Cervical radiculopathy and spinal cord indentation caused by vertebral artery loop

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Abstract

Cervical radiculopathy and spinal cord indentation caused by vertebral artery loop formation is a rare entity. We report here an 85-year-old woman had a four-year history of neck pain and left arm weakness. Radiologic evaluation showed vertebral artery loop formation compressing the adjacent nerve root and spinal cord. Accurate radiologic diagnosis of this condition is crucial to reduce unexpected surgical complications.

INTRODUCTION

Vertebral artery (VA) loop formation is one of the rare vascular causes of cervical radiculopathy. It can be congenital or acquired. Most cases are asymptomatic and diagnosed incidentally. Cervical magnetic resonance imaging (MRI) is an effective diagnostic tool to evaluate causes of cervical radiculopathy but to detect foraminal stenosis, which causes a bony compression on the nerve, computed tomography (CT) is an advantageous imaging method. Also, vascular imaging studies, including magnetic resonance angiography (MRA), computed tomography angiography (CTA), are needed to confirm the vascular origin and detail of nerve compression.1

Here, we present a rare case of cervical radiculopathy caused by the aberrant course and loop formation of the left vertebral artery that indented the spinal cord at the C3-C4 level. The patients gave signed consent for publishing this report.

CASE REPORT

An 85-year-old woman was admitted to our surgery outpatient department with intermittent neck pain, gradually progressive weakness in the left arm, and numbness in the left hand for about 4 years. She used analgesic medication occasionally to relieve her pain. Her medical and family history was unremarkable. Furthermore, there was no cervical trauma history. On physical examination, she was normotensive; her distal upper extremity pulses and heart rate were normal. She had pain in her cervical region and her neck movements were restricted in all directions. Her neck pain along the base of the neck was radiated to the superior aspect of the shoulder and posteriorly scapula. Neurological examination revealed hypoesthesia at the lateral side of left upper extremity(C5-C7 dermatomes), and fine finger movement of the left hand was moderately impaired. Her left upper extremity motor examination was left shoulder abduction 3/5, left elbow extension 4/5, left elbow flexion 3/5, left wrist flexion and extension 4/5 in strength.

There were no motor and sensory abnormalities of other extremities. No pyramidal tract signs and cranial nerve deficits were observed. Deep tendon reflexes were bilaterally normoactive. No pathological reflexes were detected. Speech and memory were intact, and also laboratory tests including complete blood count, renal and liver functions, erythrocyte sedimentation rate, C-reactive protein, rheumatoid factor were within normal limits. Hematological test for infectious agents including HIV and hepatitis was negative. Serum protein electrophoresis results which are used to diagnose some diseases such as multiple myeloma was normal in our patient.

Initial cervical MRI without contrast on 3T showed a large flow void structure in intradural space at the C3-C4 level. The left C3-C4 neural foramina compressed the left C4 nerve root by this flow void structure. (Figure 1) On T2 weighted images, there was no hyperintense signal in the spinal cord, suggestive of edema or myelomalacia. To evaluate her bony and vascular details, CT
and CTA were performed. CT scan revealed a dominant left vertebral artery looped medially and indented into the left side of the spinal cord (Figure 2). Additionally, this scan showed an anomalous course of the V2 segment of left vertebral artery. Also, there was an enlarged intervertebral foramen and erosion of the adjacent bone at this level. Another incidental finding on the opposite side was right vertebral artery tortuosity without foraminal widening. No pathological conditions were observed in the other neural foramina.

The patient had degenerative changes, but we did not identify any congenital bony and soft tissue abnormality, such as Klippel Feil syndrome.

Surgical intervention was recommended and all surgical complications were explained to the patient by the neurosurgeon; but she refused operation due to her old age.

**DISCUSSION**

The vertebral artery (VA) is divided into four segments classically. The first three segments are extracranial (V1, V2, V3) and the last is intracranial (V4). Many vertebral artery variations in the origin of VA and its entry into the transverse foramen have been reported in the literature. However, they are usually found incidentally without clinical symptoms. We could not find any other case involving VA loop formation with spinal cord indentation at the C3-C4 level in the literature to the best of our knowledge.

Cervical radiculopathy is the clinical condition resulting from irritation, damage or compression...
of the nerve root in the cervical region of the spine. Patients with radiculopathy typically present with neck pain, arm pain, or both. A variety of conditions or injuries can cause radiculopathy. Unusual causes of cervical radiculopathy include vascular, congenital, metabolic and neoplastic conditions. In the literature, vertebral artery loop formation is an infrequent cause of radiculopathy.\(^4\)

Neural foramina widening is usually caused by benign lesions, commonly neurofibroma and schwannoma. However, rare lesions, including vertebral artery loop formation can also cause this condition. VA loop formation and other vascular anomalies, such as aneurysms and arteriovenous malformation, display a signal void on T1 and T2 weighted images. MRA or CTA should be performed in order to make the differential diagnosis of vascular pathology. Using MRA, the vertebral artery can be visualized clearly and the existence of loop formation can be determined.\(^5\)

VA loop formation was the first described by Hadley in 21 cadavers. In four of these cadavers there was bone erosion of the cervical vertebra as in our case.\(^6\) Therefore, in our opinion, it is possible that chronic pulsatile compression resulted in focal bone erosion.

VA loop formation is usually detected incidentally during cervical trauma or pain investigation. Symptomatic VA loop is very rarely and the symptoms are caused by adjacent nerve root compression. Paksoy et al. reported that the incidence of symptomatic VA loop formation was 7.51% in their 173 patients.\(^7\)

The pathophysiology of this condition is not fully understood. Öga et al. reported that VA loop formation may be associated with spondylotic changes.\(^8\) Sakaida et al. suggested that a correlation between the elongation of VA and severity of disc space narrowing.\(^9\) Another study hypothesized that hemodynamic conditions such as hypertension and atherosclerosis might be a cause of VA loop formation. In several study cervical trauma was blamed for an underlying mechanism.\(^10\) But there was no history of cervical trauma or hypertension in our case. She had only spondylotic changes in the bony structure.

Cervical myelopathy refers to compression on the cervical spinal cord. Any space-occupying lesion within the spinal canal with the potential to compress the spinal cord can cause cervical myelopathy. In our case, there were no severe cervical myelopathy symptoms despite indentation on the left side of the spinal cord. She had only weakness in the left arm and numbness in the left hand.

In conclusion, VA anomalous course and loop formation may cause radiculopathy due to compression of the nerve root. Therefore, this anomaly should be considered in the differential diagnosis of cervical radiculopathy. Surgical procedures could be an effective option to decompress the spinal cord and nerve roots. It is important to keep in mind the possibility of this rare anatomical anomaly to avoid serious surgical complications. For this reason, the course of the VA should be confirmed using radiological modalities before any surgical intervention.

**DISCLOSURE**

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**REFERENCES**