中文題目：栓子保護裝置破裂引發栓子性腦中風無殘存神經功能缺損: 病例報告

英文題目：Embolic Stroke from Carotid Embolic Protection Device Tearing without residual neurologic deficits : case report and literature review

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Abstract

In recent years, endovascular treatment of carotid artery stenosis has profited from substantial technical improvements, but the point of discussion still circles around the use of cerebral protection devices.

In this report, we try to address this issue by describing a patient with right carotid artery stenosis undergoing a carotid angioplasty and stenting (CAS) for prevention of ischemic stroke. The embolic protection device (EPD) was torn during the procedure, and the postprocedural period was complicated by embolic ischemic stroke involving the right frontal and parietal lobes, resulting in left hemiparesis and dysarthria. With timely anticoagulation and rehabilitation, the neurologic deficits improved within about 2 weeks.

Introduction

The most feared outcome of carotid atherosclerosis is ischemic stroke. Percutaneous CAS has been proposed as an alternative to CEA for the treatment of patients with carotid atherosclerosis who are considered to be poor candidates for surgery to prevent ischemic stroke. However, ironically enough, the most serious acute complication associated with CAS is stroke. In fact, the risk for stroke following CAS is higher compared with CEA, prompting the development of embolic protection devices (EPDs) in order to lower, at least, the risk of periprocedural stroke.

There are 2 basic types of EPDs. Filter devices are designed to catch debris dislodged during stent placement and are the most commonly used type of EPD. Retrograde flow devices promote flow of blood retrograde away from the internal carotid artery (ICA). Despite the theoretically effective designs of EPDs, their real-world effectiveness remains debated. Moreover, these devices have several disadvantages that could potentially lead to poor outcomes following CAS.

Case Report

A 75-year-old man was admitted to this hospital for CAS.

The patient had a history of diabetes mellitus, hypertension, dyslipidemia, and prior stroke in the right temporal lobe. Approximately 3 weeks before this admission, the patient was evaluated for carotid artery stenosis. Computed tomography angiography (CTA), performed 1 week later, revealed an 80% stenosis located in the right distal common carotid artery (CCA). An echocardiography was unremarkable.

On the day of admission, CAS was performed. Percutaneous access was obtained via the right common femoral artery, and the patient was anticoagulated with heparin. A FilterWire EZ System 190 cm (Bosten Scientific) was deployed following placement of the guide wire and sheath for distal embolic protection. A Wallstent 9 mm x 40 mm (Boston Scientific) was positioned within the right CCA and deployed. A post-stenting angioplasty was performed using a Sterling 5.5 mm x 20 mm (Boston Scientific). Removal of the EPD was difficult, and the EPD was torn during the removal.

The patient was transferred to the intensive care unit (ICU) for frequent blood pressure measurements and neurologic assessment after CAS. The postprocedural period was complicated by fever, altered mental status, and left hemiparesis. The patient became less alert. The muscle strength in the left upper and lower limbs was 2 and 3, respectively. The strength on the right side and the remainder of the examination were normal. Antibiotic therapy with ceftriaxone was initiated. A CT scan of the head showed no intracranial hemorrhage. On the next day, magnetic resonance imaging (MRI) the brain revealed new ischemic lesions in the right frontal and parietal lobes.

A 2-day course of intravenous heparin plus piracetam was administered, with improvements in muscle strength. On the 4th postprocedural day, the patient was transferred to ordinary ward. On the 7th postprocedural day, antibiotic therapy was discontinued. Rehabilitation and hyperbaric oxygen therapy were started. On the 13th postprocedural day, he had another episode of urinary tract infection with fever and was treated with cefuroxime. On the 18th postprocedural day, the patient was discharged with cefixime and dual antiplatelet therapy.

Discussion

The benefit of EPDs has not been definitively established in randomized trials, and the available evidence is conflicting. Subgroup data from 2 randomized, noninferior trials comparing CAS with CEA showed no significant difference in the outcome of stroke

However, in a MRI substudy of the ICSS trial, new ischemic lesions on diffusion-weighted MRI (DWMRI) were more prevalent in patients assigned to CAS with the use of EPDs than those assigned to CAS without cerebral protection. Several case series have concluded that cerebral protection with EPDs is feasible, safe, and effective in protecting the brain from cerebral embolism. Three systemic reviews have found that the periprocedural risk of stroke after CAS is significant lower with the use of an EPD. In uncontrolled studies, it is possible that some or all of the reduction in complication rates attributed to EPDs is due to technical progress, the growing experience of active interventionalists, and the improved periprocedural anticoagulation regimens. In support of this hypothesis, a single-center case series of patients who had CAS without protection noted a significant reduction in the 30-day complication rate through a period of 5 years. Amazingly, the reported incidence of stroke was approximately 3%, which is even comparable to stroke rate after CEA

Skepticism about the effectiveness of EPDs is based not only on the low neurological complication rate without them but also on the technical difficulties related to their use. Filter devices must pass across the stenosis, so predication is often necessary. Also, removal of protection device can be difficult once the carotid stent has been placed and causes additional microembolization. Even in the absence of these technical problems, EPDs do not prevent all emboli from reaching the distal cerebral vessels. In a prospective report, small and asymptomatic ischemic lesions were seen on DWI 48 hours after carotid stent placement in more than 60% of procedures performed with an EPD. It has been postulated that disintegration of macroemboli may occur in the filter and lead to paradoxical increase in the microembolic load.

This case reminds us the importance of evidence-based medicine, particularly in an era where application of newly developed medical technology should be cautious. At first glance, it seems reasonable to apply protection systems. From a procedural point of view, nevertheless, current protection systems may only reduce but certainly do not eliminate plaque embolization. It is surprising that the use of EPDs had been obligatory in some large clinical trials such as the CREST and EVA-3S trials. We must all perceive that our decisions and practices are not guided by strong evidence. Further controlled studies are required to help us refine our proposed interventions.