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【 CASE REPORT 】

Endovascular Coil Embolization for Recurrent Bow Hunter's Stroke

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Abstract:
Bow hunter's stroke is a rare cause of vertebrobasilar infarction. There is no consensus regarding the optimal treatment. We herein report a case of bow hunter's stroke successfully treated by endovascular treatment. A 70-year-old man presented with central vertigo. Magnetic resonance imaging (MRI) showed posterior circulation infarcts. Dynamic angiography revealed thrombus formation and hypoperfusion of the right vertebral artery upon head rotation to the left. Endovascular parent artery occlusion of the right vertebral artery was performed, and there was no recurrence at follow-up MRI. Endovascular parent artery occlusion might be a useful treatment for bow hunter's stroke.

Key words: artery-to-artery occlusion, bow hunter's stroke, coil embolization, parent artery occlusion, vertebrobasilar infarction

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Introduction

Vertebral artery (VA) occlusion upon head rotation is a rare cause of infarction in the posterior cerebral circulation. This mechanism is known as bow hunter's stroke (1) and is generally associated with hemodynamic changes. Recently, artery-to-artery (A-to-A) embolism has been suggested as another cause of bow hunter's stroke (2), but the mechanism underlying bow hunter's stroke remains controversial.

Conservative management, surgery, and endovascular therapy have been reported as treatment options. However, there is no consensus on treatment strategy because of the rarity of the disease (3).

We herein report a patient with bow hunter's stroke who presented with repeated A-to-A embolism and was successfully treated by endovascular coil embolization.

Case Report

A 70-year-old man presented to the emergency room with central vertigo without any inducement. Magnetic resonance imaging (MRI) revealed an acute infarction in the territory of the right anterior inferior cerebellar artery (AICA) (Figure a). Magnetic resonance angiography demonstrated right AICA occlusion and VA fenestration on the left side. Laboratory tests, Holter electrocardiography monitoring, and transthoracic echocardiography were performed. There was no evidence of cardiac embolism or other embolic sources. In addition, there were no risk factors for atherosclerosis, smoking, hypertension, hyperlipidemia, diabetes, or obesity. He had no significant family history. He had a medical history included laminectomy for C5 level cervical spondylosis, but there were no neurological sequelae. Because no apparent cause of infarction was identified, we attributed the infarction to embolic stroke of an undetermined source.

Medical treatment with antiplatelet therapy was first pro-

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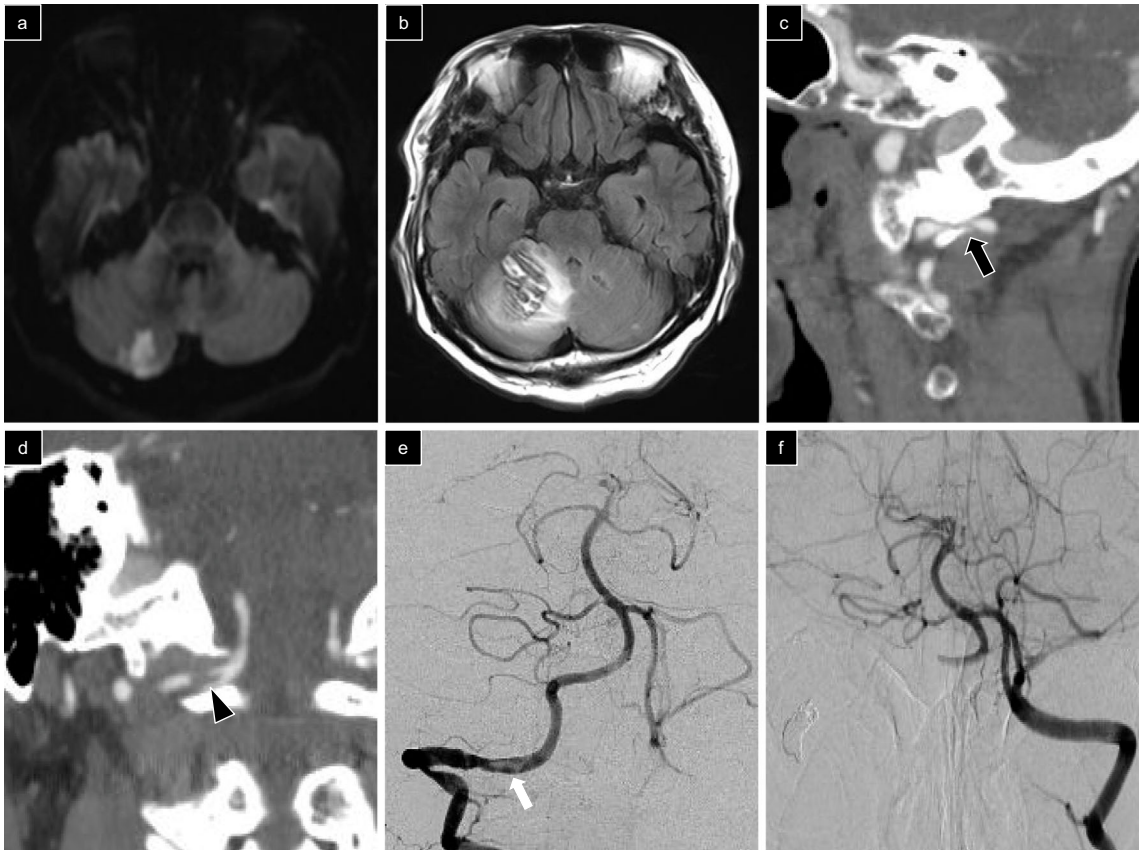

Figure. **Neuroradiological findings. a) Diffusion-weighted imaging of the initial infarct reveals a high-intensity region in the territory of the right anterior inferior cerebellar artery. b) Fluid-attenuated inversion recovery imaging of the recurrent infarct reveals a high-intensity region in the territory of the right superior cerebellar artery with hemorrhagic transformation and chronic left cerebellar infarctions. c) Sagittal computed tomography angiography showing right vertebral artery stenosis compressed by an osteophyte of the atlantooccipital joint (arrow). d) Coronal computed tomography angiography showing a filling defect indicating a thrombus distal to the stenosis of the right vertebral artery (arrowhead). e) Digital subtraction angiography of the right vertebral artery with the head rotated to the left shows hypoperfusion. Thrombotic translucencies are observed at the right vertebral artery (white arrow). f) Digital subtraction angiography of the left vertebral artery after coil embolization. The right vertebral artery has been occluded with coil embolization proximal to the thrombus.**

posed. He was admitted to our hospital two months after the initial presentation due to dysesthesia of his left arm. At that time, a small infarct in the left cerebellar hemisphere and the right temporal lobe was apparent on MRI. We changed the medical treatment to another antiplatelet agent. Approximately four months after the second stroke presentation, he presented with a recurrent infarct in the right temporal lobe after receiving a neck massage. The day after admission, he developed dysarthria and dysmetria. Follow-up MRI revealed a new infarction with hemorrhagic transformation in the right cerebellar hemisphere (Figure b). Computed tomography (CT) and CT angiography revealed abnormalities in the cervical spine and right VA stenosis due to compression by an osteophyte of the atlantooccipital joint (Figure c, d). Duplex color-coded ultrasonography did not reveal any abnormalities in the neutral position. However, there was no end-diastolic flow in the right VA upon head rotation to the left, unaccompanied by symptoms. Dynamic angiography depicted hypoperfusion of right VA with head rotation to the left (Figure e). Furthermore, a thrombotic translucency indicating a thrombus was observed distal to the stenotic site (Figure e). No other vascular abnormalities, including dissection, were evident.

We attributed the cause of stroke to an embolic mechanism through thrombus formation from the arterial wall injury at the stenosed site. In this case, we assumed that the risk of stroke recurrence was high due to the presence of a right VA thrombus. Although treatment was warranted immediately, surgical decompression and fusion were difficult because he was being treated with dual antiplatelet agents. After a thorough discussion of all treatment options, endovascular coil embolization of the right VA was performed (Figure f). Before performing coil embolization, we checked the efficiency of the collateral circulation by endovascular

balloon occlusion in the awake state. After endovascular treatment, we changed the medical treatment to apixaban for secondary stroke prevention. Furthermore, we instructed the patient to avoid prolonged head rotation. The postoperative course was uneventful. At two-month follow-up MRI, he had no stroke recurrence.

Discussion

We clarified two important clinical issues in this case. First, bow hunter's stroke can be caused not only by hemodynamic changes but also embolic mechanisms. Second, endovascular approaches are useful for treating bow hunter's stroke, especially when the patient is using antithrombotic agents.

Bow hunter's stroke is a symptomatic vertebrobasilar insufficiency resulting from mechanical occlusion or stenosis of the VA during head rotation. Recently, non-hemodynamic mechanisms have been suggested as another cause of bow hunter's stroke (2). However, the specific mechanisms remain unclear. Kawasaki et al. reported that repeated intimal injury and vascular damage promote platelet aggregation and activate coagulation mediators, resulting in thrombus formation (4). Furthermore, previous reports have found that patients with infarction in the posterior cerebral circulation upon head rotation had mobile thrombi (2, 5-7). These reports support the formation of thrombi due to vascular damage during head rotation.

In our case, duplex ultrasonography and dynamic angiography during head rotation detected hypoperfusion of the VA with no symptoms. In addition, angiographic findings suggested that an adequate vertebrobasilar flow was maintained via the contralateral flow during head rotation and revealed thrombotic translucency, indicating a thrombus. Because no symptoms were triggered by head rotation or thrombus formation, we concluded that the cause of bow hunter's stroke in this case was A-to-A embolism.

Bow hunter's stroke is rare, and there are no standard management options. Current treatments include conservative therapy with antiplatelet or anticoagulation agents and a neck collar, surgical decompression, surgical fusion, and endovascular therapy (3). In a previous report, none of 19 patients with conservative therapy developed posterior circulation stroke (8). In contrast, Rastogi et al. previously reported that only 37% of patients who received a conservative therapeutic approach had complete recovery from their symptoms (9). Thus, the efficacy of conservative therapy remains controversial.

Endovascular therapies described in previous reports have included coil embolization, angioplasty with stenting, and a microvascular plug (5, 6, 10-16). Endovascular therapy can be less invasive than surgical therapy, and the range of head motion is not reduced. Angioplasty with stenting has been used to treat arterial stenosis, which has been implicated as a cause of hemodynamic occlusion (15). However, stent stenosis or occlusion may be caused when the VA is occluded during head rotation. In contrast, coil embolization, which we performed, has been used to occlude the parent artery to eliminate the risk of thromboembolism while sustaining the posterior circulation (5, 15, 16). Interestingly, in previous cases that used coil embolization, patients had no symptoms caused by head rotation (5, 6, 10, 16). Furthermore, in two of four cases, the formation of a mobile thrombus was confirmed (5, 6). Coil embolization was used to occlude the culprit artery, eliminating the risk of thromboembolic complications (5). For coil embolization, angiograms are needed to verify the efficiency of the collateral circulation by balloon test occlusion. When there is efficient collateral circulation, coil embolization serves to prevent the recurrence of bow hunter's stroke. Coil embolization can thus be considered in cases in which conservative treatment has failed, especially when surgery is difficult because of antiplatelet therapy. Furthermore, coil embolization may prevent recurrence caused by embolic mechanisms in cases that have a confirmed mobile thrombus.

Conclusion

Coil embolization for the treatment of bow hunter's stroke is feasible and might be safe and clinically effective. However, there are no large clinical series assessing the outcome of endovascular therapy. Large randomized clinical trials are warranted to evaluate the effectiveness of coil embolization.

The authors state that they have no Conflict of Interest (COI).

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