Hyperperfusion Syndrome After Stent Placement for Subclavian Artery Stenosis

—Case Report—

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Abstract

A 60-year-old woman presented with a rare case of hyperperfusion syndrome after stent placement for subclavian artery stenosis manifesting as dizziness due to vertebrobasilar insufficiency. Three days after undergoing stent placement to treat the severely stenotic (90%) right subclavian artery, she suffered intracranial hemorrhage related to hyperperfusion syndrome. Preoperative single-photon emission computed tomography findings of low cerebral perfusion and poor perfusion reserve might indicate the possibility of hyperperfusion syndrome after stenting in patients with subclavian artery stenosis.

Key words: subclavian artery stenosis, hyperperfusion syndrome, stent, perfusion computed tomography, single photon emission computed tomography
Introduction

Hyperperfusion syndrome may occur in patients with carotid artery stenosis treated by endarterectomy or stent placement. This complication is rare, but may be fatal if intracranial hemorrhage develops. Risk factors for hyperperfusion syndrome are diminished cerebrovascular reserve, postoperative hypertension, and hyperperfusion lasting for more than several hours after endarterectomy or stent placement. Only 3 cases of hyperperfusion syndrome after stent placement for subclavian artery stenosis have been reported, but the risk factors remain unclear. We report another case and discuss the risk factors based on cerebral perfusion study.

Case Report

A 60-year-old woman had undergone radiation therapy 16 years earlier for carotid lymphoma. Magnetic resonance (MR) angiography revealed right common carotid artery occlusion (Fig. 1), severe stenosis of the left common carotid artery (80%), and right innominate artery stenosis (90%) attributable to this earlier radiation therapy. However, she had no neurological deficits. Eighteen years after radiation therapy, she reported dizzy spells when standing, and coldness of her right hand. Her blood pressure was 100/78 mmHg in the left arm but could not be measured in the right arm. Although angiography showed severe right subclavian artery stenosis without progression, the neurological sign was recognized. Blood in the right vertebral artery flowed antegradely, and the perfusion pressure was low due to severe stenosis of the innominate artery. Technetium-99m hexamethylpropyleneamine oxime (740 MBq) single photon emission computed tomography (99mTc-HMPAO SPECT) showed decreased cerebral blood flow (CBF) and poor vasoreactivity in the left occipital region after acetazolamide challenge (Fig. 2). CBF in the cerebellum was normal without crossed cerebellar diaschisis. Despite treatment with aspirin, her dizziness due to vertebrobasilar insufficiency worsened and she required treatment to address the innominate artery stenosis. She took aspirin and clopidogrel for the 2 weeks preceding the procedure.

A 5-Fr short introducer was inserted in the right brachial artery and a 5-Fr catheter was passed into the right subclavian artery. A PercuSurge guidewire (Medtronic, Santa Rosa, California, USA) was navigated into the right vertebral artery through the 5-Fr catheter. This vessel was protected from micro-debris during stent deployment at the time of stent placement. An 8-Fr long sheath was inserted into the right external iliac artery. She received an intravenous bolus injection of 5000 U of heparin just before PercuSurge balloon inflation, followed by intravenous infusion of 500–1000 U/hr for the duration of the procedure to ensure that the activated coagulation time was maintained at 2 times the baseline. An 8-Fr guiding catheter was introduced at a proximal site of the innominate artery. After inflation of the PercuSurge balloon, the subclavian stenotic lesion was crossed with a guidewire fed through the 8-Fr guiding catheter (Fig. 3A). Pre-dilation was performed with a balloon catheter for 10 seconds using 8-atm pressure. The stent was then deployed and expanded. The stent was deployed accurately, and no stent malpositioning was observed. The total procedure time was 2 hours.

**Fig. 1** Magnetic resonance angiogram showing occlusion of the right internal carotid artery.

**Fig. 2** Technetium-99m hexamethylpropyleneamine oxime single photon emission computed tomography scans showing an area of hyperperfusion in the left occipital region (A, arrow), and poor vasoreactivity after acetazolamide challenge (B, arrow).

**Fig. 3** A: Pre-treatment angiogram of the right innominate artery showing right common carotid artery occlusion and severe stenosis of the right subclavian artery. B: Post-treatment angiogram of the right innominate artery showing dilation of the stenotic lesion in the right subclavian artery.
deployed to cover the stenotic lesion of the innominate artery but not the orifice of the right vertebral artery to avoid obstruction of flow in the right vertebral artery. Lastly we performed post-dilation with a balloon catheter for 10 seconds using 8-atm pressure. The PercuSurge balloon was deflated after removing debris by suction. Angiography suggested that the stenotic region of the right innominate artery was effectively dilated (Fig. 3B) and that antegrade flow in the right vertebral artery was improved. Left carotid angiography showed normal pre- and post-treatment findings (Fig. 4).

She experienced a short, transient period of hypotension after stent placement. MR imaging performed the day after the procedure showed no cerebral infarction or hemorrhagic complications. Three days after the procedure her blood pressure exceeded 160 mmHg, and she manifested right homonymous hemianopsia and slight confusion on the 4th day. Computed tomography (CT) showed subcortical hemorrhage and fluid-attenuated inversion recovery MR imaging demonstrated hyperintensity in the left occipital lobe (Fig. 5). Perfusion CT revealed increases in CBF and cerebral blood volume (CBV), and shortening of the mean transit time (MTT), indicative of hyperperfusion syndrome (Fig. 6A–C). Her blood pressure was kept under 120 mmHg by the continuous delivery of nicardipine hydrochloride and midazolam sedation. Phenytoin, an anticonvulsant agent, was also administered. Nine days after treatment, her confusion and hemianopsia improved. Perfusion CT showed improved CBF, CBV, and MTT (Fig. 6D–F). She was discharged 3 weeks after treatment with no neurological deficits. Her dizzy spells while standing and coldness of her right hand disappeared, and right and left blood pressure showed no difference.

**Discussion**

Most reported patients with hyperperfusion syndrome had undergone endarterectomy or stenting of the internal carotid artery, or superficial temporal artery-middle cerebral artery anastomosis; all had been treated for carotid artery stenosis. Hyperperfusion syndrome has also occurred after treatment for vertebral artery stenosis. Our review of the literature found only 3 patients with hyperperfusion syndrome after treatment for subclavian artery stenosis. One patient had been treated with a subclavian-carotid artery bypass, the other 2 had undergone endovascular surgery; cerebral perfusion study of hyperperfusion syndrome was not performed. Our patient manifested increases in cortical blood flow and CBV, and shortened MTT. These findings led us to suspect hyperperfusion. After 5 days of treatment with nicardipine hydrochloride, midazolam, and phenytoin, cortical blood flow and CBV were normalized, MTT was increased, and hyperperfusion had abated.
Symptoms of hyperperfusion syndrome are headache, seizures, confusion, focal neurological signs, and intracerebral hemorrhage.\(^{5}\) Hyperperfusion syndrome reflects an increase in CBF.\(^{8}\) Pathological findings are fibrinoid necrosis of the small arteries, intraluminal fibrin deposition, endothelial swelling, and extravasation. Risk factors for hyperperfusion syndrome are preoperative failure of autoregulation and blood-brain barrier disruption.\(^{4}\) Significant risk factors also include patient age, pretreatment cerebral vasoreactivity, and asymmetry index.\(^{6}\)

Our patient manifested areas of hypoperfusion in the left occipital region on \(^{99m}\)Tc-HMPAO SPECT before the procedure, with decreased cerebral vasoreactivity after acetazolamide challenge. We were able to localize the hyperperfusion syndrome to that area. Our findings may represent evidence of severe perfusion disturbance as a result of hyperperfusion syndrome after treatment for subclavian artery stenosis. There was no evidence of hemorrhagic infarction; cerebral infarction was not recognized just after treatment. Based on the present case, we recommend careful blood pressure control to avoid hyperperfusion syndrome in patients undergoing treatment for subclavian artery stenosis.

References


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