Crevice sign as an indicator of plaque laceration associated with postoperative severe thromboembolism after carotid artery stenting: a case report

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ABSTRACT

Carotid artery stenting (CAS) is increasingly utilized in patients with carotid artery stenosis. Various intraprocedural and postprocedural complications have been reported in the literature. We present a case of symptomatic major thromboembolism after CAS. The intraprocedural angiogram showed extraordinary slow filling of the contrast medium into the plaque, which we named as “crevice sign.” An 83-year-old man presented repeat right amaurosis fugax for 6 months. The radiological examinations revealed 85% stenosis of the origin of the right internal carotid artery. The patient underwent right CAS. The procedure was performed without any problems; however, the angiogram showed slow filling of contrast medium into the carotid plaque through the stent (crevice sign). Sixty minutes later in the ward, the patient presented sudden onset of left hemiparesis and aphasia. Emergency catheter angiography did not show in-stent thrombus, major artery occlusion, or the crevice sign. Magnetic resonance imaging on the next day revealed wide acute infarction of the right cerebral hemisphere. Physicians should be aware of the intraprocedural crevice sign so that a subsequent catastrophic ischemic event can be prevented.

Keywords: carotid artery stenosis, carotid artery stenting, complication, plaque, thromboembolism

INTRODUCTION

Since the introduction of carotid artery stenting (CAS), the treatment options for carotid stenosis have changed. CAS is increasingly utilized in patients with carotid artery stenosis.1,2) Thromboembolic events, the majority of which occur during the periprocedural period, can frequently complicate CAS procedures and are closely related to plaque manipulation with intraarterial devices, which results in plaque rupture, subsequent superimposed thrombus formation, and embolization of plaque debris.3,4) Disruption of the plaque during manipulation in CAS procedures is more likely to occur in so-called vulnerable plaques with large lipid cores and thin fibrous caps.5)
Here, we report a case of a patient who presented with symptomatic major thromboembolism after CAS. The intraprocedural angiogram showed extraordinary slow filling of the contrast medium into the plaque, which we named as “crevice sign.” To the best of our knowledge, this is the first report of a case with such a radiological findings and clinical course. The possible causes, preventive techniques, and management of patients exhibiting the intraprocedural crevice sign are discussed to provide awareness to physicians so that a subsequent catastrophic ischemic event can be prevented in similar cases.

CASE REPORT

An 83-year-old man presented with repeat right amaurosis fugax once a week for >6 months. There were no other symptoms related to a possible transient ischemic attack. The patient was suffering from laryngeal cancer that was treated with local radiotherapy 12 years earlier. The patient has been medicated with an antihypertensive drug since the age of 70 years. Angiography revealed 85% stenosis of the origin of the right internal carotid artery (ICA) (Fig. 1). Cervical magnetic resonance imaging (MRI) did not show vulnerable plaque (Fig. 2). The patient was placed on dual antiplatelet therapy with clopidogrel (75 mg daily) and aspirin (100 mg daily) for 14 days before the CAS procedure. The patient underwent right CAS under local anesthesia.

At first, an 8-Fr short sheath was introduced into the right femoral artery. Intravenous heparin was then administered to achieve an activated clotting time of ≥250 s. An 8-Fr Optimo balloon guiding catheter (TOKAI Medical, Aichi, Japan) was advanced into the right common carotid artery. Under temporary occlusion of the right common carotid artery by the Optimo, a Guardwire PercuSurge (200 cm; Medtronic, Minneapolis, MN, USA) was cautiously navigated.
into the distal ICA through the stenotic lesion. Then, the distal balloon of the Guardwire was also inflated for embolic protection. Next, atropine sulfate (0.5 mg) was administered, and a pre-stenting balloon (3.5 × 40-mm angioplasty balloon) was inflated at the stenotic lesion. A Carotid WALLSTENT (Boston Scientific, Natick, MA, USA) was then deployed. Post-stenting dilatation was performed at the narrowest area using a 4.5 × 30-mm angioplasty balloon until nominal pressure was achieved because the distal ICA was 4.9 mm in diameter. There were a few tiny
Fig. 4 Second right carotid angiogram showing no crevice sign or in-stent thrombus (A) and no major intracranial artery occlusion (B).

Fig. 5 Magnetic resonance images showing wide acute ischemic lesions on the right cerebral hemisphere (A, B, and C), and no major arterial occlusion (D).
Crevice sign during carotid artery stenting

Pieces of yellow debris in the forced aspirated blood before release of the protective balloons. The maximum ICA occlusion time was 6 min 20 s. The procedure was performed without any difficulties; however, the final carotid angiogram showed slow filling of contrast medium into the carotid plaque through the stent, called the crevice sign. (Fig. 3). Intracranial angiography confirmed that there was no major arterial occlusion. Because the patient was conscious and showed no neurological deterioration, the procedure was completed.

Sixty minutes later in the ward, the patient presented sudden onset of left hemiparesis and aphasia. Emergency catheter angiography did not show in-stent thrombus, major artery occlusion, and the crevice sign (Fig. 4). MRI performed on the next day revealed wide acute infarction of the right cerebral hemisphere (Fig. 5). The patient was administered intravenous argatroban and edaravone. The patient had a modified Rankin Scale score of 4 on postoperative day 32 and was transferred to a rehabilitation facility.

DISCUSSION

Some authors have reported that carotid plaque composition changes with age and is independent of symptomatic presentation, cardiovascular risk factors, and medication use. Van Lammeren et al.6) reported that age-related changes in carotid plaque stability from the examination of 1,385 plaques from patients who had undergone carotid endarterectomy. Carotid plaques from elderly patients have relatively unstable characteristics, such as large lipid cores, decreased smooth muscle cell content, and heavy calcifications. These plaque characteristics constitute a high-risk for periprocedural stroke and inadequate stent expansion.7-8) Consequently, these findings in our case should have indicated the need to give special care to the 83-year-old patient.

We misunderstood preoperatively that the patient’s carotid plaque was not extremely vulnerable. Generally, radiation-induced carotid plaques do not appear vulnerable. Some authors have reported CAS for radiation therapy-associated carotid stenosis may be performed safely. Sadek M et al. reported that, compared with non-radiation cases, the durability of CAS and the characteristics of captured embolic particles were not altered by a history of neck radiation therapy.9) Ravin RA et al. reported that despite increased embolic particles size in cases with neck radiation therapy, CAS could be performed safely with no increased morbidity.10) Conversely, Sano N et al.11) reported that their radiologic and histologic analyses revealed that plaques of radiation-induced stenosis were often accompanied by the formation of vulnerable plaque. They concluded that carotid endarterectomy could prevent complications in such cases. Although the plaque images of MRI did not appear to be unstable in our patient, the plaque was lacerated by the undersized angioplasty balloon. Further accumulation of similar cases may explain the discrepancy in previously reported findings.

In our case, the crevice sign indicated major plaque laceration. The carotid angiogram showed slow filling of contrast medium into the carotid plaque through the stent mesh. There was no intraprocedural in-stent thrombus or major arterial occlusion. Because the patient showed no neurological deterioration, we had completed the procedure normally. However, our experience in this case led us to conclude that the procedure should not be completed in future cases if the crevice sign is observed.

We think overlap stenting is one of the best alternatives in these cases. Nishihori et al.12) reported the feasibility and safety of overlap stenting for in-stent restenosis after CAS. We believe emergency additional stenting can resolve the problem of plaque disruption. Overlooking the implications of the crevice sign can potentially lead to a catastrophic ischemic complication immediately after the procedure. In our case, because the second angiogram did not show the
crevice sign, we think that a significant amount of debris from tiny thrombi may have migrated during the process of healing after plaque laceration. We believe that high amount of debris was migrated into the right hemisphere because there was no major artery occlusion. Although hemodynamic infarction was suspected according to the diffusion-weighted images, it was excluded because the patient was tolerated against the carotid artery occlusion during the procedure.

CONCLUSIONS

The intraprocedural angiogram showed extraordinarily slow filling of the contrast medium into the plaque, a finding that was named crevice sign. To the best of our knowledge, this is the first report of a case with such radiological findings and clinical course. We believe emergency overlap stenting can resolve the problem of plaque disruption in similar cases. Physicians should be aware of the implications of crevice sign so that they can take steps to prevent a subsequent catastrophic ischemic event.

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