

The Neurocritical and Neurosurgical Care of Subdural Hematomas

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Abstract Subdural hematomas (SDHs), though frequently grouped together, can result from a variety of different etiologies, and therefore many different subtypes exist. Moreover, the high incidence of these lesions in the neurocritical care settings behooves practitioners to have a firm grasp on their diagnosis and management. We present here a review of SDHs, with an emphasis on how different subtypes of SDHs differ from one another and with discussion of their medical and surgical management in the neurocritical care setting. In this paper, we discuss considerations for acute, subacute, and chronic SDHs and how presentation and management may change in both the elderly and pediatric populations. We discuss SDHs that arise in the setting of anticoagulation, those that arise in the setting of active cerebrospinal fluid diversion, and those that are recurrent and recalcitrant to initial surgical evacuation. Management steps reviewed include detailed discussion of initial assessment, anticoagulation reversal, seizure prophylaxis, blood pressure management, and indications for intracranial pressure monitoring. Direct surgical management options are reviewed, including open craniotomy, twist-drill, and burr-hole drainage and the usage of subdural drainage systems. SDHs are a common finding in the neurocritical care setting and have a diverse set of presentations. With a better understanding of the fundamental differences between subtypes of SDHs, critical care practitioners can better tailor their management of

both the patient's intracranial and multi-systemic pathologies.

Keywords Subdural hematoma · Acute subdural hematoma · Subacute subdural hematoma · Chronic subdural hematoma · Operative management · Mass effect · Intracranial pressure · Review

Introduction

Subdural hematomas (SDHs) result from a variety of etiologies. Their clinical impact ranges from relatively minor to potentially life threatening, and significant nuances exist in their management. Given the high incidence of SDH in patients with neurocritical care needs, it is important for clinicians with routine exposure to these patients to have a firm grasp on their diagnosis and management. We present a review of SDHs, with particular focus on the various subtypes and how they differ in etiology, epidemiology, presentation, and management.

Classification

All SDHs are defined by the presence of blood between dura and arachnoid layers covering the brain. Aside from the anatomic location of blood collection, relatively few characteristics are consistent across all subtypes of SDHs. SDHs can be further classified by the time course of development (acute, subacute, chronic, acute on chronic), etiology (traumatic, spontaneous, secondary to other pathologies), age of the patient, and other special considerations (e.g., anticoagulation-induced, bilateral,

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recurrent). Often, multiple variables co-exist, and a given hematoma may harbor attributes of several subtypes.

The origin of subdural bleeding has been classically attributed to disruption of bridging veins running from the cortical surface to the overlying dura, although this is not always the case, as discussed below.

Acute Subdural Hematomas

General Considerations in Adults

In the young, healthy adult brain, acute SDHs typically follow significant head trauma and are observed in 12–29 % of severe traumatic brain injury (TBI) cases [1–4]. The presence of an acute SDH should be suspected in patients who present with decreased or progressively worsening levels of consciousness, and those with unilateral neurologic deficits (e.g., dilated pupil, motor weakness or posturing, or focal seizure activity). Diagnosis is confirmed on noncontrast computed tomography (CT), where an acute subdural hematoma (SDH) appears as a hyperattenuating mass overlying the cerebral convexity, falx cerebri, or tentorium cerebelli, classically with a crescent-

like or “half-moon” appearance that crosses cranial suture lines (Fig. 1). When present, acute SDHs are associated with high morbidity and mortality, with mortality rates as high as 50–90 % in patients who present with Glasgow Coma Scale (GCS) scores 8 or less [2, 4–8]. The prognosis for patients presenting with higher GCS scores is noticeably better, with studies reporting a 0 % mortality rate in those with a GCS score of 13 or above and a mortality rate of 21–46 % in patients with intermediate GCS scores [6, 7, 9]. On the whole, the presence of SDH in an acute traumatic injury portends poor prognosis [7, 9–11]. Among all patients with acute SDHs, fewer than 25 % ultimately achieve a full recovery without any major neurologic deficits [2, 4, 8, 9, 12, 13].

Several reasons exist for the significant morbidity and mortality associated with acute SDHs. As opposed to in elderly individuals or others with preexisting brain atrophy, blood vessels in the subdural space of young adults are protected by both the overlying skull and ample underlying brain parenchyma. As such, acute SDHs require antecedent high-energy collision or acceleratory forces. Not surprisingly, such mechanisms frequently generate other concurrent brain injuries [14–16]. Up to 83 % of patients

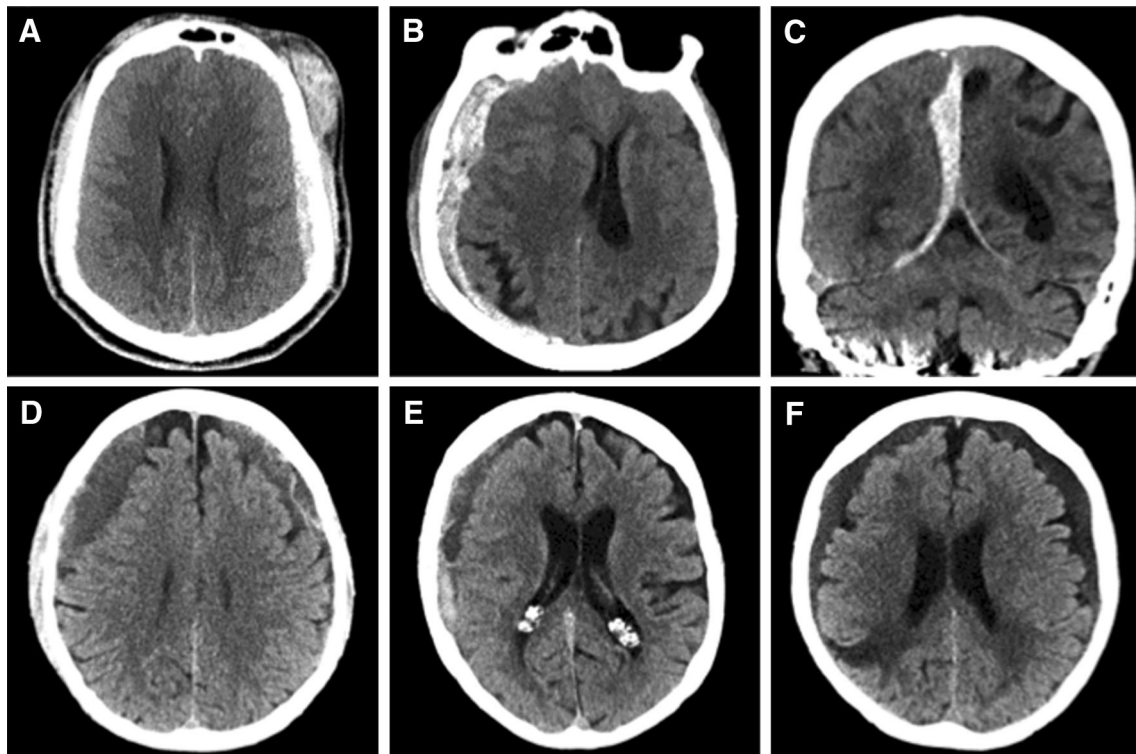


Fig. 1 Classic examples of SDH on CT scan, including **a** right-sided acute SDH in a 19-year-old male, note the associated diffuse cerebral edema, **b** right-sided acute SDH in a 93-year-old female, **c** acute parafalcine SDH in a 78-year-old male on aspirin and clopidogrel, **d** bilateral subacute SDHs in a 56-year-old male, **e** chronic SDHs

found in a 88-year-old male following a fall (no reported history of previous head trauma), and **f** acute on chronic SDH in a 79-year-old male who had fallen 2 weeks earlier, but presented with rapid-onset left-sided facial droop and hemiparesis

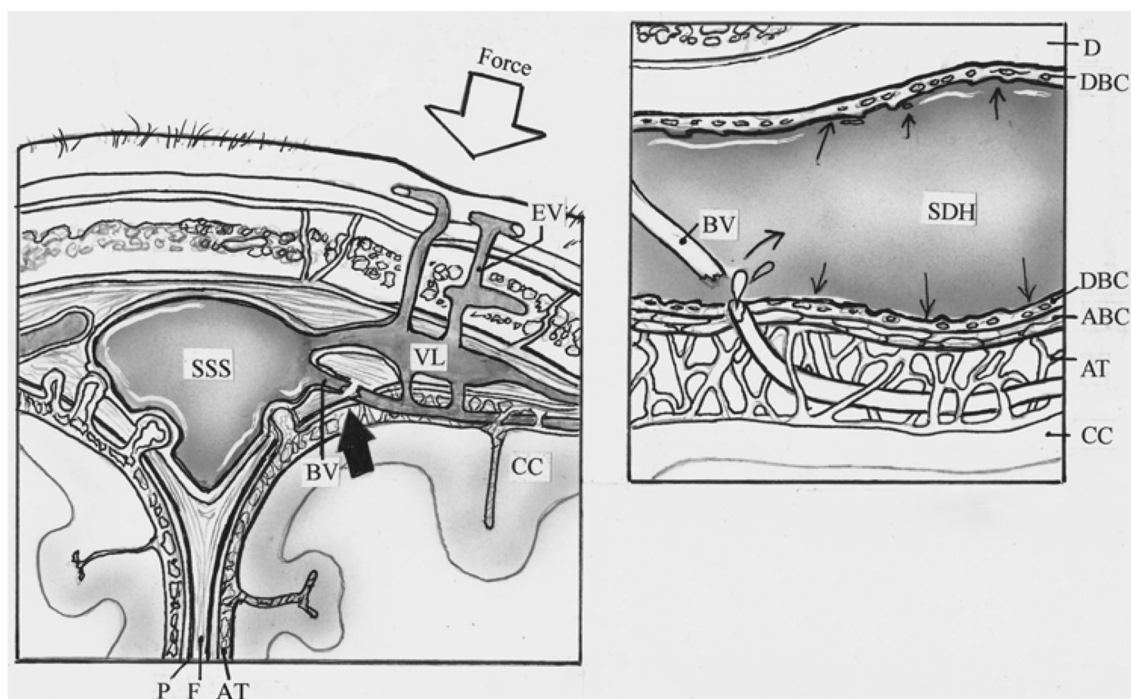


Fig. 2 Formation of a subdural hematoma. *Left* coronal section taken at the level of the SSS demonstrating tearing (*solid arrow*) of a bridging vein (BV) after forceful impact to the skull. *Right* detail of bridging vein tear (*curved arrow*) and accumulation of blood within the dural border

cell layer (*straight arrows*). ABC arachnoid barrier cell layer, AT arachnoid trabeculae, CC cerebral cortex, D dura, DBC dural border cell layer, EV emissary vein, F falx, P pia, VL venous lacunae. From [125]. Copyright Remi Nader. Published with permission

presenting with acute SDHs have associated brain contusions, and a substantial number have evidence of diffuse axonal injury [5, 14, 17]. Furthermore, a significant proportion of acute SDHs arise from hemorrhagic conversion of brain contusions and disruption of cortical arteries [2, 18, 19]. Thus, associated damage to brain parenchyma as well as potentially severe systemic injuries often compound the direct impairment caused by subdural bleeding in determining eventual outcome.

Considerations in Elderly Patients

Acute SDHs in older adults can present similarly to those in younger adults, with a few unique considerations. Due to age-related cerebral atrophy, the subdural space increases, placing cortical veins under increased tension and thus, more prone to injury. Therefore, SDHs occur not only more frequently in older adults but may also present with greater clot thickness and more midline shift [20, 21] (Fig. 2). Moreover, older adults harbor more medical comorbidities, elevating the risk of significant medical complications (myocardial infarction, pneumonia, etc.) during a patient's hospital course.

As such, it is not surprising that mortality from acute SDH is consistently elevated with increasing age. Adults

over 60 years of age have significantly higher rates of mortality following acute traumatic SDH, with some studies reporting a four times higher mortality in elderly than in younger patients [5, 7, 8, 20–22].

Considerations in Pediatric Patients

Infants and toddlers also harbor a higher incidence for SDHs compared to older children and adults [23–26]. This is attributable, in part, to nonaccidental trauma in infants, the so-called “shaken baby syndrome.” [23, 26] Abuse accounts for greater than 50 % of SDHs in toddlers and infants, and the finding of an SDH in a child should raise the concern for potential child abuse [27, 28].

The exact pathophysiology for the increased susceptibility of infants to develop SDHs is still not fully understood [29]. The unfused calvarial bones in infants allow for large fluctuations in dural intravenous pressure during even normal head movement, posing a possible role for venous hypertension in SDH formation [30]. Moreover, in cases of nonaccidental trauma, it has been hypothesized that periods of ischemia may also occur, leading to endothelial injury and subsequent bleeding risk [31].

SDH management in children similarly prioritizes monitoring of clinical decline and evacuating hemorrhages

with symptomatic mass effect. Patients with small lesions, no neurologic deficits, and no evidence of midline shift may be observed. Larger symptomatic lesions should prompt surgical evacuation or potentially needle aspiration if an open fontanel still persists [32–34]. It should be noted, however, that in infants, open craniotomy with surgical evacuation bears a higher risk of hypovolemic shock due to low total blood volume. While this consideration alone is not a contraindication to surgical evacuation, it nevertheless should be kept in mind during the intra-operative and post-operative care of young patients.

Subacute Subdural Hematoma

Hematomas begin to liquefy at 2 weeks after formation. Subacute SDHs represent evolving hematomas weeks after an initial insult as liquefaction of the initial clot causes symptomatic expansion [35]. Much like acute SDHs, subacute SDHs should be suspected in patients with a history of trauma, who then present with delayed onset of decreased mental status, unilateral focal deficits, or other signs of increased intracranial pressure (ICP). On non-contrast CT scan, a subacute SDH appears as an isodense mass, which may have been present on previous imaging studies; their density may be equal to that of normal brain tissue, and they may be missed on CT review. Care should be taken to distinguish them from other fluid collections of the subdural space that can arise in a subacute fashion, such as subdural empyemas or hygromas [36, 37]. The former can be distinguished by the presence of fever, leukocytosis, meningismus, and other signs of intracranial bacterial infection and the latter by its noted similarity to cerebrospinal fluid (CSF) on all imaging modalities.

Much like acute SDHs, subacute SDHs are typically associated with trauma. However, they carry a lower associated burden of mortality and morbidity, with some studies reporting a mortality rate one third of that encountered in acute SDHs [38]. Much remains unknown about these entities, however, and the vast majority of the literature is currently devoted toward the study of either acute or chronic SDHs. As such, there is currently little evidence to guide how management of subacute SDHs should differ from the management of acute or chronic SDHs [39–41].

Chronic Subdural Hematoma

SDHs that arise over the course of weeks to months are defined as chronic and manifest with a markedly different tempo of symptoms and management strategy [42]. The incidence of chronic SDHs is estimated at approximately 0.001–0.002 % per year, with a bias toward males and the elderly [43–45]. They commonly present in patients with

increased subdural spaces (such as from aging-related brain atrophy), and often there is a history of only minor head trauma or no recallable antecedent trauma [42, 46–48]. Presenting symptoms range from behavioral disturbances and headaches to focal seizures and unilateral focal deficits [45, 48]. On CT scan, chronic SDHs can be identified as hypo-attenuating masses that maintain the crescentic appearance of their acute counterparts.

The pathophysiology of chronic SDHs differs from that of acute hematomas, and it is important to keep in mind that normal SDH absorption relies on counter pressure from the brain parenchyma and the normal anatomic absence of a potential subdural space [47]. Thus, contributive factors in chronic SDHs include chronically lowered ICP from aggressive ventricular shunting (leading to engorgement and stretching of bridging veins), widened potential space between dura and underlying brain parenchyma (as can also be seen from aggressive ventricular shunting and can also occur with brain atrophy from aging, chronic alcoholism, or other causes), and a lack of underlying parenchymal swelling (as is seen in cases of relatively minor trauma) [46]. In the initial formation of the hematoma, minor trauma leads to disruption of venous structures, resulting in small amounts of subdural blood accumulation that cannot be absorbed in normal fashion.

Over time, chronic SDHs may enlarge [46]. The exact mechanisms for enlargement are not fully elucidated, but one hypothesis attributes continued bleeding to the formation of neomembranes from the inner layer of the dura to invest around the hematoma [49]. Neomembranes are triggered by cleavage of the dura-arachnoid space and consist of an inner and outer layer, with the latter being highly vascularized tissue that can contribute to repeated low-volume bleeding [50]. There is also reason to believe that this outer membrane secretes anti-thrombotic and fibrinolytic substances into the inner hematoma space, allowing the hematoma to remain liquefied and expand over time [51, 52].

Mortality associated with chronic SDHs is significantly lower than that of acute SDHs, with estimates ranging from less than 1–5 % in most studies [45, 53]. However, these numbers may underestimate the true morbidity burden of chronic SDHs, particularly in the elderly. One study of elderly chronic SDH patients (mean age 80.6 years) measured an initial 16.7 % in-hospital mortality rate but found that when followed after discharge, mortality rose to 26 % at 6 months and 32 % at 1 year post-hospitalization [54].

Acute on Chronic Subdural Hematomas

Acute on chronic SDHs occurs in the setting of known SDH that expands due to subsequent trauma. Clinically,

these patients often have a known or suspected chronic SDH (e.g., elderly patients with complaints of progressively worsening headaches and mental status) and then develop an acute worsening of their headache or decline in neurologic status. CT scan may reveal a lesion that features both acute (hyperdense) and chronic (hypodense) material in the subdural space. Limited literature on this population suggests that an acute component compounds as many as 8 % of chronic SDHs [55]. Similar to acute SDHs, acute on chronic SDHs require rapid treatment to avoid the potentially dangerous sequelae of an enlarging clot burden.

Bilateral Subdural Hematomas

Bilateral chronic SDHs are found in 10–25 % of patients with chronic SDHs and merit special mention for several reasons [47, 48, 56, 57]. Unlike unilateral lesions, bilateral chronic SDHs rarely present with hemiparesis or focal neurologic deficits and are less likely to produce midline shift [57, 58]. As such, the clinical diagnosis of these lesions is more difficult as symptoms may be nonspecific for intracranial masses (e.g., headache, progressive global mental decline). Moreover, as the brain parenchyma is compressed bilaterally, the direction of mass effect trends away from lateral shifts and subfalcine herniation and toward downward shifts and central herniation, a potentially fatal complication if not diagnosed and corrected in a timely fashion. Additionally, when encountered outside of a traumatic setting, bilateral SDHs should prompt suspicion of intracranial hypotension and precipitate investigations into a source of spontaneous CSF leak or overly aggressive CSF shunting. Symptoms and signs that augment suspicion for intracranial hypotension include positional headaches and apparent dural thickening on imaging. In such instances, repair of the CSF leak or reduction in the degree of CSF shunting may lead to spontaneous resolution of the subdural collection without need for more aggressive interventions.

Recurrent Subdural Hematomas

An estimated 10–25 % of patients with chronic SDHs will experience recurrence following evacuation [53, 59–63]. Many factors have been linked to recurrence, including age, anticoagulation usage, persistent post-operative midline shift, large original hematoma size, bilateral lesions, septated or mixed-density lesions, and presence of post-operative seizures [56, 59, 61, 63]. Although the reliability of any single factors to predict recurrence varies between studies, taken as a whole, they suggest that residual hematoma following treatment is an important causative factor in chronic SDH recurrence. They further indicate

that initial surgical evacuation may prove difficult in large lesions, septated lesions, and mixed-density lesions [64].

When present, recurrent SDHs are treated similarly to primary lesions, with repeat surgical evacuation a preferred option. Repeat surgical evacuation, even with the exact same methodology as the initial evacuation, is often effective and results in successful treatment in greater than 70 % of patients [53]. Continued recurrence may prompt placement of a subdural-peritoneal shunt for longer term lesion control.

Anticoagulation-Associated Subdural Hematomas

Systemic anticoagulation is a known risk for the development of both acute and chronic SDHs [65, 66]. In population studies, patients on oral anticoagulation therapy are estimated to have a 4 to 15-fold increased risk for SDH [67, 68]. Moreover, for patients on warfarin, an increase in prothrombin ratio from 2.0 to 2.5 has been measured to increase the risk of subdural hemorrhage by greater than sevenfold [69]. Limited evidence suggests that direct thrombin inhibitors may pose an equivalent risk of SDH formation compared with warfarin, in a dose-dependent manner [70]. Although direct thrombin inhibitors pose certain advantages over warfarin, caution must be exerted in the setting of intracranial hemorrhage and the neurocritical care population as no current methods exist to reverse the effects of direct thrombin inhibitors [71].

Although patients on anticoagulation represent a heterogeneous group, SDHs in the setting of anticoagulants are not only more frequent but also present in a more sudden and severe fashion. These lesions can expand rapidly, and unless anticoagulation is quickly reversed, can result in severe symptoms and death.

Secondary Subdural Hematomas

Rarely, SDHs are encountered secondary to a primary vascular, malignant, or other structural abnormalities. From a vascular perspective, SDHs have been reported secondary to dural arteriovenous fistulas, arteriovenous malformations, and aneurysmal rupture (particularly aneurysms arising from the posterior communicating artery) [72–76]. Instances of these are exceedingly rare and data are limited to case reports and small case series. In patients with no other risk factors, spontaneous SDH formation can be investigated for potential underlying vascular etiologies using CT angiogram and potentially invasive angiography. Acute, symptomatic lesions warrant immediate surgical attention and appropriate correction of the underlying lesion.

Malignant lesions have also been known to lead to SDH formation, with primary intrinsic tumors, meningeal

tumors, multicentric lymphomatous tumors, and dural metastases all having been implicated in SDH formation [77, 78]. The incidence of SDH in systemic cancer is low, with one study reporting only two incidents in a series of 2508 consecutive autopsy specimens [79]. The pathophysiology of these hematomas has been hypothesized to involve either direct hemorrhage from the tumor itself, or metastatic obstruction of intradural veins, leading to dilation and rupture of collateral capillaries on the inner layer of the dura [80, 81]. A diagnosis of malignancy-associated SDH should be suspected in patients with a known history of metastatic cancer and rapidly progressive focal neurologic deficits or decrease in mental status. Management should be based on the presence of compressive symptoms as well as the individual goals of care in the setting of metastatic disease.

Other structural brain abnormalities can also predispose patients to SDH formation. The most commonly encountered of these are arachnoid cysts, which are known to occur in approximately 2.4 % of chronic SDHs [82]. The exact mechanism for the association between arachnoid cysts and SDHs remains currently unclear and has been hypothesized to involve potential bleeding from disruption of the outer wall of the cyst [83, 84]. Management of arachnoid cyst-associated SDH also remains unclear, with authors reporting success with evacuation of either the hematoma alone or in conjunction with the associated arachnoid cyst [82, 84].

Cerebral Spinal Fluid Collections in the Subdural Space

Not all subdural fluid collections are filled with blood, however. There are a number of clinical circumstances in which there is accumulation of cerebral spinal fluid (CSF) in the subdural space. These circumstances include spontaneous accumulation in some patients, certain pediatric patients, and finally some patients who have undergone complicated intracranial operations. These extra-axial collections have been referred to as subdural hygromas, subdural fluid collections, external brain tamponade, or external hydrocephalus [85–87]. All of these descriptions have an implied etiological connection to hydrocephalus [88, 89]. In our evaluation, the more accurate association is with derangements in intracranial CSF dynamics, which at times does not reach the level of hydrocephalus. Given the focus of this manuscript, we will direct our attention to the presentation and management of those collections that present in the post-operative setting, typical of the neurocritical care setting. The vast majority of these collections follow decompressive craniectomy, although some do present outside of this setting.

The etiology of these extra-axial collections in the setting of decompressive craniectomy is difficult to ascertain.

One theory proposes that surgery violates the dural and bony tissue planes and creates the contiguity and communication among the normally distinct subgaleal, epidural, subdural spaces with the subarachnoid space needed for the formation of these collections. This derangement in cerebral hydro dynamics is only accentuated when the normal “closed vault” pressure provided by the intact skull is removed, making the accumulation of these collections more prominent in those patients who have received a craniectomy. Therefore “extra-axial” is the most appropriate anatomical description of the space where post-craniectomy fluid collections occur [89].

Malignant middle cerebral artery (MCA) infarction has been shown to respond favorably to decompressive craniectomy with significant supporting evidence from well-conducted prospective trials, including DECIMAL, DESTINY, and HAMLET [86, 90, 91]. Given this evidence, an increased number of patients are being treated with this modality. These and other studies have also demonstrated that extra-axial fluid collections are known consequences of decompressive hemicraniectomy, often requiring further management [92].

Decompressive craniectomy is also prominently utilized in other settings where uncontrolled ICP is not responsive to medical therapy, such as trauma, subarachnoid hemorrhage, and occasionally tumors associated with significant edema. Most prominent amongst these clinical settings is the application of decompressive craniectomy in trauma. The recent DECRA trial has renewed interest in this controversial surgery [93]. The trial demonstrated that after decompressive craniectomy, patients had decreased ICP and the length of stay in the Intensive Care Unit but increased unfavorable outcomes. Work published by the senior author demonstrates how these collections can, at times, behave in a very aggressive fashion causing mass effect, herniation, and significant worsening in patient clinical status [88, 89]. We described this entity as Craniectomy-Associated Progressive Extra-Axial Collections with Treated Hydrocephalus (CAPECTH; Fig. 3). It is clear that the benefits of the decompressive craniectomy in these settings will not be realized until we deal with the complications associated with the procedure, including the management of these extra-axial collections.

Management

Immediate Assessment and Nonoperative Interventions

Common to all acute presentations, the ABCs of critical care must be secured. This refers to securing the airway, controlling blood pressure, and circulation or coagulation.

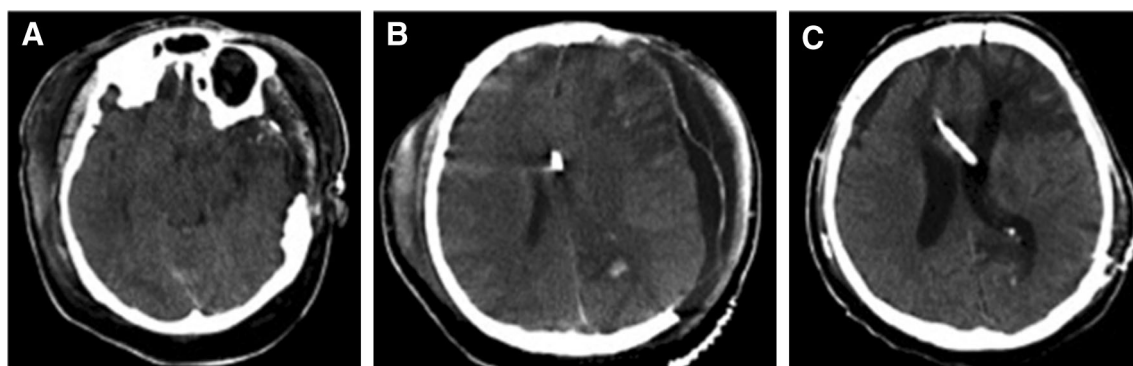


Fig. 3 A 50-year-old male with severe traumatic brain injury and hemorrhage who developed an extra-axial collection ipsilateral to the craniectomy. Axial noncontrast head CT scans showing **a** brainstem compression from mass effect; **b** a large collection slightly denser

than cerebrospinal fluid with significant mass effect; and **c** the collection resolved after cranioplasty and ventriculoperitoneal shunt placement

Although many of the concepts for this acute critical care medicine are similar for all patients, there are important differences that the practitioner must pay special attention to. In an attempt to increase awareness and education in this important topic, numerous collaborative efforts have been instituted, including the formation of the Emergency Neurological Life Support (ENLS) by the Neurocritical Care Society [94, 95].

Mechanical airway ventilation is critical to ensure that critical organs receive proper oxygenation. The central nervous system (CNS) is the most sensitive tissue to the effects of hypoxia, with irreversible damage beginning at 3 min when the PaO_2 drops below 30 mm Hg [96]. Another important role of adequate ventilation is to ensure proper removal of CO_2 , as it is known that hypercapnia may have deleterious results in the neurologically devastated patient, including vasodilation and resultant increases in ICP. The specifics of airway management are beyond the scope of this publication, but readers are guided to important literature on this topic [97]. It has been suggested that any patient with a Glasgow Coma Scale (GCS) of 8 or below should be intubated and that approximately 30 % of patients with intracerebral hemorrhage will require to be mechanically intubated [98]. Another advantage of mechanical ventilation is that allows for safer use of sedating medicines, which are helpful in the initial management of increased ICP by lowering patient agitation and decreasing brain tissue metabolic demands.

After a patient's airway has been secured, attention should be directed to controlling blood pressure to a limited extent. This is because although uncontrolled hypertension has been noted to have deleterious effects on the CNS, aggressive treatment may result in ischemic damage [99]. Similar to recent guidelines for patients with intracerebral hemorrhage, it has been suggested that lowering the

systolic blood pressures to around 140 mm Hg may be beneficial [100, 101].

As previously mentioned, reversal of anticoagulation is also important in the setting of anticoagulation-associated hematomas, and administration of fresh frozen plasma (FFP), platelets, protamine sulfate, and/or vitamin K can help stem the aggressive progression of these SDHs. Two classes of drugs that have garnered some attention in the reversal of anticoagulation are pro-thrombin complex concentrates (PCCs) and recombinant factor VIIa (rFVIIa). PCCs have been used to counteract warfarin and although results seem to be equivocal between using them and FFP, they have the added advantage of being low-volume, limiting the adverse effects of volume overload in these patients, which could include unwanted cerebral effects such as worsening cerebral edema, or more systemic effects such as heart failure [102]. Although there was much initial excitement about the use of rFVIIa in the reversal of anticoagulation-related intracerebral hemorrhages, further studies showed that rFVIIa did not generate thrombin as effectively as PCCs and furthermore caused more thromboembolic events [103]. Thus, for patients known to be on Coumadin that present with SDHs and the decision has been made for operative management, it is our practice to administer a PCC with a goal of an international normalized ratio (INR) of 1.4 or below, and this has been shown in small series to decrease the amount of time needed to correct the coagulopathy and time to surgical intervention [104]. It should be noted that the time of effect of PCCs is only a few hours and redosing may be necessary or supplementation with vitamin K. The use of newer oral anticoagulants such as thrombin inhibitors are becoming increasingly popular due to the fact that levels do not have to be continually monitored, but no specific antidotes are widely available in the US [107]. Clinicians should be

aware of these newer drugs and the timely consultation of hematologists is a common occurrence in the management of these patients.

Patients on antiplatelet therapy such as aspirin have an increased risk of bleeding and subsequent enlargement of their SDHs, and this risk is increased when they are on other antiplatelet agents such as Plavix. Platelet infusions for patients on antiplatelet therapy did not show any improvements in mortality or functional outcomes in patients with intracranial hemorrhage but is still widely practiced [105, 106]. Similarly, patients with low platelet counts such as those with myelodysplasia are particularly difficult patients to treat and current recommendations suggest that a platelet level of $100,000 \text{ mL}^{-1}$ is required for invasive neurosurgery, and the level should be maintained to $50,000 \text{ mL}^{-1}$ for 2 days after such invasive procedures [108]

It is only after securing the ABCs of critical care that it is safe to think about acquiring imaging to make a diagnosis. For its ability to quickly diagnose most intracerebral pathologies, a noncontrast CT is the initial imaging modality of choice in these instances. Although MRI may be helpful, especially in helping with subdurals that are related to other entities such as neoplasms (see section on secondary SDHs), the time needed for their acquisition makes it less favorable in the acute setting. Following the diagnosis of a SDH, a primary concern is ICP management. As originally formulated over two centuries ago, the Monro–Kellie hypothesis states that the intracranial space has a fixed volume comprised of brain parenchyma, CSF, and intracranial blood [109]. In the setting of a SDH, clot burden and potentially post-traumatic edema further reduce the available amount of intracranial space. After the buffering ability of CSF and cerebral blood flow auto-regulation are exhausted, cerebral ischemia and herniation become worrisome complications. As such, ICP management is an important pillar in the management of all SDH patients. As will be seen in the discussion below, there are important differences in the management of increased ICPs in patients with SDHs compared to those with traumatic brain injuries (TBIs) or other space-occupying lesions.

Unlike the trauma guidelines which suggest that the insertion of a fiberoptic ICP wire or external ventricular drain (EVD) may be helpful in the setting of a patient with GCS of 8 or below and an abnormal CT scan, the placement of such devices may be more detrimental for patients with SDHs, as the drainage of CSF may cause further retraction of draining veins and worsening of the SDH, similar to the subdural collections seen in patients with aggressive ventriculoperitoneal shunts. It is for these reasons that the use of hypertonic saline and mannitol—two therapies that are well documented to help with ICPs are

not useful in the context of SDHs (see below). By acting as another constituent in the cranial vault, the SDH has a direct role in increasing ICPs, and it is for this reason that operative management plays such a crucial role (see below).

Post-traumatic seizures are a potentially serious complication, especially within 7 days following the initial insult. SDHs are a known risk factor for early traumatic seizures in TBI patients, with as many as 24 % of patients with traumatic SDHs presenting with an early post-traumatic seizure [110]. Similarly, for acute or acute-on-chronic subdural patients, the rate of post-operative seizures has been reported to be as high as 25 % [111].

Patients with SDHs, particularly those with involvement of the temporal lobe, tentorium, and other epileptogenic regions, frequently require anti-seizure prophylaxis, given a previously reported association between initiation of pharmacologic prophylaxis and reduced incidence of early post-traumatic seizures [112, 113].

Although previously popular, phenytoin for post-traumatic seizure prophylaxis has been gradually supplanted by levetiracetam given its more favorable adverse effect profile [114]. Data to support this switch are somewhat lacking, but at least one retrospective study found that levetiracetam has a similar efficacy profile as phenytoin in preventing seizures in patients with acute or subacute SDHs, while being associated with a lower risk of adverse effects [115]. Equivalent efficacies may not persist for more severe lesions, however. For patients with midline shift noted on initial head CT, levetiracetam has been associated with a higher risk of electrographic seizures compared to phenytoin [115].

In chronic SDH patients, about 11 % develop seizures following surgery for hematoma evacuation, although this rate varies and has been reported to be as high as 19 % [42, 116]. The use of antiepileptic drugs for seizure prophylaxis in chronic SDH patients remains controversial.

To date, no controlled prospective study has studied the effects of timing, dosage, and type of antiepileptic medication on post-operative seizures in SDH patients. One study found that 2.4 % of patients who received prophylactic anticonvulsive medications developed seizures, as compared to 32 % of patients who did not receive therapeutic levels of prophylactic medication [116]. Another study found that, although preoperative antiepileptic drug prophylaxis was associated with a lower risk of post-operative seizures in chronic SDH patients treated with burr-hole drainage, the use of such medication did not affect discharge outcomes [117]. Given the strong correlation between post-operative seizures and mortality, as well as the significant increase in morbidity in patients with new seizures, there is a need for more controlled studies to

establish standard guidelines in managing post-operative seizures in the setting of SDH [116].

It is because of these complex issues and the association of acute SDHs and severe trauma that it is our practice to admit most symptomatic SDHs to our neurocritical intensive care unit (NICU) until operative management of the SDHs is arranged.

Operative Intervention in Acute Subdural Hematomas

The management of acute SDHs is influenced by the clinical course, radiologic findings, and patient prognosis. The latter consideration warrants special emphasis as, due to the association between acute SDHs and very severe trauma, an unfortunately high number of patients with acute SDHs are unsalvageable and thus are not surgical candidates. Current guidelines recommend surgical evacuation with open craniotomy in cases of acute SDH with maximal thickness of greater than or equal to 1 cm or greater than or equal to 0.5 cm of midline shift [1]. Lesions that do not meet the aforementioned criteria may still prompt operative evacuation if they have a GCS score of 8 or less and meet one of the following conditions: (1) a decrease in GCS score by 2 or more points from the time of injury to hospital presentation, (2) asymmetric or fixed and dilated pupils, or (3) patients with ICP greater than 20 mm Hg [1].

It is important to note that these guidelines are based on observational data, and no randomized controlled trials exist to guide surgical management of acute SDHs. Clot thickness and midline shift have both been linked to poor outcomes, though the exact numerical values at which morbidity and mortality begin to rise vary across studies [5, 11, 17, 20]. Likewise, clot thicknesses of greater than 1 cm and greater than 0.5 cm of midline shift have been associated with the failure of nonoperative therapy and the need for delayed surgery in multiple studies [3, 118, 119]. Thus, consistent evidence suggests that larger hematomas benefit from surgical evacuation. The evidence for surgical evacuation for smaller hematomas is more limited. In one prospective series of 65 patients, the 15 patients with ICP less than 20 mm Hg and with hematomas less than 5 mm of thickness were initially treated nonoperatively. Three of the 15 died from their injuries (two from progression of intracranial pathology, one unlisted) and two subsequently required surgical treatment. Decrease in GCS from time of injury was linked to failure of nonoperative management [3].

Despite advancements in diagnostic and therapeutic options over the years, acute traumatic subdural in adults remains a serious, life-threatening condition that is typically associated with underlying structural brain injury. In addition to prompt surgical evaluation and hematoma

evacuation when indicated, medical management of cerebral edema and ICP are equally important in maximizing patient outcomes after this serious injury. It is for this reason that many, if not all patients post-operatively are readmitted to the NICU after operative management. With the successful obliteration of the potential space of the SDH and the interval placement of subdural drains, more traditional methods of ICP management such as hypertonic therapy and placements of EVD are safer. It is also our practice to leave drains in the subdural space as well as in the subgaleal space for 1 or 2 days to help reduce the chance of recurrence, borrowing from the evidence of efficacy of drains in chronic subdural evacuations (see below).

Operative Intervention in Chronic Subdural Hematomas

Management of chronic SDHs is somewhat controversial, with debate on the optimal method of surgical evacuation. While reports of successful nonoperative management of chronic SDHs exist, most authors agree that the majority of chronic SDHs do require surgical drainage as their very existence suggests compromise in the body's ability to naturally reabsorb the hematoma [120]. Moreover, the natural history of these lesions is to frequently expand over time, leading to further cerebral compression [46]. In one randomized controlled trial, mannitol therapy was compared to surgical evacuation of chronic SDHs, the trial was terminated due to failure of mannitol alone in all nonoperative patients [121]. In an interesting study, a single institution hypothesized that hyperfibrinolysis plays a major role in liquefaction of chronic SDHs. Thus, they gave 21 patients an oral regimen of the antifibrinolytic drug, tranexamic acid. They showed over an 80 % reduction in the volume of chronic subdurals and no patients' subdurals increased in size [122]. Larger studies are needed to corroborate these results. No specific guidelines currently exist on the indications for surgery in chronic SDHs, all symptomatic lesions causing focal symptoms should be considered potential operative candidates.

Methods of surgical evacuation include open craniotomy (involving removal of a bone window in the operating room and direct hematoma drainage, irrigation, and/or neomembrane resection), burr-hole craniotomy (involving a large hole drilled into the cranium in the operating room, and subsequent dural puncture and irrigation), and twist-drill craniotomy (involving a smaller hole placed in the cranium, which can be performed at bedside, with subsequent placement of a drainage catheter). Any of these procedures can also be augmented with the placement of a subdural drain, to allow for continued post-

operative drainage of hematoma contents. To date, no class I evidence exists to compare the various ways of gaining access through the cranium [53, 123]. There is little evidence to suggest that excision of the hematoma membrane improves outcomes, making the use of large opened craniotomies for this purpose less frequent [95]. Moreover, it should be noted that evidence exists to support the use of subdural drains. In one randomized controlled trial of 269 patients undergoing burr-hole drainage, drain placement was associated with lower rates of hematoma recurrence and reduced mortality [124].

As the optimal method of chronic SDH evacuation remains an area of continued research, other patient-specific factors may heavily influence a clinician's approach to SDH drainage. In patients who are unstable for transport to the operating room or are poor candidates to undergo general anesthesia, twist-drill craniotomy may have significant advantages over more invasive methods.

Operative Intervention in Cerebral Spinal Fluid Collections in the Subdural Space

The treatment alternatives for management of CSF collections vary to a great extent on which clinical setting they accompany. Collections that accompany replacement of the bone flap tend to follow a much more benign course, as is the case with those that accompany decompressive procedures for MCA infarction. These same collections in the setting of trauma and subarachnoid hemorrhage can have a much more aggressive clinical course and therefore require a different approach.

In the setting of a benign evolution of these collections, it is often sufficient to manage with careful observation and close follow-up. This will avoid unnecessary surgical intervention while monitoring that they resolve spontaneously. In the intermediate setting of persistent collections that cause clinical alterations, the choices for management include temporary drainage of the collection itself, treatment of underlying hydrocephalus with a ventriculoperitoneal shunt, or draining of the subdural collection itself with a subdural to peritoneal shunt. In our own published experience with this intermediate group of patients following MCA infarction and decompression, we found that prompt cranioplasty, when permitted by the clinical situation, resolved these collections without additional interventions [89].

In the setting where these collections follow an aggressive course with mass effect and clinical deterioration, it is often the case where multiple management techniques are required, including local drainage, treatment of hydrocephalus with ventriculoperitoneal shunts, treatment of local collections with subdural-peritoneal shunts and rapid cranioplasty. For those patients in this

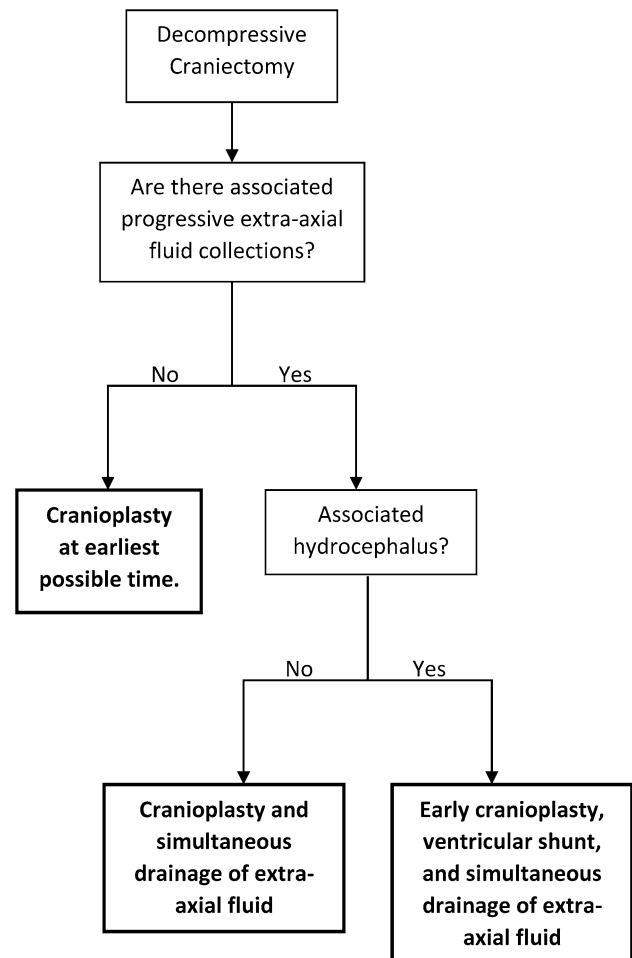


Fig. 4 A treatment algorithm for patients with Craniectomy-Associated Progressive Extra-Axial Collections with Treated Hydrocephalus (CAPECTH)

setting, we have developed a treatment algorithm (Fig. 4) [88].

Follow-Up

Following stabilization of patients with SDH, regular follow-up is an important step not to be missed. As previously mentioned, hematoma recurrence and progression are not uncommon, and follow-up with serial imaging is critical to ensure that recurrent hematoma formation is caught and addressed before it becomes symptomatic. An immediate post-operative CT scan can demonstrate the amount of remaining clot-burden left and serves as a baseline for future comparisons if a patient develops symptoms suspicious for SDH recurrence. Follow-up imaging within 1–2 weeks is also commonly pursued in otherwise asymptomatic patients to monitor for signs of progression or recurrence.

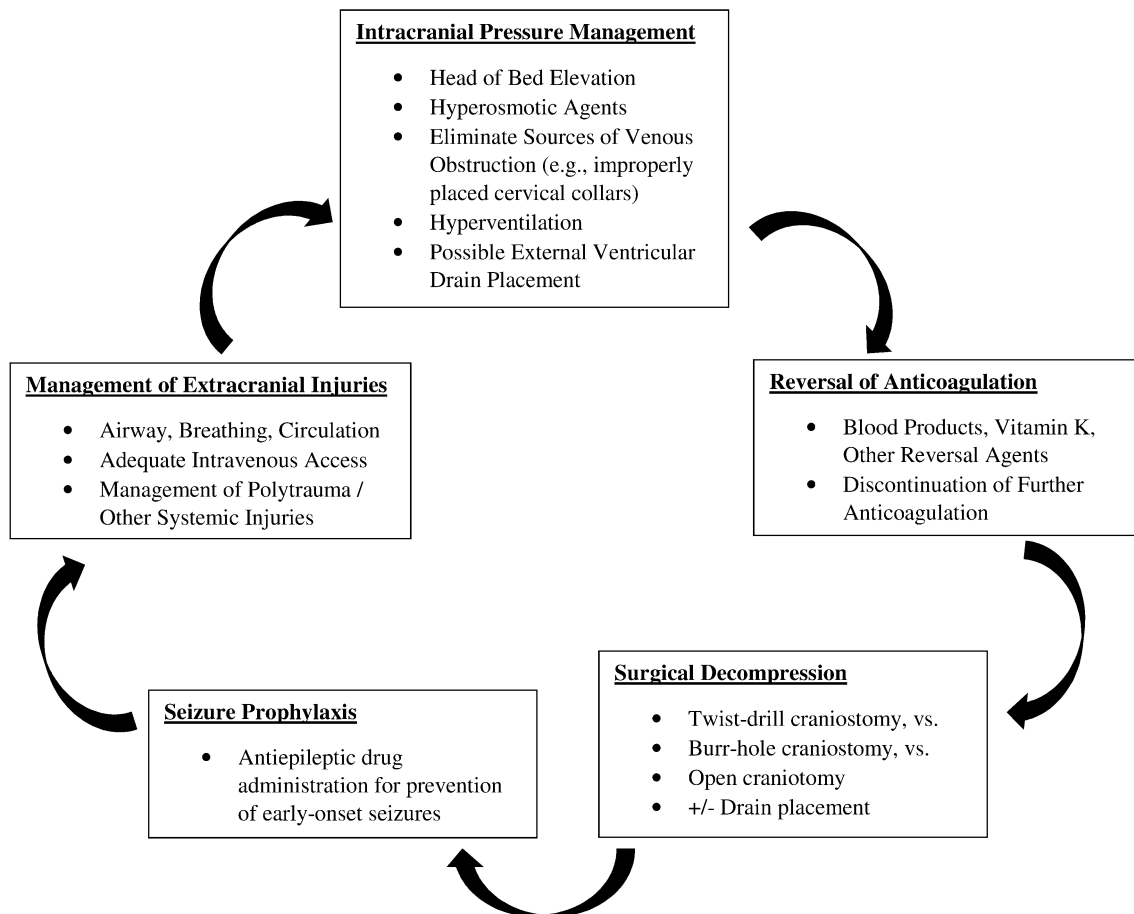


Fig. 5 Principles in SDH management

Conclusions

SDHs are a common finding in neurocritical care patients and represent heterogeneous pathologies with different etiologies, epidemiology, and management strategies. It is clear that a multi-faceted approach, including critical care and surgical management is needed for the optimal care of these often very sick patients (Fig. 5). Properly selected patients can benefit substantially from clot evacuation and alleviation of mass effect, but it is important not to overlook the importance of management of other concurrent injuries both intracranially and elsewhere in the body.

Compliance with Ethical Standards

Conflict of interest The authors declare that they have no conflict of interest.

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