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Review article

# Evidence based diagnosis and management of chronic subdural hematoma: A review of the literature

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ABSTRACT

Chronic subdural hematomas are encapsulated blood collections within the dural border cells with characteristic outer "neomembranes". Affected patients are more often male and typically above the age of 70. Imaging shows crescentic layering of fluid in the subdural space on a non-contrast computed tomography (CT) scan, best appreciated on sagittal or coronal reformats. Initial medical management involves reversing anticoagulant/antiplatelet therapies, and often initiation of anti-epileptic drugs (AEDs). Operative interventions, such as twist-drill craniostomy (TDC), burr-hole craniostomy (BHC), and craniotomy are indicated if imaging implies compression (maximum fluid collection thickness >1 cm) or the patient is symptomatic. The effectiveness of various surgical techniques remains poorly characterized, with sparse level 1 evidence, variable outcome measures, and various surgical techniques. Postoperatively, subdural drains can decrease recurrence and sequential compression devices can decrease embolic complications, while measures such as early mobilization and re-initiation of anticoagulation need further study. Non-operative management, including steroid therapy, etizolam, tranexamic acid, and angiotensin converting enzyme inhibitors (ACEI) also remain poorly studied. Recurrent hemorrhages are a major complication affecting around 10–20% of patients, and therefore close follow-up is essential.



## 1. Introduction

First described by Virchow in 1857, chronic subdural hematomas (cSDH) are encapsulated blood collections with a characteristic outer "neomembrane" [1]. cSDH often appear weeks (usually 3 or more) after minor head trauma, are bilateral in approximately 20% of cases, and are associated with significant morbidity and mortality [2,3]. Many approaches – both surgical and non-surgical – have been proposed to treat cSDH, with no current established standard of care. This review aims to identify evidence-based methods for diagnosing and managing cSDH.

## 2. Epidemiology

cSDH has a 3:1 male to female predilection, occurring at mean age of 77 years [6–8]. The overall incidence of cSDH in the general US population is approximately 5 per 100,000 [4], and the rate substantially increases to 58 per 100,000 in patients over 70 years of age [5]. Importantly, with the number of individuals over the age of 65 expected to double by the year 2050, the overall incidence of cSDH is also likely to rise [5,9].

Historically, head trauma was considered the most common cause of cSDH. However, with increased use of both anticoagulant and antiplatelet medications, cSDH frequently occurs in the absence of any identifiable trauma [5–8]. Other important risk factors include: advanced age, excessive alcohol consumption, epilepsy, low intracranial pressure (both spontaneous and iatrogenic), hemodialysis, and bleeding diathesis [2,5,9–11]. The elderly are particularly predisposed to cSDH because of frequent brain atrophy. Such atrophy stretches the bridging veins, making them more susceptible to tearing [12]. Given the broad presentation of this increasingly common and life-threatening condition, cSDH should be on the differential for any neurological change in an elderly patient.

## 3. Pathophysiology

cSDH are encapsulated structures with an inner and outer membrane. The inner membrane is thin and avascular while the outer membrane is thick and rich with immature, fragile blood vessels. Classically, cSDH are thought to occur within the "subdural"

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compartment – a potential space that exists between the dura and arachnoid mater. However, multiple investigations utilizing electron microscopy have challenged this notion by demonstrating cSDH to develop within a sub-layer of the dura mater itself, the dural border cells. These cells form the innermost layer of the dura mater and are immediately adjacent to the barrier cells of the arachnoid mater. Unlike the more external dural layers, the dural border cells are flattened fibroblasts with minimal extracellular collagen [13,14]. Such features make this layer one of the weakest in the meninges and thus a natural cleavage plane for fluid to collect after an insult. This theory has been supported by both animal models and human data that have identified dural border cells within both the inner and outer membranes of cSDH [14–17].

Several mechanisms have been identified by which blood or fluid can accumulate within the dural border cell layer. The most common mechanism involves head trauma, which causes tearing of bridging veins and resultant acute subdural hematoma (aSDH) [18]. Hemorrhage localizes to the dural border cell layer because it is a natural cleavage plane as described above as well as the site where the walls of the bridging veins are the thinnest [18]. As the acute hematoma expands, it incites an inflammatory response that drives clot fibrinolysis, production of granulation tissue, and release of angiogenic factors such as vascular epithelial growth factor and placental growth factor [19,20]. The granulation tissue that forms becomes rich in immature, fragile, and leaky blood vessels. Blood extravasation from these vessels then contributes to hematoma expansion. Subsequently, the characteristic tissue capsule of cSDH, the "neomembrane", forms over the course of 3–4 weeks [20,21]. Moreover, evidence from human cSDH samples identifying dural border cells in the outer hematoma membrane suggests that formation of this neomembrane is due to proliferation of dural border cells [22,15–17]. Like granulation tissue, this neomembrane is also full of immature and leaky blood vessels that contribute to hematoma expansion.

Of all aSDH, only 20% appear to become chronic [23,24]. One hypothesis suggests a balance between the body's natural resorptive mechanisms for hematomas and continued hematoma expansion. It has been proposed that if a neomembrane develops and matures quickly, the blood vessels stabilize and recurrent bleeding halts allowing for the hematoma to be fully resorbed over time. However, if the neomembrane develops slowly, the hematoma is likely to continue to expand and become a cSDH.

A second mechanism for cSDH development starts with a subdural hygroma – a fluid collection that accumulates within the dural border cell layer secondary to leakage of cerebrospinal fluid from the arachnoid layer. Hygromas can develop spontaneously, but are often associated with asymmetric skulls that place increased tension on the dura and arachnoid layers. Similar to the pathogenesis observed with aSDH, hygromas induce an inflammatory response that leads to proliferation of the dural border cells and angiogenesis. Microhemorrhages from these new blood vessels convert the hygroma into a hematoma [22].

## 4. Radiographic characteristics

The initial diagnostic study of choice for a cSDH is a non-contrasted head computed tomography (CT) scan. On CT, a cSDH presents as crescentic layering of fluid in the subdural space. Importantly, sagittal and coronal reformats are better measures of fluid volume and should be obtained in all cases, if possible (Fig. 1). Notably, unlike epidural hematomas, cSDH typically cross suture lines. There can also be mass effect with associated midline shift (MLS). The density of fluid is based on many factors, most notably the type and chronicity of fluid in the subdural space. Fluid within this space can be either blood or cerebrospinal fluid (CSF).

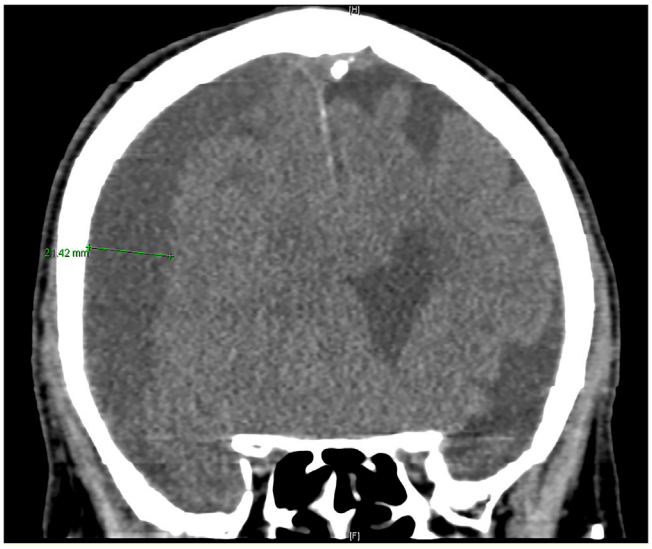


**Fig. 1.** Coronal non-contrast computed tomography showing chronic subdural hematoma with width of 21.42 mm.

In the case of external hydrocephalus, subdural hygromas, or early in the postoperative period, the fluid is likely CSF. On CT, this fluid will be isodense with the ventricular CSF. If the fluid is blood, the age of hemoglobin breakdown plays an important role in radiographic characterization, with cSDH typically appearing as a hypodense collection on CT. This is due to hemoglobin degradation into hemosiderin and hemochromes [25].

A special consideration should be made for bilateral cSDH as these will often present with minimal to no MLS, but with significant mass effect. The lack of MLS is due to equal, but opposite, forces on the brain parenchyma. In the setting of bilateral cSDH, the ventricles may appear squeezed secondary to the opposing mass effect.

## 5. Pre-surgical management

### 5.1. Reversing anti-coagulation

Given the potential for hematoma expansion, the activated prothrombin time (aPTT) and international normalized ratio (INR) results should be normalized prior to surgical intervention in order to prevent perioperative complications as well as cSDH recurrence. A number of common conditions and their treatments can alter the aPTT and INR. The most common of these is atrial fibrillation and treatment with warfarin. Prior to surgical intervention, INR should be reversed to less than 1.4.

If immediate reversal is required, four factor prothrombin complex concentrates (PCC), made up of factors 2, 7, 9, and 10, can be given to rapidly normalize INR. Fresh frozen plasma (FFP) contains all coagulation factors and can also be used to rapidly reverse warfarin. However, FFP may only partially reverse warfarin based on factor concentration and takes longer for warfarin reversal based on intravenous (IV) volume burden [26,27]. PCCs are often preferred due to a higher quantity of factors, with the goal of overwhelming inhibition by an influx of factors involved in the coagulation cascade. In addition, IV vitamin K can be administered to stimulate hepatic synthesis of these cofactors over time, and should also be given as an adjuvant to PCCs and/or FFP [28].

In an elective situation, warfarin should be held for at least five days prior to surgery, with an INR check on the day of surgery. If the INR continues to be mildly elevated (>1.4–2.0), IV vitamin K may be sufficient to eliminate the coagulopathy [29]. Those with