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Case Report

Exclusively epidural spinal metameric arteriovenous shunts: case report and literature review

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Abstract

BACKGROUND CONTEXT: Spinal arteriovenous metameric syndrome (SAMS) is a subgroup of spinal arteriovenous malformations (AVMs). Most SAMS cases have intra- and extradural AVMs and suffer from hematomyelia, subarachnoid hemorrhage, or venous congestive myelopathy.
PURPOSE: To present a rare case of SAMS in which spinal AVMs were exclusively epidural. We reviewed previous literature and evaluated the feasibility of a treatment strategy using endovascular interventions, followed by surgical obliteration.
STUDY DESIGN: A case report and literature review of SAMS.
METHODS: We report a case of a 15-year-old boy suffering from SAMS in which epidural venous ectasia because of extradural AVMs caused spinal cord compression.
RESULTS: The patient was successfully treated with multiple sessions of transarterial embolization followed by open surgery. After the treatment, his neurologic deficits resolved. Postoperative angiography confirmed complete obliteration of extradural AVMs.
CONCLUSIONS: Although exclusively epidural spinal AVM is an uncommon type of SAMS, combined endovascular and surgical interventions can be an effective treatment for AVMs to achieve better radiologic outcomes and complete resolution of patient symptoms.

Keywords: Arteriovenous malformation; Cobb syndrome; Epidural arteriovenous shunt; Endovascular treatment; Metameric; Myelopathy; Spinal cord

Introduction

Spinal arteriovenous metameric syndrome (SAMS) is a rare clinical form of spinal arteriovenous malformation (AVM) whose hallmarks include spinal vascular malformations and skin nevi in the same dermatome [1–3]. Spinal arteriovenous metameric syndrome is also termed Cobb syndrome based on a case described by Stanley Cobb in 1915 [4]. Most patients with SAMS suffer from intradural spinal vascular pathology [5], which is consistent with the fact that it was classified as "*extradural-intradural arteriovenous malformations*" in one of the most widely used classification schemes [6].

Here we report an unusual case of SAMS exclusively involving epidural spinal AVM. The patient suffered myelopathy because of midthoracic spinal cord compression. Multiple sessions of endovascular interventions followed by surgical obliteration of extradural arteriovenous shunts (AVSs) led to complete regression of the epidural venous engorgement and a good clinical outcome.

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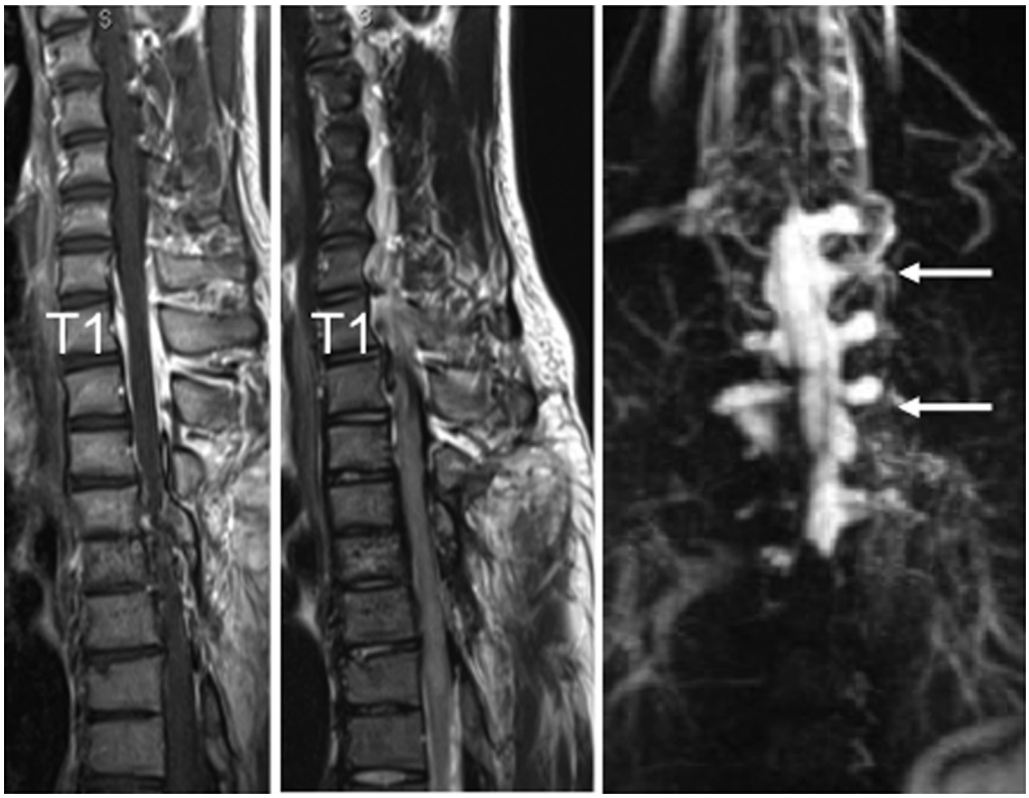

Fig. 1. Magnetic resonance image of the patient's cervical and thoracic spine on admission. (Left) T1-weighted gadolinium enhanced sagittal image showing enlargement of the epidural venous plexus from C7 to T3. (Middle) T2-weighted sagittal image showing no abnormal flow void along the spinal cord surface. There was no T2 high-intensity area in the spinal cord. (Right) Magnetic resonance angiogram demonstrating ectasia of the epidural venous plexus (arrows). ''T1'' indicates (Left and Middle) the vertebral level.

Case report

Presentation

A 15-year-old boy with a history of acute thoracic (T4–T7) epidural hematoma when he was 1 year old was admitted to and treated at our department. At 1 year of age, he had developed acute flaccid paraparesis and the hematoma was surgically resected. According to his record, no vascular malformation was confirmed during the surgery. Although the patient was followed for 5 years postoperatively, angiography was not performed. No spinal or paraspinal vascular anomaly was detected in magnetic

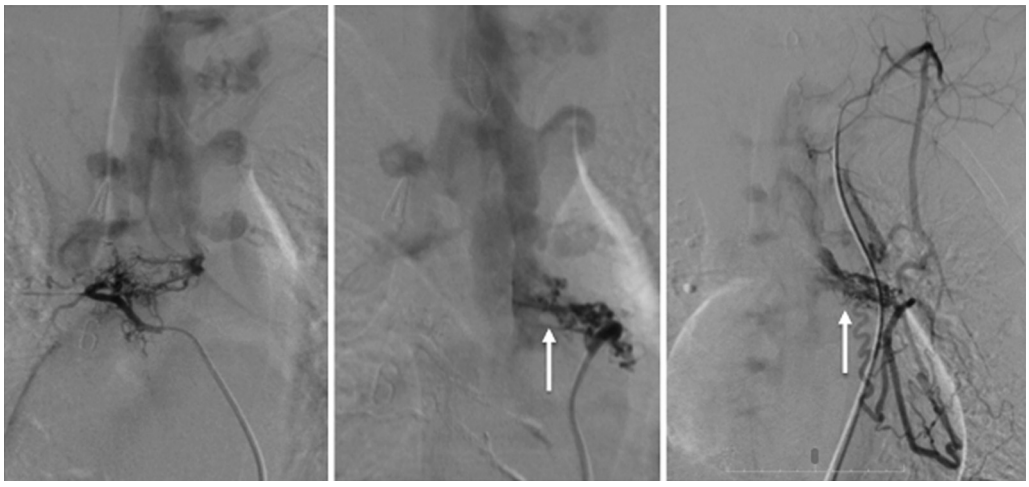

Fig. 2. Preoperative digital subtraction spinal angiograms. Selective angiograms through (Left) right T5 intercostal, (Middle) left T5 intercostal, and (Right) left descending scapular arteries. Epidural arteriovenous shunts were along the left T5 root sleeve (arrows). (Left and Middle) An enlarged epidural venous plexus was also apparent.

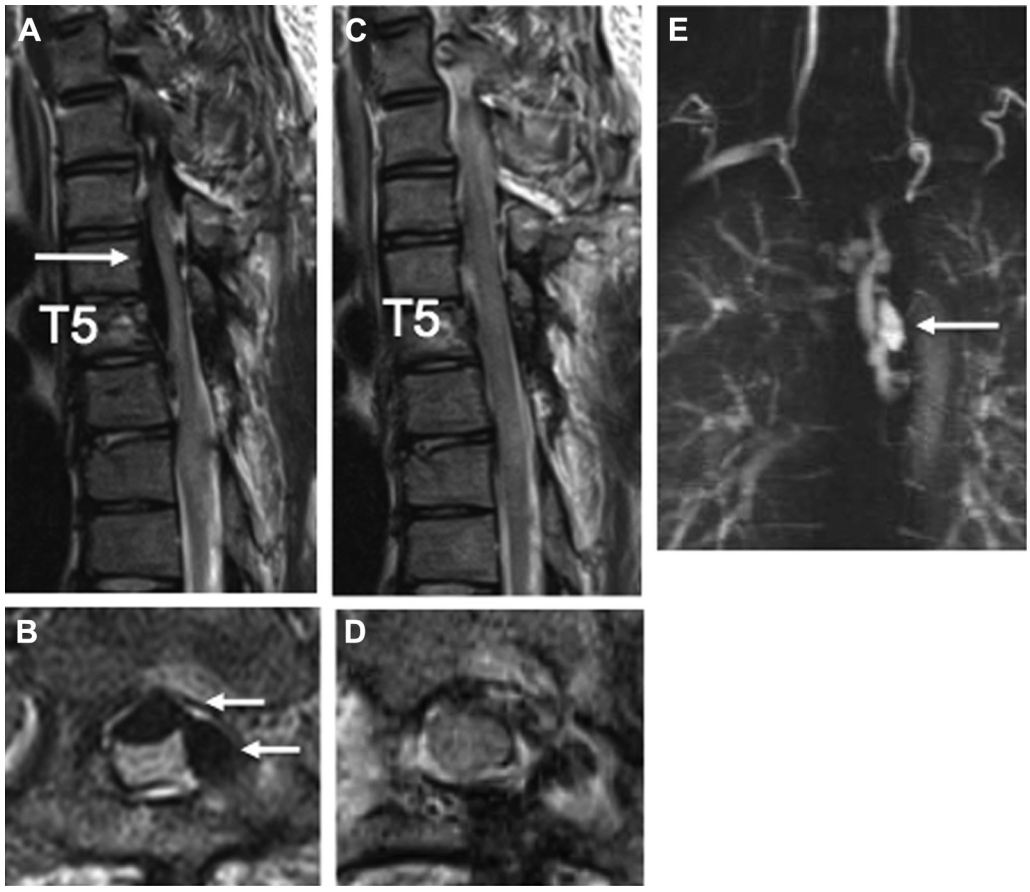

Fig. 3. Magnetic resonance images of the lower cervical and thoracic spine (A, B) before and (C, D) after multiple sessions of endovascular intervention. (A, B) T2-weighted sagittal image (A) showing a newly emerged, low-intensity mass compressing the spinal cord at T4 and T5 levels (arrow) when the patient presented with myelopathy. ''T5'' indicates the vertebral level. An axial image of the (B) T5 level demonstrating a distorted spinal cord and intramedullary T2-hyperintensity. Note the enlarged venous structure compressing the ventral and left lateral spinal cord (arrows). (C, D) Sagittal (C) and axial (D) T2-weighted images confirmed the decrement of the epidural venous plexus and decompression of the spinal cord. (E) Magnetic resonance angiogram demonstrating the remnants of the arteriovenous shunt and venous pouch (arrow).

resonance imaging (MRI) or computed tomography during the clinical course. At 6 years of age, he did not have any neurologic deficits and had normal growth. Thereafter, he was not seen in the outpatient clinic until his referral at 15 years of age.

On presentation at 15 years of age, the patient complained of paresthesia in the left back and arm. He also felt numbness and muscle weakness on the ulnar side of the left hand. The patient did not have any family history.

Examination

On admission, a neurologic examination revealed reduced superficial sensation in areas over the left C8 to T3 and bilateral T4 to T7 dermatomes. The grasping power was diminished in his left hand; however, he had no weakness in his other extremities. There was no Romberg sign or pathologic reflexes. Patellar and Achilles tendon reflexes were normal, and he did not have difficulty in walking or urinating. On his back, there was pigmentation along the previous incision.

Magnetic resonance imaging of the thoracic spine revealed an enhanced area in the epidural space that was most prominent from C7 to T3 vertebral levels (Fig. 1). T2-weighted MRIs showed no abnormal intensity in the spinal cord. There was no intradural signal flow void indicating intradural vascular pathology. Because magnetic resonance angiograms indicated engorgement of the epidural venous plexus, the patient underwent spinal angiography.

Spinal angiograms showed multiple epidural and paraspinal AVSs in the left T4–T7 vertebral segments (Fig. 2). Feeding arteries were bilateral costocervical, bilateral intercostal from T5–T8 segments, and the left descending scapular artery, which caused engorgement of the epidural venous plexus in the upper thoracic levels. Draining routes were through azygos and hemiazygos veins.

Clinical course

The aforementioned examinations led to the diagnosis of SAMS in the midthoracic vertebral level. The patient did

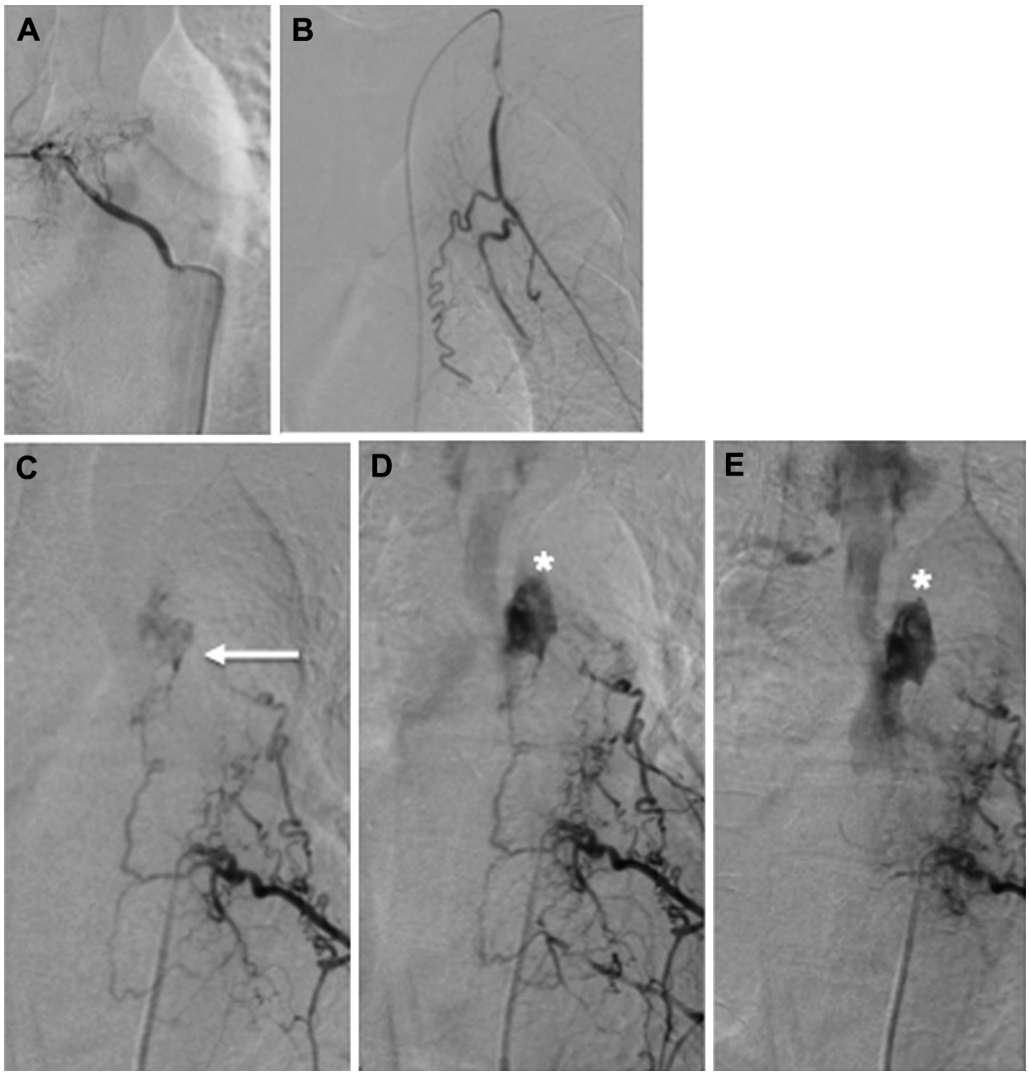

Fig. 4. Digital subtraction angiograms after endovascular intervention. (A, B) Arteriovenous shunts (AVSs) and enlarged venous structures were not evident through selective angiography of the right T5 intercostal (A) and left descending scapular (B) arteries. (C–E) A remnant of the AVS was revealed by left T7 selective angiography. Shunts were localized around the venous pouch at the left T5 in the arterial phase (arrow in C). Emergence of the venous pouch (asterisks) followed by the epidural venous plexus were demonstrated in the late arterial (D) and early venous phase (E).

not show signs of myelopathy, and his symptoms were considered to be because of left C8 radiculopathy. *We speculated C8 nerve root might be compressed by the engorged epidural venous plexus*. Because his symptoms improved gradually, the patient and his family preferred conservative treatments.

Four months after examination, the patient developed difficulty in walking and urinating. Although the patient's paresthesia and motor weakness in his left arm had resolved, he had recently developed motor weakness in his bilateral iliopsoas muscles. Deep tendon reflexes were now brisk in the bilateral lower extremities, and he required a walker for short distances. At this time, the MRI demonstrated enlargement of the epidural venous structure ventral and left of the T4 and T5 spinal cord. The spinal cord was compressed and intramedullary T2-hyperintensity areas were apparent (Fig. 3).

Endovascular interventions

Because the patient now suffered from progressive myelopathy, the decision was made to begin endovascular treatments intended to reduce the shunting flow through AVS and decrement the mass effect of the engorged epidural venous plexus. Transarterial embolizations using *n*-butyl cyanoacrylate (NBCA) were performed over three sessions. After selective catheterization, NBCA (30%) was delivered through segmental arteries of bilateral T4, T5, and T6, the left T7, and bilateral costocervical and left deep scapular arteries.

After endovascular treatment, the enlarged epidural vascular structure at T5 disappeared (Fig. 3). The patient recovered motor strength in his lower extremities and could walk independently, albeit with hyperreflexia. Angiograms showed remarkably reduced blood flow through AVS.

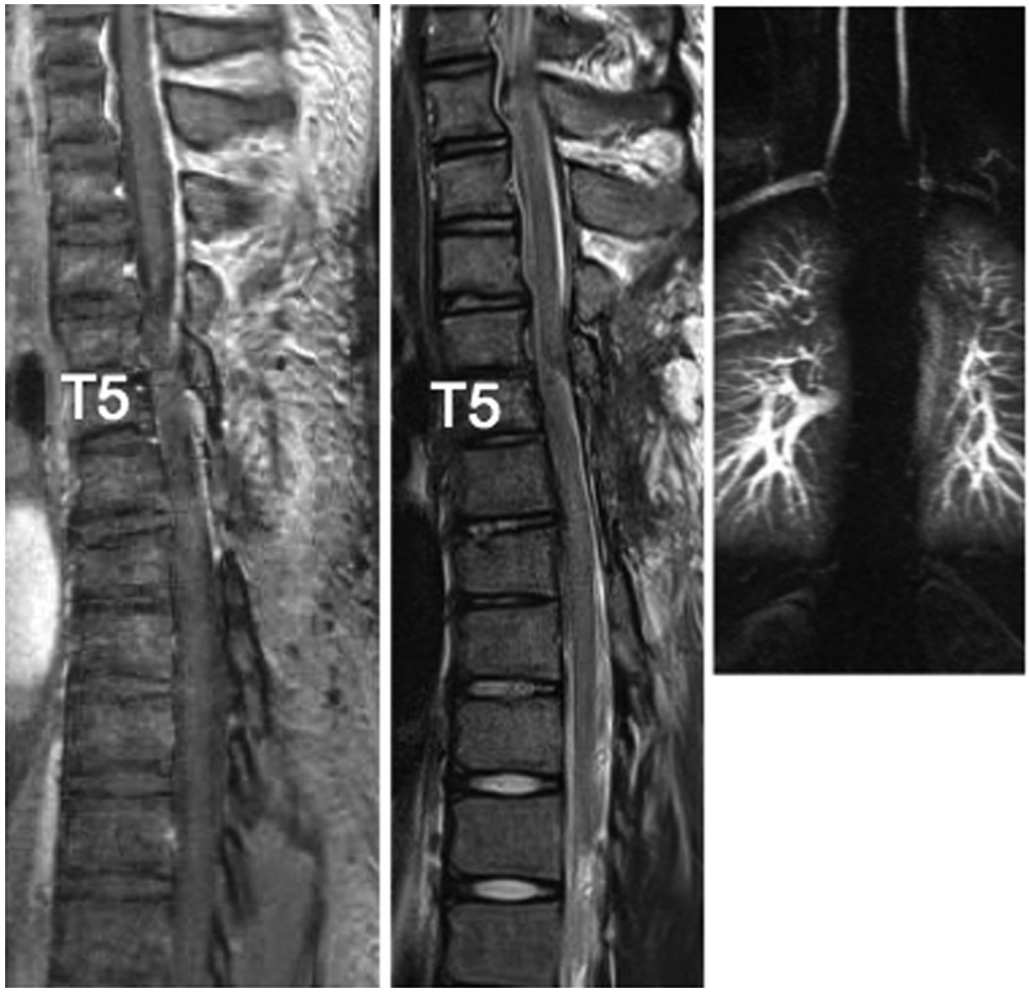

Fig. 5. Magnetic resonance image of the lower cervical and thoracic spine 6 months postoperatively. (Left) T1-weighted gadolinium enhanced sagittal image showing disappearance of the epidural venous plexus. (Middle) T2-weighted sagittal image demonstrating a decompressed spinal cord. There was no T2 high-intensity area in the spinal cord. "T5" indicates (Left and Middle) the vertebral level. (Right) Magnetic resonance angiogram demonstrating no abnormality.

Arteriovenous shunt remnants were localized to the venous pouch medial to the left T5 root sleeve (Fig. 4).

Surgery

Open surgery exposed the venous pouch and obliterated the shunting points around its wall. For monitoring motor and sensory evoked potentials, the patient was positioned prone. Left T5 hemilaminectomy disclosed the pouch. Indocyanine green videoangiography demonstrated early filling of the pouch and epidural venous plexus. The wall of the venous pouch was coagulated circumferentially and detached from the feeder arteries. Finally, it was dissected where the venous pouch flowed into the epidural venous plexus. Indocyanine green videoangiography confirmed the obliteration of the extradural AVS in the surgical field.

Postoperative course

The patient did not develop new neurologic deficits after surgery. Postoperative angiograms demonstrated complete remission of epidural shunts. Six months after intervention, the patient's symptoms and signs of myelopathy completely subsided. Magnetic resonance imaging revealed no recurrence of the spinal AVM (Fig. 5).

Discussion

Spinal arteriovenous metameric syndrome, also termed juvenile metameric or Cobb syndrome, is characterized by multiple vascular malformations that are derived from the same spinal metameric segments [1,2,4,5]. Arteriovenous malformation can be localized in the bone, epidural space, spinal cord, paraspinal soft tissues or muscles, subcutaneous tissues, and skin [1,5,6]. Possible events creating vascular malformations in the same metamere include genetic mutation in the neural crest before migration [7]. Thus, it is classified as "genetic nonhereditary" in criteria proposed by Rodesch et al. [3]. It is estimated that SAMS accounts for approximately 6% to 19% of all spinal cord AVM cases [1,3,5].

Table
Previously reported 24 cases of spinal arteriovenous metameric syndrome

Case	Authors, year	Age, sex	Vertebral level	Pathology	Treatment	Clinical outcome	Radiologic outcome
1	Miyatake et al., 1990 [8]	15 y, F	T5–T7	Intra- and extradural AVM	Embolization	Improved but hypesthesia remained	No remnants, but L1 feeding artery occlusion
2	Shim et al., 1996 [9]	23 y, F	T5–T11	Intra- and extradural AVM	Surgical excision	Improved	Not reported
3	Soeda et al., 2003 [10]	5 mo, F	T8–L3	Intra- and extradural AVM	Embolization	Not able to stand	Residual angioma
4	Pascual-Castroviejo et al., 2002 [11]	17 y, M	C7–T6	Intra- and extradural AVM	Conservative	Not applicable	Not applicable
5	Clark et al., 2008 [12]	8 y, F	T12, L3–L5	Intra- and extradural AVM	Conservative	No data	No data
6	Johnson and Petrie, 2009 [13]	34 y, M	T9–T11	Intra- and extradural AVM	Coiling and embolization	No data after recurrence	Not reported
7	Dilme-Carreras et al., 2010 [14]	12 y, M	T8–L1	Intra- and extradural AVM	Conservative	Not applicable	Not applicable
8	Gomez et al., 2011 [15]	34 y, M	C6–T5	Intra- and extradural AVM	Embolization	Improved but urinary symptoms remained	Residual AVM
9	Schirmer et al., 2012 [16]	17 y, M	T12–L5	Intra- and extradural AVM	Embolization and surgical excision	Improved but ambulating with cane	Residual AVM
10	Linfante et al., 2012 [17]	14 y, F	T5–T8	Intra- and extradural AVM	Embolization	Improved, able to ambulate	Residual AVM
11	Niimi et al., 2013 [5]	19 y, M	C2–C5	Intra- and extradural AVM	Embolization	Improved but not completely cured.	Residual AVM
12	Niimi et al., 2013 [5]	24 y, F	C7, T1	Intra- and extradural AVM	Conservative	Not applicable	Not applicable
13	Clinton et al., 2003 [18]	19 y, M	T2–T5	Intramedullary cavernous angioma	Surgical excision	Gradual improvement	Total resection
14	Gatzonis et al., 2010 [19]	32 y, F	T1,T4–T8	Intramedullary cavernous angioma	Surgical excision	Improved but not completely cured.	Not reported
15	Matsui et al., 2014 [20]	42 y, M	T12–S1	Intramedullary cavernous angioma	Surgical excision	Neurologically free	Residual angiomas
16	Maramattom et al., 2005 [21]	17 y, M	C5–C7	Perimedullary AVS	Embolization	No ambulation	Complete obliteration
17	Sayuthi et al., 2006 [22]	28 y, M	C7–T2	Intramedullary AVM	Embolization and surgical excision	No motor function	Complete obliteration
18	Niimi et al., 2013 [5]	39 y, M	L4–S2	Intradural AVM	Embolization	Improved but not completely cured.	Residual AVM
19	Johnson and Petrie, 2009 [13]	29 y, M	T5–T10	Extradural venous angioma	Surgical excision	Neurologically free	Totally resected
20	Basappa, 1996 [23]	26 y, F	C5–T5	Extradural venous angioma	Surgical excision	Improved	Not reported
21	Romeo et al., 2009 [24]	52 y, F	C3–C5	Epidural hematoma	Surgical excision	Good improvement	Not reported
22	Spiotta et al., 2011 [25]	M	L1–L5	Exclusively epidural AVM	Embolization and surgical excision	Neurologically free	Small residual nidus.
23	Fairhall et al., 2010 [26]	22 y, M	T6–T9	Exclusively epidural AVM	Embolization and surgical excision	Neurologically free	Complete obliteration
24	Present case	15 y, M	T1–T7	Exclusively epidural AVM	Embolization and surgical excision	Neurologically free	Complete obliteration

F, female; M, male; AVM, arteriovenous malformation; AVS, arteriovenous shunt.

SAMS

The Table summarizes 24 cases of previously reported SAMS in English literature after 1990 [5,11–25]. It can occur in any spinal segment, although lesions are frequently localized to the cervical and thoracic spinal levels. Furthermore, most spinal cord vascular pathologies are intradural. Among the 24 cases in the Table, 18 had intradural pathology (75.0%), including intramedullary AVM, perimedullary AVS, and cavernous angiomas [5,18–22]. Common clinical presentations of SAMS include subarachnoid hemorrhage, hematomyelia, or myelopathy because of venous hypertension of the spinal cord [5]. A higher incidence of intradural vascular pathology accounts for these symptoms. When patients suffer extradural pathology, as demonstrated in our case, extradural venous engorgement compresses the spinal cord or nerve root causing myelopathy or radiculopathy, respectively [13,23–26].

Treatment: endovascular

Because of its rarity and poorly understood pathophysiology, optimal management of SAMS remains to be established [10,21]. In general, endovascular interventions are the first treatment of choice as many authors have argued that surgical resection carries high morbidity [17,22,26]. In fact, surgical excisions were specifically indicated to remove intramedullary cavernous angiomas (three cases) [18–20], extradural venous angiomas (two cases) [13,23], or hematoma (one case) [24] in previous reports (Table). Complete obliteration of fistulas in SAMS is not a primary goal of the treatment because of the complexity and diversity of AVMs [5,27]. Rather, intervention is aimed at halting the progression of neurologic symptoms and minimizing neurologic sequelae.

As shown in the Table, among eight cases treated solely with endovascular interventions [5,8,10,15–21], none of them achieved complete fistula obliteration and good clinical outcome at the same time. With the recent development of endovascular techniques and materials used in interventions [28,29], selective catheterization of a small feeding artery, including the anterior spinal artery, can be safely performed [30]. Previously reported cases (Table) demonstrated that embolization with NBCA was a safe and feasible option in SAMS treatment [31,32]. Careful assessment of patient symptoms and causative vascular pathology is required to perform embolization at the appropriate time.

Treatment: surgery

In five cases, including our case, surgical excision was combined with endovascular interventions to treat intra- and extradural pathologies [16,22,25,26]. After surgical excisions, three cases (60%) achieved complete obliterations of the AVM. Although one case retained severe motor impairment [22], four cases had good neurologic outcomes. Importantly, those who had complete resolution of neurologic symptoms and satisfactory radiologic outcomes shared the same characteristics; they all had exclusively epidural AVMs [25,26]. As shown in the present case, blood flow into the AVM decreased remarkably after transarterial embolization. Arteriovenous malformations became angiographically simplified and were more easily localized, which favored surgical treatment. Only after endovascular treatment were we able to locate the shunts on the wall of the venous pouch next to the T5 root sleeve and safely perform the surgical resection.

Conclusions

The subgroup of SAMS with exclusively extradural AVMs can achieve good treatment outcomes compared with patients with SAMS with intra- and extradural AVMs. Therefore, it is important to properly diagnose whether AVMs are truly and completely extradural in patients with SAMS. If so, we recommend applying multiple sessions of transarterial embolizations, followed by surgical resection.

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