Vertebral Artery Stenting for Acute Multiple Cerebral Infarctions Caused by Vertebral Artery Dissection After Massage: A Case Report

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Case Report

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Abstract

Background: Vertebral artery dissection (VAD) is a special cause of ischaemic stroke in young patients. Cervical vertebrae massage, especially improper pulling manipulation, is one cause of vertebral artery dissection. The best therapy for acute VAD, whether conservative antithrombotic therapy or emergency endovascular therapy, has always been a topic of discussion.

Case presentation: The case of a young woman with acute multiple posterior circulation ischaemic cerebral infarction caused by left vertebral artery V4 segment dissection after improper neck massage is reported. Due to the serious condition of the patient, we performed emergency vertebral artery stent implantation at the site of dissection. The symptoms of the patient were significantly relieved on the second day. She recovered well in the later period without adverse complications or intravascular restenosis in the following year.

Conclusions: Endovascular therapy may be an effective measure to quickly relieve clinical symptoms and reduce serious complications in patients with acute VAD. Additional clinical observations are needed.

Introduction

Vertebral artery dissection (VAD) is recognized as a special cause of ischaemic stroke in younger patients. The incidence of VAD is estimated to be 2.6 to 3 per 100,000[1]. The majority of dissections are intracranial. It is estimated that vertebral artery dissection may be responsible for at least 20% of ischaemic strokes in young people[2]. Patients with arterial dissection usually present with acute neck and head pain and dizziness. Due to the various and complicated symptoms, there are difficulties in the timely diagnosis of VAD, which is critical for the prevention of more serious complications. Neck massage-related attacks are helpful for diagnosis[3]. Vascular ultrasonography, cranial vascular computed tomography angiogram (CTA), and digital subtraction angiography (DSA) are helpful for the diagnosis of VAD[4]. Treatment of VAD depends on multiple factors, including clinical manifestations, onset time, anatomical manifestations, number of damaged blood vessels, and contraindications to specific treatments[5]. Therapy includes thrombolytic therapy, antithrombotic agents (antiplatelet or anticoagulant agents), endovascular therapy, and surgery. To date, anticoagulant therapy has been recommended as the main treatment[5–6], while endovascular therapy is suggested when the condition is aggravated[7].

Herein, we report a young female who suffered head and neck pain after a neck massage, which was acutely aggravated after one night of rest and resulted in multiple acute cerebral infarctions in the posterior circulation. She recovered well after emergency treatment with vertebral artery stenting. No recurrence or adverse reactions occurred during the 1-year follow-up.

Case Presentation

A 32-year-old female was brought to the emergency department of our hospital by ambulance on March 22, 2021. She awoke with sudden severe left-side head and neck pain (VAS score: 10 points), dizziness, nausea, and vomiting. She underwent neck massage on the previous day and suffered sudden pain in the left head and neck after the cervical vertebrae pulling method. The visual analogue scale (VAS) score was approximately 5 points and was relieved after resting at home. The patient had only a medical history of hyperlipidaemia for 2 years and was treated with atorvastatin calcium.
We received the patient at 14:00 and found that the patient had severe pain in the left head and neck, radiating to the top of the head, which was aggravated by turning the head, dizziness, frequent vomiting, difficulty in standing and walking, and consciously blurred vision. Physical examination findings were as follows: Blood pressure 164/94 mmHg, consciousness, fluent speech, bilateral coarse horizontal nystagmus, bilateral finger-nose test (+), heel-kneetibia test (+), and Romberg’s sign (+). The NIHSS score was 4 (1 point for Questions, 1 point for Commands, and 2 points for Ataxia). Her brain CT showed a patchy area of hypodense white matter in the deep right temporal lobe (Fig. 5A). Brain MRI showed multiple acute cerebral infarction lesions in the bilateral cerebellum, brainstem, medial temporal lobe, and occipital lobe (Fig. 1B-C).

The patient entered the interventional radiotherapy room at 15:00. The Seldinger technique was used to complete the right femoral artery puncture and catheterization under local anaesthesia. A 6F arterial sheath (Terumo, Japan) was placed, and a single-curved catheter and loach catheter were inserted through the arterial sheath. Under the guidance of the guide wire, the catheters were positioned in the aortic arch, the proximal ends of the bilateral subclavian arteries, the bilateral common carotid arteries, and the bilateral vertebral arteries for angiographic examination. Angiography showed suspected arterial dissection in the V4 segment of the left vertebral artery, with severe stenosis of the blood vessel, a stenosis rate of approximately 85% and a stenosis length of approximately 8 mm. Forwards flow indicated TICI grade 1. A local, distinct rectangular notch could be seen as an obvious manifestation of dissection (Fig. 2A-B).

The endovascular treatment process was as follows. Considering that local thrombus shedding would lead to acute cerebral infarction and recurrence of cerebral infarction, a local stent was placed at the left vertebral artery V4 segment. We used a Command microguide wire (0.014mm*190mm, Abbott, U.S) and Rebar18 microcatheter (Ev3, U.S) to carefully pass through the stenosis of the dissection for placement at the distal end of the dissection. Then, we withdrew the microguide wire and sent an self-expanding Solitaire AB stent (Ev3, U.S) along the Rebar18 one piece microcatheter (6mm*30mm). After adjusting the position of the stent, the microcatheter was withdrawn to release the stent. A re-examination angiography showed that the stenotic part of the local blood vessel was in contact, and the dissection notch had disappeared (Fig. 2C-D).

After returning to the ward, the patient was relieved from headache, nausea and vomiting. Her NIHSS score decreased to 1 (1 point for ataxia). After 7 days of hospitalization, the patient’s symptoms were completely relieved, and her NIHSS score decreased to 0. After endovascular treatment, she orally took aspirin tablets (0.1 g per day) and clopidogrel bisulfate tablets (75 mg per day) for 3 months and then changed to aspirin tablets (0.1 g per day) for 1 year.

At the 1-month follow-up after the operation, the patient was asymptomatic and was able to work and live normally. No new cerebral infarction lesions were found in the brain MRI. Three months and 6 months after the operation, re-examination cranial vascular CTA showed that the local blood vessels were unobstructed, and the dissection had disappeared (Fig. 3A-B, C-D). After 1 year, the patient’s condition was stable without recurrence, and she could work and live normally. No new cerebral infarction lesions were found in the re-examination of the brain MRI (Fig. 4A-B). The re-examination of the cranial vascular DSA showed that the local blood vessels were smooth without thrombosis or stenosis (Fig. 4C-D).

**Discussion And Conclusions**

VAD accounts for only 2% of ischaemic strokes but is the leading cause of ischaemic stroke in people under 45 years of age[8]. The mechanisms of ischaemic stroke induced by VAD include the following: 1) tearing of the blood...
vessel wall, which compresses the surrounding tissue and leads to ischaemia, 2) lumen stenosis and occlusion caused by dissection, 3) detached embolism after secondary thrombosis, and 4) haemodynamic disorders[9].

The patient presented a moderate headache, and then developed severe head and neck pain, ataxia, dizziness, nausea, and vomiting the following day. The symptoms of VAD were in accordance with a previous report[10]. Brain MRI examination revealed multiple and sporadic acute cerebral infarction lesions in the brain. No distal vascular occlusion was found on the examination. The formation of a thrombus in the interlayer, which broke off and embolized the circulating vessels, was considered to be the underlying cause[11].

Typical imaging features of VAD through DSA include eccentric stenosis with external duct dilatation, beaded or segmental stenosis, intramural haematoma, double-lumen sign, intimal valve, and formation of dissecting aneurysm. Among them, the double-lumen sign and intimal flaps are the symptoms of a direct diagnosis of arterial dissection[12]. It has also been reported that irregular lumen stenosis or occlusion, thin line sign, rat tail sign, and pseudoaneurysm in DSA are the diagnostic signs of arterial dissection[13]. In this case, the DSA presented irregular luminal stenosis, clear distal vascular dilatation and an obvious right-angled vascular notch at the distal end of the vessel, which is not typical imaging features but is expected to provide a new imaging manifestation for arterial dissection.

Since the vertebral artery runs through the intervertebral foramen, VAD is often caused by mild mechanical stimulation, such as neck hyperextension or hyperflexion, or even head-turning[14]. The patient in the case was a young female with symptoms that appeared after neck massage with the pulling method. There is rarely a clear relationship between neck massage and VAD, but some studies have found that incorrect, oblique angle in cervical pulling manipulation, can lead to acute intimal damage and dissection of the vertebral artery. Some studies have also found that there are three segments of the vertebral artery that can easily peel off to form a dissection[15]: the origin of the subclavian artery, the area where it penetrates the intervertebral foramen, and the area where it passes through the dura of the skull base (the most common area, which is consistent with this case) [16]. The risk of carotid artery dissection and stroke during neck massage is related to cervical spine manipulation and the strength of neck rotation in pulling manipulation[17], but there is currently no clinical recommendation on the range of rotation angle and strength of cervical spine manipulation. The improper technique may require further investigation.

There are only a few reports about VAD related to neck massage in the literature. Six previously reported cases are summarized in Table 1[18–22]. Among the 6 patients, 5 patients had acute cerebral infarction with neurological deficits remaining. One patient had no cerebral infarction with bilateral carotid artery and bilateral vertebral artery dissection. Only one patient received emergency endovascular therapy due to acute vertebral artery occlusion[22], while the others received drug treatment. The symptoms and prognosis are shown in Table 1.
Table 1
Cases of vertebral artery dissection related to neck massage.

<table>
<thead>
<tr>
<th>Case</th>
<th>Trigger</th>
<th>Symptoms</th>
<th>Vessel</th>
<th>Cerebral infarction lesions</th>
<th>Treatment options</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>M.Bashar Katirji, et al. [18]</td>
<td>Chiropractic manipulation</td>
<td>Neck pain, vertigo, vomiting, hemiparesis, ataxia</td>
<td>Right vertebral artery dissection</td>
<td>Right cerebellum</td>
<td>Low molecular weight heparin for anticoagulation</td>
<td>Ataxia and hypoesthesia remained after 20 months</td>
</tr>
<tr>
<td>M.Bashar Katirji, et al. [18]</td>
<td>Chiropractic manipulation</td>
<td>Neck pain, nausea, vomiting, vertigo, left hand numbness, slurred speech, difficulty swallowing</td>
<td>Right vertebral artery dissection</td>
<td>Right medulla</td>
<td>Medicine treatment</td>
<td>Speech disorders, dysphagia and ataxia remained after 3 months</td>
</tr>
<tr>
<td>Stephen J. Phillips, et al. [19]</td>
<td>Chiropractic manipulation</td>
<td>Dizziness, speech disturbance, left-sided hemiplegia</td>
<td>Bilateral vertebral artery dissection</td>
<td>Right pons</td>
<td>Medicine treatment</td>
<td>Hemiplegia of left side remained after 16 days</td>
</tr>
<tr>
<td>R N Nadgir, et al. [20]</td>
<td>Chiropractic manipulation</td>
<td>Headache, ataxia, dysarthria, hypoesthesia</td>
<td>Left vertebral artery dissection</td>
<td>Right thalamus</td>
<td>Low molecular weight heparin for anticoagulation</td>
<td>Hemianesthesia of left side remained after 1 month</td>
</tr>
<tr>
<td>Andrea L. Chakrapani, et al. [21]</td>
<td>Chiropractic manipulation</td>
<td>Neck pain, ptosis</td>
<td>Bilateral internal carotid arteries</td>
<td>None</td>
<td>Warfarin, Clopidogrel, aspirin•</td>
<td>Improved completely after 4 weeks; recovered completely after 1 year of follow-up</td>
</tr>
<tr>
<td>Jiang-Qiong Ke, et al. [22]</td>
<td>Chiropractic manipulation</td>
<td>Complete quadriplegia, complete facial and bulbar palsy, dyspnoea</td>
<td>Bilateral vertebral artery dissection</td>
<td>Bilateral pons</td>
<td>Emergency endovascular therapy</td>
<td>Hemiplegia of both sides remained after 27 days</td>
</tr>
</tbody>
</table>

The treatment of VAD should take into account the condition of stroke and arterial dissection. According to the AHA/ASA Guidelines for the Early Management of Acute Ischaemic Stroke published in 2019, intravenous thrombolysis using alteplase within 4.5 h of onset is safe and reasonable in patients with acute ischaemic stroke with suspected carotid artery dissection[23]. For the occurrence outside of the intravenous thrombolysis therapy window (more than 4.5 h), antithrombotic therapy, including antiplatelet aggregation and anticoagulant therapy, is mainly recommended for treating ischaemic stroke and transient ischaemic attack and preventing the recurrence of artery dissection[24]. Studies have shown that 14% of patients with VAD experience an exacerbation of symptoms within 1 month, and most of them are alleviated at least 6 months later[25].
Endovascular therapy has become an important choice for the treatment of carotid and vertebral artery dissection in recent years but has mostly been evaluated in small samples[26]. The Chinese Guidelines for the Diagnosis and Treatment of Carotid Artery Dissection (2015) recommended that endovascular therapy could be chosen for carotid artery dissection, while stroke events were not controlled by drug therapy[27]. In this case, the patient was a young female with an acute onset and aggravated progression. Considering the critical condition, we adopted emergency endovascular therapy by placing a self-dilating Solitaire AB stent at the VAD and then applied aspirin and clopidogrel. The treatment process, in this case, can provide clinical ideas for endovascular treatment of stroke events caused by acute VAD so that the patients can receive more timely and effective treatment and more quickly achieve remission. However, additional cases and longer periods of follow-up are needed.

In conclusion, it is particularly important to collect the patient's previous medical history to determine some special pathogens for the diagnosis of VAD. Endovascular therapy may be an effective way to quickly relieve clinical symptoms and reduce serious complications in patients with acute VAD. In our experience, the timely diagnosis and emergency endovascular therapy for the young female patient resulted in the rapid relief of symptoms and effective prevention of progression, in addition to reducing the possibility of permanent disability or even death from VAD.

**Abbreviations**

VAD  vertebral artery dissection  
IAD  intracranial arterial dissection  
VAS  visual analogue scale  
CTA  computed tomography angiogram  
DSA  digital subtraction angiography  
NIHSS  National Institutes of Health Stroke Scale  
MRI  magnetic resonance imaging  
CT  computed tomography  
DWI  diffusion-weighted imaging  
ADC  analogue to digital converter.

**Declarations**

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**Authors’ contributions**
Yx C and Ss W contributed to the writing and editing of the manuscript. Xs H collected the medical data and the follow-up information of the patient. Ss W, Gl L and XJ performed the endovascular treatment, BL supervised the findings of this work, and all authors have read and approved the final manuscript.

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**Availability of data and materials**

The data that support the findings of this case are available on reasonable request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

**Ethics approval and consent to participate**

Not applicable.

**Consent for publication**

Written informed consent for the publication of this case report and any accompanying images was obtained from the patient. A copy of the written consent is available for review by the Editor of this journal.

**Competing interests**

The authors declare that they have no competing interests.

**References**


**Figures**

![Figure 1](image)

**Figure 1**

A: CT showed patchy low-density areas in the deep white matter of the right temporal lobe, suggesting ischaemic changes (black arrow). B-C: Speckles and flecks of long T1 and T2 signals, high DWI signals, and low ADC signals were observed in the bilateral deep temporal lobe, right occipital cortex, pons, and bilateral cerebellar hemispheres. Multiple acute cerebral infarctions were considered (black arrows).
Figure 2

A-B: There was significant eccentric stenosis and localized thickening of the posterior segment of the left vertebral artery in the proximal V4 segment, with a local obvious rectangular notch (black arrow), consistent with arterial dissection. C-D: After local stent implantation, the vascular eccentric stenosis improved, the vascular wall was significantly smooth, and the local notch disappeared (black arrow), but the distal vessels remained dilated, and the proximal vessels were observed to be vasospasms due to the passage of the guide wire.
Figure 3

A-B: Three months after the operation, the local stent shadow in the V4 segment of the vertebral artery (white arrow) passed through the foramen magnum, and the blood flow in the stent was smooth without stenosis. C-D: Re-examination 6 months later showed that the V4 segment of the vertebral artery passed through the foramen magnum with a local stent shadow (white arrow), and the blood flow in the stent was unobstructed without stenosis.
Figure 4

A-B: One year after the operation, no new cerebral infarction lesions were found in the re-examination of the brain MRI. C-D: DSA imaging showed a stent implantation shadow in the V4 segment of the vertebral artery. Compared with the DSA imaging findings in March 2021, the local lumen had completely returned to normal, the proximal and distal vessels had the same diameter, the vessel wall was smooth, and the endovascular notch had completely disappeared.