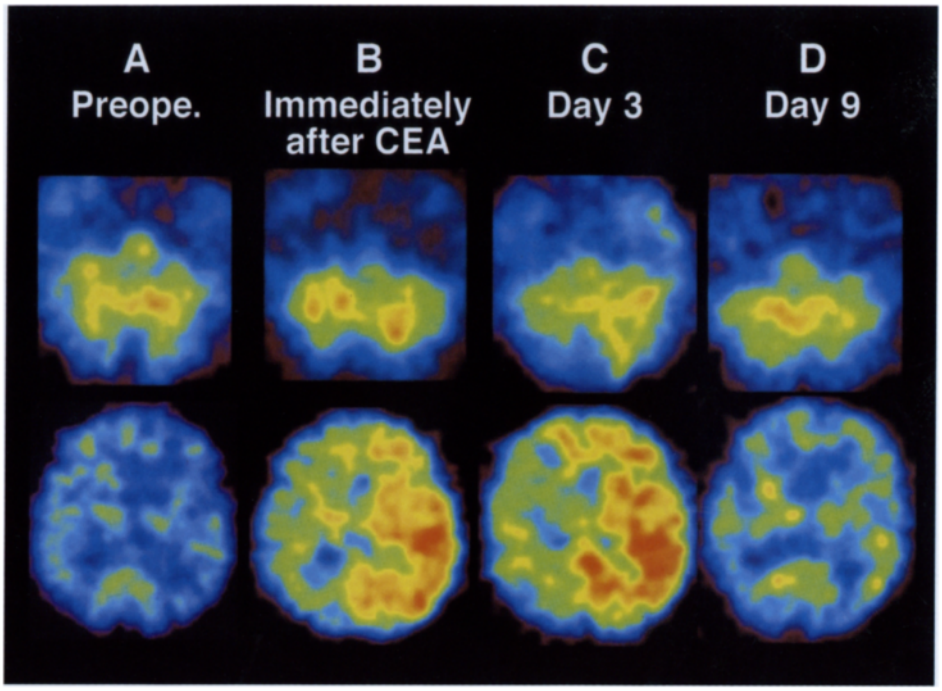

Fig. 1 Serial quantitative cerebral blood flow images using [^{123}I]*N*-isopropyl-*p*-iodoamphetamine single photon emission computed tomography (SPECT) in a patient with cerebral hyperperfusion syndrome after left carotid endarterectomy (CEA). A, Preoperative perfusion in the bilateral cerebral hemispheres was reduced compared with that in the cerebellum. B, While perfusion in the left cerebral hemisphere was markedly increased immediately after CEA, perfusion in the bilateral cerebellar hemispheres remained equal. C, Hyperperfusion in the left cerebral hemisphere persisted, and right cerebellar hemispheric perfusion was reduced on the 3rd postoperative day. D, Hyperperfusion in the left cerebral hemisphere and hypoperfusion in the right cerebellar hemisphere resolved on the 9th postoperative day.

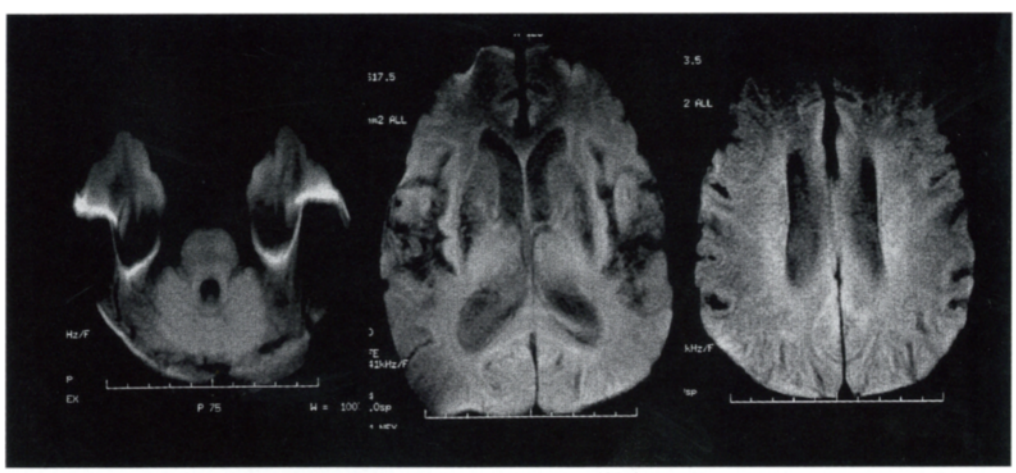

Fig. 2 Diffusion-weighted magnetic resonance images obtained after the appearance of crossed cerebellar diaschisis showed no hyperintense lesion in the left cerebral hemisphere (A, B) or the right cerebellar hemisphere (C).

lesions on MR imaging is a novel one and suggests that cerebral hyperperfusion following CEA may reduce cerebral metabolism, even when asymptomatic.

The incidence of post-CEA hyperperfusion obtained using CBF measurement was 8–12%.[6,16] However, patients with cerebral hyperperfusion that occurs early after CEA do not all always experience hyperperfusion syndrome.[9,10,16,17] In addition, while strict control of blood

pressure in the postoperative period may be effective in preventing intracerebral hemorrhage due to hyperperfusion, hyperperfusion syndrome without intracerebral hemorrhage may develop even with low blood pressure.[9,10,18] Thus, it is not clear which patients may develop hyperperfusion syndrome. In the present case, SPECT performed immediately after CEA revealed no CCD despite the presence of cerebral hyperperfusion, and symptoms due to cerebral hyperperfusion, (e.g., hyperperfusion syndrome) developed on the day after the appearance of CCD. Therefore, the preceding CCD may suggest the development of occult hyperperfusion syndrome as a result of reduction of cerebral metabolism due to cerebral hyperperfusion.

While post-CEA hyperperfusion on SPECT images has been often described,[10,16–18] cerebellar perfusion during cerebral hyperperfusion has not been reported. We believe that transient crossed cerebellar diaschisis secondary to post-CEA hyperperfusion such as that in our patient is detected more often when SPECT imaging is performed early and repeatedly after CEA.

In conclusion, the present case suggests that cerebral hyperperfusion after CEA causes CCD. The appearance of CCD under the presence of cerebral hyperperfusion may suggest the development of occult hyperperfusion syndrome.

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