

Case Report

Atypical Hyperperfusion Encephalopathy in Post-Carotid Stenting

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Hyperperfusion encephalopathy (HPE) is a condition due to increased perfusion of the brain which is clinically characterized by headache, seizures, and other neurologic signs associated with increased (or not) systemic blood pressures and edema in the subcortical white matter (predominantly in the occipital lobe). Patients with critical carotid stenosis treated with endarterectomy or carotid artery stenting may develop a HPE syndrome of the ipsilateral hemisphere which closely resembles the unilateral HPE and that usually involves the vascular area subjected ipsilaterally to the carotid stenosis. We present here a case of a 62-year-old woman who developed atypical hyperperfusion syndrome after a carotid stenting for high-grade carotid artery stenosis. In our patient, the HPE involved bilaterally both hemispheres, even though the treatment of the carotid stenosis was unilaterally. Some authors have hypothesized that a high dose of contrast, in combination with an unidentified personal vulnerability, may result in the rupture of the blood-brain barrier, carrying the CA into the cerebral parenchyma (both hemispheres), leading to the encephalopathy. The course and prognosis of HPE in post-carotid stenting are excellent with conservative treatment and full recovery usually occurs within 24 to 48 hours.

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During endovascular carotid intervention, brain damage may occur because of hemodynamic or embolic events, acute carotid occlusion, and, much rarely, hyperperfusion encephalopathy (HPE) [1]. HPE is a transient, rare clinical condition in which perfusion to the brain is acutely increased with or without systemic hypertension [1]. Very few cases are reported [2–5] during endovascular carotid intervention.

Here we report a case of a 62-years-old woman who came to our attention because of a history of high blood pressure and transient ischemic attacks in the distribution of the anterior circulation. The ultrasound of cerebral blood vessels revealed an irregular fibrocalcific plaque at the origin of the right internal carotid artery (ICA), causing $\geq 70\%$ stenosis with a peak systolic velocity (PSV) of 290 cm/s and ICA/common carotid artery PSV ratio 3.8 [6]. The Transcranial Doppler revealed an intracranial compensation through flow inversion in the A1 segment of the ipsilateral anterior cerebral artery.

The patient was therefore treated with ticlopidine 250 mg twice a day, and a week later the angiography, performed with

50 mL of iomeprol (300 mg/dL), a nonionic tri-iodinated radiographic contrast agent (CA), confirmed the severity of the stenosis, quantified at 85%, on the basis of the NASCET criterion [7]. During the angiographic session, the stent (Carotid Wallstent Monorail) was positioned at the origin of the right ICA, with the administration of further doses of CA for a total of 200 mL of iomeprol and 5000 U.I. of heparin IV. Soon after the procedure, the patient rapidly worsened, developing mental confusion, headache, mild left hemiparesis, and deviation of the head and eyes to the right. A cranial CT scan without CA performed two hours after the procedure showed swelling of both hemispheres (Figure 1(a)). The duplex ultrasound confirmed the appropriate stent position. Brain MRI limited to diffusion-weighted sequences did not relieve restriction of diffusion of water molecules.

The following day, the patient improved rapidly, and a new CT scan was unchanged. The neurological examination returned to normal. A CT scan of the brain performed five days later showed the resolution of the edema and the normal

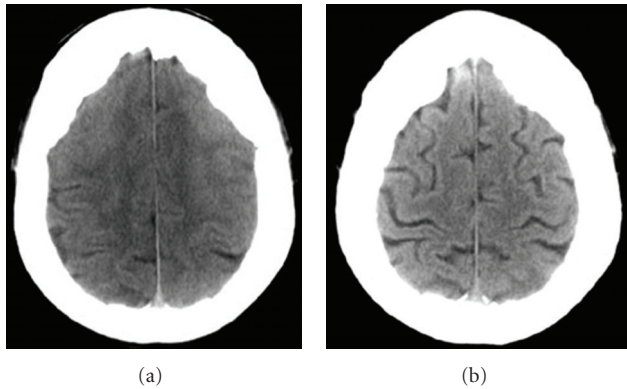


FIGURE 1: (a) A brain CT scan two hours after the clinical onset mental confusion, headache, and mild left hemiparesis following the carotid stenting. There is marked cerebral cortical swelling with disappearance of cortical sulci. (b) A CT scan of the brain one month prior the angiographic examination.

appearance of the cortical sulci (Figure 1(b)). A month after the procedure, the carotid duplex ultrasonography showed normal canalisation, with the stent correctly positioned in the ICA.

HPE is a condition due to increased perfusion of the brain which is clinically characterized by headache, seizures, and other neurologic signs associated with increased (or not) systemic blood pressures and edema in the subcortical white matter (predominantly in the occipital lobe). However, the edema may rarely involve also the cortex. HPE is usually reversible, although infarction or hemorrhage may complicate its natural course [1].

Patients with critical carotid stenosis treated with endarterectomy may develop an HPE syndrome of the ipsilateral hemisphere which closely resembles the unilateral HPE and that usually involves the vascular area subjected ipsilaterally to the carotid stenosis. Cerebral blood flow to the hemisphere in these cases may be chronically reduced, and the intracranial vessels tend to be maximally dilated in order to maintain adequate brain perfusion. Thus, when the carotid stenosis is relieved, breakthrough of autoregulation may occur and therefore cause such syndrome (unilateral HPE in post-endovascular carotid intervention). In our patient, the HPE involved bilaterally both hemispheres, even though the treatment of the carotid stenosis was unilaterally.

Only few report, show bilateral involvement of the hemispheres after interventional procedure with administration of contrast agent [1, 8, 9].

Some authors have hypothesized that a high dose of contrast, in combination with an unidentified personal vulnerability, may result in the rupture of the blood-brain barrier, carrying the CA into the cerebral parenchyma, leading to the encephalopathy and to the secondary neurological and radiological signs [10]. The different osmolarity of the contrast agent may facilitate fluid extravasation and the formation of cerebral edema [4, 10]. However, this theory has some limitations.

Another complication of the contrast agent application is an abnormal brain parenchymal contrast enhancement

(reversible) caused by leakage of the iodinated contrast agent into the extracellular space, with a neuroradiological hyperdensity of the affected hemisphere [3].

The course and prognosis of HPE in post-carotid stenting are excellent with conservative treatment, and full recovery usually occurs within 24 to 48 hours.

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