Chapter 19
Intracranial Hypotension

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19.1 Introduction

Intracranial hypotension (IH) is a rare cause of chronic subdural hematomas (CSDHs). The most common clinical presentation is headaches that often have a positional component and worsen when upright. According to the International Classification of Headache Disorders (ICHD-3), the diagnosis of IH requires a temporal relation of headaches to low cerebrospinal fluid (CSF) pressures [16]. Patients should be evaluated for low CSF pressures as well as for evidence of spinal fluid leaks on imaging.

There are several etiologies associated with IH, including post-traumatic, iatrogenic, and spontaneous. Post-traumatic, degenerative, and iatrogenic causes often have an identifiable cause of the spinal fluid leak, while cases of spontaneous IH may require more diagnostic workup. Spontaneous IH is rare and described most often in small retrospective reviews and literature reviews. Best estimates place the incidence of spontaneous IH at 5 per 100,000 per year [40].

Regardless of the etiology, diagnosis is difficult because the most common presenting symptom of IH is headaches. Both clinically and radiographically there are several other pathologies that present similarly and can confound the diagnosis. The focus of this chapter is IH presenting as CSDHs or hygromas. IH is an important etiology on the differential for CSDHs because they are managed differently from other subdural collections. While traditionally, CSDHs are managed with surgical evacuation, if IH is present surgery can result in worsened symptoms.

In this chapter we will review the presenting symptoms, clinical findings, radiology findings, and management of subdural hematomas (SDHs)/collections in the setting of IH.

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19.2 Pathogenesis

While IH can be spontaneous in nature, many cases described in the literature are secondary to invasive procedures. Some of the common iatrogenic causes include lumbar punctures, durotomies during spine surgery, and intradural spinal and cranial surgery [11, 22, 23, 53]. Any surgery or procedure where the dura is opened or breached can potentially result in IH if a spinal fluid leak persists. Likewise, traumatic etiologies of IH are secondary to dural injury. Some cases of traumatic IH have been linked to innocuous trauma [39]. There have also been case reports of degenerative spinal pathology that has resulted in dural defects leading to IH [6, 46, 49].

On the other hand, spontaneous cases of IH are thought to be secondary to an underlying weakness in the dura that results in a spinal fluid leak. Spontaneous cases of IH have been associated with connective tissue disorders but they are not linked directly to a specific disease process. Spontaneous etiologies have also been associated with spinal meningeal diverticula. These are outpouchings of the dura at the nerve root sheath which are thought to be susceptible to rupture and injury. However, interestingly, a recent retrospective series showed there was no difference in the presence and number of diverticula seen in patients with IH compared to those without it [20].

Regardless of the etiology, the mechanism through which low intracranial pressure develops is through a spinal fluid leak. One consequence of IH is the development of subdural fluid collections or hematomas. The data regarding the prevalence of subdural collections in the setting of IH is limited to retrospective reviews and ranges from 19.8 to 50% [5, 10, 22, 39, 45, 50].

The mechanism through which subdural collections or hematomas develop in the setting of IH is not well studied. One hypothesis is that there is a relative loss of pressure and buoyancy in the intracranial space that causes stretching of bridging veins making them susceptible to shear forces [22]. Another theory is that IH causes the development of thin-walled and fragile vessels within the dura that are prone to rupture. A small sample of six meningeal biopsies of diffusely enhancing dura showed small thin vessels on the subdural side of the dura in a loose amorphous fibroblastic matrix [22, 32]. CSDHs have also been shown to be encapsulated and divided by membranes in which there is neovasculature that has been associated with re-bleeding and expansion [17, 18, 51].

19.3 Clinical Presentation

The most common presentation of IH is headaches. Sometimes the headaches can have a positional component and worsen when patients are upright. However, when patients develop subdural collections from IH, there are a variety of additional

Table 19.1 Symptoms of intracranial hypotension

Headaches (postural or non-postural)
Neck stiffness
Dizziness or vertigo
Focal weakness or paresthesia
Visual deficits (such as diplopia)
Hearing disturbances (such as tinnitus)
Depressed level of consciousness

symptoms that can develop (Table 19.1). These symptoms range from mild focal neurologic deficits to severe alterations in mental status. Additional symptoms that patients may present with include but are not limited neck stiffness, dizziness, vertigo, focal neurological deficits such as weakness or paresthesia, visual field deficits, diplopia, hearing disturbance, tinnitus, and depressed level of consciousness [25, 41, 45]. In very rare cases, IH may mimic neurodegenerative disease such as dementia, Parkinsonism, and memory deficits [3].

The presenting symptoms are often nonspecific for IH. Therefore, a thorough clinical history can be very helpful in raising clinical suspicion for the diagnosis. Some clinical histories that have been associated with spontaneous IH include weight lifting, straining, violent sneezing or coughing, increased intra-abdominal pressure, and chiropractic manipulation [37, 38].There are reports in the literature that attribute up to 50% of patients presenting with SDHs due to IH to prior lumbar puncture [22]. There are also a number of reports of IH developing after spine or posterior fossa surgery [23, 49, 53].

A recent retrospective study showed that patients that presented with SDHs due to IH tended to have fewer underlying medical comorbidities, additional radiological signs of spontaneous IH such as meningeal enhancement, and had smaller volume of subdural hemorrhage compared to SDHs secondary to other pathologies [19]. Age is a demographic factor that has been studied as a risk factor for SDHs but studies conflict whether or not younger or older patients are at more risk for SDHs due to IH [19, 50].

One rare presentation of IH described in the literature is spontaneous cerebral venous thrombosis. Although venous sinus thrombosis itself is rare, there are several case reports in the literature that attribute the etiology to IH [35, 52, 54]. Treating venous sinus thrombosis secondary to IH can become challenging when CSDHs are present. The pathophysiology is not well understood, but one theory is that decreased intracranial pressure increases cerebral blood volume and leads to stasis in the venous system [52]. Standard venous sinus thrombosis treatment requires systemic anticoagulation; however, in the setting of IH and subdural hemorrhage, it may place patients at increased risk for complications. The successful treatment of cerebral venous thrombosis in the setting of IH requires identifying and treating the underlying cause of the IH [54].

19.4 Radiographic Imaging

Imaging plays an important role first in diagnosing IH and also identifying the cause. Because the clinical history can have an insidious onset, cranial imaging often plays an important role in establishing the diagnosis. If the clinical history does not reveal an obvious etiology for IH, then additional imaging of the spine may be needed in order to establish a diagnosis.

19.4.1 Cranial Imaging

Computed tomography (CT) of the head is often the first imaging completed in patients presenting with neurological symptoms or deficits. In IH, CT scans are often negative for any pathology, however can reveal subdural collections. Most commonly, subdural collections secondary to IH will appear hypodense (Fig. 19.1). They will often appear as bilateral collections and their appearance will be similar to chronic subdural hematomas or hygromas. They can be difficult to detect on CT imaging if the collections are small or appear isodense to the brain. Sometimes, the collections will appear subacute and have a mixed density, loculated appearance.

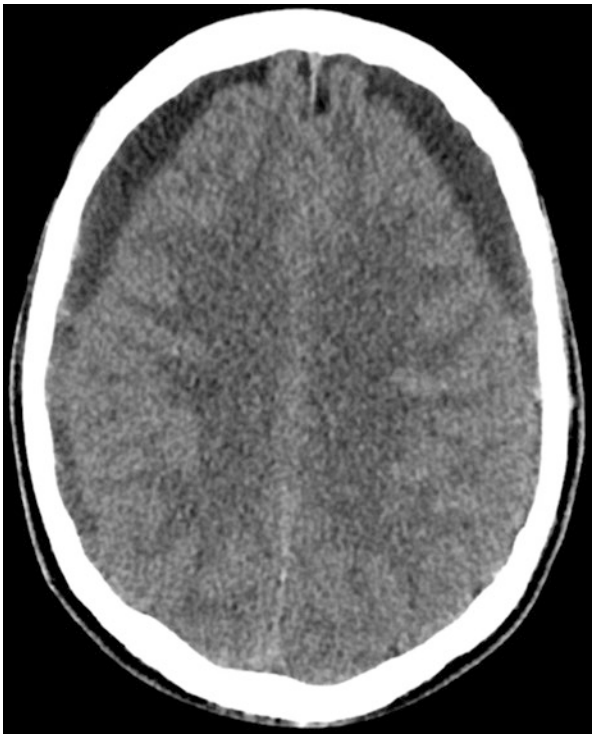

Fig. 19.1 Axial CT head of bilateral chronic subdural hematomas secondary to intracranial hypotension

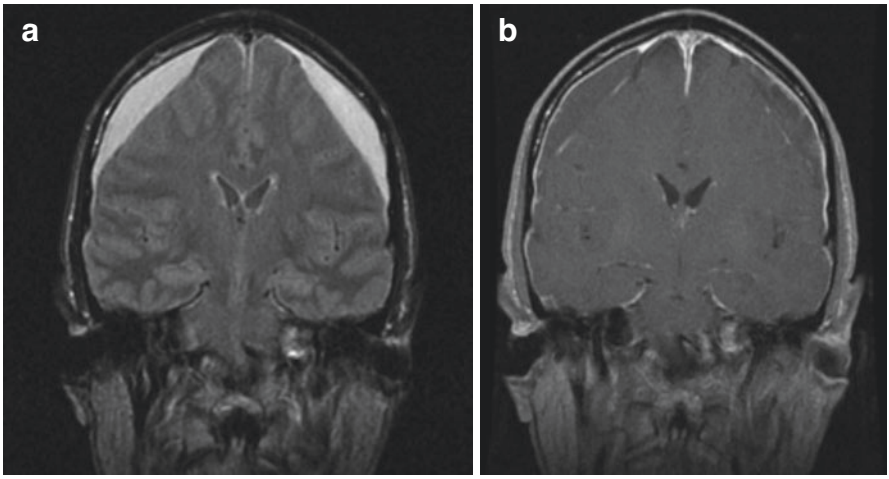

Fig. 19.2 Coronal MRI brain findings for intracranial hypotension. (**a**) FLAIR imaging revealing bilateral chronic subdural hematomas with compression of the frontal lobes. (**b**) T1 contrast weighted imaging with diffuse dural enhancement

CT imaging can also sometimes detect brain descent. This can be visualized by the appearance of cerebellar tonsillar crowding around the foramen magnum, obscured cisternal spaces, or ventral brainstem crowding. These findings can be misdiagnosed as a Chiari malformation.

Magnetic resonance imaging (MRI) of the brain in the setting of IH often reveals classic and reproducible findings. T1 contrasted sequences may reveal diffuse dural or pachymeningeal enhancement (Fig. 19.2). This finding can be considered pathognomonic. In some retrospective studies this finding was observed in 95% of the patients that were diagnosed with IH [1, 25, 31, 50]. The reason for which dural contrast enhancement occurs is not well understood but it is a finding that can also be seen in patients after shunting procedures [7].

A proportion of patients with IH may also have small subdural collections or hematomas that can be visualized on MRI of the brain that may not be apparent on CT imaging. These collections are often bilateral in nature but can present as unilateral subdural collections or hematomas [15, 34]. These collections can be subdural hygromas or CSF collections or can be hemorrhage that is chronic in nature. In a cohort of 40 patients, Schievenk et al. describes that 50% of patients had abnormal subdural collections and of those patients 60% had subdural hygromas while 40% had SDHs [41]. The underlying mechanism and explanation of why some patients will develop hematomas while others hygromas is not well understood.

MRI is more sensitive than CT imaging for detecting features of IH and a variety of additional imaging findings have been described (Fig. 19.3). Classically there is pontine flattening that can be quantified as a pontomesencephalic angle less than 50° or as a mammillopontine distance less than 5.5 mm. Crowding the basal cisterns and be present as well as an interpeduncular angle at the level of the mammillary

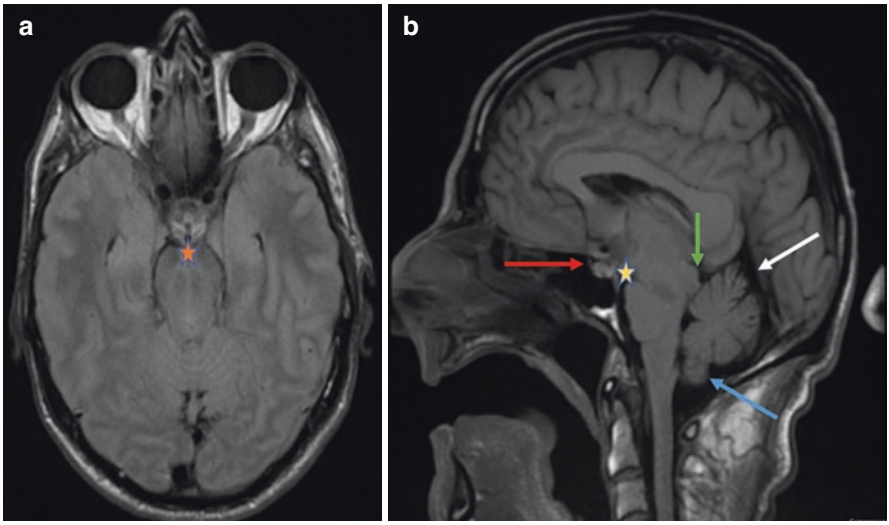

Fig. 19.3 Additional MRI findings associated with intracranial hypotension. (**a**) T1 axial imaging at the level of the mammillary bodies can show an interpeduncular angle <40° (orange star). (**b**) Venous engorgement and congestion of the straight sinus (white arrow), tectum displaced below the level of the tentorium (green arrow), cerebellar tonsillar descent (blue arrow), enlargement of the pituitary gland (red arrow), continue flattening with a pontomesencephalic angle <50° and mammillopontine distance less than 5.5 mm (yellow star)

bodies less than 40°. Both cerebellar tonsilar descent and descent of the tectum below the level of the tentorium are also suggestive of IH.

IH is a state of low pressures within the calvarium due to CSF hypovolemia and it is thought that a hyperemic compensatory mechanism occurs that results in venous engorgement [10]. Kim et al. describes that congestion and engorgement of both the straight dural sinus and the transverse dural sinus can be useful in the detection of IH [19]. Also venous sinus thrombosis has been associated as a complication of IH and may be visualized in these cases [35, 36, 54]. Finally, the pituitary gland may be conspicuously hyperemic or contrast enhancing on a post contrast sequence on MRI adding to the theory that these findings are related to a hyperemic state within the brain in the setting of low pressures [11, 25, 31].

19.4.2 Spinal Imaging

Once the diagnosis of IH has been established, additional spinal imaging may be needed if there is not a clear etiology from the patient's history. CSF leaks can have an insidious presentation. They may present as intermittent, low flow, or high flow leaks and the choice of imaging modality can affect the visualization and localization of these leaks [21]. CT myelogram, radionuclide cisternogram, digital subtraction myelography, and magnetic resonance imaging with or without myelography are all modalities that have been used in order to detect the source of CSF leak. All

of these imaging modalities involve the injection of intrathecal contrast with the exception of the T2-weighted fat saturation MRI. CSF leaks can have a variety of appearances such as epidural collections, small collections around nerve roots, or paraspinal collections.

CT myelography has been regarded as the traditional study for identifying CSF leaks because of its widespread availability and familiarity [21]. This involves the injection of iodinated contrast into the thecal sac through a lumbar puncture and subsequent or concurrent CT imaging of the spine. CSF leak rates can be variable. High flow leaks can show widespread extravasation of contrast from the thecal sac and localizing the egress point can be difficult. In these cases, dynamic CT myelography can be used. Using this technique serial CT scans of the spine are obtained both during and after injection of contrast allowing the visualization of early extravasation of contrast [27].

Spinal MRI has been more recently described in the literature as a comparable method to CT myelography for identifying spinal CSF leaks. Because CSF as well as fat is hyperintense on T2-weighted imaging, fat suppressed T2-weighted imaging can be used to identify CSF leaks. Spinal MRI has been useful in identifying high flow leaks which appear as an abnormal fluid (T2 hyperintense) epidural collection or collection adjacent to a nerve root [44]. There is some evidence to support that MRI is comparable to CT myelography. Starling et al. reported that 91.7% of patients with confirmed CSF leak on CT myelogram were also identified to have a CSF leak on spinal MRI [44]. Wang et al. also showed a high concordance rate between the two modalities [47]. MR myelography using intrathecal gadolinium has also been described; however, intrathecal injection of gadolinium is considered off-label use by the FDA [4].

Finally, both radionuclide cisternogram and digital subtraction myelography are less commonly used modalities for identifying CSF leaks. Digital subtraction myelography may be useful in the real-time detection, and localization of CSF leaks however is time and resource intensive. Radionuclide cisternogram seems to have fallen out of favor as an imaging modality for detecting spinal CSF leaks due to CT and MR imaging modalities having higher imaging resolution [21].

While spinal imaging can often identify a discrete leak that can be targeted for treatment. Sometimes there is no overt leak that can be identified. One pathology that has been found in association with spontaneous IH is perineural cysts (Fig. 19.4). These are spinal meningeal diverticula that occur at the nerve root sheath. They are thought to be a weakened portion of the dura that can be susceptible to elevated intracranial pressure or shear injury.

19.5 Treatment

The primary treatment for IH is to repair an identifiable CSF leak. Because IH is a rare entity and subdural collections only occur in a subset of patients, the literature regarding treatment algorithms is sparse. However, there have been a number of retrospective studies addressing treatment strategies and their effectiveness.

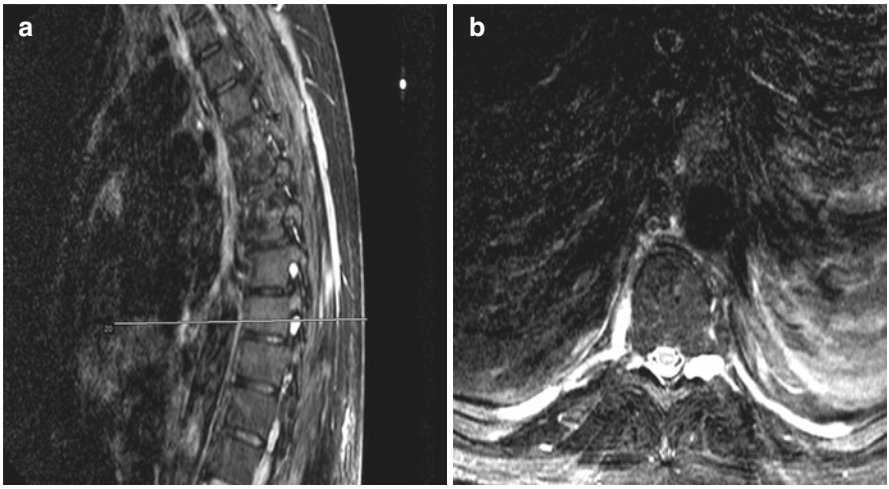

Fig. 19.4 T2-weighted imaging of the spine. Sagittal (**a**) and axial (**b**) imaging showing a perineural cyst

19.5.1 Conservative Management

Conservative management for IH involves bed rest and adequate hydration. The head of the bed is often kept flat. Effort is made to avoid straining, and analgesics may be employed for symptom control. Takahashi et al. describe that in a retrospective analysis of 55 patients with subdural hematomas in the setting of IH, 23.6% of patients were successfully treated via conservative measures [45].

19.5.2 Epidural Blood Patch

Epidural blood patches are an effective treatment modality for IH. Epidural blood patches can be performed under fluoroscopic guidance to inject autologous blood into the epidural space. These procedures can be targeted to a specific level if a discrete leak or abnormality is discovered on imaging. Numerous retrospective studies have shown that epidural blood patches are not only effective in relieving symptoms of IH but also can result in radiographic improvement on follow-up imaging. The literature describes the success rate of epidural blood patches to be anywhere between 46.8 and 100% and including patients that required multiple or repeat blood patch procedures [13, 14, 26, 30].

However, not all patients diagnosed with IH may be diagnosed with a targetable CSF leak [41, 42, 45]. Epidural blood patches can be performed as a "targeted" or "blind" procedure. Targeted epidural blood patches are performed precisely at the identified level of a CSF leak while blind epidural blood patches are performed at

the level of the lumbar spine [2, 28]. The efficacy of targeted versus blind epidural blood patches has not been well studied but reported efficacy in both groups appears promising. He et al. demonstrated that 87.9% of patients had symptomatic relief after the first targeted epidural blood patch one or two vertebral levels below the identified leak [14]. Levi et al. showed also that 89.1% of patients had symptomatic relief after blind lumbar epidural blood patches and included patients who underwent repeat procedures [24].

19.5.3 Fibrin Glue

Fibrin glue epidural injections have also been described in the literature as an alternative or adjunct to epidural blood patches. There have been reports described of fibrin glue mixed with autologous blood, fibrin glue alone, or fibrin glue use after epidural blood patch failure for the treatment of IH [8, 29, 43]. However, the literature on its use is sparse and further investigation is needed to evaluate its efficacy and safety.

19.5.4 Open Surgical Repair

Conservative measures and epidural patches can be effective treatment options; however, refractory cases IH may require an open surgical approach if there is an identifiable source of CSF leak. There are very few cases reported in the literature for open surgical repairs of CSF leaks for IH [12, 15, 46, 49]. The specific method of repair varies depending on the location of the leak, but even in the case reports, open surgical methods are reserved as a therapeutic modality in patients that fail multiple epidural blood patches or for those who present with acute neurological decline. Also, the majority of these cases involve ventral degenerative pathologies of the spine such as disc osteophyte complexes. Both anterior and posterior approaches to the spine have been described. Some interventions even required a partial corpectomy in order to reach the dural defect for primary or secondary repair.

19.5.5 Subdural Evacuation

CSDHs and collections have been shown to radiographically improve after the underlying cause of IH has been treated. These collections have been shown to resolve without surgical evacuation [9]. However, some CSDHs caused by IH can enlarge to a significant enough size to cause midline shift, altered level of consciousness, and herniation. In these cases, where patients present in neurologic extremis, subdural evacuation may be indicated. However, subdural evacuation

without addressing the underlying etiology may provide little benefit and can result in worsened outcomes.

Schievenk et al. and Ferrante et al. describe patients that underwent subdural evacuations alone, some due to delayed diagnoses, and reported that these patients had no improvement in symptoms or had significant neurological decline [9, 15, 41]. Fortunately, the majority of patients in these studies did have neurological recovery after the underlying IH was addressed.

There is no consensus or guidelines on how to manage large symptomatic CSDHs in the setting of IH. Takahashi et al. and Yoon et al. describe their clinical experience and recommend utilizing a combination of blind or targeted epidural blood patches with evacuation of the SDH in sequence [45, 52]. Whether or not the SDH should be evacuated before or after the underlying IH is addressed has not been studied. Extreme cases of IH may present with downward herniation syndrome. When downward herniation is suspected in the setting of IH, basic steps should be taken to reverse the pathology. The patient should be positioned with the head down in trendelenberg position. Intravascular volume should be expanded. Two case reports also describe that infusion of saline into the thecal sac can be a useful salvage maneuver in patients that show signs of downward herniation [33, 48].Then the underlying etiology should be addressed.

19.6 Conclusion

CSDHs are commonly encountered in the field of neurosurgery. While CSDHs caused by IH is a rare entity, recognizing the diagnosis is important in order to provide patients with the appropriate treatment. Because IH is rare, there are often delays in diagnosis. The clinical history is important in raising suspicion for IH. The diagnosis often requires multimodal cranial and spinal imaging. The vast majority of subdural collections will resolve by addressing the underlying cause of IH. Except for patients presenting in neurologic extremis with rapidly declining exams, caution should be given towards surgical evacuation until the source of the CSF leak has been identified and treated. Surgical intervention on SDHs secondary to IH can result in worsened neurologic symptoms.

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