Cerebral hyperperfusion syndrome after carotid stenting
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Section: Interventional radiology
Imaging Technique: Ultrasound-Colour Doppler
Imaging Technique: CT
Imaging Technique: Ultrasound-Power Doppler
Case Type: Clinical Cases
Authors: ZV Milosevic, B Zvan, M Zaletel, M Surlan
Patient: 72 years, female

Clinical History:

Symptomatic patient (minor ischaemic stroke) with ipsilateral 90% stenosis of the internal carotid artery.

Imaging Findings:

The patient was referred in September 2001 after a minor ischaemic stroke. In 1995 she had had an anterior circulation cerebrovascular accident, but she had made a good recovery. Her medical history included long-lasting arterial hypertension for more than 20 years and hyperlipoproteinaemia. There was no history of cigarette smoking or excessive alcohol intake. In spite of regular ACE-inhibitor treatment her blood pressure fluctuated. She had been treated with aspirin 100mg and statin 20mg daily.

When assessed in hospital before carotid angioplasty, she had a residual expressive dysphasia, as well as a mild right facial and limb weakness. Her blood pressure was 180/110mmHg. A brain computed tomography scan (CT) revealed widened liquor spaces and pre-existed old ischaemic lesions up to 1mm in size, located deep in the left cerebral hemisphere, in the left frontal lobe and subcortically in the left parietal lobe. There was no evidence of haemorrhage. All haematological and biochemical tests were normal, with a normal platelet count and coagulation screen. Duplex ultrasonography performed in 2001 revealed a 90% stenosis of the left internal carotid artery (ICA) produced by echolucent plaque type I according to the accepted international classification (Fig. 1).

Discussion:

The patient underwent left carotid stenting through the femoral approach under local anaesthesia. Intra-arterial digital subtraction angiography confirmed 95% stenosis of the left ICA (Fig. 2a). 5000IU of heparin IV was administered. Glycopyrrolate and 0.5mg atropine IV was administered during the procedure. The stenosis was predilated with a low-profile coronary balloon (4 x 20mm Bypass Speedy Monorail Catheter, Boston Scientific Corp) and stented with a 7 x 30 Carotid Wallstent Monorail (Boston Scientific Corp). The stent was dilated with a 5.5 x 20mm Bypass Speedy Monorail Catheter (Boston Scientific Corp) to firmly embed it into the vessel wall. The blood pressure varied between 160/90mmHg and 175/105mmHg during the procedure but there were no residual adverse neurological sequelae.

A postprocedural angiogram showed no significant stenosis or dissection (Fig. 2b). Over the following 24 hours, the patient was treated with aspirin and clopidogrel; her blood pressure varied between 140 to 160/95mmHg and she
was clinically stable. On the following day she was discharged. She did not continue antihypertensive therapy when she was at home, because she decided that she did not need this therapy after carotid stenting.

After 2 days she was urgently re-admitted because of a grand mal type epileptic seizure. After the seizure she had transient right-sided hemiplegia. Her blood pressure at the time of admission was 180/100mmHg. An urgent brain CT revealed a small haemorrhage in the right frontal lobe (Fig. 3). Colour and Power Doppler ultrasound of the ICA revealed a visibly patent vessel (Fig. 4), but the peak systolic velocity was elevated at 2.3m/s, with an end diastolic velocity of 1.2m/s. The patient was managed conservatively. Hypertension was easily controlled with 10mg enalapril twice daily and antiepileptic therapy was introduced. She recovered completely after 2 weeks.

In this case we do not have pathological evidence to support hyperperfusion injury as a cause of the haemorrhage after carotid stenting, but clinical features and postprocedural systemic hypertension, together with the lobar appearance of the haemorrhage support a hyperperfusion mechanism of injury.

Cerebral hyperperfusion syndrome (HS) has been widely reported in the surgical literature as an infrequent complication of carotid endarterectomy (CEA) with an incidence of approximately 0.6%. It may also occur after percutaneous transluminal carotid angioplasty and stenting with a similar causal mechanism and clinical features to those related to CEA. HS is caused by a probable failure of vascular autoregulation. Autoregulatory failure results in the cerebral arterioles being maximally dilated over a long period of time, with subsequent loss of their ability to constrict when normal perfusion pressure is restored. HS manifests as ipsilateral headaches, seizures, or intracerebral haemorrhage. Risk factors such as high grade stenosis, contralateral carotid occlusion, poor collateral flow, chronic ipsilateral hypoperfusion, preoperative and postoperative hypertension, and perioperative use of anticoagulant or antiplatelet agents have been reported. In the patient with neurological symptoms following carotid stenting, it is important to consider cerebral HS as a differential diagnosis to embolic or haemorrhagic stroke since early recognition and meticulous control of blood pressure may prevent progression of cerebral haemorrhage and death. A brain CT scan may be considered to be essential after any atypical complication of angioplasty.

**Differential Diagnosis List:** Cerebral hyperperfusion syndrome after carotid stenting

**Final Diagnosis:** Cerebral hyperperfusion syndrome after carotid stenting

**References:**

Figure 1

Description: Colour Doppler ultrasound shows a 90% stenosis of the left ICA. Origin:
Figure 2

Description: Lateral view of the left carotid artery bifurcation. High grade circumferential, atherosclerotic stenosis of the internal carotid artery origin before carotid stenting, Origin:

Description: No residual stenosis after carotid stenting. Origin:
Figure 3

Description: CT of the brain demonstrates a small haemorrhage in the left frontal region. Origin:
Figure 4

Description: Power Doppler ultrasound of the ICA shows a visibly patent vessel. Origin: