



Cervical Nerve Root Cavernoma – Case Report and Literature Review

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Abstract

Spinal cavernous angiomas are uncommon vascular malformations in the spine accounting for 5%-12% of all spinal vascular lesions. When present in an intradural extra-medullary location, these usually present with radicular pain and neurological deficits due to mass effect (myelopathy). Herein, we present an atypical presentation of cavernous angioma in a 54-year-old man with tinnitus, headache and sensorineural hearing loss. We have also reviewed 51 cases of intradural extramedullary cavernous angiomas including our case with respect to demographic and clinical profile. A 54-year-old man presented with tinnitus in the left ear and occipital headache with neck pain and slight weakness of left-hand grip along with atrophy of thenar muscles. His pure tone audiometry (PTA) test revealed mild left sensorineural hearing loss. Magnetic resonance imaging (MRI) of cervical spine showed T2WI heterogeneously hyperintense left intradural extramedullary lesion at C7 vertebral body level. It was avidly enhancing with contrast. The patient underwent C7 laminectomy with a midline durotomy and complete excision of the lesion under neuromonitoring with sacrifice of the C8 sensory root. His symptoms improved following the surgery. The diagnosis of a cavernoma in an unusual location in the presence of cranial nerve dysfunction needs a high degree of diagnostic suspicion. Most of these cavernomas have a nerve root origin or attachment. The optimal treatment is microsurgical *en bloc* resection which leads to an effective resolution of both the symptoms.

Keywords: Cavernous hemangiomas, Cavernous angioma, Spinal nerve root

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Introduction

Vascular lesions constitute around 6%-7% of all spinal intradural tumours.¹ Cavernous angiomas or cavernomas are uncommon vascular malformations in the spinal cord, accounting for just 5%-12% of all spinal vascular lesions.² The cavernous malformations occur within vertebral bodies in most cases with only 3%-5% being intracanalicular.³ The most common location for intracanalicular cavernomas is the thoracic epidural space. They are rarely found in intramedullary, intradural extramedullary, the foraminal and extraforaminal locations.⁴ Intradural extramedullary or nerve root cavernomas are extremely rare, with their description mostly limited to few case reports. Usually, nerve root cavernomas present with localised neck or back pain, with or without features of radiculopathy or myelopathy.

Herein, we describe a case of cervical nerve root cavernoma with atypical presentation operated at our centre. We also present a review of existing literature with emphasis on the challenges in the management of this rare entity.

Case Report

A 54-years-old man, presented with complaints of tinnitus in his left ear, with intermittent suboccipital and neck pain for one month. Tinnitus increased in duration and frequency over the last 20 days. The headache was also associated with two episodes of vomiting. The patient also had weakness of left-hand grip along with atrophy of thenar muscles. The sensory examination and deep tendon reflexes were normal. There were no signs of myelopathy.

Pure Tone Audiometry examination revealed mild left sensorineural hearing loss. Contrast magnetic resonance imaging (MRI) of the Brain was normal. Contrast MRI of the cervical spine showed an ovoid heterogeneous, intradural extra-medullary soft tissue lesion, measuring ~14×7×7 mm, ventrolateral to the left hemicord at C7 vertebral body level (Figure 1). The lesion was isointense on T1 and iso to hyperintense on T2 weighted image and was avidly enhancing with contrast and showed significant blooming on the gradient sequences. There was no extension through the neural foramina. The lesion caused mass effect on left hemicord.



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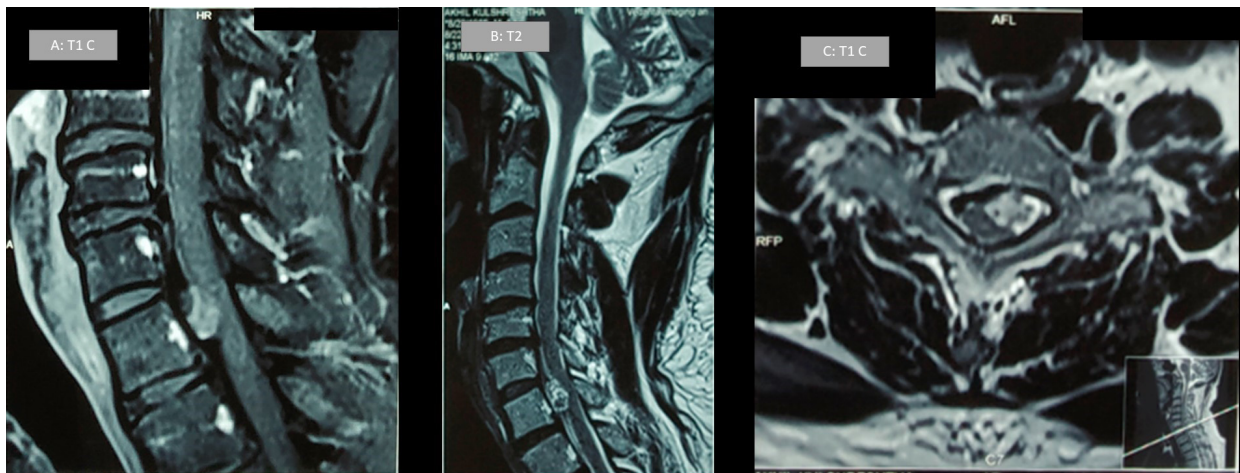


Figure 1. MRI images of the Patient With left C8 Nerve Root Cavernoma. A: T1 contrast image showing avid enhancement on contrast image with posteromedial displacement of the cervical cord. B: T2 weighted image showing heterogeneously hyperintense lesion with classical popcorn appearance. C: T1 C Axial showing contrast enhancing lesion with mass effect on the spinal cord

The patient underwent C7 laminectomy and a linear midline durotomy under neuromonitoring. After gentle retraction of the cord, a multilobulated, pinkish, well defined, lesion was seen densely adherent to the C8 sensory nerve root. Grossly, it had the classic whorled, mulberry like appearance suggestive of a cavernoma (Figure 2). The lesion was completely free of the cord or the dura. A complete excision of the lesion was performed. The C8 sensory nerve root had to be sacrificed as the lesion was originating from the nerve root and was densely adherent. The patient recovered well following surgery with mild improvement in handgrip. His headache improved significantly but he still had persisting mild tinnitus. There were no procedure related complications.

On histopathological examination, the lesion was composed of variably sized, dilated vascular channels with hyalinized walls lined by a single layer of endothelium. There was no muscular layer or a defined arterial vessel wall. Areas of small calcification and thrombus within the lumen were also noted. The immunohistochemistry was positive for CD31, CD34 (markers of endothelial cells) and smooth muscle actin (SMA). A low mitotic index (Ki-67 index of 2%) and the above findings were consistent with the diagnosis of a cavernous haemangioma (Figure 3).

At six months follow-up, the patient remained symptom free along with resolution of tinnitus.

Discussion

Cavernous angiomas are hamartomatous vascular lesions with dilated, thin-walled sinusoidal vascular spaces lacking intervening connective tissue. Intradural extramedullary cavernomas are rare entities, with origin from the vasculature of nerve roots, the inner surface of the dura, or the pial surface of the spinal cord⁵. In our review of the available literature, we found 50 similar reported cases of intradural extramedullary cavernomas.^{5,6-49} Based on analysis of these cases including our case (n=51,

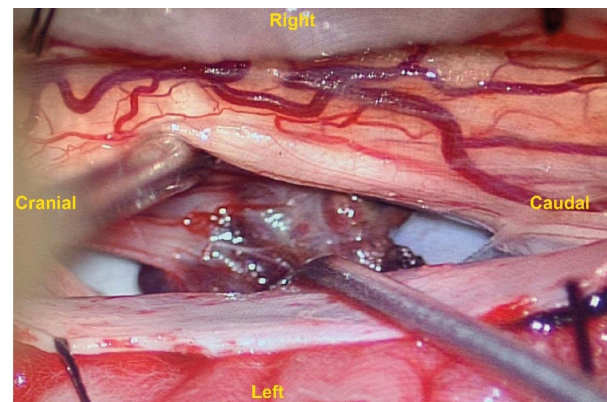


Figure 2. Multilobulated Vascular Lesion Seen Anterior to, and After Medially Displacing the Cord. The lesion is seen densely adherent to the ventral nerve root

Table 1), the mean age at presentation was 53.19 years (range 18 to 79 years). There were 33 men and 18 women. Most cavernomas were located in lumbar cord 27 (53%), followed by thoracic 16 (31%) and cervical spine 8 (16%). Most (n=47, 92.16%) had an attachment or took origin from the spinal nerve root.

The presenting symptoms of cavernoma can be broadly grouped into local or axial pain, radicular, myelopathy and symptoms due to recurrent bleeds (subarachnoid hemorrhage, hydrocephalus, cranial nerve dysfunction). In our case, the compression was significant, but the patient was asymptomatic. The case presentation was unique as the primary symptoms of headache and tinnitus were not explainable by the C8 root lesion.

The lesion originated from the nerve root similar to neurofibromas and schwannoma which are the most probable differential radiological diagnoses. The other differential diagnosis in this case was metastatic lesion with carcinomatous meningitis. A tuberculoma with tubercular meningitis (especially in endemic regions) can also have symptoms of radicular/myelopathic

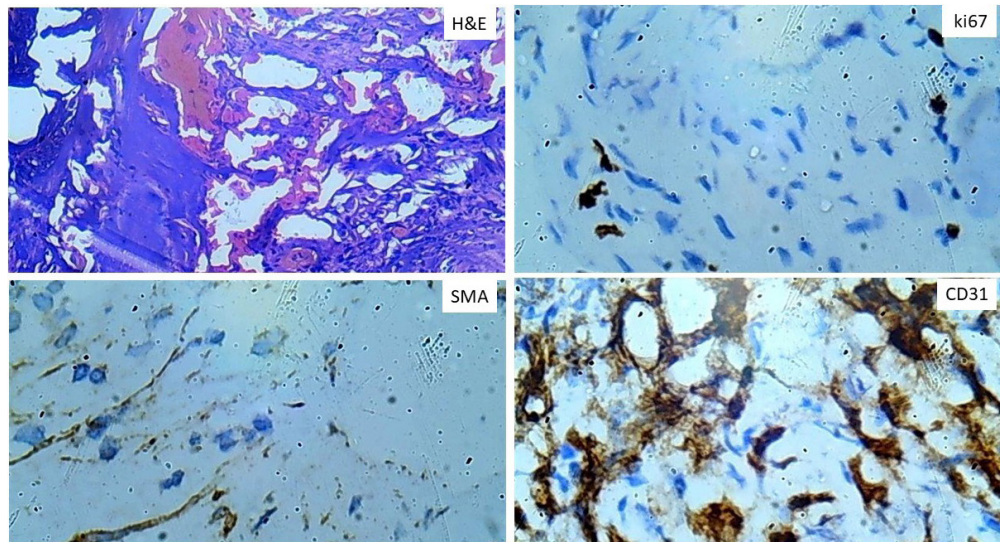


Figure 3. Histopathological Examination Showing Variably Sized, Dilated Vascular Channels With Hyalinized Walls Lined by a Single Layer of Endothelium (H&E) With Immunohistochemistry Positive for CD31 Cells and SMA (Smooth Muscle Actin) and a Low Mitotic Index (Ki-67 index of 2%)

Table 1. Demographic and Clinico-radiological Profile of the Patients in Previously Described Reports

Study	Year	Age	Gender	Location	Vertebral Levels Involved	Root Attachment	Presenting Symptoms
Hirsch et al ⁶	1965	20	M	Lumbar	L2-3	Yes	SAH, sensory deficit, motor deficit, sphincter dysfunction
Pansini et al ⁷	1966	46	M	Lumbar	L2	Yes	Low back pain, radicular pain, sensory deficit, motor deficit, sphincter dysfunction
Heimberger et al ⁸	1982	24	M	Thoracic	T2-3	Yes	SAH
Ueda et al ⁹	1987	28	M	Lumbar	L1-2	Yes	SAH, back pain
Pagni et al ¹⁰	1990	46	M	Lumbar	T 12-L1	Yes	Low back pain, radicular pain
Ramos et al ¹¹	1990	67	F	Lumbar	L3	Yes	Hydrocephalus
Mastronardi et al ¹²	1991	49	F	Thoracic	T4	Yes	Sensory deficit, motor deficit
Mori et al ¹³	1991	65	M	Thoracic	T1	Yes	Low back pain, SAH, sensory deficit, motor deficit
Acciarri et al ¹⁴	1992	54	F	Cervical	C2-C3	Yes	Neck pain, SAH
Sharma et al ¹⁵	1992	63	M	Thoracic	T12	Yes	Low back pain, motor deficit, sphincter dysfunction
Bruni et al ¹⁶	1994	28	M	Lumbar	L2	Yes	Low back pain, SAH
Cervoni et al ¹⁷	1995	26	M	Lumbar	L1-2	Yes	SAH
Cervoni et al ¹⁷	1995	32	M	Lumbar	L5	Yes	Radicular pain
Padovani et al ¹⁴	1997	54	F	Cervical	C2-C3	Yes	Severe headache with neck pain
Rao et al ¹⁸	1997	60	M	Lumbar	L1-3	Yes	Motor deficit
Zander et al ¹⁹	1998	51	F	Lumbar	L4-5	No	Radicular pain, motor deficit
Duke et al ²⁰	1998	49	F	Lumbar	L4	Yes	Low back pain, sensory deficit, sphincter dysfunction
Holtzman et al ²¹	1999	56	F	Lumbar	S1	Yes	Low-back pain, radicular pain, sensory deficit
Nowak et al ²²	2000	63	F	Thoracic	T12	Yes	Radicular pain, sensory deficit
Roncaroli et al ²³	2000	74	M	Lumbar	L2-3	Yes	Motor Deficit
Shin et al ⁵	2000	66	F	Thoracic	T8-9	No	Lower back pain, motor deficit
Choi et al ²⁴	2001	28	M	Lumbar	L1	Yes	Back pain, radicular pain, sensory deficit, motor deficit
Choi et al ²⁴	2001	52	M	Thoracic	T5-T6	No	Neural claudication, motor deficit
Choi et al ²⁴	2001	51	M	Thoracic	T4-T5	No	Neural claudication, radicular pain, motor deficit, sensory deficit
Nozaki et al ²⁵	2003	51	M	Cervical	C5-6	Yes	Sensory deficit, motor deficit
Falavigna et al ²⁶	2004	44	F	Lumbar	L3-4	Yes	Lower back pain; sensory deficit; sphincter dysfunction
Abdullah et al ²⁷	2004	32	F	Thoracic	T9-10	Yes	Lower back pain, motor deficit
Crispino et al ²⁸	2005	65	M	Thoracic	T1-2	Yes	Motor deficit, upper thoracic back pain

Table 1. Continued

Study	Year	Age	Gender	Location	Vertebral Levels Involved	Root Attachment	Presenting Symptoms
Kim et al ²⁹	2006	59	M	Lumbar	L1-2	Yes	Low back pain, radicular pain, sensory deficit
Er et al ³⁰	2006	67	M	Thoracic	T11-L1	Yes	Back pain, sensory deficit, motor deficit, sphincter dysfunction
Rachinger et al ³¹	2006	56	M	Cervical	C7	Yes	Neck pain, radicular pain
Caroli et al ³²	2007	71	M	Lumbar	L4	Yes	Lower back pain, sensory deficit
Cecchi et al ³³	2007	75	F	Lumbar	L3-4	Yes	Sensory deficit
Miyake et al ³⁴	2007	18	M	Lumbar	L1	Yes	Lower back pain, radicular pain
Kivelev et al ³⁵	2008	44	M	Cervical	C5 C6	Yes	Neck pain, sensory deficit, motor deficit, Brown-Sequard syndrome involving the C5–6 level
Yi et al ³⁶	2009	67	M	Lumbar	L2-3	Yes	Lower back pain, radicular pain
Chun et al ³⁷	2010	74	M	Lumbar	L4	Yes	Radicular pain
Jin et al ³⁸	2011	55	M	Thoracic	T12	Yes	Headache, dizziness, and bilateral sensorineural hearing loss caused by an intracranial superficial hemosiderosis
Nie et al ³⁹	2012	57	F	Lumbar	L1	Yes	Lower back pain; radicular pain
Popescu et al ⁴⁰	2013	60	M	Lumbar	L4	Yes	Lower back pain
Mataliotakis et al ⁴¹	2014	79	M	Lumbar	L2-L3	Yes	Sensory deficit, motor deficit
Katoh et al ⁴²	2014	36	M	Lumbar	L1	No	Progressive hearing impairment, severe headache, bilateral sensorineural hearing impairment, ataxia, hyperreflexia, mild cognitive dysfunction, superficial siderosis, hydrocephalus
Takata et al ⁴³	2014	60	M	Thoracic	T2	Yes	Radicular pain, progressive gait disturbance
Rizzi et al ⁴⁴	2015	49	F	Lumbar	L5-S1	Yes	Low back pain, sensory deficit
Henderson et al ⁴⁵	2016	65	F	Cervical	C6	Yes	Neck pain, radicular pain
Zhu et al ⁴⁶	2016	59	F	Thoracic	T8	Yes	Backache, sensory deficit
Pétillon et al ⁴⁷	2018	76	F	Cervical	C7-C8	Yes	Worsening neck pain
Apostolakis et al ⁴⁸	2018	77	M	Lumbar	L3	Yes	Lower back pain
Ziechmann et al ⁴⁹	2018	55	M	Thoracic	T3-T4	Yes	Radicular pain, back pain
Vicenty et al ⁵⁰	2019	56	M	Thoracic	T2	Yes	Sensory deficit, motor deficit
Present Case	2019	54	M	Cervical	C7	Yes	Tinnitus, sensory neural hearing loss, headache and neck pain

SAH, Subarachnoid haemorrhage.

involvement with hydrocephalus +/- cranial nerve dysfunction.

We hypothesize that these symptoms of headache and cranial nerve dysfunction may be related to recurrent microhaemorrhages within the lesion and in subarachnoid spaces thereby causing impairment in flow of the cerebrospinal fluid. Superficial siderosis, a progressive degenerative disorder can occur due to deposition of hemosiderin along the leptomeninges and has been known to cause neurological dysfunction. The involvement of the VIII cranial nerve is one of the commonest symptoms, with cerebellar and pyramidal signs also occurring frequently. The toxic effects of hemosiderin affects selectively vulnerable neurons and glia in this condition.⁵¹ Jin and colleagues reported a similar case with headache, dizziness and progressive hearing loss due to intracranial superficial hemosiderosis associated with a cavernous angioma in the cauda equina on the level of the T12 vertebral body.³⁸

MRI is the study of choice to detect these lesions and usually the first study done when the initial clinical

presentation is of neural compression. The cavernomas appear heterogeneously iso to hyperintense on T1-weighted images. They can be iso to hyperintense on T2-weighted images with a peripheral rim of hypointensity (prior haemorrhage); gadolinium enhancement is frequent but not necessary.¹² MRI also shows blooming on the gradient recalled echo sequences suggesting presence of hemosiderin from prior haemorrhages.

In cases where the presentation is atypical in form of symptoms of SAH, superficial siderosis and hydrocephalus, the diagnostic workup is not straight forward and the finding of cavernoma may be unexpected. In the latter case, the workup may include lumbar puncture, pure tone audiometry and contrast MRI of brain and spine. Cavernomas are usually not seen during conventional digital subtraction angiography (DSA) and hence DSA is not the imaging modality of choice.

The treatment of choice is microsurgical *en bloc* resection of the lesion. Because of their attachment to the nerve root, it may not always be possible to excise clearly or separate the cavernoma from the nerve root. Hence,

the root may have to be sacrificed. In our case too, the cavernoma was densely adherent to the C8 sensory nerve root and could not be excised without sacrificing the nerve root.

There is no role of endovascular treatment like embolization for cavernomas owing to their cytoarchitecture making them angiographically occult.⁵²

Conclusion

Spinal nerve root cavernomas are extremely rare. They usually present with radicular or myelopathy symptoms due to compression. Repeated rupture or micro-bleeds can cause raised intracranial pressure features along with cranial nerve deficits. The diagnostic workup is not straight forward in these latter cases and the presence of cavernoma may be an unusual finding. The diagnosis of a cavernoma in an unusual location in the presence of cranial nerve dysfunction needs a high degree of diagnostic suspicion. Most of these cavernomas have a nerve root origin or attachment. The optimal treatment is microsurgical *en bloc* resection which leads to an effective resolution of both the symptoms.

Competing Interests

None to be declared.

Ethical Approval

Written informed consent was obtained to report the case.

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