

letters

Thalamic hemorrhage six days after carotid artery stenting

To the Editor: Hyperperfusion syndrome (HPS) is a serious and potentially fatal complication following carotid artery revascularization procedures. The onset of this syndrome following carotid endarterectomy (CEA) is variable, ranging from immediate up to 4 weeks postoperatively. In carotid artery stenting (CAS), most cases happen immediately or in the early phase after stent implantation, and late onset intracerebral hemorrhage is an unusual presentation.^{1,2} Cerebral blood flow (CBF) and perfusion increase in almost all patients following CEA and CAS, but hyperperfusion is usually defined as a more than 100% increase in CBF compared to the pre-operative baseline.³

There is general belief that autoregulatory failure and cerebral hyperperfusion persists for some time after revascularization, but eventually normalizes.⁴ This syndrome is characterized by symptoms like headache, seizure, confusion, focal neurological signs due to cerebro-

vascular accidents and ipsilateral intracerebral hemorrhage.^{5,6} Some basic mechanisms that could result in ICH after these procedures are: 1) cerebral hyperperfusion syndrome, 2) a preoperative cerebral ischemic event, 3) a cerebral infarction and 4) the use of postoperative anticoagulation therapy.⁷

A 74-year-old male presented with right side hemiparesis and dysarthria one week before admission. These symptoms lasted a few hours and resolved completely. He denied any history of stroke and vascular risk factors included arterial hypertension, high blood cholesterol and smoking. All laboratory tests, including blood and coagulation parameters were normal, and blood pressure on admission was 120/80 mm Hg. A carotid artery Doppler ultrasound scan revealed stenosis of the left internal carotid artery. Catheter angiography confirmed 75% stenosis of the left internal carotid artery according to The North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria with post-stenotic segmental dilatation. The

patient underwent successful carotid artery stenting, which was performed without cerebral protection. Despite unfavorable and difficult aortic arch anatomy (bovine type), the operation went very well without complications. The morphologic result was good, without residual stenosis (**Figure 1**). The patient received aspirin 80 mg and clopidogrel 75 mg daily (which was started 5 days before CAS) plus full anticoagulation during the procedure. After the procedure, the patient was closely observed and his blood pressure was monitored for 2 days. On the third day the patient was neurologically intact and discharged home with clopidogrel 75 mg daily. On the sixth postoperative day, he developed clinical signs of HPS, including headache and right side hemiplegia. A brain CT scan showed intracranial hemorrhage in the left thalamus with some extension to posterior limb of internal capsule (**Figure 2**). The arterial blood pressure of the patient was within normal limits. All coagulation tests including serum fibrinogen were in the normal range.

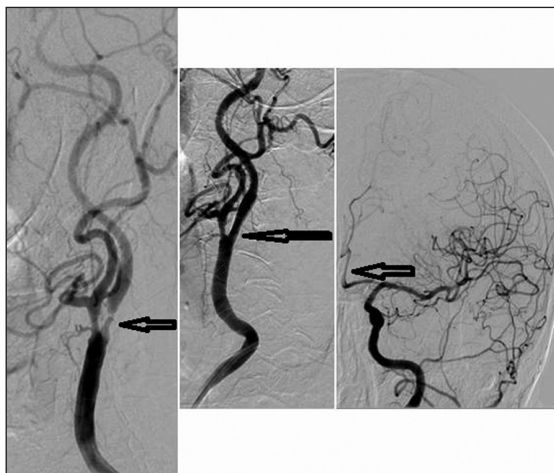


Figure 1. Lateral angiography of the left internal carotid artery, before (left) and after (middle) stenting. Intracranial frontal view showed normal A1 segment (right).



Figure 2. Plain axial CT scan, showed left thalamic hemorrhage with some extension to posterior limb of internal capsule.

The patient was treated conservatively and recovered partially, and at the 2-month follow-up there was mild right limb paresis. If we consider this case HPS, there is an unusual late presentation, six days after carotid artery stenting in a normotensive patient. Although there are claims that severe ipsilateral stenosis, impaired collateral blood flow secondary to an advanced occlusive disease in other extracranial cerebral vessels or an incomplete circle of Willis, peri- and postoperative hypertension are main risk factors for developing HPS and intracerebral hemorrhage after CAS, our case showed that this complication may happen even in a patient without these risk factors and also as a late presentation, six days after intervention.

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