Acta Neurochir (2010) 152:1981–1984
DOI 10.1007/s00701-010-0758-7

CASE REPORT

Spontaneous acute spinal subdural hematoma: spontaneous recovery from severe paraparesis—case report and review

Michael Payer · Reto Agosti

Received: 4 June 2010 / Accepted: 23 July 2010 / Published online: 11 August 2010

Abstract Spontaneous idiopathic acute spinal subdural hematomas are highly exceptional. Neurological symptoms are usually severe, and rapid diagnosis with MRI is mandatory. Surgical evacuation has frequently been used therapeutically; however, spontaneous recovery in mild cases has also been reported. We present a case of spontaneous recovery from severe paraparesis after spontaneous acute SSDH, and review the English-speaking literature.

Keywords Spinal · Subdural hematoma · Spontaneous · Paraparesis

Introduction

Spontaneous idiopathic acute spinal subdural hematomas (SSDHs) are highly exceptional. As neurological symptoms are usually severe, rapid diagnosis with MRI is mandatory [2, 9]. In therapeutic management, the main question is whether surgical evacuation is necessary or not. We present a case of spontaneous subtotal recovery from severe paraparesis after spontaneous acute SSDH, and review the English speaking literature.

M. Payer (✉)
Neurosurgery Klinik Hirslanden,
Witellikerstrasse 40,
8032 Zürich, Switzerland
e-mail: mpayer@hotmail.com

M. Payer
Department of Neurosurgery, University Hospital of Geneva,
Geneva, Switzerland

R. Agosti
Kopfwehzentrum Hirslanden,
Zürich, Switzerland

Case report

A 59-year-old man, under acetylic salicylic acid and clopidrogrel after cardiac stenting, presented with spontaneous acute left-dominant paraparesis sub T8 with an average muscle strength of M3 and severe sphincter dysfunction. Gadolinium-enhanced MRI revealed an acute SSDH from T2-9 anterior to the spinal cord (Fig. 1). No underlying tumor or vascular malformation was seen. Because of the extensive cranio-caudal hematoma extension and poor medical condition, particularly in the fear of causing postoperative recurrent subdural or epidural hematoma because of acetylic salicylic acid and clopidogrel, conservative treatment was chosen under constant readiness for surgery in case of progressive worsening. Clopidogrel was discontinued in accordance with the treating cardiologist, while aspirine was continued. Under initial bed rest and steroids, leg strength fluctuated between grades M 1–3 for 2 days and improved thereafter. Full muscle strength was regained at 1 year; ataxia, temperature/pain sensation, and urge incontinence had well improved. MRI at 5 weeks showed hematoma resorption (Fig. 2), and at 16 weeks, cystic arachnoiditis was observed with spinal cord deformity and without further re-bleedings (Fig. 3).

Discussion

SSDHs are uncommon and often result from major or minor spine trauma or from spine puncture including spinal anesthesia. “Spontaneous” acute SSDHs are even more rare

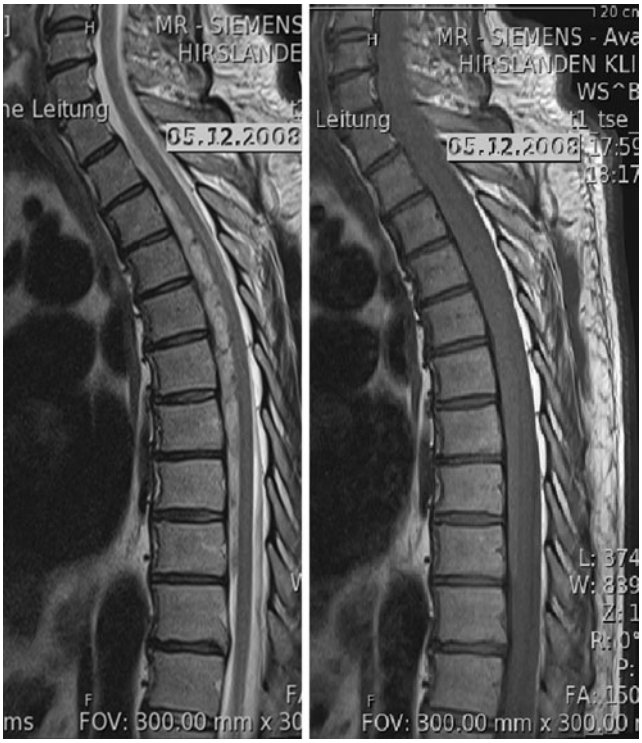

Fig. 1 Sagittal T2- and T1-weighted MRI showing acute SSDH primarily anterior to the spinal cord from T2–9

and have mostly been observed in conjunction with coagulopathy or anticoagulant therapy, intraspinal tumor, and vascular anomaly such as an aneurysm or dural arteriovenous fistula [3, 9, 12]. Spontaneous SSDHs without detectable structural lesion or anticoagulation are less numerous again, and we detected 16 properly documented cases in the readily available English-speaking literature (Table 1).

SSDHs are predominantly found in the thoracic spine, usually extending over several vertebral levels [2, 3, 6, 9]. Severe acute local back pain with sensorimotor deficits and sphincter disturbance are the typical clinical manifestation [3, 9, 12]. MRI is the imaging of choice; it shows the hematoma location in relation to the dura and spinal cord, its cranio-caudal extension, and usually shows or rules out an underlying tumor or AVM [9, 11, 12]. Acute SSDHs (1–3 days) are isointense on T1 and mixed on T2 images; in the subacute stage (4–7 days), they show high intensity on both T1 and T2, and diffuse high signal is seen in the chronic stage after weeks to months [9, 11]. While epidural hematomas are usually found posterior to the spinal cord in a lentiform shape, SSDHs are commonly found anterior-lateral to and around the spinal cord in a semi-circular pattern with compression and little displacement [3, 9, 11].

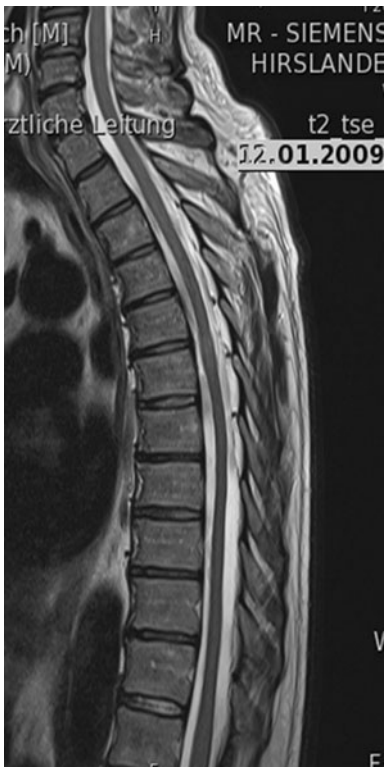

Fig. 2 Sagittal T2-weighted MRI at 5 weeks after the acute bleeding, showing resorption of the thoracic SSDH

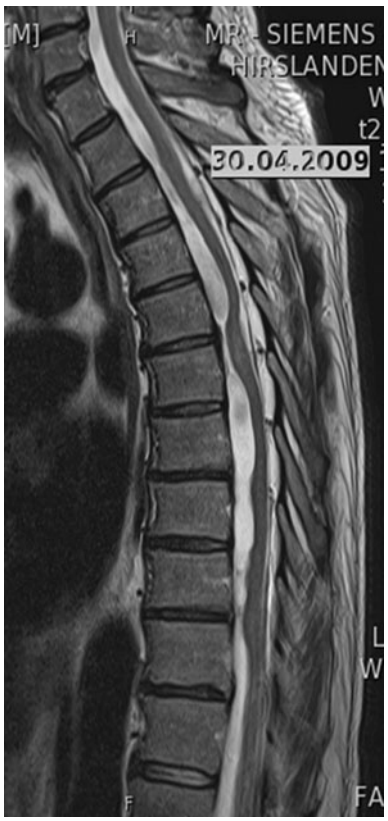

Fig. 3 Sagittal T2-weighted MRI at 16 weeks after the acute bleeding, showing cystic arachnoiditis with spinal cord deformity and without further re-bleedings

Table 1 Published cases of idiopathic spontaneous acute SSDH

Article	Year	Age, sex	Hematoma location	Bleeding cause, risk factors	Preop neuro deficit	Angio	Treatment (spinal)	Recovery
Swann [16]	1984	46, f (case 1)	TL junction	Unknown	Transient mild paraparesis	Yes	Lumbar puncture	Complete recovery
Kalina [4]	1995	60, f	T7-S2, anterior	Unknown, polycythemia vera	Mild paraparesis	No	Conservative	Complete recovery
Kang [5]	2000	49, f	T5-L3, anterior	Unknown	Transient mild paraparesis	No	Conservative	Complete recovery
Küker [11]	2000	81, m (case 4)	Mid T spine	Unknown	Paraparesis (M3/5)	No	Surgery	Complete recovery
		56, f (case 6)	Thoraco-lumbar, anterior	Unknown	Paraparesis (M1–3/5)	Yes	Surgery	Good recovery
Kirsch [9]	2000	47, m (case 1)	T4-L5, antero-lateral	Unknown	Paraparesis	Yes	Lami T11-L1	Improved
		42, m (case 2)	CCJ–L3, around SC	Unknown	Paraplegia	No	Lami T2–5	No recovery
		34, m (case 3)	T1–4, around SC	Unknown	Only pain and paresthesia	Yes	Conservative	Complete recovery
Yamada [17]	2003	38, f	T1–7, anterior	Unknown	Mild paraparesis	Yes	Conservative	Complete recovery
Konitsiotis [10]	2003	60, f	T3-L5, anterior-lateral	Unknown, essential thrombocythaemia	Only pain	No	Conservative	Pain subsided
Cha [1]	2005	72, f	T3–6, posterior-lateral	Unknown; aspirin+low molecular heparin	Paraplegia	Yes	Lami T3–5	No relevant recovery
Kyriakides [12]	2007	44,m	T2–6, anterior	Unknown	Paraplegia	No	Lami	Subtotal recovery
Kim SD [6]	2008	48, f	T1–4, mainly anterior	Unknown, FMD	Paraplegia	No	Lami T1–4	No recovery
Ozdemir [15]	2008	50, m	T4–8, anterior	Unknown	Paraparesis (M3–4)	No	Lami T4–6	Complete recovery
Kakitsubata [3]	2009	66, m	T11/12, anterior-lateral	Unknown	Only pain	No	Conservative	Pain subsided
Oh [14]	2009	59, f	C3–6, posterior-lateral left side	Unknown	Left-sided hemiparesis	No	Conservative	Complete recovery

Lami laminectomy, *SC* spinal cord, *FMD* fibromuscular dysplasia

Angiography has been used inconsistently to rule out AVF, AVM, or aneurysm, and was performed in six of the 16 retrieved cases (Table 1). While some authors recommend spinal angiography in the presence of spontaneous SSDH whenever available [3, 12, 15], others judge that gadolinium injected MRI, particularly if repeated, should show vascular malformations directly or by flow void signals [7, 17]. Many papers do not discuss the indication for spinal angiography [1, 6, 9–11, 14], and it remains a case to case decision based on availability, degree of neurosurgical emergency, and suspicion level of vascular malformation.

The pathomechanism in spontaneous idiopathic SSDHs is little understood [2, 11]. It is commonly thought that either the hematoma originates from rupture of valveless radiculomedullary veins in the subarachnoid space after increased intra-abdominal or intra-thoracic pressure (coughing or straining) or from minor trauma and that it subsequently breaks through the thin arachnoid into the subdural space [9, 12, 15]. This hypothesis could explain clinical signs of SAH in many patients with SSDH, the reported combination of SSDH and SAH, and the potential dilution of such hematomas by the CSF [9, 12]. Conversely, SSDH has been thought to arise from the few thin, delicate extra-archnoidal vessels located on the inner dural surface and then breaking through the arachnoid into the subarachnoid space, and it is usually impossible to determine the origin [3, 7, 13]. In either case, the diluting effect of the CSF prevents clot formation, unless the hematoma is sufficiently large to block CSF flow [8, 12].

In idiopathic SSDHs, therapy is limited to the hematoma management, as there is no underlying pathology to address surgically. Platelet dysfunction has been shown to be associated with SSDH not only in our case but also in a cases with polycythemia vera [4] and essential thrombocythemia [10]. Discontinuation of anti-aggregating medication, however, must be weighed against potential thrombotic complications, and depends on the individual indication of such a treatment. Patients with mild symptoms

or in poor general conditions should be treated conservatively [3, 9, 15]. There is an ongoing controversy, however, whether surgical or conservative treatment should be preferred in severe neurological deficit. While most authors advocate rapid surgical evacuation via laminectomy, posterior dural opening, and hematoma removal in such cases [6, 9, 11, 12, 15], several spontaneous recoveries have been reported in cases with moderate neurological deficits [4, 14, 17], and surgical evacuation in a large review of 106 cases with all types of SSDHs has yielded a poor outcome in 58% [2]. Kyriakides in his review concluded that "patients with incomplete neurologic deficit tend to recover with or without surgery. Patients who exhibit signs of deterioration are usually likely to benefit from percutaneous drainage or surgery" [12]. Our current case shows that substantial neurological recovery can occur even in severe paraparesis; however, we do not conclude that conservative treatment should generally be chosen in severe neurological deficit with "difficult" medical conditions. Initial conservative treatment and close observation can be a treatment option as has been shown previously for "mild" paraparesis and hemi-paresis [4, 14, 17]. Indeed, the current authors were initially not sure whether they should go for surgery in their case, but were very reluctant to do so primarily because of acetylic salicylic acid and clopidogrel.

We agree to the hypothesis that spontaneous recovery from SSDH may be explained by a progressive craniocaudal distribution and pressure drop of the hematoma within the subdural space on one hand and dilution of the hematoma by CSF in the presence of arachnoidal tearing on the other hand [8, 12, 17]. The current case further confirms that sphincter disturbance is the least likely to be improved, as has been found before [11]. Paraplegia carries a poor prognosis even with surgery [6, 12].

In conclusion, there is a general agreement that in mild symptoms, conservative treatment is preferred in spontaneous idiopathic acute SSDH. A clear trend for surgical evacuation via laminectomy and posterior dural opening is found in the literature in cases of a severe neurological deficit; however, the current case demonstrates that good neurological recovery can occur even in severe paraparesis. Patients with paraplegia and sphincter disturbance have the poorest prognosis regardless of surgical or conservative treatment.

Conflicts of interest None

References

1. Cha YH, Chi JH, Barbaro NM (2005) Spontaneous spinal subdural hematoma associated with low-molecular-weight heparin. Case report. J Neurosurg Spine 2(5):612–613
2. Domenicucci M, Ramieri A, Ciappetta P (1999) Nontraumatic acute spinal subdural hematoma: report of five cases and review of the literature. J Neurosurg 91(1 Suppl):65–73
3. Kakitsubata Y, Theodorou SJ, Theodorou DJ, Miyata Y, Ito Y, Yuki Y, Honbu K, Maehara T (2010) Spontaneous spinal subarachnoid hemorrhage associated with subdural hematoma at different spinal levels. Emerg Radiol 17(1):69–72
4. Kalina P, Drehobl KE, Black K, Woldenberg R, Sapan M (1995) Spinal cord compression by spontaneous spinal subdural haematoma in polycythemia vera. Postgrad Med J 71(836):378–379
5. Kang HS, Chung CK, Kim HJ (2000) Spontaneous spinal subdural hematoma with spontaneous resolution. Spinal Cord 38 (3):192–196
6. Kim SD, Park JO, Kim SH, Lee YH, Lim DJ, Park JY (2008) Spontaneous thoracic spinal subdural hematoma associated with fibromuscular dysplasia. J Neurosurg Spine 8(5):478–481
7. Kim JS, Lee SH (2009) Spontaneous spinal subarachnoid hemorrhage with spontaneous resolution. J Korean Neurosurg Soc 45(4):253–255
8. Kim YH, Cho KT, Chung CK, Kim HJ (2004) Idiopathic spontaneous spinal subarachnoid hemorrhage. Spinal Cord 42 (9):545–547
9. Kirsch EC, Khangure MS, Holthouse D, McAuliffe W (2000) Acute spontaneous spinal subdural haematoma: MRI features. Neuroradiology 42(8):586–590
10. Konitsiotis S, Glantzouni A, Argyropoulou MI, Tsapoga T, Elisaf M, Efremidis SC (2003) Acute spontaneous spinal subdural haematomas in a patient with essential thrombocythaemia. J Neurol 250(9):1109–1111
11. Küker W, Thiex R, Friese S, Freudenstein D, Reinges MH, Ernemann U, Kringes T, Skalej M (2000) Spinal subdural and epidural haematomas: diagnostic and therapeutic aspects in acute and subacute cases. Acta Neurochir (Wien) 142(7):777–785
12. Kyriakides AE, Lalam RK, El Masry WS (2007) Acute spontaneous spinal subdural hematoma presenting as paraplegia: a rare case. Spine 32(21):619–622
13. Morandi X, Carsin-Nicol B, Brassier G, Scarabin JM (1998) MR demonstration of spontaneous acute spinal subdural hematoma. J Neuroradiol 25(1):46–48
14. Oh SH, Han IB, Koo YH, Kim OJ (2009) Acute spinal subdural hematoma presenting with spontaneously resolving hemiplegia. J Korean Neurosurg Soc 45(6):390–393
15. Ozdemir O, Calisaneller T, Yildirim E, Caner H, Altinors N (2008) Acute spontaneous spinal subdural hematoma in a patient with bilateral incarcerated inguinal hernia. Joint Bone Spine 75 (3):345–347
16. Swann KW, Ropper AH, New PF, Poletti CE (1984) Spontaneous spinal subarachnoid hemorrhage and subdural hematoma. Report of two cases. J Neurosurg 61(5):975–980
17. Yamada K, Nakahara T, Yamamato K, Muranaka T, Ushio Y (2003) Nontraumatic spinal subdural haematoma occurring in a postpartum period. Acta Neurochir (Wien) 145(2):151–155