Chapter 2
Cranial Acute and Subacute Subdural Hematomas

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2.1 Introduction

Head injuries are a common mode of presentation to neurosurgical departments. Major changes in medical practice have been observed over the last decades [11]. In the 1990s, the advancements in modern diagnostic methods and medical technologies [15, 35] have intensely altered the management of patients with head trauma [14], and an improved understanding of the pathophysiologic events in cerebrovascular diseases has also occurred [6, 28, 39]. A better understanding of the pathophysiology of events after acute subdural hematoma (aSDH) has led to better patient outcomes. Currently, trauma is still a major public health problem [7] with high morbidity and mortality rates [16]. The frequency of aSDH is 11–20% in patients with head injury [31], but it occurs in about one-third of patients with severe traumatic brain injuries [3], and its mortality rate ranges between 50 and 90% [36]. There is no emergency in neurosurgical practice as worrisome as a large aSDH [36]. This review aims to assess the current knowledge of acute and subacute SDH.

2.2 Acute Subdural Hematoma

Blunt cranial traumas are commonly seen in every community and can be seen in any age group [27]. The cranium is a critical surgical region because of its content [17]. After a cranial trauma, impairments in physical, cognitive, psychological, and behavioral functioning and early complications can be seen [20] such as acute

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subdural hemorrhage which refers to the accumulation of fresh hematoma between the dura and arachnoid membranes, usually due to tearing of the bridging cortical veins [2].

2.3 Subacute Subdural Hematoma

Delayed deterioration within 24 h after trauma may be observed in some patients with aSDH. A conversion process from aSDH to subacute subdural hematoma (sASDH) may occur [33]. Hematomas usually begin to liquefy by 2 weeks after formation [12]. In these patients, aSDH is initially absorbed and the volume is reduced, but later, there can be an increase in the mass effect of the subarachnoid hemorrhage (SAH), the density of the hematoma may decrease, and the midline shift may increase on computed tomography (CT) [33]. New neurologic deficits can be seen in patients with subacute SDH.

2.4 Pathophysiology

Atrophic brain volume and the large subdural space of elderly patients constitute the enlarged subdural space [19]. In these patients, the enlarged subdural space may compensate for the increase in hematoma volume and cerebral edema before neurological deterioration occurs [32]. The timely detection of neurological deterioration is an important issue in neurosurgical practice [5]. For that reason close neurological [23] and radiological observation should be performed in a patient with head trauma [21].

The cerebrum is one of the most important organs of the human body [8] and is located in the cranium. This structure controls all of the central nervous system [8]. The volume of the cranium cannot be changed. Inside of cranium, the sum of the volume of the brain, cerebrospinal fluid, and intracranial blood is constant [26]. This principle is known as the Monro-Kellie doctrine or hypothesis and was defined in 1783 by Monro and Kellie. In a normal cranium without an aSDH, there is an equilibrium inside of the cranium. If the volume of one of the components within the cranium changes following a SDH, an increase in intracranial pressure (ICP), the onset of coma and herniation may occur. The normal level for intracranial pressure is 5–15 mmHg. Coma may be seen at the onset of severe head injury in 25–50% of cases [2]. Coma and high rate mortality from an aSDH may depend on many factors such as the Glasgow Coma Score/Scale (GCS) at the time of presentation, the degree of mass effect of the SDH, and extent of the midline shift, and the presence of increased ICP and cerebral edema. Increased ICP (above 20 mmHg) may lead to poor neurological outcome [32]. Measurement of the midline shift and the ICP has been used in assessing the severity of the SDH. The hematoma volume is an important issue when located in the posterior fossa [38]. The blood-brain barrier

is necessary for normal brain function [4, 14]. The disruption of this barrier may occur following acute and subacute SDH. Cerebral perfusion pressure (CPP) can also be altered in patients with acute SDH. CPP is important for delivery of oxygen to cerebral tissue and it is affected by cerebral blood flow which may be decreased immediately after a SDH [22].

The GCS was originally devised for patients with head trauma to evaluate impaired consciousness or coma [18]. This scale has become the worldwide standard for the assessment of the patient with a head injury. The GCS has several disadvantages such as the limited verbal reaction of intubated patients [23] with aSDH. Both parasympathetic and sympathetic nervous system disorders appear to be important factors in pupillary diameter changes [5, 29]. Pupillary changes can occur in patients with SDH from uncal herniation due to mass effect leading to compression of the oculomotor nerve and the brainstem [24]. For that reason, measuring and comparing pupil diameter by testing the reactivity to light can help diagnose oculomotor nerve injury after an aSDH, but pupillary changes can also occur due to direct orbital/ocular trauma [24].

2.5 Imaging

A non-contrast CT scan is an important radiological modality in the diagnosis of aSDH. Brain magnetic resonance imaging (MRI) can be preferred in cases with a thin aSDH, and tentorial and interhemispheric aSDH. Cerebral MRI is more sensitive than head CT for hematoma detection in these cases with a thin aSDH [2]. In an aSDH, a crescent-like or "half-moon" appearance that crosses cranial suture lines may be seen [12]. Later, delayed hematoma expansion may also be detected [32].

2.6 Management

Management of SDH is still a controversial issue because evidence-based guidelines and randomized controlled trials are lacking [31]. Decisions regarding surgery are based on SDH location, size, mass effect, midline shift, acuity, patient age, medical comorbidities, and the extent of neurological deficits [31]. Some factors such as age, comorbidities, and SDH evacuation have been identified as predictors of clinical outcome [31].

2.7 Role of Surgery

Primary or secondary brain injury may occur from an aSDH [27]. The aim of the surgical approach is to resolve the cerebral herniation in patients and to reduce secondary ischemic injury, minimally [22]. In some cases, the apparent resolution

of sASDHs may be seen. Because of the mass effect on the brain of a thick clot in an aSDH, decompressive craniotomy and hematoma evacuation are useful procedures. However, in the recent decades, decompressive craniectomy has been performed as an alternative surgical procedure to decompressive craniotomy [30]. Generally, the neurologic status of the patient with an aSDH on initial presentation, the hemorrhage size and its associated midline shift, and the presence of cerebral edema on CT are important factors that influence the surgical type of procedure such as a decompressive craniotomy or craniectomy [1]. The theoretical advantage of decompressive craniectomy is the ability to have more effective control of the increased ICP, and to improve in cerebral perfusion pressure and brain partial pressure of oxygen [1]. Continuous oxygen delivery and CO_2 clearance are paramount for the maintenance of normal brain function and tissue integrity [14]. The timing of surgery has often been regarded as an important factor for the clinical outcome of the patient with aSDH [34]. Surgical indications are an aSDH with >10 mm thickness or >5 mm midline shift, a deteriorating patient with the GCS <8, unilateral or bilateral fixed dilated pupils, or evidence of elevated increased ICP >20 [9]. Out of these parameters, mass effect is a significant indication for surgical intervention regardless of patient GCS [25]. Figure 2.1a shows the CT images of a patient with an aSDH; his hematoma thickness is 12.88 mm, and the patient also has

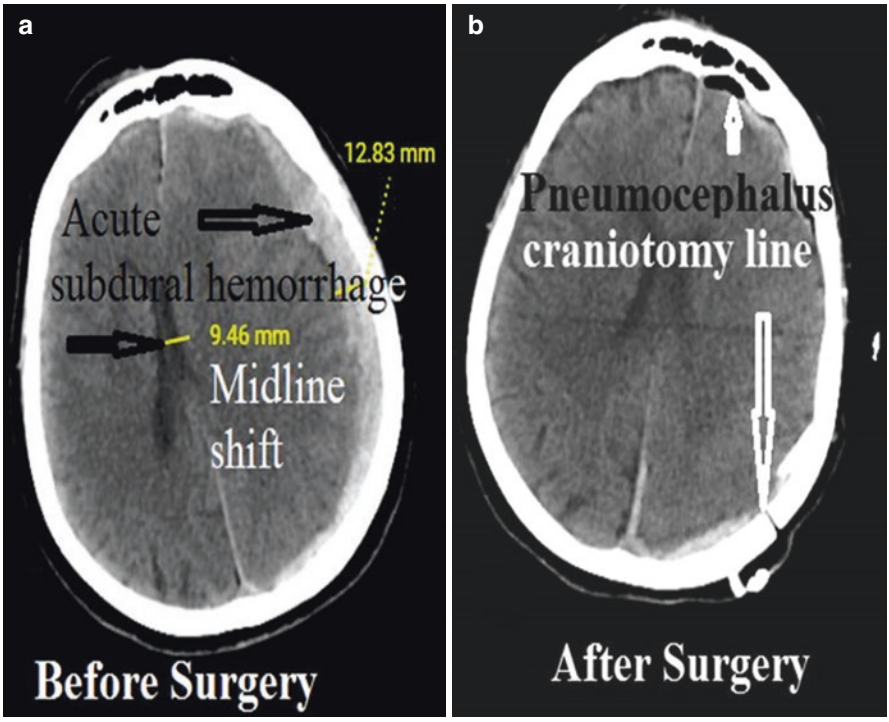

Fig. 2.1 (**a**) Shows the computed tomography images of a patient with acute subdural hematoma. His hematoma thickness is 12.88 mm, and the patient also has a 9.46 mm midline shift. (**b**) Shows the resolution of the midline shift after surgery

9.46 mm midline shift. Figure 2.1b shows the resolution of the midline shift after surgery.

Nonoperative management can be preferred in patients with thin aSDHs (clot thickness <10 mm) without significant mass effect (midline shift <5 mm) and minimal to no neurological deficits [25].

Seizures are a serious complication in patients with SDH [37]. The prevalence of seizures in SDHs is reported to be 24% in acute SDHs and 11% in chronic SDHs [37]. The use of anti-epileptic drugs for sASDH patients is a controversial issue [32].

2.8 Conclusion

The morbidity and mortality of patients following acute SDH are still high. In patients with decreased consciousness, and those with unilateral neurologic deficits (e.g., dilated pupil, motor weakness, or posturing) following a severe head injury, the presence of an acute SDH should be suspected [12]. Close clinical and radiologic follow-up is necessary to detect the rapid expansion of an aSDH. Given the important morbidity and mortality after an aSDH, it is necessary to correctly assess the damage to the human brain, which is difficult to perform on live human patients with aSDH, due to ethical issues. There are reasons to focus on experimental studies [28]. To understand the biomechanical, molecular, and cellular effects of traumatic brain injury, several injury models have been used in experimental studies [27]. The experimental studies may provide a better understanding of the effects of acute and subacute SDH than those studies of human subjects. If we consider the nervous system as a great orchestra that can express a complete range of rhythms and melodies and the most complex harmonic combinations [10], we will find it easier to understand how any traumatic acute subdural hematoma may be translated into an alteration of the rhythmic systems that synchronize the brain after disruption of the blood-brain barrier and a change in cerebral perfusion pressure. To examine the outcomes of a study, it is necessary to have testable hypotheses [13]. Outcomes are then expressed with respect to the implied goals [13]. The outcome of aSDHs has been dismal because of combined diffuse axonal injury and the accompanying increased ICP [32]. Well-orchestrated, evidenced-based, multidisciplinary studies are needed to achieve the best outcome following aSDH.

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