

Chronic Subdural Hematoma ICU Management

Jeremy T. Ragland, MD, Kiwon Lee, MD*

KEYWORDS

• Chronic subdural hematoma • ICU • Cerebral spinal fluid • Anticoagulants • Antiplatelet agents

KEY POINTS

- A chronic subdural hematoma (cSDH) is a collection of old (>3 weeks) blood products and blood breakdown products that have accumulated in the subdural space, which is a potential space between the dura mater and the arachnoid layers of the meninges.
- cSDH is increasingly common due to the combination of more frequent use of anticoagulant and antiplatelet agents and the advanced age of many people in the population.
- The incidence of cSDH is estimated to be between 3.4 and 58 per 100,000 person-years, depending on the age of the population, with the average presenting age 63 years.
- The United Nations predicts that the percentage of the population above the age of 65 is expected to double between 2010 and 2050.
- cSDH is more prevalent in men, with a 3:1 male-to-female ratio.

CASE PRESENTATION

A 50-year-old man with a history of hypertension and alcohol abuse with prior withdrawal-related seizures presented to an outside hospital facility with confusion and headache after a witnessed fall. Further questioning revealed that he suffered a fall approximately 8 weeks prior to presentation. Since then he has been experiencing progressive dizziness and intermittent headaches. For the 10 days prior to presentation, he noticed progressive right arm weakness. On the day of presentation, after drinking a few beers, bystanders noted that he was confused and then suffered a fall from standing without head strike or loss of consciousness. The emergency medical service was activated, and he was taken to an outside hospital facility for evaluation. Initial vital signs were notable for no fever with blood pressure

152/105 mm Hg and a pulse of 102 beats per minute. He was awake and alert but confused. He was able to state his name and provide the history surrounding his fall but was unable to state his current location or the date. He was very combative. His right pupil was 6 mm and left pupil was 5 mm and both were reactive to light. He had pronator and downward drift in the right arm with decreased strength in all muscle groups. He had full strength in the left arm and both legs. His Glasgow Coma Scale (GCS) score was 14. His initial blood testing was notable for normal electrolytes, slightly elevated liver function tests, and mild anemia with normal platelet count and normal coagulation studies. CT scan of the head showed large leftsided cSDH (Fig. 1). He was immediately transported to the authors' facility for further management.

Departments of Neurosurgery and Neurology, Division of Neurocritical Care McGovern Medical School, 6431 Fannin Street, Medical School Building 7.152, University of Texas Health Science Center at Houston, Houston, TX 77030, USA

* Corresponding author. E-mail address: kiwon.lee@uth.tmc.edu

Neurosurg Clin N Am 28 (2017) 239–246 http://dx.doi.org/10.1016/j.nec.2016.11.006 1042-3680/17/© 2016 Elsevier Inc. All rights reserved.



Fig. 1. Patient's initial CT scan. Non-contrast-enhanced CT scan of the head showing an acute-on-chronic left subdural hematoma with a maximum thickness of 2.4 cm and 7 mm of left-to-right midline shift.

INTRODUCTION

A cSDH is a collection of old (>3 weeks) blood products and blood breakdown products that have accumulated in the subdural space, which is a potential space between the dura mater and the arachnoid layers of the meninges.¹ cSDH is increasingly common due to the combination of more frequent use of anticoagulant and antiplatelet agents and the advanced age of many people in the population.² The incidence of cSDH is estimated to be between 3.4 and 58 per 100,000 person-years, depending on the age of the population.^{1,3-6} The average presenting age is 63 years.⁶ The United Nations predicts that the percentage of the population above the age of 65 is expected to double between 2010 and 2050.² The US census reported that 12% of the population (or 35.9 million people) were over the age of 65 in 2003. This is expected to rise to approximately 20% of the population (or 72 million people) by the year 2030.⁷ The incidence of cSDH is expected to follow accordingly. In elderly individuals, cSDH has been identified as a sentinel event linked to underlying systemic pathology with 1-year mortality similar to that of a hip fracture.² cSDH is more prevalent in men, with a 3:1 male-to-female ratio.2,3,8,9 cSDH is bilateral in 20% to 25% of cases.⁶

cSDH occurs at the dural border cell layer, which is between the dura mater and arachnoid mater (**Fig. 2**).^{1,2,7} Generally, 2 predisposing factors must be present for a cSDH to develop: (1) decreased brain volume (eg, in elderly patients and patients abusing alcohol) and (2) previous head trauma.^{2,7} Experts believe there are several causes that lead to the formation of cSDH. The 2 most common are thought to be an acute subdural hematoma and a subdural hygroma (a collection of cerebral spinal fluid in the subdural space).^{1,2,6,7} In acute subdural hematoma, incomplete resorption of the blood products leads to the formation of cSDH. In both causes, a substantial inflammatory response infiltrates the subdural space. This inflammation includes angiogenic factors that lead to neovascularization. Although these friable capillaries are forming, they are subject to microhemorrhages, which result in cSDH. Many risk factors are thought to contribute to the formation of cSDH, including advanced age, history of falls, alcohol abuse (due to brain atrophy, increased fall risk, and coagulopathy), epilepsy, low intracranial pressure states, and hemodialysis.^{1,2} As discussed previously, there has been an increase in the use of anticoagulants and antiplatelet agents. This has led to a higher incidence of both atraumatic cSDH (whereby the antecedent trauma is so minor that it is not recalled) and recurrent cSDH.²

Some sources refer to cSDH as the "great imitator" given its heterogenous presenting symptoms and that its symptom onset can range from days to weeks prior to presentation.^{2,10} In a pooled cohort of 205 randomized controlled trial participants, presenting symptoms included gait disturbance and falls, cognitive decline, limb weakness, and acute confusion (Box 1).⁸ In the same cohort, a majority of patients presented with a GCS score 13 to 15 (81%) whereas 12% had a GCS score 9 to 12, and 7% had a GCS score less than or equal to 8.8 A diagnosis of cSDH is usually made using CT scan of the head, on which cSDH usually appears as a hypodense, crescentshaped collection along the convexity of the brain.^{1,2,7} There may be the presence of hyperdense or isodense material associated with the hypodense collection owing to the descriptive acute-on-chronic hematoma terms. and subacute-on-chronic subdural hematoma, respectively.¹¹



Fig. 2. Schematic of the ultrastructure of the meninges. SAS, subarachnoid space. (*Adapted from* Haines DED. On the question of a subdural space. Anat Rec 1991;230(1):3–21; with permission.)

PREOPERATIVE MANAGEMENT

Initial assessment of a patient with a cSDH should include evaluations of the airway, breathing, and circulation. Any acutely life-threatening issues should be appropriately managed. As a general rule, a GCS score of less than 8 warrants endotracheal intubation. A detailed history should be obtained with a focus on prior falls and head trauma as well as the use of antiplatelet or anticoagulant agents. A physical examination should be conducted, including a calculation of GCS score and evaluation for focal neurologic deficits. Initial laboratory evaluation should include chemistries and electrolytes, complete blood cell count, liver function tests, and coagulation studies. A noncontrast CT scan of the head should be completed to make a diagnosis of cSDH.^{1,2,7}

Reversal of Coagulopathies

Once the patient is stabilized, the focus should move to the management of any coagulopathies. The initial step is the cessation of any anticoagulant or antiplatelet agents. The next step is the decision to reverse any coagulopathy that may exist. One series of 88 patients showed that patients with cSDH who presented on anticoagulant medications experienced a significantly longer hospital stay (11 vs 7.5 days, P = .040), although the percentage with a good modified Rankin scale at discharge was not significantly different between the groups.⁷ General consensus is that patients with cSDH presenting on anticoagulant medications should undergo rapid reversal to prevent hematoma expansion and to allow for urgent neurosurgical intervention; this even applies to patients on anticoagulation for prosthetic heart valves.^{7,12} Oral warfarin, a vitamin K antagonist, is the most frequently used anticoagulant medication associated with cSDH.¹³ The vitamin K antagonists are reversed using prothrombin complex concentrate (PCC), fresh frozen plasma (FFP), and vitamin K.^{14,15} Traditionally, FFP has been the first-line therapy in the reversal of vitamin K antagonists. Its use, however, can predispose to additional medical complications given its large volume of infusion.^{7,15,16} Alternatively, PCC can be infused over a few minutes and has a volume exponentially smaller than FFP.7,15,17 With both PCC

Box 1 Presenting symptoms

- Acute confusion
- Aphasia
- Collapse
- Drowsiness or coma
- Falls
- Gait disturbance
- Headache
- Incontinence
- Limb weakness
- Mental deterioration
- Seizure
- Speech impairment
- Status epilepticus
- Visual disturbance
- Vomiting

Adapted from Santarius T, Kirkpatrick PJ, Ganesan D, et al. Use of drains versus no drains after burr-hole evacuation of chronic subdural haematoma: a randomised controlled trial. Lancet 2009;374(9695):1071; with permission.

and FFP, vitamin K should be used an adjunctive therapy to facilitate the liver in producing new clotting factors.¹⁵ For cases in which a more gradual reversal technique is acceptable, vitamin K can be used as monotherapy.¹² Most institutions have guidelines on the use of these agents. The clinical situation and the acuity of any necessary surgical intervention guide the decision on which agents to use.

Novel oral anticoagulants (NOACs) include factor Xa inhibitors and direct thrombin inhibitors. There is limited experience with the reversal of these agents. The best antidote for NOACs is time in the absence of life-threatening acute hematoma expansion or the need for emergent surgical intervention. The Neurocritical Care Society guidelines suggest using activated charcoal and PCC for NOAC reversal, but this is based on lowquality evidence and this decision should be made with input from a hematologist.¹⁵ The direct thrombin inhibitor dabigatran now has a Food and Drug Administration-approved antidote, idarucizumab. It is suggested that the reversal of dabigatran involve activated charcoal, hemodialysis, and PCC (all low-quality evidence) and idarucizumab (moderate-quality evidence).¹⁵ Laboratory assays have been developed that can aid in assessing the anticoagulant effect of NOACs. Diluted thrombin time for direct thrombin inhibitors and factor Xa levels for factor Xa inhibitors are inconsistently available and the extent of their clinical use is not fully established.¹⁸

For antiplatelet therapy, it has been established that 7 to 10 days are necessary to allow for the production of fully functional platelets to replace the inhibited ones in circulation.¹⁹ When emergent surgical intervention is necessary, platelet transfusion is recommended to acutely reverse the effects of antiplatelet agents although there is scant evidence to support this.²⁰ Some centers combine platelet transfusion with a single dose of desmopressin, but this too is based on minimal evidence.²¹

Mannitol and Hypertonic Saline

Mannitol and hypertonic saline are agents used to create an osmotic gradient between the brain and the plasma, ultimately removing free water from the brain in an effort to decrease the intracranial pressure. In acute subdural hematoma, there is a theoretic concern of SDH expansion with the use of hypertonic and hyperosmotic agents because it is thought that the full brain has a tamponade effect on the SDH. Despite this theoretic concern, the temporary use of hypertonic and hyperosmotic agents in patients actively experiencing cerebral herniation or impending herniation should be advocated as a temporizing measure prior to surgical intervention.

Seizure Prophylaxis

Only a few studies have examined the rate of seizures in patients with cSDH.²²⁻²⁵ The preoperative incidence of seizures ranged from 2% to 19%; postoperative seizure incidence was 1% to 23%.^{2,7} Two studies reported no significant difference in the seizure rate related to the use of prophylactic anticonvulsive medications (ACMs).7,23,24 The investigators concluded that there was no benefit in using prophylactic ACM except in patients at high risk of seizures, such as patients abusing alcohol.^{23,24} A third study showed that 2.4% of patients treated with therapeutic prophylactic ACMs experienced a seizures compared with 32% of patients who were not adequately treated with prophylactic ACMs.²⁵ The investigators concluded that this significantly increased the morbidity and mortality for the patients who experienced new-onset seizures and therefore recommended the use of prophylactic ACMs in cSDH.²⁵ A fourth study found that preoperative administration of prophylactic ACMs was the only independent predictor of decreased incidence of postoperative seizures.²² Despite this,

there was no effect on discharge outcomes, suggesting that, if given, prophylactic ACMs should be administered prior to surgical intervention.⁷ Given these mixed results, practices vary in their use of prophylactic ACMs. The authors' practice is to treat with seizure prophylaxis for 7 days, especially in patients who are undergoing surgical intervention or have an increased risk of seizures, such as alcoholics or those with significant underlying traumatic brain injury.

Corticosteroids

Because inflammation plays a role in the pathophysiology of cSDH, multiple studies have examined the use of corticosteroids as monotherapy to avoid surgery and in combination to surgical intervention.26-30 Medical management of cSDH is discussed in a See Jan Claassen's article, "Chronic Subdural Medical Management", in this issue, so monotherapy is not discussed in this article. European studies have shown the use of preoperative and postoperative steroids reduces the rate of cSDH recurrence after surgical treatment and decreases 6-month mortality.^{2,28} An additional retrospective study suggests that local application of methylprednisolone into the hematoma cavity at the time of surgery may reduce hematoma recurrence.³¹ The evidence is insufficient, however, to draw any significant conclusions, and further study with randomized controlled trials should be conducted. It is the authors' practice not to use steroids as monotherapy or as adjunctive therapy to surgical intervention in the treatment of cSDH. The use of corticosteroids in the treatment of cSDH is generally not recommended due to lack of evidence.

Indication for Surgical Intervention

Clinical presentation and radiographic appearance both influence the decision for surgical intervention. A small cSDH in an asymptomatic patient is best managed with clinical and radiographic observation in a closely monitored setting.^{1,7} Should a patient develop significant neurologic symptoms, however, surgical intervention is advised.^{1,2} Although the radiographic size of the cSDH plays a role in the decision for surgery, there is no absolute size greater than which a surgical intervention is mandated.^{1,7} The spontaneous resolution of cSDHs of significant size has been reported in a few case series.^{1,7,32,33} The investigators described these patients as advanced in age (>70 years) with significant cerebral atrophy and no clinical or radiographic evidence of increased intracranial pressure.32,33

Management of asymptomatic patients with cSDHs large enough to cause brain compression and/or midline shift remains a controversial topic. Despite the lack of evidence to establish a size cutoff, it is generally accepted that hematoma volume with a maximum thickness greater than 1 cm, or greater than the thickness of the skull, should undergo surgical evacuation.^{1,6}

Patients with neurologic symptoms that can be attributed to a radiographically proved cSDH warrant immediate surgical evacuation. These symptoms are dictated by the location of the cSDH but can include aphasia, neglect, contralateral hemiparesis, and partial seizures. For patients with a large cSDH causing mass effect on the left frontotemporal convexity, it is not uncommon to observe a profound aphasia; some of these patients have severe global aphasia with or without contralateral hemiparesis. An isolated aphasia with intact level of alertness is often due to the chemical irritation of the dominant hemisphere and does not necessarily mean the patient is experiencing a stroke or status epilepticus. Although it is prudent to rule out acute ischemic stroke and seizures for anyone with acute aphasia, such a symptom is frequently seen with dominant hemispheric convexity subdural hematomas whether acute or chronic. Large hematomas, or those in a frontotemporal location, can lead to brainstem compression resulting in anisocoria, coma, and death. The presence of these symptoms associated with a cSDH necessitates emergent surgical intervention. Surgical options include burr hole craniostomy, twist drill craniostomy, and craniotomy, all of which are discussed in See William Mack's article, "Minimally Invasive Surgical Approaches for Chronic Subdural Hematomas and See Louis Kim's article, Craniotomy for treatment of chronic subdural hematoma", in this issue.

POSTOPERATIVE MANAGEMENT Postoperative Imaging

The timing and frequency of postoperative CT scans in cSDH are topics of controversy. Practitioners must weigh the advantage of daily CT scans (objective monitoring of the cSDH drainage) versus the disadvantages (radiation exposure to patients and health care costs). One study used early (within 48 hours) postoperative CT to predict outcome at discharge.³⁴ Because it found that (1) the only predictive factor of postoperative SDH volume was preoperative SDH volume and (2) early postoperative SDH volume on CT did not correlate with outcome at discharge, the investigators concluded that routine postoperative CT scan

may be unnecessary.³⁴ In the authors' practice, a CT scan of the head is completed 24 hours after operative intervention. A second routine CT scan is performed just prior to planned removal of the subdural drain unless the patient has a clinical change or there is an issue with the drainage catheter output. Obtaining daily scans for a patient who is neurologically doing well is of low yield and generally not recommended.

Mobilization

The timing of mobilization after surgical treatment of cSDH remains a controversial issue. Although early mobilization decreases the risk of medical complications, its benefit has to be weighed against the risk of recurrent cSDH, which is thought to be prevented by bed rest.⁷ Practices vary with regards to early mobilization versus prolonged bed rest. One study using historical controls showed significantly fewer medical complications with early mobilization (postoperative day 1) compared with delayed mobilization (postoperative day 3) although there was no difference in the rate of recurrence.³⁵ A second study also found no significant difference in the rate of recurrence between patients who remained recumbent for 3 days postoperatively compared with those who assumed a sitting position on postoperative day 1.36 Alternatively, a third study examined elevating the head of bed to 30° to 40° compared with strict supine position in the first 3 postoperative days and showed a significant reduction in the rate of recurrent cSDH in the strict supine group (19% vs 2.3%).37 There was no significant difference in other position-related medical complications, including pneumonia, atelectasis, deep vein thrombosis, and decubitus ulcers.³⁷

Venous Thromboembolism Prophylaxis

Patients with cSDH are at risk for complications related to venous thromboembolism (VTE). Unless contraindicated, all patients should have intermittent pneumatic compression devices placed on their legs. There is controversy surrounding the use of chemical VTE prophylaxis. One study showed an increase in recurrence of cSDH after the use of a prophylactic dose of low-molecularweight heparin (18.8% vs 32.1%) although it is unclear from the study on what postoperative day the prophylaxis was started.38 A separate institution recommends starting chemical VTE prophylaxis only after the closed drainage system has been removed and there is a follow-up CT scan showing no acute or recurrent hemorrhage.7 Despite this, many ICUs routinely start chemical VTE prophylaxis 24 to 72 hours after surgical intervention.² More studies are needed to examine the ideal time to start chemical VTE prophylaxis after surgery and to compare the risks and benefits of low-molecular-weight heparin with unfractionated heparin. It is reasonable to use chemical VTE prophylaxis starting 24 hours after operative intervention for cSDH in stable patients.

Resuming Antiplatelet and Anticoagulant Agents

Many patients with cSDH are also taking antiplatelet and anticoagulant agents. Because these agents are generally taken to prevent heart disease and ischemic stroke, there is pressure on practitioners to restart these medications as early as safely possible. There are varying opinions on not only when to restart these medications but also whether the doses should be adjusted on restarting. Currently these decisions are made on a patient-by-patient basis.² Variables that should be considered are patient age, indication for antiplatelet or anticoagulant agents, risk for future falls and head trauma, and underlying cause of cSDH. Comparison between CHA2DS2-VASc score and HAS-BLED score can provide some objective data to aid in the decision of restarting warfarin in patients with atrial fibrillation.³⁹⁻⁴¹ The CHA2DS2-VASc score evaluates the risk for cerebral thromboembolic events in patients with atrial fibrillation, and the HAS-BLED score estimates the risk of major bleeding in patients on anticoagulation for atrial fibrillation.40,41 Limited data suggest safety in restarting warfarin 72 hours after surgical intervention. There are sparse data when it comes to the use of NOACs.

Case Summary

The authors' patient was admitted to the neuroscience ICU. Valproic acid was started for seizure prophylaxis. The cSDH was managed with burr hole craniostomy and closed drainage system completed in the operating room. His postoperative course was complicated by alcohol withdrawal requiring high doses of benzodiazepines. After adequate drainage, the subdural drain was removed. He was discharged to a rehabilitation facility after recovering from alcohol withdrawal.

REFERENCES

 Soleman J, Taussky P, Fandino J, et al. Evidencebased treatment of chronic subdural hematoma. In: Sadaka F, editor. Traumatic brain injury. Rijeka(Croatia); 2014. p. 249–81.

- Kolias AG. Chronic subdural haematoma: modern management and emerging therapies. Nat Rev Neurol 2014;10(10):570–8.
- Cousseau DH. Chronic and subacute subdural haematoma. An epidemiological study in a captive population. Rev Neurol 2001;32(9):821–4 [in Spanish].
- Asghar MM. Chronic subdural haematoma in the elderly-a North Wales experience. J R Soc Med 2002;95(6):290-2.
- Kudo HH. Chronic subdural hematoma in elderly people: present status on Awaji Island and epidemiological prospect. Neurol Med Chir (Tokyo) 1992; 32(4):207–9.
- Greenberg M. Chronic subdural hematoma. Handbook of neurosurgery. 3rd edition. New York: Thieme; 2010. p. 899–902.
- Ducruet AF. The surgical management of chronic subdural hematoma. Neurosurg Rev 2012;35(2):155–69.
- Santarius T, Kirkpatrick PJ, Ganesan D, et al. Use of drains versus no drains after burr-hole evacuation of chronic subdural haematoma: a randomised controlled trial. Lancet 2009;374(9695):1067–73.
- Gelabert-González M. Chronic subdural haematoma: surgical treatment and outcome in 1000 cases. Clin Neurol Neurosurg 2005;107(3):223–9.
- 10. Potter JF. Chronic subdural hematoma-the "great imitator". Geriatrics 1977;32(6):61-6.
- Yousem DM. Head trauma. Neuroradiology: the requisites. 3rd edition. Philadelphia: Elsevier Health Sciences; 2010. p. 173–6.
- 12. Hanley JP. Warfarin reversal. J Clin Pathol 2004; 57(11):1132–9.
- Ansell J. The pharmacology and management of the vitamin K antagonists. Chest 2004;126(3):204S–33S.
- Cartmill MM. Prothrombin complex concentrate for oral anticoagulant reversal in neurosurgical emergencies. Br J Neurosurg 2000;14(5):458–61.
- 15. Frontera JA. Guideline for reversal of antithrombotics in intracranial hemorrhage a statement for healthcare professionals from the neurocritical care society and society of critical care medicine. Neurocrit Care 2015;24(1):6–46.
- Lankiewicz MW. Urgent reversal of warfarin with prothrombin complex concentrate. J Thromb Haemost 2006;4(5):967–70.
- Vigué B. Ultra-rapid management of oral anticoagulant therapy-related surgical intracranial hemorrhage. Intensive Care Med 2007;33(4):721–5.
- Heidbuchel H. Updated European Heart Rhythm Association practical guide on the use of non-vitamin-K antagonist anticoagulants in patients with nonvalvular atrial fibrillation: executive summary. Eur Heart J 2016;17:1467–507.
- Wada M. Influence of antiplatelet therapy on postoperative recurrence of chronic subdural hematoma: a multicenter retrospective study in 719 patients. Clin Neurol Neurosurg 2014;120:49–54.

- Handin RI. Hemostatic effectiveness of platelets stored at 22°C. N Engl J Med 1971;285(10):538–43.
- Ranucci M. Platelet mapping and desmopressin reversal of platelet inhibition during emergency carotid endarterectomy. J Cardiothorac Vasc Anesth 2007;21(6):851–4.
- Grobelny BT. Preoperative antiepileptic drug administration and the incidence of postoperative seizures following bur hole-treated chronic subdural hematoma Clinical article. J Neurosurg 2009;111(6): 1257–62.
- Ohno KK. Low incidence of seizures in patients with chronic subdural haematoma. J Neurol Neurosurg And Psychiatry 1993;56(11):1231–3.
- Rubin GG. Epilepsy in chronic subdural haematoma. Acta Neurochir (Wien) 1993;123(1–2):39–42.
- 25. Sabo RA. Chronic subdural hematomas and seizures: the role of prophylactic anticonvulsive medication. Surg Neurol 1995;43(6):579–82.
- Frati A. Inflammation markers and risk factors for recurrence in 35 patients with a posttraumatic chronic subdural hematoma: a prospective study. J Neurosurg 2004;100(1):24–32.
- 27. Santarius TT. Working toward rational and evidencebased treatment of chronic subdural hematoma. Clin Neurosurg 2010;57:112–22.
- Berghauser Pont LM, Dammers R, Schouten JW, et al. Clinical factors associated with outcome in chronic subdural hematoma: a retrospective cohort study of patients on preoperative corticosteroid therapy. Neurosurgery 2012;70(4):873–80.
- Sun TF, 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.
- Berghauser Pont LM, Dirven CM, Dippel DW. The role of corticosteroids in the management of chronic subdural hematoma: a systematic review. Eur J Neurol 2012;19(11):1397–403.
- Xu X. Local application of corticosteroids combined with surgery for the treatment of chronic subdural hematoma. Turk Neurosurg 2013;25(2):252–5.
- Göksu EE. Spontaneous resolution of a large chronic subdural hematoma: a case report and review of the literature. Ulusal Travma Acil Cerrahi Derg 2009; 15(1):95–8.
- Parlato CC. Spontaneous resolution of chronic subdural hematoma. Surg Neurol 2000;53(4):312–5 [discussion: 315–7].
- Ng HY. Value of routine early post-operative computed tomography in determining short-term functional outcome after drainage of chronic subdural hematoma: an evaluation of residual volume. Surg Neurol Int 2014;5(1):136.
- Kurabe S. Efficacy and safety of postoperative early mobilization for chronic subdural hematoma in elderly patients. Acta Neurochir (Wien) 2010;152(7):1171–4.

Ragland & Lee

- Nakajima HH. The role of postoperative patient posture in the recurrence of chronic subdural hematoma: a prospective randomized trial. Surg Neurol 2002;58(6):385–7 [discussion: 387].
- **37.** Abouzari M. The role of postoperative patient posture in the recurrence of traumatic chronic subdural hematoma after burr-hole surgery. Neurosurgery 2007;61(4):794–7.
- Tahsim-Oglou Y. Factors predicting recurrence of chronic subdural haematoma: the influence of intraoperative irrigation and low-molecular-weight heparin thromboprophylaxis. Acta Neurochir (Wien) 2012;154(6):1063–8.
- Chari A. Recommencement of anticoagulation in chronic subdural haematoma: a systematic review and meta-analysis. Br J Neurosurg 2014; 28(1):2–7.
- 40. Olesen JB. Risks of thromboembolism and bleeding with thromboprophylaxis in patients with atrial fibrillation: a net clinical benefit analysis using a 'real world' nationwide cohort study. Thromb Haemost 2011;106(4):739–49.
- **41.** Pisters RA. Novel user-friendly score (HAS-BLED) to assess 1-year risk of major bleeding in patients with atrial fibrillation. Chest 2010;138(5):1093–100.