MR Findings Of Vertebral AVM
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Citation

Abstract
Vertebral AVM’s are a rare entity. They are mostly commonly encountered in 2nd part of vertebral artery. Mostly they have old history of penetrating trauma. AVM’s due to blunt injury or congenital cause is very rare. They may be asymptomatic or cause symptoms due to compression to the surrounding structures or the spinal cord. Color doppler ultrasound findings show high velocity, low resistance monophasic flow and also demonstrates feeders and draining vessels. Contrast MR Angiography of neck-Time of flight(TOF) images show the whole extent, origin and its relations with the spinal cord better along with feeders and draining vessels. It is drained by epidural veins and paraspinal veins.

INTRODUCTION
Vertebral AVM/AVF is a rare lesion consisting of an abnormal shunt and communication between the extra cranial vertebral artery (or one of its muscular or radicular branches) and neighboring veins.[2]

We describe an interesting case with AVM.

CASE REPORT
An 18 year-old female presented with a 1-year history of progressive increase in swelling on left side of the neck. She has history of tingling sensation on left upper limb since 6months. She gives history of blunt injury to the neck and upper limb before 10 years. The patient’s medical history was otherwise unremarkable. On neurological examination, all four limbs show normal sensory and motor function except for tingling sensation of the patient.

Colour Doppler and Contrast Magnetic resonance (MR) imaging of the neck was performed.

Figure 1
Fig.1- Vertebral AVM. Grey scale ultrasound image with 10MHz probe of neck shows cystic fluid filled anechoic structures adjacent to the common carotid artery.
Figure 2
Fig. 2 – Vertebral AVM. Color Doppler ultrasound image shows high grade vascularity in the above mentioned cystic spaces in the neck.

Figure 3
Fig. 3 – Vertebral AVM. Color doppler ultrasound shows High grade velocity, low resistance, monophasic spectrum in the vascular structures.

Figure 4
Fig. 4 – Vertebral AVM. Color doppler ultrasound shows Feeder from Common carotid artery showing high grade flow also with good diastolic component.

Figure 5
Fig. 5 – Vertebral AVM. Color doppler ultrasound shows High grade vascularity with pulsatility with absent cardiac pressure waves in left Internal jugular vein.
**Figure 6**

Fig. 6 – Noncontrast MR T2 coronal [TE-80, TR-4170] shows hypointense, dilated, tortuous left vertebral artery forming a thin walled blood filled cavity.

**Figure 7**

Fig. 7 - Noncontrast MR T2 coronal [TE-80, TR-4170] shows dilated epidural veins compressing and pushing the spinal cord towards right side which is caused by drainage of AVM of left vertebral artery.
Figure 8
Fig. 8 – Non contrast T2 Sagittal [TE-82, TR-4310] shows dilated, tortuous left vertebral artery AVM which shows hypointense blood.

Figure 9
Fig. 9 – Non contrast MR PD Axial [TE-32, TR-3500] shows left vertebral artery AVM with arterial feeders communicating with epidural veins which have become dilated.
**Figure 10**
Fig. 10 - Non contrast MR PD Axial [TE-32, TR-3500] shows left vertebral artery AVM with arterial feeders communicating with epidural veins which have become dilated.

**Figure 11**
Fig. 11 – Contrast MR Angiography TOF [TE-7, TR-30] shows enhancing non- thrombosed AVM with arterial feeders communicating with epidural veins through the intervertebral foramen which have become dilated causing compression of spinal cord which is pushed towards right side.
Figure 12
Fig. 12 - Contrast MR Angiography T2 FLASH 3D Axial [TE-1.49, TR-3.9] shows enhancing non-thrombosed AVM with arterial feeders communicating with epidural veins through the intervertebral foramen which have become dilated causing compression of spinal cord which is pushed towards right side.

Figure 13
Fig. 13 - Contrast MR Angiography T2 FLASH 3D Axial [TE-1.49, TR-3.9] shows dilated enhancing left paraspinal veins and epidural veins carrying the drainage of the AVM.

Figure 14
Fig. 14 - Contrast MR Angiography T2 FLASH 3D Coronal [TE-1.49, TR-3.9] shows enhancing non-thrombosed tortuous cavitary left vertebral AVM with arterial feeders communicating with epidural veins through the intervertebral foramen which have become dilated causing compression of spinal cord which is pushed towards right side.
Figure 15
Fig. 15 – Maximum intensity projection image of Contrast MR Angiography T2 FLASH 3D Coronal [TE-9.3, TR-27] shows the dilated enhancing AVM in the neck along the course of vertebral artery.

Figure 16
Fig. 16 - Maximum intensity projection image of Contrast MR Angiography TOF [TE-7, TR-30] shows the dilated enhancing AVM in the neck along the course of vertebral artery

FINDINGS
Ultrasound and Colour Doppler imaging showed thin walled, dilated, elongated cavity showing high-grade vascularity. Spectrum showed high velocity, low resistance monophasic flow. There is also a small feeder with a width of 2mm from the common carotid artery supplying the feeder, which is confirmed on MR imaging. Internal jugular vein showed pulsatile high velocity, low resistance flow and is arterialized with absence of normal pressure waves. PSV measured is 1.28m/s.

MR imaging showed thin walled, dilated, tortuous 2nd part of vertebral artery up to the level of inferior end of medulla oblongata communicating with the ventral epidural veins and left paraspinal veins. Dilated epidural veins extend from the level of C5 to C1 and decrease the epidural space causing compression over spinal cord and its shift towards right. There is no bone erosion seen. Maximum Intensity Projection images show malformation in its whole extent in the neck.
DIAGNOSIS: Left Vertebral artery arteriovenous malformation (AVM).

DISCUSSION
Anatomy: Vertebral artery, one on each side of the body enter deep to the transverse process of the level of the 6th cervical vertebrae (C6). It then proceeds superiorly, in the transverse foramen (foramen transversarium) of each cervical vertebra until C1. Inside the skull, the two vertebral arteries join up to form the basilar artery at the base of the medulla oblongata. The vertebral artery may be divided into four parts:

First part: The first part runs upward and backward between the Longus colli and the Scaleneus anterior. In front of it are the internal jugular and vertebral veins, and it is crossed by the inferior thyroid artery; the left vertebral is crossed by the thoracic duct also. Behind it are the transverse process of the seventh cervical vertebra, the sympathetic trunk and its inferior cervical ganglion.

Second part: The second part runs upward through the foramina in the transverse processes of the upper six cervical vertebrae and is surrounded by branches from the inferior cervical sympathetic ganglion and by a plexus of veins which unite to form the vertebral vein at the lower part of the neck.

Third part: The third part issues from the latter foramen on the medial side of the Rectus capitis lateralis, and curves backward behind the superior articular process of the atlas, the anterior ramus of the first cervical nerve being on its medial side. This part of the artery is covered by the Semispinalis capitis and is contained in the suboccipital triangle—a triangular space bounded by the Rectus capitis posterior major, the Obliquus superior, and the Obliquus inferior.

Fourth part: The fourth part pierces the dura mater and inclines medial ward to the front of the medulla oblongata; it is placed between the hypoglossal nerve and the anterior root of the first cervical nerve and beneath the first digitation of the ligamentum denticulatum.

Predisposing factors: The most common cause of vertebral AVF is penetrating neck injury, usually a knife or gunshot wound; however, blunt cervical trauma is implicated in some cases. Iatrogenic forms of trauma have also caused vertebral AVF/AVMs; these include direct percutaneous puncture of a carotid or vertebral artery for diagnostic angiography, vertebral artery injury during insertion of central venous catheters, and complications of cervical interbody fusion. Many vertebral AVFs are spontaneous, likely related to congenital abnormalities of the arterial wall. Diseases associated with this lesion include fibromuscular dysplasia (increased vascular fragility) and neurofibromatosis (mesodermal dysplasia). In cases of spontaneous vertebral AVF, the involved feeding artery is usually markedly enlarged, presumably due to a longstanding high flow state. The long intraforaminal course and posterior location of the vertebral artery account for its rare involvement in penetrating neck injuries. Injuries to the first segment (V1) of the vertebral artery (extending from the origin of the artery to its entry into the transverse foramen) usually involve other major structures and are associated with high immediate morbidity. Most traumatic vertebral AVF/AVMs involve the second (intraforaminal) segment (V2) of the vertebral artery. Spontaneous fistulas typically affect the third segment (V3) (extending from the point at which the vertebral artery exits the transverse foramen of C2 to the point at which it enters the foramen magnum). Vertebral AVF/AVMs may also develop an extensive collateral blood supply.

Symptoms: Clinical symptoms produced by vertebral AVF/AVMs are related to the flow rate of the shunt, the chronicity of the lesion and the venous drainage pattern. Vertebral AVF/AVMs are frequently asymptomatic and discovered incidentally as a bruit in the cervical region. Tinnitus, a common symptom, caused by turbulent flow and probably relates to ascending venous drainage to the jugular foramen. Serious neurologic symptoms are uncommon. Brain stem signs (eg, vertigo, vertebrobasilar insufficiency) are probably due to arterial steal. Spinal cord dysfunction can be the result of direct cord compression by dilated, pulsatile veins or of spinal cord ischemia due to arterial steal or venous hypertension. Intradural venous drainage of the fistula causes increased pressure in and engorgement of the pial veins of the spinal cord; the increased pressure is transmitted to the intrinsic veins of the cord, reducing the intramedullary arteriovenous pressure gradient. This in turn leads to decreased tissue perfusion and hypoxia of the cord. Additional consequences of chronic venous hypertension include cord edema, disruption of the blood-cord barrier, and progressive myelopathy. The dilated epidural draining veins of a vertebral AVF can cause cervical radiculopathy by compressing the exiting cervical nerve roots. Traumatic vertebral AVF/AVMs can manifest as massive hemorrhage, expanding hematoma, pseudoaneurysm, or airway
obstruction. [2]

**DIAGNOSIS**

**IMAGING**

- Conventional angiography is the definitive radiological examination for vertebral AVM, the diagnosis can usually be made noninvasively with MR imaging and MR angiography. Abnormalities that can be identified with these modalities include markedly enlarged epidural or paraspinal veins, enlarged extra spinal or vertebral arteries proximal to the fistula, and spinal cord or nerve root compression. Spinal cord edema may be seen if venous hypertension is present in the coronal venous plexus and medullary veins. [2]

- Color Doppler Ultrasonography reflects hemodynamic state of the AVM. In an AVM, the diastolic flow velocity is increased because of the depression of peripheral vascular resistance: waveform with a low pulsatility index is produced. The draining vein show increased flow velocity and pulsatile waveform. [3]

- MR Angiography is the imaging of choice nowadays. The mainstay of MRA has been time of flight or phased contrast angiography. There are two forms of time of flight: two dimensional (2D TOF) and three dimensional (3D TOF). 2D TOF is most commonly used for carotid neck imaging due to its superior flow-related enhancement. TOF imaging is acquired in a much shorter time than PC imaging, allowing more time for acquisitions and better resolution. PC imaging can have better background suppression, allowing better depiction of slower flow and smaller vessels. The images are acquired without a breath hold and with manual injection of contrast. The 3D images obtained during the arterial phase of the bolus passage are identified by means of visual inspection and the data set subtracted from the pre-contrast data set after the background signal of fat is eliminated. MIP images are then generated. Timing from the start of the acquisition to the arrival of the contrast bolus is critical in contrast MRA. It can be acquired using a timing bolus or alternatively using an automated bolus detection and scan-triggering scheme such as SMARTPREP (GE Medical Systems, Milwaukee, WI, USA). [4]

Differential Diagnosis: - Vertebral AVF/AVM should be distinguished from other vascular malformations of the spinal cord such as spinal Dural AVM, spinal cord arteriovenous malformation, spinal cord (perimedullary) AVM, and cavernous malformation. In particular, vertebral AVF/AVM can be readily distinguished from the more common spinal Dural AVM, which is characterized by a thoracolumbar location and dilated intradural veins. Cervical Dural AVF is rare. [2]

Treatment: - Endovascular treatment of vertebral AVM is the simplest and most reliable method of occlusion. This is accomplished with detachable balloons, although coils, particles, liquid adhesives, and autologous clot have also been used.

Complications of endovascular treatment include ischemic events (eg, spasm, dissection, emboli) and reactions to contrast material. Neurological complications may also arise after abrupt closure of a long-standing fistula; this is due to reestablishment of normal arterial flow to a region that is chronically ischemic and unable to regulate cerebral blood flow (normal perfusion pressure breakthrough). [2]

**References**

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