Chapter 2
Chronic Subdural Hematoma

Andrew Ajisebutu and Gregory Hawryluk

Clinical Scenario

A 75-year-old man is brought to the Emergency Department (ED) by his daughter-in-law, who has noted a declined in his function. Over the past 3 weeks, he has exhibited memory lapses and gait impairment and has spent an increased amount of time sleeping. Today, his daughter-in-law noticed that he had some difficulty with word-finding.

2.1 History and Neurologic Exam

The first priority of any physician confronted with a patient with a suspected CSDH should be to obtain an accurate history, with special attention to some relevant questions:

- *Etiology*: Is there a history of blunt trauma? If so, what was the mechanism and its proximity to the current presentation? In elderly patients, an assessment of mobility and fall history is important. Recognize that even mild trauma can precipitate CSDH formation in older patients.

A. Ajisebutu (✉)
Neurosurgery Resident, University of Manitoba, Winnipeg, Manitoba, Canada
e-mail: aajisebutu@manitoba-physicians.ca

G. Hawryluk
Neurosurgery, University of Manitoba, Winnipeg, Manitoba, Canada
e-mail: ghawryluk@hsc.mb.ca

P. B. Raksin (ed.), *Acute Care Neurosurgery by Case Management*,
https://doi.org/10.1007/978-3-030-99512-6_2

- *Patient demographics*: Is this an elderly patient, with age-related atrophy predisposing to CSDH formation? In a younger patient, are there risk factors for subdural formation, such as alcohol use or prior brain injury?
- *Medications*: Is there a history of antiplatelet or anticoagulant use? If so, what is the indication and what are the potential consequences of withholding the agent(s) in question?
- *Presentation*: Is the onset of symptoms acute or more insidious? Do symptoms suggest subacute progression? Are symptoms consistent with focal or lateralizing deficits?

On review of systems, the patient denies headache, nausea/vomiting, and dizziness. He does endorse experiencing a mild "numbness" of his dominant right hand and, as a result, has had difficulty with fine motor tasks like fastening buttons. He lives at home with his spouse. Prior to this episode, he had been independent with all activities of daily living and ambulated without walking aids. He has a history of hypertension and diet-controlled type II diabetes. He takes aspirin 81 mg daily, which his family states is for "heart health." On physical exam, he is awake, alert, and oriented to self and place, but not to date. His speech is slow and, at times, halting. On motor exam, he has a slight right-sided pronator drift.

The clinical presentation of CSDH is heterogeneous; therefore, a high index of suspicion must be maintained for a structural pathology when approaching patients presenting to the emergency department—even when focal findings are not apparent. CSDH is a relatively common neurosurgical condition; the overall reported incidence ranges anywhere from 1.75 to 20/100,000 people/year [1–4]. CSDH is generally a disease of the elderly; a study that examined Japanese patients between 2005 and 2007 reported an overall incidence of 20.6/100,000; when stratified by age, the incidence rose to 76.5 in patients 70–90 years of age, and 127.1 in those over 80 [2]. As populations age, there appears to be an increase in the overall incidence, primarily due to the higher prevalence of falls and anticoagulant use in this population. One group in the United States (US) created a mathematical model based on US, Japanese, and Finnish data, estimating that by 2030 we may see an over two-fold increase in incidence as the population ages [5]. However, CSDH is not seen exclusively in elderly patients. Young or middle-aged patients may develop chronic subdural collections, typically after trauma, in the setting of an acute subdural hematoma that liquefies over time, or with certain risk factors that predispose to premature brain atrophy such as alcoholism—which, itself, can be associated with coagulopathy and thrombocytopenia. Patients with renal failure and secondary platelet dysfunction may also be predisposed to CSDH formation.

Historically, it was believed that a difference in osmolarity between CSDH fluid and cerebrospinal fluid (CSF) established an oncotic pressure gradient that, in turn, drove CSDH expansion; however, published evidence has served to disprove this theory [6]. The current proposed mechanism of CSDH growth is that, following the initial injury and maturation of the hematoma, a neomembrane forms on both the dural and arachnoid surfaces of the clot, leading to its encapsulation. Neomembrane

formation involves the formation of new, fragile blood vessels through the process of neovascularization. This process precipitates microhemorrhages, and, along with hyperactivation of the fibrinolytic system, is responsible for continued growth and expansion of the collection [7]. Of additional importance, CSDHs contain low concentrations of coagulation factors, such as fibrinogen and plasminogen, and relatively high concentrations of coagulation breakdown products, supporting the idea that CSDHs act as contained "disseminated intravascular coagulation chambers" [8].

Making the diagnosis of CSDH may be challenging at times, as the clinical manifestations can be diverse and non-specific. The most common presenting complaints include gait disturbance, confusion, and limb weakness. Over half of patients who present with CSDH requiring surgical intervention complain of gait disturbance and falls; a third will have unilateral hemiparesis, and many will present with confusion or mental deterioration [9]. Many assume headaches to be a universal symtom of CSDH, however they only occur in 20–30% of patients [9]. Most patients will present with some combination of these symptoms. Other symptoms, such as incontinence, vomiting, seizures, aphasia, anisocoria, and visual disturbances are less common but do occur in about 2–10% of patients. Most patients present with a Glasgow Coma Scale (GCS) score of 13–15, although a significant minority (approximately 20%) may present with a GCS below 13. Approximately, 5–7% of those patients will be comatose (GCS <8) at presentation [9, 10]. Another 20–30% with CSDH, however, will be completely asymptomatic. Table 2.1 illustrates common presenting symptoms, stratified by frequency of occurrence.

A careful, comprehensive history must be obtained from both the patient and family when evaluating patients with suspected CSDH. Trauma is the most common cause of CSDH; however, it is important to understand that the inciting event can be as trivial as a sneeze. The timing of the traumatic event with respect to presentation varies, but it is most commonly on the order of several weeks. It is also important to recognize that nearly 40% of patients deny a history of trauma [11]. A detailed neurological exam should be obtained for all patients with suspected CSDH, with careful attention paid to level of consciousness and the presence of lateralizing signs.

Certain risk factors predispose patients to the development of CSDH, and their identification on history can be important in making the diagnosis. Table 2.2 summarizes both fixed and potentially modifiable risk factors for development of CSDH:

Advanced age. By far the most commonly reported risk factor in the literature is older age. Many epidemiologic studies of CSDH report higher rates in older age cohorts—where advanced age generally is defined as 55 and older [12]. The contribution of age as a risk factor has multiple facets. It has been theorized that brain atrophy plays a major role in the pathophysiology of the development of CSDH; minor trauma leads to the tearing of bridging veins that traverse from the cortex to the dura—the point at which they are thinnest and most vulnerable [13]. This causes small hemorrhages that accumulate within the potential space between the dura and the arachnoid. Recurrent trauma, particularly in the

Table 2.1 Common symptoms associated with CSDH presentation

Presentation of CSDH	
Symptoms	Rate
Gait disturbance	~50%
Mental deterioration	~30%
Unilateral limb weakness	~30%
Headache	~20–30%
Drowsiness or coma	~10%
Speech impairment	~5–10%
Seizure	~<5%

Adapted from Santarius et al. [9]

Table 2.2 Fixed and variable risk factors for the development of CSDH

Fixed risk factors
• Advanced age
• Male sex
Variable risk factors
• Excessive alcohol consumption
• Coagulopathy
• Trauma

"frequent fallers" prevalent in this age group, compounds this risk [14]. Not only is the incidence of CSDH higher in patients with advanced age, but disease severity (in terms of the degree of neurological deficit) tends to be worse at the time of admission [2].

Male gender. Most epidemiologic studies identify a male predominance for the diagnosis of CSDH. The reason for this gender disparity is unclear; however, it has been theorized that other risk factors—such as trauma and alcohol use—are also more prevalent among men. Hematoma recurrence is similarly affected; one review of over 300 cases in South Korea quoted a recurrence rate of 10.2% among males, yet only 3.1% among females.

Alcohol consumption. Excessive alcohol consumption is often quoted as a risk factor for CSDH formation. Its effect is related to a number of factors—it increases the rates of trauma, fall, and acute subdural formation; it promotes brain atrophy; and it can be associated with coagulopathy or thrombocytopenia [15, 16].

Coagulopathy. The most pertinent modifiable risk factor for the development of CSDH is the use of anticoagulation or antiplatelet therapy. Elderly patients have much higher rates of atrial fibrillation, coronary artery disease, and stroke, and as such, frequently have indications for blood-thinning medications [17, 18]. However, this also increases the risk of developing CSDH. One review of national insurance databases in Australia showed that patients anticoagulated with warfarin had a 40 times higher risk of CSDH development [19]. It is difficult to ascertain whether anticoagulation/antiplatelet use increases the likelihood of developing a CSDH, increases the severity of an existing CSDH, or both. At least one study found that the average time interval between trauma and the first operation for CSDH was significantly shorter for patients who had received antiplatelet/anticoagulant medications than for those who had not, suggesting that these medications do have an effect on disease severity and clinical presentation [19]. A careful history of anticoagulation use should be obtained, as it impacts both the likelihood of the diagnosis and the subsequent treatment. Similarly, significant medical comorbidities—such as severe hepatic failure or renal failure—may be accompanied by coagulopathy and/or thrombocytopenia that may predispose to CSDH formation and affect its course [15].

2.2 Differential Diagnosis

Our elderly patient on antiplatelet therapy has presented with a gradual, subacute decline in his cognitive function. His physical exam demonstrates speech arrest and lateralizing symptoms. At this point, the differential diagnosis remains broad. However, the presence of lateralizing complaints may increase the likelihood of a structural brain etiology. We should consider both likely and less likely diagnoses, as well as the investigations needed to narrow our differential diagnosis.

Given the often vague history and variation in presentations, CSDH has been referred to as the "great imitator" [20]. Patients harboring a CSDH may present with a constellation of neurologic symptomatology: sensorimotor changes, dysphasia, and neuropsychiatric changes. It is important that clinicians maintain a broad differential diagnosis incorporating structural pathology when confronted with these patients, both when CSDH is suspected and when it is not.

For any patient presenting to the emergency department with decreased level of consciousness, lateralizing symptoms, and speech changes, stroke must be near the top of the differential. Though CSDH—by definition—develops gradually, patients may present with acute neurologic symptoms mimicking stroke. Prompt neurologic

assessment and imaging are essential to exclude an acute stroke that may require urgent intervention. More generally, CSDH can present like any intracranial space-occupying lesion, such as a tumor or intracerebral hemorrhage. This possibility, likewise, would prompt an urgent neurological assessment and neuroimaging.

It may also happen that a patient presents initially to a primary care provider, rather than to the emergency department, with subacute symptoms of gradual confusion and memory or mood changes—more suggestive of dementia than bleed. It is important to remember that the age groups and demographics of these two conditions may overlap. In such cases, a detailed neurologic exam may demonstrate lateralizing findings—making CSDH somewhat more likely—or at least prompt further investigation with neuroimaging. When lateralizing symptoms are not present, a structural cause still remains a possibility. Time course of symptom onset may be revealing. While one would expect CSDH-related symptoms to evolve over a period of weeks, a time line of several months to years would be more typical for dementia. One would also expect expansion of the hematoma to result in more rapid progression of symptoms as compared with dementia. Patients (particularly, elderly) presenting with confusion should be screened for other conditions, such as urinary tract infections or pneumonia, that may present with altered mental status.

The diagnosis of CSDH is usually evident by non-contrast CT imaging. However, a particularly important diagnosis that should be ruled out is subdural empyema. Presentation with a history of recent neurosurgical intervention, fever, constitutional symptoms, an elevated white blood cell count, immunosuppression, or intracranial mass effect disproportionate to the size of the extra-axial collection should prompt concern for this entity. Subdural empyema must be quickly identified, as prompt surgical intervention is often indicated. Magnetic resonance imaging (MRI) can be used to more clearly differentiate a CSDH from a subdural empyema.

An additional entity to consider in the setting of a hypodense extra-axial collection is subdural hygroma, which is a collection of CSF in the subdural space. Hygromas occur spontaneously, and are believed to form due to the splitting of the arachnoid and the dura at points of tension, allowing CSF to fill this otherwise potential space [21]. Subdural hygroma is generally a benign condition that does not require intervention, although it is possible to have acute bleeding into these fluid collections, which may, in turn, warrant surgical evacuation depending on the clinical context.

2.3 Diagnostic Evaluation

Non-contrast CT head is the best first step in the diagnostic evaluation for potential CSDH. CSDHs appear as crescentic collections that spread out within the extra-axial space between the dura and underlying brain. The density of the collections, measured in Hounsfield units (HU), provides an idea of the chronicity of the lesion: truly chronic collections appear hypodense (<30 HU), while those with a subacute component may appear more isodense (HU of 30–60), so much so that even

experienced physicians may overlook them if they do not review the images thoroughly [22]. In such cases, a post-contrast study may be helpful; enhancement of the cortical vessels more clearly defines the adjacent extra-axial space and its contents as distinct from brain.

The radiographic appearance of CSDH does have some clinical relevance to both treatment and recurrence rate. Some authors have attempted to characterize CSDHs into distinct subtypes, based on CT appearance: *homogenous*, wherein the CSDH retains the same HU throughout; *laminar*, in which a high-density component (thought to consist of fresh blood) runs along the inner membrane; *separated*, wherein a gradient is formed between the thin components and the thicker components of the CSDH; and finally, *trabecular*, in which the hematoma appears to be loculated, with a mix of isodense and hypodense components (Fig. 2.1a–d). Nakaguchi et al. hypothesized that these differing configurations represent distinct stages of the disease process and potentially impact recurrence rates. Recurrence rates among the separated subtype were high (36%), while those among the trabecular subtype were near zero. Homogenous and laminar subtypes were intermediate in behavior, with reported recurrence rates of 15% and 19%, respectively. In our experience, collections that are isodense or darker with respect to the brain are liquid and readily amenable to drainage.

The diagnosis of CSDH can be made solely on the basis of a non-contrast CT scan. If there is a clinical suspicion for subdural empyema, MRI brain pre- and post-gadolinium may provide additional detail to permit differentiation from simple hematoma. The post-gadolinium T1 sequence may demonstrate peripheral enhancement of the collection. A collection of infectious origin should demonstrate restricted diffusion (i.e., appear bright) on the diffusion-weighted imaging (DWI) sequence, whereas simple hematoma should not. This distinction is relevant both to surgical and medical management. A craniotomy is necessary in the case of suspected empyema, where organized phlegmon is unlikely to be amenable to burr hole drainage. Likewise, empiric broad-spectrum antimicrobial therapy would be appropriate in the setting of suspected infection.

For this particular patient, screening blood studies were unremarkable. Non-contrast CT scan of the head revealed a 1 cm thick, crescent-shaped, hypodense collection on the left side, associated with 0.25 cm of midline shift—consistent with a chronic subdural hematoma (Fig. 2.2).

2.4 Clinical Decision-Making and Next Steps

In this case, CT imaging identified the presence of a left-side CSDH with associated mass effect. How should this symptomatic (confusion, aphasia) elderly patient receiving antiplatelet therapy best be managed? What should be done with his antiplatelet agent? Is operative or nonoperative intervention appropriate? If operative, what type of procedure should be performed? What adjuncts are available, and are they necessary (or advisable)?

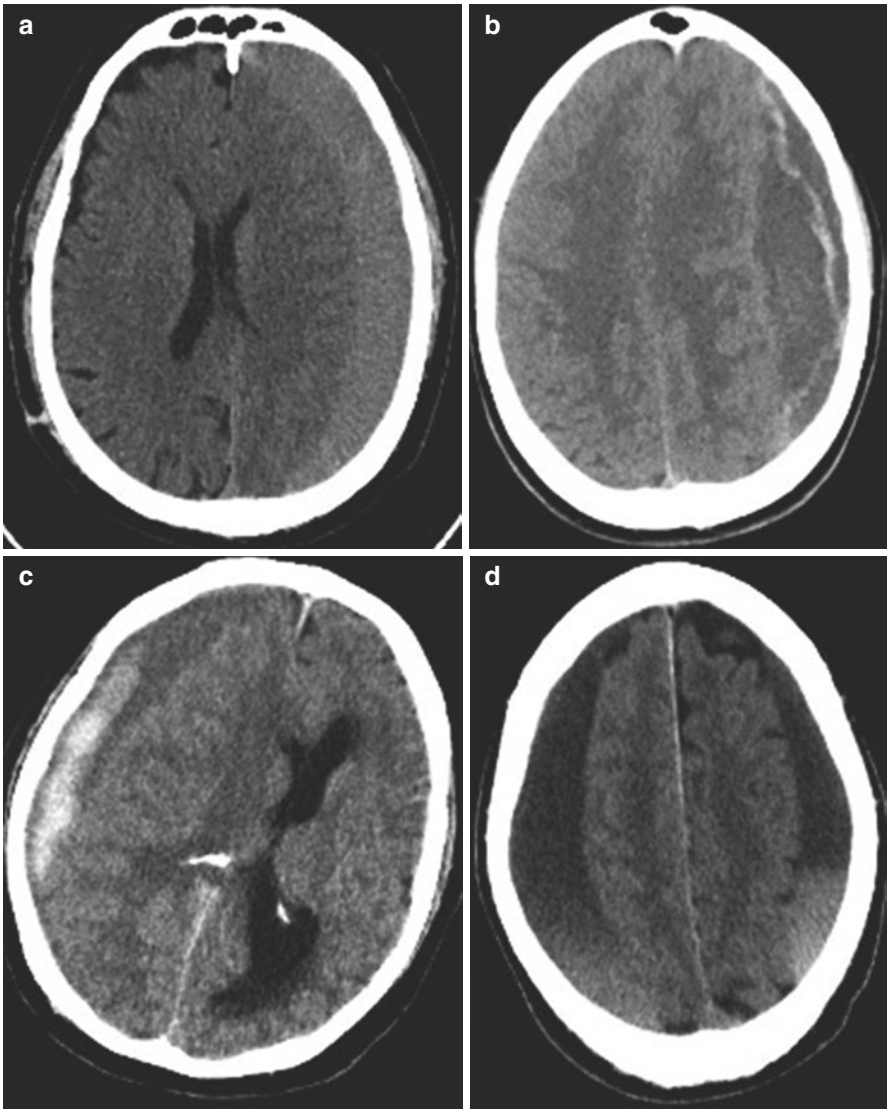

Fig. 2.1 (**a**–**d**) CSDH subtypes on CT scans. Proposed subtyping of CSDH by Nakaguchi et al. (J Neurosurg, 2001). (**a**) Homogenous—the CSDH general maintains the same HU throughout. (**b**) Trabeculated—the CSDH has a septated, mixed appearance with iso- and hypodense components. (**c**) Laminar—the CSDH has high-density components along its inner membrane. (**d**) Separated—the CSDH forms a gradient, representing its thinner and thicker components

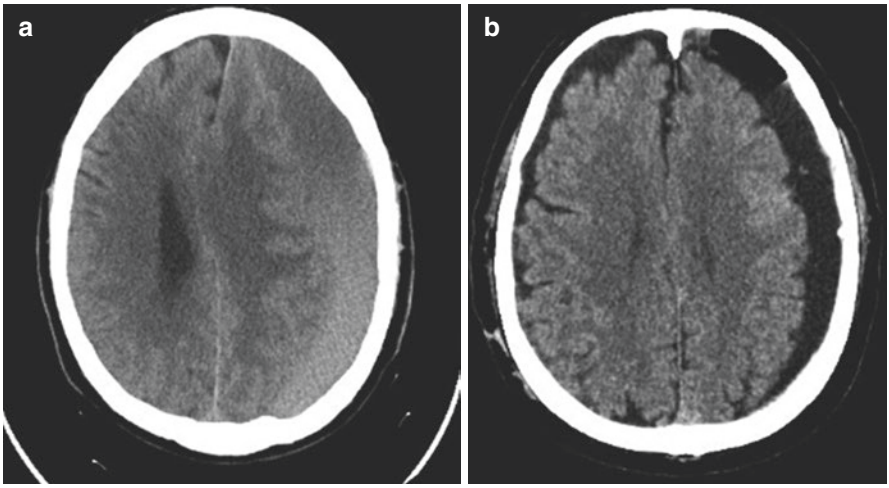

Fig. 2.2 (**a**) CT scan demonstrating a mixed iso- and hyperdense extra-axial collection, consistent with a chronic subdural hematoma. There is significant sulcal effacement and midline shift present. (**b**) Post-operative CT scan demonstrating single bur hole placement for evacuation of the CSDH. The subdural collection was thoroughly irrigated with saline; there is some evidence of post-operative pneumocephalus, common after these procedures. The midline shift and sulcal effacement have resolved, and a subdural drain has been placed (see arrow)

After arriving at the proper diagnosis, clinicians must decide on the appropriate course of treatment. The first step is to decide whether invasive or conservative therapy is indicated. What these two pathways share is medical optimization. Many patients, particularly elderly patients, have significant medical comorbidities, such as hypertension, congestive heart failure, renal or hepatic diseases that must be addressed and optimized prior to any intervention. Moreover, it is not uncommon for patients to present on anticoagulation or antiplatelet therapy. These agents should be held, and correction may be considered depending on the agent, indication, and planned intervention. Both anticoagulation and antiplatelet therapy have negative impacts on outcome: patients presenting on these medications have longer stays in hospital, higher rates of recurrence, and higher rates of mortality [23–26].

The method of reversal relies heavily on the mechanism of the coagulopathy. Patients on vitamin K antagonists, such as warfarin, may be reversed with a combination of vitamin K and prothrombin complex concentrate (PCC) or fresh frozen plasma (FFP), with a goal of reducing the International Normalized Ratio (INR) to <1.4. PCC helps to avoid fluid overload, as was typically seen when FFP was administered to these patients in the past. For direct oral anticoagulants, such as dabigatran and rivaroxaban, reversal agents do exist (idarucizumab and andexanet alpha, respectively); however, they are costly and not universally available. These agents

should be held, and when possible, surgical intervention should be delayed for 24–48 h. Reversal with PCC in emergent situations may show some benefit [27].

In the setting of antiplatelet therapy, operative intervention should be delayed to up to 7–10 days, if possible, to allow for replenishment of functional platelets. There is little to no evidence that platelet transfusions are beneficial in this setting and may, in fact, be harmful. Desmopressin (DDAVP) has been proposed as an agent that could be utilized in the setting of platelet dysfunction due to its ability to increase plasma von Willebrand factor, as well as to promote platelet adhesion. Currently, the Neurocritical Care Society and Society of Critical Care Medicine support the use of DDAVP in intracranial hemorrhage in patients exposed to antiplatelet agents although their utility in the setting of CSDH is less clear [28]. The timing for resumption of antiplatelet and anticoagulant agents is somewhat controversial—published studies suggest re-introduction variously at 5–7 days, 2 weeks, or 1 month after a bleed and/or invasive intervention [29]. Certainly, the correct answer depends on the indication for these medications, and risk stratification can be done using standardized scoring tools, such as the CHADSVASC score [30]. An individualized approach is recommended.

The mainstay of treatment for CSDH remains surgical. Patients with symptomatic CSDH benefit from surgical intervention: 70–80% of patients report a favorable outcome, though recurrence is seen in as many as 30%. There are a variety of surgical techniques that can be utilized, including twist-drill craniotomy (TDC) or craniostomy, burr hole craniotomy (BHC), and mini craniotomy. These techniques vary with respect to the size of the bony opening for access, where the procedure is performed (either in a sterile operating theater or bedside), and the drainage system utilized afterwards, if any. Surgery is generally safe; a meta-analysis published by Ducruet et al. quoted a complication rate of 2.5%, 3.9%, and 9.3% for craniostomy, craniotomy, and burr hole, respectively. They found a mortality rate higher for craniotomy (12.2%), when compared to TDC (5.1%) or BHC (3.8%). The rates of recurrence were as follows: BHC 11.7%, craniotomy 19.4%, and TDC 28.1% [26]. This particular meta-analysis gives a somewhat varied picture and suggests that no one technique is superior to another. Surgeons should exercise clinical judgment to ascertain which treatment is optimal for which patient. In patients with multiple comorbidities, a single burr hole may be best, as the procedure can be completed under local, with modest sedation, and is associated with a lower complication rate than mini craniotomy. Craniotomy is generally reserved for patients failing one or more attempts at burr hole drainage, those with a significant acute component, or those with problematic septations.

Each surgical option is imbued with certain technical nuances that may influence outcomes. For BHC, some advocate for two burr holes over simply one. Systematic reviews on the subject have not demonstrated clear evidence to support one versus the other [31, 32]. When performing craniotomies, an inner membranectomy can be performed, with the thought that this may facilitate brain re-expansion, along with the reabsorption of CSDH components by cortical and dural glymphatic/lymphatic pathways [33]. However, it is a common belief that this benefit must be balanced against the risk of seizures inherent to membranes stripping. The choice of

anesthesia, either using generalized anesthetic or local with sedation, is also a topic for debate; general anesthetics may pose some risk, particularly for patients with significant medical comorbidities. However, because these patients often present with confusion and agitation, it may be ill-advised to proceed without adequate sedation given the risk of patient movement during the procedure. The use of conscious sedation is, however, a good option for patients who carry a high risk with generalized anesthetic, but who may not tolerate the use of local anesthetic alone. Prospective randomized trials currently underway aim to assess the risk and benefits of general anesthesia (the NEURANESTH and GAS trials). The use of subdural drains has been investigated via a randomized control trial conducted by Santarius et al.; this study demonstrated a clear reduction in recurrence with the use of a drain [9]. Since that time, subperiosteal and subgaleal drains have been studied; published data suggest at least noninferiority of these techniques compared to subdural drain placement [34, 35]. It is the authors' preference to evacuate CSDH using a single burr hole, coupled with a high volume of intraoperative irrigation through a subdural drain—until the effluence runs clear. If there is concern about the ability to safely place a drain in the subdural location, the authors will leave one in the subgaleal space, given the recent supportive literature for that approach.

There are many situations in which surgical intervention may not be appropriate as first-line intervention. Nonoperative management should be reserved for patients for whom the benefits of surgery are felt to be outweighed by the risks; this may be true of patients with multiple comorbidities and poor baseline functioning. In those patients, a careful, patient-centered approach that includes other services (such as geriatric or palliative care medicine) should be undertaken, including detailed conversations with family members surrounding goals of care. For patients with small, asymptomatic collections, nonoperative management is often appropriate. Close follow-up with repeat imaging can be considered; generally, a CT scan at 1–2 weeks is performed to ensure stability of the subdural, followed by another at a 3-month interval. Spontaneous resolution of CSDH is possible and has been reported in the literature [36].

The non-surgical management of CSDH is an expanding field. Middle meningeal artery (MMA) embolization is a relatively new treatment modality for CSDH (Fig. 2.3a–d); the rationale for this approach is based on the concept that recurrent hemorrhage from the CSDH membrane is responsible for its evolution and that blood flow to the membrane originates from the MMA. This blood flow can be disrupted through embolization of this artery. Embolization versus conventional treatment was compared in a trial performed by Ban et al. Patients were prospectively enrolled in the study and underwent MMA embolization. Asymptomatic patients received MMA embolization alone, while those with symptoms also underwent surgery. This cohort was compared against a retrospective group treated in the conventional manner. The authors demonstrated significantly lower rates of treatment failure in patients who underwent embolization (1.4% vs %27.5) and a low rate of surgical rescue among those asymptomatic patients who underwent embolization as the sole modality of treatment (1.4% vs 18.8%) [37].

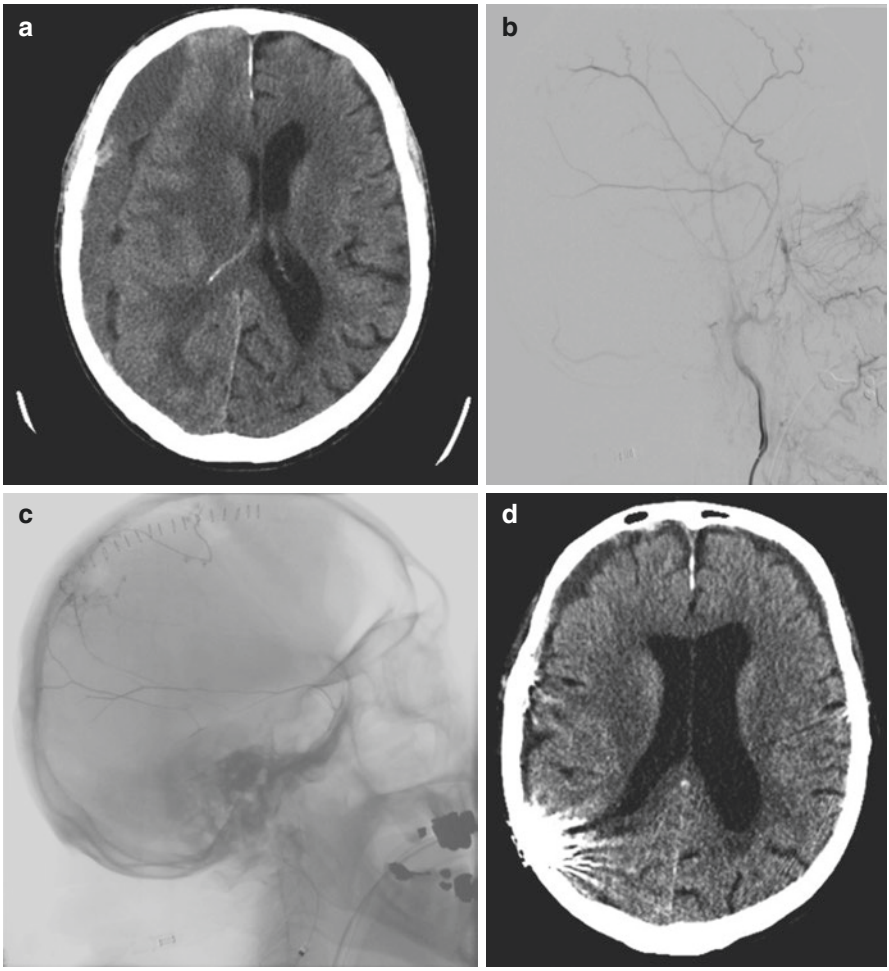

Fig. 2.3 (**a**–**d**) The use of MMA embolization in CSDH management. (**a**) CT scan demonstrating a mixed iso- and hyperdense extra-axial collection, consistent with a chronic subdural hematoma. There is significant sulcal effacement and midline shift present. (**b**) External carotid injection utilized for road mapping during the injection of non-adhesive liquid embolic agent (SQUID) material for MMA embolization. (**c**) Post embolization of the middle meningeal artery. Evidence of a mini craniotomy performed prior to embolization can be appreciated. (**d**) One month follow-up CT scan revealing resolved CSDH with hyperintense artifact representing the embolic material

A larger, multicentered clinical prospective study of MMA embolization as primary or rescue treatment has been performed. In this trial, surgical treatment options were left to the discretion of the attending physicians; surgery was offered to patients deemed clinically symptomatic (those with weakness grade 4/5 or worse and/or midline shift over 5 mm), and MMA embolization was utilized as an adjunct. The authors reported a 6.5% recurrence rate within 90 days, and a 9.4% complication rate, which included asymptomatic and symptomatic recurrence (2.2% and 5.1%,

respectively), asymptomatic MMA rupture (0.7%), post op seizure (0.7%), and facial droop (0.7%) [38]. Other published trials have been predominantly comprised of small case series that appear to support the use of MMA embolization in conjunction with surgical intervention for the reduction of recurrence [39, 40]. This may be a promising avenue for treatment for both asymptomatic patients and patients with recurrent CSDH; however, larger randomized trials must be completed and are currently underway. At this time, it is the preference of the senior author to reserve MMA embolization for recurrent hemorrhage.

Other adjuvant therapies may also be considered. The role of steroids in CSDH management is somewhat controversial. Existing retrospective and prospective studies do suggest that there may be role for steroids as an adjunct to reduce recurrence rates [41, 42]. However, a recent multicenter, randomized trial conducted in the United Kingdom compared oral dexamethasone treatment to placebo. The majority of patients in this study underwent surgical evacuation in addition to steroid treatment. They found that although patients treated with dexamethasone demonstrated lower rates of recurrence, they also had fewer favorable outcomes and more adverse events at 6 months [43]. This result may reflect the older population in which CSDH is most prevalent and which also tends to have higher rates of frailty and comorbidities; the results of this trial suggest that caution must be employed when considering steroids in the management of CSDH. Other agents, such as tranexamic acid, angiotensin-converting enzyme inhibitors, and platelet-activating factor receptor antagonists (such as etizolam), have been proposed [44–46]. Although they have yet to be become common place treatments, they remain active areas of research. Anecdotal evidence from our institution suggests that steroids may be more efficacious when instituted for CSDH believed present for only a short period of time—prior to the formation of membranes. Considering published evidence and personal experience, the senior author will consider offering dexamethasone therapy to select patients judged to have poor operative risk when a structurally complex CSDH is not evident and membranes are not suggested on imaging.

The use of prophylactic antiepileptic drugs (AEDs) lacks general consensus, and there remains a relative paucity of evidence to support their use [47]. On the one hand, that rate of seizures among patients with CSDH may be between 2% and 19% [48]. However, AEDs have been associated with increased incidence of falls in patients above the age of 65, and therefore, are not without risk [49]. The authors prefer to administer a 7-day course of AED prophylaxis to patients undergoing surgical drainage of CSDH, especially if membrane stripping is performed.

In this case, based on discussions with the patient and his family, operative intervention was chosen. The patient was admitted to hospital, and his aspirin was held. A pre-operative consultation with the anesthesiologist was arranged to optimize comorbidities and select the modality of anesthesia. Given the presence of aphasia and agitation, it was decided that this would not be an ideal case for a bedside craniostomy or awake burr hole, and so the patient was brought to the operating room for a generalized anesthetic. We opted for single burr hole drainage, coupled with high-volume intraoperative irrigation through a subdural drain. The patient

tolerated the procedure well. Post-operative CT scan revealed near total resolution of the CSDH. The drain was removed the next day. The patient's speech deficits and weakness resolved. He was discharged on post-operative day #3 after being clearing by our physiotherapy, occupational therapy, and speech language pathology teams. He continued to do well in follow-up, and, to date, has had no clinical recurrence.

2.5 Clinical Pearls

- CSDH may present with a constellation of neurological and neuropsychiatric symptoms and should be suspected in any patient—particularly if elderly—with a subacute decline.
- Many patients presenting with CSDH deny a history of trauma or point to an event that seemed trivial at the time.
- Surgical intervention—whether by burr holes or mini craniotomy—remains the mainstay of treatment for symptomatic CSDHs.
- There is growing evidence that middle meningeal artery embolization may provide a viable option for adjuvant or sole therapy in select CSDH patients.

References

1. Asghar M, Adhiyaman V, Greenway MW, Bhowmick BK, Bates A. Chronic subdural haematoma in the elderly—a North Wales experience. J R Soc Med. 2002;95(6):290–2.
2. Karibe H, Kameyama M, Kawase M, Hirano T, Kawaguchi T, Tominaga T. [Epidemiology of chronic subdural hematomas]. No Shinkei Geka. 2011;39(12):1149–53.
3. Foelholm R, Waltimo O. Epidemiology of chronic subdural haematoma. Acta Neurochir (Wien). 1975;32(3–4):247–50.
4. Kudo H, Kuwamura K, Izawa I, Sawa H, Tamaki N. Chronic subdural hematoma in elderly people: present status on Awaji Island and epidemiological prospect. Neurol Med Chir (Tokyo). 1992;32(4):207–9.
5. Balser D, Farooq S, Mehmood T, Reyes M, Samadani U. Actual and projected incidence rates for chronic subdural hematomas in United States Veterans Administration and civilian populations. J Neurosurg. 2015;123(5):1209–15.
6. Weir B. Oncotic pressure of subdural fluids. J Neurosurg. 1980;53(4):512–5.
7. Holl DC, Volovici V, Dirven CMF, Peul WC, van Kooten F, Jellema K, et al. Pathophysiology and nonsurgical treatment of chronic subdural hematoma: from past to present to future. World Neurosurg. 2018;116:402–411.e2.
8. Sugiura M, Mori N, Sugimori T, Imanaga H, Kitamura K, Kohno H. [Intracranial hematoma secondary to chronic DIC (author's transl)]. No Shinkei Geka. 1982;10(3):295–303.
9. Santarius T, Kirkpatrick PJ, Ganesan D, Chia HL, Jalloh I, Smielewski P, et al. Use of drains versus no drains after burr-hole evacuation of chronic subdural haematoma: a randomised controlled trial. Lancet (London, England). 2009;374(9695):1067–73.
10. Mori K, Maeda M. Surgical treatment of chronic subdural hematoma in 500 consecutive cases: clinical characteristics, surgical outcome, complications, and recurrence rate. Neurol Med Chir (Tokyo). 2001;41(8):371–81.

11. Sousa EB, Brandão LFS, Tavares CB, Borges IBC, Neto NGF, Kessler IM. Epidemiological characteristics of 778 patients who underwent surgical drainage of chronic subdural hematomas in Brasília, Brazil. BMC Surg. 2013;13:5.
12. Farhat Neto J, Araujo JLV, Ferraz VR, Haddad L, Veiga JCE. Chronic subdural hematoma: epidemiological and prognostic analysis of 176 cases. Rev Col Bras Cir. 2015;42(5):283–7.
13. Yamashima T, Friede RL. Why do bridging veins rupture into the virtual subdural space? J Neurol Neurosurg Psychiatry. 1984;47(2):121–7.
14. Baechli H, Nordmann A, Bucher HC, Gratzl O. Demographics and prevalent risk factors of chronic subdural haematoma: results of a large single-center cohort study. Neurosurg Rev. 2004;27(4):263–6.
15. Gelabert-González M, Iglesias-Pais M, García-Allut A, Martínez-Rumbo R. Chronic subdural haematoma: surgical treatment and outcome in 1000 cases. Clin Neurol Neurosurg. 2005;107(3):223–9.
16. Yang W, Huang J. Chronic subdural hematoma: epidemiology and natural history. Neurosurg Clin N Am. 2017;28(2):205–10. Available from: https://www.sciencedirect.com/science/article/pii/S1042368016300997.
17. De Rosa R, Piscione F, Galasso G, De Servi S, Savonitto S. Antiplatelet therapy in very elderly and comorbid patients with acute coronary syndromes. J Geriatr Cardiol. 2019;16(2):103–13. Available from: https://pubmed.ncbi.nlm.nih.gov/30923541.
18. Ng KH, Hart RG, Eikelboom JW. Anticoagulation in patients aged ≥75 years with atrial fibrillation: role of novel oral anticoagulants. Cardiol Ther. 2013;2(2):135–49. Available from: https://pubmed.ncbi.nlm.nih.gov/25135392.
19. Torihashi K, Sadamasa N, Yoshida K, Narumi O, Chin M, Yamagata S. Independent predictors for recurrence of chronic subdural hematoma: a review of 343 consecutive surgical cases. Neurosurgery. 2008;63(6):1125–9; discussion 1129.
20. Potter JF, Fruin AH. Chronic subdural hematoma—the "great imitator". Geriatrics. 1977;32(6):61–6.
21. Lee K-S. Natural history of chronic subdural haematoma. Brain Inj. 2004;18(4):351–8.
22. Markwalder TM. Chronic subdural hematomas: a review. J Neurosurg. 1981;54(5):637–45.
23. Forster MT, Mathé AK, Senft C, Scharrer I, Seifert V, Gerlach R. The influence of preoperative anticoagulation on outcome and quality of life after surgical treatment of chronic subdural hematoma. J Clin Neurosci. 2010;17(8):975–9.
24. Pieracci FM, Eachempati SR, Shou J, Hydo LJ, Barie PS. Degree of anticoagulation, but not warfarin use itself, predicts adverse outcomes after traumatic brain injury in elderly trauma patients. J Trauma. 2007;63(3):525–30.
25. Yasuda CL, Morita ME, Nishimori FY, Yasuda AM, Alves HL. [Chronic subdural hematoma: study of 161 patients and the relationship with coagulation abnormalities]. Arq Neuropsiquiatr. 2003;61(4):1011–4.
26. Ducruet AF, Grobelny BT, Zacharia BE, Hickman ZL, DeRosa PL, Andersen KN, et al. The surgical management of chronic subdural hematoma. Neurosurg Rev. 2012;35(2):155–69; discussion 169.
27. Cuker A, Burnett A, Triller D, Crowther M, Ansell J, Van Cott EM, et al. Reversal of direct oral anticoagulants: guidance from the anticoagulation forum. Am J Hematol. 2019;94(6):697–709.
28. Frontera JA, Lewin JJ, Rabinstein AA, Aisiku IP, Alexandrov AW, Cook AM, et al. Guideline for reversal of antithrombotics in intracranial hemorrhage: executive summary. A statement for healthcare professionals from the Neurocritical Care Society and the Society of Critical Care Medicine. Crit Care Med. 2016;44(12):2251–7.
29. Hawryluk GWJ, Furlan JC, Austin JW, Fehlings MG. Individual characteristics and management decisions affect outcome of anticoagulated patients with intracranial hemorrhage. World Neurosurg. 2014;81(5–6):742–51.
30. Gage BF, van Walraven C, Pearce L, Hart RG, Koudstaal PJ, Boode BSP, et al. Selecting patients with atrial fibrillation for anticoagulation: stroke risk stratification in patients taking aspirin. Circulation. 2004;110(16):2287–92.

31. Smith MD, Kishikova L, Norris JM. Surgical management of chronic subdural haematoma: one hole or two? Int J Surg. 2012;10(9):450–2.
32. Belkhair S, Pickett G. One versus double burr holes for treating chronic subdural hematoma meta-analysis. Can J Neurol Sci. 2013;40(1):56–60.
33. Sahyouni R, Mahboubi H, Tran P, Roufail JS, Chen JW. Membranectomy in chronic subdural hematoma: meta-analysis. World Neurosurg. 2017;104:418–29. Available from: https://pubmed.ncbi.nlm.nih.gov/28512051.
34. Soleman J, Lutz K, Schaedelin S, Kamenova M, Guzman R, Mariani L, et al. Subperiosteal vs subdural drain after burr-hole drainage of chronic subdural hematoma: a randomized clinical trial (cSDH-Drain-Trial). Neurosurgery. 2019;85(5):E825–34.
35. Häni L, Vulcu S, Branca M, Fung C, Z'Graggen WJ, Murek M, et al. Subdural versus subgaleal drainage for chronic subdural hematomas: a post hoc analysis of the TOSCAN trial. J Neurosurg. 2019:1–9. https://doi.org/10.3171/2019.5.JNS19858.
36. Kim HC, Ko JH, Yoo DS, Lee S-K. Spontaneous resolution of chronic subdural hematoma: close observation as a treatment strategy. J Korean Neurosurg Soc. 2016;59(6):628–36.
37. Ban SP, Hwang G, Byoun HS, Kim T, Lee SU, Bang JS, et al. Middle meningeal artery embolization for chronic subdural hematoma. Radiology. 2018;286(3):992–9.
38. Kan P, Maragkos GA, Srivatsan A, Srinivasan V, Johnson J, Burkhardt J-K, et al. Middle meningeal artery embolization for chronic subdural hematoma: a multi-center experience of 154 consecutive embolizations. Neurosurgery. 2021;88(2):268–77.
39. Waqas M, Vakhari K, Weimer PV, Hashmi E, Davies JM, Siddiqui AH. Safety and effectiveness of embolization for chronic subdural hematoma: systematic review and case series. World Neurosurg. 2019;126:228–36.
40. Ng S, Derraz I, Boetto J, Dargazanli C, Poulen G, Gascou G, et al. Middle meningeal artery embolization as an adjuvant treatment to surgery for symptomatic chronic subdural hematoma: a pilot study assessing hematoma volume resorption. J Neurointerv Surg. 2020;12(7):695–9.
41. Sun TFD, Boet R, Poon WS. Non-surgical primary treatment of chronic subdural haematoma: Preliminary results of using dexamethasone. Br J Neurosurg. 2005;19(4):327–33.
42. Mehta V, Harward SC, Sankey EW, Nayar G, Codd PJ. Evidence based diagnosis and management of chronic subdural hematoma: A review of the literature. J Clin Neurosci. 2018;50:7–15.
43. Hutchinson PJ, Edlmann E, Bulters D, Zolnourian A, Holton P, Suttner N, et al. Trial of dexamethasone for chronic subdural hematoma. N Engl J Med. 2020;383(27):2616–27.
44. Ghajar J, Hariri RJ, Narayan RK, Iacono LA, Firlik K, Patterson RH. Survey of critical care management of comatose, head-injured patients in the United States. Crit Care Med. 1995;23(3):560–7.
45. Kageyama H, Toyooka T, Tsuzuki N, Oka K. Nonsurgical treatment of chronic subdural hematoma with tranexamic acid. J Neurosurg. 2013;119(2):332–7.
46. Weigel R, Hohenstein A, Schlickum L, Weiss C, Schilling L. Angiotensin converting enzyme inhibition for arterial hypertension reduces the risk of recurrence in patients with chronic subdural hematoma possibly by an antiangiogenic mechanism. Neurosurgery. 2007;61(4):783–8.
47. Kotwica Z, Brzeiński J. Epilepsy in chronic subdural haematoma. Acta Neurochir. 1991;113(3–4):118–20.
48. Ohno K, Maehara T, Ichimura K, Suzuki R, Hirakawa K, Monma S. Low incidence of seizures in patients with chronic subdural haematoma. J Neurol Neurosurg Psychiatry. 1993;56(11):1231–3.
49. Ferreri S, Roth MT, Casteel C, Demby KB, Blalock SJ. Methodology of an ongoing, randomized controlled trial to prevent falls through enhanced pharmaceutical care. Am J Geriatr Pharmacother. 2008;6(2):61–81.