Rom J Morphol Embryol 2025, 66(4):741–745 ISSN (print) 1220–0522, ISSN (online) 2066–8279 doi: 10.47162/RJME.66.4.13

CASE REPORT

RJME Romanian Journal of Morphology & Embryology http://www.rjme.ro/

Cauda equina cavernoma: case report and review of literature

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Abstract

Background: Cavernous malformations (CMs) are benign, low-flow vascular malformations of the central nervous system, most commonly located intracranially. Spinal CMs are rare, and involvement of the *cauda equina* is exceptionally uncommon. Owing to their rarity and nonspecific clinical presentation, *cauda equina* cavernomas pose a diagnostic challenge and are often misdiagnosed as more common intradural extramedullary tumors. *Case presentation*: We report the case of a 55-year-old woman presenting with several weeks of low back pain radiating to the right gluteal region, associated with urinary dysfunction consistent with a neurogenic bladder. Imaging findings revealed a well-circumscribed intradural extramedullary mass at the L1–L2 level, causing significant compression of the *cauda equina*. The patient underwent surgery, and total resection was achieved. *Conclusions*: *Cauda equina* CMs are rare entities that should be considered in the differential diagnosis of intradural extramedullary lesions, particularly in patients presenting with radicular pain and autonomic dysfunction. Magnetic resonance imaging (MRI) is the diagnostic modality of choice, while complete microsurgical resection remains the treatment of choice, offering excellent neurological outcomes when performed early.

Keywords: *cauda equina*, intradural extramedullary lesion, spinal vascular malformation, microsurgical resection.

Introduction

A cavernous malformation (CM), also referred to as cavernoma, cavernous angioma, or cavernous hemangioma, is a benign, low-flow vascular malformation rather than a true neoplasm, characterized by clusters of dilated, blood-filled sinusoidal spaces lined by a single layer of flattened endothelial cells and lacking smooth muscle or elastic tissue within the vessel wall [1–3]. Histopathologically, these vascular channels are closely apposed and separated by minimal or absent intervening neural parenchyma, a feature that distinguishes CMs from other vascular anomalies [2, 4]. On gross examination, the lesion typically appears as a well-circumscribed, red-blue, spongy mass with a "mulberry"- or "popcorn"-like architecture, although it remains non-encapsulated despite its sharp demarcation from surrounding tissues [3, 4]. CMs demonstrate considerable variability in size, ranging from a few millimeters to several centimeters in diameter, a characteristic that contributes to their heterogeneous clinical and radiological presentation [1].

Epidemiological studies estimate that CMs affect approximately 0.2% to 0.9% of the general population, with variations due to methodology and imaging ascertainment, and many lesions remaining clinically silent in asymptomatic individuals [5, 6]. Population-based magnetic resonance imaging (MRI) research suggests an overall prevalence near 0.44%, with slight male predominance observed in some cohorts, though sex ratios vary between studies [6]. CMs can arise anywhere along the central nervous system (CNS) axis, including the cerebral hemispheres, brainstem, cerebellum, and spinal cord, and, albeit much less commonly, have been reported in extracranial locations, such as the retina and liver, reflecting their systemic vascular malformation nature [5, 7]. Within the CNS, the vast majority of CMs are supratentorial, with estimates around 70%, while infratentorial and posterior fossa locations constitute a smaller proportion [1]. Spinal cord CMs represent a relatively rare subset, accounting for approximately 3% to 5% of all CNS cavernomas, and are predominantly intramedullary when present [5, 8]. Cavernomas involving the *cauda equina* are exceedingly rare, with only a limited number of cases documented in the literature to date, reflecting both their unusual anatomical location and the overall rarity of these lesions in the lumbosacral region [9, 10].

The clinical presentation of *cauda equina* CMs is heterogeneous and has been classically categorized into three main clinical syndromes: *cauda equina* syndrome,

subarachnoid hemorrhage (SAH) syndrome, and intracranial hypertension (ICHT) syndrome [9]. Among these, *cauda equina* syndrome is by far the most frequent manifestation, reported in approximately 90% of published cases, whereas the SAH syndrome has been described in 28.5% of patients, and ICHT syndrome represents the rarest clinical presentation, occurring in roughly 9.5% of cases [9]. The simultaneous occurrence of all three syndromes is exceptional, having been documented in only a single reported patient (4.7%) in the available literature [9].

The *cauda equina* syndrome results from direct compression of the lumbosacral nerve roots by the lesion itself or by associated hemorrhage and typically manifests with low back pain and/or radicular (sciatic) pain, which represent the most common presenting symptoms, followed by motor weakness, sensory deficits, and sphincter or sexual dysfunction [9, 11, 12]. In contrast, the SAH syndrome is characterized by acute headache, nuchal rigidity, nausea, and vomiting, secondary to subarachnoid dissemination of blood products, which exert a chemical irritative effect on neural structures and may ascend toward the posterior fossa through the cerebrospinal fluid pathways [9, 13]. ICHT syndrome is usually associated with hydrocephalus and presents with headache, visual disturbances (blurred or double vision), altered mental status, gait instability, and coordination deficits, reflecting impaired cerebrospinal fluid circulation [13].

Symptom onset in *cauda equina* cavernomas may be acute, subacute, or chronic, with many patients experiencing episodic and fluctuating neurological deficits [14]. This clinical variability is largely explained by the propensity of these lesions to undergo recurrent micro- or macro-hemorrhages, leading to sudden increases in lesion volume or local pressure and resulting in acute nerve root compression with abrupt neurological deterioration [11, 12]. Subsequent resorption of hemorrhagic products may lead to partial or complete symptom regression. However, repeated bleeding episodes can cause progressive fibrosis, chronic neural compression, and ultimately irreversible neurological deficits [9, 14].

MRI examination is universally regarded as the "gold standard" modality for the diagnosis of CMs, owing to its superior sensitivity and specificity compared with computed tomography (CT) and conventional angiography, and its ability to detect even small lesions and hemosiderin deposits [3, 15]. The widespread adoption and enhanced resolution of MRI over the past two decades have contributed substantially to the observed increase in reported incidence of cavernomas, with many lesions now detected as incidental findings during imaging performed for unrelated clinical indications [16]. On MRI, CMs classically demonstrate a multiloculated, heterogeneous "popcorn-like" appearance on both T1-weighted and T2-weighted sequences, reflecting the presence of blood degradation products at varying stages of evolution within the lesion [3]. Surrounding the core of mixed signal intensity, there is typically a hypointense rim on T2-weighted imaging, representing hemosiderin deposition from previous microhemorrhages, which is a hallmark feature and a key differentiator from other vascular lesions [3, 16]. In addition to conventional T1 and T2 sequences, susceptibility-sensitive sequences, such as gradient-recalled echo (GRE) and susceptibility-weighted imaging (SWI), further enhance detection of small lesions and delineation of hemosiderin-laden tissue [15]. MRI also provides valuable anatomical information regarding the lesion's relationship to dural and spinal compartments, as well as its intra- *versus* extramedullary location, although precise identification of nerve root involvement in the *cauda equina* region can be challenging due to the close packing of multiple nerve roots [3]. In contrast, angiography is typically uninformative for CMs because of their slow flow and lack of high-flow vascular nidus, and CT scanning often fails to characterize these lesions definitively, though occasional hyperdense foci from calcifications may be seen [15, 16].

From a macroscopic perspective, spinal intradural extramedullary CMs may originate from the vascular plexus of the nerve roots, the inner aspect of the *dura mater* or the pial surface of the spinal cord, likely reflecting aberrant angioblastic development of periradicular vessels rather than neoplastic proliferation [17, 18]. Unlike arteriovenous malformations, these lesions lack a large feeding artery and a prominent draining vein and are characterized by slow intralesional blood flow [17]. Although the majority of spinal cavernomas arise within the vertebral body or epidural space, only approximately 3% of CMs involve the intradural compartment, and within this subgroup, the extramedullary location is particularly uncommon [9]. Common differential diagnoses for intradural extramedullary masses include schwannomas, ependymomas (including the myxopapillary subtype), meningiomas, paragangliomas, astrocytomas, lymphomas, hemangioblastomas, gangliogliomas, and metastatic lesions, underscoring the diagnostic challenge posed by these vascular anomalies [18]. *Cauda equina* cavernomas are typically intradural and extramedullary, and may be intimately adherent to one or more nerve roots, which can complicate surgical dissection and occasionally require sacrificing the involved nerve root to achieve complete excision [19].

The "gold standard" for treatment of symptomatic intradural extramedullary cavernomas is microsurgical resection, since gross total removal significantly reduces the risk of recurrent hemorrhage and neurological deterioration associated with persistent or residual lesion [9, 17, 18]. These malformations have limited potential for spontaneous regression and are prone to growth and repeated micro-bleeding, making conservative management generally unfavorable [18]. With modern microsurgical techniques and intraoperative monitoring, complete resection is achievable in the majority of cases with acceptable morbidity; in reported series, many patients experienced clinical improvement postoperatively and no intraoperative mortality has been documented in the literature to date [9, 17].

Case presentation

We report the case of a 55-year-old Caucasian woman, with a medical history of well-controlled arterial hypertension, chronic venous insufficiency of the lower limbs, and claustrophobia, who was referred to our Neurosurgical Department with a several-week history of low back pain radiating to the right gluteal region, associated with episodes of urinary retention alternating with intermittent overflow incontinence.

On neurological examination, the patient presented no focal motor deficits. Deep tendon reflexes were preserved and symmetric bilaterally, including both patellar and Achilles reflexes. There was no objective sensory deficit, and both the straight leg raise test were negative. A urological evaluation, including pre- and post-void bladder ultrasonography, demonstrated findings consistent with a neurogenic bladder.

An initial diagnosis of L5–S1 lumbar disc herniation was suspected based on a non-contrast CT scan, which revealed degenerative changes and reduced disc height at the L5–S1 level but no clear evidence of nerve root compression. Further evaluation was performed with lumbar spine MRI, both native and contrast-enhanced, under mild sedation due to the patient's claustrophobia. MRI revealed a subdural, intradural mass measuring 11.8×18.5×5.4 mm, which was relatively well-circumscribed and heterogeneous. The lesion appeared slightly hyperintense on T2-weighted and short *tau* inversion recovery (STIR) sequences, isointense on T1-weighted images, and demonstrated moderate diffusion restriction, without contrast enhancement following gadolinium administration. The mass was located posterolaterally within the spinal canal at the L1–L2 level, without intraforaminal extension, and caused severe secondary lumbar canal stenosis at this segment (Figure 1, A and B).

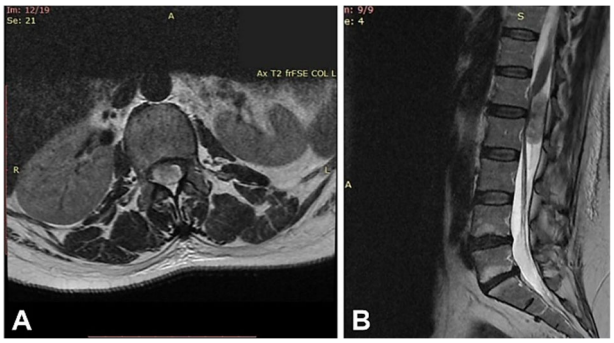

Figure 1 – ***Preoperative magnetic resonance imaging (MRI): (A) Axial view; (B) Sagittal view.***

The patient was informed of the suspected diagnosis based on clinical examination and paraclinical investigations. The surgical procedure, potential risks, and expected benefits were thoroughly explained to both the patient and her family. After receiving all relevant information, the patient and her family provided informed consent for the operation.

Surgical intervention was performed under general anesthesia, with the patient in the prone position. Accurate localization of the L1 and L2 vertebral levels was achieved using intraoperative fluoroscopy. A midline posterior approach was performed, followed by L1 and L2 laminectomies. After adequate exposure, the *dura mater* was opened longitudinally along the midline. Under the operative microscope, the intradural space was carefully explored. A soft, friable, brownish-violet mass was identified occupying the posterolateral portion of the dural sac. The lesion was mobile and exerted significant compression on the *cauda equina* nerve roots, displacing them anterolaterally toward the left side (Figure 2).

The tumor was meticulously dissected from the surrounding neural structures using microsurgical techniques. Although the lesion was not firmly adherent to individual nerve roots, the *cauda equina* roots at this level exhibited a diffuse brownish discoloration, most likely secondary to previous hemorrhagic episodes. The mass was progressively debulked and ultimately completely excised without the need for sacrifice of any nerve root (Figures 3 and 4).

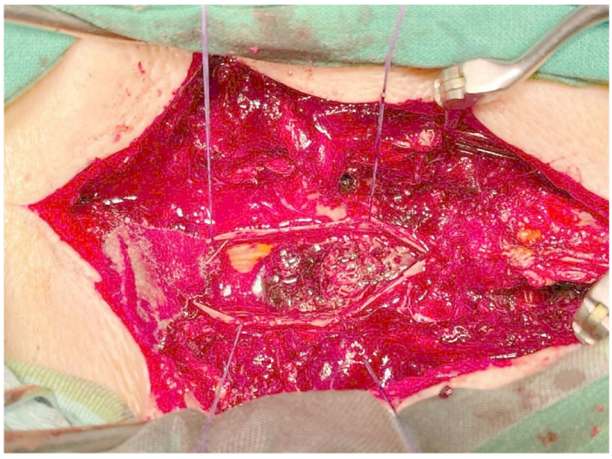

Figure 2 – ***Intraoperative view. Exposure of soft, friable, brownish-violet mass after dural opening.***

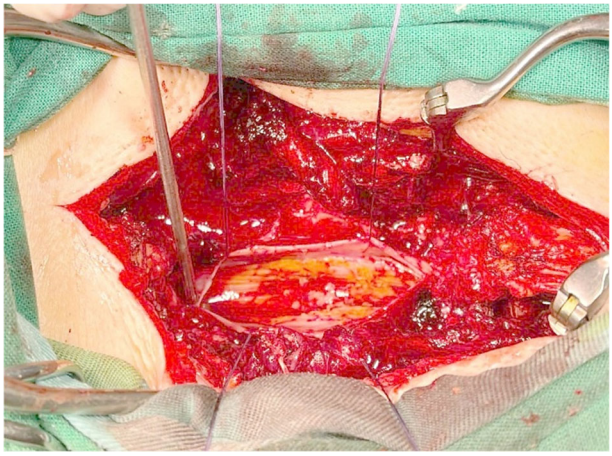

Figure 3 – ***Intraoperative aspect of cauda equina after en bloc removal of the lesion.***

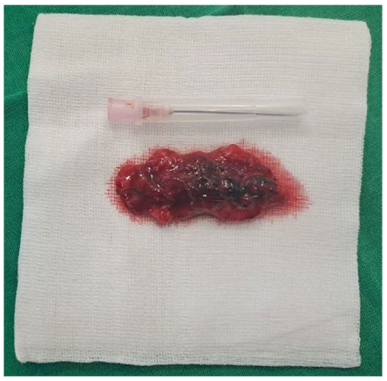

Figure 4 – ***En bloc resection of the lesion.***

Following lesion removal, the intradural space was carefully inspected to confirm gross total resection and adequate decompression of the *cauda equina*. The *dura mater* was closed in a watertight fashion, and the wound was closed in anatomical layers.

The postoperative clinical and neurological state was favorable. The patient experienced significant improvement in her lumbar and radicular pain. Bladder function progressively recovered, with complete resolution of urinary

dysfunction within three months after surgery. The patient declined pre- and postoperative neurophysiological studies. Complete imagistic screening was recommended to exclude additional lesions. However, due to persistent claustrophobia, she agreed only to a cerebral MRI, which revealed no intracranial abnormalities.

Discussions

Cauda equina CMs are extremely rare intradural extramedullary lesions, representing a small fraction of all spinal cavernomas, with most lesions located intramedullary or within vertebral bodies [6, 9]. Their clinical presentation is often insidious, and may include radicular pain, motor or sensory deficits, and autonomic dysfunction such as neurogenic bladder, reflecting the compressive effects on the lumbosacral nerve roots [9, 12]. The onset of symptoms may be acute or subacute, frequently associated with small hemorrhages, which explains the episodic and fluctuating nature of neurological deficits [12].

MRI remains the "gold standard" imaging modality, with lesions typically demonstrating the "popcorn" appearance, a heterogeneous signal core with a surrounding hemosiderin ring, which is pathognomonic for cavernomas [6, 9, 15]. Conventional CT and angiography are largely uninformative due to the slow-flow nature of these lesions [6]. In our patient, MRI allowed precise localization of the lesion, its intradural extramedullary position, and assessment of canal compression, which was crucial for surgical planning.

The differential diagnosis of *cauda equina* masses is broad, including schwannomas, ependymomas, meningiomas, paragangliomas, and metastases [6]. The unique vascular characteristics and intraoperative appearance of cavernomas – soft, friable, brownish-violet, multilobulated lesions – aid in distinguishing them from other intradural masses. Complete microsurgical resection remains the treatment of choice, aiming at neural decompression and prevention of recurrent hemorrhage or progressive neurological deterioration [6, 15]. In our case, total excision was achieved with careful microsurgical dissection, preserving all nerve roots, resulting in full recovery of pain and bladder function within three months.

This case emphasizes the importance of early MRI evaluation in patients with atypical lumbar pain and urinary dysfunction, particularly when initial CT findings are inconclusive. Although rare, *cauda equina* cavernomas should be included in the differential diagnosis of intradural extramedullary masses, as prompt surgical intervention can result in excellent neurological outcomes.

Conclusions

Cauda equina CMs, although rare, may present with radicular pain, neurogenic bladder, and subtle neurological deficits. MRI is the diagnostic modality of choice, providing detailed anatomical localization and aiding surgical planning. Microsurgical resection is safe and effective, with the potential for complete recovery of neurological function. Early recognition and total excision are critical to prevent recurrent hemorrhage and progressive neurological impairment.

Conflict of interests

The authors declare that they have no conflict of interests.

References

[1] Bhangoo RS, Crockard HA. Cavernous angiomas of the brain stem and spinal cord. J Clin Neurosci, 1998, 5(Suppl):20–25. https://doi.org/10.1016/s0967-5868(98)90005-4 PMID: 18639094

[2] Gross BA, Du R. Cerebral cavernous malformations: natural history and clinical management. Expert Rev Neurother, 2015, 15(7):771–777. https://doi.org/10.1586/14737175.2015.1055323 PMID: 26098013

[3] Wang KY, Idowu OR, Lin DDM. Radiology and imaging for cavernous malformations. Handb Clin Neurol, 2017, 143:249–266. https://doi.org/10.1016/B978-0-444-63640-9.00024-2 PMID: 28552147

[4] Cortés Vela JJ, Concepción Aramendía L, Ballenilla Marco F, Gallego León JI, González-Spínola San Gil J. Cerebral cavernous malformations: spectrum of neuroradiological findings. Radiologia, 2012, 54(5):401–409. https://doi.org/10.1016/j.rx.2011.09.016 PMID: 22197483

[5] Rigamonti D, Hadley MN, Drayer BP, Johnson PC, Hoenig-Rigamonti K, Knight JT, Spetzler RF. Cerebral cavernous malformations. Incidence and familial occurrence. N Engl J Med, 1988, 319(6):343–347. https://doi.org/10.1056/nejm198808113190605 PMID: 3393196

[6] Flemming KD, Graff-Radford J, Aakre J, Kantarci K, Lanzino G, Brown RD Jr, Mielke MM, Roberts RO, Kremers W, Knopman DS, Petersen RC, Jack CR Jr. Population-based prevalence of cerebral cavernous malformations in older adults: Mayo Clinic study of aging. JAMA Neurol, 2017, 74(7):801–805. https://doi.org/10.1001/jamaneurol.2017.0439 PMID: 28492932 PMCID: PMC5647645

[7] El Allouchi Y, Hamadi H, Benantar L, Aniba K. Management of central nervous system cavernomas: an experience of the Department of Neurosurgery at the Ibn Tofail Hospital, Mohammed VI University Hospital. Cureus, 2022, 14(12): e33028. https://doi.org/10.7759/cureus.33028 PMID: 36721533 PMCID: PMC9880495

[8] Sun I, Pamir MN. Spinal cavernomas: outcome of surgically treated 10 patients. Front Neurol, 2017, 8:672. https://doi.org/10.3389/fneur.2017.00672 PMID: 29326642 PMCID: PMC5742471

[9] Apostolakis S, Mitropoulos A, Diamantopoulou K, Vlachos K. Cavernoma of the *cauda equina*. Surg Neurol Int, 2018, 9:174. https://doi.org/10.4103/sni.sni_212_18 PMID: 30210907 PMCID: PMC6122280

[10] Popescu M, Grigorean VT, Sinescu CJ, Lupascu CD, Popescu G, Sandu AM, Plesea IE. *Cauda equina* intradural extramedullary cavernous haemangioma: case report and review of the literature. Neurol Med Chir (Tokyo), 2013, 53(12):890–895. https://doi.org/10.2176/nmc.cr2012-0309 PMID: 24097094 PMCID: PMC4508733

[11] Santoro A, Piccirilli M, Frati A, Salvati M, Innocenzi G, Ricci G, Cantore G. Intramedullary spinal cord cavernous malformations: report of ten new cases. Neurosurg Rev, 2004, 27(2):93–98. https://doi.org/10.1007/s10143-003-0302-9 PMID: 14714194

[12] Cai Z, Hong X, Dai W, Zhang Z, Liang Q, Ding X, Sun W. Surgical outcomes of symptomatic intramedullary spinal cord cavernous malformations: analysis of consecutive cases in a single center. Neurospine, 2023, 20(3):810–821. https://doi.org/10.14245/ns.2346430.215 PMID: 37798975 PMCID: PMC10562213

[13] Gross BA, Lin N, Du R, Day AL. The natural history of intracranial cavernous malformations. Neurosurg Focus, 2011, 30(6):E24. https://doi.org/10.3171/2011.3.FOCUS1165 PMID: 21631226

[14] Li D, Wu ZY, Liu PP, Ma JP, Huo XL, Wang L, Zhang LW, Wu Z, Zhang JT. Natural history of brainstem cavernous malformations: prospective hemorrhage rate and adverse factors in a consecutive prospective cohort. J Neurosurg, 2020, 134(3):917–928. https://doi.org/10.3171/2019.12.JNS192856 PMID: 32168479

[15] Kuroedov D, Cunha B, Pamplona J, Castillo M, Ramalho J. Cerebral cavernous malformations: typical and atypical imaging characteristics. J Neuroimaging, 2023, 33(2):202–217. https://doi.org/10.1111/jon.13072 PMID: 36456168

[16] Smith ER. Cavernous malformations of the central nervous system. N Engl J Med, 2024, 390(11):1022–1028. https://doi.org/10.1056/NEJMra2305116 PMID: 38477989

[17] Calderon C, Baron J, Ramdass A, Ramcharan R, Ramnarine D. A rare case of cavernous malformation of the *cauda equina* a case report. Int J Surg Case Rep, 2024, 114:109200. https://doi.org/10.1016/j.ijscr.2023.109200 PMID: 38154230 PMCID: PMC10800712

[18] Takami H, Kumar R, Brown DA, Krauss WE. Histologic features and prognosis of spinal intradural extramedullary Ewing sarcoma: case report, literature review, and analysis of prognosis. World Neurosurg, 2018, 115:448–452.e2. https://doi.org/10.1016/j.wneu.2018.04.015 PMID: 29654955

[19] Ismaiel N, Ibrahem H, Jabbour G, Joha M, Abdulrahman M, Alshehabi Z. Spinal intradural extramedullary cavernoma: a case report. Int J Surg Case Rep, 2024, 115:109274. https://doi.org/10.1016/j.ijscr.2024.109274 PMID: 38219515 PMCID: PMC10826290

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Received: October 1, 2025

Accepted: March 3, 2026