Cervical Radiculopathy Caused by Vertebral Artery Loop Formation: A Case Report and Review of the Literature

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Vertebral artery loop formation causing encroachment on cervical neural foramen and canal is a rare cause of cervical radiculopathy. We report a case of 61-year-old woman with vertebral artery loop formation who presented with right shoulder pain radiating to her arm for 2 years. Plain radiograph and computed tomography scan revealed widening of the right intervertebral foramen at the C5-6 level. Magnetic resonance imaging and angiogram confirmed the vertebral artery loop formation compressing the right C6 nerve root. We had considered microdecompressive surgery, but the patient’s symptoms resolved after conservative management. Clinician should keep in mind that vertebral artery loop formation is one of important causes of cervical radiculopathy. Vertebral artery should be visualized using magnetic resonance angiography in suspected case.

KEY WORDS: Vertebral artery · Vertebral artery loop · Vascular compression · Vascular anomaly · Cervical radiculopathy · Magnetic resonance angiography.

INTRODUCTION

The common causes of cervical radiculopathy are supposed to be cervical disc herniation or cervical spondylosis, but it can also be developed by other infrequent causes such as tumors, cysts, vascular malformations and congenital absence of the pedicle. Vertebral artery loop formation (VALF) is one of those infrequent causes which can erode the cervical neural foramen and compress the cervical nerve root causing cervical radiculopathy. We present a case report of patient with a VALF causing widening of the right C5-6 intervertebral foramen and compressing right C6 root leading to radiculopathy.

CASE REPORT

A 61-year-old woman presented with right shoulder pain radiating to her arm and intermittent headaches for 2 years. There was no history of any cervical spine injury or family history of neurofibromatosis. Her symptoms were aggravated by the Spurling maneuver and partially relieved on shoulder abduction. No neurologic deficit was detectable upon clinical examination. The symptom was prevalent concordant to C6 dermatomal innervation. Initial cervical radiograph (Fig. 1) and axial computed tomography (CT) scan (Fig. 2) showed

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Fig. 1. Left posterior oblique cervical radiograph shows a widening of the intervertebral foramen on the right side at C5-6 caused by erosion of the superior margin of the right C6 pedicle, inferior margin of the right C6 pedicle, and the posterolateral portion of the C6 body (arrow).
widenng of the right C5-6 intervertebral foramen and erosion of the adjacent bony structures. T2-weighted axial and sagittal magnetic resonance (MR) images showed a signal-void vascular structure in the right C5-6 neural foramen compressing the right C6 nerve root (Fig. 3). There was another incidental finding of vertebral artery (VA) migration into the left lateral portion of C4 vertebral body (Fig. 4). A MR angiography (MRA) showed a right VALF migrated into the right C5-6 neural foramen and a left VALF on the left lateral side of the C4 body (Fig. 5). We considered microdecompressive surgery to relieve her symptoms, but it was resolved after conservative management.

**DISCUSSION**

Infrequent causes of cervical radiculopathy include congenital, cystic, metabolic, neoplastic, and vascular conditions. Among these causes, VALF can cause bony erosion, neurovascular compression, or verteobasilar insufficiency. Hadley first described the bony erosion of cervical vertebrae caused by VALF in four of the 21 cadavers. Paksoy et al. reported the incidence of the VALF as 7.51% in their 173 patients, presenting with symptoms of cervicobrachial pain. Even though the real incidence of this abnormality is very low, it is not negligible. If we have mistaken his symptom as common radiculopathy due to disc herniation or spondylosis, it may have led to a possible VA injury casualty during the surgery.

Sato et al. found 10 cases of anomalous VA course, in their 1669 angiograms in 1436 cases, at the C1-2 level. In these cases, angiography depicted the VA not passing through the transverse foramen of the atlas, but running medioposterior to it, and MR images and CT findings demonstrated the VA running intradurally between the atlas and axis. Bruene et al. reported 2% rate of incidence of VALF in their 250 cases of the V2 segment (transversey segment) of the VA. Although there are several reports of it as a rare cause of cervical radiculopathy, they do not indicate the incidence of this anomaly.

The mechanism of VALF is not clear. Oga et al. reported that VALF is developed in association with cervical spondylotic changes. Hemodynamic stress such as hypertension and atherosclerosis were suggested as other possible causes. Sakaia et al. hypothesized that the narrowing of the disc space can cause the elongation of the VA, leading to the VALF. High pulsatile arterial pressure causes migration of
VA and erosion of the adjacent bony structures. With this hypothesis, they also further suggested that the radiculopathy caused by the VALF can recur after the decompressive surgery. Some authors reported cases that cervical trauma might be a cause of VALF. But there were no history of cervical trauma or hypertension in our case. The patient had only cervical spondylosis (disc space narrowing).

In plain radiograph, VALF may show erosion of adjacent bony structures with sharp and sclerotic margins induced by high pulsatile arterial pressure. A common radiographic finding of this lesion is enlargement of the affected intervertebral neural foramen. CT may show VALF as a mass enlarging the intervertebral foramen or another adjacent bony structure. It can be more prominent in intravenous contrast-enhanced CT as a hypodense mass. In MR image, VALF or other vascular malformation displays a signal void. If there is any possibility of VALF or other vascular malformation in CT or MR image, MRA or angiography should be performed to differentiate these vascular lesions. MRA is a non-invasive procedure performed without any contrast and having no risk due to ionizing radiation. Thus, MRA is useful for evaluating VALF. There was one report about the usefulness of multidisc CT angiography compared with MRA and angiography, in demonstrating VALF. They reported that it is less expensive than MRA, safer than angiography, and can show the exact relation between the vertebral artery and the spine.

Various symptoms can occur depending on the level of the anomaly. If the anomalies are at higher levels of the VA, the clinical presentation will be of dysphagia, glossopharyngeal neuralgia, Horner’s syndrome, neurogenic hypertension, occipital neuralgia, sensation of pharyngeal mass, and spasmodic torticollis. The anomalies at the lower levels can cause symptoms of cervicobrachial neuralgia. Cervicobrachial neuralgia produced by vascular compression presents with paraesthesia and dysaesthesia of the fingers without a triggering factor, the lack of nocturnal symptoms, and the rarity of neurologic deficits. Paksoy et al. reported that hypertension could be a triggering factor in one case. VALF may also be a rare cause of verteobasilar insufficiency.

Among the VALF reported in the literature, the authors briefly reviewed the radiologic features for the 50 anomalies in 40 patients who presented with symptoms. The symptomatic cervical levels involved with this anomaly were C1-C2, C3-C4, C4-C5, 10 at C5-6, and 8 at C6-C7 level; 25 anomalies were on the left side, 19 were on the right, and 3 were bilateral. Six patients had VALF at two levels, while one patient had the anomaly at three levels. Consequently, this anomaly could occur at any cervical level along the vertebral artery, but the actual cervical levels involved with this anomaly that produce symptomatic were more frequent at the mid-cervical like C4-5 or C5-6 as in our case.

Surgical procedures reported in the literature are microvascular decompression through the anterolateral or posterior approach, foraminotomy with sectioning of the compressed rootlet, and vascular reconstruction through the anterolateral approach. Although surgical treatment is an effective treatment option, several cases successfully treated conservatively only, were reported as our case.

The projected reason for this symptomatic improvement only with conservative care might be the reduced local pulsatile arterial pressure in the loop formation area, spontaneous resolution of impingement of the loop away from the neural tissue, or the lesion itself was originally asymptomatic one. Therefore, it can be inferred that the reason for spontaneous resolution as in our case would be more related to the latter, asymptomatic one because no specific intervention has been performed to either decrease the size or pressure of the anomalous loop.

CONCLUSION

VALF and subsequent erosion of the intervertebral neural foramen can produce cervical radiculopathy. Although it is a rare cause of cervical radiculopathy, clinicians should keep in mind that it can be an important cause of cervical radiculopathy, especially when the plain radiograph of the cervical spine shows the enlargement of the intervertebral foramen. Thus, if overlooked, a serious complication like VA injury may occur during surgery.

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References

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