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# Cauda Equina Cavernous Angioma

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## Abstract

Cavernous angiomas are rare vascular lesions, most occurring supratentorially, with spinal cavernomas being even rarer. They have a varied magnetic resonance imaging (MRI) appearance which is usually not diagnostic. A 42-year-old man presented with progressive low backache for the past 5 years and was found to have a mild right ankle weakness. MRI showed an intradural extramedullary lesion at the L1 vertebral body level. Intraoperatively, a dark reddish-blue mulberry-like lesion was found attached to a nerve root that had to be sacrificed during the excision. Histopathology confirmed that it was a cavernous angioma and the patient had no added deficits postoperatively. Cauda equina cavernomas are extremely rare and hence not thought of as a differential preoperatively. They are relatively simple to remove but will mostly need the sacrifice of the adherent nerve root. Most cases do well postoperatively with stable deficits and some improving.

**Keywords**
- cauda equina
- spinal cavernoma
- vascular lesions

## Introduction

Cavernous angiomas are a rare vascular lesion in the central nervous system (CNS) with most occurring supratentorially. They are even rarer in the spine with most occurring in the vertebrae or extradurally.[1] Cauda equina cavernomas are the rarest with less than 50 case reports. They usually present with cauda equina syndrome and have no typical appearance on magnetic resonance imaging (MRI). We present our case of a cavernoma in the cauda equina followed by a review of the literature.

## Case Report

### History and Examination

A 42-year-old man presented with dull aching low back pain for the past 5 years which was progressive, aggravated at night, when lying down (more when turned to the left) with relief on standing or walking. He had difficulty standing on his toes and had a history of slippage of footwear on the right. On examination, he had weakness in plantar flexion of the right foot and dorsiflexion of the right large toe with no sensory deficits. Laboratory findings were unremarkable. MRI (►**Fig. 1**) revealed an intradural extramedullary lesion at the L1 vertebral body level, 2 cm below the conus which was T1 isointense, T2 heterogeneous with mild homogenous enhancement. Under the presumption of being a schwannoma, the patient was taken up for surgical excision of the lesion.

### Surgery

L1 and L2 laminectomy and midline durotomy were done. A $1.9 \times 1.2 \times 1.2$ cm multilobulated, firm, vascular lesion with mulberry-like appearance was identified arising from the nerve root (►**Fig. 2**). Extracapsular delineation of the lesion was done circumferentially. The nerve root cranial and caudal to the lesion was coagulated and cut on either side and the lesion was removed in toto. Water-tight dural closure was done. The patient had a smooth postoperative period with significant resolution of preoperative pain with no fresh deficits.

### Histopathology

Examination under the microscope (►**Fig. 3**) showed dilated cavernous blood vessels with organizing thrombi of varying

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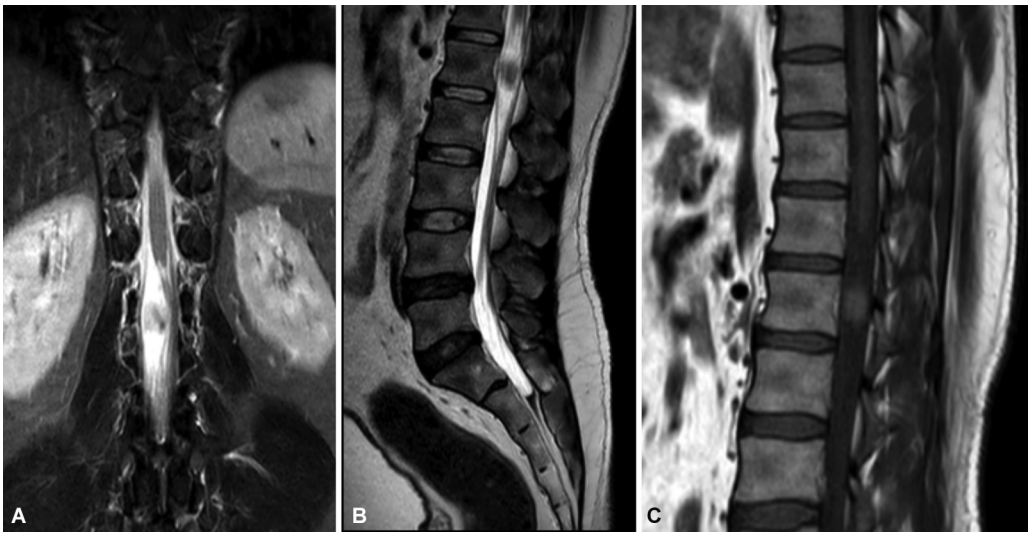


**Fig. 1** (A) T2 coronal showing a heterogeneous lesion. (B) T2 sagittal showing a hypointense intradural lesion at the L1 vertebral body level. (C) Homogeneous mildly enhancing lesion is seen.

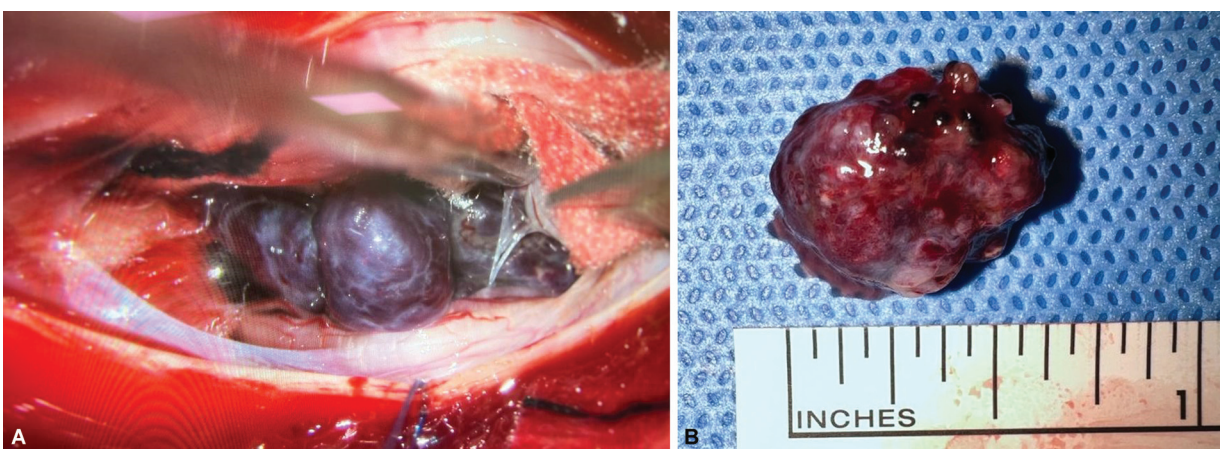


**Fig. 2** (A) Intraoperative photograph after opening the arachnoid. (B) Lesion after en masse excision.

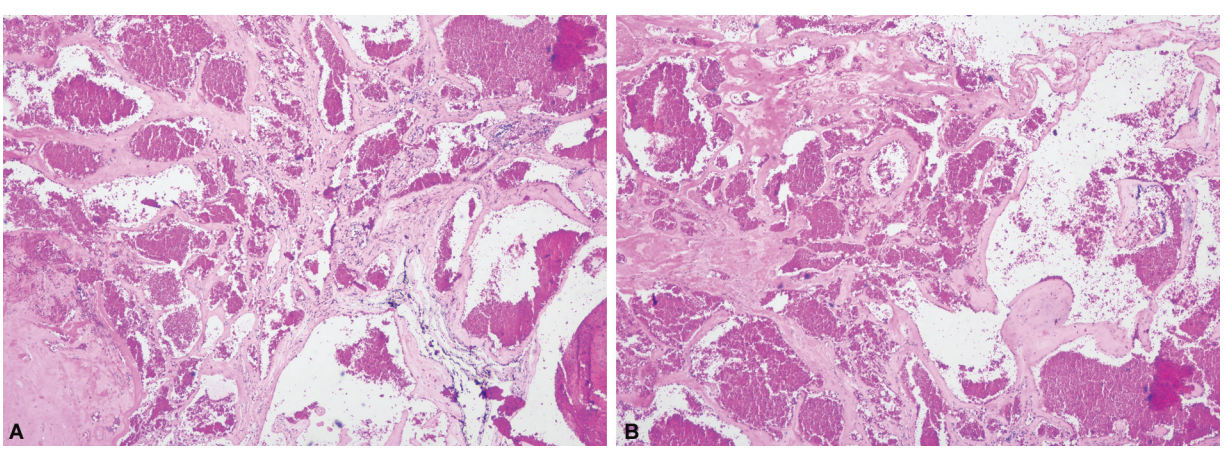


**Fig. 3** (A, B) Microscopic photograph (×100) of hematoxylin and eosin–stained slide of the lesion showing abnormally dilated vascular channels consistent with cavernous angioma.

 

stages surrounded by fibrous stroma and focal areas of calcification which was consistent with cavernous hemangioma.

## Discussion

CNS cavernomas are a rare entity with an overall frequency of 0.41%[2] and spinal cavernomas being even rarer; 6 to 7% of spinal tumors are vascular lesions, of which 5 to 12% are cavernomas.[3] There are less than 50 case reports of cauda equina cavernomas (►**Table 1**). Like cavernomas in other locations, in the cauda equina, there is a male predominance (1.7:1). The median age of diagnosis is 46 years with the youngest case seen in a 13- year-old girl.[4]

The most common presentation was with low back pain with or without radicular lower limb pain with some also presenting with lower limb weakness, paresthesias, or

**Table 1** Review of all previous case reports of cauda equina cavernomas with their demographic details, presentation, and MRI findings

| Author | Year | Age | Sex | Level | Symptom | T1 | T2 | Contrast |
|---|---|---|---|---|---|---|---|---|
| Hirsch et al | 1965 | 20 | M | L2–3 | SAH, pain, weakness, anesthesia, sphincter dysfunction | – | – | – |
| Pansini and Lo | 1966 | 46 | M | L2 | Pain, anesthesia, sphincter/erectile dysfunction | – | – | – |
| Ueda et al | 1987 | 28 | M | L1–2 | SAH, pain | Hyper | – | – |
| Pagni et al | 1990 | 46 | M | D12–L1 | Low back pain + sciatica | – | – | – |
| Ramos et al | 1990 | 67 | F | L3 | Sphincter dysfunction + difficulty in walking | – | – | – |
| Acciarri et al | 1993 | 31 | M | L2 | Radicular pain | – | – | – |
| Bruni et al | 1993 | 28 | M | L2 | SAH, headache, vomiting, neck stiffness | Hyper | – | – |
| Cervoni et al | 1995 | 26 | F | L1–2 | SAH + sudden headache, lumbar pain | – | – | Enhancing |
| | | 32 | M | L5 | Left sciatica + left foot drop + left L5 hypoesthesia | – | – | Enhancing |
| Makino | 1995 | 67 | M | L2 | Headache, unsteady gait | Hyper | Iso | Slightly enhancing |
| Moreno | 1995 | 63 | M | | Low backache, sphincter dysfunction, sensory deficits, knee, and ankle reflexes diminished | Hypo | Hyper | – |
| Rao et al | 1997 | 60 | M | L1–3 | Low backache, paresthesia, paraparesis | – | – | – |
| | | 65 | F | L3–5 | Sudden paraparesis after trivial injury, sensory deficit below L3 | – | – | – |
| | | 35 | F | D11–12 | Sudden-onset paraparesis, sensory deficit below L3 | – | – | – |
| | | 32 | M | L2–3 | Left sciatica | Hyper | Hypo | Enhancing |
| Mastronardi et al | 1997 | 41 | M | L5 | Low backache, left ankle jerk diminished | Hyper slightly | Isointense | |
| Duke et al | 1998 | 49 | F | L4 | Low back pain | Hyper | Hyper | Not enhancing |
| Falavigna et al | 2004 | 44 | F | L4 | Pain, anesthesia, sphincter/erectile dysfunction | – | – | Hetero |
| Crispino | 2005 | 65 | M | T12–L1 | Due to coexisting cervical lesion | Iso | Hyper | Homogeneous |
| Miyake et al | 2007 | 18 | M | L1 | Low back pain radiating to bilateral lower limbs | Iso | Hyper | Nonenhancing |
| Cecchi et al | 2007 | 75 | F | L3–4 | Painful paresthesias | Iso–hypo | Iso–hypo | Heterogeneous |

(*Continued*)

 

**Table 1** (*Continued*)

| Author | Year | Age | Sex | Level | Symptom | T1 | T2 | Contrast |
|---|---|---|---|---|---|---|---|---|
| Caroli et al | 2007 | 71 | M | | Low back pain and left leg radiation, left L4 hypoesthesia | Hyper | Hypo | Nonenhancing |
| Er | 2007 | 67 | M | T11–L1 | Lower limb paresthesia, sphincter and bowel dysfunction, difficulty walking | Hyper | Hypo | Nonenhancing |
| Chun | 2007 | 74 | F | L3 | Neurogenic claudication and lumbar tenderness | – | Hetero | Enhancing |
| Jin et al | 2011 | 55 | M | D12–L2 | Headache, dizziness, SNHL | Iso | Hyper | No CE |
| Nie | 2012 | 57 | F | L1 | Acute low back pain, lower limb weakness, sphincter dysfunction | High | Iso | Hetero |
| Popescu | 2013 | 60 | F | L4 | Low back pain, bilateral radiculopathy, bilateral L4–S1 hypoesthesia | – | Hyper | – |
| Takeshima | 2013 | 44 | M | L2–3 | Low back pain, bilateral sciatica, neck pain | Iso | Hypo | – |
| Yang et al | 2013 | 42 | M | L3 | Low back pain, weakness | Iso–hypo | Iso–hyper | Mild |
| | | 31 | F | L2–3 | SAH, headache, right leg pain | Iso–hypo | Iso–hyper | Heterogeneously |
| | | 28 | F | L1–2 | SAH, bilateral leg pain | Iso–hypo | Iso–hyper | Heterogeneously |
| | | 27 | M | L2 | Low back pain, radiation to right, urinary disturbance | Iso–hyper | Iso–hypo | Mild |
| | | 23 | M | L1–2 | SAH, headache, bilateral leg pain, low backache | Iso–hyper | Iso–hypo | Heterogeneously |
| | | 49 | F | L2–3 | Right leg pain, numbness, urinary disturbance | Iso–hypo | Iso–hyper | Heterogeneously |
| | | 33 | M | L2 | Low back pain, right leg pain, numbness | Iso–hyper | Iso–hypo | Heterogeneously |
| | | 51 | F | L1–2 | Low back pain, bilateral leg pain, and numbness | Iso | Iso–hyper | Mild |
| | | 42 | F | L2–3 | Bilateral leg pain and numbness | Iso | Iso–hyper | Mild |
| | | 59 | M | L3 | Low back pain, left leg pain, and weakness | Iso to hyper | Iso to hypo | Mild |
| Mataliotakis | 2014 | 79 | M | L2–3 | Back pain, neurogenic claudication, paresthesia, both lower limb weakness | Hetero | Iso | – |
| Kumar et al | 2016 | 21 | M | L3–4 | Low backache, ascending sensory loss, weakness | Hypo | – | Enhancing |
| Rizzi et al | 2016 | 49 | F | L2 | Low back pain, bilateral sciatica, paraparesis | Hyper | Hypo | Enhancing |
| Yaltirik et al | 2016 | 13 | F | L1 | Back pain, right side EHL, and dorsiflexion weakness | Hyper | Hyper | Enhancing |
| Katoh et al | 2017 | 36 | M | L1 | Sensorineural hearing loss, headache, and hydrocephalus | – | Iso | Nonenhancing |
| Golnari | 2017 | 60 | M | L2 | Back pain radiating to left leg, headache | | Hypo | Mild CE |
| Apotolakis et al | 2018 | 77 | M | L3 | Low back pain | NA | NA | NA |
| Hughes et al | 2021 | 50 | M | L4 | Bilateral leg weakness, bladder, and bowel dysfunction | Iso | Hyper | Enhancing |

Abbreviations: CE, contrast-enhanced; EHL, extensor hallucis longus; MRI, magnetic resonance imaging; NA, not available; SAH, subarachnoid hemorrhage; SNHL, sensorineural hearing loss.

bowel/bladder dysfunction. Yang et al[5] pointed out the different patterns in the presentation of the patients with some experiencing a slow progressive course, while others had acute onset of symptoms. This may be due to the different pathophysiologies, as repeated bleeds, endothelial proliferation, and neoangiogenesis leading to slow progression while a large subarachnoid hemorrhage or an acute swelling of the lesion due to thrombosis in the acute setting. Subarachnoid hemorrhage was seen in 7 of the 46 patients who we reviewed.

There are two reports of patients presenting with headache and an unsteady gait who were found to have hydrocephalus which resolved after the removal of the lesion. Ramos et al[6] stated that the hydrocephalus was due to hyperviscosity of the cerebrospinal fluid (CSF) which must be either due to increased proteins in the CSF, slowed flow, or inflammation impeding its absorption. Jin et al[7] showed that headache resolved after lesion excision in a patient with superficial siderosis, impressing on the fact that the headache was due to the lesion.

MRI was the modality of choice in most case reports with no typical appearance and a wide range of differences as seen in ►**Table 1**. The presence of a hemosiderin ring may give a clue to the nature of the pathology but may not be specific since the blood products are degraded faster outside the blood–brain barrier. It should be known that ependymomas can also cause subarachnoid bleeds.[8] Contrast enhancement is also variable which may be related to which tissue the lesion arises from. MRI does not show flow voids and neither does angiography reveal cavernomas due to either thrombosis or poor flow.[9] Previously, myelogram used to be done which would show the location of a lesion but not its nature. In patients who have multiple bleeds, there may be features of superficial siderosis that can be easily missed.

Microscopically, there are large, dilated hyalinized vascular channels that often show thrombosis, perivascular hemosiderin deposition, and calcifications. Intralesional bleeds lead to the formation of cystic spaces that can be so severe that the lesion mimics a neurenteric cyst.[10] Five case reports showed nerve fibers within the lesion. Cavernomas of the spine can arise not only in the bone, extradural space, and intradural space but can also arise from the pia, be attached to the nerve root, within the nerve root, periradicular vessel, ligamentum denticulatum, or filum.

Bruni et al[11] pointed out that the lesions become symptomatic due to the dynamic mobility of the lumbar spine, with the squashing of the lesion within the canal. Enhi and Love[12] stated that these types of lesions develop after traumatic or other stimuli, while Willis et al considered it a hamartoma thinking of it as a congenital lesion.

All lesions in the case reports reviewed were symptomatic and were surgically excised en masse. The lesion was usually adherent to one nerve root as in our case necessitating the sacrifice of that root. Postoperatively, all had an excellent outcome with most improving after surgery and a few with stable deficits.

## Conclusion

Cavernomas are very rare in the cauda equina and require a high index of suspicion to diagnose preoperatively. The exact pathophysiology of these lesions is yet to be elicited. After removal, the outcomes of these lesions are generally good.

**Conflict of Interest**
None declared.

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