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Case Report

A case report: C2 radiculopathy induced by neck flexion due to the cord compression of C2 segmental type vertebral artery relieved by microvascular decompression

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Abstract

Background: Decompression of an anomalous vertebral artery (VA) may effectively treat cervical myelopathy/radiculopathy due to resultant spinal cord or nerve compression. Here we report a case of C2 radiculopathy induced by neck flexion due to cord compression of the C2 segmental type VA relieved by microvascular decompression.

Case Description: A 30-year-old female presented with left occipitalgia, sensory abnormalities in the left upper and lower extremities, and neck pain induced by neck flexion. The magnetic resonance imaging (MRI) revealed an abnormal flow void, confirming that the VA was compressing the spinal cord at the C1 level. Three-dimensional computed tomography (3D-CT) showed an anomalous course of the left VA, which entered the spinal canal between the axis and atlas. Microvascular decompression was performed by transposing the artery (e.g., anchoring it to the dura using PTEF): this effectively relieved cord compression.

Conclusion: An anomalous VA rarely causes cervical radiculopathy induced by neck flexion. When it occurs, microvascular decompression effectively relieves pressure resulting in a resolution of symptoms.

Key Words: Anomalous vertebral artery, C2 segmental type vertebral artery, cervical radiculopathy, microvascular decompression



INTRODUCTION

C2 radiculopathy may be attributed to and cervical compression may be attributed to an anomalous vertebral artery (VA). These patients may present with symptoms consistent with other neurovascular compression syndromes; for example, hemifacial spasm, trigeminal neuralgia, and glossopharyngeal neuralgia. Previously, microvascular decompression of anomalous VA had effectively treated these disorders.^[2,3] Among them, only one reported pain at the nape of the neck induced by exercise.^[5] Further, in these prior reports, the youngest patient was 36 years old.^[1]

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Here we report a 30-year-old female who presented with radiculopathy induced by neck flexion. Magnetic resonance imaging (MRI) assessment and CT revealed that it was due to compression of the C2 nerve root by a segmental type VA. She was successfully treated by microvascular surgical decompression.

CASE REPORT

A 30-year-old female exhibited neck pain induced by neck flexion. This was considered to be C2 radiculopathy. The neurological examination was normal. SEP and MEG of the upper extremities were normal. The cervical x-ray was unremarkable, but the motion-sensitized driven-equilibrium (MSDE)-turbo spin echo (TSE)^[6] and T2-weighted MRI revealed an abnormal flow void, consistent with an anomalous VA, (e.g., documented on fusion images [LEXI, Tokyo, Japan] using MRI and three-dimensional computed tomography [3D-CT] angiography) [Figure 1a]. The VA that entered the spinal canal between the axis and atlas and compressed the spinal cord at the C1 level [Figure 2a and b]. The intradural course of the left VA formed the vascular loop and compressed the cord at the C1 level.

She underwent microvascular surgical decompression of the cord by performing a VA mobilization procedure. This required a suboccipital craniectomy and Cl laminectomy (e.g., midline skin incision). The left VA was accessed through a paramedian dural incision. The left VA was mobilized laterally and anchored to the dura mater by PTEF tape (2 mm; BARD) [Figure 3]. Dural closure utilized a fascia flap to prevent shortening of the distance between the anchor site and the dural closure site.

Postoperatively the patient was pain-free [Table 1]. Postoperative MSDE-TSE and T2-weighted MRI showed that the left VA was separated from the surface of the cord [Figure 2c and d]. The postoperative fusion MRI



Figure 1: Fusion images by MRI and three-dimensional computed tomography (3D-CT) angiography. (a) Preoperation, (b) postoperation. (a) shows the anomalous course of the left VA, which entered the spinal canal between the axis and atlas (arrowhead). There was no fenestration of the VA and no laterality of the diameter of the VA. The intradural course of the left VA formed a vascular loop and compressed the spinal cord at the CI level (arrow). (b) shows that the left VA was separated from the surface of the cord accompanied by CI laminectomy (arrow)

and 3D-CT angiography [Figure 1b] also revealed that the left VA was separated from the surface of the cord following the C1 laminectomy.

DISCUSSION

According to the previous reports describing the compression of the cervical cord or nerve roots by an elongated VA or VA anomaly inducing myelopathy or radiculopathy, such as trigeminal neuralgia and hemifacial spasm, the symptoms observed were radiculopathy, and/ or myelopathy at the level of the cord compression. Most of the cases were treated by microvascular surgical decompression, with improvement of symptoms obtained in all cases.^[2,3] We experienced a case of C2 radiculopathy induced by neck flexion due to cord compression of the C2 segmental-type VA traveling medially and entering the spinal canal between C1 and C2. This unique course represents a C2 segmental VA first reported by Tokuda et al. in 1985.^[4] They reported that 2 of 300 patients free from disease at the craniocervical junction had a C2 segmental VA. Surgical treatment in these reports was almost always transposition of VA. Other various materials were also reported.^[2,7] In our case, the left VA was anchored to the dura mater by PTEF tape. An important point to ensure is that the anchor site should be kept from moving with dural closure. Therefore, we used a fascia flap to prevent shortening of the distance between the anchor site and dural closure site.

The characteristic features of our case are the young age (the youngest among the previous reports) and the



Figure 2: Pre- and postoperative MRI findings. (a) Motion-sensitized driven-equilibrium-turbo spin echo (MSDE-TSE), (b) T2-WI: preoperative magnetic resonance imaging (MRI). (c) MSDE-TSE, (d) T2-weighted image (T2-WI): postoperative MRI. (a) shows that the cervical cord at the CI level is compressed by the left vertebral artery (VA). (b) shows an abnormal flow void indicating that the left VA is compressing the spinal cord at the CI level without a signal change in the spinal cord. (c) and (d) show that the left VA was separated from the surface of the cord. The compression of the cord was relieved after the operation



Figure 3: Intraoperative photographs. (a) The vascular loop of the left VA compressed the cervical cord at the CI level and C2 dorsal nerve roots (arrow). (b) Impression by the left VA on the cord and stretched C2 nerve roots was observed (arrow). (c) The left VA was mobilized laterally and anchored to the dura mater by PTEF tape to relieve the cord compression

symptom of neck pain induced by neck flexion. Only one other report^[5] described the same symptom as in our case.

Although the cause of neck pain induced by neck flexion is rarely radiculopathy/myelopathy due to compression of the cervical cord by the VA, surgical microvascular decompression can be effective when this is the case.

CONCLUSION

Anomalous VA is a rare cause of cervical radiculopathy induced by neck flexion. Microvascular decompression is an effective treatment for this disorder inducing symptom relief.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Table	1: T	he	results	in ar	ı asses	sment o	f pain	by	various
scale	S								

	pre-operation	post-operation
Short Form McGill Pain Questionnaire (SF-MPQ)	18/45	2/45
Visual Analogue Scale (VAS)	80mm	25mm
Present Pain Intensity (PPI)	3 (distressing)	2 (discomforting)

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Conflicts of interest

There are no conflicts of interest.

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