Subdural Hematoma

18

Mehmet Turgut, Sinan Sağıroğlu, and Ali Akhaddar

18.1 Introduction

Since acute subdural hematoma (aSDH) is a pathology that is frequently diagnosed post-traumatic in emergency services, data on its frequency cannot be determined exactly and it varies between countries [1]. The incidence of non-traumatic aSDH cases is thought to be 3–5% [2]. Coombs et al. [3] in their literature review on non-traumatic aSDH screened 193 cases and stated that there was only a small number of cases reported in the current literature. 171 of the cases were over 40 years old and predominantly male. Arterial (61.5%), idiopathic (10.8%), coagulopathy (10.1%), oncologic (5.4%), spontaneous intracranial hypotension (5.4%), cocaine abuse (2.0%), arteriovenous malformation (1.4%) and arachnoid cyst, spontaneous occlusion of the Circle of Willis, brittle bone disease, meningioma, lifting heavy objects (0.07% each) are reported. Symptoms begin with gradually worsening severe headache and may evolve into symptoms of raised intracranial pressure (ICP) such as abducens palsy [3]

In epidemiological studies on chronic subdural hematoma (cSDH), incidence has been reported as 1.72–20.6 per 100.000, although it increases with the elderly [4–7]. The leading risk factors are advanced age, male gender, trauma/fall, anticoagulant/antiaggregant use, diabetes mellitus, alcohol abuse, epilepsy, and cardiovascular disease [1, 3, 6–9]. In cSDH, unlike aSDH, there is a process that spreads over time [1, 6]. In young patients, it often presents with a headache not accompanied by a neurological deficit [5, 9]. Common causes of admission are headache, gait disturbance, limb weakness and paralysis, altered mental status (delirium, confusional state, drowsiness, or coma), speech impairment, and epilepsy [5, 9, 10].

In the pediatric population, the most common cause of subdural hematoma (SDH) is shaken baby syndrome with a rate of 21/100.000 [11]. Caretaker abuse is most likely associated with retinal hemorrhages and additional physical injuries [12]. Other causes have been reported as trauma, surgical complications, fetal SDH, traumatic birth, aneurysm, arachnoid cyst (AC), hematological diseases causing coagulopathy, glutaric aciduria, galactosemia, and hypernatre-

M. Turgut (✉)
Department of Neurosurgery, Aydın Adnan Menderes University Faculty of Medicine, Efeler, Aydın, Turkey

Department of Histology and Embryology, Aydın Adnan Menderes University Health Sciences Institute, Efeler, Aydın, Turkey

S. Sağıroğlu
Department of Neurosurgery, Aydın Adnan Menderes University Faculty of Medicine, Efeler, Aydın, Turkey

A. Akhaddar
Department of Neurosurgery, Avicenne Military Hospital of Marrakech, Mohammed V University in Rabat, Rabat, Morocco

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mia [12]. There are few cases of idiopathic SDH in teenagers [2, 13, 14]. The authors suggest that spontaneous intracranial hypotension could be the etiology after the Valsalva maneuver [14].

SDH can have a range of symptoms that include headache, confusion, memory loss, difficulty speaking, weakness gait imbalance, and even coma [10]. However, it is also possible for a SDH to be present without any of these symptoms, or symptoms may have been relieved and forgotten at admission [14–16]. This is more likely to occur in cases where the hematoma is small or develops slowly over time. In this section, we will focus on rare cases in which intracranial SDH can be detected incidentally on radiological studies taken for patients with unrelated clinical presentations, apart from the causes of SDH that are frequently mentioned in the literature and that come to mind immediately today.

18.2 Subdural Hematoma in Adult Population

The main reason for the development of aSDH is trauma, and depending on the study, the incidence is 5–25% after severe head trauma [1]. Epidemiologic studies found around 24% of aSDH to be spontaneous, the rest is primarily of traumatic origin [1, 17]. Spontaneous SDHs generally have an underlying cause [2]. Very few cases were reported with negative work-up for underlying disease and considered idiopathic [2, 3, 14] (Fig. 18.1).

18.2.1 Idiopathic Subdural Hematoma

A few cases were reported in the literature with idiopathic aSDH or cSDH [2, 3, 14]. All of the cases were previously healthy, with no documented vascular malformation, coagulopathy, drug abuse, or oncologic etiology. Common factors in these cases are young age, a profession in which physical actions equivalent to the Valsalva maneuver are highly likely routine [2, 3, 14, 18, 19]. Four of these patients, also engage in vigorous physical activity [2, 3, 18]. Some papers reported the use of nonsteroidal anti-inflammatory drugs which may cause enlargement of SDH by hindering platelet function [3, 18, 19]. SDH specimens containing large blasts of atypical lymphoid proliferation or myeloid process may be seen [3].

Illicit drugs have been reported with SDH in literature such as cocaine and methamphetamine abuse [20, 21]. Both drugs are sympathomimetic

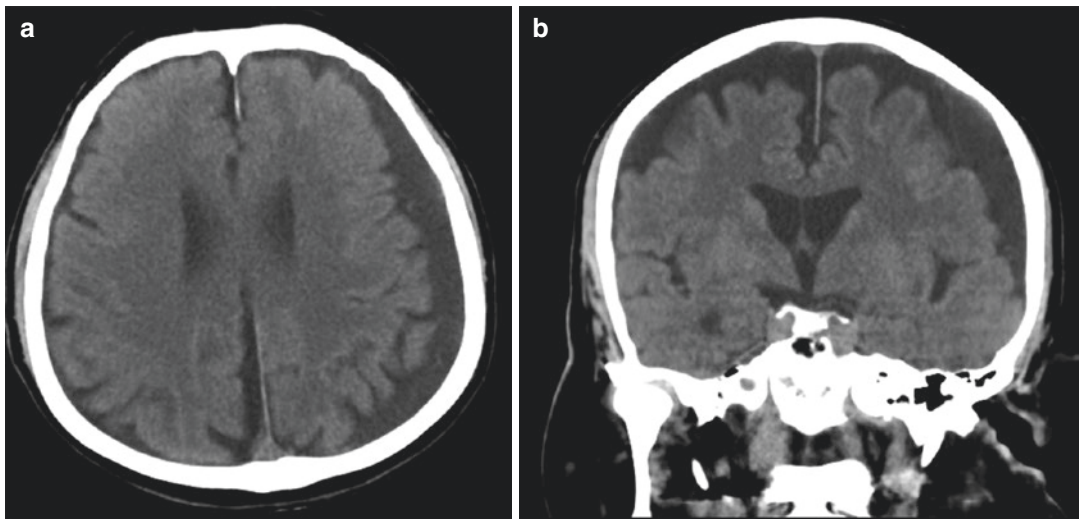

Fig. 18.1 Axial (**a**) and coronal (**b**) computed tomography (CT) images of a case of chronic subdural hematoma (cSDH) of an asymptomatic patient

and hypothesized to cause hypertensive SDH. High altitude may cause SDH in unaccustomed individuals [22, 23]. Increased cerebral blood flow and as a consequence, increased venous pressure might explain the pathogenicity of SDH [24, 25]. While reasons are unknown, momentary intracranial hypertension or hypotension, increased venous pressure, submaximal dynamic exercise, Valsalva maneuver, or dehydration are suggested mechanisms for the tearing of bridging veins [14, 18, 19, 25].

18.2.2 Arterial Hemorrhages and Vascular Malformations

Rupture of the cortical artery is the leading etiology in spontaneous aSDH [3]. Multiple studies found male predominance for arterial aSDH [26, 27]. Patients refer to their sudden severe headache as "the worst headache ever," similar to the thunderclap headache of subarachnoid hematoma [28]. There are several theories of which artery may rupture without trauma. One theory is that arterial twigs arising from cortical artery or attachment of these twigs to the arachnoid, which is thought to be the result of a previous microhemorrhage, is a structural weakness and flimsy to pressure difference, such as hypertension and sudden movement [27–32]. This theory is supported by direct intraoperative and autopsy observation of defects in the cortical cerebral wall [27, 29, 32, 33]. Hypertension is the main risk factor for these hematomas, especially combined with alcoholism [26, 27, 29, 30, 34]. The most common anatomic location of rupture is arteries at or near the Sylvian fissure [27, 29, 34] (Fig. 18.2).

Aneurysm, arteriovenous malformation (AVM), dural arteriovenous fistula (dAVF), and moyamoya disease are rare conditions accompanied by aSDH [35–42]. A ruptured aneurysm may present with SDH with an incidence of 2–5.8%, while pure aSDH is a rare finding [3, 33, 35, 39, 43]. Cortical arterial aneurysms are likely to result in aSDH but few reports are available, while other locations such as internal carotid artery (ICA), posterior communicating artery (PCoA), ICA-PCoA bifurcation, anterior cerebral artery (ACA),

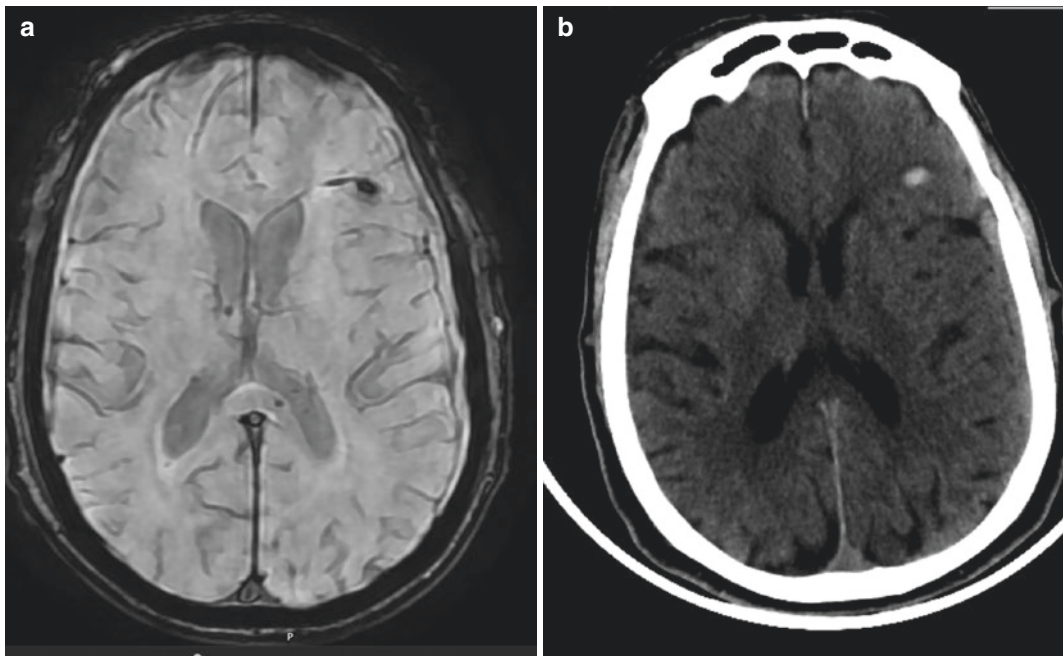

Fig. 18.2 A vascular malformation accompanied with a subacute subdural hematoma. Axial susceptibility weighted imaging sequence magnetic resonance imaging (**a**) and CT (**b**) images

anterior communicating artery (ACoA), middle cerebral artery (MCA) reported frequently [36, 39, 42–44]. High blood pressure during aneurysm rupture, especially in the setting of previous self-contained bleeds causing arachnoid adhesions, may lead to arachnoid rupture, resulting in aSDH [39, 42, 43, 45]. A recent study suggests that previous unremembered microtraumas may cause rupture and self-limiting hemorrhage in the distal part of cortical arteries of Sylvian fissure, which develop into pseudoaneurysms. A second minor trauma or hypertension may cause aSDH [46].

Mycotic aneurysm incidence is 2.5–4.5% among all aneurysms [47]. Infective endocarditis is a known risk factor [47–50]. While extremely rare, aSDH secondary to mycotic aneurysms of distal MCA had been reported. The infective SDH mechanism is explained with distal eventual localization of the mobile vegetation, causing cortical aneurysm, or arterial rupture without the evidence of an aneurysm [47–50]. Dural AVF is extremely rare to present with acute or chronic SDH and may be responsible for unexplained recurrent cSDH [38, 40, 41]. Dural AVF resulting from a middle meningeal artery (MMA) has been most frequently associated with SDH [41]. One explanation is bleeding from the venous part of dAVF originating from MMA may lead to cSDH [38]. Another recent study suggests that local venous pressure is amplified with arterial pressure due to the shunt, which may cause rupture of dural veins, resulting in SDH [40]. Endovascular embolization may be a sufficient treatment for such cases [38, 41]. AVM is another extremely rare cause of SDH [37, 42]. Adhesions of AVM to the arachnoid and strain of the arachnoid are thought to cause SDH [37, 51].

Moyamoya disease is an unusual condition in individuals with stenosis of supraclinoidal ICA and in those with collateral circulation [52]. While extremely rare to present with SDH, and with a variety of vascular malformations of moyamoya, rupture of an occult aneurysm or moyamoya vessel itself, high cerebral venous pressure, rupture or transdural anastomoses [52–54]. On the other hand, cerebral venous thromboses (CVTs) account for 0.5–1% of all strokes, which are known to result in intracranial hypertension, brain edema, venous infarction, and SAH, and may rarely present with SDH [55–57]. One explanation is venous hypertension may rupture dural veins, similar to dAVF [25, 40, 58].

Today, it is strongly suggested to take an angiogram in non-traumatic aSDH, if the neurological status of the patient can tolerate the time delay, to rule out underlying vascular malformations as it may change the surgical approach and survival of the patient [29, 33, 34, 37–39, 42, 45, 53, 59]. The initial angiogram may be negative for microaneurysms [36]. As an endnote, arterial ruptures may mimic saccular aneurysms in digital subtraction angiography through extravasation of contrast into SDH, especially in settings of anti-aggregation or antiplatelet drugs [27, 29, 30, 33, 34].

18.2.3 Oncologic Etiology

Shekarchizadeh et al. [44] reported that acute SDH due to neoplastic disease has an incidence of 7.8% of all spontaneous SDHs. Various primary intracranial tumors and metastatic tumors with dural involvement have been documented to present with SDH [44, 60]. While 20% of dural metastases are clinically silent, about 41% percent of dural involvement is also accompanied by SDH. Dural metastases may be an extension of skull metastases with an incidence of 57%, primarily lung, prostate, breast carcinomas, and Ewing sarcoma [60–64]. Hematogenous spread with 43% incidence, associated with advanced stages and with lung involvement [60, 62]. Another type of dural spread is from brain parenchyma, mostly seen in nonocular malignant melanoma [62] Sporadic cases of SDH as an accidental finding of malignancy have been reported [61, 65, 66] (Fig. 18.3).

Hematologic malignancies have a high incidence of 31% for SDH [67]. Acute myeloid leukemia (AML), lymphoma, acute promyelocytic leukemia(APL), and acute lymphocytic leukemia (ALL) may present with SDH [67–69]. Repeated lumbar punctures for intrathecal therapy, especially with coagulopathic cases may be the culprit [68, 69]. Various hematological oncologies

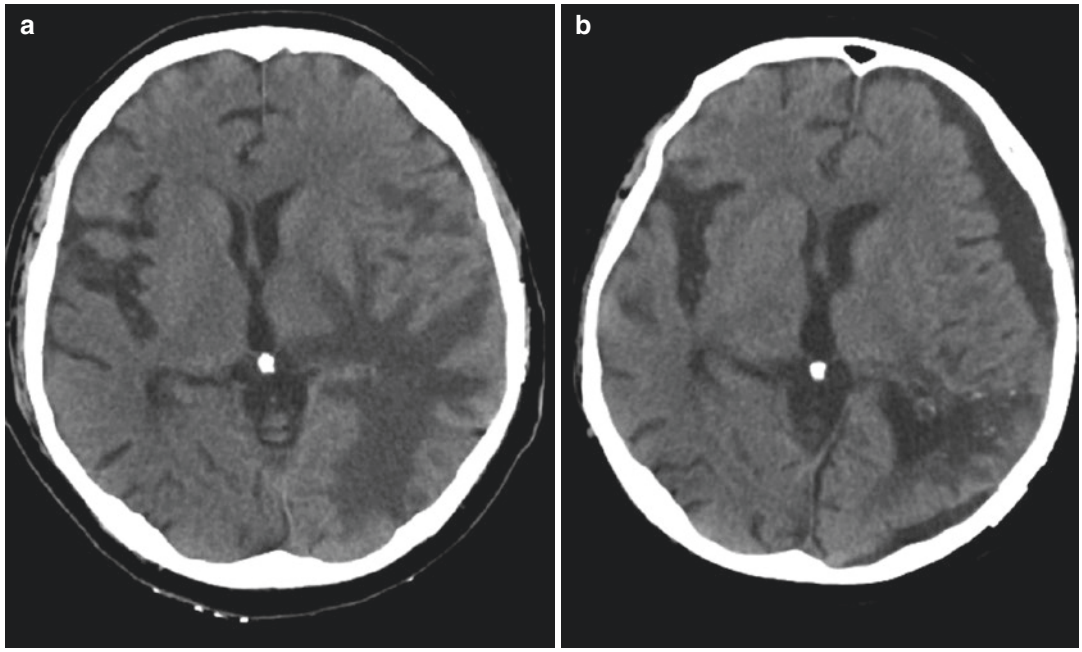

Fig. 18.3 The images show development of cSDH in a patient with glioblastoma multiforme over a 10-month period, with the pre-operative CT image (**a**) and the post-operative CT image (**b**)

such as chronic lymphocytic leukemia (CLL), and chronic myeloid leukemia (CML), have been suggested SDH as an initial finding of the underlying disease [70, 71]. Meningiomas are the most frequent primary tumor of the brain with an extremely rare presentation with SDH [72, 73]. Hematoma pathogenesis is unclear whether primary tumoral bleeding, vascular insult due to lesion mass, or secreted pro-angiogenic factors are the primary mechanisms [72, 73]. As a rule, SDH with surrounding membrane should be examined histopathologically if malignancy is suspected in patients such as elder patients, a history of weight loss, accompanied thrombocytopenia, or unexplained recurrence [64, 71, 74].

18.2.4 Spontaneaous İntracranial Hypotension

Spontaneous intracranial hypotension is an entity presenting with postural headache and low opening lumbar puncture pressure [75, 76]. Annual incidence is 5 in 100.000 per year [77]. Cases reported in the literature suggest spontaneous intracranial hypotension (SIH) associated with aSDH has an incidence of 5–10 and favors young and middle-aged with good neurological outcomes if treated promptly [3, 76]. Ferrante et al. [78] found the incidence of SDH with SIH was 16% and 97% of these were bilateral SDH. Hypotension may be idiopathic, as a result of cerebrospinal fluid (CSF) leak, or possibly postexertional to resistance exercise [19]. CSF leak may be spontaneous, or due to possibly trivial trauma, invasive procedures, or connective tissue diseases like Marfan's syndrome or Ehler–Danlos syndrome [68, 75, 78, 79]. Gadolinium-enhanced brain MRI and myelography of the central nervous system are recommended to differentiate [75, 80].

In this context, the critical point that the physician should pay attention to is whether the patient has a CSF leak because the treatment method is determined accordingly [75, 76]. SIH should come to mind in unexplained, bilateral SDH in middle-aged and young patients [78, 81, 82]. Without treatment of underlying CSF leakage, hematoma evacuation can lead to hematoma recurrence and even rapid deterioration after surgery [76, 78, 82].

18.2.5 Anticoagulants, Antiaggregants, and Coagulopathy

SDH may be the incidental finding of an underlying coagulopathy, which is mostly seen in the elderly, with a relatively high mortality rate [3, 59]. Thrombocytopenia may cause aSDH or cSDH in all age-groups [59, 83–85]. Several oncologic cases, with thrombocytopenia, have also been reported to result in SDH [65, 66, 74]. Liver cirrhosis also may lead to coagulopathy and increase the risk of SDH development [86].

Many infective pathogens are known to cause thrombocytopenia or coagulopathy with a variety of mechanisms. Kleib et al. [87] described a case of aSDH caused by coagulopathy due to Crimean-Congo hemorrhagic fever. Raj et al. [88] reported incidental SDH discovered in the setting of thrombocytopenia in a patient with malaria. Sandouno et al. [89] reported an interesting case of systemic lupus erythematosus (SLE) with the initial finding of aSDH. Jokonya et al. [90] reported human immunodeficiency virus (HIV) with cSDH in young patients. The authors propose coagulopathy due to retroviral infection as the culprit, while stating that dampened liver function via pathogen or antivirals may also cause coagulation factor deficiencies or dehydration that may result in cSDH.

cSDH with idiopathic thrombocytopenic purpura (ITP) may benefit from medical treatment without surgery if initial symptoms were mild, as restoring platelet function leads to the remission of hematoma [83–85]. Coagulation factor deficiencies such as factor I (fibrinogen), II, V, VII, VIII (hemophilia A), IX (Hemophilia B), X, XI, XII, von Willebrand disease have been proposed to create a predisposition to SDH [86, 89, 91–95]. Coagulopathy is associated with worse initial symptoms and poor outcomes in SDH [59].

Liver cirrhosis is a systemic disease with hindered factor synthesis, with a risk of 1–2% development of intracerebral hemorrhage [86, 96]. Hemophilia-related intracranial hematoma occurs at all ages but tends to favor the pediatric population [95]. Warfarin, which is a vitamin K antagonist, has a higher incidence of non-traumatic SDH, compared to traumatic cases [4, 17]. The use of warfarin may lead to an arterial rupture in hypertensive patients or due to minor trauma, making it more significant risk factor when compared to antiplatelet aggregation agents [4, 29].

Anticoagulant and antiaggregant treatment somewhat increase the incidence of cSDH, yet figures might be overestimated [4, 17, 59]. Antiplatelet therapy, commonly used in stroke patients and coronary diseases, is also a risk factor for SDH development and expansion [29, 59]. Anticoagulants have been shown to increase the mortality of SDH in hemodialysis patients [97]. Heparinization is a known risk factor and has a high incidence of SDH development, especially in patients undergoing routine hemodialysis [59, 97].

18.3 Subdural Hematoma in Pediatric Population

SDH is a rare condition in the young population, and the incidence of SDH in infants is increased compared to the rest of the pediatric population and is found to be 16.5/100.000 [11, 98]. However, it is not easy to diagnose in young children and infants. While it may be asymptomatic in newborns, it may present with clinical symptoms such as convulsions, apnea, and bradycardia [99, 100]. Infants and young children may have general symptoms such as fatigue, irritability, nausea, lethargy, and confusion [11, 101, 102].

18.3.1 Labor, Benign External Hydrocephalus, and Macrocephaly

Vaginal labor, whether traumatic or atraumatic, has been a known risk factor for perinatal intracranial hemorrhages [99, 103]. Compression during labor, tearing of falx or tentorium, and tearing of bridging veins are thought to result in SDH [104]. Blood products envelop the dural capillary bed, which is the primary CSF evacuation mechanism (minor CSF pathway) until the latter half of the first age [102, 105]. This hindrance of the minor

CSF pathway leads to hygroma formation, which leads to macrocephaly [102, 106]. This mechanism may be vice versa, rebleeding of stretched bridging veins due to hygroma resulting in recurrence of SDH [106, 107]. Thus, SHD may be an incidental finding of macrocephaly [106, 108].

While these mechanisms may explain SDH without a history of trauma, child abuse called shaken baby syndrome (SBS) should always be kept in mind. Occipital or infratentorial location of the SDH is a common sign of asymptomatic SDH. Likewise, the prognosis of incidental SDH is benign, and spontaneous resolution is expected by 2–3 years of age [99, 103, 109, 110].

18.3.2 Fetal Subdural Hematoma

There are limited reports on the suspected fetal origin of SDH, while it has 32% mortality [111]. One of the key features of this entity is the lack of apparent trauma or difficulty of birth, as almost all fetal SDH newborns are delivered with cesarean section [112–114]. The reports of vaginal birth with documented intrauterine SDH are scarce [115, 116]. Some reports document intrauterine intracranial hemorrhage and an increase in head circumference [114, 116]. Suspected etiology for in utero SDHs are maternal injury, bleeding disorders, and intracranial vascular malformations, yes most of the time prepartum history and postnatal evaluations fail to document such findings [113, 116, 117]. These reports show that however rare, routine intrauterine ultrasound evaluation may find incidental hydrocephalus which is revealed postnatally accompanying SDH [113, 116, 117]. Prenatal US features are intracranial echogenicity (42%), enlarged lateral ventricles (38%), presence of an intracranial mass (31), macrocephaly, (24%), displacement of falx cerebri from the midline (20%), intracranial fluid-filled collection (11%), reversed diastolic flow in MCA(11%) [111]. In such suspicious findings, fetal MRI could be considered may prove useful for revealing underlying pathology [115]. A multidisciplinary approach and swift response to minimize neurological sequelae to such cases are advised.

18.3.3 Arachnoid Cysts

AC is considered to be one of the most common intracranial masses with an incidence of 1% [118]. The annual risk for hemorrhage in the middle cranial fossa is thought to be below 0.1% [119]. AC is a known source of headaches in children, and gradual worsening may be the clue for hemorrhage [75, 120]. Hemorrhage may be spontaneous or after minor head trauma [120]. One of the theorized mechanisms is AC wall which is less compliant and this may cause to the rupture of bridging veins or unsupported veins around the AC wall, leading to SDH [121]. One of the alternative explanations is a one-way valve mechanism of CSF flow into AC or secretions of the AC wall itself may cause increased cyst pressure and rupture of the vascular AC wall [121, 122].

AC is a common entity and is mostly an incidental finding in children with headaches, with a benign outcome. Physicians should keep in mind AC is a predisposing factor for SDH development, especially after minor trauma. When patients who followed up with AC come with an advanced headache, control imaging should not be delayed.

18.3.4 Vascular Malformations

Fetal vascular malformations such as AVM, and aneurysms are well-researched and common causes of intracranial hemorrhage in the pediatric population [123]. Although aneurysms, dAVFs, and AVMs are known etiology of SDH in adults, vascular malformations seem rarely a cause of SDH in the pediatric population [3, 45]. Only a handful of cases with SDH had an aneurysmal origin [117, 124, 125]. AVMs in children are the main cause of spontaneous intracranial hemorrhage, and SDH is mostly reported to be a component of complex intraparenchymal-subarachnoid-SDH seen in AVM rupture [123, 126]. Development of aSDH is accepted to be the rupture of arterialized bridging vein from an AVM [126]. There is one pediatric case of SDH developed after AVF embolization. The authors commend rapid deflation of

giant malformation may be the culprit [127]. CVT has a prevalence of 0.67 per 100.000 among the pediatric population [128]. While the rare occurrence of CVT with SDH limits the number of cases, recent reports postulate previous SDH with mass effect is the reason for the development of CVT in infants, not vice versa [129, 130].

18.3.5 Hematologic Diseases and Coagulation Disorders

The factors affecting the coagulation pathway are on a wide spectrum and it is difficult to follow all diseases up to date. One of the well-reported conditions is SDH in the setting of hemophilia, both type A (factor VIII) and B (factor IX). Hemophilia is reported to have a central nervous system bleeding rate of 7.5%, and the incidence increases to 85.2% in the severe forms. SDH accounts for 19–29.8% of intracranial hematomas in hemophilia patients [95, 131]. Trauma accounts for 54.3% of SDH in hemophilia [95].

Hemophilia is a serious risk factor for an intracranial hematoma in general, carrier women should have genetic counseling. The delivery method should be decided accordingly by a multidisciplinary approach. And prophylaxis should start as soon as possible to avoid further complications [132]. Other factor deficiencies seldom reported with intracranial hematomas such as fibrinogen (factor I), factor VII, X, XIII, and von Willebrand factor [92–94, 131]. Accompanying hemorrhages such as muscular or retinal hematomas makes it difficult to differentiate unknown coagulopathy or nonaccidental trauma such as SBS [92, 94]. Idiopathic thrombocytic purpura is a very common hematologic condition in children and an extremely rare cause of intracerebral hematoma, approximately 1–10/1.000.000 [133]. Only a few cases were reported in the pediatric population with SDH [134–136].

Various types of leukemia may present with SDH as a first sign, and lack of abuse history and repeated infections may be the clue for neoplastic disease in children [137]. These hematological cancer types are acute lymphocytic leukemia (ALL), acute myeloid leukemia (AML), juvenile myelomonocytic leukemia, acute monocytic leukemia, and other variants [17, 137–142]. In earlier studies, ALL is found to be predisposing to cSDH, and the authors claimed morphological studies did not support lumber puncture as a cause [142]. Initial hemorrhage mechanism is due to intradural bleeding secondary to thrombocytopenia. Meningeal spread of leukemia is thought to be a prominent factor in sustaining SDH [139, 142].

18.3.6 Metabolic Disorders

Glutaric aciduria type 1 (GA1) is a rare metabolic disorder with an incidence of 20–30% of subdural hematoma development [143]. Microcephalic macrocephaly is the initial sign of the disease most of the time [144]. A recent review of GA1 with SDH found that 40% of the cases were related to trauma [145]. The probable pathogenesis of SDH is thought to be the rupture of bridging veins due to cerebral atrophy [144–147]. Another very rare disease, D-2-hydoxyglutaric aciduria type 1 has been diagnosed after incidental SDH, which was initially suspected to be abuse due to retinal hemorrhages and bilateral SDH [148]. In a clinical investigation of 9 infants with neuronal ceroid lipofuscinosis (NCL), 4 of 9 patients had incidental SDH without additional symptoms. NCL causes progressive brain atrophy, which is associated with stretching and tearing of the bridging veins [149]. Menkes disease is a disease of copper absorption and transport abnormality, characterized by seizures, developmental delay, and kinky hair [150–152]. This rare disease is another example of neurodegeneration and atrophy, resulting in stretching of the bridging veins [146, 150–152]. Lastly, SDH alone does not provide evidence for abuse or a genetic disorder [146]. A variety of genetic diseases rarely causes SDH, except for GA1, and may provide a challenge for physicians because of similarities with non-incidental trauma. Herewith, we compiled known cases to keep in mind the probability of underlying genetic origin.

18.4 Conclusion

SDH is one of the most common diseases a neurosurgeon encounters during practice. Acute forms usually result from trauma and chronic forms are seen mostly in the elderly after minor head trauma, which every neurosurgeon is accustomed to. Non-traumatic SDH is an overlooked entity, while incidence is around 24% for aSDHs. If an aSDH is suspected to be of arterial origin, craniotomy should cover the Sylvian fissure, which is the most probable origin of the hematoma. Underlying pathologies such as aneurysms, AVM, dAVF, and moyamoya disease require additional pre-operative planning and may require advanced surgical techniques compared to regular aSDH or cSDH operations. While rare in the pediatric population, vascular anomalies may also play a role in SDH development. Mycotic aneurysms have a chance to resolve with anti-biotherapy alone, and surgery can be reserved for cases with mass effects or deterioration. cSDH associated with dAVFs and with CSF leak may cause unexplained recurrence and the patient may go through several cranial surgeries to no avail. Additional evaluation of the patients such as an angiogram or enhanced MR myelography should come to mind in such cases. Bilateral unexplained SDH in middle-aged and young patients might be the clue for SIH. SDH may be a sign of a primary intracranial tumor, metastasis of cancer, or hematologic oncology.

Pre-operative evaluation of blood work-up should be carefully examined because several hematologic malignancies cause SDH, which may be the initial finding of the underlying disease. If there is a suspicion of oncological etiology, hematoma and surrounding membranes should be examined histopathologically. Various infections, factor deficiencies, liver cirrhosis, autoimmune diseases, anticoagulants, and anti-aggregant drugs have been reported with SDH. A comprehensive history of the patient should be taken carefully, and a hematologic work-up including bleeding time and hematology consult for additional tests are advised. In pediatric patients, hematologic diseases such as hemophilia, factor deficiencies, ITP, or malignancies may be accompanied by SDH. The neurosurgeon must be scrupulous with pediatric cases, as the majority of SDH is caused by abuse, which is accompanied by retinal hemorrhages and additional physical injuries. There are several diseases in the differential diagnosis of SBS. Fetal screening with ultrasound might find suspicious findings, and further investigation with MRI may be considered for revealing underlying pathology. A multidisciplinary approach, planning of delivery, and early intervention may minimize neurological sequelae. Vaginal labor itself may change the cranial vault due to compression, which may result in asymptomatic SDH, and have a benign outcome. Macrocephaly is a subject for research on SDH in newborns and infants.

Today, the most common genetic diseases are included in newborn screening. However, screening tests vary from one country to another, physicians should also keep in mind rare diseases may present initially with SDH. AC is a common intracranial mass that is mostly found as an incidental finding. The neurosurgeon may come upon the new onset of headache in followed-up patients with AC. SDH in the setting of AC should come to mind and control imaging is advised. In this chapter, the goal is to inform the neurosurgeon of the incidental appearance of SDH in literature. Proper diagnosis of the underlying disease is crucial for appropriate treatment, and benefit of the patient.

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