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Keywords: natural history; spinal arteriovenous malformation; perimedullary arteriovenous fistula; spinal metameric arteriovenous malformation; arteriovenous shunt

Abbreviations: mALS = modified Aminoff and Logue scale; PMAVF = peri-medullary arteriovenous fistula; SAVM = spinal arteriovenous malformation; SCAVS = spinal cord arteriovenous shunt; SMAVMs = spinal metameric arteriovenous malformation

Introduction

Spinal cord arteriovenous shunts (SCAVSs) are pathological connections between spinal cord arteries and veins without a normal intervening capillary network. They can be subclassified into intramedullary spinal arteriovenous malformations (SAVMs), perimedullary arteriovenous fistulas (PMAVFs) and spinal metameric arteriovenous malformations (SMAVMs) (Bao and Ling, 1997; Rodesch *et al.*, 2002; Kim and Spetzler, 2006; Krings, 2010). These lesions may lead to severe neurological deficits resulting from haemorrhage, spinal venous congestion or cord compression (Rosenblum *et al.*, 1987; Rodesch *et al.*, 2004; Flores *et al.*, 2017; Ho *et al.*, 2018).

Treatment strategy of these lesions is controversially discussed as these are rare diseases with challenging treatment that may in itself result in potentially disabling complications (Boström *et al.*, 2007, 2009; Krings, 2010, Krings *et al.*, 2010; Flores *et al.*, 2017; Ho *et al.*, 2018). Complication rates reported in the literature vary between 5% and 25% irrespective of treatment modality (Flores *et al.*, 2017). Only with comprehensive understanding of the natural history can treatment-associated hazards be weighed against the prognosis, leading to an appropriate decision-making in the individual case. Unfortunately, SCAVSs are very rare diseases: based on an estimation of data from major German referral centre, an analysis of a US hospital database and our own referral pattern, the incidence of SCAVSs is believed to be between 1 and 2.5 per million per year (Thron, 2001; Lad *et al.*, 2009). Given the rarity of SCAVSs, few studies have thus far investigated their natural history (Aminoff and Logue, 1974; Gross and Du, 2013*a*, *b*, 2014; Lee *et al.*, 2014).

The present observational study in an unselected, consecutive patient cohort with SCAVSs admitted to three institutes was undertaken to reveal the natural history of this complex disease, which would provide valuable evidence to inform clinical decision-making.

Materials and methods

The China-INI spinal vascular malformations database is an ongoing prospectively maintained database capturing clinical data on consecutive patients with spinal vascular malformations admitted to three referral centres (Xuanwu Hospital, Beijing Haidian Hospital, and Beijing United Family Hospital). As tertiary centres specializing in spinal vascular diseases, our institutions draw patients from across China. This study was reviewed and approved by the ethics committee of our institutions with waiver of informed consent from patients given its retrospective nature.

The study included patients with symptomatic SCAVSs who were initially admitted to Xuanwu Hospital between January 2007 and December 2017, and to Haidian and Beijing United Family Hospitals between February 2014 and December 2017. Patients were eligible if: (i) their SCAVSs was not detected incidentally; (ii) their symptoms were attributed to the SCAVSs; (iii) they had been initially treated in our centres with an interval of at least 1 month between onset and treatment, or they were not treated; (iv) the location of the shunt was intradural; and (v) the level of lesion was from C1 to the tip of conus medullaries.

Patients with spinal epidural vascular malformations, spinal dural arteriovenous fistulas, spinal radicular arteriovenous fistulas, filum terminale arteriovenous fistulas, spinal cavernous malformations or paravertebral spinal shunts were not included in this study. Patients with concurrent tethered cord, herniated disc, spinal tumour or any other kind of disease that could impair the spinal cord function were excluded, as well as patients without complete spinal digital subtraction angiography (DSA) data. Patients younger than 1 year old were not eligible because their spinal cord function could not be evaluated objectively.

Baseline clinical characteristics, including age of onset and sex, were derived from the database. Angio-architecture features were determined from DICOM DSA. Lesion subtypes included SAVM, PMAVF and SMAVM. Lesion location was classified into the craniocervical cord (C1–C2), the mid-cervical cord (C3–C5), the cervicothoracic cord (C6–T2), the mid-thoracic cord (T3–T9) and the thoracolumbar cord (from T10 to the tip of conus medullaries). The relationship between the major part of each lesion and the spinal cord was defined as ventral, lateral, central, and dorsal.

The observational period was defined as the interval between onset and invasive treatment or last follow-up (for patients who did not receive an invasive treatment). The clinical course during the observational period was evaluated using the modified Aminoff and Logue scale (mALS) (Aminoff and Logue, 1974). The onset pattern was dichotomized into acute and gradual: an acute onset was defined as an increase in mALS of >1 point within 1 day or severe sudden spinal pain of >4 on the numerical rating scale.

We defined the endpoint of the natural history analysis as either further clinical deterioration during the observational period, or invasive treatment (if no deterioration occurred) or last follow-up (if neither deterioration nor invasive treatment occurred). The follow-up period was the interval between

above. SCAVSs differ from brain arteriovenous malformations in that gradual deterioration unrelated to haemorrhage is more common than acute deterioration (Flores *et al.*, 2017; Ho *et al.*, 2018). Thus, in our analysis, clinical deterioration was further divided into acute and gradual, using the same definition for acute versus gradual as used in onset description. Patients with multiple time points of deterioration were censored at their first event. Although observation was continued after an endpoint was reached, further data were not included in the natural history analysis.

To assess the risk of clinical deterioration after treatment, data of treatment and post-treatment follow-up in this series were analysed. Follow-up plan was at discharge, 1 month, 6 months and at yearly intervals through direct interview or telephone contact. Patients with more than 6 months follow-up or death were enrolled in this part of the analysis. Permanent complications were defined as clinical deterioration that occurred within 2 weeks after treatment and sustained for more than 6 months or death.

Statistical analysis

Differences between groups were tested using Pearson's χ^2 test for categorical variables and the Wilcoxon rank sum test for continuous variables. Multivariate models included variables that were significant at $P \leqslant 0.1$ in the univariate analysis for the outcome of interest. The annual risk of clinical deterioration was calculated

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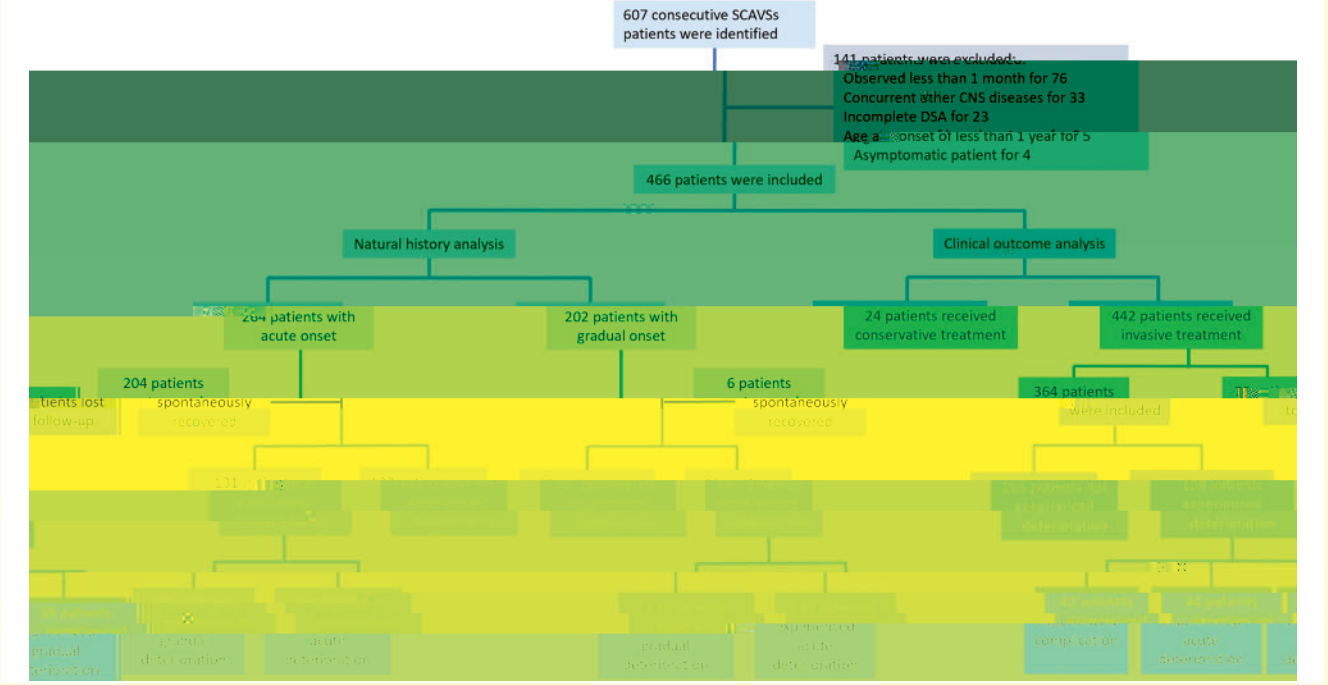

Figure 1 Flow diagram of the clinical course of intradural spinal cord arteriovenous shunts.

Table 1 Baseline characteristics

Characteristic	Total (*n* = 466)	SAVM (*n* = 269)	SMAVM (*n* = 118)	PMAVF (*n* = 79)	*P*-value
Male	283 (60.7)	161 (59.9)	70 (59.3)	52 (65.8)	0.593
Age, years					<0.001
1–14	87 (18.7)	36 (13.4)	24 (20.3)	27 (34.2)	
15–28	226 (48.5)	139 (51.7)	70 (59.3)	17 (21.5)	
>28	153 (32.8)	94 (34.9)	24 (20.3)	35 (44.3)	
Observational period	12.0 (2.3, 48.0)	12.0 (2.3, 48.0)	12.0 (3.0, 48.0)	8.3 (2.0, 32.0)	0.397
Lesion location					<0.001
C1-C2	30 (6.4)	25 (9.3)	4 (3.4)	1 (1.3)	
C3-C5	78 (16.7)	55 (20.4)	18 (15.3)	5 (6.3)	
C6-T2	60 (12.9)	31 (11.5)	22 (18.6)	7 (8.9)	
T3-T9	107 (23.0)	61 (22.7)	31 (26.3)	15 (19.0)	
Lower than T9	191 (41.0)	97 (36.1)	43 (36.4)	51 (64.6)	
Relationship between lesion and spinal cord[a]					–
Ventral	120 (25.8)	64 (23.8)	26 (22.0)	30 (38.0)	
Lateral	78 (16.7)	51 (19.0)	16 (13.6)	11 (13.9)	
Central	122 (26.2)	78 (29.0)	44 (37.3)	0 (0.0)	
Dorsal	146 (31.3)	76 (28.3)	32 (27.1)	38 (48.1)	
Onset pattern					<0.001
Acute	264 (56.7)	164 (61.0)	71 (60.2)	29 (36.7)	
Gradual	202 (43.3)	105 (39.0)	47 (39.8)	50 (63.3)	

[a]The relationship between PMAVF lesions and the spinal cord did not include the 'Central' group, so the difference between three subtypes was not analysed.

Deterioration of spinal cord function

Acute versus gradual deteriorations were observed in 122 (26.2%) versus 212 patients (45.5%) respectively. Twenty-two patients (4.7%) presented with both acute and gradual deterioration during their observational period. The analysis of risk factors of clinical deterioration included three parts.

First, to explore the total risk of clinical deterioration (coined 'general' deterioration), the endpoints of acute and gradual deterioration were combined. For patients who had experienced both patterns, the endpoint was

defined as the earlier event. During the total follow-up period of 1016.24 person-years, 312 patients experienced either acute or gradual clinical deterioration, yielding an annual general deterioration rate of 30.7%. The cumulative clinical deterioration rate was 72.1% in 4 years after initial onset. The risk of general clinical deterioration was higher during the first few months after onset, decreasing thereafter (Fig. 2). The annual general deterioration rate was almost 2.5 times higher during the first 6 months (76.4%) compared to the entire follow-up period.

The second endpoint we evaluated was acute deterioration. During the total follow-up period of 1237.7 person-years, 122 patients experienced acute clinical deterioration, yielding an overall annual rate of 9.9%. The cumulative rate was 32.5% in 4 years after initial onset. The risk of acute deterioration was highest during the first few months after onset. The annual acute deterioration rate was more than two times higher during the first 6 months (21.2%) compared to the entire follow-up period (Fig. 2 and Table 3). Log-rank test and Cox multivariate analysis indicated that an acute onset was the only risk factor for subsequent acute deterioration [hazard ratio (HR) 1.957 (95% CI: 1.324–2.894); $P = 0.0008$] (Fig. 3, Tables 3 and 4, and Supplementary Fig. 2). The annual acute deterioration rates of SAVMs, PMAVFs and SMAVMs were similar. Pairwise comparison log-rank test and multivariate analysis failed to show any significant difference between them (Supplementary Tables 2 and 3). Twenty-three patients (18.9%) with acute deterioration experienced trigger incidents, 60.9% of which directly increased the thoracic and abdominal pressure.

The third endpoint we evaluated was gradual deterioration. During the total follow-up period of 1197.1 person-years, 212 patients experienced gradual deterioration, yielding an annual gradual deterioration rate of 17.7%. The cumulative gradual deterioration rate was 56.2% in the 4 years after initial onset. The risk of gradual deterioration was highest during the first few months after onset, regard-

Table 3 Annual and cumulative rate of acute deterioration

Characteristics	Patients, *n*	Annual deterioration rate (%)										Cumulative deterioration rate (%)	Log-rank *P*-values		
		1–6 months		1–12 months		13–48 months		>48 months		Whole F/U period		1–48 months	Whole F/U period		
		Acute	Gradual	Acute	Gradual	Acute	Gradual	Acute	Gradual	Acute	Gradual	Acute	Gradual	Acute	Gradual
Total	466	21.2	52.1	17.9	39.7	7.3	15.1	7.5	8.2	9.9	17.7	32.54	56.2		
Sex														0.741	0.006
Female	183	18.2	29.2	15.6	23.1	7.0	15.6	7.2	7.2	9.4	13.4	30.90	50.2		
Male	283	23.0	67.3	19.3	51.4	7.7	14.7	7.8	9.1	10.2	21.0	33.51	60.0		
Age at onset, years														0.072	<0.001
1–14	87	27.4	35.3	19.2	26.4	3.7	7.2	7.3	5.0	7.9	8.7	24.00	35.6		
15–28	226	23.7	51.0	22.5	37.8	8.5	13.5	9.3	9.8	12.4	17.8	38.83	53.9		
>28	153	14.2	62.8	10.3	50.6	8.3	26.2	4.1	11.1	7.6	28.8	28.72	71.2		
Onset pattern														<0.001	<0.001
Gradual onset	202	12.8	81.7	10.4	62.7	5.3	22.3	5.6	13.0	6.7	29.8	22.35	71.6		
Acute onset	264	29.7	24.7	26.1	17.9	9.4	9.1	9.1	5.8	12.9	9.2	41.87	36.3		
Lesion location														0.439	0.001
C1–C2	30	30.8	10.1	29.1	11.5	8.7	14.0	0.0	8.0	10.9	11.3	42.70	45.3		
C3–C5	78	22.7	22.2	26.0	24.4	8.8	8.5	7.7	8.3	12.1	11.8	40.10	44.6		
C6–T2	60	32.6	32.4	23.9	24.3	6.9	9.1	7.4	10.9	10.7	13.3	33.30	40.9		
T3–T9	107	18.4	55.6	11.0	37.3	7.3	13.9	4.9	7.5	6.8	15.3	28.89	52.2		
Lower than T9	191	17.4	74.3	15.0	56.4	6.6	21.4	11.7	7.7	10.6	24.7	29.24	68.8		
Subtype														0.748	0.002
SAVM	269	23.5	39.4	18.8	29.5	6.9	16.0	8.3	6.1	10.3	14.2	31.97	53.5		
SMAVM	118	20.4	49.6	17.4	38.9	10.5	14.5	5.0	15.3	9.7	21.2	39.30	55.7		
PMAVF	79	14.2	106.4	15.1	86.0	3.1	11.4	9.1	9.2	8.6	29.4	21.25	66.1		
Relationships between lesion and spinal cord														0.697	0.001
Ventral	120	33.7	47.1	22.9	35.9	6.8	12.2	7.4	9.2	10.3	15.9	34.42	49.6		
Lateral	78	22.3	46.2	21.7	40.4	4.3	10.6	6.0	10.6	8.7	17.1	27.68	51.2		
Central	122	10.4	27.6	15.2	22.9	9.5	13.8	9.9	5.8	11.0	12.2	38.24	48.3		
Dorsal	146	20.6	82.4	14.5	59.9	7.0	23.4	6.3	8.8	8.9	27.1	25.30	71.0		

F/U = follow-up.

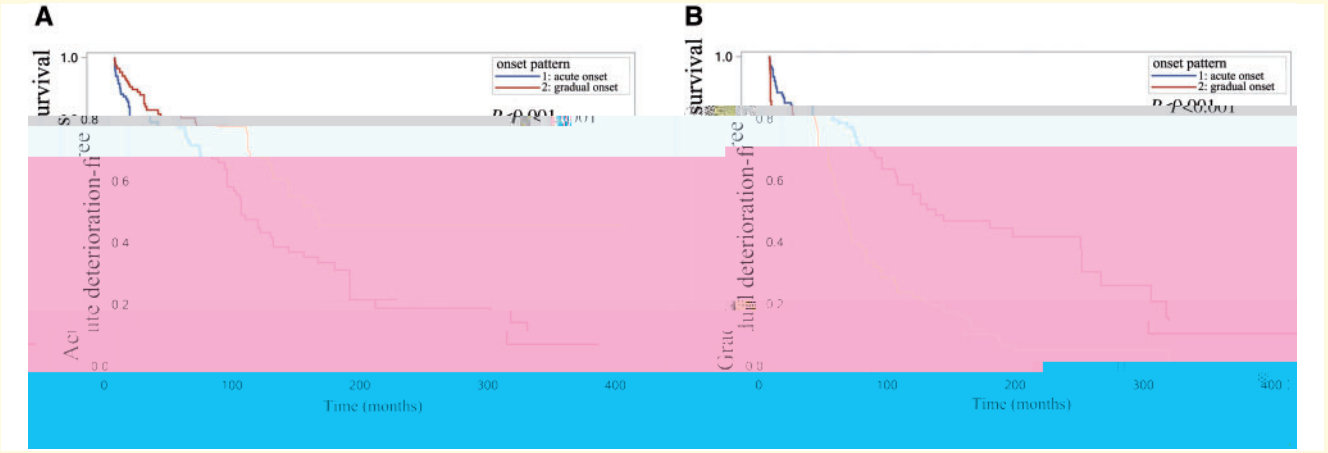

Figure 3 Kaplan-Meier curves demonstrating cumulative rates of acute (A) and gradual (B) clinical deterioration for patients as the function of follow-up time in months, patients were stratified by onset pattern.

Table 4 Multivariable analysis for risk factors of deterioration

Characteristics	Hazard ratio during the whole F/U period (95%CI)	
	Gradual deterioration	Acute deterioration
Male	1.354 (1.008–1.820)*	1.126 (0.777–1.632)
Age		
1–14	Ref[a]	Ref[a]
15–28	1.806 (1.145–2.848)*	1.362 (0.839–2.213)
>28	2.142 (1.347–3.405)**	0.849 (0.466–1.549)
Onset pattern		
Acute onset	Ref[a]	1.957 (1.324–2.894)**
Gradual onset	2.350 (1.711–3.229)***	Ref[a]
Subtype		
SAVM	Ref[a]	Ref[a]
SMAVM	1.247 (0.897–1.733)	0.939 (0.611–1.441)
PMAVF	1.649 (1.093–2.488)*	0.964 (0.520–1.786)
Lesion location		
C1–C2	0.801 (0.373–1.717)	1.485 (0.619–3.560)
C3–C5	0.792 (0.476–1.318)	1.643 (0.902–2.994)
C6–T2	0.854 (0.508–1.435)	1.506 (0.804–2.821)
T3–T9	Ref[a]	Ref[a]
Lower than T9	1.141 (0.799–1.629)	1.454 (0.858–2.465)
Relationships between lesion and spinal cord		
Ventral	Ref[a]	Ref[a]
Lateral	1.356 (0.857–2.146)	0.774 (0.435–1.379)
Central	1.079 (0.704–1.656)	0.936 (0.573–1.531)
Dorsal	1.407 (0.981–2.018)	0.906 (0.547–1.499)

*$P > 0.05$; **$P < 0.005$; ***$P < 0.001$.
[a]Compared as reference.

risk factor of subsequent acute deterioration. In contrast, the majority of patients with gradual onset of symptoms did not spontaneously recover and they were at the highest risk for gradual deterioration. Moreover, Cox multivariate analysis indicated that gradual onset was the strongest predictor for further gradual deterioration. The difference between acute and gradual onset prompt, therefore, a differentiated treatment strategy at our institutions.

The majority of patients who present with acute onset of symptoms harbour a spinal haemorrhage (Rodesch *et al.*, 2004; Ho *et al.*, 2018). For these patients, determining the necessity of emergency decompression surgery is the first challenge for clinicians. Our data indicated that 77% of acute onset patients presented with a favourable spontaneous recovery in the early period after onset. This observation is comparable with the results reported by Rodesch

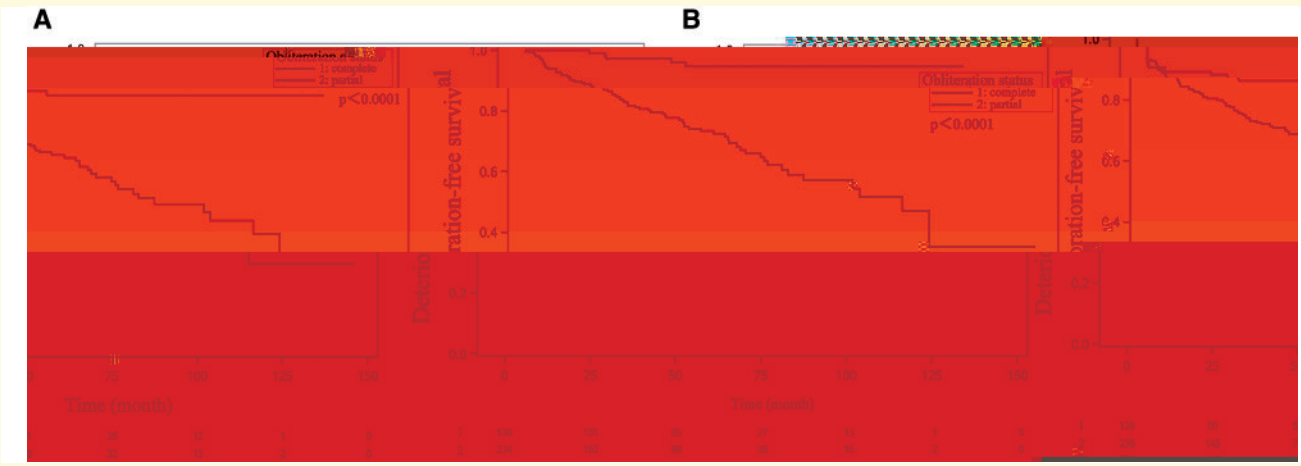

Figure 4 Kaplan-Meier curves demonstrating cumulative rates of post-treatment clinical deterioration for all patients as the function of follow-up time in months, patients were stratified by obliteration status. (**A**) Overall post-treatment clinical deterioration; (**B**) post-treatment clinical deterioration with permanent complications were excluded.

et al. They found a spontaneous recovery rate after haemorrhagic presentation of >70%. Because the limited operative field of emergency surgery resulting from haematoma and swollen cord may lead to higher surgical complication rates and lower obliteration rates of the underlying arteriovenous shunts, these authors argued that emergency open surgery was not indicated (Rodesch *et al.*, 2004). However, given our data in a large cohort of patient, we believe this conclusion may benefit from a more differentiated approach. More than 15% of acute onset patients did not spontaneously recover in the observational period of >2 months. Predictive factors for failure to recover were an initial mALS of >3, age of onset older than 28 years, mid-thoracic and non-ventral lesions. We presume that the above-mentioned factors reflect relatively limited compensatory capability of spinal cord function or a severe primary injury of the spinal cord. For example, the vascularization of mid-thoracic segment is the weakest of the cord (Lasjaunias *et al.*, 2001) and the size of mid-thoracic spinal canal is relatively small among the whole spinal segments (Kang *et al.*, 2012), which means that these segments of the spinal cord are more vulnerable when a haemorrhage occurs. Thus, we believe that, while for the majority of patients emergent open surgery is not necessary, a specific subset of patients does benefit from this strategy. The risk of acute deterioration is significantly increased during the first few months after acute onset, which is similar to brain arteriovenous malformation (Mast *et al.*, 1997; Hernesniemi *et al.*, 2008; da Costa *et al.*, 2009). Therefore, although an emergent open surgery is not necessary for the majority of patients with acute onset, we suggest an early endovascular embolization of weak points such as intranidal aneurysms to prevent subsequent haemorrhage.

For patients presenting with gradual deterioration, the subsequent risk of further deterioration was very high especially during the early period after onset and spontaneous recovery was rather rare. The mechanism of this phenomenon is likely multifactorial and includes venous congestion, progressive venous thrombosis or mass effect (Flores *et al.*, 2017; Ho *et al.*, 2018). However, this finding has important implications for early invasive treatment of these patients particularly if they harbour additional risk factors for further deterioration. Given the pathological mechanism, surgical complications caused by a haematoma are of no concern for these patients; thus, early surgical or endovascular management are advised according to angiographic and anatomical characteristics of the lesions.

The differences in angio-architecture of SAVMs, SMAVMs and PMAVFs affect the complexity of treatment. Therefore, differences in natural history between these entities are of interest. SMAVMs were previously considered as more unstable lesions given their complex nidus anatomy. Niimi *et al.* (2013) investigated the clinical course of 28 SMAVMs patients and found that their haemorrhagic rate was higher compared to non-metameric lesions. This is comparable with our results. In addition, these lesions were more prone to present with multiple hemorrhages, although this result did not reach statistical significance (Niimi *et al.*, 2013). Our data did not show significant differences between the three subtypes of SCAVSs regarding their risk for acute deterioration. However, gradual deterioration was seen significantly more often in PMAVFs compared to SAVMs and SMAVMs. We believe that this is related to PMAVFs presenting more often with venous congestion rather than haemorrhage (Flores *et al.*, 2017; Ho *et al.*, 2018). Compared to the other two subtypes, the angio-architecture of most PMAVFs is relatively simple which relates to a relatively easier therapeutic intervention with higher obliteration rates and lower risk of complication (Lee *et al.*, 2014). We therefore propose that PMAVFs should be completely treated in the early

period after symptom onset. Multivariate analysis showed that male gender and increasing age of onset were independent risk factors for gradual deterioration after initial onset. These phenomena have not been reported before and their mechanism remains unclear; however this finding may have to be factored into management decisions. We found that a large proportion of patients who presented with acute onset or acute deterioration experienced a trigger event with raised thoracic or abdominal pressure. Although we do not have sufficient statistical evidence, we believe it is wise to avoid such events for patients harbouring SCAVSs.

Outcomes of intervention were reported in various previous articles and obviously vary widely between different groups and are different in nidus-type arteriovenous malformations as compared to perimedullary fistulous type arteriovenous malformations. In the Korean cohort, clinical deterioration rate immediately after treatment was 25% and, for SAVMs patients, no recovery was documented at last follow-up (Cho *et al.*, 2013). In the experience of the Toronto group, complication rates of $\sim$ 4% were reported for both surgery and endovascular management (Lee *et al.*, 2014). In the Barrow series, the permanent complication rate of 13.6% was found for SAVMs (Rangel-Castilla *et al.*, 2014). Our experience is consistent with previous reports, as in this series we found that permanent complication rate related to treatment was 11.5% and the total post-treatment annual deterioration rate was 8.4%. Comparing favourably to the natural history, the treatment results support our approach of early invasive treatment for SCAVSs.

Our data indicate that residual lesions may still harbour the risk of post-treatment clinical deterioration. Therefore, complete obliteration is the goal of SCAVSs treatment, which may be difficult in some cases (Lee *et al.*, 2014). The major challenge of the operation is to accurately recognize the angioarchitecture under the microscope and to determine the residual lesion during the procedure. Based on our experience, intraoperative DSA combined with methylene blue angiography is a key technique to achieve complete obliteration with neurological function preservation (Tani *et al.*, 2001; Osanai *et al.*, 2017). The intraoperative DSA can localize the AVM nidus precisely, confirm complete obliteration right after resection and identify residual shunting. Methylene blue angiography can reveal the angioarchitecture of the SCAVSs and identify residual shunting in the operative field of view. In addition, intraoperative neurophysiological monitoring is a reliable tool to preserve spinal function (Li *et al.*, 2018).

There are limitations to our study. We cannot eliminate the possibility that the pattern of referral to our institutes might have affected the results. However, as the only referral centres specialized in spinal vascular diseases within the whole country since 2000, most diagnosed or suspected SCAVSs in China are transferred to our departments. Thus, we believe that our SCAVSs cohort is representative of the general SCAVSs population.

Since most of our patients eventually received surgical or endovascular treatments, patients were inevitably followed for a limited period of time, and 50% of patients in our study were observed within 12 months. Thus, our natural history findings should be interpreted cautiously, especially for patients with a longer history of presentation. Because patients with a long history of mild symptom or transient manifestation may not seek medical counselling unless symptoms deteriorate, one may presume that the risk of deterioration for these patients may be overestimated. However, because of the poor natural history, especially during the early period after onset, and the available modern treatment options, it may not be possible to obtain prospective data of the long-term, untreated clinical course of SCAVSs.

Another limitation of the study is that the endpoints lack objective criteria such as haemorrhage, which is widely used in the natural history study of brain arteriovenous malformations (Mast *et al.*, 1997; Hernesniemi *et al.*, 2008; da Costa *et al.*, 2009). However, the diagnosis of spinal haemorrhage is more difficult than brain haemorrhage. First, compared to the brain, the size of the spinal cord is small, and the signal of flow voids and oedema may obscure small bleeds. Second, spinal subarachnoid hemorrhage with a small volume might be difficult to detect on MRI or CT. In addition, haemorrhage is only one potential cause of SCAVSs to become symptomatic and thus relying only on haemorrhage to determine the natural history is not sufficient for SCAVSs. The mALS was initially designed to assess spinal cord function for patients with spinal vascular diseases and was widely used in clinical practice. It could be obtained by some simple inquiries rather than physical examination, which makes it the best tool to assess the spinal function for observational studies of SCAVSs.

Conclusions

The natural history of symptomatic spinal cord arteriovenous shunts is poor, especially in the early period after onset, and an early intervention is therefore recommended. Our data on stratifying factors which affect the natural history may prove valuable for the decision-making process in individual patients with these complex lesions. We propose emergency open surgery for acute onset patients harbouring non-spontaneous recovery risk factors. For acute onset patients with spontaneous recovery, emergency open surgery is avoided; however, early endovascular embolization may be considered to reduce the risk of subsequent haemorrhage during the early period after the onset. For gradual onset patients, especially for patients harbouring risk factors for subsequent deterioration, early surgical or endovascular managements should be performed according to the angiographic and anatomical characteristics of the lesions. The acute deterioration risks of SAVMs, PMAVFs and SMAVMs are similar, while PMAVFs harbour a

significantly higher risk of gradual deterioration, which necessitates an early invasive treatment.

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Competing interests

The authors declare no competing financial interests.

Supplementary material

Supplementary material is available at *Brain* online.

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