Subdural Hematoma

Past to Present to Future Management

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Foreword

This timely book comprehensively covers one of the most frequent afflictions and yet sorely neglected issues in Neurosurgery. In particular the chronic variant of the subdural hematoma (SDH), or as Virchow coined it the "pachymeningitis hemorrhagica interna" (1857), has increased in proportion to our longevity. Additional amplifying factors as well as the diversity of clinical presentations are systematically covered in this volume.

While books about arteriovenous malformations and aneurysms are numerous, despite being relatively rare entities, a thorough analysis in one volume on SDHs is rare, despite the prevalence in real life. Also, SDHs most often represent the first surgical experience of many neurosurgeons, past, present, and future.

And most often this is good, since frequently a small operation can yield an almost instantaneous recovery. However, all of us have also seen our patients on the slippery slope of recurrent hematomas that defy your every best therapeutic effort with uncontrollable deterioration and dismal outcomes. Chronic subdural hematoma (CSDH) can provide a humbling experience.

And while few would call themselves a "CSDH" surgeon, aren't we all? And if we are, our obligation is to further our knowledge and art. Improving the lot of a patient with SDH can be easy and gratifying with a "miracle" recovery. Not being able to help a patient despite our best efforts may be fate, but it is our duty to try everything to improve the odds and it is due diligence to treat the issue with scientific rigor and sincerity.

Turgut, Akhaddar, Turgut, and Hall provide this foundation. Their book will become a standard tool for our younger colleagues, but also a reminder for all to improve our art where it matters most.

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Preface

Subdural hematomas (SDHs) are an interesting disease that is still widely discussed regarding its management despite it being the most common pathology in surgical practice and one of the first surgical procedures taught in residency training in neurosurgery. The incidence of this clinical entity is gradually increasing worldwide due to the extended life expectancy for adults and the common usage of antithrombotic therapy for various medical conditions. SDHs are primarily manifest in three forms, acute, subacute, and chronic, which are influenced by their etiology and the individual patient developing the malady. The pathophysiology of the development of SDHs can be described with respect to their location in the cranial vault or the spinal canal. The various histopathological changes seen in SDHs differ based on the age of the collection since its inception. There are specific predisposing medical risk factors that can lead to the occurrence of SDHs.

The clinical presentation of the SDHs is related to its intracranial location and can be different when the specific age group affected is considered. In intracranial SDHs, the anticipated modes of presentation, such as seizures, can result as can other unusual forms of presentation, such as psychiatric disorders, while spinal cord compression syndrome can occur at the spinal level of origination. The presence of neurological findings should necessitate some form on neuroimaging study in the form of computed tomography or magnetic resonance imaging to determine the underlying source of the hemorrhage. A careful clinical history can also yield a potential etiology for the SDH if a lumbar puncture or cerebrospinal fluid diversion surgery is performed, electroconvulsive therapy is administered, and an underlying hematological disorder or spontaneous intracranial hypotension is present. In some cases, specific intracranial abnormalities have been identified in association with SDHs such as arteriovenous malformations and arachnoid cysts. Not unexpectedly, head trauma and sports-related injuries can result in the development of SDHs, even with minor trauma in the elderly.

The management of SDHs is primarily surgical in nature with different anesthetic techniques being administered in order to successfully treat the hemorrhagic collection. New invasive procedures that include endoscopic drainage and middle meningeal artery embolization have been effectively employed to SDHs. Medical

management in the form of corticosteroid therapy and the administration of tranexamic acid have been utilized to manage SDHs. Despite improved treatment for SDHs, postoperative complications can occur and lead to disease recurrence. Ultimately, some form of postoperative rehabilitation therapy is necessary for the elderly population that sustains a SDH, and the prognosis and clinical outcome are often influenced by a successful course of treatment. From a medicolegal point of view, it should always be kept in mind that various acts of malpractice, such as shaken baby syndrome, in addition to diagnostic and treatment errors, may complicate the management of patients with SDHs.

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Chapter 1
Review of Craniospinal Acute, Subacute, and Chronic Subdural Hematomas

Ali Akhaddar

1.1 Introduction

A subdural hematoma (SDH) appears due to an accumulation of blood into the space between the dura mater and the arachnoid layer. The dura represents the pachymeninx that protects the brain within the cranial cavity and both the spinal cord and nerve roots inside the spinal canal. Most SDHs result from a rupture of bridging veins in the subdural space under certain circumstances, the most important of which is trauma in addition to some medical comorbidities and risk factors such as antiplatelet or anticoagulant agents.

SDHs represent a common and well-known entity in neurosurgical practice with various localizations, heterogeneous clinical manifestations, different neuroimaging features, as well as diverse vital and functional considerations. However, in order to simplify SDH classification, three main categories of patients must be distinguished depending on the topography and age of the hematoma:

– "Primo" Cranial acute/subacute SDH
– "Secundo" Cranial chronic SDH
– "Tertio" Spinal SDH

However, different associations are possible as discussed throughout this book.

The distinction between acute and subacute SDH on one hand and subacute and chronic SDH on the other hand is not always evident; it depends on time and neuroimaging features.

Both young and aging populations are affected by cranial SDH. Younger patients typically experience acute traumatic SDH secondary to high-energy mechanisms of injury, while older patients are more likely to present chronic SDH generally

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resulting from a minor head injury with or without predisposing conditions (brain atrophy, alcoholism, coagulopathies…). Cranial acute SDHs are frequently life-threatening while chronic ones have a better prognosis if correctly managed.

Spinal localization is even more uncommon than intracranial forms of SDH with less than 260 reported cases in the literature to date [61].

A summary of cranial and spinal SDHs is given in this chapter, taking into consideration the age of hematoma and the potential association of its various forms.

1.2 Cranial Acute and Subacute Subdural Hematoma

Acute and subacute SDHs are usually associated with severe head injuries and may result from avulsed bridging veins, lacerated brain, or ruptured cortical vessels. Also, up to one third of head-injured patients with acute SDH may have a significant polytrauma [24, 40]. These hematomas can also be associated with other intracranial lesions such as contusions and intracerebral hematomas, brain swelling, diffuse axonal injuries, or epidural hematomas. Some acute/subacute SDHs may occur in patients without a history of trauma but who have bleeding disorders (receiving anticoagulant therapies or having coagulation diseases) or other unusual underlying etiologies as neoplasms or vascular malformations [13, 16, 27, 32, 41, 58]. In addition, a few cases have been described following cranial or spinal surgery [10, 12, 52, 62, 64].

Symptoms may be secondary to compression of underlying brain, injured brain parenchyma, cerebral edema, and midline shift. Frontal and temporal lobes are the most common localizations. However, some acute/subacute SDHs may be interhemispheric, along the tentorium, or in the posterior fossa [19]. A wide range of clinical presentations may occur such as altered consciousness, seizure, and neurological deficits, as well as other cardio-respiratory and systemic disorders.

In emergency situation, non-contrast computed tomography scan (CT scan) is considered the gold initial procedure in cranial injured patients because it is more accessible, faster, and less expensive. The classic intracranial SDH is an extra-axial crescentic mass with a concave inner margin that follows the surface of the brain usually with associated edema, mass effect, and midline shift. Severe presentations are accompanied by cerebral contusions, intraparenchymal hematomas, uncal herniation, effacement of basal cisterns, and dilatation of the contralateral temporal horn. Distinction between acute and subacute bleeding is occasionally confused. However, most acute forms (less than 3 days old) are hyperdense and most subacute hematomas (between 3 days and 2 weeks old) are isodense or with mixed density as compared to brain parenchyma (Figs. 1.1 and 1.2). In the same way, some forms of SDH cannot be distinguished from epidural subdural hematoma on CT scan and can even sometimes coexist [4]. When a vascular lesion is suspected, a prompt angiographic CT scan or angio-magnetic resonance (MR) (less commonly a brain angiography) can be useful to localize and diagnose the etiology [13, 18, 58]. Other

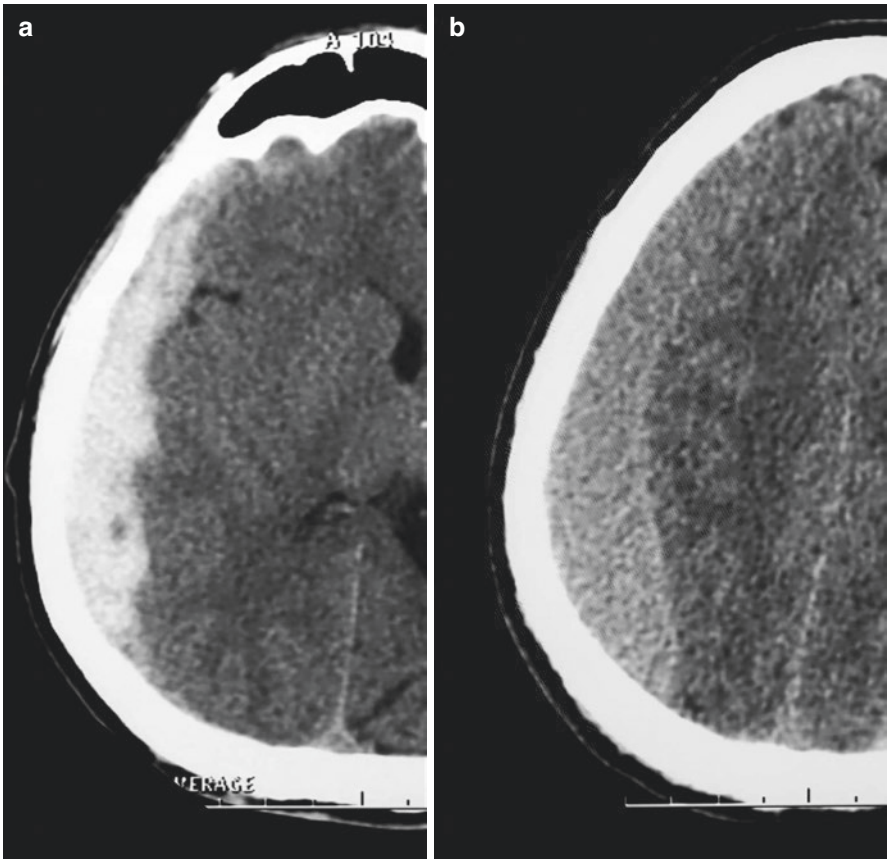

Fig. 1.1 Computed tomography appearance of acute (**a**) and subacute (**b**) subdural hematomas

testings can be useful as laboratory investigations in order to search for an occult coagulopathy or hematologic disease.

The majority of patients with acute SDH need neurocritical care [19, 36]. When surgical procedures are discussed, the neurosurgeon should always consider the underlying etiology of the bleeding, patient's general conditions and any coagulopathy that could be associated. The treatment of acute/subacute SDHs depends on the size and rate of growth of the hematoma, as well as the underlying brain damage. Some small subdural hematomas (thickness less than 10 mm in adult and less than 5 mm in children) can be managed conservatively as the blood collection may resolve spontaneously [28, 76]. Others, especially subacute and liquefied forms, can be treated by bur hole(s) and drainage. However, because of the consistency of blood clots (coagulum), large or symptomatic acute SDHs require a craniotomy for hematoma evacuation and a control of the bleeding (Fig. 1.3) [19]. Sometimes, a supplement decompressive craniectomy may be necessary [40, 60]. Postoperative complications can include high intracranial pressure, brain swelling,

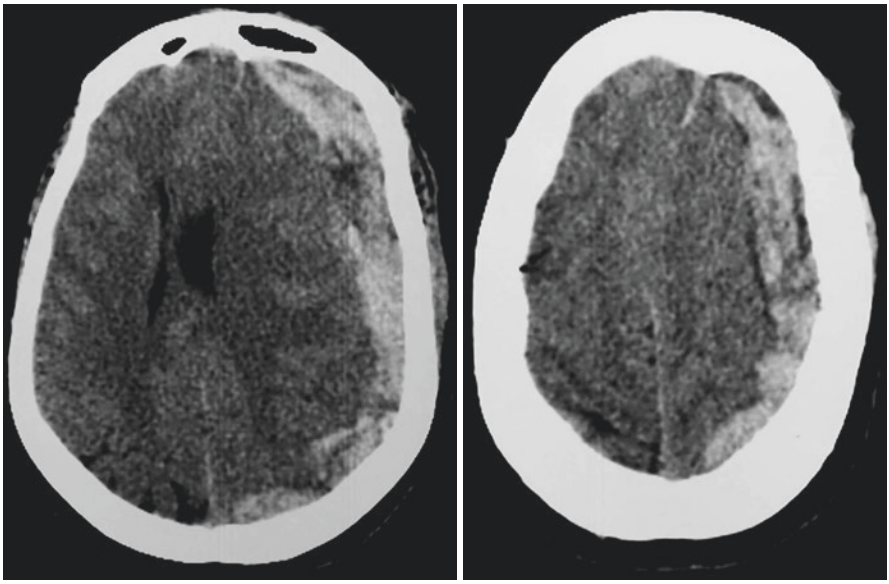

Fig. 1.2 Head axial CT scan: spontaneous (non-traumatic) left acute subdural hematoma in a patient receiving anticoagulant agent

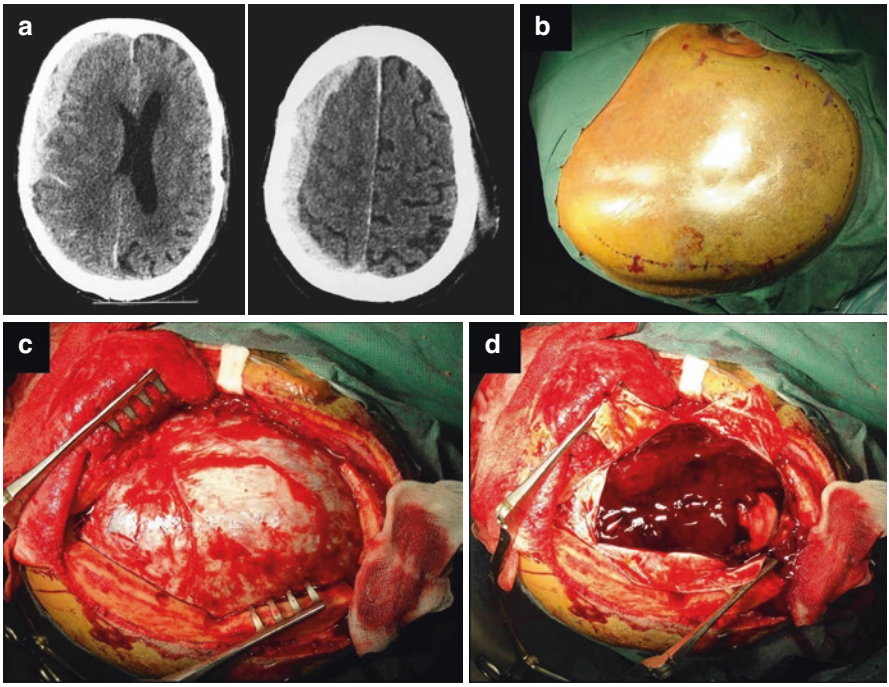

Fig. 1.3 Axial CT scan: post-traumatic acute subdural hematoma on the right side (**a**). Head position with incision mark (question-mark-shaped) on the scalp (**b**). Operative view after removing the fronto-parieto-temporal bone flap (**c**). After opening the dura in a cruciform fashion, the acute hematoma (blood clots) was revealed on the subdural space (**d**)

various forms of new intracranial bleeding, subdural recurrence, infections, seizures, and cardiopulmonary consequences. Therefore, a multidisciplinary postoperative critical care is often needed [36, 70].

The prognosis is habitually much worse than chronic SDH and epidural hematoma. The extent of primary brain lesions underlying the acute subdural hematomas seems the most significant factor affecting the outcome [22, 33, 70]. However, an aggressive medical management and a rapid surgical decompression may improve the initial neurological status [33, 40]. [For further details about cranial acute and subacute SDHs, please refer to Chap. 2 of the present book.]

1.3 Cranial Chronic Subdural Hematoma

Chronic form of SDH is a collection of old blood products (more than 3 weeks old) that have accumulated in the subdural space generally in the elderly due to brain weight diminution and subdural space expansion with age. The incidence rate of this intracranial hematoma is about 80–120 cases per 100,000 persons in aged population [11, 65]. However, this value is expected to increase considerably from 2020 to 2040 to the point that chronic SDH will become the most frequent cranial neurosurgical pathology among adults by the year 2030 in the United States of America [11, 59].

Intracranial chronic SDH had multiple potential etiologies (head injury, neoplasm, vascular malformation, intracranial hypotension,…) and risk factors (chronic alcoholism, seizure, cerebrospinal fluid shunt, cranial/spinal surgery, coagulopathies, male gender…). However, its physiopathology is not completely understood [5, 11, 34, 38, 59]. Three centuries ago, chronic SDH was recognized as a stroke [75]. One century later, it became an inflammation [63, 69, 75]. Then, a traumatic origin was accepted in the early twentieth century (traumatic tearing of the bridging veins which connect the brain cortex with the dura mater) [63, 71]. After that, various further suggestions such as osmotic pressure or effusion were advanced. Some investigators have also found that both coagulation and fibrinolysis systems were excessively activated in chronic SDH. Also, it has been proposed that more complex factors are simultaneously implicated including angiogenesis, inflammation, recurrent microbleedings, exudates, and local coagulopathy [21, 25].

Otherwise, several and various risk factors have been associated with the occurrence of this intracranial hematoma. Lately, a number of authors have assumed the role of “cranial morphology” (symmetrical or asymmetrical) on the location and development of chronic SDH (refer to Chap. 6 of the present book) [5].

Usually clinical evolution is divided into three separate stages: the initial traumatic incident (often minor and sometimes unnoticed), the latency phase, and the real clinical presentation period. Therefore, patients with chronic SDH can be asymptomatic or can have very mild symptoms such as headache, confusion, language difficulties, nausea, vomiting, vertigo, asthenia, progressive mental deterioration, gait disturbance, limb weakness, or incontinence. They may also present acute and grave symptoms with varying degrees of hemiplegia, seizures, or even coma [6, 7].

Cranial chronic SDH is usually diagnosed by CT scan. This hematoma classically appears as a concavo-convex pericerebral fluid collection along the cranial convexity. Most commonly the density of the collection is low; however, isodense and mixed or heterogeneous density lesions are also seen (Fig. 1.4a–d). Chronic SDHs are often unilateral with significant mass effect and midline shift; nevertheless, some others can be bilateral, interhemispheric, but rarely in the posterior fossa or adjacent to the skull base. Although rare, some hematomas may be organized, calcified or even ossified (Fig. 1.4e, f) [73]. In some unusual presentations, supplementary post-contrast CT scan and even better MR imaging offer important characteristics in determining the exact topography of the hematoma, its relationship with contiguous anatomic structures, and its potential underlying etiologies (e.g., tumors, vascular malformations, inflammatory lesions, infections...) (Figs. 1.5, 1.6, and 1.7) [15]. In addition, other testings can be useful as laboratory investigations in order to search for an occult coagulopathy or hematologic disease. Therefore, diagnosis of chronic SDHs can be challenging due to the variable clinical presentations of the disease and potentially subtle neuroimaging appearances (refer to Chap. 26 of the present book). For that reason, a high index of suspicion needs to be kept in mind to avoid mismanagement and possible complications of this common neurosurgical entity.

Although there is a part for nonoperative medical management strategies (Figs. 1.8 and 1.9) [Refer to Chap. 27 of the present book], surgical closed-system drainage remains the basis of current therapy in symptomatic patients. This surgical technique considerably decreases the possibility of recurrence of chronic SDH, length of hospital stay, postoperative complications, and mortality.

Formal surgical options are widely used such as burr holes (Fig. 1.10) (two, single large or trephination), twist drill craniostomy, and craniotomy (Fig. 1.11) with or without subdural closed-system drainage. Other various surgical procedures have been described worldwide but the experiences are limited and the encouraging results lack perspective [66]. Among the surgical techniques reported, we list: hollow screws, subduroperitoneal shunt, implantation of an ommaya reservoir for repetitive punctures/aspiration of subdural collections, Subdural Evacuating Port

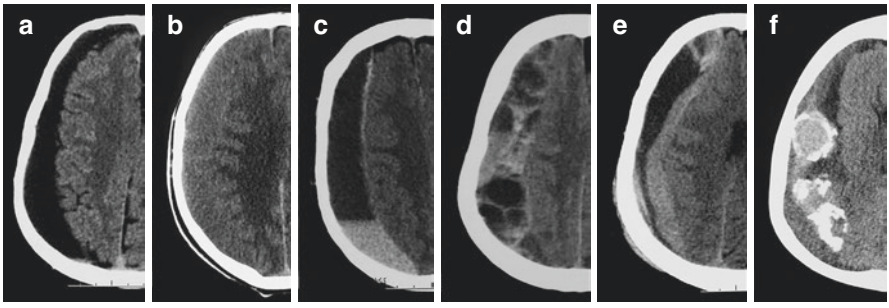

Fig. 1.4 Computed tomography appearance of various types of intracranial chronic subdural hematomas. (**a**) Low density. (**b**) Isodensity. (**c**) Acute on chronic SDH. (**d**) Mixed density with multilayer loculations. (**e**) Organized SDH. (**f**) Calcified/ossified SDH

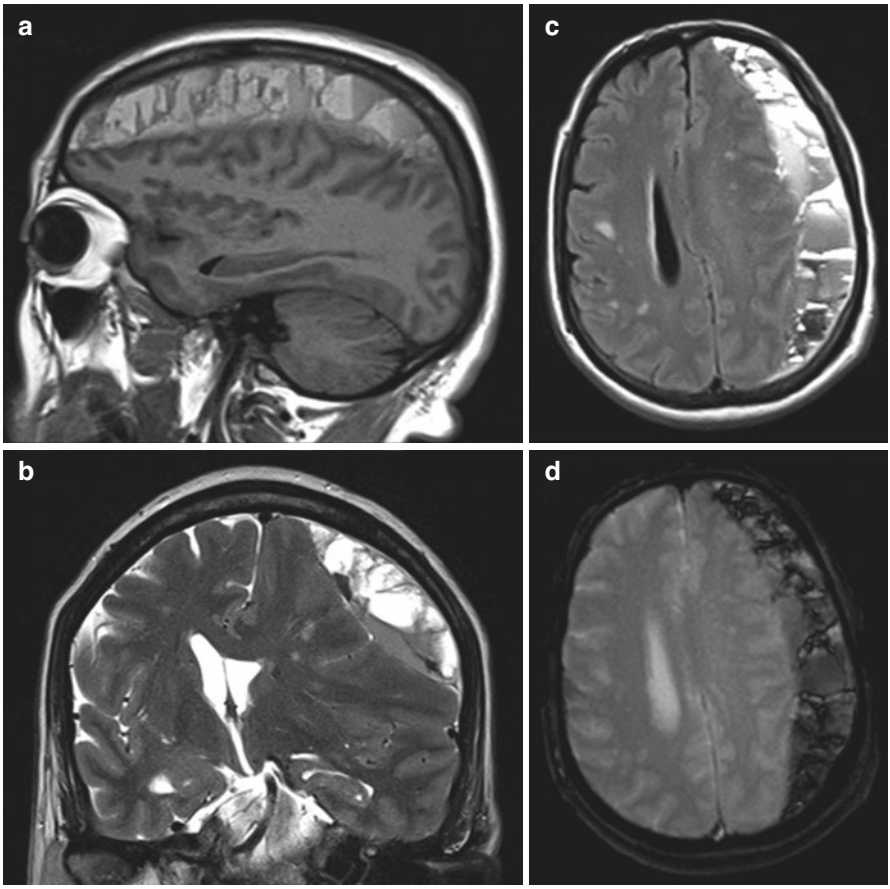

Fig. 1.5 MRI appearance of a heterogeneous mixed intracranial chronic SDH with multilayer loculations on the left side (**a**–**d**)

System (SEPS), endoscopic hematoma removal, and middle meningeal artery embolization [14, 30, 78].

Surgical evacuation of the chronic SDHs is indicated for symptomatic patients or when the hematoma had a maximum thickness superior than 10 mm with brain mass effect. Although surgery for intracranial chronic SDH is thought to be a relatively simple and safe procedure with a low complication rate, reported incidences of postoperative complications can reach 38% of operated patients [Refer to Chap. 33 of the present book]. Complications include those directly related to surgery or surgical techniques (recurrence, seizures, new intracranial bleeding, tension pneumocephalus, and infection), while others are called nonsurgical (common medical) complications. All these problems can adversely impact morbidity and mortality as well as contribute substantially to the costs of treatments and the hospital stay [65].

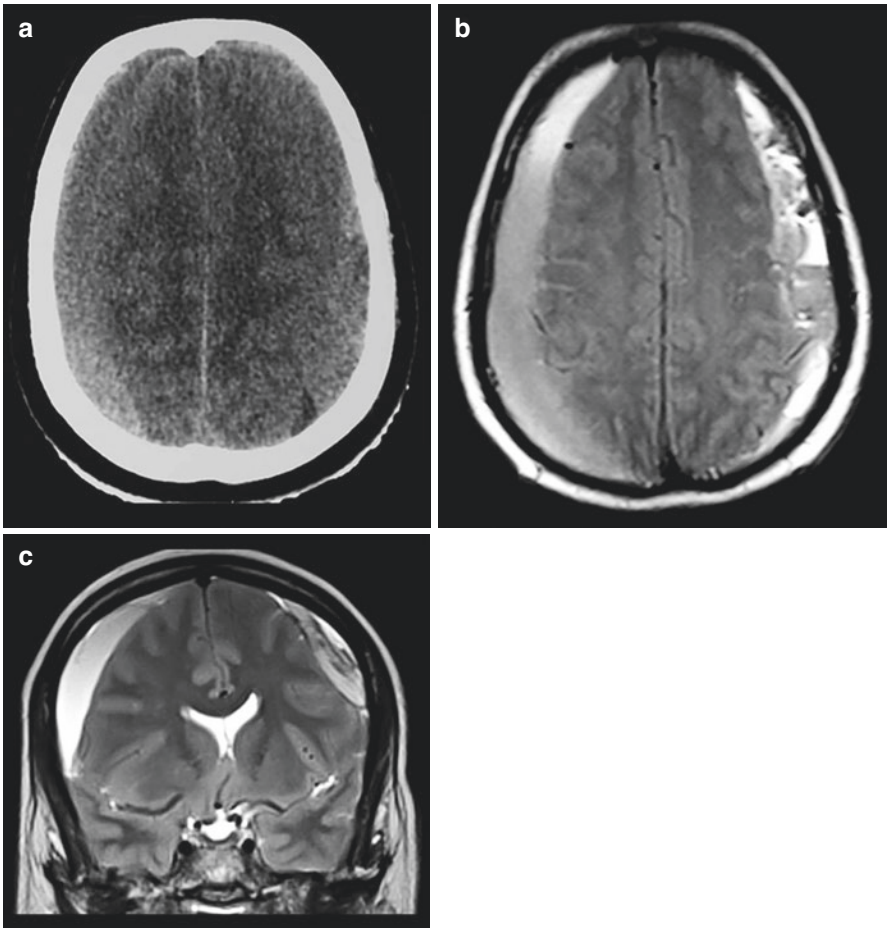

Fig. 1.6 Bilateral intracranial isodense chronic subdural hematoma on CT scan (**a**). Better distinction of the SDH on MRI: axial (**b**) and coronal (**c**) T2-weighted images

Benefit of antiepileptic agents is unclear. For some practitioners, seizure prophylaxis is used systematically. In our practice, like most neurosurgeons, antiepileptic drugs are not recommended if there are no seizures. If a late seizure occurs, a long-term therapy is required. The efficacy of corticosteroids is currently recognized [72]. They can be used as a monotherapy or as an adjunct to surgery. Coagulopathies should be corrected taking into consideration its risks/benefits.

The prognosis of chronic SDH is normally much better than the one of acute SDH. Morbidity and mortality rates of patients operated on for a chronic SDH depend mostly on the surgical technique, the patients' age and comorbidities, and the initial neurological status. The overall postoperative favorable outcome is reported to be up to 90% with younger patients usually attaining better outcomes

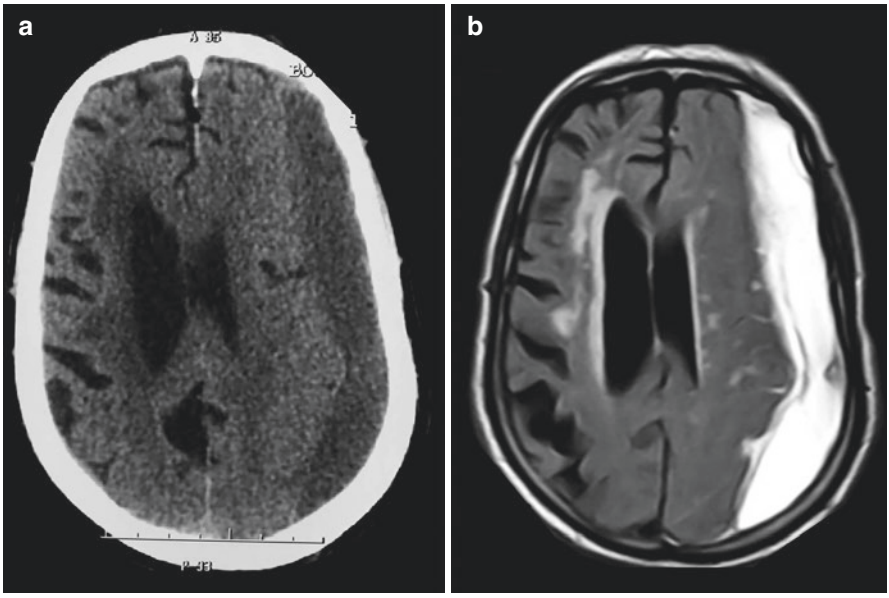

Fig. 1.7 Intracranial chronic SDH on the left side in the same patient on axial CT scan (**a**) and inversion recovery sequence (MRI) (**b**)

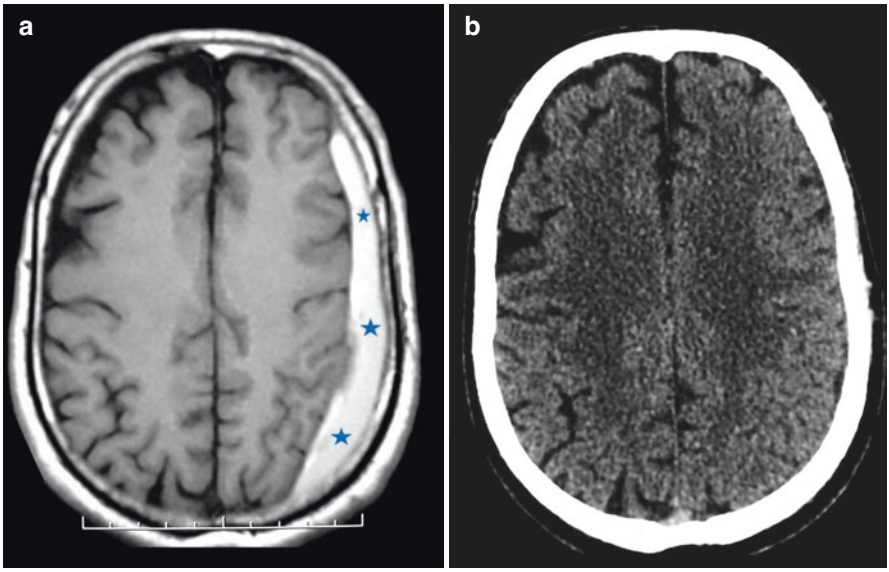

Fig. 1.8 Paucisymptomatic 53-year-old man with an intracranial left chronic SDH on T1-weighted MRI (**a**). This patient was treated by corticosteroids (hydrocortisone) and oral rehydration for 8 weeks. Control CT scan (at the end of the second month following the initial MRI) revealing the complete regression of the subdural collection (**b**)

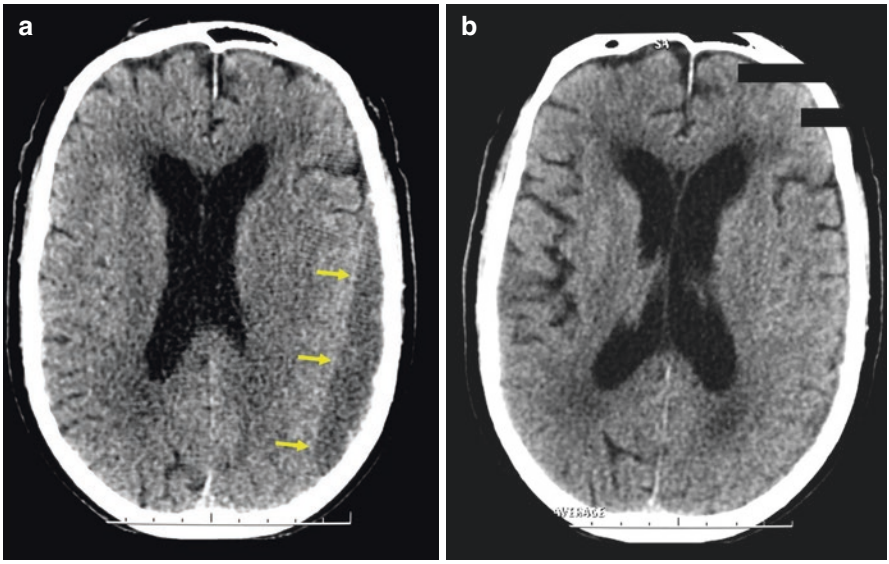

Fig. 1.9 Paucisymptomatic intracranial chronic SDH in 72-year-old man treated by corticosteroids and oral rehydration for 6 weeks. Initial CT scan (**a**) and control CT scan 2 months later showing the disappearance of the hematoma (**b**)

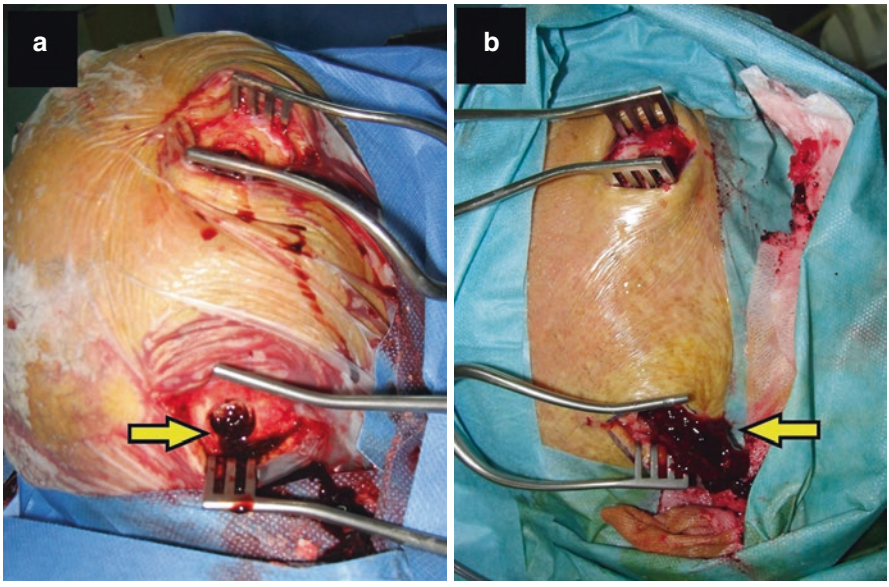

Fig. 1.10 Operative view. Surgical evacuation of chronic subdural hematomas through two burr holes. First case: evacuation of a brownish "motor oil" subdural fluid (arrow) (**a**). Another case: the chronic subdural hematoma contains darker clots (arrow) (**b**)

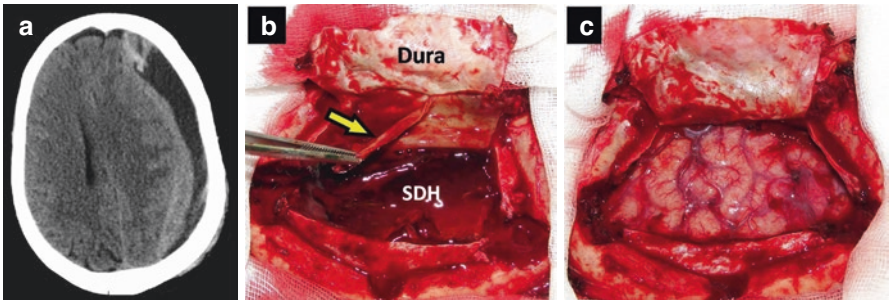

Fig. 1.11 Organized chronic/subacute subdural hematoma on axial CT scan (**a**). This patient was treated by large craniotomy and dura opening. We can see the organized SDH with its thick fibrous capsule (arrow) (**b**). Operative view of the cortical brain parenchyma following hematoma evacuation and extended membranectomy (**c**)

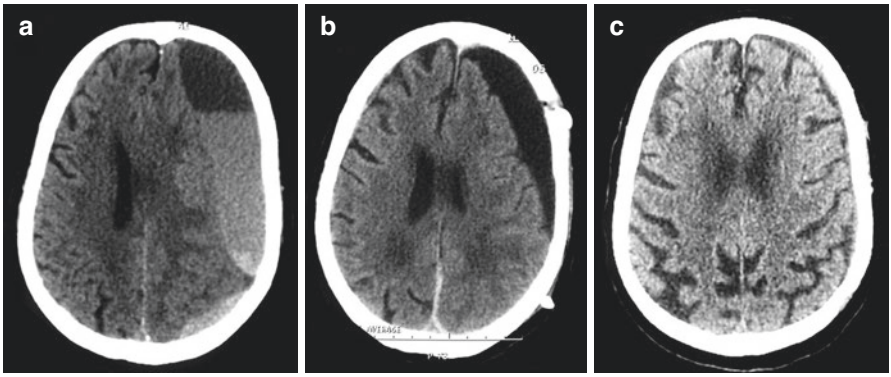

Fig. 1.12 Axial CT scan before (**a**), at third postoperative day (**b**), and 3 months after evacuation of a chronic subdural hematoma using two burr holes (**c**) with a good outcome in an 83-year-old woman

compared to the elders [66]. Also, patients with high subdural collection pressures tend to have faster cerebral expansion and better neurological improvement than cases with low pressures (Fig. 1.12).

1.4 Spinal Subdural Hematoma

The prevalence of spinal SDH is much lower than the one of cranial SDH because the intraspinal subdural space lacks important blood vessels or bridging veins which could be a cause for subdural bleeding [35, 54, 68]. Nevertheless, damage to radicular veins that cover the spinal subdural space may be the source of bleeding [61]. On the other hand, subdural hematomas are among the most uncommon types of spinal

hematomas. In a systematic review of 613 patients with spinal hematomas conducted by Kreppel et al. in 2003, only 4.1% of the cases had a subdural blood collection while 75.2% (461 patients) had epidural hematomas followed by 15.7% (96 patients) with subarachnoid hematomas [46]. In a more recent review of the literature, all reported cases of spinal SDH are about 259 [35]. Chapter 40 of the present book provides an in-depth review of knowledge of the management of spinal SDH.

There are several different conditions or etiologies that can cause spinal SDH, including bleeding disorders, hematologic disease, anticoagulation therapy, traumatic injury, iatrogenic injury (lumbar puncture, epidural anesthesia, following spine or even cranial surgery), intraspinal arterio-venous malformations, and intraspinal tumors [1, 2, 8, 20, 35, 39, 48, 51, 53, 55, 61]. In some cases, the exact cause of the bleeding is not known and the SDH is then called "idiopathic" [3, 44].

Clinical symptoms of spinal SDH are not specific and vary depending on the size, level, and cause of hematomas. Apart from post-traumatic forms, most cases will present acute or subacute installation of back pain and varying degrees of neurological symptoms such as radiculopathy, leg weakness, and sphincter disturbances. Although rare, a few cases of spinal SDH also had simultaneous intracranial SDH (see below).

Long unrecognized pathology, spinal SDH has surely benefited from the contribution of MR imaging scanning that is currently the neuroimaging modality of choice [23]. MR imaging can show both the subdural hematoma, its location, and may reveal the underlying tumoral or vascular etiology. The hematoma itself has a variable T1 and T2 signal depending on the age of the bleeding (Fig. 1.13). The axial view is particularly important for differentiation between subdural and epidural hematomas. Spinal epidural hematoma has a convex lens-like form and is typically localized posteriorly to the spinal cord while subdural hematoma is usually found ventrally and laterally to and around the spinal cord in a semi-circular

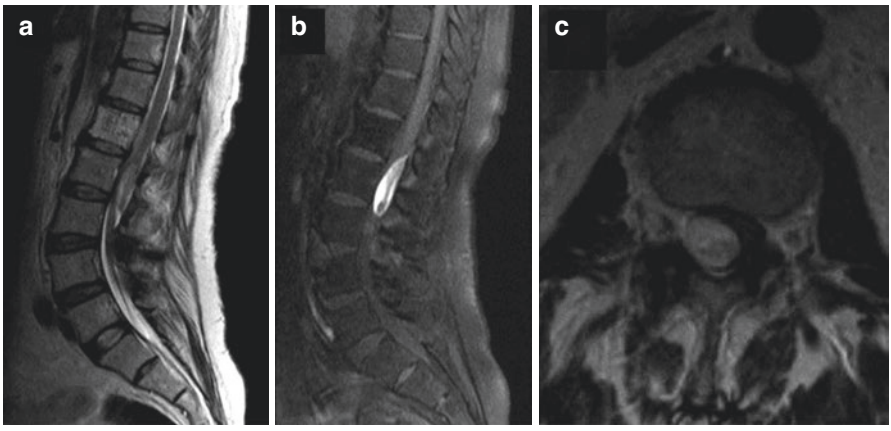

Fig. 1.13 (**a**) Preoperative images from a sagittal T2-weighted MRI, (**b**) sagittal T2-weighted FSE MRI, and (**c**) axial T2-weighted MRI revealed a spinal subdural hematoma at L2. (Reproduced from Kobayashi K et al. Eur Spine J (2017)26:2739–43; with permission) [44]

"cap sign" pattern, or in tri-radiate pattern called "inverted Mercedes Benz sign" at lumbar area [43, 47]. However, clear distinction between epidural and subdural forms of spinal hematomas before surgery can be difficult and many cases were only diagnosed intraoperatively. If a vascular malformation is suspected, a prompt spinal selective angiography should be performed or at best an angio-MR to reduce iatrogenic problems [1].

Based on many publications, treatments of spinal SDH are various [9, 49, 61]. When surgery is discussed, the neurosurgeon should always take into account the underlying etiology of the bleeding, spinal stability, patient's general conditions, and any coagulation disorders that could be associated. Generally, it appears that in patients with severe neurological symptoms, surgical decompression and evacuation have usually been achieved with good results (Fig. 1.14). However, surgical procedure should be done without delay unless the patient's conditions do not allow any anesthesia or operation. In paucisymptomatic or asymptomatic cases, both conservative and surgical treatments have allowed successful outcome. Therefore, these cases could be managed conservatively with attentive clinical follow-up. An interesting mini-invasive procedure should be mentioned: lumbar puncture for spinal SDH involving the lumbar and/or sacral column [37, 49, 56].

It is obvious that the main predictive factor of outcome will depend on neurological disorders at initial clinical presentation. Finally, patients with lumbar spinal SDH had a better outcome than those with cervical or thoracic ones.

1.5 Combination of Cranial and Spinal Subdural Hematomas

Although rare, neurosurgeons should consider the possibility of occurrence of spinal SDH in association with cranial SDH. To the best of our knowledge, less than 50 cases of craniospinal SDHs have been reported in the literature (Table 1.1) [17, 26, 29, 31, 35, 37, 42, 45, 57, 67, 68, 74, 77]. For Kokubo et al., this entity is underdiagnosed. In their prospective study, 1.19% (2 from 168) of patients surgically treated for cranial chronic SDH had concomitant lumbar SDH but these were asymptomatic [45].

This rare association can be separated into three forms as follows:

- *Type 1:* Concomitant spinal SDH and cranial SDH.
- *Type 2:* Spinal SDH developing following cranial SDH.
- *Type 3:* Cranial SDH developing following spinal SDH evacuation.

Consequently, the pathogenesis of combination of cranial and spinal SDH remains ambiguous, but a variety of hypothesis has been suggested according to the three forms:

In *type 1*, both hematomas developed as a result of trauma instead of common isolated spinal SDH, which mostly occurred owing to coagulopathy or other non-traumatic etiologies. In *type 2*, gravitational migration of the intracranial SDH

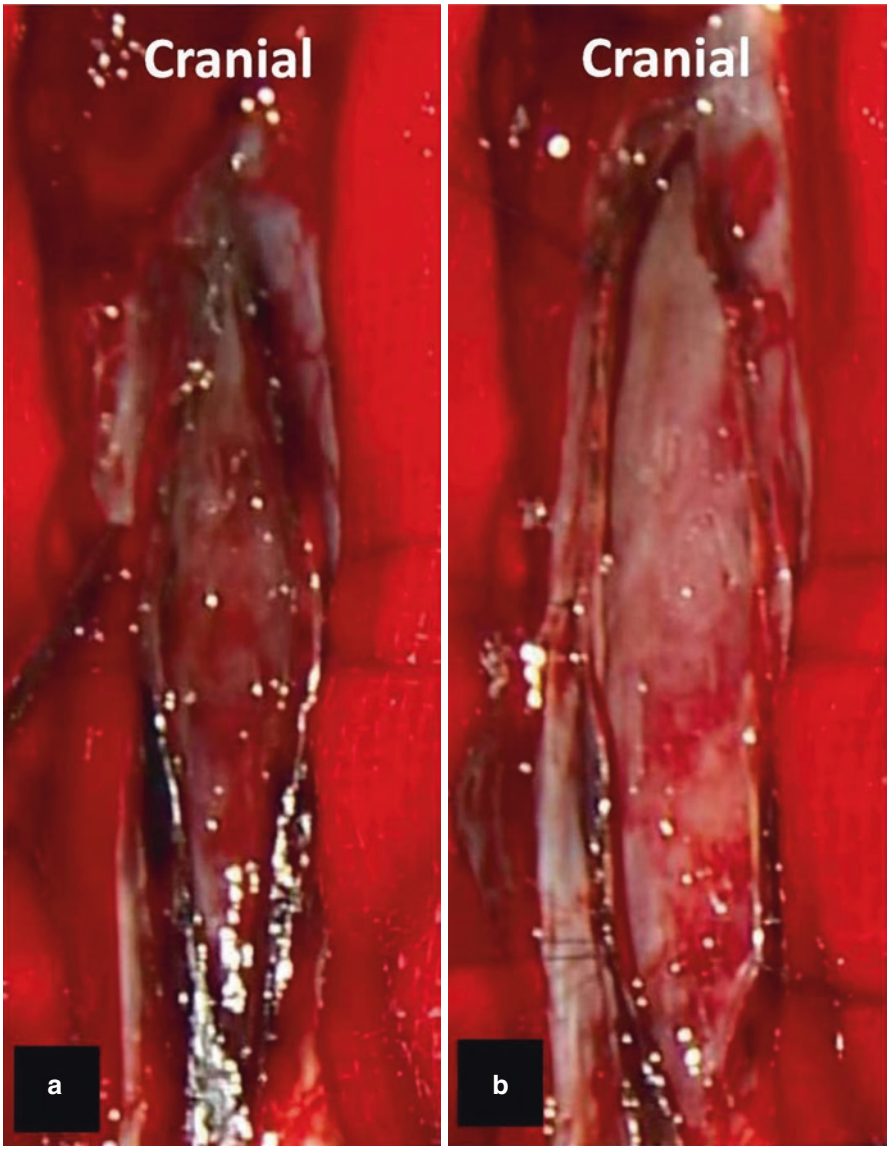

Fig. 1.14 Intraoperative photographs. (**a**) The dura and arachnoid membrane were exposed, and the spinal cord was centrally retracted to reveal a hematoma in the ventral space. This led to diagnosis of subarachnoid hematoma. (**b**) After removal of the hematoma, the spinal cord was decompressed (Reproduced from Kobayashi K et al. Eur Spine J (2017)26:2739–43; with permission) [44]

Table 1.1 Literature review of 48 patients with cranial and spinal subdural hematoma association

First author, year [reference]	Age/ sex	Predisposing event/ factors	Succession of detected lesions	Location of cranial SDH	Location of spinal SDH	Treatment of cranial SDH	Treatment of spinal SDH	Outcome
Lee, 1996 [49]	15 y/M	Trauma	Cranial	Unilateral (ASDH)	Lumbar	Conservative	Lumbar punctures	Good
Shimada, 1996 [35]	68 y/ Unk	Trauma	Cranial	Unilateral	T5-S2 (ASDH)	Conservative	Surgery	Good
Leber, 1997 [37]	54 y/M	Trauma	Cranial	Bilateral	L1-S2	Conservative	Surgery	Good
Tillich, 1999 [37]	54 y/M	Trauma	Cranial	Bilateral	T12-S2	Conservative	Surgery	Good
Kirsch, 2000 [43]	42/M	Attempting suicide	Spinal	Posterior fossa	C1-L3 (ASDH)	Conservative	Surgery	Poor
Hung, 2002 [35]	12 y/M	Trauma	Cranial	Unilateral (ASDH)	L1-L5	Conservative	Conservative	Good
Lecouvet, 2003 [68]	31 y/M	Metastatic melanosarcoma treated	Spinal	Unilateral. Posterior fossa	L1-S2	Surgery	Conservative	Improvement
Bortolotti, 2004 [35]	23 y/F	Trauma	Cranial	Unilateral	L4-S2	Conservative	Surgery	Good
Ahn, 2005 [3]	4 y/M	Trauma	Simultaneous	Unilateral. Posterior fossa	Cervico-thoracic	Conservative	Conservative	Improvement
Yamaguchi, 2005 [37]	59 y/M	Antiplatelet therapy	Simultaneous	Bilateral. Posterior fossa	T11-S1	Conservative	Conservative	Good
Jimbo, 2006 [74]	72 y/M	Anticoagulant therapy	Spinal	Multiple	L4-S2	Conservative	Surgery	Good
Sari, 2006 [67]	56 y/M	Trauma	Cranial	Interhemispheric	L1-S2	Conservative	Surgery	Good
Lee, 2007 [26]	68 y/F	No	Cranial	Unilateral	L4-S1	Surgery	Surgery	Good

(continued)

Table 1.1 (continued)

First author, year [reference]	Age/ sex	Predisposing event/ factors	Succession of detected lesions	Location of cranial SDH	Location of spinal SDH	Treatment of cranial SDH	Treatment of spinal SDH	Outcome
Morishige, 2007 [56]	54 y/M	No	Simultaneous	Unilateral. Posterior fossa	C1-S2	Surgery	Lumbar puncture	Good
Broc-Haro, 2008 [68]	44 y/M	No	Cranial	Unilateral (subacute SDH)	L2-L5 (ASDH)	Surgery	Conservative	Good
Gruber, 2008 [35]	4 m/M	Trauma (shaken baby)	Cranial	Unilateral (ASDH)	T10-L4	Ventricular shunt	Surgery	Improvement
Jain, 2008 [37]	12 y/M	Aplastic anemia	Simultaneous	Posterior fossa	C1-S3	Conservative	Conservative	Good
Nakajima, 2009 [37]	65 y/F	Trauma	Simultaneous	Unilateral	T12-S1	Surgery	Conservative	Good
Wong, 2009 [29]	73 y/F	Trauma	Cranial	Unilateral	T4-T10	Conservative	Conservative	Good
Yang, 2009 [37]	35 y/F	No	Simultaneous	Unilateral	L3-S1	Surgery	Surgery	Good
Hagihara, 2010 [31]	47 y/M	Trauma. Antiplatelet therapy	Cranial	Bilateral	L3-S1	Surgery	Conservative	Good
Kim K, 2010 [37]	24 y/F	Trauma	Simultaneous	Bilateral	L4-S2	Conservative	Conservative	Good
Nagashima, 2010 [37]	66 y/M	No	Spinal	Bilateral	L1-S1	Surgery	Conservative	Good
Nagashima, 2010 [37]	60 y/M	No	Cranial	Bilateral	L3-S2	Surgery	Conservative	Unk
Moscovici, 2011 [57]	88 y/M	Trauma	Cranial	Bilateral (ASDH)	L5-S1	Conservative	Surgery	Improvement
Wajima, 2012 [77]	78 y/F	Trauma Antiplatelet therapy	Cranial	Unilateral. Interhemispheric. Posterior fossa (ASDH)	S1-S2	Surgery	Conservative	Good

Wang, 2012 [37]	67 y/F	Antiplatelet therapy	Simultaneous	Unilateral	L4-S1	Surgery	Conservative	Good
Ji, 2013 [29]	47 y/F	Trauma	Simultaneous	Tentorium	L5-S2	Conservative	Conservative	Good
Jibu K, 2013 [29]	73 y/M	No	Simultaneous	Bilateral	L3-S2	Surgery	Conservative	Good
Li, 2013 [50]	26 y/M	Trauma	Cranial	Unilateral (ASDH)	T4-S1	Conservative	Conservative	Good
Moon, 2013 [37]	39 y/F	No	Spinal	Unilateral	L1-S2	Surgery	Conservative	Good
Kim, 2014 [35]	62 y/M	Trauma	Cranial	Unilateral	L2-L5	Conservative	Conservative	Good
Kokubo, 2014 [45]	83 y/M	Myelodysplastic syndrome	Cranial	Bilateral	L5-S1	Surgery	Conservative	Good
Kokubo, 2014 [45]	70 y/M	No	Cranial	Bilateral	S1	Surgery	Conservative	Good
Lin, 2014 [29]	70 y/M	No	Simultaneous	Bilateral	L4-S1	Surgery	Conservative	Good
Treister, 2014 [29]	15 y/M	Trauma	Simultaneous	Unilateral. Interhemispheric	T11-L4	Conservative	Conservative	Good
Cui, 2015 [17]	45 y/M	No	Spinal	Bilateral	L4-S3	Conservative	Surgery	Good
Kim MS, 2015 [42]	82 y/F	Trauma	Cranial	Bilateral	L3-L4	Surgery	Conservative	Good
Köksal, 2015 [35]	20 y/M	Trauma	Cranial	Unilateral (ASDH)	T10-L2	Conservative	Conservative	Improvement.
Kwon, 2015 [35]	57 y/M	Trauma	Spinal	Unilateral (ASDH)	L2-S1	Surgery	Surgery	Good
Kanamaru, 2016 [37]	67 y/M	Trauma	Cranial	Bilateral	L4-S1	Surgery	Surgery	Good
Matsumoto, 2016 [54]	58 y/M	Trauma	Cranial	Unilateral. Posterior fossa	T1-S1	Surgery	Surgery	Good

(continued)

Table 1.1 (continued)

First author, year [reference]	Age/ sex	Predisposing event/ factors	Succession of detected lesions	Location of cranial SDH	Location of spinal SDH	Treatment of cranial SDH	Treatment of spinal SDH	Outcome
Ichinose, 2018 [37]	40 y/M	Trauma	Simultaneous	Bilateral	L2-S1	Surgery	Lumbar puncture	Good
Satyarthee, 2018 [68]	14 y/M	Anaplastic anemia	Simultaneous	Posterior fossa	Thoraco-lumbar	Conservative	Conservative	Good
Uto, 2018 [74]	77 y/M	Anticoagulant and antiplatelet therapy	Spinal	Unilateral	L4-S1	Surgery	Conservative	Good
Fugita, 2019 [26]	63 y/F	Trauma	Cranial	Unilateral	L4-S1	Surgery	Conservative	Good
Golden, 2019 [29]	56 y/M	Trauma	Spinal	Bilateral (subacute)	T12-S1 (subacute)	Surgery	Surgery	Good
Hsieh, 2020 [35]	35 y/M	Trauma	Cranial	Bilateral + parafalcine (ASDH)	Thoraco-lumbar	Conservative	Conservative	Improvement

Abbreviations: *ASDH* Acute subdural hematoma, *CSDH* Chronic subdural hematoma, *m* months, *y* years, *M* male, *F* female, *C* cervical, *L* lumbar, *S* sacral, *T* thoracic, *Unk* unknown

downward to form a hematoma in the spinal canal (especially the lumbosacral region) has been supposed [3, 45, 50]. It was also speculated that the newly formed spinal SDH may be caused by tearing of the bridging veins in the posterior fossa especially following surgical evacuation of the cranial SDH. Lastly, in *type 3*, intracranial hypotension caused by spinal SDH evacuation may lead to the development of the cranial hematoma. In addition, the well-known frontal cerebral atrophy in aged patients may lead to a latent space for collection of cranial SDH [29, 35, 74].

Based on our review of the literature (Table 1.1), there was no precise age of presentation. The mean age reported was 49.37 years (range, 4 months–88 years) with male predominance: there was a 35:12 male/female ratio (undetermined sex in one case). Eleven patients (22.9%) had no predisposing event or risk factors. The origin of subdural hematomas was traumatic in 28 cases (58.3%) and seven patients (14.5%) were under anticoagulant and/or antiplatelet therapy. Hematologic disease was involved in three patients (6.2%). Both SDHs were diagnosed simultaneously (*type 1*) in 14 patients (29.1%). However, cranial SDH was detected firstly (*type 2*) in 25 cases (52.2%), and spinal SDH was detected before the cranial one (*type 3*) in 9 cases (18.7%). Intracranially, most of the hematomas were supratentorial and unilateral, whereas lumbosacral area was habitually involved in the spinal SDH.

Regardless of the type of craniospinal association, the appropriate treatment of both localizations of SDH is imprecise. Previous publications described both conservative and surgical approaches with overall very good results. However, most of the cases respond very well to conservative options especially for spinal SDH. Twenty-nine patients (60.4%) with spinal SDH were managed conservatively. Sixteen patients (33.3%) underwent surgical spinal evacuation but a lumbar puncture was attempted in only three cases (6.3%) [37, 49, 56]. This last technique is simple and useful for well-liquefied spinal SDH.

In all cases, neurosurgeons should be vigilant: patients with cranial SDH who develop neurological symptoms in the lower extremities should have MRI evaluation to eliminate spinal SDH. If neurologic symptoms are severe, or patient worsening, urgent surgical evacuation should be considered.

1.6 Conclusion

Subdural hematoma is a common heterogeneous pathologic entity with various manifestations that is more complex than previously thought. It includes cranial, spinal, acute, subacute, and chronic forms with sometimes mixed combination of each type. Since the age of the population, the number of road accidents, and the need of anticoagulation therapies will rise, then an increase in incidence of SDH rate is expected in the near future especially in intracranial localizations. Although conservative medical management strategies can be applied, surgical decompression of SDH with or without drainage remains the most used therapy for many

symptomatic cases. However, there is still some debate regarding the best strategy for treatment. Consequently, supplementary investigations focusing on etiopathogenesis and pathophysiology should be conducted to get a better management of SDH. When surgery is discussed, the neurosurgeon should always take into account the underlying etiology of the bleeding, clinical signs and symptoms, hematoma appearance and its localization, patient's general conditions, and any coagulation disorders that could be associated.

References

1. Abut Y, Erkalp K, Bay B. Spinal subdural hematoma: a pre-eclamptic patient with a spinal arteriovenous malformation. Anesth Analg. 2006;103:1610. https://doi.org/10.1213/01.ane.0000246274.96202.c7.
2. Abuzayed B, Oğuzoğlu SA, Dashti R, Ozyurt E. Spinal chronic subdural hematoma mimicking intradural tumor in a patient with history of Hemophilia a: case report. Turk Neurosurg. 2009;19:189–91.
3. Ahn ES, Smith ER. Acute clival and spinal subdural hematoma with spontaneous resolution: clinical and radiographic correlation in support of a proposed pathophysiological mechanism. Case report. J Neurosurg. 2005;103(2 Suppl):175–9. https://doi.org/10.3171/ped.2005.103.2.0175.
4. Akhaddar A. The yin-yang shaped image following head injury. Pan Afr Med J. 2013;16:133. https://doi.org/10.11604/pamj.2013.16.133.3555.
5. Akhaddar A, Bensghir M, Abouqal R, Boucetta M. Influence of cranial morphology on the location of chronic subdural haematoma. Acta Neurochir (Wien). 2009;151:1235–40. https://doi.org/10.1007/s00701-009-0357-7.
6. Akhaddar A, Karouache A, Boucetta M. Bilateral chronic subdural hematoma misdiagnosed as neuroleptic malignant syndrome. Emerg Med J. 2010;27:233. https://doi.org/10.1136/emj.2008.071001.
7. Akhaddar A, Boucetta M. Reversible tetraparesis due to bilateral chronic subdural haematoma. Age Ageing. 2010;39(Suppl). https://doi.org/10.1093/ageing/el_105.
8. Akiyama Y, Koyanagi I, Mikuni N. Chronic spinal subdural hematoma associated with antiplatelet therapy. World Neurosurg. 2017;105:1032.e1–5. https://doi.org/10.1016/j.wneu.2016.11.128.
9. Al B, Yildirim C, Zengin S, Genc S, Erkutlu I, Mete A. Acute spontaneous spinal subdural haematoma presenting as paraplegia and complete recovery with non-operative treatment. BMJ Case Rep. 2009;2009:bcr02.2009.1599. https://doi.org/10.1136/bcr.02.2009.1599.
10. Amagasaki K, Takusagawa Y, Kanehashi K, Abe S, Watanabe S, Shono N, et al. Supratentorial acute subdural haematoma during microvascular decompression surgery: report of three cases. J Surg Case Rep. 2017;2017(2):rjx004. https://doi.org/10.1093/jscr/rjx004.
11. Balser D, Farooq S, Mehmood T, Reyes M, Samadani U. Actual and projected incidence rates for chronic subdural hematomas in United States Veterans Administration and civilian populations. J Neurosurg. 2015;123:1209–15. https://doi.org/10.3171/2014.9.JNS141550.
12. Berger A, Constantini S, Ram Z, Roth J. Acute subdural hematomas in shunted normal-pressure hydrocephalus patients—management options and literature review: a case-based series. Surg Neurol Int. 2018;9:238. https://doi.org/10.4103/sni.sni_338_18. PMID: 30595959; PMCID: PMC6287333.
13. Caton MT Jr, Wiggins WF, Nuñez D. Non-traumatic subdural hemorrhage: beware of ruptured intracranial aneurysm. Emerg Radiol. 2019;26:567–71. https://doi.org/10.1007/s10140-019-01691-2.