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Cavernous angioma of the cauda equina: A case report

Annelies Mondelaers ^{a,b,g}, Thomas Vermeulen ^{c,g}, Eline De Smet ^{d,g}, Maarten Vanloon ^{e,f,g,*}, Tomas Menovsky ^{f,g}

^a NeuroVascular Center Antwerp, Antwerp University Hospital, Edegem, Belgium

^b Faculty of Medicine and Health Sciences, Research Group mVision, University of Antwerp, Antwerp, Belgium

^c Huisartsenpraktijk Den Biezerd, Niel, Belgium

^d Dept. of Radiology, Antwerp University Hospital, Edegem, Belgium

^e Faculty of Health, Medicine and Life Sciences, University Maastricht, The Netherlands

^f Dept. of Neurosurgery, Antwerp University Hospital, Edegem, Belgium

^g Dept. Translational Neurosciences, University of Antwerp, Antwerp, Belgium

ABSTRACT

Cavernous angiomas are benign vascular malformations that are occasionally found in the central nervous system. They comprise about 3% of all subdural spinal cord tumors, with only 24 cases of cavernomas of the cauda equina described in the literature.

This paper reports the case of a 55-year-old man who presented with back pain radiating to both legs. The patient followed several sessions of physiotherapy with only short pain relief. A lumbar magnetic resonance imaging showed a lesion with diameter of 12 mm located at the L1/L2 level under the conus medullaris. The patient underwent L2 and partially L1 laminectomy with complete resection of the lesion. The pathohistological examination was consistent with a cavernous angioma of the cauda equina. Full recovery of the patient was obtained without any neurological deficit.

1. Introduction

Cavernous angioma (CA), also known as cavernoma, cavernous hemangioma, and cavernous malformation (National Library of Medicine Medical Subject heading Unique ID D006392), are benign neurovascular malformations comprised of clustered dilated capillaries. CAs originate from blood vessel progenitors whose development is stopped. [1,26] They are histologically characterized by grossly dilated vascular spaces (known as 'caverns') lined by a single layer of endothelium, lacking mural elements of mature vascular architecture, and displaying features of chronic hemorrhage in adjacent neuroglial parenchyma. [15,20,35,40] Impaired blood flow may cause intralesional thrombosis, calcification and recanalization, which can be observed with histopathological analysis.[15].

About 0.4–0.8 % of the general population present with CAs. [9,30,31,34] Nowadays, the gold standard radiological modality for diagnosis and follow-up is magnetic resonance imaging (MRI). This highly sensitive and specific technique clearly demonstrates the typical popcorn-like appearance of a CA with hemosiderin, underlining dilated caverns with mixed signals of blood at different stages of organization and calcifications.[12] Hemosiderin deposition is a typical feature of cavernomas and it is suggested to arise from clinically silent small

bleedings that create cavities around the blood vessel, subsequently leading to hemosiderin accumulation.[36] CAs are dynamic lesions and show great variability in size ranging from a few millimeters to several centimeters.[8,15,28,39] These lesions are seldom diagnosed preoperatively and are mostly discovered as incidental findings during MRI examinations.[9,27,28,34].

Cavernous malformations may be observed in any part of the central nervous system (CNS). However, they are mostly located in the supratentorial cerebral compartment. [2,26] They can also present on the skin, in the spinal cord, retina or liver. [17,22] Spinal cavernomas are rare entities representing only 5–12 % of spinal cord vascular lesions and often originate in the vertebrae with sporadic extension into the extradural cavity. [14,32] Even more uncommon are cavernomas of the cauda equina, with only 24 cases described in the literature. [4,16].

Cavernomas can occur either in a sporadic form harboring a solitary hemorrhagic vascular lesion or as clustered lesions, or in an autosomal dominant familial form [22] The hereditary form results from a heterozygous germline loss-of-function mutations in one of three genes-*CCM1/KRIT1*, *CCM2/Malcavernin*, and *CCM3/PDCD10*-leading to multifocal lesions throughout the brain and spinal cord. These genes encode proteins which are implicated in modulating junction formation between vascular endothelial cells. It was reported that mutations in any

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^{*} Corresponding author at: Faculty of Health, Medicine and Life Sciences, University Maastricht, The Netherlands. *E-mail address:* maarten_vanloon@hotmail.com (M. Vanloon).



Fig. 1. Midsagittal CT-image of the lumbar spine. Discrete bulging of the L4-L5 and L5-S1 discs is seen (arrowheads). No intraspinal mass is detected.

of these genes impair the function of a protein complex, hereby leading to weakening of these junctions and increased leakage from vessels. [10,21,22].

The pathogenesis of CAs are not completely understood. Different mechanisms may contribute to the development of CAs, including inflammation and immune-mediated processes, angiogenesis and vascular permeability, microbiome-driven factors, and lesional anticoagulant domains. In-depth characterization of these mechanisms has led to the identification of plasma biomarkers and new putative therapeutic targets.[5].

The most common clinical manifestations of CAs include seizures (40–70 %), focal neurological deficits (35–50 %) or hemorrhage (9–56 %).[3,11,33,37,43] However, up to 44 % of the affected individuals are asymptomatic.[27,28] The estimated annual risk of hemorrhage for CAs is between 1.7 and 4.5 %.[6] If the hemorrhage causes severe neurological signs and symptoms or the bleeding recurs, surgical intervention should be considered. Early treatment is warranted due to the risk of sudden neurological impairment due to hemorrhagic lesions. Surgery is the main and preferred treatment in case of superficial lesions or intractable seizures.[7].

Spinal cavernomas are mostly diagnosed when patients experience neurological deficits. Clinical presentation depends on the location and growth rate of the lesion, but typical features include spinal pain, radiculopathy, progressive paraparesis, acute paraplegia, and progressive myelopathy.[26] Clinical symptoms manifest due to progressive enlargement of the lesion. Spinal cord cavernous malformations may even cause bladder and bowel problems. Microsurgery is warranted, especially for lesions greater than 5 mm and located posteriorly, as conservative treatment of spinal cavernomas may result in neurological



Fig. 2. Midsagittal CT-image of the lumber spine, 10 years after initial CT-scan. A hyperdense mass is seen within the spinal canal at the L1-L2 level (arrow).

deterioration due to the risk of bleeding and compression.[38].

In this report, the authors describe the clinic-radiological and surgical features of a case presenting with an intradural extramedullary cavernoma of the spinal cord.

2. Case report

2.1. Clinical presentation

A 55-year-old male patient presented with a history of low back pain radiating to the lower extremities. The symptoms lasted for more than 10 years. His history included appendicitis, pneumonia, and surgery of the shoulder. Neurological examination revealed no sensorimotor deficits. Initial Computed Tomography (CT) of the lumbar spine at the start of the symptoms revealed only minimal bulging of the disk at L4-L5 and L5-S1 with no direct compression of the nerve roots. (Fig. 1) The patient followed several rehabilitation sessions, which resulted in short pain relief. Ten years later, due to progressively worsening of the pain, CT of the lumbar spine was repeated and revealed a hyperdense mass at the L1-L2 level. (Fig. 2).

Subsequent MRI was performed and revealed an intradural extramedullary lesion with a diameter of 12 mm, located under the conus medullaris. The well-marginated mass appeared very hypointense on T2-weighted images and showed high signal intensity on T1-weighted images. No bone involvement was observed. A radiological diagnosis of melanocytic meningioma was considered, and microsurgical resection was recommended. (Fig. 3).



Fig. 3. A, B. Sagittal and axial T2-weighted MR images. A well demarcated and very hypointense mass is located centrally in the spinal canal (arrow) without detectable contact with the dura or bone involvement. There is only minimal cerebrospinal fluid around the nerve roots, indicative of compression (arrowheads). C,D. Sagittal T1-weighted MR images before and after contrast administration. The lesion appears hyperintense without significant enhancement after contrast administration.

2.2. Surgery

The patient was positioned in prone. Neuromonitoring was used to help prevent nerve damage. The level of the cavernoma was checked by radioscopy before skin incision. Iodination and infiltration with epinephrine-containing Xylocaine 1 % was performed. Iodination was repeated. Incision of the skin and dissection of underlying tissue was performed until the spinous processes and lamina of L1/L2/(L3) were reached. The patient underwent an L2 and partially L1 laminectomy. Ultrasound imaging was performed intraoperative to determine the



Fig. 4. Intra-operative view of the intradural cavernoma.



Fig. 5. A. Hematoxylin and eosin (H&E) staining, 50 µm. B. H&E, 100 µm. C. Immunostain S100 expression in pre-existent nerve.

exact location of the lesion. Midline durotomy was executed under magnification. Visualization of the lesion revealed features of cavernous angioma. The cavernoma was characterized by small clusters of capillaries at caudal side and a capsulated, well-demarcated mass which appears as an old hematoma containing hemosiderin deposits with variable density (Fig. 4). Cavernoma tissue was approached and completely resected with sparing of the nerve root closely adherent to the lesion. After resection, the conus medullaris and the filum terminale were visible. No signs of bleeding and nerve damage was observed in the resection cavity. The post-operative course was uneventful.

2.3. Pathohistological examination

Macroscopically, the excised lesion showed a nodular, lobular aspect with some presence of blood components. A thin membranous capsule was formed around the nodule. Microscopic analysis documented characteristics of fibrous connecting tissue. Some dilated vascular structures were observed, positioned adherent and between pre-existent nerve bundles (focal S-100-positive structure) with normal aspect. Histopathological examination confirmed the presence of a partially fibrous-transformed cavernoma (Fig. 5).

2.4. Postoperative course

The patient was conservatively managed with bedrest, anticoagulants, and an analgesic. Immediately after surgery, the patient developed ocular redness and pain in the left eye with visual impairment, pointing to the occurrence of corneal abrasion. The patient showed favorable progress in neurological recovery after complete resection. No additional neurologic deficits were reported at discharge on day 4 after surgery. During his follow-up clinic visit one and a half months after surgery, the patient complained of some sensorimotor deficit in both feet. However, these clinical symptoms were only temporary. The patient suffered from intermittent headache attacks and experienced some discomfort at the site of the scar and location of the resection. A twomonth follow-up MRI scan revealed total resection of the lesion without any residual mass (Fig. 6).

3. Discussion

Cavernous angiomas are abnormally vascular malformations of the CNS presenting with dilated thin-walled sinusoidal spaces, lined by a single layer of endothelium with little or no intervening nervous tissue.



Fig. 6. A. Sagittal STIR image. Post-operative soft tissue changes and small epidural elongated fluid collection superficial of the dura (arrow) as well as bone oedema in the residual spinous process L2 (arrowhead) are present. B. Axial T2-weighted image at the surgical level. No residual mass is seen with resolution of nerve root compression.

[25] These lesions may emerge from blood vessels of the nerve roots, the inner surface of the dura mater, and the pial surface of the spinal cord, with only one case described as originating from the filum terminale.[42] It is suggested that these vascular anomalies probably arise from the aberrant development of periradicular vessels.

In recent years, the proportion of cavernomas that were incidentally diagnosed has likely increased due to the increasing accessibility of MRI. MRI is the primary imaging modality as this technique has excellent contrast and structural resolution, is able to visualize all compartments, and is capable of analyzing the presence of enhancement, cystic change, and blood products. A wide variety of MRI appearances exists with significant overlap of the imaging features. MRI can help differentiate intra- or extradurally and intra- or extramedullary lesions. However, the precise nerve root involvement of the cauda equina is often difficult to assess due to the close proximity of several nerve roots.

Spinal vascular malformations are usually located within the vertebral bodies, however, about 3 % of CAs are situated intradurally, mostly intramedullary but seldom extramedullary.[24,29] The first case of intradurally extramedullary CA was described in 1903.[23] Intradural extramedullary spinal CAs exhibit characteristics features on MRI, which differentiates them from other spinal cord tumors and vascular malformations. The existence of mixed subacute and chronic hemorrhage, marked by mixed high-and low-signal intensity components is typical for these lesions.[19,33] The hemosiderin accumulation is clinically relevant, because it is detected on MRI images as a hypointense signal ring around the lesion.[36] The differential diagnosis includes different tumors of the cauda equina region, *i.e.* schwannoma, paraganglioma, myxopapillary ependymoma, meningioma, medulloblastomas, metastasis, primitive neuroendocrine tumor, hemangioblastoma, astrocytoma, and ganglioglioma.[41].

Here, we report the 25th case of a CA of the cauda equina involving a 55-year-old male.[4,16] Drazin et al. described that about half of the cauda equina lesions were reported in patients who received prior radiotherapy.[16] Our patient did not receive any radiotherapy in the past. The absence of radiation exposure suggests that the patient's symptoms and imaging findings were not attributed to a radiationrelated cause, contrasting with the predisposing factor highlighted in existing literature. Our patient complained for several years of low back pain which is a typical clinical symptom secondary to local compression of the nerve fibers of the cauda equina. Both CT and MRI imaging demonstrated a well-demarcated mass at L1/L2 level. The patient underwent L2 and partially L1 laminectomy with gross total resection of the lesion with preservation of neural tissue. Intra-operative findings were consistent with intradural extramedullary cauda equina cavernoma. Pathohistological examination with microscopic appearance of lobules packed with capillary-sized vessels provided histological evidence of cavernous angioma.

Total resection is crucial as cavernoma residues may cause neurological deterioration due to their tendency to grow and bleed. Current microsurgical techniques allow complete surgical resection with acceptable morbidity. In addition, no cases of mortality are described in the literature.[38] Despite careful microsurgical dissection, preservation of the nerve root is not always feasible due to encapsulation within the lesion.[13] In our patient, the lesion did not originate within the nerve root and thus complete surgical resection could be performed.

4. Conclusion

This report presents a case of a cauda equina cavernoma in a 55-yearold patient who suffered from low back pain for 10 years. The intradural

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extramedullary lesion measured 12 mm diameter and was located at the L1/L2 level under the conus medullaris. The vascular lesion harvested the typical features of a cavernous angioma, showing a well-demarcated mass with clustered dilated capillaries adherent to a nerve root. Microsurgical procedure succeeded in gross total lesion resection with favorable neurological outcome. Postsurgical MRI evaluation showed evidence of no mass recurrence.

CRediT authorship contribution statement

Annelies Mondelaers: Writing – review & editing, Writing – original draft, Visualization, Methodology, Investigation, Conceptualization. Thomas Vermeulen: Writing – review & editing, Writing – original draft, Visualization, Validation, Methodology, Investigation, Conceptualization. Eline De Smet: Writing – review & editing, Validation, Supervision, Methodology, Conceptualization. Maarten Vanloon: . Tomas Menovsky: Writing – review & editing, Writing – original draft, Visualization, Validation, Methodology, Investigation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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