Introduction

In approximately 15% of patients with intracranial subarachnoid haemorrhage (SAH), the cause of the haemorrhage is another than a ruptured intracranial saccular aneurysm. Two-thirds of these patients, thus comprising 10% of all patients with intracranial SAH, have a non-aneurysmal perimesencephalic haemorrhage. The remaining 5% of intracranial SAH...
is caused by a variety of rare conditions such as a (transmural) arterial dissection, a cerebral arteriovenous malformation, a cranial dural arteriovenous fistula, a mycotic/septic aneurysm, pituitary apoplexy, cocaine abuse, sickle cell disease, a coagulation disorder or trauma [1, 2]. In addition, spinal arteriovenous shunts (SAVS), in particular if localized in the cervical region, have been reported to present with symptoms and signs suggesting an intracranial cause of the SAH [1, 2].

Most commonly, SAVS present with gradually worsening sensory disturbances, diffuse back and muscle pain, weakness and sphincter disturbances. Acute onset of symptoms is mostly attributed to spinal haemorrhage either into the subarachnoid space or intramedullary and rarely to venous thrombosis [3, 4]. SAVS can be divided into arteriovenous fistulas (AVF) and arteriovenous malformations (AVM). Spinal AVF commonly presents with signs and symptoms of progressive myelopathy. Spinal AVM may also present with pain, acute myelopathy, or radiculopathy [5]. Spinal SAH is reported in approximately half of symptomatic spinal cord AVM [6, 7] and is frequently accompanied by intracranial signs and symptoms [8].

The aim of this study was to investigate the clinical characteristics and type and location of the SAVS of patients with SAVS who present with symptoms and signs suggesting an intracranial and not a spinal cause of the SAH. We also aimed to assess the proportion of patients with intracranial SAH in whom the cause is a SAVS.

Methods

Review of the literature

To identify patients with intracranial SAH caused by SAVS we performed a literature search using PubMed (up to December 2005) with the following keywords: spinal arteriovenous malformation(s), spinal arteriovenous shunt(s), spinal vascular malformation(s) and subarachnoid haemorrhage. Additional articles were found by searching the reference lists of relevant articles.

We included all articles reporting patients diagnosed with intracranial SAH caused by a SAVS with extracranial arterial supply, without signs and symptoms directly suggestive of spinal pathology. Patients were included when a diagnosis of intracranial SAH was reported. Preferably the diagnosis was confirmed on CT but patients with confirmation of SAH by cerebrospinal fluid investigation only or patients, in whom the details on how the diagnosis of intracranial SAH was reached were lacking, were not excluded. Articles in languages other than English were excluded.

Two authors (J.v.B, D.C.G.S) independently reviewed and extracted the following data on eligible patients: demographic characteristics, method of diagnosis of SAH, medical history, presenting symptoms, findings at neurological examination on admission, neuro-imaging, angio-architectural features of SAVS, classification of SAVS (according to Spetzler et al. [5]), treatment, clinical and radiological follow-up (including obliteration after treatment), and clinical outcome, which we aimed to classify according to the Glasgow Outcome Scale (GOS) [9]. In case of disagreement between these two authors, extracted items were reviewed together to reach consensus. If necessary, a third author (C,J,M,K) resolved the disagreement between the first two authors. The included cases were subdivided into three subgroups: cranio-cervical junction SAVS (CCJ, between the foramen magnum and C2), cervical SAVS (C3-C8), and thoracolumbar SAVS.

Search SAH database

The records of all patients registered in the SAH database of the University Medical Center Utrecht from January 1985 to January 2005 were reviewed to find additional cases. The same inclusion and exclusion criteria as for inclusion of cases reported in the literature were applied. The method of data-extraction was identical to that for the patients reported in the literature.

Data analysis

The data were analyzed using SPSS for Windows, version 12.0.1 (SPSS Inc.). We used the Students T-test to compare mean ages between the different subgroups of patients. We used proportions with corresponding 95% confidence intervals (CI) to assess the estimated prevalence of SAVS among patients registered in our SAH database and to compare clinical and radiological characteristics of the patient subgroups.

Results

The literature search yielded 28 articles. In the SAH database, consisting of 2142 patients with SAH in the studied period, we found one patient (0.05%, 95% CI 0.006–0.3%). Of the 2142 patients, 1740 (81.2%) patients were diagnosed with an aneurysmal SAH, 149 (7.0%) with a perimesencephalic haemorrhage, and 130 (6.1%) with another cause, including the one SAVS. In 123 (5.7%) patients no definite cause was identified. All patients underwent four-vessel angiography if CT angiography demonstrated no abnormalities but we have not systematically searched for SAVS by MRI screening in every patient with a negative angiogram.

A summary of the 36 patients with intracranial SAH caused by SAVS is presented in Table 1. In 14 of the 36 patients (39%), the SAVS was located at the CCJ, in 11 patients (30.5%) at the cervical level, and in another 11 (30.5%) at the thoracolumbar level. In only one of the 11 patients with a thoracolumbar SAVS, the malformation was located at the midthoracic level.

Twenty-two patients (61%) were men. Mean age of patients at presentation was 36 years (range 4–72 years). Patients with a SAVS at the CCJ presented at older age than patients with a SAVS in the cervical or thoracolumbar region (mean difference, 33 years; 95% CI 21–44 years; p-value < 0.0001). The delay between the (first) SAH and the diagnosis of a SAVS...
varied from 0 to 15 years. A delayed diagnosis had occurred mostly in cervical (n = 5; delay of 2–15 years) and thoracolumbar SAVS (n = 4; delay of 2–5 years). Twenty-six patients (72%) had only one episode of SAH, the remaining ten patients had two or more episodes (range 2–12 SAH). In all ten patients with two or more episodes, the diagnosis was made only after a repeated episode. The presenting symptoms, findings on examination, CT findings, and treatment results of all 36 patients are listed in Table 2.
Clinical presentation

Most patients (n = 27, 75%) presented with headache. In two of the remaining nine patients, no clinical details about the first SAH were reported but these two patients did present with headache after subsequent SAH [19, 22]. In the other seven patients, primarily patients with cervical or thoracolumbar SAVS, there was no information on whether or not patients also had headache at the time of presentation. Two of these seven patients presented with impaired consciousness [28, 32]. In one of them, generalized tonic seizures, abnormal breathing pattern, and bilateral papiledema with retinal haemorrhages were reported [28]. In one other patient, who presented with a mild hemiparesis, CT of the brain showed SAH [27, 29]. The reported signs and symptoms that may have added to the diagnosis of intracranial SAH in another two patients for whom there was no information on the presence or absence of headache, were vomiting and dizziness in one patient [14] and nuchal rigidity in another [29]. In two patients no further information was available [19].

A period of unresponsiveness was found in eight patients (22%), three of whom had a thoracolumbar SAVS. Back pain associated with the symptoms and signs suggesting an intracranial cause was found only in patients with a thoracolumbar SAVS; it occurred in four (36%) of the patients with thoracolumbar SAVS. Neck pain was reported in nine patients (25%), four of whom had thoracolumbar SAVS. In eight of these nine patients, the neck pain was associated with headache. In the ninth patient, a 13-year-old boy, acute pain in the neck was the only symptom during the first episode of SAH. At the second episode he also had headache and vomited. At this second episode, a cervical vascular malformation was diagnosed after vertebral angiography [22]. Only one of the 11 patients with a cervical SAVS complained of pain between the shoulder blades after this patient had a

Table 2: Clinical characteristics, diagnostic tests, and treatment in 36 patients presenting with SAH due to SAVS

|                       | All patients n = 36 | CCJ SAVS n = 6 | Cervical SAVS n = 14 | Thoracolumbar SAVS n = 11 |
|-----------------------|---------------------|---------------|---------------------|--------------------------|
|                       | Present | Absent | Unknown | Present | Absent | Unknown | Present | Absent | Unknown |
| **Symptoms**          |         |        |         |         |        |         |         |        |         |
| Headache              | 27a     | 75a    | 13      | 1       | 6      | –       | 5a      | 8      | –       |
| Nausea                | 9       | 25     | 4       | 10      | 1      | –       | 10      | 4      | –       |
| Vomiting              | 11a     | 31a    | 4       | 10      | 3      | –       | 8a      | 4      | 1       |
| Unresponsiveness      | 8       | 22     | 4       | 9       | 1      | 1       | 9       | 3      | 1       |
| Neck pain             | 9       | 25     | 2       | 11      | 3      | –       | 8       | 4      | 1       |
| Back pain             | 4       | 11     | –       | 1       | 13     | –       | 10      | 4      | –       |
| **Neurological examination** |         |        |         |         |        |         |         |        |         |
| Impaired consciousness| 4       | 11     | 2       | 4       | 8      | –       | 1       | 10     | 2       |
| Nuchal rigidity       | 13a     | 36a    | 5       | 9       | 2      | 1       | 8a      | 6      | 1       |
| Focal neurological deficit | 6a     | 17a    | 1       | 8       | 5a     | 3       | 3       | 5a     | 2       |
| Brain CT              |         |        |         |         |        |         |         |        |         |
| Performed             | 25      | 69%    | 11      | 3       | NA     | –       | 4       | 6      | NA      |
| Confirming SAH        | 17      | 47%    | 9       | 2       | 6      | –       | 2       | 6      | –       |
| **Blood distribution (CT)** |         |        |         |         |        |         |         |        |         |
| Basal cisterns        | 7       | 19     | 3       | –       | 4      | –       | 4      | –       |
| Perimesencephalic     | 1       | 3      | 1       | –       | –      | –       | –      | –       |
| Intraventricular      | 10      | 28     | 6       | 4       | –      | –       | 4      | –       |
| Third ventricle       | 3       | 8      | 1       | –       | –      | 2       | –      | 2       |
| Fourth ventricle      | 9       | 25     | 5       | –       | –      | 4       | –      | 4       |
| Cisterna magna        | 2       | 6      | 1       | 1       | –      | –       | –      | –       |
| SAH, not specified    | 6       | 17     | 3       | 1       | 2      | –       | 2      | –       |
| Negative CT           | 8       | 22     | 2       | 2       | 4      | –       | 4      | –       |
| **Treatment SAVS**    |         |        |         |         |        |         |         |        |         |
| Surgery               | 23      | 64     | 12      | 6       | 5      | –       | 5       | –       |
| Embolization          | 4       | 11     | –       | 1       | 4      | 3       | 3      | –       |
| Embolization and surgery | 3      | 8      | –       | 1       | 1      | –       | 2      | –       |
| Radiation             | 1       | 3      | –       | 1       | –      | –       | –      | –       |
| None                  | 5       | 14     | 2       | 2       | 1      | –       | –      | –       |
| Results treatment SAVS n = 31 |         |        |         |         |        |         |         |        |         |
| Partial obliteration  | 4       | 13     | –       | 2       | 2      | –       | 2      | –       |
| Complete obliteration | 17      | 55     | 9       | 3       | 5      | –       | 5      | –       |

*a*CCJ Craniocervical junction, NA, Not available, SAH Subarachnoid haemorrhage, SAVS Spinal arteriovenous malformation, *a*At first presentation, *a*For three patients details of their first SAH were not described but information about subsequent SAH were documented [19, 22]
second SAH [24]. Two patients (6%) had a history of transient spinal cord signs before the SAH; one of these patients had a transient paraplegia 6 years before SAH, and the other had a history of quadriplegia 11 years before the SAH (diagnosed as Pertussis and Poliomyelitis) [26, 37]. One patient mentioned, after further questioning, difficulty with urination in the year before presentation with a SAH [34].

Six patients had neurological deficits at the time of the first episode of SAH. A hemiparesis was present in three patients [14, 24, 27]. One patient presented with a weakness of all extremities, but more pronounced on one side [26]. Two patients presented with a mild weakness in one leg only [34, 37]. The sixth patient had a peripheral facial nerve palsy [12]. Papilledema [20, 28] and retinal haemorrhages [28] were found in three patients (9%). Presence of nuchal rigidity was reported in two patients (18%) with cervical SAVS, in five patients (36%) with CCJ SAVS, and in six patients (55%) with a thoracolumbar SAVS from a total of 15 patients in whom information on the presence or absence of nuchal rigidity was reported.

Seven patients developed neurological deficits after their first SAH but before diagnosis [19, 27, 36]. In four patients, this was after additional episodes of SAH [19, 22]. The signs and symptoms included quadriplegia [19, 27], paraparesis with weakness of one arm [19], hemiparesis [19], paresis of one leg [36], sphincter [22, 27] and sensory [19, 36] disturbances, and decreased unilateral abdominal reflexes [22].

### Diagnostic procedures and neuro-imaging

A brain CT was done in 25 (69%) of the 36 patients, and showed SAH in 17 (47%). Details about the delay between initial symptoms and timing of CT were not mentioned. In 12 patients (34%), the diagnosis of SAH was based upon a positive lumbar puncture. In seven (20%) patients, the diagnostic method was not mentioned. In four of them there was headache or pain in the neck, and in three patients details on the clinical presentation were not given. Six of these seven patients presented with a first episode of SAH in the fifties [22], sixties [19] or early seventies [33] of the last century, when CT scanning was not available. The seventh patient presented with severe headache from SAH, but a four-vessel cerebral angiography showed no abnormalities, and subsequent cervical spinal MRI scan revealed a vascular malformation [21].

The distribution of blood on the brain CT is summarized in Table 2. Intracranial SAH on CT was frequently seen in CCJ SAVS (in nine of 11 patients in whom a CT was performed), but also in the majority (six of ten patients in whom a CT was performed) of patients with thoracolumbar SAVS and in half of patients with a cervical SAVS (in two of four patients in whom a CT was performed).

In 13 of the 14 patients with a CCJ SAVS cerebral angiography revealed the diagnosis, in the remaining patient after spinal angiography. The diagnosis of a cervical SAVS was made after cerebral angiogram in five of the 11 patients, on a diagnostic laminectomy [19, 27] in three, on spinal MRI in two [20, 21], and after myelography in one patient [26]. Three CCJ SAVS [10, 11, 18] and nine cervical SAVS [19, 24, 25] were not diagnosed directly after the first cerebral angiogram because initially not all four cerebrospinal vessels were catheterized selectively. Of the 11 patients with thoracolumbar SAVS, seven patients were diagnosed with MRI, two after spinal angiography, and two with myelography.

### Treatment and clinical outcome

Patients with a SAVS at the CCJ were treated surgically (86%) or received no treatment to obliterate the SAVS (14%). Embolization was performed only in cervical and thoracolumbar SAVS. Three patients were reported to await treatment and two patients [18, 26] did not receive treatment of the SAVS. Five of the 36 patients (14%) were treated because they developed hydrocephalus. Three patients needed temporary drainage by means of an external ventricular drain [10, 28]. A ventriculoperitoneal drain was placed in one patient [14]. Another patient was treated with acetazolamide to reduce intracranial pressure [20].

Information on outcome was available for 33 patients. Twenty-six patients (79%; 95% CI 61–91%) made a good recovery (GOS 5), four patients remained moderately disabled (GOS 4), and two patients were severely disabled (GOS 3). One patient died of shunt-related complications [10].

### Discussion

This study of 36 patients with SAVS presenting with signs and symptoms suggesting an intracranial cause of SAH showed that intracranial SAH from a SAVS can occur at any age and that the SAVS can be located at any level: at the craniocervical junction, the cervical level and at the thoracolumbar level. Most patients with intracranial SAH from a SAVS have a good recovery.

SAVS have been reported as a rare cause of intracranial SAH. However, mainly patients with cervical SAVS have been described as being indistinguishable from patients with an intracranial source
of bleeding [1, 2, 44]. Our search retrieved 11 patients with a thoracolumbar SAVS, emphasizing that SAVS localized at any level of the spinal cord can present with signs and symptoms of intracranial SAH.

We found no SAVS located between the thoracic levels two and eight. Because of the small number of patients included in this study this is most likely due to chance. In general, SAVS frequently occur in the midthoracic region [7, 45]. Dorsal intradural spinal AVF are found mainly in the low thoracic and lumbar regions in contrast to other types of SAVs that are spread more equally along the cord [7, 46, 47]. In this study, two dorsal intradural spinal AVF were located in the thoracolumbar region [28, 30], but in two other patients dorsal intradural spinal AVF were situated in the cervical region [13, 27].

The pathological mechanism of intracranial SAH from a SAVS remains unclarified. The most straightforward mechanism is migration or extension of subarachnoid blood from the spinal to the intracranial level [26, 28, 31]. Haemorrhage may be caused by venous hypertension when arterialized blood flows via the medullary vein to the valveless coronal venous plexus and radial vein [11, 25]. Another hypothesis suggests that the vein around the midbrain is compressed or stretched by the tentorial incisura when, eg., physical exercise elevates the ICP, which then leads to aggravation of venous hypertension with subsequent rupture of the vein [13]. In patients with intracranial drainage of their SAVS, the relatively fast venous flow may cause formation of a varix on the draining vessel, which may result in intracranial SAH after rupture [16]. Ascending venous drainage was associated with an increased risk of SAH in six patients with CCJ perimedullary and dural AVF [15]. In this light, it is not surprising that in patients with SAVS also other cranial symptoms and signs than those suggestive of intracranial SAH caused by rupture of a saccular aneurysm have been reported, such as intermittent double vision, slurred speech, and nystagmus [8].

A limitation of this study is that in only about half of the reported patients with SAVS presenting with intracranial SAH the diagnosis was confirmed on brain CT. However, in all 36 patients the authors were convinced of the diagnosis of intracranial SAH and at first instance searched for a saccular aneurysm.

It remains questionable whether in patients with SAH without an intracranial source of the haemorrhage SAVS should be searched for, since intracranial SAH caused by SAVS is very rare. Four-vessel angiography can detect a CCJ or cervical SAVS and should always be performed before investigating the spinal cord. MRI screening of the spine in all patients with (repeated) negative angiograms will probably have a low yield and be very expensive. In a cohort of 15 patients with an aneurysmal pattern of SAH and three negative four-vessel angiograms, but no imaging of the spine, no episodes of new episodes of SAH occurred during an average follow-up of 65 months [48]. We suggest that in patients with an intracranial SAH without identifiable cause, close attention should be given to sometimes subtle clues in the history and examination (such as back pain, pain between shoulder blades, and difficulty with urination) that may point to a spinal cause of the SAH. A delayed diagnosis can have negative implications for patients, because they may develop new or progressive neurological deficits that may or may not be related to new episodes of SAH. In patients with multiple episodes of SAH and repeated negative cerebral four-vessel angiograms, further investigations (e.g., MRI) should probably be considered to exclude a SAVS as a rare but possible cause, even in the absence of symptoms or signs of medullary or spinal root involvement. In those rare cases the full length of the spinal cord should be investigated.

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Reference

1. Rinkel GJ, van Gijn J, Wijdicks EF (1993) Subarachnoid hemorrhage without detectable aneurysm. A review of the causes. Stroke 24:1403–1409
2. van Gijn J, Rinkel GJ (2001) Subarachnoid haemorrhage: diagnosis, causes and management. Brain 124:249–278
3. Ferch RD, Morgan MK, Sears WR (2001) Spinal arteriovenous malformations: a review with case illustrations. J Clin Neurosci 8:299–304
4. Kringts M, Mull M, Gilbsch JM, Thron A (2005) Spinal vascular malformations. Eur Radiol 15:267–278
5. Spetzler RF, Detwiler PW, Rini HA, Porter RW (2002) Modified classification of spinal cord vascular lesions. J Neurosurg Spine 96:145–156
6. Niimi Y, Berenstein A, Setton A, Pryor J (2000) Symptoms, vascular anatomy and endovascular treatment of spinal cord arteriovenous malformations. Intervent Neuroradiol 6:199–202
7. Rosenblum B, Oldfield EH, Doppman JL, Di Chiro G (1987) Spinal arteriovenous malformations: a comparison of dural arteriovenous fistulas and intradural AVM’s in 81 patients. J Neurosurg 67:795–802
8. Caroscio JT, Brannan T, Budabin M, Huang YP, Yahr MD (1980) Subarachnoid hemorrhage secondary to spinal arteriovenous malformation and aneurysm. Report of a case and review of the literature. Arch Neurol 37:101–103
9. Jennett B, Bond M (1975) Assessment of outcome after severe brain damage. Lancet 1:480–484
10. Aviv RI, Shad A, Tomlinson G, Niemann M, Deedwania P, Molyneux AJ, Byrne JV (2004) Cervical dural arteriovenous fistulae manifesting as subarachnoid hemorrhage: report of two cases and literature review. AJNR Am J Neuroradiol 25:854–858
11. Do HM, Jensen ME, Cloft HJ, Kallmes DF, Dion JE (1999) Dural arteriovenous fistula of the cervical spine with subarachnoid hemorrhage. AJNR Am J Neuroradiol 20:348–350
12. Endo T, Sato K, Takahashi T, Kato M (2001) Acute hypotension and bradycardia by medulla oblongata compression in spinal surgery. J Neurosurg Anesthesiol 13:310–313
13. Hashimoto H, Iida J, Shin Y, Hironaka Y, Sakaki T (2000) Spinal dural arteriovenous fistula with perimesencephalic subarachnoid haemorrhage. J Clin Neurosci 7:64–66
14. Hosoda K, Fujita S, Kawaguchi T, Yamada H (1994) A transcglyceryl approach to the arteriovenous malformation at the ventral cervicomedullary junction: report of three cases. Neurosurgery 34:748–752
15. Kai Y, Hamada J, Morikoa M, Yano S, Mizuno T, Kuratsu J (2005) Arteriovenous fistulas at the cervicomedullary junction presenting with subarachnoid hemorrhage: six case reports with special reference to the angiographic pattern of venous drainage. AJNR Am J Neuroradiol 26:1949–1954
16. Kinouchi H, Mizoi K, Takahashi T, Oshikuma S, Fujimoto S, Seki T, Miyasaka K (2002) Iliac artery: a direct approach to perimedullary arteriovenous fistulas of the anterior cervical spinal cord. J Neurosurg 96:157–161
17. Hiioka O, Lidvall H (1958) Arteriovenous aneurysms of the spinal cord: a report of two cases investigated by vertebral angiography. J Neurosurg 15:84–91
18. Morimoto T, Yoshida S, Basugi N (1992) Dural arteriovenous malformation in the cervical spine presenting with subarachnoid hemorrhage: case report. Neurosurgery 31:118–120
19. Odom GL (1962) Vascular lesions of the spinal cord: malformations, spinal subarachnoid and extradural hemorrhage. Clin Neurosurg 8:196–236
20. Willinsky R, Terbrugge K, Lasjaunias P, Montanera W (1990) The variable presentations of craniocervical and cervical dural arteriovenous malformations. Surg Neuro 34:118–123
21. Clark RS, Orr RA, Atkinson CS, Towbin RB, Pang D (1995) Retinal hemorrhages associated with spinal cord arteriovenous malformation. Clin Pediatr (Phila) 34:281–283
22. Goren P, Stroobant M (1983) Spinal cord arteriovenous malformations with significant intramedullary components. J Neurosurg 59:471–478
23. Koch C, Gottschalk S, Giese A (2004) Dural arteriovenous fistula of the lumbar spine presenting with subarachnoid hemorrhage. Case report and review of the literature. J Neurosurg 100:385–391
24. Maggioni F, Rossi P, Casson S, Fiore D, Zanchin G (1995) Initially migraine-like manifestation of a ruptured spinal arteriovenous malformation. Cephalalgia 15:237–240
25. Mandzia JL, terBrugge KG, Faughnan ME, Hyland RH (1999) Spinal cord arteriovenous malformations in two patients with hereditary hemorrhagic telangiectasia. Childs Nerv Syst 15:80–83
26. Parkinson D, West M (1977) Spontaneous subarachnoid hemorrhage first from an intracranial and then from a spinal arteriovenous malformation. Case report. J Neurosurg 47:965–968
27. Rosenow J, Rawanduzy A, Weitzner I Jr., Coulshed WT (2000) Type IV spinal arteriovenous malformation in association with familial pulmonary vascular malformations: case report. Neurosurgery 46:1240–1244
28. van Santbrink H, de Witt Hamer PC (2003) Spinal AV malformation. Lancet 361:1766
29. Wataki S, Inoh S, Iwanaga H, Nagai M, Sato T, Izumi J (1992) Successful surgical obliteration of a huge intradural arteriovenous fistula of the spinal cord in a child. Childs Nerv Syst 8:347–350
30. Williams FC, Zabramski JM, Spetzler RF, Rekate HL (1991) Anterolateral transthoracic transvertebral resection of an intramedullary spinal arteriovenous malformation. Case report. J Neurosurg 74:1004–1008
31. Meisel HJ, Lasjaunias P, Brock M (1996) Multiple arteriovenous malformations of the spinal cord in an adolescent: case report. Neuroradiology 38:490–493
32. Vates GE, Quinones-Hinojosa A, Halbach VV, Lawton MT (2001) Conus perimedullary arteriovenous fistula with intracranial drainage: case report. Neurosurgery 49:457–461
33. Defreyne L, Achten E, Vandenckhove T, Kunnen M (1999) Rupture of a cervical spinal cord arteriovenous malformation: a rare complication of endovascular embolisation. Eur Radiol 9:734–737
34. Hash CJ, Grossman CB, Shenkman HA (1975) Concurrent intracranial and spinal cord arteriovenous malformations. Case report. J Neurosurg 43:104–107
35. Sugiu K, Meguro T, Nakashima H, Ohmoto T (2001) Successful embolization of a spinal perimedullary arteriovenous fistula with cellulose acetate polymer solution: technical case report. Neurosurgery 49:1257–1260
36. Hoffman HJ, Mohr G, Kusunoki T (1976) Multiple arteriovenous malformations of spinal cord and brain in a child. Case report. Childs Brain 3:317–324
37. Warlow CP, Dennis MS, van Gijn J, Hankey GJ, Sandercock PAG, Bamford JM, Wardlaw JM (2001) What caused this subarachnoid haemorrhage? Stroke: a practical guide to management. Blackwell Science Ltd, Oxford
38. Jellema K, Canta LR, Tijssen CC, van Rooij WJ, Koudstaal PJ, van Gijn J (2003) Spinal dural arteriovenous fistulas: clinical features in 80 patients. J Neurol Neurosurg Psychiatry 74:1438–1440
46. Symon L, Kuyama H, Kendall B (1984) Dural arteriovenous malformations of the spine. Clinical features and surgical results in 55 cases. J Neurosurg 60:238–247

47. Yaşargil MG, Symon L, Teddy PJ (1984) Arteriovenous malformations of the spinal cord. Adv Tech Stand Neurosurg 11:61–102

48. Ruigrok YM, Rinkel GJ, van Gijn J (2002) CT patterns and long-term outcome in patients with an aneurysmal type of subarachnoid hemorrhage and repeatedly negative angiograms. Cerebrovasc Dis 14:221–227