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Case Report

Therapeutic strategy of severe circular calcified carotid plaque with hemodynamic impairment: A patient treated by carotid endarterectomy following balloon angioplasty to prevent hyperperfusion

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ABSTRACT

Background: Cerebral hyperperfusion syndrome (HPS) is a serious complication. Recently, staged angioplasty has been reported as an effective strategy to avoid HPS. Severe calcification has been reported as contraindication of carotid artery stenting (CAS). In these cases, carotid endarterectomy (CEA) might be an alternative second stage treatment. We present a case of severe circular calcified plaque with hemodynamic impairments, treated with CEA following percutaneous transluminal angioplasty (PTA) to prevent HPS.

Case Description: A 77-year-old woman presented with severe stenosis at the proximal left internal carotid artery. A CT scan of the neck demonstrated circular calcification. ^{123}I-iodoamphetamine single-photon emission computed tomography (^{123}I-IMP SPECT) showed reductions in cerebral blood flow (CBF) and cerebral vascular reserve in the left hemisphere. Staged therapy was subsequently performed as this patient had a high risk of HPS after conventional CAS or CEA. In the first stage, PTA was performed under local anesthesia. Two days after the procedure, ^{123}I-IMP SPECT revealed improvements in CBF. There were no neurological morbidities. CEA was then performed under general anesthesia 7 days later, for the second stage. We found a calcified plaque with a large thrombus at its proximal end. A hematoxylin-eosin stain of the thrombus showed mostly intact and partially lytic blood cells. Postoperative ^{123}I-IMP SPECT revealed CBF was improved, with no hyperperfusion immediately and 2 days after CEA. The patient was discharged with no neurological deficits.

Conclusion: CEA following PTA for severe circular calcified plaque can be an alternative treatment strategy to prevent HPS. A disadvantage is the formation of thrombi. Early CEA should be considered if thrombus formation is suspected.

Keywords: Calcified plaque, Carotid endarterectomy, Hyperperfusion, Staged angioplasty

INTRODUCTION

Cerebral hyperperfusion syndrome (HPS) is a serious complication of carotid endarterectomy (CEA) or carotid artery stenting (CAS) and can lead to mortality or severe morbidities such as headache, psychological symptoms, focal neurologic deficits, epilepsy, and intracerebral hemorrhage.[7,8,11] Recently, staged angioplasty has been reported as an effective strategy to avoid

HPS.[4,6] Staged angioplasty is a two-stage treatment that is composed of percutaneous transluminal angioplasty (PTA) in the first stage, and CAS in the second stage.[16] The interval between the first and second stages has been reported as 2 or 4 weeks;[15] however, the optimal interval has still not been found. Several factors have been reported as contraindication of CAS, one of which is severe calcification.[1,9] In these cases, CEA might be an alternative second stage treatment. We present a case of severe circular calcified carotid plaque with hemodynamic impairments, treated with CEA following PTA to prevent hyperperfusion.

CASE PRESENTATION

A 77-year-old woman presented to our hospital with a history of hypertension, hyperlipidemia, and diabetic mellitus. She also had occlusion of the left central retinal artery due to ipsilateral carotid stenosis 3 years prior, after which no further ischemic events had occurred. Neck MRA demonstrated severe stenosis of the proximal left internal carotid artery (ICA), and the external carotid artery was not detected [Figure 1a]. Intracranial MRA showed weak flow signal intensity of the left ICA and left middle cerebral artery [Figures 1b and c], and neck computed tomography revealed severe circular calcification [Figure 1d]. ^{123}I-iodoamphetamine single-photon emission computed tomography (^{123}I-IMP SPECT), additionally, showed reduced CBF and cerebral vascular reserve [Figures 1e and f] in response to acetazolamide in the left hemisphere.

Staged therapy was subsequently performed, as this patient had a high risk of HPS following conventional CAS or CEA. Dual antiplatelet therapy (DAPT) (aspirin 100 mg and clopidogrel 75 mg) was administrated 2 weeks before PTA. The procedure was performed under local anesthesia. After intravenous administration of 5000 U heparin, the lesion was crossed with a 0.014 inch guide wire, and submaximal angioplasty was performed using a 2.5 mm × 12 mm noncompliant Gateway® balloon (Stryker Neurovascular, Kalamazoo, Michigan). The stenotic lesion was sufficiently expanded immediately; however, elastic recoil was observed ten minutes later. PTA was then performed using a 3.0 mm × 12 mm Gateway® balloon. After expanding the stenotic lesion, we observed that flow delay was improved [Figures 2a-c]. Two days after first stage PTA, ^{123}I-IMP SPECT revealed improvements in CBF [Figure 2d]. In addition, no neurological morbidity was observed during the interval between the first and second stages.

In the second stage, CEA was conducted under general anesthesia 7 days after the first stage, using real-time near-infrared spectroscopy (NIRS) monitoring. The carotid sheath was dissected, and the arteriotomy was completed. Furui's double balloon shunt system was used to maintain blood flow from the common carotid artery to the ICA. When the ICA

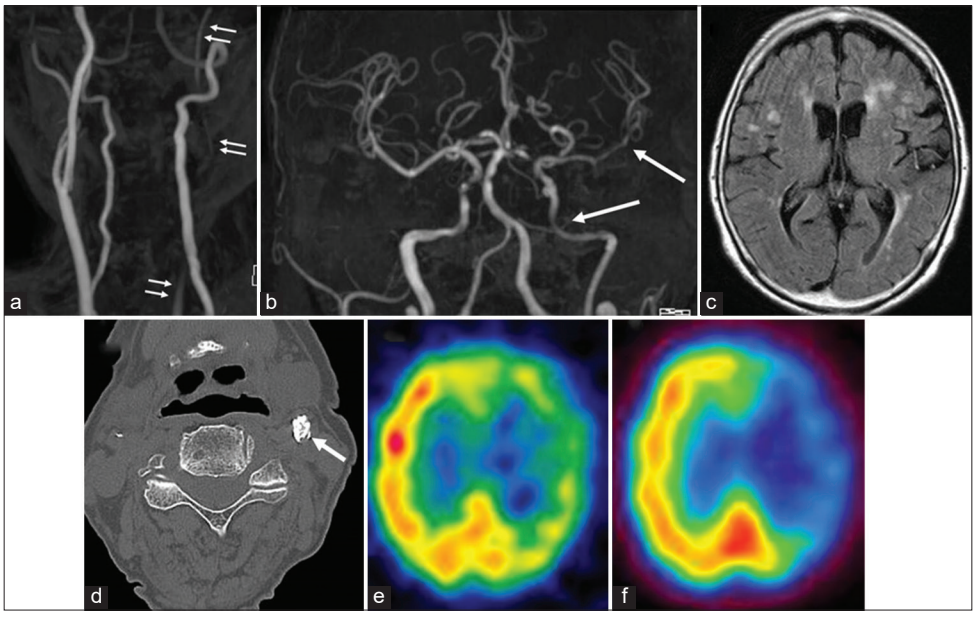

Figure 1: (a) Neck MRA revealing severe stenosis of the proximal left internal carotid artery (ICA) (arrow), and undetected external carotid artery. (b) Intracranial MRA displaying weak flow signal intensity of the left ICA and left middle cerebral artery (MCA) (arrow). (c) MRI FLAIR showing hyperintense vessel sign in the left MCA. (d) Neck noncontrast CT demonstrating severe circular calcification (arrow). (e and f) 123I-iodoamphetamine single-photon emission computed tomography showing reductions in cerebral blood flow (CBF) and (f) CBF to acetazolamide in the left hemisphere.

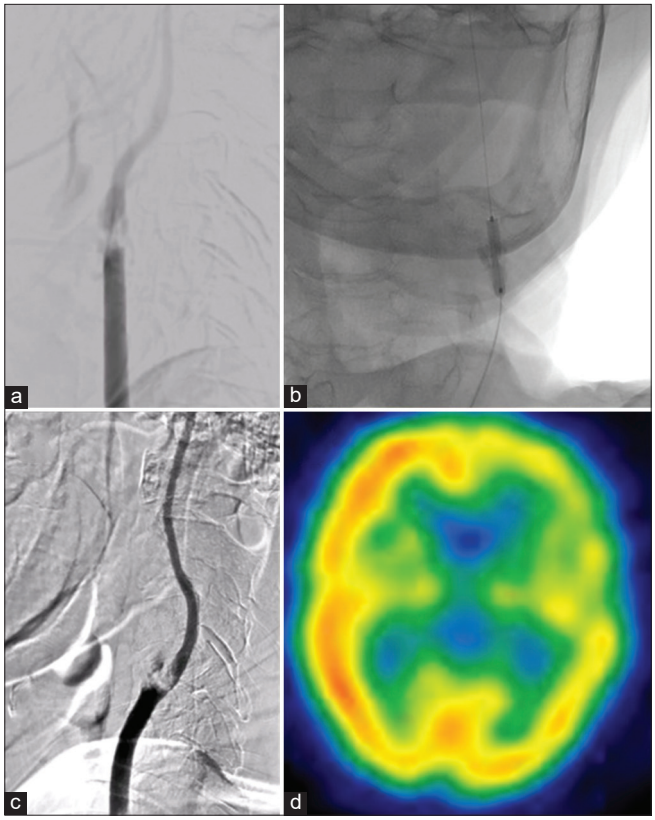

Figure 2: (a) Left common carotid artery angiography before first-stage percutaneous transluminal angioplasty (PTA) showing slow flow in the internal carotid artery (ICA), and occlusion of the left external carotid artery. (b) PTA being performed. (c) Improved flow within the ICA. (d) Improvements in the left hemisphere cerebral blood flow through ^{123}I-iodoamphetamine single-photon emission computed tomography, but with impairments still detected.

was clamped, left cerebral hemisphere NIRS dropped by 10%. However, once reperfusion to ICA was delivered through the shunt system, NIRS returned to baseline. Further, we found a calcified plaque with a large thrombus at its proximal end [Figure 3a]. The plaque was, then, extracted, and the ICA was closed. The skin incision was then closed. Intracranial and neck MRA showed normal flow signals [Figures 3b and c]. CBF was improved, and hyperperfusion was not detected in postoperative ^{123}I-IMP SPECT immediately and 2 days after CEA [Figure 3d]. The postoperative course was uneventful, and the patient was, then, discharged with no neurological deficits. She continued to receive DAPT until 6 months after completing CEA.

Histopathology

The extracted thrombus was found to consist of calcified plaque. A hematoxylin-eosin stain of the thrombus showed mostly intact and partially lytic blood cells, without necrotic tissue [Figure 3e]. Therefore, the thrombus was assumed to be between 1 and 5 days old, as per previous reports.[10,13] We, therefore, found that the thrombus was formed after the first therapeutic stage.

DISCUSSION

In this report, we present a patient with severe circular calcified carotid plaque accompanied by hemodynamic impairments, which were treated with CEA following PTA. This could be an alternative strategy to treat carotid stenosis and prevent HPS. In addition, in this case, thrombus formation occurred following first stage PTA. We have, further, discussed the role and the advantages and disadvantages of staged angioplasty for severe calcified plaques.

Staged revascularization

Conventional staged angioplasty is a two-stage treatment, composing of PTA in the first stage and CAS in the second stage.[6,15] In the present case, our patient was found to have severe circular calcified plaque as a contraindication of CAS. This can lead to dissection with vessel perforation or rupture,[12] stent fracture,[1] underexpansion,[2] apposition of stent,[2] and in-stent restenosis.[14] However, in patients with CAS complications, CEA could be an alternative second-stage treatment. However, a disadvantage of this strategy is acute occlusion or restenosis after the first stage. Following endothelial damage and platelet activation, there is a release of factors and adhesion molecules which affect smooth muscle cell proliferation. These factors lead to thrombus formation. In our case, thrombus formation occurred following the first PTA despite administering DAPT. Therefore, after completing the first stage of PTA, imaging such as ultrasound, MRA, or CT angiography should be performed routinely to detect thrombus formation. Early CEA should then be performed if thrombus is detected.

Although the risk and benefit should be consider in each patient, we believe staged revascularization with CEA following PTA could bring more benefit rather than conventional CEA or CAS. Uchida *et al*. reported 43 patients treated with staged angioplasty (CAS following PTA).[15] In their series, only one patient had morbidity worsened their mRS. Further, no patients treated by staged angioplasty experienced hyperperfusion symptoms. These results supported the benefit of staged revascularization with CEA following PTA. In previously, one case was reported the patient treated with CEA following PTA.[4] They successfully treated symptomatic carotid stenosis and hyperperfusion was prevented. In their case, unstable plaque was the reason they performed CEA rather than CAS. In any reason for high risk for CAS, CEA can be alternative treatment method of staged therapy following PTA.

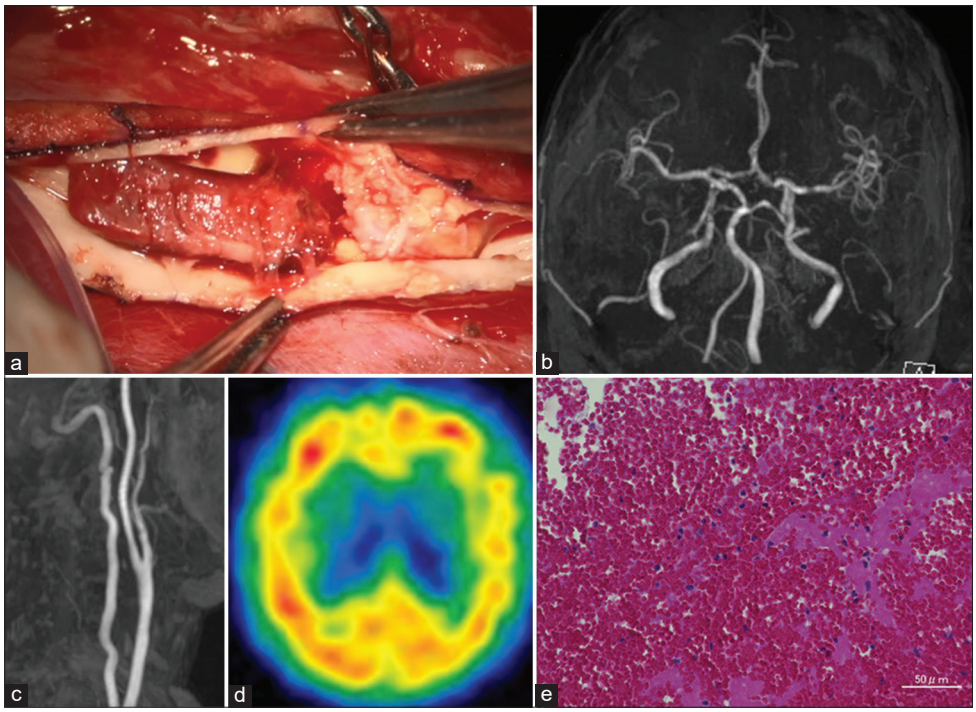

Figure 3: (a) Operative view of carotid endarterectomy (CEA) showing calcified plaque and thrombus formation proximal to the plaque. (b and c) Improvements in intracranial and neck MRA following CEA. (d) ^{123}I-iodoamphetamine single-photon emission computed tomography 2 days after CEA showing normalized cerebral blood flow. (e) H-E stain of thrombosis revealing mostly intact and partially lytic blood cells, without necrotic tissue.

In our case, first PTA was performed without distal protection, because lumen was too narrow and filter device or protection balloon was not reach to distal of lesion. It is safer using protection device to prevent distal embolism, if filter device or protection balloon can be navigated. In some cases, PTA for circumferential calcified lesions may failed to dilate the lesion. In these cases, CEA should be performed, followed by postoperative hypotensive therapy. If HPS is observed, general anesthesia should be used to prevent the following HPS.

Hyperperfusion and the role of first stage PTA

Impaired cerebral autoregulation is a known mechanism of hyperperfusion. With concomitant single-stage CEA/CAS, CBF over the under-perfused area would drastically increase. Normal cerebral autoregulation constricts the cerebral artery in response to a sudden increase in blood flow, to maintain normal cerebral perfusion. Patients with impaired hemodynamic function additionally cannot maintain a stable CBF through vasoconstriction in response to a sudden increase in cerebral perfusion pressure after revascularization.[5,8,11] Derdeyn *et al*. have reported the clinical implications of cerebral blood volume (CBV) parameters in patients with ICA occlusion; they suggested that an oxygen extraction fraction (OEF) increases in the area of an ICA occlusion which often occurs in the absence of CBV elevation. Patients with both increased OEF and CBV are more profound brain ischemia than those with increased OEF and normal CBV.[3] To this end, these patients had higher risk of developing hyperperfusion. Therefore, the main role of initial PTA in a staged angioplasty would be CBV decrease by dilatation of stenosis. Further, studies should be conducted to establish methods of evaluating CBV decrease during PTA.

CONCLUSION

CEA following PTA for severe circular calcified plaque can be an alternative treatment strategy to prevent HPS However, the optimal interval period between the initial PTA and CEA is unclear. A disadvantage of this staged procedure is the formation of thrombi. Early CEA should be considered if thrombus formation is suspected.

Declaration of patient consent

Institutional Review Board (IRB) permission obtained for the study.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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