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Natural History of Acute Subdural Hematoma

Rafael A. Vega, MD, PhD, Alex B. Valadka, MD*

KEYWORDS

- Acute subdural hematoma • Anticoagulation • Computed tomography • Glasgow coma scale • MRI
- Nonoperative management • Traumatic brain injury

KEY POINTS

- Although guidelines for surgical decision-making in patients with acute subdural hematomas (ASDHs) are widely available, the evidence supporting these guidelines is weak, and management of these patients must often be individualized.
- Smaller ASDHs in patients in good neurologic condition usually can be successfully managed without surgery.
- Large ASDHs with minimal mass effect in patients with minimal symptoms also may be considered for nonoperative management.
- The literature is divided about the effects of anticoagulant and antiplatelet medications on rapid growth of ASDHs and on their likelihood of progression to large chronic subdural hematomas, but it is reasonable to reverse the effects of these medications promptly.
- Close clinical and radiologic follow-up is needed in these patients, both acutely to detect rapid expansion of an ASDH, and subacutely to detect formation of a large subacute or chronic subdural hematoma.

INTRODUCTION

Few emergencies in neurosurgery are as worrisome as a large acute subdural hematoma (ASDH) (**Fig. 1**). Many of these lesions require immediate evacuation, regardless of the time of day or day of the week. Fortunately for the affected patients, the vast majority of ASDHs are relatively small, thus allowing initial management to consist of nonsurgical observation.

The decision that emergency surgery is not needed is soon followed by an obvious question: what will happen to the ASDH? The ideal scenario is that it will gradually resorb and the patient will be left with no deficits. But the worst-case scenario is rapid and unexpected enlargement of the hematoma, causing the patient to undergo neurologic

deterioration and immediate surgical evacuation. Over a more protracted course, another suboptimal outcome is progression of a small ASDH to a large chronic subdural hematoma (CSDH) (**Figs. 2 and 3**), which may enlarge to such an extent that surgical evacuation becomes necessary (**Fig. 4**).

Despite the frequency with which neurosurgeons have to make these decisions, and despite the likely increase in incidence of small but potentially worrisome ASDHs as the population ages, becomes more prone to falls, and consumes more antiplatelet and anticoagulant medications, relatively little work has been reported on the natural history of ASDHs. Most published reports are based on retrospective reviews of case series or of large public registries.

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Fig. 1. Representative CT images of a left convexity ASDH with no underlying skull fracture (*axial, coronal, and sagittal, from left to right, respectively*). There is a small amount of left-to-right midline shift with mass effect on the left lateral ventricle. The basilar cisterns are preserved, without signs of herniation.

DEMOGRAPHICS

Traumatic brain injury (TBI) is a significant cause of mortality and permanent disability across the globe. TBI represents a spectrum of disease processes that ranges from concussion to large intra-axial and extra-axial intracranial hematomas, including ASDH.

For patients with TBI in general, outcomes have improved somewhat over the past decades because of better organization of emergency medical systems, greater speed and quality of computed tomography (CT) scanning, and refinements in general critical care practices.

However, mortality and outcome in patients with ASDH have not seen as much improvement. Mortality has been reported to range from 50% to 90%¹⁻⁴ or even higher in some series that include patients receiving anticoagulant therapies.⁵ Associated intracranial and extracranial injuries are common and may contribute to

increased morbidity and mortality.⁶ Overall outcomes may worsen in coming years as the population ages.

SPECIFIC TYPES OF ACUTE SUBDURAL HEMATOMA

The natural history of a subdural hematoma (SDH) is influenced by whether it is traumatic or nontraumatic in origin (**Table 1**). A retrospective cohort study based on several statewide administrative claims databases analyzed more than 27,000 patients with conservatively managed SDH. This study included both traumatic SDH, identified by such International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) discharge codes as 852.2x or 852.3x, and non-traumatic SDH, coded as 432.1.⁷ Approximately 71% of all cases were traumatic SDHs, and 29% were nontraumatic. This latter group had higher rates of subsequent SDH-related hospitalization,

Fig. 2. Sequential change of CT density of an ASDH and its proposed mechanism. (*From Lee KS. Natural history of chronic subdural haematoma. [Review]. Brain Injury 2004;18: 354; with permission.*)

Fig. 3. Schematic representation of the origin and pathogenesis of SDHs and the relationship among the 3 traumatic subdural lesions. SDG, subdural hygroma. (From Lee KS. Natural history of chronic subdural haematoma. [Review]. Brain Injury 2004;18:356; with permission.)

surgery, and death. The overall readmission rate within 90 days for SDH was approximately 1 in 8.

Parafalcine and Tentorial Acute Subdural Hematoma

Certain subtypes of traumatic ASDH and certain patient characteristics taken together may portend a benign course. Howard and colleagues⁸ found that isolated thin parafalcine and tentorial ASDHs in younger patients with mild TBI (Glasgow Coma Scale [GCS] score 13–15) did not enlarge, even when patients were taking antiplatelet or anticoagulant agents, or both. They suggested that such patients could be managed on a standard medical/surgical ward and did not require observation in an intensive care unit or intermediate care unit. However, only 65 patients met inclusion criteria for their study, which represented 8% of all patients admitted to their Level I trauma center with an ICD-9 code for SDH after closed head injury.

Posterior Fossa Acute Subdural Hematoma

Although rare, traumatic posterior fossa ASDHs are often associated with poor outcome. In a retrospective analysis of their experience with 10 patients, Takeuchi and colleagues⁹ reported a 90% poor outcome rate and a 50% mortality rate in patients with posterior fossa ASDH. Half of their patients exhibited coagulopathy. Their review of the literature revealed that posterior fossa SDHs were associated with occipital impacts and fractures, low GCS score, additional intracranial lesions (especially supratentorial lesions and intracerebellar hematomas), a significant rate of lesion evolution within the first 2 postinjury days, and high rates of poor outcome and mortality. Similarly, Oliveira de Amorim and colleagues¹⁰ retrospectively identified 4 cases of traumatic posterior fossa ASDH from their own institution and added an additional 57 cases from the literature. More than half the patients had an initial GCS score below 8, and unfavorable outcomes were recorded in 63%.

Fig. 4. The left axial CT image illustrates a CSDH. The image on the right depicts the original ASDH (arrows) from which the chronic hematoma originated.

Table 1 Etiologies of acute subdural hematomas	
Type	Causes
Trauma	Motor vehicle collisions, falls, assaults, accidents
Coagulopathy or medical anticoagulation	Warfarin, heparin, hemophilia, liver disease, thrombocytopenia
Nontraumatic hemorrhage	Cerebral aneurysm, arteriovenous malformation, tumor (especially meningioma or dural metastasis)
Postsurgical	Craniotomy, CSF shunting
Intracranial hypotension	Lumbar puncture, lumbar CSF leak, lumboperitoneal shunt, spinal epidural anesthesia
Inflicted injury	Usually pediatric age group; also can be seen in the elderly
Spontaneous or unknown	Rare

Abbreviation: CSF, cerebrospinal fluid.

Nontraumatic Acute Subdural Hematoma

Numerous nontraumatic conditions may cause ASDH, either as a result of direct bleeding into the subdural space or as an extension of an intraparenchymal hematoma into the subdural space. These conditions include (1) ruptured intracranial aneurysm, (2) ruptured cortical artery, (3) hypertensive cerebral hemorrhage, (4) neoplasm, (5) hematologic disorder, (6) anticoagulant or thrombolytic therapy, (7) cerebral amyloid angiopathy, (8) dural arteriovenous fistula, and (9) acquired immune deficiency syndrome (Table 2).¹¹

INITIAL DECISION-MAKING

The decision about whether to pursue nonoperative management of a patient with ASDH or to proceed with surgery is guided by the clinical examination and by imaging findings. The bedside examination can be repeated as often as desired. However, repeating imaging at frequent intervals can be unduly burdensome. It also increases costs and adds to a patient’s cumulative radiation exposure. Many reports have demonstrated success in not obtaining follow-up CT scans in certain patients whose neurologic examinations remain at or near normal. This strategy is especially

well-suited for patients with very small traumatic hemorrhagic lesions and clinically mild injuries. Very thin, low-volume SDHs may fall into this category.

With larger ASDHs, concern about potential enlargement may prompt follow-up CT scanning roughly 4 to 6 hours after an initial scan. Significant expansion of an ASDH may lead to immediate surgery before the patient exhibits associated clinical findings. Less dramatic growth of the lesion may lead to plans for another scan several hours later to determine if the clot has stabilized, or if it has continued to enlarge and may thus require surgical evacuation. Deciding to take a patient for surgical evacuation of an ASDH because of lesion growth on imaging studies, rather than change in neurologic examination, may occur more commonly in elderly patients. Age-associated cerebral atrophy may allow such patients to harbor relatively large hematomas without initially demonstrating symptoms. But after 1 or 2 days, edema of compressed brain tissue and of the hematoma itself may lead to neurologic worsening even if the active bleeding that created the hematoma has ceased.

Antiplatelet and Anticoagulant Medications

A patient’s use of anticoagulants and/or newer antiplatelet medications would seem to be an obvious risk factor for enlargement of a small ASDH. However, some published reports fail to support this assumption or even provide data to refute it. The retrospective design of these studies and of their data collection may partially explain such findings. Aggressive reversal of these medications should be the neurosurgeon’s standard approach, modified as necessary by the particular circumstances of each individual case.

Ivascu and colleagues¹² reported that even an emergency department protocol for prompt physician evaluation of patients known to be taking anticoagulant medications does not improve outcomes without a concomitant protocol for aggressive therapeutic intervention. The generalizability of these results may be limited because types of intracranial hemorrhage other than ASDH also were included, and more importantly, many anticoagulants other than warfarin are now in widespread use. Reversal of these other agents is very difficult or even not possible in some cases. But the underlying message that speedy treatment is essential is one that should be remembered by emergency physicians and others who see these patients soon after trauma.

The influence of anticoagulant or antiplatelet medications in the transformation of a small ASDH into a CSDH that is large enough to require

Table 2
Nontraumatic causes of acute subdural hematomas

Type	Epidemiology	Brief Discussion
Ruptured intracranial aneurysm	2%–10% of ruptured aneurysms result in SDH. Typically women.	In most cases there is an associated subarachnoid hemorrhage. The location of the responsible aneurysm is most often the internal carotid artery or the anterior communicating artery.
Ruptured cortical artery	Spontaneous rupture occurs predominantly in men. Accounts for 80% of reported cases.	The location in all cases reported is typically a vessel at or near the Sylvian fissure, originating from branches of the middle cerebral artery. The anatomic arrangement that predisposes to spontaneous rupture includes a fragile right-angled artery or an artery “bridging” the subdural space.
Hypertensive intracerebral hemorrhage	Reported to occur in 6% of hypertensive hemorrhages and most often involves cerebellar hematomas.	SDH may result from extension of an intracerebral hypertensive hemorrhage rupturing through the cerebral cortex to involve the subdural space. Hypertension may also result in SDH by causing rupture of a cortical artery.
Neoplasm	Most common malignancies associated with SDH are leukemia and metastatic carcinoma.	A variety of neoplasms are associated with SDH. These include primary tumors of the brain and its coverings, hematologic malignancies, and metastatic tumors. In some cases, SDH may be the first symptoms of the tumor.
Hematological disorder	Typical age of the individual is <18 y, but may also occur in neonates and infants.	In most cases of disorders of the hematopoietic system, factor deficiency (inherited or acquired) is the most common type. Individuals with Factor VIII and IX deficiency have factor activity levels of 0–1 U/dL. Fatal nontraumatic SDH has been reported in an individual with lupus anticoagulant.
Other conditions	—	Nontraumatic SDH has been described in conditions that include therapy with anticoagulants, thrombolytic therapy, cerebral amyloid angiopathy, dural arteriovenous fistulas, and acquired immune deficiency syndrome.

Abbreviation: SDH, subdural hematoma.

surgical drainage is unclear. It is possible that any such effect varies both with the specific medication and/or also with the dosage. Laviv and Rappaport¹³ retrospectively analyzed their experience with conservative management of patients with ASDH. They reported that, of 21 patients taking anticoagulants, antiaggregants, or clopidogrel, 20 (95%) later underwent surgical drainage of a CSDH. Overall, the group that developed surgically treated CSDHs had thicker initial hematomas than the nonoperated group.¹³

NONOPERATIVE MANAGEMENT

Significant medical comorbidities are common in elderly patients and are often of sufficient severity to preclude an intervention as aggressive as a craniotomy. The cerebral atrophy that accompanies normal aging offers some protection from ASDH by creating extra intracranial volume into which an acute hematoma can expand, thus allowing nonoperative management to be successful in many cases.

The greatest risk of nonoperative management is the potential for neurologic deterioration. This risk, and the likelihood of subsequent permanent neurologic impairment, must be considered in the context of the risks of surgical intervention. There appears to be little acute risk of other complications of nonoperative management of these patients. Seizures have not been reported to occur more commonly in patients with an unevacuated ASDH. The same is true regarding ischemic injury to compressed underlying brain tissue.

A related question is the setting in which such patients should be observed. There are at least 2 factors that must be considered.

- The first is early detection of neurologic deterioration. Because of the need to detect such an event quickly and then to initiate immediate action, many patients are better off in an intensive care unit or similar setting in which they can be assessed frequently by trained examiners.
- The other factor is prompt access to neurosurgical intervention. Securing prompt access does not necessarily require that all such patients must be physically transferred to a hospital with around-the-clock access to a neurosurgical operating room. If foolproof mechanisms have been created for immediate transfer of worsening patients to a neurosurgical facility, then observation at the original hospital may be reasonable in many cases because relatively few such patients will suffer neurologic deterioration.

Progression to Chronic Subdural Hematoma After Conservative Management

Occasionally, patients with large ASDHs who would normally be taken for surgical evacuation may be too frail or may have such significant underlying comorbidities that subjecting them to general anesthesia and craniotomy is not a viable option, even if they display unilateral weakness or other signs of mass effect. Allowing several days to pass so that the solid acute hematoma undergoes significant liquefaction as it progresses to a subacute hematoma may be the best approach in some of these cases because the liquefied hematoma often can be drained through a bur hole or other minimally invasive approach under local anesthesia.

In a retrospectively analyzed series of 177 patients, Lee and colleagues¹⁴ compared 16 patients (9%) in whom nonoperatively managed ASDH progressed to CSDH requiring surgical evacuation with 161 patients in whom ASDH resolved with conservative management. These investigators found that older age and larger hematoma size were associated with progression of ASDH to symptomatic CSDH. They also found a slightly higher hematoma density as measured in Hounsfield units. They did not find a correlation with use of anticoagulant or antiplatelet agents, but the numbers of such patients were small, and abnormal clotting parameters were treated with blood products, thus obscuring any potential effect of these medications.

In another series of 27 patients with traumatic ASDH who were managed nonoperatively, 1 (4%) deteriorated and required craniotomy.¹⁵ Four patients (15%) demonstrated evolution into CSDH and underwent bur hole drainage 15 to 21 days after injury. The remaining 22 patients (81%) required no additional treatment. No patients developed seizures. Mean duration of follow-up was 6 weeks.

Spontaneous Resolution of Acute Subdural Hematoma

Case reports stretching back over decades have documented apparent resolution of ASDHs in patients with obvious neurologic compromise from the hematomas, including coma, asymmetric motor findings, and unilateral pupillary dilatation. Concurrent injuries, underlying medical comorbidities, and/or family refusal to consent to surgery represent reasons why emergency craniotomy was not performed.

Wen and colleagues¹⁶ described their experience with such a patient and identified 19 additional cases in the literature. They reported that

the following features seem to characterize most of these cases: (1) coma less than 12 hours in duration after the initial injury; (2) absence of cerebral contusion; (3) thin width and wide distribution of the hematoma; (4) presence of a low-density band between the hematoma and the inner table of the skull on initial CT scan; and (5) lack of severe initial TBI, with initial GCS score greater than 8.

Fujimoto and colleagues¹⁷ retrospectively reviewed 56 patients with ASDH with midline shift of more than 10 mm and clot thickness larger than 10 mm who did not undergo immediate surgery, most commonly because of advanced age, poor general condition, or family wishes, or because patients did not exhibit significant neurologic symptoms. Rapid spontaneous resolution of the ASDH was defined as neurologic improvement within 24 hours and decrease of hematoma thickness by more than 5 mm within 96 hours. Eighteen patients (32%) demonstrated rapid spontaneous resolution. Multivariate analysis found that preinjury use of antiplatelet agents and presence of a low-density band between the hematoma and the inner wall of the skull on initial CT scan were independent predictive factors for rapid spontaneous resolution.

Commonly proposed explanations for rapid decrease in size and mass effect of an ASDH include a tear in the arachnoid that permits cerebrospinal fluid to wash away the acute blood, and/or redistribution rather than true resorption of the acute blood, which may be facilitated by a decrease in intracranial pressure after treating physicians' medical interventions. Interestingly, however, others have reported that decreasing brain compliance by adjusting upward the valve setting of an already-present ventriculoperitoneal shunt was associated with disappearance of an ASDH within 6 days.¹⁸ Of course, the very fact that these cases are unusual enough to merit publication demonstrates how rare it is for a sizable ASDH to resolve rapidly. Immediate surgery is the recommended treatment for these patients.

OUTCOMES WITH NONOPERATIVE MANAGEMENT

Acute deterioration has been recognized for decades as a potential outcome of nonoperative management of patients with acute traumatic intracranial hemorrhage. The terms "talk and deteriorate" or "talk and die" vividly describe this phenomenon. In 1987, Rockswold and colleagues¹⁹ reported on 33 of 215 patients with severe TBI who exhibited such deterioration. Twenty-five (76%) of these 33 patients underwent surgery, and of those 25, 14 had ASDHs. More aggressive

use of early follow-up CT scanning in recent years seems to have lowered the rate of such events, but they still occur with sufficient frequency that virtually every neurosurgeon has personal experience with this type of patient.

In a 1994 review at their Level I trauma center of ASDH management in 83 patients with GCS score 11 to 15, Croce and colleagues²⁰ found that nonoperatively managed patients had fewer focal neurologic deficits, smaller hematomas, and less cisternal effacement. Outcomes did not differ between groups. Of the 58 patients managed non-surgically, 6% later required surgery for CSDH.

Bullock and colleagues attempted nonoperative management in 23 conscious patients (3%) of 837 with traumatic ASDH.² In a retrospective analysis, they report that 6 (26%) of the 23 later underwent burr hole drainage of their hematomas at a mean of 15 days after injury. The ASDHs in the 6 operated patients were significantly larger than in the other 17 patients. The investigators proposed the following criteria for attempting nonoperative management of patients with traumatic ASDH: (1) GCS score greater than or equal to 13; (2) absence of associated intraparenchymal hematomas, contusions, or edema; (3) midline shift less than 10 mm; and (4) absence of basal cisternal effacement.

Ellenbogen and colleagues reviewed outcomes in 1427 patients with ASDH referred to their Level I trauma center. Such a population would be expected to have an overall greater severity of injury than that seen at most hospitals. GCS score exceeded 12 in only 58% of patients. Mean age was 58 years. A total of 248 (17%) patients underwent surgical evacuation of their SDHs. Mean length of stay was 9.6 days, and 40% spent 2 or more days in the intensive care unit. Overall inpatient mortality was 16% and was essentially the same in operated and nonoperated groups. Medical complications occurred in 28% of the overall series and most commonly consisted of pneumonia, urinary tract infection, or acute respiratory distress syndrome. Complications did not differ between operated and nonoperated patients. At discharge, 94% of patients had a GCS score of more than 12. The investigators note that their mortality rate was lower than that reported in other studies of traumatic ASDH and that their rates of deep venous thrombosis and pulmonary embolism were lower than expected. They suggest that earlier diagnosis of ASDH and improvement in care from regionalization of emergency medical systems may explain their findings.²¹

Kim and colleagues²² described 98 patients with mild TBI (GCS score 13–15) and ASDH in whom outcome was assessed by enlargement of the SDH on subsequent imaging and by the need

for subsequent surgical evacuation. Roughly two-thirds of these patients demonstrated regression of the SDH. The other 35% underwent delayed surgical evacuation at a median of 17 days after injury. Larger hematoma volume and midline shift were associated with the need for delayed hematoma evacuation. Interestingly, subarachnoid hemorrhage and cerebral contusions were more common in the nonoperative group. Reasons for the higher failure rate of conservative management in this series as compared with those in other reports are unclear and may relate to a lower threshold for taking patients to the operating room for delayed evacuation of an SDH.

Feliciano and colleagues²³ reported outcomes of nonoperated ASDH in a series of 38 patients, roughly half of whom were younger than 65 years. In 87% of these patients, midline shift was 5 mm or less, and hematoma thickness was 10 mm or less. Six patients in this group were taking antiplatelet or anticoagulant medications, and some were treated with blood products. Use of these medications was not associated with outcome in this subgroup with smaller hematomas. None of these patients required surgery. In the other 5 patients with hematoma thickness of more than 10 mm or shift greater than 5 mm, surgery was not performed because of excessive cardiac risk or withholding of consent by relatives. One patient in this group required surgery for neurologic deterioration.

Bajsarowicz and colleagues²⁴ retrospectively reviewed 646 patients with traumatic ASDH who were initially treated conservatively. Forty-two patients eventually required delayed surgical intervention after a median of 9.5 days, most commonly for symptomatic enlargement of the hematoma (median 14 days) or development of increased intracranial pressure (median 3.5 days). Factors associated with deterioration were thicker SDH, greater midline shift, location at the cerebral convexity, history of falls, and alcohol abuse.

SUMMARY

Published literature and widespread clinical experience make it clear that most ASDHs can be managed nonoperatively. This is especially true for smaller lesions in patients with good neurologic status. Even in patients who present with severe neurologic deficits or coma, the benefit of evacuating small ASDHs is often unclear. These ASDHs usually resolve spontaneously.

Large ASDHs may require a different approach. When a comatose patient is found to have a large ASDH associated with compression of the basal cisterns, significant midline shift, or other signs of

mass effect, immediate surgery is the default management plan.

More problematic is the management of large ASDHs in patients with no or minimal symptoms. Conservative management may be considered in patients in good neurologic status and, despite the size of the ASDH, relatively little underlying mass effect.

The effect of anticoagulant and antiplatelet medications on the natural history of ASDH is not clear from the literature. Prudence would suggest that reversal of such agents be considered in all patients with ASDH. Likewise, a lower threshold for early repeat CT scanning may be appropriate in patients with ASDH who are taking such medications.

Even if nonoperative management is successful in the acute phase, the risk remains for subsequent progression of an ASDH into a CSDH that is large enough to require surgery. Again, the literature is unclear on the effects of anticoagulants or antihypertensives on such a progression. Scheduled CT scanning 1 to 2 weeks after development of an ASDH may detect this process before clinical symptoms appear.

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