CASE REPORT

Anterior decompression of persistent vertebral artery occlusion caused by the cervical facet joint originated osteophyte

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Background: Persistent vertebral artery occlusion caused by compression of cervical facet joint originated osteophyte is exceptional rare. The authors sought to achieve adequate decompression of the vertebral artery (VA) with less stability decrease and movement restriction via the anterior approach, and to the authors' knowledge, no case of anterior decompression of this condition has been reported, and combination of intraoperative indocyanine green (ICG) angiography in the setting of VA decompression is also rare.

Case presentation: A 77-year-old man presented continuous vertigo, unsteady gait and dysphagia with no relationship to the head movement. Preoperative computed tomography angiography (CTA) and digital substraction angiography (DSA) examination revealed the left vertebral artery was severely compressed at $C_{4\text{-}5}$ level with approximately 95% occlusion due to a left $C_{4\text{-}5}$ facet joint originated large osteophyte. Successful anterior decompression was performed without fusion and intraoperative ICG fluorescence angiography proved excellent blood flow. After surgery, vertebrobasilar insufficiency symptoms remarkably improved with no neurological deficits and no recurrence at 12 months' follow-up.

Conclusions: The authors' therapeutic strategy of anterior decompression was successful in treating VA compression due to facet joint overgrowth with adequate exposure, no stability decrease and movement restriction, and lower rates of neck pain and blood loss.

Key words: Anterior decompression; Cervical facet joints; Vertebral artery occlusion; Vertebrobasilar insufficiency

Introduction

Vertebrobasilar insufficiency (VBI) is characterized by syncope, vertigo, tinnitus, hypoacusis, dysphagia, visual blurriness, sensory and motor deficits[1–3]. These symptoms can be caused by both intrinsic and extrinsic factors. Extrinsic mechanical compression of vertebral artery (VA) caused by cervical spondylotic osteophyte is one of the etiological factors and symptoms usually induced by rotational head movement, which is named as rotational vertebral artery occlusion (RVAO). These osteophytes mainly originate from cervical uncovertebral joints[4,5]. Though this kind of compression have been decompressed successfully via the anterior approach[4,5], the facet joint originated persistent bony compression of VA and corresponding anterior decompression is exceptional rare. This case report, with consent from the patient, describes anterior cervical decompression of persistent vertebral artery occlusion caused by posterolateral cervical facet joint originated osteophyte compression which is not related with head and neck movement.

Case Report

Clinical Presentation and Examination

A 77-year-old man presented with 6 months history of continuous vertigo, unsteady gait and dysphagia, and these symptoms were not related to his head movement. He is a

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retired farmer and his social history included never smoking, socially drinking. There were no radiculopathy symptoms of the upper limbs. No positive sign was found on neurological examination. He had been treated with flunarizine and betahistine at the local hospital, but without benefit. The Dizziness Handicap Inventory (DHI) score was 82. Preoperative vertebral artery computed tomography angiography (CTA) examination revealed the V2 segment of the left VA was severely compressed at $C_{4\text{-}5}$ level close to the left C_4 transverse foramen with approximately 95% occlusion due to an anteromedially protruding left $C_{4\text{-}5}$ facet joint originated large osteophyte (Fig. 1A–C). No uncovertebral joint originated bony compression was found (Fig. 1C). 50% stenosis of the V4 segment of the right VA was also found (Fig. 1D). Preoperative cranial magnetic resonance angiography (MRA) showed poor blood flow of V4 segment of the left VA, as well as 50% stenosis of the V4 segment of the right VA consistent with the CTA (Fig. 1E). Digital subtraction angiography (DSA) was then performed. During the procedure, we found there was occlusion of the V4 segment of the right VA (Fig. 1F,G), but not very severe relative to the preoperative CTA and MRA and no indication for angioplasty or stenting. Meanwhile, 95% occlusion of the V2 segment of the left VA at the level of $C_{4\text{-}5}$ was confirmed, and no evidence of internal factors such as atherosclerosis and embolization was found (Fig. 1H,I). We did not find neck tumors through preoperative CT and MRI. No evidence of cerebral infarction was found by brain magnetic resonance imaging (MRI). The cause of his symptoms were determined to be hemodynamic insufficiency due to left VA compression. In order to prevent permanent neurological deficits caused by vertebrobasilar ischemia attack, left VA decompression surgery was indicated.

Surgical Procedure

The surgery approach was basically the same as our routinely performed anterior cervical discectomy/corpectomy and fusion (ACDF/ACCF). After endotracheal intubation general anesthesia, the patient was kept supine on the operating table. After fluoroscopy localization, a horizontal incision was made in a natural skin crease between the midline and sternocleidomastoid muscle. After a careful soft tissue dissection, the platysma was longitudinally splited. The carotid sheath was retracted laterally, the esophagus and trachea were retracted medially. The left longus colli was splited and the anterior aspect of the vertebral body and left transverse process of C_4 was exposed. Under the operating microscope, the anterior margin of the left transverse process and osteophytes origin from left C4/5 facet joint were carefully excised by ultrasonic osteotome instead of high speed burr to protect surrounding soft tissue. Then the fibrous adhesion around the VA was excised. After decompression (Fig. 2A), intraoperative indocyanine green (ICG) fluorescence angiography proved excellent blood flow of the left VA (Fig. 2B). Postoperative CTA revealed that the osteophyte and anterior margin of the left C_4 transverse foramen was excised (Fig. 2C–E) and no compressive lesion of the left VA (Fig. 2E,F). Surgery time was 3 h and 34 min.

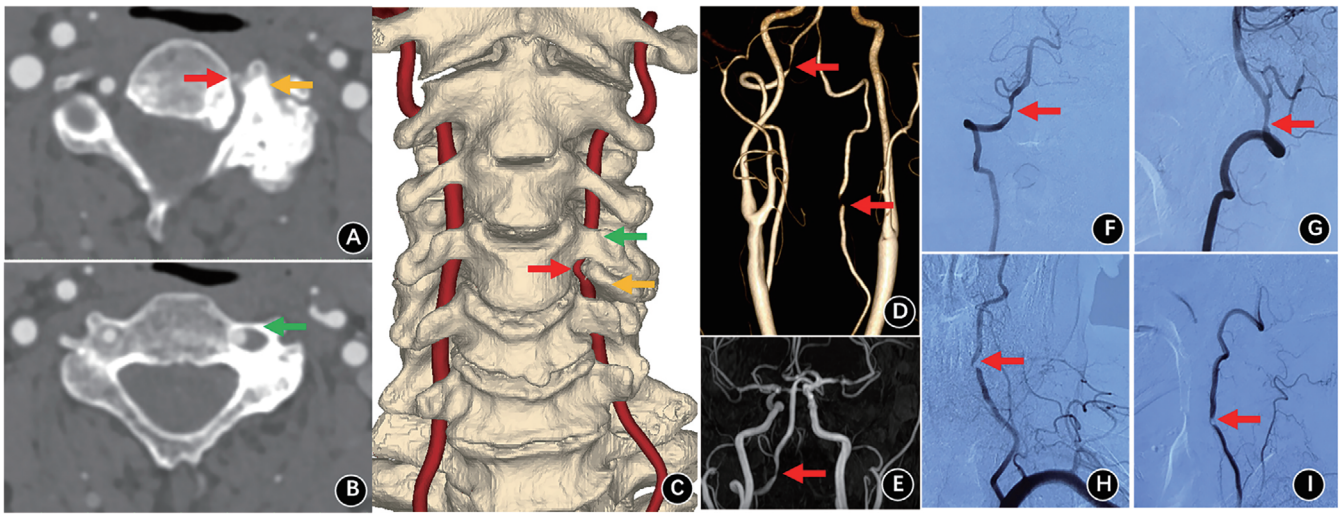

Fig. 1 (A–C) preoperative axial CT scan and 3D reconstruction images of the left C4/5 facet joint originated osteophyte (yellow arrow) compressing the left vertebral artery (VA) (red arrow) and intact anterior margin of the left C4 transverse foramen relative to postoperative CT images (green arrow). (D) preoperative CTA showing severe stenosis of the V2 segment of the left VA (red arrow) and the V4 segment of the right VA (red arrow). (E) preoperative MRA showing poor blood flow of the V4 segment of the left VA and stenosis of the V4 segment of the right VA (red arrow). (F, G) acceptable atherosclerotic stenosis of the V4 segment of the right VA (red arrow). (H, I) preoperative DSA showing 95% occlusion of the V2 segment of the left VA (red arrow).

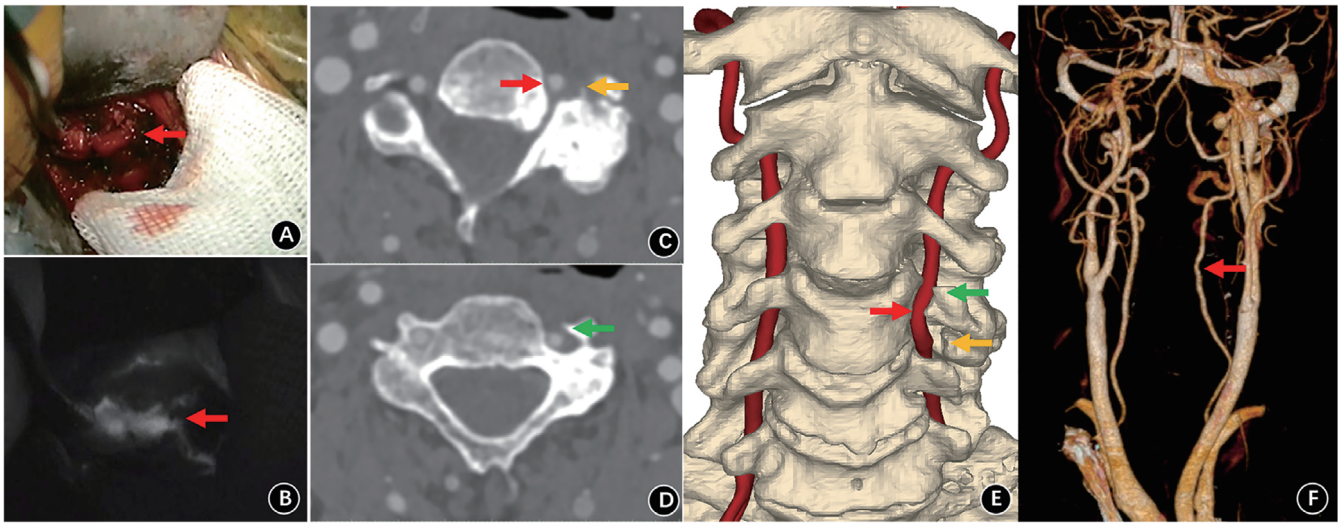

Fig. 2 (A) intraoperative photo after decompression of the left vertebral artery (VA) (red arrow). (B) intraoperative post-decompression ICG fluorescence angiography revealing excellent blood flow (red arrow). (C–E) Postoperative axial CT scan and 3D reconstruction images showing the left C4 facet joint originated osteophytic compression (yellow arrow) and anterior margin of the left C4 transverse foramen (green arrow) was excised and post-decompression left VA (red arrow). (F) Postoperative CTA showing no compressive lesion of the left VA (red arrow).

Postoperative Outcome

Antiplatelet therapy was given after surgery. No Philadelphia collar was used, and the patient was allowed to move his head freely. The patient stated he felt significant improvement of his symptoms, with no neurological deficits and restriction of neck movement. Histologically, the removed tissue consisted of cartilage and bone tissue. The DHI score 1 week after surgery was 2. There was no recurrence of his vertebrobasilar symptoms and no symptoms of cervical instability at 12 months' follow-up. We did not consider invasive postoperative DSA because adequate vascular decompression had been proved by intraoperative ICG angiography and postoperative CTA, and the symptoms improved very well.

Discussion

VBI are mainly caused by intrinsic factors, like atherosclerosis, emboli, etc. Extrinsic factors like osteophytic overgrowth, tumor or tendinous bands are rare[4–6]. Neither open nor endovascular intervention should be performed in asymptomatic VA stenosis, but in patients with symptomatic VA stenosis, invasive intervention should be considered for lesions 50% in patients with recurrent symptoms[7]. In this case, the patient has typical symptom of VBI and bilateral VA stenosis caused by different etiological factors. The right intracranial VA stenosis was caused by intrinsic atherosclerosis, and the left VA stenosis was caused by extrinsic osteophyte compression with 95% occlusion. In order to prevent permanent neurological deficits caused by vertebrobasilar ischemia attack, intervention towards the left VA was indicated. Since transluminal procedure is not applicable to resolve the bony compression of the left VA, open decompression surgery was chosen. In VBI patients, osteophytes mostly originate from uncovertebral joints[4,5] and symptoms usually induced by head and neck movement. Patients with persistent symptoms caused by posterior facet joint originated osteophytes are exceptional rare, and VA compression can be well alleviated by excision of osteophytes.

The surgical approach should be tailored to each individual patient based on the characteristic of the compression[1–4,8]. To those cases with uncovertebral joints originated osteophytes, after uncovertebral joints resection and partial discectomy, instrumented fusion was needed to reconstruct the stability of the cervical spine. To this end, both anterior and posterior approaches may achieve VA decompression. However, facetectomy needs to be performed in the posterior approach, while integrity of the facet joints play an important role in the stability of cervical spine[9–11], internal fixation may need to be performed[12], which results in postoperative restriction of the neck movements, higher rates of neck pain; and blood loss was also reported[13]. Therefore, we performed anterior decompression surgery to achieve adequate exposure of the VA which results in a complete and direct decompression, while not leading to iatrogenic cervical instability. Also, the anterior aspect of transverse foramen and perivascular fibrous tissue can be directly removed and ICG fluorescence angiography can be administrated via the anterior approach. ICG fluorescence angiography is rarely used in VA decompression surgeries of VBI and we administrated intraoperative ICG fluorescence angiography to confirm the adequacy of the VA decompression. Intraoperative DSA is not acceptable as noninvasive ICG fluorescence angiography, which is a useful adjunct and may supplant intraoperative

catheter angiography in anterior VA decompression surgeries[14]. Tissue-specific untrasonic osteotome was used to make precise osteotomy and minimize the possibility of adjacent VA and soft tissue injury, as soft tissue is not affected by the ultrasonic oscillation[15].

Conclusion

We promptly performed surgery and symptoms quickly improved with no neurological deficits. Surgery was strongly suggested to this patient and a surgical plan was tailored on the characteristics of the compression. We propose that anterior decompression is an effective approach for the treatment of vertebral artery compression due to cervical facet joint overgrowth which provides adequate exposure, with no stability decrease and movement restriction, and lower rates of neck pain and blood loss. High-tech ICG fluorescence angiography and untrasonic osteotome are useful and effective tools in this kind of surgery to shorten surgery time and minimize surgical injury.

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Authorship Declaration

All authors listed in this manuscript meet the authorship criteria according to the latest guidelines of the International Committee of Medical Journal Editors, and all authors are in agreement with the manuscript.

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