Neurosurg Focus 21 (1):E10, 2006

Clinical presentation and optimal management for intramedullary cavernous malformations

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Object. Intramedullary cavernous malformations (CMs) account for approximately 5% of all intraspinal lesions. The purpose of this study was to define the spectrum of presentation for spinal intramedullary CMs and the results of microsurgery for these benign but clinically progressive lesions.

Methods. Retrospective chart review was performed in 26 patients with histologically diagnosed CMs. All patients had undergone preoperative magnetic resonance (MR) imaging studies. All patients were treated with a laminectomy and microsurgical resection of the malformation.

Conclusions. The MR imaging findings are diagnostic for intramedullary CMs; these lesions abut a pial surface and have a characteristic imaging pattern. Spinal intramedullary CMs present with either an acute onset of neurological compromise or a slowly progressive neurological decline. Acute neurological decline occurs secondary to hemorrhage inside the spinal cord. Chronic progressive myelopathy occurs due to microhemorrhages and resulting gliotic reaction to blood products. Surgery and total removal of the lesion tends to halt progression of symptoms.

KEY WORDS • cavernous malformation • intramedullary lesion • spine • vascular malformation

CAVERNOUS malformations are well-circumscribed lesions that consist of closely packed, capillary-like vessels, without intervening brain or spinal tissue. This term is synonymous with cavernous angiomas, cavernous hemangiomas, and the older term cavernoma. These lesions are angiographically occult vascular malformations and thus are difficult to diagnosis without MR imaging. Microscopically, they are characterized by thin-walled sinusoidal vascular channels. They have mixed signal intensity on T_1-weighted MR images. Hemosiderin deposits cause a low-signal-intensity ring surrounding these lesions on both T_1- and T_2-weighted images.[1,4,7,18] On gross inspection, they have a mulberry-like appearance and are stained with hemosiderin.[29]

The intraspinal location accounts for approximately 5% of all CMs. Regardless of their location, CMs are histologically identical,[7] but their clinical presentation varies depending on the site at which they are found. These intramedullary lesions, in such a particularly precarious location, are more likely to give rise to clinically significant neurological findings, unlike their intracranial counterpart.

We previously reported our small series of intramedullary CMs,[14] but we have expanded the patient population and the long-term follow-up duration. We retrospectively reviewed our larger series of 26 patients with spinal lesions treated surgically during the same period. The clinical presentation and outcome after surgical intervention are presented and discussed.

Clinical Material and Methods

The charts of 26 patients treated between 1991 and 2005 for intramedullary CM were reviewed. The study included nine female and 17 male patients ranging in age from 8 to 73 years (mean age 38 years) who presented with a variety of symptoms before surgery (Table 1). The most common presentation was a motor deficit, followed by sensory loss.

A dorsal approach was made through an osteoplastic laminotomy or laminectomy. The bone removal was limited to the site of the malformation. Prior to opening the dura mater, the location of the malformation was insonated with the intraoperative ultrasonography device. This surgical adjunct guided the extent of bone removal. A blue discoloration was evident on the dorsal or dorsolateral surface of the spinal cord in all patients. The myelotomy was performed over this area and then the lesion was removed in an inside-out fashion similar to the surgical method for intramedullary astrocytomas. A gliotic plane surrounds the capillary sinusoids and demarcates the malformation from normal spinal cord. However, in patients who had a chronic presentation, the spinal cord appeared atrophic, edematous, and had a very soft consistency.

The outcome data we obtained included the patient's current neurological status compared with results of their

Abbreviations used in this paper: CM = cavernous malformation; MR = magnetic resonance.

TABLE 1
*Clinical characteristics of 26 patients with spinal CMs**

Case No.	Age (yrs), Sex	Lesion Location†	Symptoms: Type	Symptoms: Duration (mos)	Outcome: Postop	Outcome: Long-Term	FU (mos)	Other CMs
1	67, M	T-3	lt LE weakness	36.0	worse	improved	120.0	no
2	28, M	C-5	lt UE weakness	0.5	same	same	115.0	no
3	13, M	T-4	lt LE weakness	3.0	same	same	98.0	7
4	73, M	T-8	myelopathy	240.0	worse	same	96.0	no
5	33, M	T-7	Brown-Séquard	180.0	worse	same	89.0	no
6	38, F	T-9	Brown-Séquard	24.0	worse	improved	76.0	no
7	26, F	C7–T1	lt UE paresthesia	2.0	improved	same	64.0	no
8	37, M	T-11	bladder incontinence	3.0	worse	improved	60.0	no
9	38, M	T-7	myelopathy	96.0	worse	worse	59.0	no
10	64, F	T-8	myelopathy & lt LE weakness	120.0	worse	improved	58.0	no
11	8, M	C-1	myelopathy	0.5	same	same	48.0	6
12	32, F	T-2	lt LE weakness	8.0	same	same	50.0	no
13	42, F	C-4	rt-sided weakness	0.5	improved	improved	47.0	no
14	46, F	T-10	progressive myelopathy	276.0	worse	improved	44.0	no
15	44, M	C-2	rt-sided weakness	0.5	improved	improved	42.0	no
16	49, M	T4–5	pain, rt LE weakness, sensory loss	6.0	worse	same	47.0	no
17	30, M	C-3	lt-sided paresthesias	0.75	same	same	30.0	4
18	40, M	T8–9	myelopathy, misdiagnosed as MS	33.0	same	improved	48.0	no
19	29, F	T9–10	acute rt LE weakness, bladder incontinence	0.5	same	improved	60.0	no
20	47, M	C2–4	myelopathy	24.0	worse	worse	40.0	no
21	46, M	C-5	shoulder pain	5.0	worse	same	4.0	no
22	18, M	C7–T1	chronic rt LE weakness	48.0	worse	same	9.0	no
23	45, F	T4–5	rt LE weakness, sensory loss	1.0	same	improved	34.0	no
24	36, F	C2–3	neck pain, sensory	1.5	improved	improved	32.0	no
25	18, M	T1–2	rt LE weakness	8.0	improved	improved	20.0	no
26	40, M	T6–7	lt LE weakness, myelopathy	30.0	worse	improved	6.0	no
mean	38.0			44.1			53.7	

* FU = follow up; LE = lower extremity; MS = multiple sclerosis; UE = upper extremity.
† Totals for location: eight cervical and 18 thoracic.

preoperative examination and any evidence for new symptoms referable to the lesion or to other CMs. The mean follow-up period for the study group was 4.5 years (range 4 months–10 years).

Illustrative Cases

Case 19

History and Examination. This 29-year-old woman presented to the emergency room with onset of back pain and right radicular pain. Results of the initial examination included right lower-extremity weakness (3/5) in dorsiflexion and perineal hypesthesia. She underwent MR imaging, which demonstrated an intramedullary hemorrhage at T-10 (Fig. 1). She was discharged with orders for outpatient physiotherapy.

Operation and Postoperative Course. The patient returned to undergo surgery 4 weeks after the initial admission. At that time, she continued to experience weakness in her distal lower extremity, but her urological function had improved. The CM was removed through a simple osteoplastic laminotomy. There was no change in the results of her neurophysiological monitoring. Her lower-extremity neurological function improved immediately after surgery, and neuroimaging studies confirmed the gross-total resection.

Case 26

History and Examination. This 40-year-old man presented with a chronic history of progressive myelopathy. He initially presented with left lower-extremity spasms and numbness that had lasted for several years. For the last 8 or 9 months, however, he had experienced a left foot drop and urinary urgency. Admission MR images of his thoracic spine revealed a lesion with mixed signal characteristics on T_1-weighted sequences. A dark halo on T_2-weighted images was consistent with a hemosiderin ring, which was characteristic for an intramedullary CM. There was no enhancement after gadolinium administration. The spinal cord appeared minimally expanded (Fig. 2).

The patient's strength was judged to be 5/5 on the right side. He had decreased light touch and proprioception in the left lower extremity. His strength was 2/5 in plantar and dorsiflexion. He also was hyperreflexic in the left lower extremity, with several beats of clonus.

Operation. The patient underwent a T6–7 osteoplastic laminotomy and complete excision of the malformation. The ultrasonography device was used to insonate the margins, and then the dura mater was opened. A myelotomy was centered over the malformation. The lesion was then removed in an inside-out fashion until the gliotic white matter was identified. The patient's spinal cord appeared quite atrophic and edematous, although there was a good separation plane between the malformation and the cord.

Postoperative Course. Postoperatively, the patient experienced increased weakness in the proximal left lower extremity, which improved over several months with physi-

FIG. 1. Case 19. Preoperative MR images of an intramedullary CM with an acute presentation. *Left:* Sagittal T_2-weighted image demonstrating the intramedullary lesion with hemorrhage. *Right:* Sagittal T_1-weighted image confirming the presence of subacute hemorrhage and blood products.

cal therapy. He was able to walk with the assistance of a cane. He had no new pain or dysesthesia related to the surgery.

Results

These malformations were located throughout the spinal axis; eight were found in the cervical cord and 18 in the thoracic cord. The mean duration of symptoms before surgery was 44.1 months (range 2 weeks–23 years).

All patients were screened with MR imaging studies. The lesion size varied from 8 to 45 mm (mean 16.1 mm). Angiography was not performed in any patient, nor was this diagnostic study necessary to narrow the differential diagnosis. All patients underwent a laminectomy or osteoplastic laminotomy with an attempt made to attain gross-total resection of the lesion. Neurophysiological monitoring was used for all patients. Complete resection was achieved in 25 of the 26 patients, and none had a surgical complication.

Five patients experienced immediate improvement in their neurological status postoperatively, whereas in 11 a transient decline in neurological function necessitated physiotherapy. Although their number was not statistically significant, patients with chronic symptoms were more likely to experience postoperative deficits requiring inpatient physiotherapy.

The mean follow-up duration was 4.5 years (range 4 months–10 years). Although 50% of patients worsened immediately after surgery, 12 (46%) reported improvement from their preoperative functional status at their last follow-up evaluation. Another 12 (46%) reported improved motor strength compared with their immediate preoperative function. Functional status was worse postoperatively in only two patients (8%). The immediate and long-term neurological outcome after surgery is reported in Table 2.

Postoperative imaging of the spine was performed in all patients, as was follow-up MR imaging of the entire neuraxis. Of the 26 patients, three had multiple lesions: one patient had seven intracranial lesions located throughout the supratentorial and infratentorial compartments; the second patient had six intracranial lesions; and the third had four supratentorial lesions. The remainder of the spinal axis in these patients revealed no other intraspinal malformations. None of the patients had been previously treated for their intracranial lesions or required treatment for these incidentally discovered CMs. These three patients were all younger than the median age.

Discussion

Cavernous malformations can occur throughout the central nervous system, but are most commonly located in the supratentorial compartment. The intramedullary location is relatively rare, but recent MR imaging studies have demonstrated that lesions in this region are more common than

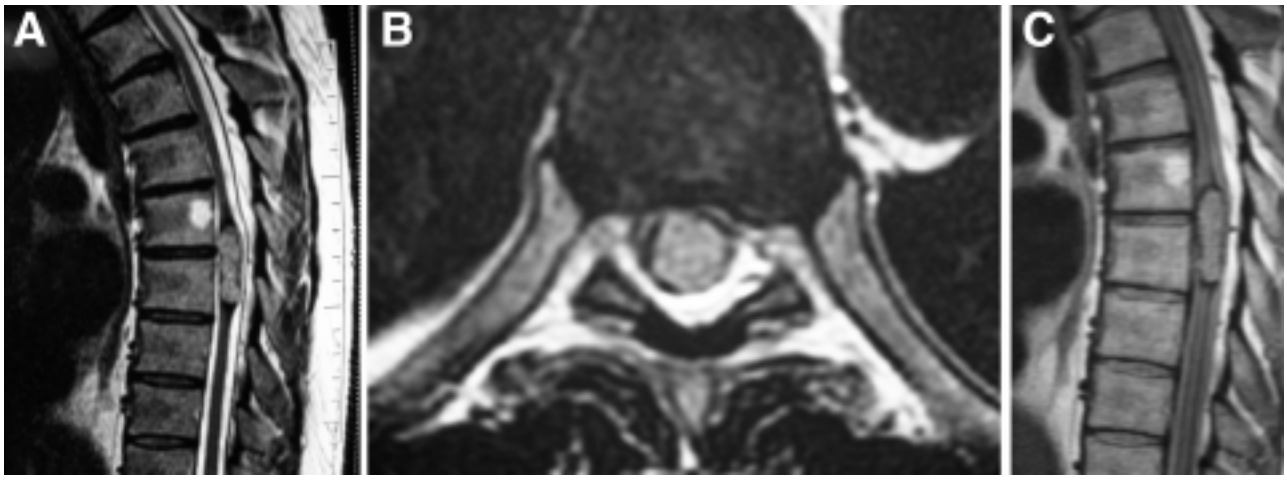

FIG. 2. Case 26. Preoperative MR images of an intramedullary CM with a chronic presentation. A: Sagittal T_2-weighted image revealing the intramedullary lesion with hemosiderin ring. B: Axial T_2-weighted image confirming the intramedullary location and width of the minimally expanded spinal cord. C: Sagittal T_2-weighted image also demonstrating hemosiderin and blood products extending up the central canal.

TABLE 2

Outcome in 26 patients treated surgically for intramedullary CMs

Postop Status	Outcome (%)	
	Immediate	Long-Term FU
improved	5 (19)	12 (46)
unchanged	8 (31)	12 (46)
worsened	13 (50)	2 (8)

originally thought.[18] It is likely that the myelopathy previously diagnosed as idiopathic was caused by these intraspinal CMs, because these lesions were difficult to identify on computed tomography and myelography studies.[16] More recent estimates indicate that CMs constitute 5% of vascular malformations.[32]

There have been previous cases of intramedullary CMs reported in the literature (Table 3), and there has been an increased number of case reports and small series in the MR imaging era.[2–6,8–10,12–15,17,19,20,22–28,30–41] A female/male ratio of 2:1 had been previously reported.[1] However, we previously calculated an equal distribution for all spinal intramedullary CMs reported in the literature.[14] The present study, with its more detailed review of the literature, also supports our hypothesis that there is an equal female/male distribution for predominantly nonfamilial intraspinal CMs. Interestingly, there have been several lesions identified during pregnancy.

In our experience, it appears that patients with multiple intracranial lesions are more likely to present at an earlier age with a clinically significant spinal source for the hemorrhage. The mean age for the three patients with multiple lesions was 17 years, which is significantly less than the mean age for the entire cohort. These patients with multiple CMs had multiple intracranial lesions (mean 5.7 lesions) but only one intraspinal one. No patient had familial CMs. Patients with multiple CMs constituted only 12% of this series. This is in contrast to a recent study in which it was reported that as many as 40% of patients with spinal CMs may harbor an intracranial one.[11] Thus, we recommend complete neuraxis imaging when a patient presents with an intraspinal lesion. We also saw no hemorrhages or seizures related to these intracranial lesions during the follow-up period.

Appearance on Neuroimaging

These lesions have a characteristic appearance on MR

TABLE 3

*Literature review of histologically proven intramedullary CMs**

Authors & Year	No. of Patients	Mean Age (yrs)	M/F Ratio	Presentation		Symptom Duration (mos)	Long-Term Outcome		
				Acute	Chronic		Worse	Same	Improved
Bicknell, et al., 1978	1	32	0:1	0	1	120.0	1	0	0
Tyndel, et al., 1985	1	27	0:1	1	0	0.5	0	0	1
Cosgrove, et al., 1988	5	41	2:3	1	4	47.0	1	4	0
McCormick, et al., 1988	6	33	4:2	2	4	24.0	1	2	3
Vaquero, et al., 1988	2	26	0:2	0	2	30.0	1	1	0
Villani, et al., 1989	3	38	1:2	1	2	NA	0	1	2
Zentner, et al., 1989	2	37	1:1	0	2	90.0	1	1	0
Barnwell, et al., 1990	1	37	1:0	0	1	36.0	0	1	0
Lopate, et al., 1990	2	30	0:2	1	1	29.0	0	0	2
Mehdorn & Stolke, 1991	2	34	0:2	1	1	18.0	0	0	2
Fazi, et al., 1992	2	37	1:1	0	2	102.0	0	1	1
Ogilvy, et al., 1992	6	43	2:4	4	2	4.0	0	0	6
Anson & Spetzler, 1993	6	35	2:4	2	4	25.0	0	3	3
Canavero, et al., 1994	1	48	0:1	0	1	168.0	1	0	0
Lunardi, et al., 1994	5	42	2:3	3	2	29.0	NA	NA	NA
Cantore, et al., 1995	6	53	5:1	0	6	110.0	1	3	2
Gordon, et al., 1995	3	35	1:2	0	3	70.0	NA	NA	NA
Harrison, et al., 1995	1	36	1:0	NA	NA	NA	0	1	0
Spetzger, et al., 1995	9	43	6:3	2	7	150.0	0	2	7
Turjman, et al., 1995	10	47	3:7	NA	NA	NA	NA	NA	NA
Furuya, et al., 1996	4	52	2:2	1	3	9.0	0	0	4
Padovani, et al., 1997	4	46	3:1	1	3	NA	0	1	3
Visteh, et al., 1997	17	40	8:9	6	11	NA	1	6	10
Cristante& Hermann, 1998	12	34	4:8	1	11	20.0	2	3	7
Tu, et al., 1999	7	30	5:2	1	6	NA	0	1	6
Zevgaridis, et al., 1999	7	39	3:4	4	3	34.0	1	1	5
Nagib & O'Fallon, 2002	2	12	1:1	2	0	0.2	0	0	2
Barrena-Caballo, et al., 2003	12	33	8:4	3	9	30.0	0	2	10
Sandalcioglu, et al., 2003	10	35	3:7	5	5	29.0	0	6	4
Santoro, et al., 2004	10	41	5:5	4	6	29.0	0	1	9
Bakir, et al., 2005	1	14	1:0	1	0	0.5	0	0	1
present study	26	38	17:9	12	14	44.0	2	12	12
totals (%)	186	36.5	92:94	59 (34)	116 (66)	44.6	13 (8)	53 (31)	102 (61)

* NA = not available.

images.[4] That is, they have mixed signal intensity on T_1-weighted images,[1,18] whereas microhemorrhages and hemosiderin deposits cause a low-signal-intensity ring surrounding the lesions on T_1- and T_2-weighted images. Gradient echo sequences demonstrate the hypointense signal from the hemorrhage.

Clinical Presentation

Patients may receive a diagnosis of asymptomatic intramedullary CM, although the classic presentation is either acute or chronic progressive clinical symptoms. Patients can present with acute neurological deficits that occur because of a hemorrhage within the spinal cord parenchyma.[27] A second presentation, which is more common (66%), is slowly progressive myelopathy. The proposed mechanism for this myelopathy is microhemorrhages and resultant reactive gliosis. Some patients, however, have a combined presentation in which an acute deterioration is followed by neurological improvement. Subsequently, a gradual decline in function and worsening myelopathy occurs, which may be punctuated with recurrent acute hemorrhages.

Treatment Options

Resection has been advocated in all previously published series. Cavernous malformations have a significant rebleeding rate associated with further neurological decline. This hemorrhage rate for symptomatic intramedullary CMs has been reported as 1.4 to 4.5% per year.[30,41] These vascular malformations lead to a progressive myelopathy that can be arrested by surgery.[39] The neurological decline secondary to chronic myelopathy is not as reversible as the acute presentation, although others have reported general improvement in neurological function in all patients.[19,24,30,32,40] It may be that the recurrent hemorrhages irritate the normal spinal cord, so that it becomes gliotic and edematous. The pain associated with these intraspinal lesions does not respond as well to surgery. In a recent publication, investigators found that the long-term control or improvement in pain is only 50%.[21]

There are no alternatives in the treatment of CMs; surgery is the mainstay treatment. The long-term results of microsurgical resection include a stable or improved functional status in 92% of patients, and it is a rare patient whose neurological function deteriorates after surgery. A gliotic plane develops between the lesion and the spinal cord, allowing for relatively safe removal. The lesions usually come to a pial surface, and in all our cases, we were able to use a dorsal laminectomy or osteoplastic laminotomy. A myelotomy is made directly over the discolored area where the malformation comes to the dorsal cord surface. The CM is then removed in an inside-out fashion until the gliotic white matter plane is reached. In patients who have a chronic presentation, the lesion is still surrounded by the gliotic plane, but the spinal cord is extremely atrophic and has a yellow edematous appearance.

Conclusions

Intramedullary spinal CMs are relatively uncommon lesions but are a surgically curable cause of myelopathy. The chronic myelopathy does not generally reverse with surgery, but its progression is halted. These patients are in an extremely precarious situation at the time of presentation, and thus we advocate early surgery to prevent further neurological decline. In patients with an acute hemorrhage, a waiting period of several weeks may facilitate the resection. We do not advocate surgery for incidentally identified lesions or in an asymptomatic patient because of concerns about postoperative pain.

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Manuscript received March 20, 2006.
Accepted in final form May 30, 2006.

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