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Cavernous Angiomas of the Cauda Equina: Clinical Characteristics and Surgical Outcomes

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Abstract

Cavernous angioma (CA) is a rare hamartomatous vascular lesion, consisting of abnormal, dilated, and packed sinusoidal vascular channels without interposed nervous tissue. CAs of the cauda equina are exceedingly rare and have been previously reported in the literature as case reports. The aim of this study was to discuss the clinical presentation and the outcomes of microsurgery for these rare lesions. We retrospectively reviewed the records of 10 patients who underwent microsurgery for CAs of the cauda equina. All patients had performed pre- and postoperative magnetic resonance imaging (MRI). CAs of the cauda equina generally exhibited mixed intensity on T_1- and T_2-weighted images. Contrast-enhanced T_1-weighted images showed heterogeneous enhancement. The hemosiderin ring which surrounded the cauda equina CA was rare. Gross total resection was achieved in all cases. All patients were followed up, with a mean duration of 41.1 months. Long-term neurological function was improved in nine patients and remained stable in one patient. No recurrence was observed on MRI. CAs should be considered in the differential diagnosis of cauda equina tumors. Because of the excessive vascularity of CAs, en bloc resection is recommended. For symptomatic patients, early surgery should be performed before neurological deficits deteriorate.

Key words: cauda equina, cavernous angioma, long-term outcome, surgical resection

Introduction

Cavernous angioma (CA) is a rare hamartomatous vascular lesion, consisting of dilated thin-walled sinusoidal vascular spaces lacking intervening nervous tissue.[1,2] These lesions are usually located in the tentorium cerebellum and subcortical white matter. In the spine, CA predominantly affects the vertebral bodies with occasional extension into the epidural space.[3] Only about 3% of spinal CAs occur in the intradural space, most of which are intramedullary.[4]

CAs of the cauda equina are very rare. To our knowledge, only 14 cases have been described in the English literature since the earliest report in 1987,[3,5–16] and most previous studies are case reports with an associated literature review. We took into account only the lesions arising from below conus medullaris, excluding the lesions that originated from conus medullaris with subsequent caudal extension to the cauda equina. The detailed clinical features are summarized in Table 1. Because of the rarity of cauda equina CAs, the treatment experience is limited. In this series, we present the data of 10 patients with histologically proven cauda equina CAs and their long-term outcomes from a single center.

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Methods

The study was approved by Institutional Review Board of Beijing Tiantan Hospital, Capital Medical University. We retrospectively reviewed the medical records and radiological studies of 10 patients with cauda equina CAs who underwent microsurgery in our department from 2007 to 2013. All patients had performed pre- and postoperative MRI of the spine as the standard radiological investigation. Patients were included in the study based on the following criteria: (1) there was an intradural-extramedullary lesion present in the preoperative imaging studies, (2) there was an intraoperative confirmation of a pure cauda equina lesion, and (3) there was a postoperative pathological diagnosis of CA.

Surgery was performed in all patients through posterior approach with intraoperative monitoring of somatosensory and motor evoked potentials.

Table 1 Review of cavernous angiomas in the cauda equina previously reported in the English literature

Authors & year	Age (yrs)/ sex	Location	Symptoms	Therapy	Origin	Outcome	FU
Ueda et al. (1987)[16]	28/M	L1–2	SAH (headache); LBP	GTR	Root	Good recovery	3 weeks
Ramos et al. (1990)[14]	67/F	L3	Hydrocephalus	GTR	Filum terminale	Good recovery	3 years
Bruni et al. (1994)[5]	28/M	L2	SAH	GTR	Root	Good recovery	7 days
Cervoni et al. (1995)[8]	26/F	L1–2	SAH	GTR	Root	Good recovery	Discharge from hospital
Cervoni et al. (1995)[8]	32/M	L5	Pain	GTR	Root	Incomplete recovery	6 months
Rao et al. (1997)[15]	60/M	L1–3	Motor deficit	GTR	Root	Good recovery	Not mentioned
Duke et al. (1998)[10]	49/F	L4	LBP; sensory deficit; sphincther dysfunction	GTR	Root	Good recovery	3 months
Falavigna et al. (2004)[3]	44/F	L3–4	LBP; sensory deficit; sphincther dysfunction	GTR with attached root	Root	Good recovery	6 months
Caroli et al. (2007)[6]	71/M	L4	LBP; sensory deficit	GTR	Root	Good recovery	1 year
Miyake et al. (2007)[11]	18/M	L1	LBP; bil legs pain	GTR with attached root	Root	Good recovery	4 months
Cecchi et al. (2007)[7]	75/F	L3–4	Bil legs numbness	GTR with attached root	Root	Good recovery	Not mentioned
Chun et al. (2010)[9]	74/M	L4	Sciatic pain	GTR with attached root	Root	Good recovery	Not mentioned
Nie et al. (2012)[12]	57/M	L1	LBP; bil legs pain	GTR	Root	Good recovery	6 months
Popescu et al. (2013)[13]	60/M	L4	LBP	GTR	Root	Good recovery	2 years

Age, sex, location, and degree of surgical resection are shown for each case. Authors and year of publication are shown for each case reported. bil: bilateral, FU: follow-up, GTR: gross total resection, L: lumbar, LBP: low back pain, Lt: left, Rt: right, SAH: subarachnoid hemorrhage.

Histological specimens were sent to the Department of Pathology for histological confirmation.

Follow-up data for all patients were obtained during individual office visits and telephone interviews. Modified Japanese Orthopedic Association (JOA) scores (Table 2) were applied to assess neurological function.[17] This assessment and MRI were performed before surgery, at discharge, at 3 months after surgery and annually thereafter. All cases were reassessed at a last clinical follow-up in February 2014.

Results

I. Clinical presentation

This case series consisted of five men and five women; and the mean age at the time of the operation was 40.3 years (range, 23–59 years). During the same period, 3,072 patients were diagnosed with intraspinal tumors and 157 patients were diagnosed with intraspinal CAs. The duration of symptoms preceding the initial diagnosis ranged from 3 days to 10 years. The clinical course of cauda equina CAs showed two patterns: a chronically progressive pattern with or without sudden deterioration (n = 7), and an acute pattern (n = 3). The main clinical symptoms of chronically progressive pattern included root pain in seven patients, sensory change in four patients, motor deficit in three patients, and sphincter dysfunction in two patients. Three patients developed subarachnoid hemorrhage (SAH) with acute onset of symptomatology (headache, low-back as well as leg pain, nuchal rigidity, and vomiting without alteration in consciousness). Lumbar puncture revealed bloody cerebrospinal fluid (CSF) in the three patients. The preoperative JOA score was 12.3 ± 1.85 (range, 9–14). The detailed clinical profiles are summarized in Table 3.

Table 2 Modified Japanese Orthopedic Association scale[17]

Section	Score (points)
Motor function of upper extremity	
Unable to feed oneself	0
Unable to use knife and fork; able to eat with a spoon	1
Able to use knife and fork with much difficulty	2
Able to use knife and fork with slight difficulty	3
Normal	4
Motor function of lower extremity	
Unable to walk	0
Can walk on flat floor with walking aid	1
Can walk up and/or down stairs with handrail	2
Lack of stability and smooth gait	3
Normal	4
Sensory function of upper extremity	
Severe sensory loss or pain	0
Mild sensory loss	1
Normal	2
Sensory function of lower extremity	
Severe sensory loss or pain	0
Mild sensory loss	1
Normal	2
Sensory function of trunk extremity	
Severe sensory loss or pain	0
Mild sensory loss	1
Normal	2
Bladder function	
Unable to void	0
Marked difficulty in micturition (retension)	1
Difficulty in micturition (frequency, hesitation)	2
Normal	3

Total score of 17 points.

II. Radiological findings

Cranial computerized tomography (CT) of the three cases of acute pattern disclosed nothing abnormal. Preoperative MRI was available for all patients. On MRI, these lesions were lobulated or nodular shaped, well-circumscribed, and grow eccentrically within the spinal canal. The lesion was isointense in two cases, iso- to hyperintense in four cases, and iso- to hypointense in four cases on the T_1-weighted images. The T_2-weighted images revealed the lesion was iso- to hypointense in four cases, iso- to hyperintense in six cases. In two cases, the lesion was surrounded by a hypointense hemosiderin ring on the T_2-weighted images. Contrast-enhanced T_1-weighted images revealed five cases with markedly heterogeneous enhancement and five cases with mild heterogeneous enhancement. According to the preoperative MRI, only two cases were diagnosed as CAs (two cases, 20%). The differential diagnosis included ependymomas (six cases, 60%) and schwannomas (two cases, 20%). Pre- and postoperative MR images in two patients (case 1 and case 10) are illustrated in Figs. 1–4.

III. Operation and pathological findings

All patients underwent a laminectomy through the posterior approach. After opening the dura mater, the lesions were located in the cauda equina and seemed to arise from attached nerve roots. No attachment to dura mater was found. The intraoperative findings were uniform, showing well-circumscribed, purple-reddish, mulberry or nodular shaped lesions with rich blood supply (Fig. 5). In one case of acute pattern, a reddish pigment suggesting subacute

Table 3 Characteristics of 10 patients with cavernous angiomas of the cauda equina

Case no.	Age (yrs)/ sex	Clinical symptoms	Duration of illness	Site	MRI findings			Preoperative diagnosis	Treatment	Modified JOA scores			FU (mos)
					T_1WI	T_2WI	+GA			Pre-	Post-	Last-FU	
1	42/M	LBP; bil legs pain and weakness	2 years	L3	Iso-hypo	Iso-hyper	Mildly; heterogeneous	Ependymoma	GTR with attached rootlet	11	13	16	71
2	31/F	SAH (Headache; nuchal rigidity; LBP; rt leg pain)	3 days	L2–3	Iso-hypo	Iso-hyper	Markedly; heterogeneous	Ependymoma	GTR	14	14	17	61
3	28/F	SAH (Headache; nuchal rigidity; LBP; bil legs pain)	4 days	L1–2	Iso-hypo	Iso-hyper	Markedly; heterogeneous	Ependymoma	GTR	13	14	16	57

(*Continued*)

Table 3 (Continued)

Case no.	Age (yrs)/ sex	Clinical symptoms	Duration of illness	Site	MRI findings			Preoperative diagnosis	Treatment	Modified JOA scores			FU (mos)
					T_1WI	T_2WI	+GA			Pre-	Post-	Last-FU	
4	27/M	LBP with radiation to rt leg; difficulty in urination	10 years	L2	Iso-hyper	Iso-hypo; hypointense ring	Mildly; heterogeneous	Cavernous angioma	GTR	9	7	9	53
5	23/M	SAH (headache; nuchal rigidity; LBP with radiation to bil legs)	5 days	L1–2	Iso-hyper	Iso-hypo	Markedly; heterogeneous	Ependymoma	GTR	13	13	17	48
6	49/F	Rt leg pain and numbness; difficulty in urination	4 years	L2–3	Iso-hypo	Iso-hyper	Markedly; heterogeneous	Ependymoma	GTR	9	7	15	37
7	33/M	LBP; rt leg pain and numbness	2 years	L2	Iso-hyper	Iso-hypo	Markedly; heterogeneous	Ependymoma	GTR with attached rootlet	14	14	17	33
8	51/F	LBP with radiation to bil legs; bil legs numbness	2 years	L1–2	Iso	Iso-hyper	Mildly; heterogeneous	Schwannoma	GTR	13	15	16	26
9	42/F	Bil legs pain and numbness	1 year	L2–3	Iso	Iso-hyper	Mildly; heterogeneous	Schwannoma	GTR	14	14	17	17
10	59/M	LBP; lt leg pain and weakness	1 year	L3	Iso-hyper	Iso-hypo; hypointense ring	Mildly; heterogeneous	Cavernous angioma	GTR with attached rootlet	13	15	17	8

bil: bilateral, FU: follow-up, GA: Gadolinium administration, GTR: gross total resection, hyper: hyperintense, hypo: hypointense, iso: isointense, JOA: Japanese Orthopedic Association, LBP: low back pain, Lt: left, pre: preoperative, post: postoperative, Rt: right, SAH: subarachnoid hemorrhage, WI: weighted image.

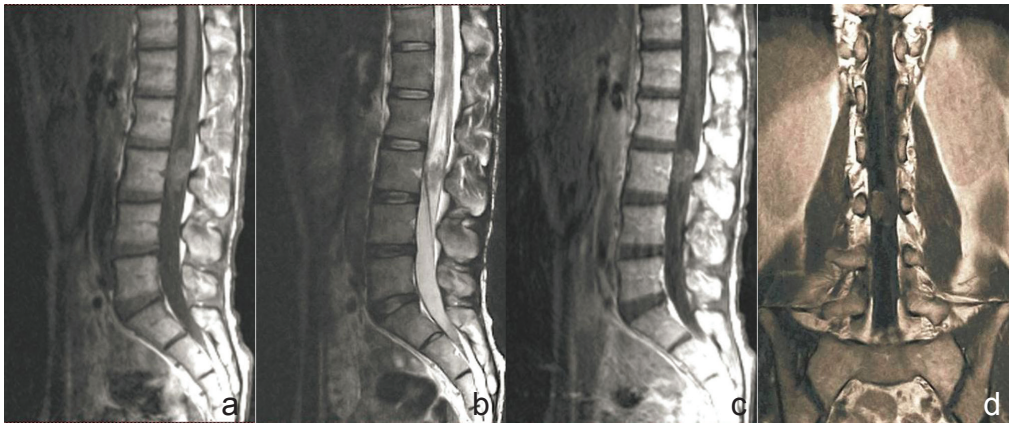

Fig. 1 Case 1: Preoperative magnetic resonance imaging revealed a well-circumscribed lesion adherent to the nerve roots at the L3. The lesion was iso- to hypointense on T_1-weighted image (WI) (a) and iso- to hyperintense on T_2WI (b). Heterogeneous enhancement was observed on the contrast-enhanced T_1WI (c). Coronary contrast-enhanced T_1WI demonstrated the lesion was located on the right-side of the spinal canal (d).

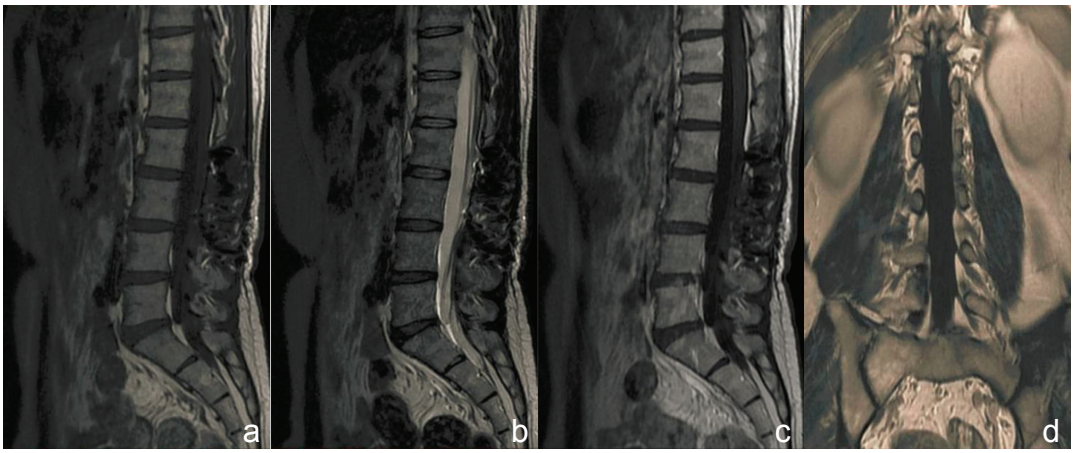

Fig. 2 Five years after surgery, magnetic resonance imaging showed no recurrence of the lesion and the cauda equina had decompressed (a: T_1WI, b: T_2WI, c: contrast-enhanced T_1WI, d: coronary contrast-enhanced T_1WI). WI: weighted image.

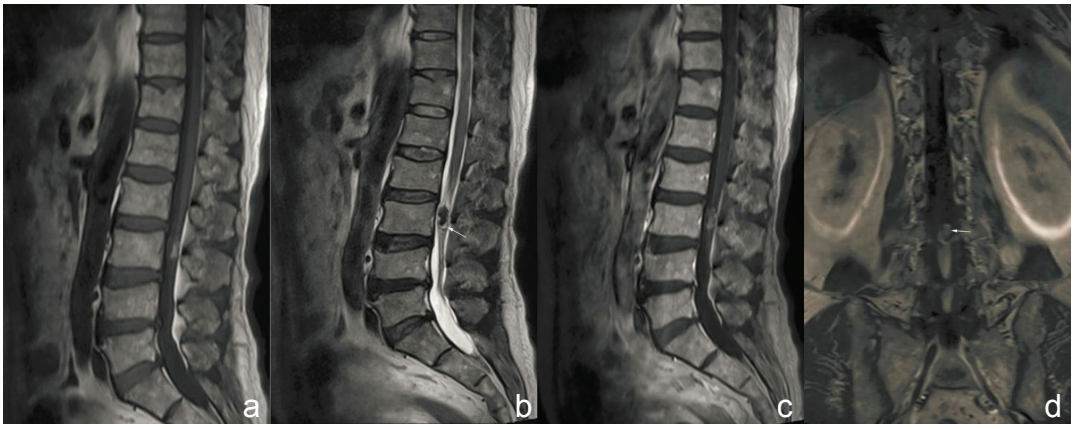

Fig. 3 Case 10: Preoperative magnetic resonance imaging revealed a well-circumscribed lesion at L3. The lesion was iso- to hyperintense on T_1WI (a) and iso- to hypointense on T_2WI (b). The thin irregular hypointense hemosiderin ring (*arrow*) was observed on T_2WI. Heterogeneous enhancement was observed on the contrast-enhanced T_1WI (c). Coronary contrast-enhanced T_1WI demonstrated the lesion (*arrow*) was located on the left-side of the spinal canal (d). WI: weighted image.

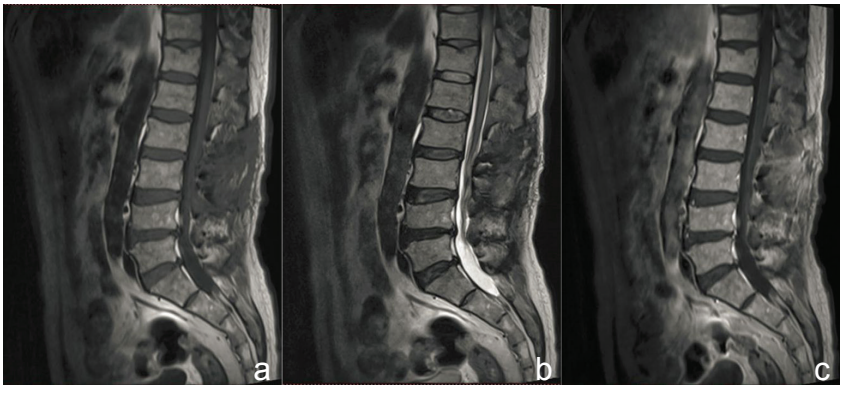

Fig. 4 Eights months after surgery, magnetic resonance imaging showed no recurrence of the lesion and that the cauda equina had decompressed (a: T_1WI, b: T_2WI, c: contrast-enhanced T_1WI). WI: weighted image.

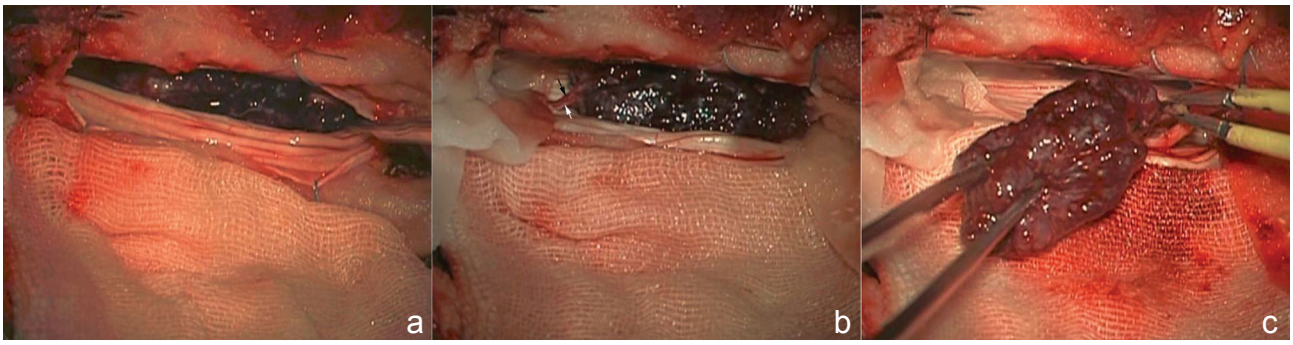

Fig. 5 Case 1: Intraoperative photographs. A L2–3 laminotomy and midline dural opening showed a purple-reddish, mulberry-shaped lesion behind nerve roots in the cauda equina (a). The attached nerve rootlets (*white arrow*) were closely adhered to the lesion and a cluster of vessels was visualized on the lesion (*black arrow*) (b). The nerve rootlet of origin was transected to remove the lesion en bloc (c).

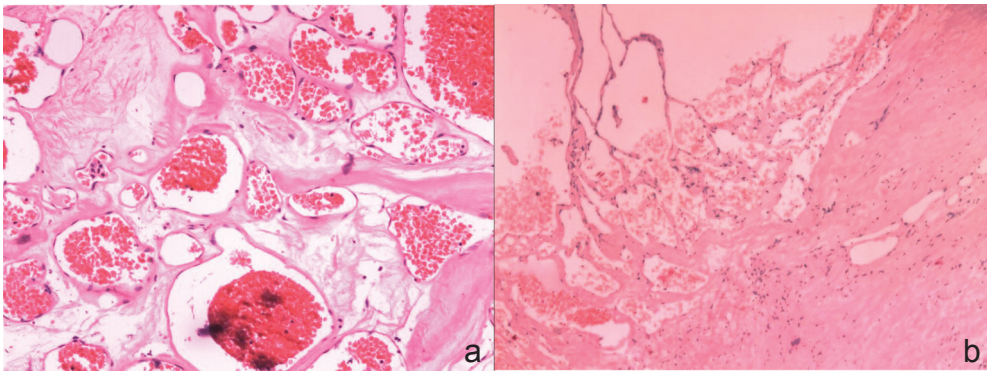

Fig. 6 Photomicrographs of the surgical specimens illustrated that all lesions consisted of variable sized dilated vascular channels with thin or hyalinized wall, which were lined by flattened endothelial cells and contain blood clots or organizing thrombi. (a: case 1, b: case 10. Hematoxylin and eosin stain, original magnification ×200).

hemorrhage was found under the arachnoid membrane. The bulk would gradually shrink through bipolar coagulation of its surface. Gross total resection (GTR) was achieved in all cases. In seven patients (70%), the lesion was easily dissected from attached nerve roots, and the rootlets were preserved. In the other three patients (30%), the attached thin rootlets appeared densely adherent to the lesion, which made it impossible to preserve. In these difficult cases, the lesions were completely resected after sacrificing rootlets. Microscopically, each lesion was composed of a large number of thin-walled vascular channels in collagenous connective tissue, lined by a single layer of epithelial cells. The vascular channel contained red cells or thrombi and the intervening area in the vascular spaces did not reveal any neural tissue (Fig. 6).

IV. Postoperative evaluation

Soon after surgery, four patients experienced immediate improvement and four patients remained unchanged from the preoperative condition. Neurologic deterioration after surgery was seen in two patients with sphincter dysfunction (case 4 and case 6). All patients received medication treatments (30 mg edaravone, intravenous injection, twice daily; 100 mg vitamin B1, intramuscular injection, once daily; 500 mg mecobalamin, oral administration, three times daily; 50 mg diclofenac sodium enteric-coated tablets, oral administration, three times daily, when necessary). Meningitis occurred in one patient after surgery, and was treated successfully by antibiotics and lumbar drainage. Five patients received rehabilitation, which was initiated at a median of 18 days (range 15–33 days) post-surgery. During a mean follow-up period of 41.1 months, neurological status had markedly improved in nine patients and was stable in one patient compared with their preoperative neurological deficits. However, some patients did complain of motor deficit (one case, 10%), sensory deficit (two cases, 20%), and sphincter dysfunction

(one case, 10%). At the last assessment, the postoperative JOA score was 15.7 ± 2.3 (range, 9–17) of the 10 patients. The final JOA score was significantly improved ($p < 0.05$) over the preoperative JOA score. However, the final JOA score remained stable in one patient (10%). Postoperative MRI results showed no recurrence in all cases. Surgical outcomes and assessment of neurological functions are summarized in Table 3.

Discussion

I. Review of the literature

Table 1 provides a summary of the 14 reported cases of cauda equina CAs. Patients in their 3rd–6th decades of life were the most affected, with an unexplained slight male predominance. Progressive radiculopathy was the major symptom in 10 cases. Acute SAH was observed in three cases.[5,8,16] As a unique case, one patient had symptoms of elevated intracranial pressure resulting from hydrocephalus caused by small repeated hemorrhages.[14] Intraoperatively, lesions in 13 cases were adherent to the nerve roots and one lesion was adherent to filum terminale. In the three patients presented with SAH, the mass was surrounded by a recent bleeding in one case and a brown pigment suggesting repeated hemorrhage under the arachnoid membrane in another case.[5,16] In addition, we found no other differences in operative findings, pathologic characteristics, and radiological features between the slow progressive pattern and acute pattern. GTR was achieved in all cases, and resection was performed in four cases where the lesion was tightly adhered to the nerve roots. Excellent postoperative recoveries were seen in 92.9% cases.

II. Epidemiology and clinical futures

CAs are not true neoplasms, as they grow on the basis of hypertrophy and not due to cellular anaplasia. Spinal CAs are relatively rare, accounting for 5–12% of spinal vascular anomalies.[4] Cauda equina CAs are extremely rare. Department of Neurosurgery, Beijing Tiantan Hospital is the most renowned and largest neurosurgical center in China. As such, we have more opportunities to see cases of rare spinal vascular lesions. Our series adds 10 cases, which is a substantial addition to the existing literature. Because many lesions may be asymptomatic, the actual incidence of cauda equina CAs is difficult to know. In the current study, symptomatic cauda equina CAs constituted 0.32% of all intraspinal tumors and 6.4% of all intraspinal CAs. The age ranged from 23 years to 59 years, with a mean age of 38.5 years, which was close to that in the literature. However, there was no sex predilection, which was different from those in published literature.

The presented symptomatology also showed two patterns: a slow progressive pattern (n = 7) and an acute pattern (n = 3). The slow progressive pattern, with progressive cauda equina compression, is usually caused by slow enlargement of the lesion. The slow enlargement of the lesion was thought to be caused by a consequence of small repeated intralesional hemorrhage, endothelial proliferation, and neoangiogenesis.[7,18] In 80% of the present cases, the symptoms at onset were low-back pain and radiculopathy associated with or without motor deficit. Sphincter dysfunction eventually appeared in the late stages. Regardless of the duration, sudden deterioration might occur in the slowly progressive course. The sudden deterioration may be due to acute intralesional hemorrhage or thrombotic venous occlusion causing sudden increase in the bulk of the lesion.[10,11,19] The acute pattern, with a sudden onset of neurological deterioration, is usually caused by SAH. The bleeding rate for symptomatic intramedullary CAs has been reported to be 4.5% per year.[20] However, acute symptoms are mostly caused by intramedullary hemorrhage rather than SAH.[2] The hemorrhagic risks of cauda equina CAs are just beginning to be elucidated. In our series, 30% of cauda equina CAs developed SAH with acute onset of symptomatology. Cauda equina CAs seem more likely to cause SAH than intramedullary CAs because there is no nervous tissue preventing direct exposure to the subarachnoid space.[21] Bruni et al. postulated that apart from the vascular structure of CA, a dynamic mechanism caused by the extreme mobility of the lumbosacral spine may correlate with the tendency of SAH.[5] However, this needs to be confirmed further.

III. Pathogenesis

The occurrence of cauda equina CAs is exceedingly rare. The pathogenesis of this rare lesion is still unknown. Some theories of etiology suggested that CAs may originate from dysplasia blood vessel progenitors of the caudal nerve roots, the inner surface of the dura, and pial surface of the spinal cord.[9,19,22,23] In our series, the lesion was placed in the subarachnoid space, attached to nerve rootlet, and no dura attachment was found. Hence, we agree that this vascular malformation most likely originate from periradicular vessels of nerve rootlet.

IV. Differentiation

Spinal angiography does not reveal CAs.[15] MRI is the modality of choice for these lesions. These

lesions are usually nodular or lobulated shape and grow eccentrically within the spinal canal. Owing to intralesional hemorrhage and calcification, CAs generally exhibit mixed intensity on T_1- and T_2-weighted images. Contrast-enhanced T_1-weighted images show heterogeneous enhancement. A hypointense hemosiderin ring which surrounds the lesion on T_2-weighted images was considered to be a typical characteristic of CAs.[24] However, in our study, only two cases exhibited the thin irregular hemosiderin ring. The mechanism of this phenomenon may be more rapid removal of blood products outside the blood-brain barrier.[25]

Ependymomas and schwannomas occur at a greater frequency in the cauda equina.[26] It is difficult to differentiate CAs from cauda equina ependymomas, which also cause intralesional hemorrhage or SAH.[16,27] However, the distribution of caudal nerve roots in the thecal sac may help in distinguishing these tumors.[28] Ependymomas always push the roots to the periphery of the thecal sac, whereas CAs often push the roots together in an eccentric fashion. Schwannomas are also located eccentrically on spinal nerve roots.[29] However, schwannomas always have a smooth contour and cystic change, extending into the paraspinal region with intravertebral foramen widen, which could be the clue to the differential diagnosis of CAs.[30]

Although CAs are considered to have these radiographic features which should be able to distinguish them from other cauda equina tumors, definitive preoperative diagnosis may still be challenging based only on MRI. An accurate diagnosis still depends on pathological examinations. Histologically, CAs must be distinguished from capillary hemangiomas.[6] Capillary hemangiomas are composed of capillary-sized blood vessels lined by simple endothelium without atypia.[31] CAs consist of large, dilated hyaline vascular channels arranged in diffuse patterns. They often show thrombosis, perivascular hemosiderin deposition, and calcification.[6,32] In the present study, all histological characteristics were consistent with CAs and no significant differences in pathologic characteristics were found between the two clinical patterns.

V. Treatment

Given the tendency of hemorrhage and neurologic worsening, microsurgical resection should be performed for symptomatic patients. GTR was achieved in all reported cases and our series. Once removal was achieved, the surgery field must be carefully examined to identify any residues of the lesion which might give rise to recurrence and further bleeding. Because of the excessive vascularity of CAs, en bloc resection is advocated and piecemeal resection should be avoided.[6] In our study, most lesions were easily dissected from adherent nerve roots, and en bloc resection was achieved with preservation of nerve rootlets. However, if CAs are densely adhered to attached nerve rootlets, dissection of the rootlets that are assumed to be the origin of the lesion is unavoidable. In two cases of our series, the attached rootlets were densely adhered to CAs, which made it impossible to preserve. Nonetheless, resection of the thin rootlets in the current report did not compromise good postoperative recovery.

At the last evaluation, the postoperative JOA scores of most patients had significantly improved; moreover, no recurrence was observed in any patient. Although transient postoperative neurological deterioration was seen in two patients, at the six-month neurological examination, one patient evaluated as normal. However, the symptoms had no improvement in the other patient with sphincter dysfunction. Long-lasting compression and sudden intralesional hemorrhage could cause severe neurologic damage such as sphincter dysfunction. Thus, for symptomatic patients, especially those who had sudden neurological deterioration; early surgery should be performed to prevent further neurological deterioration. Besides, the use of intraoperative neuromonitoring can allow safe resection and decrease the risk of postoperative neurological deficits.[11]

Limitations

This retrospective study was performed in a single institution and the number of patients was relatively small. The best standard for evaluating our treatment policy is a prospective randomized trial, but it is not possible due to the rarity of the lesions. Additionally, detailed operative information in some early cases could not be fully ascertained since their intraoperative findings were based on the medical records without confirmation by operation videos.

Conclusion

CAs should be considered in the differential diagnosis of a middle-aged patient with cauda equina tumors, if the lesion has eccentric localization and heterogeneous enhancement on MRI. Chronically progressive cauda equina compression and acute SAH are main clinical symptoms. Because of the excessive vascularity of CAs, en bloc resection is recommended and piecemeal resection should be avoided. In order to achieve good functional results, early surgery should be performed before neurological

deficits deteriorate. Clinical and radiological follow-up is required to evaluate long-term outcomes.

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Conflicts of Interest Disclosure

The authors have reported no conflicts of interest.

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