Neurocritical Care of Acute Subdural Hemorrhage

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KEYWORDS

• Acute subdural hematoma • Acute subdural hemorrhage • Traumatic brain injury
• Advanced Trauma Life Support protocol

KEY POINTS

- Although urgent surgical hematoma evacuation is necessary for most patients with SDH, well-orchestrated, evidenced-based, multidisciplinary, postoperative critical care is essential to achieve the best possible outcome.
- Acute SDH complicates approximately 11% of mild to moderate traumatic brain injuries (TBIs) that require hospitalization, and approximately 20% of severe TBIs.
- In most cases, acute SDH is related to a clear traumatic event, but in some cases, acute SDH can occur spontaneously.
- Management of SDH in the setting of TBI typically conforms to the Advanced Trauma Life Support (ATLS) protocol with airway taking priority, and management breathing and circulation occurring in parallel rather than in sequence.

INTRODUCTION

Acute subdural hematoma (SDH) develops between the dura and arachnoid membranes, usually due to tearing of the bridging veins that extend from the surface of the brain to the dural sinuses. Most cases of SDH result from low-pressure venous bleeding that eventually arrests due to rising intracranial pressure (ICP) and clot tamponade, but it is estimated that up to 20% to 30% of cases can result from arterial rupture.[1–3] Linear translation of acceleration across the diameter of the skull in the lateral direction can produce stretch or torque injury to veins or arteries, resulting in SDH.[4]

Acute SDH complicates approximately 11% of mild to moderate traumatic brain injuries (TBIs) that require hospitalization, and approximately 20% of severe TBIs.[5–8] In most cases, acute SDH is related to a clear traumatic event, but in some cases acute SDH can occur spontaneously. Important causes of spontaneous SDH include use of anticoagulants or antiplatelet agents, and rupture of an intracranial aneurysm that is adherent to the arachnoid membrane, with consequent arterial bleeding into the subdural space (with or without associated subarachnoid hemorrhage).[9–11] Less commonly, SDH also can result from bleeding due to arteriovenous malformations, vascular meningiomas, dural metastases, and spontaneous intracranial hypotension.[12–14] Identification of a treatable cause of nontraumatic SDH is crucial, because reversal of anticoagulation may be all that is necessary in some patients, whereas others may require treatment of a cerebral aneurysm.

After traumatic acute SDH, coma is present at the onset of injury in 25% to 50% of cases,

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whereas another 12% to 38% experience progressive neurologic decline to coma over the next several hours.[15,16] Coma is primarily the result of brain shifting and distortion, followed by elevated ICP and low cerebral perfusion pressure (CPP).[17] Large acute SDH leads to compression and ischemia of the underlying brain within 4 to 6 hours in the absence of emergency evacuation.[18] Posterior fossa SDH is especially treacherous because it can precipitate upward or downward herniation extremely rapidly.[19]

EMERGENCY RESUSCITATION

Airway, Breathing, Circulation

The objectives in the emergency department are to undertake a rapid and systematic clinical assessment, and to institute immediate lifesaving treatment. Management of SDH in the setting of TBI typically conforms to the Advanced Trauma Life Support (ATLS) protocol, with airway taking priority, and management breathing and circulation occurring in parallel rather than in sequence.[20] As is the case with all forms of severe trauma, maintenance of oxygenation (Pa_{O_2} >60 mm Hg) and blood pressure (BP) at a mean arterial pressure (MAP) of 65 mm Hg or higher is the immediate priority.[21]

Deciding on the optimal timing to secure the airway can be challenging. Intubation and ventilation may allow for expedited imaging, as well as safer interventions in the unconscious or agitated patient.[22] Intubations in the setting of TBI and elevated ICP are more challenging, with higher rates of failure and complications.[23] As a general rule, rapid sequence intubation (RSI) after an initial period of preoxygenation is the preferred strategy.[24–26] RSI is the concomitant administration of a sedative and a neuromuscular blocking agent to render a patient rapidly unconscious and flaccid so as to facilitate emergent endotracheal intubation and to minimize the risk of aspiration. Multiple studies confirm the high first-pass success rate of RSI, which in experienced hands can exceed 90%.[25,26]

Induction agents provide amnesia and blunt sympathetic responses, and can improve intubating conditions. Pretreatment with intravenous (IV) lidocaine 1.5 mg/kg 3 minutes before intubation may minimize any increase in ICP that can be associated with airway manipulation. Ketamine 1.5 mg/kg IV or etomidate 0.3 mg/kg are suitable induction agents because they tend to maintain MAP and do not increase ICP. Ketamine is a dissociative anesthetic agent that provides sedation and analgesia. Ketamine preserves respiratory drive and has both a quick onset of action and analgesic properties.[27] Ketamine can cause sympathetic stimulation, and although it is known to maintain BP, there is concern that it may cause ICP elevation in TBI.[28,29] Etomidate is a sedative-hypnotic agent that is frequently used for RSI that has putative neuroprotective properties.[30] It provides no analgesic effect, so it often is given with fentanyl 1.5 μg/kg, administered during the pretreatment phase of RSI. For paralysis, a short-acting paralytic, such as succinylcholine 1.5 mg/kg IV, is ideal, because it wears off quickly and restores the neurologic examination within 10 to 15 minutes (use with caution in the setting of crush injury or hyperkalemia).

Longer-acting nondepolarizing alternatives include rocuronium (0.6–1.2 mg/kg) or cisatracurium (0.15 mg/kg).

Imaging

Neuroimaging in the form of a noncontrast computed tomography (CT) is integral to the diagnosis of acute SDH due to it widespread availability and speed. The sensitivity now approaches 100% with newer generation scanners.[31] Brain MRI is more sensitive than head CT for the detection of extremely thin SDH, and tentorial and interhemispheric SDH.[31] Acute SDH can be classified by (1) the age of the imaged blood (eg, acute, acute-on-subacute, or acute-on-chronic), and (2) by the maximal thickness of the subdural collection in centimeters (**Fig. 1**). Additional parameters to assess are the extent of midline shift, the presence of effacement of the basal cisterns, and the extent of trapping of the contralateral ventricle. Because SDH can result from a ruptured cerebral aneurysm, noninvasive or digital subtraction angiography should be performed in suspicious cases of nontraumatic acute SDH.

Reversal of Anticoagulation

Reversal of all forms of anticoagulation is a medical emergency in patients with acute SDH. Coagulation panels are typically obtained on arrival; whether or not the patient is on some form of anticoagulation or antiplatelet therapy should be discerned through the history. The Neurocritical Care Society has published the most up-to-date guidelines for the emergency reversal of anticoagulation.[32] Patients on oral anticoagulation therapy are estimated to have a 4- to 15-fold increased risk for SDH, leading to higher likelihood of hematoma expansion, an increased risk of death, and a worse functional outcome unless anticoagulation is quickly reversed.[33,34] Patients on vitamin K antagonists, such as warfarin, are optimally reversed with vitamin K 10 mg