CASE REPORT

Infected symptomatic carotid artery atheroma concurrent with bacterial endocarditis

Yamaan S Saadeh,¹ Luis E Savastano,¹ Julian L Gendreau,² Aditya S Pandey¹

¹Department of Neurosurgery, University of Michigan, Ann Arbor, Michigan, USA

²Mercer University, Macon, Georgia, USA

Correspondence to

Dr Aditya S Pandey, adityap@med.umich.edu

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SUMMARY

Optimal management of patients with stroke due to symptomatic carotid artery disease coexistent with bacterial endocarditis is still not well established. We report the case of a patient who presented with multifocal left middle cerebral artery stroke in the setting of *Enterococcus faecalis* endocarditis and was found to have near-occlusive ipsilateral carotid artery stenosis in stroke workup. Carotid artery endarterectomy was performed, and atheroma material demonstrated complicated plaque with cultures positive for *E. faecalis*. This report demonstrates that patients with cardioembolic disorders such as bacterial endocarditis with vegetations who present with stroke may benefit from evaluation for extracranial vessel stenosis. Also, additional consideration should be given to the possibility of infected atheroma in patients with symptomatic carotid stenosis with recent or active endocarditis or bacteraemia.

BACKGROUND

There is limited information on the management of symptomatic carotid artery disease that occurs due to infected carotid atheroma. Although the process of atheroma formation has been shown to be related to multiple genetic and environmental factors, there is increasing evidence that micro-organisms play a significant role in atheroma formation and progression, suggesting that there may be an infectious component contributing to carotid artery disease. In this clinical setting, there was evidence of active infection within the carotid plaque, which was suspected to have precipitated the patient's multiple strokes. We review the literature and discuss risks and benefits of carotid artery stenting versus endarterectomy for management of symptomatic carotid artery disease in the setting of bacterial endocarditis and presumed infected atheroma.

CASE PRESENTATION

History and imaging

A 66-year-old right-handed man with a history of aortic stenosis, chronic obstructive pulmonary disease, diabetes mellitus, atrial fibrillation on anti-coagulation and recent urosepsis with blood cultures growing *Enterococcus faecalis* presented with a 1 cm left frontal intraparenchymal haematoma (figure 1A). Transoesophageal echocardiogram showed aortic valve endocarditis with perivalvular abscess, and the patient was started on broad-spectrum antibiotics. MRI showed evidence of multiple

foci of diffusion restriction in the territory of the left middle cerebral artery (MCA) consistent with multiple ischaemic strokes (figure 1B). Further workup via CT angiography revealed severe occlusive disease of the left internal carotid artery (ICA) origin with 99% stenosis and imaging consistent with intraluminal thrombus (figure 1C). No mycotic aneurysm was identified. Cerebral angiography was performed, which revealed a string sign of the left ICA with >90% stenosis (figure 1D).

Procedure

Carotid endarterectomy (CEA) was recommended rather than stenting secondary to the fact that the ICA harboured thrombus and that the patient would require aortic valve replacement (thus, aspirin and clopidogrel would need to be discontinued). An awake CEA was performed with primary closure. Extensive fibrinous adhesions were encountered when the carotid sheath was opened and the ICA and common carotid artery were dissected free of soft tissue. In addition, the plaque appeared soft and friable with a yellow discolouration. A shunt was not used during the CEA as the patient remained asymptomatic. Closure was performed primarily without a patch given the patient's recent history of bacteraemia and active enterococcal endocarditis. The patient remained neurologically stable post-procedure.

Pathology

Given the history of endocarditis and the unusual degree of adhesions encountered during carotid exposure in the virgin neck, the plaque material was sent to pathology and microbiology for cultures. Pathology revealed a complicated plaque with associated intraluminal thrombus (figure 1E), and microbiology grew *E. faecalis* (same species and sensitivities as pre-existent blood cultures). No abscess was identified in the tissues analysed by histology (figure 1F).

OUTCOME AND FOLLOW-UP

Antibiotic therapy was continued postoperatively. Fourteen days post-CEA, the patient underwent mitral valve repair and aortic root replacement with debridement of an aortic root abscess. The procedure was without complication. The patient was discharged to a subacute rehabilitation facility with a plan for administration of 6 weeks of intravenous penicillin.

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Figure 1 (A) Non-contrast head CT revealed a 1 cm left frontal intraparenchymal haematoma. (B) Brain MRI showed multiple areas of diffusion restriction in the territory of the left MCA, consistent with embolic ischaemic strokes. (C) CT angiography revealed severe occlusive disease of the left internal carotid artery (ICA) origin with >90% stenosis and imaging consistent with intraluminal thrombus. (D) Cerebral angiography revealed a string sign (arrow) of the left ICA. (E) At surgery, a soft and friable plaque with intraluminal thrombus was removed. (F) Pathology demonstrated a complicated lesion with an intraluminal thrombus associated with the necrotic core.

DISCUSSION

The formation of atherosclerosis is thought to be multifactorial, comprising a combination of environmental and genetic factors.¹⁻⁴ In addition, reports of infectious agents as causative agents of atherosclerosis have been postulated.^{5,6} It is thought that the shoulder and cap of carotid plaque undergo tissue remodelling secondary to the expression of metalloproteinases as well as tissue factor.⁶ The presence of metalloproteinases and tissue factor have been associated with infectious agents. This is supported by the Bruneck Study, which revealed that those with chronic bacterial infections had a fourfold higher risk of developing carotid atherosclerosis versus those without chronic bacterial infections.⁷ In fact, Chiu reported the presence of *Porphyromonas*, *Streptococcus* and *Chlamydia* species in carotid plaques.⁸ Nonetheless, to our knowledge, ischaemic stroke due to complicated and infected carotid plaque concurrent with bacterial endocarditis has not been reported in the English literature. Here, we present an elderly patient with *E. faecalis* endocarditis and perivalvular abscess on antibiotic therapy who was found to have ischaemic strokes with haemorrhagic conversion in the setting of near-occlusive ipsilateral carotid stenosis. CEA was performed and atheroma material sent to pathology demonstrated complicated plaque with cultures positive for *E. faecalis*. In this regard, surgical management of the carotid artery was recommended, given the high degree of stenosis due to advanced plaque with associated intraluminal thrombi in the setting of multifocal ischaemic embolic strokes. Although the patient did have acute bacterial endocarditis, and therefore a cardiogenic source of emboli could be postulated, the totality of the small ischaemic strokes (>10 foci) were in the left MCA territory, and the ipsilateral carotid was subocclusive with 99% stenosis. It is also possible that a single large septic vegetation embolised from the heart and became wedged into a region of pre-existing

cervical ICA stenosis, subsequently showering small emboli into the ipsilateral anterior circulation. However, pathological analysis of the sample demonstrated a ruptured atheroma, making this theory less likely.

Regardless of the mechanism, surgical management of symptomatic carotid disease concurrent with bloodstream infections and bacterial endocarditis merits discussion. Typically, management of symptomatic high-grade carotid stenosis involves either placement of a carotid stent or performance of a CEA, depending on factors such as age, prior neck procedures or radiation, anatomical location of the carotid bifurcation, recurrent stenosis and patient preference. CEA is usually preferred, given the lower risk of future stroke, as revealed by randomised trials.^{9,10} In the setting of symptomatic carotid stenosis with recent or active endocarditis or bacteraemia, additional consideration should be given to the possibility of infected atheroma, as demonstrated in this case. Placement of a foreign body over a potentially infected plaque, such as a stent during carotid angioplasty or a patch graft during CEA, may result in prolonging a potential source of bacteraemia and possibly result in significant postoperative complications such as infected graft or septic embolic strokes.

In the context of an infected vessel, CEA is likely more desirable as placement of a stent increases the probability of persistent infection within the vessel wall as well as potential thrombosis with the latter option. In addition, most patients with endocarditis will require valvular replacement and, thus, a stented patient will be placed at higher risk of vessel thrombosis as aspirin and clopidogrel will be discontinued during the perioperative period.¹¹ Nonetheless, if CEA is performed in the setting of bacterial endocarditis and/or concern for infected atheroma, primary arterial closure without a patch graft is recommended, as this foreign body could become infected from local infection or haematogenous seeding leading to patch graft blowout and massive haemorrhage, as well as sepsis, abscess formation or stroke.^{12,13}

Learning points

- Patients with endocarditis who present with evidence of stroke may benefit from evaluation for extracranial vessel stenosis.
- Patients with bacterial endocarditis who require intervention for symptomatic carotid stenosis may be at higher risk for complications with stent placement or endarterectomy with patch angioplasty.
- Endarterectomy without patch should be considered, if possible.

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