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Case Report

Surgical clip occlusion of the V3 segment to prevent recurrent cerebral infarction associated with extracranial vertebral artery dissection: A case report

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Received : 05 July 2020
Accepted : 24 September 2020
Published : 15 October 2020

DOI
10.25259/SNI_405_2020

Quick Response Code:

ABSTRACT

Background: Recurrent cerebral infarction caused by traumatic extracranial vertebral artery dissection (EVAD) is treated medically and surgically. We report a case of EVAD that was treated using surgical clip occlusion of the V3 segment to prevent recurrent cerebral infarction.

Case Description: A 48-year-old man was admitted for a cerebral infarction caused by EVAD and was treated using 200 mg/day cilostazol. Afterward, the cerebral infarction recurred. Digital subtraction angiography revealed that initial severe stenosis of the VA ostium resulted in the final occlusion and that collateral vessels to the VA remained. We continued antiplatelet therapy, but the cerebral infarction recurred due to thromboembolism of the collateral vessels. Parent artery occlusion was planned. We exposed the V3 segment of the VA and clipped it to prevent the recurrence of cerebral infarction.

Conclusion: Surgical clip occlusion of the V3 segment was effective for treating recurrent cerebral infarction caused by traumatic EVAD that had remained an issue despite continuing medical therapy.

Keywords: Extracranial vertebral artery dissection, Occlusion clipping, Parent artery occlusion, V3 segment, Vertebral artery

INTRODUCTION

Traumatic extracranial vertebral artery dissection (EVAD) occurs less frequently than intracranial VA dissection but is recognized as an important cause of ischemic stroke. EVAD has occurred after spinal manoeuver[1,10] and has often caused thrombotic cerebral infarction.[1] Medical therapy is recommended as a first-line treatment for EVAD.[1,3,6] Despite the continuation of medical therapy, if a recurrent ischemic stroke occurs, which often presents as transient ischemic attacks or aneurysms, surgical parent artery occlusion (PAO) is performed.[2,4,5,7-9] We report a rare case of traumatic EVAD with recurrent cerebral infarction. The patient was effectively treated with surgical clip occlusion of the V3 segment of VA and had no recurrence of cerebral infarction.

CASE DESCRIPTION

A 48-year-old man without connective tissue disease received a neck massage 2 years before being admitted to our hospital. Two months after the massage, he experienced vertigo and underwent magnetic resonance imaging (MRI) at another hospital. MRI revealed bilateral cerebellar infarction; however, its cause could not be determined. One year later, he received another neck massage and experienced vertigo. He visited the same hospital, and a second MRI revealed left cerebellar infarction and poor flow to the left VA [Figures 1a and b]. Digital subtraction angiography (DSA) revealed dissection from the VA ostium to the V2 segment and anastomoses between the VA and collateral vessels distal to the VA dissection [Figure 1c]. The neurosurgeon initiated antiplatelet therapy (200 mg/day cilostazol) to prevent cerebral infarction. One month later, the neurosurgeon consulted our hospital.

MRI findings in our hospital were similar to those obtained previously, and treatment with 200 mg/day cilostazol was continued while disease progression was observed. Five months later, he experienced right tinnitus and bilateral right hemianopia. MRI revealed left occipital lobe infarction [Figure 2a]. DSA revealed partial VA ostial occlusion despite progressive stenosis. One month later, DSA revealed complete VA ostial occlusion and V2 segment dissection, in which the antegrade VA flowed slowly through collateral vessels [Figures 2b and c]. We determined that the cerebral infarction risk was less than that of VA ostial stenosis. We continued 200 mg/day cilostazol treatment. Two months later, the patient experienced vertigo, and an MRI revealed left cerebellar and right occipital lobe infarction [Figures 3a and b]. DSA produced a similar image as the one produced previously [Figures 3c and d]. We strengthened the antiplatelet therapy (75 mg/day clopidogrel and 200 mg/day cilostazol).

Surgical PAO was planned because the patient requested surgery and experienced repeated cerebral infarction despite the presence of occluding VA ostium and enhanced antiplatelet therapy. We decided to clip the V3 segment, which was more distally positioned than the collateral vessels. He underwent surgery in the supine position with his head turned to the right. This position enabled access to the VA with fewer skin incisions and with less damage to the VA venous plexus. We exposed the VA V3 segment after identifying the suboccipital triangle and confirmed VA blood flow with Doppler ultrasonography and indocyanine green video angiography. We clipped the VA using Yasargil Titanium Mini Clips (FT720T) [Figure 4]. After the surgery, the patient was neurologically intact. MRI revealed no cerebral infarction and flowed through the left VA. DSA

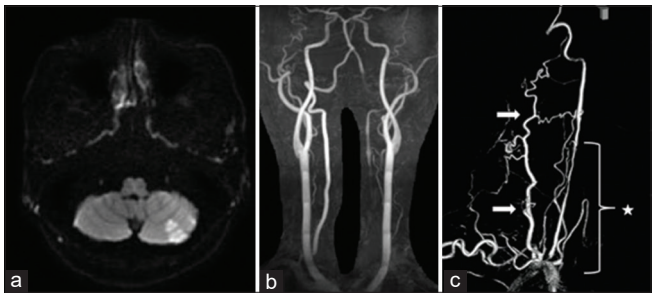

Figure 1: (a and b) Magnetic resonance images showing left cerebellar infarction and poor flow through the left vertebral artery. (c) Left subclavian artery angiography showing dissection from the vertebral artery ostium to the V2 segment, vertebral artery ostial stenosis (star), and collateral vessels (arrow).

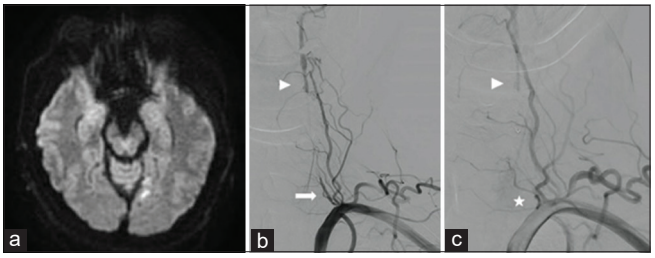

Figure 2: (a) Magnetic resonance images showing left occipital lobe infarction. (b and c) Left subclavian artery angiography showing vertebral artery flow from the collateral vessels (arrowhead), antegrade vertebral artery slow flow (arrow) from the vertebral artery ostium, and vertebral artery ostial occlusion (star).

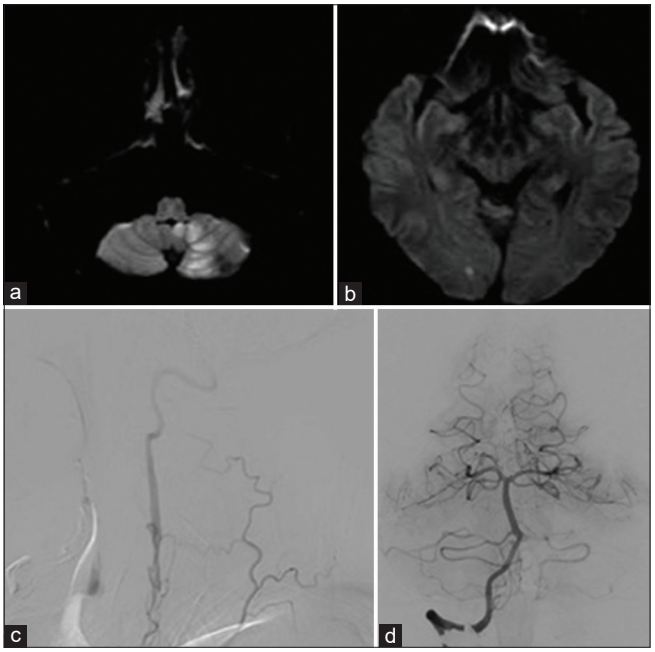

Figure 3: (a and b) Magnetic resonance images showing left cerebellar infarction and right occipital lobe infarction. (c) Left subclavian artery angiography showing antegrade vertebral artery slow flow from the collateral vessels. (d) Right vertebral artery angiography showing the dominant right vertebral artery.

revealed an absence of flow past the V3 segment and a good backflow from the right to left VA. No ischemic episodes occurred during the 20-month postsurgical follow-up period [Figure 5].

There was no requirement for IRB/ethics committee approval. The patient provided consent for the publication of this case report.

DISCUSSION

Several studies reported traumatic EVAD occurrence postspinal manoeuvers, including chiropractic manipulation and neck rotation.[1,10] These manoeuvers cause neurological sequelae resulting from cerebral ischemia. The cause of cerebral ischemia can be thromboembolism, hypoperfusion, or a combination of both. However, thromboembolism is considered the major cause of ischemic symptoms.[1]

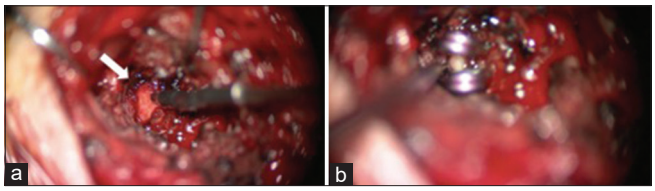

Figure 4: (a and b) Intraoperative findings reveal the V3 segment (arrow) and antegrade flow using Doppler ultrasonography. The V3 segment of the vertebral artery is clipped.

Antiplatelet or anticoagulant therapy is the recommended treatment for thromboembolism.[1,3,6] Surgical treatment is suggested for patients with traumatic EVAD accompanied by recurrent cerebral ischemia despite medical therapy.[5,7] Endovascular or direct surgery is selected on an individual basis.

Seven cases of traumatic EVAD treated with PAO were reported [Table 1].[2,4,5,7-9] Most cases occurred in younger patients, on the right side and on the V2 segment of the VA dissection. However, our case was the only one in which the cerebral infarction recurred after medical therapy. Endovascular and direct surgery was performed in three[4,7,9] and five[2,5,8] cases, respectively. Cohen *et al*.[4] reported an involved coil embolization of the right VA. A stent was used to treat the left VA due to bilateral EVAD. Two other cases were treated using coil embolization of the affected VA because the unaffected VA remained intact.[7,9] In all three cases, VA dissection was limited to the V2 segment and was approached from the VA ostium.

For direct surgery, multiple studies have reported PAO with bypass,[2,5,8] but our case was the first, in which surgical clip occlusion was executed without bypass because the unaffected VA was intact. According to Morgan and Sekhon,[8] the reason that direct surgery was selected was that the dissection ranged from the VA ostium to the V2 segment. Direct surgery is generally the most effective treatment for

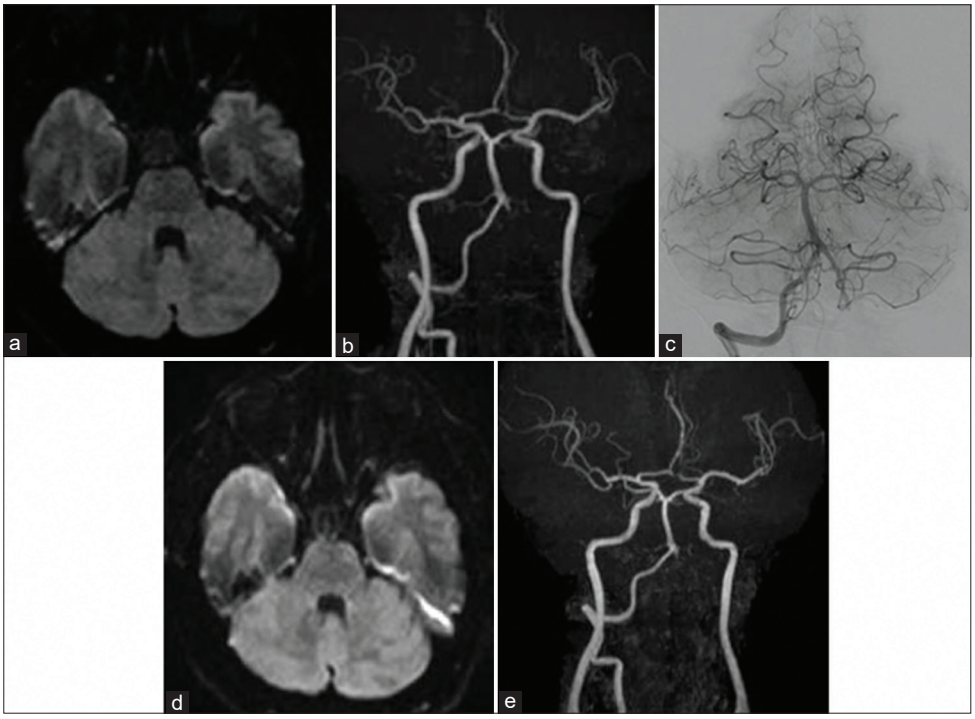

Figure 5: Magnetic resonance imaging and right vertebral artery angiography. No cerebral infarction and good backflow from the vertebral artery (right to left) can be observed. (a and b) Postoperative day 1. (c) Postoperative day 4. (d and e) Postoperative 20 months.

Table 1: Clinical profiles of previous recurrent ischemic stroke cases caused by extracranial vertebral artery dissection, and treated with parent artery occlusion.

Author	Age	Sex	Side	VA segment	Triggering event	Ischemic stroke	Opposite VA	Medicine	Recurrence	Treatment
Cohen *et al.*[4]	42	F	Bi	V2	—	Infarction	—	—	—	R: PAO (Coil), L: Stent
Matsuura *et al.*[7]	37	M	R	V2	Baseball	Infarction	Intact	Anti-coagulant	TIA	PAO (Coil)
Shimizu *et al.*[9]	39	M	R	V2	Bone anomaly	Infarction	Intact	Anti-platelet	TIA	PAO (Coil)
Morgan and Sekhon[8]	39	M	R	V2	Chiropractic	Infarction	Nonintact	Anti-coagulant	AN	PAO, VA-VA bypass
Morgan and Sekhon[8]	9	M	Bi	Rt: VA ostium-V2, Lt: V2	Trampoline	Infarction	—	Anti-coagulant	TIA	R: PAO, CCA-VA bypass
Chiche *et al.*[2]	48	F	L	V2	—	TIA	Unknown	Anti-coagulant	AN	PAO, ICA-VA bypass
Inoue and Matsuzawa[5]	33	M	R	V2	Sudden neck movement	TIA	Intact	Anti-platelet	TIA	PAO, OA-VA bypass
Our case	48	M	L	VA ostium-V2	Neck massage	Infarction	Intact	Anti-platelet	Infarction	PAO (Clip)

AN: Aneurysm, Bi.: Bilateral, F: Female, ICA: Internal carotid artery, L: Left, M: Male, OA: Occipital artery, PAO: Parent artery occlusion, R.: Right, TIA: Transient ischemic attack, VA: Vertebral artery

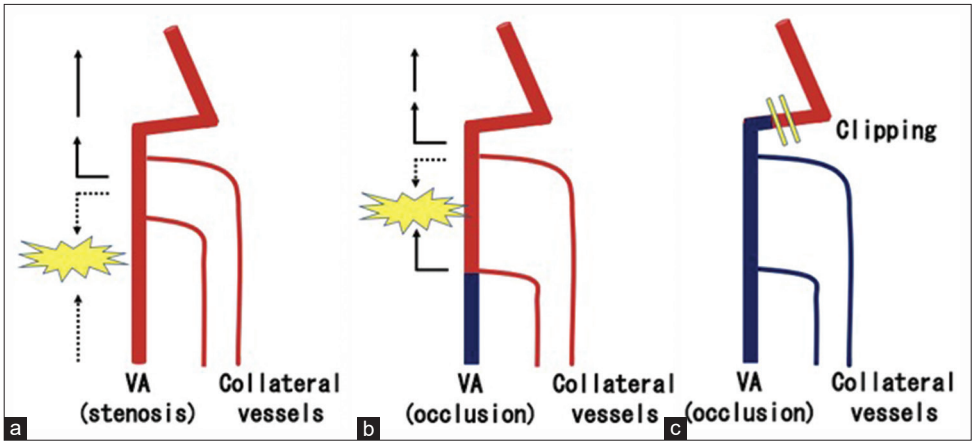

Figure 6: (a) Severe vertebral artery ostial stenosis, antegrade vertebral artery flow, and retrograde flow from collateral vessels collided and produced thromboembolism. (b) Vertebral artery ostial occlusion and collision of flow from several collateral vessels resulted in thromboembolism. (c) After surgical clip occlusion.

VA ostial occlusion because the dissection can be approached extravascularly.

Our patient experienced cerebral infarction despite VA ostial occlusion. We expected that the thrombus was formed due to a collision that occurred between the flow of several collateral vessels at the VA. Therefore, we decided to clip the V3 segment, which was more distally positioned than the collateral vessels [Figure 6]. To treat VA ostial occlusion using endovascular surgery, we were required to approach the occlusion from the unaffected VA through the intracranial VA union. No cases of surgical clip occlusion to prevent cerebral infarction for EVAD have been reported, and our surgery represents a treatment option that may be ideal for other patients, depending on the precise situations in which cerebral infarction or dissection occurs.

Surgical occlusion using only clips is only recommended when the affected VA ostium is occluded and an approach through the VA ostium is not possible; therefore, surgical occlusion without reconstruction has limitations.

CONCLUSION

Surgical PAO is selected when patients experience recurrent ischemic strokes despite continuous medical therapy. Surgical

occlusion using only clips without intracranial operation and craniotomy is a treatment option if the patient's pathology permits.

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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How to cite this article: Aiura R, Matsumoto M, Mizutani T, Sugiyama T, Tanioka D. Surgical clip occlusion of the V3 segment to prevent recurrent cerebral infarction associated with extracranial vertebral artery dissection: A case report. Surg Neurol Int 2020;11:337.