



Advances in chronic subdural haematoma management: beyond the burr hole

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Chronic subdural haematoma is associated with increased frailty, comorbidities including dementia, and a higher risk of mortality compared to age-matched controls, with incidence rising as populations age. Burr holes have long been considered the mainstay of surgical treatment for chronic subdural haematoma, but novel therapies are seeking to expand treatment beyond surgical burr hole drainage. Since 2020, several high-quality clinical trials have helped refine operative strategies, provided robust evidence against the routine use of steroids, and investigated minimally invasive treatment with middle meningeal artery embolisation. One treatment approach does not fit all patients, and stratification using risk factors for poor outcome might prove crucial in maximising benefit from adjuvant therapies such as middle meningeal artery embolisation. Results of further trials of middle meningeal artery embolisation and drug trials are awaited, and increased recognition of the need for multispecialist care should also help to improve outcomes for patients with chronic subdural haematoma in the future.

Introduction

Chronic subdural haematoma (CSDH) has become a key clinical concern worldwide, both due to its growing incidence in ageing populations and due to changes in understanding of the disease and wide application of new therapies with varied evidence. CSDH can be defined as a collection of old blood and fluid within the subdural space, overlying the brain and encapsulated within a membrane. It is associated with a higher long-term mortality compared with controls (6–9% excess-mortality at 1 year and 18% at 10 years), and with increased level of frailty and comorbidity, particularly dementia.^{1,2,3} Mortality is higher compared with controls in both surgically and conservatively managed patients, and minimising repeat surgery for recurrent CSDH reduces mortality.^{2,4}

The aim of this Review is to highlight areas of new knowledge and practice relating to CSDH that have emerged in the past 5 years, and to identify the questions that remain in this fast-moving area. We explore the clinical decision pathway in CSDH, including diagnosis and recurrence, surgical strategies, middle meningeal artery embolisation (MMAE), pharmacotherapies, and conservative management. We explore challenges and

advances in care within each management approach and discuss future directions of care.

CSDH diagnosis and recurrence

CSDH is diagnosed on imaging, usually CT, appearing as a crescent-shaped collection of old blood and fluid overlying the brain surface (ie, the outer wall of the haematoma is convex and the inner wall concave) but can expand to be planoconvex (ie, the inner wall is straight) or even biconvex. Small CSDHs can be difficult to differentiate from a hygroma (a collection of CSF), but often CSDHs contain components of new bleeding and membranes (figure 1).

Many CSDHs are found incidentally on scans after falls in older adults. When symptomatic, features can include headache, confusion, gait unsteadiness, lateralising weakness, and eventual coma, and can mimic a slowly evolving stroke. The pathogenesis of CSDH can involve an overt or covert traumatic injury to dural-border cells overlying the brain, with consequential dysregulated inflammation and angiogenesis driving CSDH accumulation.^{5,6} Incidence is increasing worldwide, with consequent societal impact through increased hospitalisation and resource use, and ultimately increased patient frailty and death over time (panel 1).

A crucial factor in CSDH recovery is symptomatic recurrence, usually within 3 months of the initial CSDH and arising from re-accumulation of blood and fluid within the same space. Retained CSDH fluid, membranes, air, or a combination thereof accumulate postoperatively, secondary to the atrophic brain in older adults being unable to re-expand, resulting in an ongoing stimulus for inflammation and CSDH recurrence.^{22–25} The role of antithrombotic medications in promoting re-bleeding is debated (panel 2).

Recurrence indicates the need for further treatment and longer hospital stay and is linked with higher mortality and worse outcome than non-recurrent CSDH.³⁷ One systematic review reported a 12%

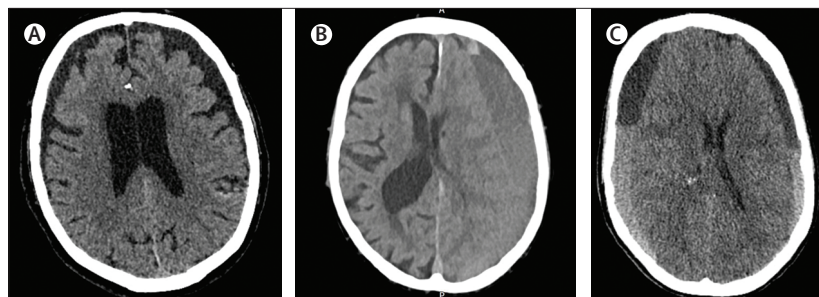


Figure 1: CT imaging examples of subdural hygroma compared with different subdural haematomas (A) Bilateral subdural hygroma, (B) left hemisphere chronic subdural haematoma with underlying mass effect and planoconvex shape (straight inner wall with convex outer wall), and (C) bilateral chronic subdural haematoma, crescentic in shape and containing separated acute blood.

recurrence rate influenced by patient factors (eg, older age and multiple comorbidities), radiological factors (eg, increased haematoma size and midline shift and density), and surgical practices.³⁸ Large population observational studies over the past 20 years in Italy and South Korea show similar recurrence rates as the systematic review (10–11%).^{39,40} Randomised controlled trials have reported postoperative recurrence rates of 7% (UK), 8–12% (Switzerland), and 13–14% (Denmark).^{15,16,41}

The search for a biomarker to help predict the progression of CSDH, its response to treatment, and the risk of recurrence is a key area of ongoing research. Reviews of observational studies have identified potential inflammatory markers in CSDH fluid linked to recurrence, including IL-6, vascular endothelial growth factor (VEGF), and fibrinolytic markers.^{5,42} The reduction of VEGF in early postoperative CSDH drain fluid in patients treated with burr holes and dexamethasone indicates that inflammatory activity in the early postoperative period might influence recurrence.⁴³ To provide individualised therapy targeting inflammatory mechanisms, both an accurate marker for recurrence and a reliable intraoperative test for said biomarker are needed. Point-of-care tests have been successful in other specialties, such as IL-6 testing for inflammation in preterm labour amniotic fluid, and are, therefore, potentially feasible.⁴⁴ A recently published trial⁴⁵ assessed the role of postoperative subcutaneous tocilizumab (monoclonal antibody to IL6), reporting a 6-month re-operative rate for recurrence of 0% with tocilizumab combined with tranexamic acid compared with 0.8% with tranexamic acid only and 6.3% with surgery alone. Although tocilizumab also showed higher rates of haematoma resolution on imaging, compared with the other two groups, the study was not powered for efficacy or safety. There were more infections in the tocilizumab arm; a known risk with monoclonal antibodies. Therefore, IL-6 biomarker targeted therapy could be preferable. Anti-VEGF therapies are established in clinical use for retinal diseases, paving the way for studying their applicability in CSDH.⁴⁶ A standardised point-of-care test for markers such as IL-6 and VEGF could be incorporated into clinical trials to aid understanding of treatment effects.

Early recognition of CSDH recurrence is important but the CSDH must be symptomatic and distinguished from asymptomatic residual CSDH. Even large residual CSDH on imaging can resolve with time and therefore recurrence should not be diagnosed on the basis of radiological findings alone. The TOSCAN study randomly assigned 361 patients to a CT group (routine postoperative scanning at 2 and 30 days) versus a no CT group (scanned only when clinically indicated);⁴⁷ the primary outcome of survival without severe disability (modified Rankin scale [mRS] score 0–3) was not improved in the CT group, and routine scanning led to a higher rate of surgical intervention than in the no CT

Panel 1: Epidemiology of chronic subdural haematoma

The incidence of chronic subdural haematoma (CSDH) is rising, primarily due to a change in population demography, with a substantial growth of the older adult population secondary to increased life expectancy.^{7–10} Worldwide, the proportion of people older than 60 years is expected to rise from 12% to 22% between 2015 and 2050.¹¹ CSDH is more prevalent in older adults (mean age 76 years at diagnosis). Finnish studies report rising incidence risk, from 7.2–12.2 per 100 000 people per year in the 1990s to 13.4–16.5 per 100 000 people per year in 2011–15.^{8,9} Similarly, incidence rose from 26.4 per 100 000 per year in 2003 to 58 per 100 000 per year in 2016 in the USA.^{11,12} Incidence also increases with age (particularly those older than 75 years), although this increase is likely to be skewed by increased use of neuroimaging identifying small CSDHs that do not require treatment.^{8,9,12} A UK study reported an increased incidence of operated CSDH from 3.5 per 100 000 per year in 2015 to 10–18 per 100 000 per year in 2040, which, when correlated to population growth statistics, results in a projected incidence growth of 53%.⁷

CSDH has a male preponderance (approximately 65% of cases are in men), which becomes more pronounced with age.^{7,9,13} Trauma (usually falls) is a prominent risk factor for CSDH, occurring in around 60% of patients, between a week and several months before diagnosis.^{9,14} Antithrombotic medications are a risk factor for CSDH, used by up to 50% of patients requiring surgery, with a 3:2 ratio of antiplatelets to anticoagulants.^{15–17} A large retrospective series reported significantly lower odds of large CSDH (>2 cm depth) and midline shift with direct oral anticoagulants compared with warfarin, suggesting their improved safety profile.¹³

Knowledge gaps surround CSDH in low-income and middle income countries, owing to a scarcity of published data and challenges understanding incidence and outcomes due in part to population movement. From the data available, patients are younger in low-income and middle-income countries than in high-income countries (mean age 54–59 years from reports in Ethiopia, Pakistan, and Mexico), still with a male preponderance and often traumatic causes.^{18–21} The younger age reflects the mean ages of these populations and patients of this age in any population are less likely to take antithrombotic medications than older patients, the use of which can reduce incidence of CSDH. Age is relevant as CSDH recurrence rates appear to be low in low-income and middle income countries (0–8%) despite not using subdural drains, probably owing to better re-expansion of the younger brain after surgery.^{18–21}

group (rate ratio [RR] 1.50; 95% CI 0.99–2.32).⁴⁷ Routine scanning therefore potentially encourages early, and perhaps inappropriate, intervention for residual fluid collections that might otherwise resolve. Resolution might also occur more slowly in older patients (age >80 years), with one observational study showing a mean of 3.7 months versus 2.8 months (in patients age <80 years) for CSDH resolution.⁴⁸ An international Delphi study due to be published this year, is working towards a standardised definition of recurrence.⁴⁹

Surgical treatment for CSDH

Multiple CSDH treatment strategies are available, depending on patient symptoms, background medical health, and preferences of the clinician and patient (figure 2). Typically, the first decision is to determine whether there is mass effect from a CSDH, for which surgery is largely considered the first-line treatment. In line with the patient journey, we discuss technical aspects of anaesthetic, surgical, and postoperative care;

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Panel 2: Antithrombotic medications

Antithrombotic medication, both antiplatelet drugs and oral anticoagulants, are commonly taken by patients with CSDH, owing to underlying atrial fibrillation and other comorbidities associated with older age. Whether these agents should be withdrawn on diagnosis before surgical intervention, as well as when to restart them postoperatively, is uncertain. Some large cohort studies have suggested that preoperative use or early recommencement of antithrombotic drugs postoperatively does not influence CSDH recurrence or poor outcome.^{26–29} However, a 2025 meta-analysis on 44 observational studies reported higher rates of CSDH recurrence for patients on oral anticoagulants (odds ratio [OR] 1.41, 95% CI 1.11–1.79) and antiplatelets (1.31, 1.08–1.58) with a higher rate of thromboembolic events across all patients receiving antithrombotic therapy compared with no such therapy (3.74, 1.08–1.58).³⁰ A balance needs to be struck between starting antithrombotic therapy at a safe interval following surgery to minimise bleeding and recurrence and waiting too long, leading to thrombotic events. Recommencement 7–14 days postoperatively has been suggested, but more robust research is needed.^{28,31,32} Low-dose aspirin is also continued despite burr hole surgery in some institutions, and a 2025 Swiss trial randomised 155 patients with CSDH to continuous aspirin (100 mg) or placebo, for 12 days from diagnosis of CSDH, with intervening burr hole surgery.³³ The primary outcome was an operation for recurrent CSDH at 6 months and no significant difference was found between groups, with 13.9% recurrence within the aspirin group compared with 9.5% recurrence within the placebo group (risk difference 4.4%, 95% CI 7.2–15.9%).³³ The thrombotic event rates were also similar between groups. These results suggest that aspirin continuation might not increase recurrence, and the trial authors recommend that bleeding and thrombotic risk are evaluated on an individual basis rather than blanket discontinuation of aspirin. From these data, the use of platelet transfusions in patients on aspirin might also seem unnecessary, although they are often given before emergency surgery despite a scarcity of evidence for benefit.³⁴ However, one study has reported that a low platelet count ($<157 \times 10^9/L$), independent of antiplatelet use, might be a risk factor for recurrence.^{35,36}

20 ongoing trials are registered in anaesthetic, surgical, and postoperative care (appendix pp 1–3).

See Online for appendix

Anaesthesia

Debate continues around optimal anaesthetic techniques during CSDH evacuation, with both general anaesthesia and local anaesthesia with sedation used in different institutions. Areas of discussion include the effect of anaesthetic regimen on outcome and the risks of general anaesthesia in an older population with associated comorbidities. Three systematic reviews on anaesthetic techniques incorporate only single-centre randomised controlled trials or retrospective studies.^{69–71} Local anaesthesia with sedation seems to be associated with shortened operative time, shortened length of hospital stay, and fewer post-operative complications,^{69–71} compared with general anaesthesia but these conclusions are based on unadjusted observational data. One randomised trial showed no significant effect of the use of local anaesthesia with sedation on readiness for discharge, but the trial had a small sample size ($n=60$) and large estimated effect (a projected 2-day reduction in length of stay), probably leading to low power.⁷² No difference between local anaesthesia with sedation and general anaesthesia was reported in a multicentre retrospective study using

propensity matching for patients treated with the minimally invasive MMAE.⁷³ Anaesthetic techniques are heterogeneous across centres, making trials difficult to design and compare, and determining the best outcome measure for such trials is challenging owing to concerns that patient-tailored care might be more important than choice of anaesthetic.⁷⁴

Surgical approach

One or two burr holes have long been considered the mainstay of surgical treatment for CSDH. Two other common surgical approaches are twist-drill craniostomy and mini-craniotomy. A randomised trial comparing these three techniques across 245 patients found that all were effective with a non-significant ($p=0.07$) reduced recurrence rate associated with burr holes (7.6%) compared with mini-craniotomy (13.1%) and twist-drill craniotomy (19.5%).⁵⁷ A meta-analysis of ten studies supports lower operative recurrence with two burr holes compared with twist-drill craniostomy (RR 0.48, 95% CI 0.24–0.94).⁷⁵

A meta-analysis of 16 studies reported lower operative recurrence with burr holes compared with twist-drill craniostomy without suction (odds ratio [OR] 1.62, 95% CI 1.08–2.42), although not with application of suction.⁷⁶ Twist-drill craniostomy (and similar subdural-evacuating port systems) is helpful as a bedside procedure for high-risk patients or if resources are scarce.⁷⁷ Neuroendoscopic-assisted burr hole drainage was reported to lower recurrence rates compared with standard burr holes (3.1% vs 13.8%; $p<0.0001$), but with longer operative times and no difference in mortality or outcomes at 6 months in meta-analysis of six observational studies.⁷⁸

Irrigation

The multicentre FINISH study randomly assigned 589 patients to two groups: a single burr hole with irrigation and a single burr hole without irrigation, both with 48 h of postoperative subdural drainage.⁵⁸ Although the study did not show non-inferiority of no irrigation, the authors recommend irrigation on the basis of a 6% reduction (95% CI 0.2–11.7) in the 6-month recurrence rate with irrigation (12.6% with irrigation vs 18.3% without irrigation).⁵⁸ Volume of irrigation fluid has not been shown to influence recurrence, with similar recurrence rates in an observational study of 525 patients (14.1% recurrence rate) using a mean irrigation volume of 852–938 mL compared with the FINISH study with mean irrigation volume of 400 mL (range 220–540 mL).^{58,79} Body-temperature irrigation significantly reduced recurrence compared with room-temperature irrigation (6% vs 14%) in a randomised trial of 541 patients.¹⁷

Drains

Subdural drains in CSDH surgery are widely used because of their association with reduced recurrence

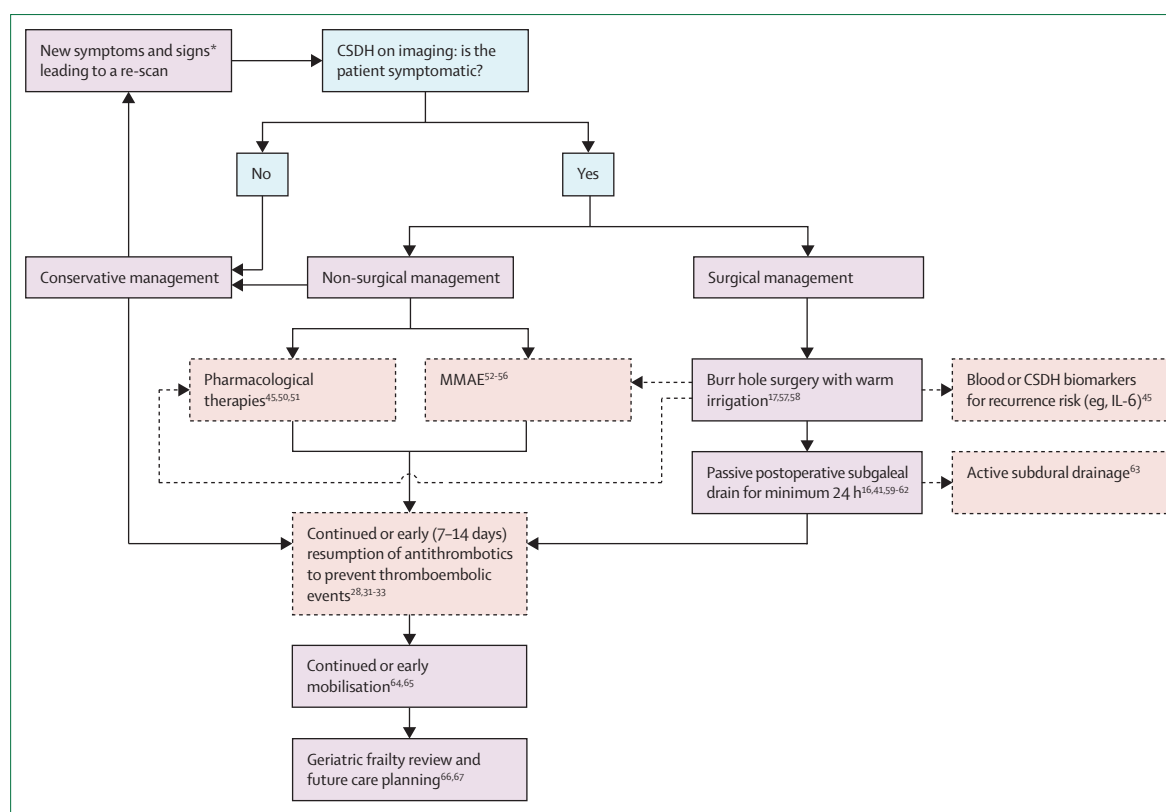


Figure 2: Current and potential future approaches to management of chronic subdural haematoma

The figure represents the opinions of the authors. Blue boxes show clinician decisions, purple boxes show standard of care,^{66,68} and orange boxes show areas of emerging research, where evidence is insufficient (due to either conflicting data or a scarcity of robust clinical trial or real-world evidence of efficacy) to support incorporation into standard-of-care, as discussed in this Review. Solid lines show main pathway options and dotted lines show additional potential future options in patients who have had surgery. Non-surgical refers to patients for whom an alternative to surgery is considered preferable owing to patient factors such as frailty and comorbidities, patients who decline surgery, or patients with symptoms that are too mild for surgery to be appropriate. CSDH=chronic subdural haematoma. MMAE=middle meningeal artery embolisation. *Symptoms and signs include weakness, unsteadiness, speech disturbance, confusion, and progressive headache.

(9.3% subdural drain vs 24% no drain, $p=0.0031$) and associated mortality.³⁷ These benefits have been validated in a meta-analysis of ten studies (comprising 1961 patients), in which subdural drains reduced recurrence (OR 0.39, 95% CI 0.28–0.55), mortality (0.65, 0.43–0.97), and gross focal neurological deficit (0.58, 0.37–0.89) compared with no drain.³⁹ However, subdural drains pose a risk of injury to the brain during blind placement through a burr hole and instead can overlie the burr hole in the subgaleal (also called subperiosteal) space.

In a single-centre review of 290 subdural drains, 17 (6%) were misplaced within the brain cortex, five of which caused haemorrhage and nine caused new neurological symptoms, compared with no complications in 170 subgaleal drains.⁶⁰ A non-inferiority randomised trial of 220 patients was underpowered but reported lower recurrence rates (8% vs 12%) and reduced complications with subgaleal drains compared with subdural drains.⁴¹ Two further observational studies reported no significant increase in recurrence or worse outcome with subgaleal drains compared with subdural drains, but fewer

complications.^{61,62} Although a meta-analysis of 15 studies reported no differences in outcomes between these two drainage techniques,⁸⁰ the potential to reduce rare but serious complications of subdural drains suggests subgaleal drains are preferable.

Drains are commonly left in place for 48 h postoperatively, but many patients improve rapidly postoperatively and can be ready for discharge within this timeframe. One study randomly assigned 420 patients to 24 h or 48 h of postoperative passive subdural drain, with no significant difference in recurrence rate (14% for 24 h vs 13% for 48 h).¹⁶ Mortality and complications were similar between the 24 h and 48 h groups, with a significantly shorter stay in the neurosurgical unit in the 24 h group (2 ± 0.9 vs 2.8 ± 1.6 days; $p<0.001$). This research group further randomly assigned 347 patients to 6 h, 12 h, and 24 h of passive subdural drainage; 3-month recurrence increased with 6 h (27%) and 12 h (20%) compared with 24 h (10%), not meeting the non-inferiority benchmark for less than 24 h drainage.⁸¹ However, a study on active subgaleal drainage, with 96 patients drained for more than 24 h

(mean 24.8 h) and 111 drained for less than 24 h (mean 12 h), found no significant difference in recurrence (12.5% for >24 h vs 11.7% for <24 h) or mortality.⁶³ Thus, active drainage might be necessary to enable shorter drainage times. A trial comparing exhaustive drainage, guided by postoperative CT scans and utilising urokinase injections for residual haematoma, with standard fixed drainage over 48 h, is registered as complete and results are awaited (NCT04573387). Because multiple aspects of drain technique, timing, and type of drainage are influential, a multicentre prospective platform randomised trial would be useful to enable synchronous comparison of these variables.

Postoperative mobilisation

Historically, patients remained on bedrest after CSDH surgery as it was thought to encourage brain re-expansion and reduce recurrence. The GET-UP trial randomised 208 patients to early mobilisation (walking as soon as clinically tolerated) versus 48 h of bedrest.⁶⁴ Patients with early mobilisation (at a mean of 5.5 h) had significantly more favourable outcomes (76.5% Extended Glasgow Outcome Scale score ≥ 5 at 1 year) compared with bedrest (58.4% Extended Glasgow Outcome Scale score ≥ 5 at 1 year). Surgical recurrence (4.8% for bed rest and 7.7% for early mobilisation) and mortality (8.7% for both groups) at 1-month were similar across groups. The number of postoperative medical complications was higher in the bedrest group than in the mobilisation group (34.6% vs 19.2%; $p=0.012$).⁶⁵

MMAE

Recognition that recurrent haemorrhage arises from the fragile neovascularisation within CSDH membranes led to targeting of the dural blood supply as a treatment. As the dura and the newly formed CSDH membranes are primarily supplied by the middle meningeal artery, endovascular embolisation of this vessel represents a logical therapeutic target. Minimally invasive MMAE can be used as an alternative to non-operative management (to prevent a small or incidental CSDH progressing) or as an adjunct to surgery (to prevent recurrence), or as a stand-alone treatment in frail patients or those aiming to avoid surgery.

The enthusiasm for this treatment approach has led to it being adopted in some high-income countries worldwide, despite initially scarce evidence. The recently published findings of five large randomised trials are useful in guiding use of MMAE: MAGIC-MT,⁵² STEM,⁵³ EMBOLISE,⁵⁴ EMPROTECT,⁵⁵ and MEMBRANE.⁵⁶ All five studies included patients with symptomatic treatment-naïve CSDHs, with the EMPROTECT trial also including recurrent cases. Each study randomised patients to usual care or the addition of MMAE with primary outcomes centred on recurrence rather than patient-reported outcomes (table 1). Importantly, the primary outcome varied across the trials such that some

studies included death and major complications in addition to recurrence, and the definition of recurrence varied from reoperation only to non-operated residual or haematoma indicating re-admission; both non-operated residual and haematoma indicating re-admission have uncertain clinical significance. All studies except MAGIC-MT were industry funded, introducing potential bias. There was also substantial selection bias among the studies, with all excluding patients with poorer baseline function (mRS >3 in EMBOLISE, EMPROTECT, and MEMBRANE, >2 in MAGIC-MT, or >1 in STEM).

In operated CSDH, EMBOLISE reported a significant reduction in reoperation at 90 days with embolisation (table 1).⁵⁴ Similarly, STEM and MEMBRANE reported fewer primary outcome events at 6 months with MMAE for the combined surgical and non-surgical groups.⁵³ Most of the primary outcomes in STEM were reoperations (51 [77%] of 66 events) and although not powered to assess subgroups, only the non-surgical group results suggested effectiveness of embolisation (OR 0.18, 85% CI 0.07–0.45), whereas the surgical group did not suggest effectiveness (0.53, 0.23–1.24). Although recurrence was not separated from other co-primary outcomes in subgroups, the rate of 23% in the surgical group is much higher than recurrence rates reported previously (around 10%).^{15,16,39–41} The MEMBRANE trial also reported fewer re-operations at 6 months with MMAE compared with standard care (3.8% versus 10.0%), although the trial was not powered for this outcome.⁵⁶ The MAGIC-MT trial did not show a difference in primary outcome between groups (table 1).⁵² Again, a larger potential effect of embolisation was seen in the non-surgical group (RR 0.39, 95% CI 0.14–0.99) but not in the surgical group, who made up 78% of the study population. Finally, EMPROTECT reported no significant difference in primary outcome with the addition of embolisation to surgery (table 1) and similar operative recurrence rates with embolisation (4.3%) and without embolisation (8.3%).⁵⁵

Surgical and non-surgical groups should be analysed separately, as many clinicians would consider not requiring surgery a watch-and-wait condition, which might never progress or require treatment, unlike CSDHs that need urgent surgery. The justification for treating patients who might never need intervention remains debated. Current practice often restricts surgery to patients with neurological deficits relating to mass effect (eg, limb weakness), overlooking other symptoms such as mild cognitive impairment, headache, and progression of frailty. The definition of recurrence is also crucial, as seen in the EMMA-Can trial of 192 patients treated with surgery with or without MMAE, in which symptomatic radiological recurrence was significantly reduced with MMAE compared with surgery alone (43% vs 28%) but re-operation rate did not differ (5.4% vs 8.6%).⁸²

A 2025 meta-analysis of EMBOLISE, MAGIC-MT, and STEM reported no significant difference in recurrence

	Participants (n)	Patient selection	Interventions		Primary outcome measure	Results of primary outcome
			Treatment	Control		
MAGIC-MT (China), 2024 ⁵²	722	Primary symptomatic CSDH with mass effect; surgical (565 patients) and non-surgical (157 patients), as per decision by surgeon	Standard care (surgical in 281 patients) plus MMAE (before any surgery) with a liquid embolic agent (Onyx)	Standard care (surgical in 284 patients; surgery was using burr holes)	Symptomatic recurrence for surgical group (re-operation or haematoma thickness >10 mm) or progression for non-surgical group (increased haematoma thickness by >3 mm from baseline or the need for surgery) within 90 days; imaging adjudication by core unblinded laboratory team	6·7% recurrence or progression in MMAE group and 9·9% recurrence or progression in control group (−3·3% absolute percentage difference, 95% CI −7·4 to 0·8)
STEM (USA), 2024 ⁵³	310	Primary symptomatic CSDH >10 mm; surgical CSDH (189 patients) and non-surgical CSDH (121 patients) as per decision by surgeon	Standard care (surgical in 91 patients) plus MMAE (before any surgery) with non-adhesive liquid embolic agent (squid)	Standard care (surgical in 98 patients; surgery was using burr holes [89%] or subdural evacuating port system [7%])	Any of: recurrent or residual haematoma (>10 mm), repeat or rescue surgery, major disabling stroke, myocardial infarction, or death from neurological cause within 180 days; imaging adjudication by core unblinded laboratory team	16% primary outcome rate in the MMAE group and 36% primary outcome rate in the control group (OR 0·36, 95% CI 0·20–0·66)
EMBOLISE (USA), 2024 ⁵⁴	400	Primary symptomatic CSDH treated with surgery	MMAE within 5 days of surgery with embolic agent (Onyx) on one or both sides as per clinician decision	Surgery alone (burr holes or craniotomy)	Either repeat surgery for CSDH recurrence or progression within 90 days (with or without symptoms); surgery at clinician discretion and outcome adjudicated by clinical events committee	4·1% recurrence or progression in the MMAE group and 11·3% recurrence or progression in the control group (risk ratio 0·36, 95% CI 0·11–0·80)
EMPROTEC (France), 2025 ⁵⁵	342	Primary surgical CSDH with high-risk for recurrence (patients with alcohol misuse disorder, liver cirrhosis, antithrombotic use, platelets <100, and no subdural drain) or recurrent CSDH requiring surgery	MMAE within 7 days of surgery with free-flow injection of embosphere trisacryl gelatin microspheres	Surgery alone (burr holes)	Recurrent CSDH requiring surgery or re-admission, that is symptomatic, with a midline shift of ≥5 mm, or that is >10 mm thick at 6 months; assessed by a surgeon, radiologist, and an independent adjudication committee	14·8% primary outcome rate in the MMAE group and 21% primary outcome rate in the control group (OR 0·64, 95% CI 0·36–1·14)
MEMBRANE (USA and China), 2026 ⁵⁶	376	Surgical (265 patients) and non-surgical (111 patients) as per decision by surgeon	Standard care (surgical in 133 surgical patients) plus MMAE	Standard care (surgical in 132 patients; surgery was with burr holes [51%], craniotomy [36%] or minimally invasive [13%])	Residual or recurrent CSDH (>10 mm or requiring surgery) at 6 months	11·6% primary outcome rate in MMAE group and 22·1% primary outcome rate in control group (OR 0·53, 95% CI 0·31–0·91; p=0·04)

CSDH=chronic subdural haematoma. MMAE=middle meningeal artery embolisation. OR=odds ratio.

Table 1: Characteristics and outcomes of MMAE trials

with embolisation compared with usual care in surgical patients (RR 0·60, 95% CI 0·19–1·88).⁸³ However, the definition of recurrence in MAGIC-MT and STEM included residual CSDH more than 10 mm regardless of symptoms or intervention, and the reoperation rate at 90 days was lower with embolisation than with standalone surgery (0·43, 0·22–0·83). The need for rescue surgery or progression was significantly reduced in non-operated patients with MMAE compared with no treatment (0·36, 0·22–0·60).

Overall, the data suggest a biological effect favouring reduced recurrence with embolisation. However, none of the studies showed a significant difference in functional outcomes, as assessed by the mRS, and data are insufficient for MMAE to be adopted as standard of care. All studies had favourable safety profiles for embolisation, with a low rate of serious adverse events (0–2·0%), although detailed procedural information was not always reported. Embolisation was not technically feasible in all cases, as seen in 20 (11·7%) of 171 participants in EMPROTECT.^{52,54,55} Targeting embolisation to high-risk populations, such as

patients requiring antithrombotic medication or those with coagulopathies, might be appropriate, as suggested by some consensus guidelines.⁸⁴

Data on the economic impact of embolisation are scarce. One retrospective analysis of 170 patients reported significantly lower 1-year hospital costs with embolisation compared with surgery as first-line treatment, driven by lower re-treatment rates with embolisation.⁸⁵ However, the embolised CSDHs were significantly smaller than those treated surgically at baseline and, despite propensity-matching, there was still bias between the cohorts, low power, and no patient-reported outcomes. The economic cost of embolisation in addition to surgery is still unclear. Further data from the 19 ongoing MMAE trials will enhance understanding of its application (appendix pp 1–3).

Pharmacological therapies

The complex cascade of mediators leading to CSDH formation mean that there are multiple pharmacological targets.⁵ Drugs with broad effects, such as steroids, have

multiple pro-inflammatory and neovascular targets, but risk greater side-effects; a more targeted approach could use a drug that affects a single influential mediator in the cascade (such as VEGF). In this section we discuss current pharmacological therapies, starting with those with the most evidence and moving on to use-combined and biomarker-led drug regimens, which are potential avenues for future research.

Steroids

Previous observational studies suggested that steroids were effective as a standalone therapy or adjunctive to surgery to treat CSDH, but more recently three large randomised trials have highlighted serious concerns with steroid therapy.^{15,86,87} One multicentre randomised trial comparing primary treatment with dexamethasone versus burr hole surgery in 252 patients with symptomatic CSDHs⁸⁶ was terminated early due to safety concerns and because the 3-month functional outcome on the mRS did not reach the non-inferiority benchmark for dexamethasone compared with surgery (OR 0.55, 95% CI 0.34–0.90). Dexamethasone was not successful as a first-line treatment, with 77 (61%) of 127 patients needing further treatment, and an increased adverse event rate.

In the Dex-CSDH trial, 748 patients were randomly assigned to dexamethasone or placebo in combination with standard therapy (including initial surgery for the presenting CSDH in 699 [93%] patients); the dexamethasone group had a lower rate of favourable outcome on the mRS at 6 months (83.9% vs 90.3%) and serious adverse events occurred in 60 (16.0%) of 375 participants in the dexamethasone group compared with 24 (6.4%) of 373 in the placebo group.¹⁵ The recurrence rate was noticeably lower with dexamethasone than with placebo (1.7% vs 7.1%), suggesting the drug did have a mechanistic effect. The HEMACORT trial, which assessed prednisolone as an adjunct to surgery, was terminated early (after recruiting 155 participants), owing to significantly more adverse events in the steroid group.⁸⁷ 6-month radiological recurrence rate was lower with steroids, suggesting steroids aid CSDH resolution postoperatively, but this did not affect reoperation rates or functional outcome at 6 months.

Given these poorer outcomes, steroids are not currently considered part of standard care for CSDH. However, these trials used relatively high-doses, and future studies of lower doses (eg, dexamethasone 2–4 mg for 7–14 days), particularly in patients with frailty and multimorbidity, could be considered. Topical steroids injected into postoperative drains have also been reported to be safe⁸⁸ and steroids might be more effective in some haematoma types (eg, without acute haemorrhage).⁸⁹

Tranexamic acid

The antifibrinolytic properties of tranexamic acid, inhibiting plasminogen and plasmin activity, might reduce microbleeds and aid CSDH reabsorption.⁹⁰ A

meta-analysis of five studies with 643 patients reported lower CSDH recurrence (OR 0.35, 95% CI 0.23–0.53; $p < 0.01$) and residual volumes on 3-month imaging with oral tranexamic acid in addition to surgery, compared with surgery-alone, with no increase in complications.⁵⁰ There was significant heterogeneity between studies, with only two randomised trials, both with low power, and further prospective trials are needed. However, a very recently published large randomised controlled trial in 382 patients having burr hole surgery for CSDH showed an absolute risk reduction in recurrence of 5.6% (95% CI 1.3%–11.4; $p = 0.018$) with tranexamic acid compared with burr hole surgery alone.⁴⁴ Two randomised trials are ongoing, assessing tranexamic acid as a primary medical treatment for CSDH (NCT03582293), and for prevention of postoperative recurrence (NCT05713630).

Other pharmacological agents

Statins have a potential role in CSDH owing to their weak anti-inflammatory effects. Meta-analyses suggest improved outcomes for conservatively managed patients with CSDH on statins, but data conflict on whether they reduce recurrence following surgery.^{91,92} One study of 60 patients reported improved resolution of conservatively managed CSDH when atorvastatin was combined with low-dose dexamethasone compared to atorvastatin alone,⁵¹ and the ATOCH-II study will assess this therapy combination in a large randomised trial (ChiCTR1900021659). ACE inhibitors are also postulated to modify CSDH pathophysiology, but a 2024 meta-analysis of five observational studies and one randomised trial suggests no reduced recurrence in patients taking ACE inhibitors compared with those not taking ACE inhibitors (OR 1.06, 95% CI 0.6–1.9).⁹³ A traditional Chinese herbal medicine (Goreisan) was reported to significantly reduce CSDH recurrence in combination with standard care compared with standard care alone (OR 0.72; $p < 0.0001$) in a meta-analysis of three randomised trials (comprising 51 participants) and five observational studies (comprising 505 participants).⁹⁴ However, the randomised trials were underpowered with a high or unknown risk of bias, and a recurrence rate around 10% in the Goreisan arm is similar to standard care in other studies.

Conservative management

In the age of pharmacotherapy, MMAE, and other interventions, it is tempting to offer all patients with CSDH on imaging some form of treatment. However, the natural history of conservatively managed CSDH remains largely unknown, since the literature focuses on patients who are treated rather than those who are observed. Nonetheless, a conservative management approach remains an option for smaller or minimally symptomatic CSDH and might be appropriate as a palliative approach in larger CSDHs in very frail patients. Even large CSDHs can resolve without any treatment, so watchful waiting can be a valid management strategy. One study of

	Presentation and early care	Intervention and hospital stay	Recovery and rehabilitation
Primary care and community team	Patient assessment and referral	Not applicable	Support post-discharge and community nursing support
Neurosurgery	Neurosurgical triage, assessment of risk factors, fitness for intervention, and urgency of transfer and intervention	Plan intervention and consider adjuvant therapies (eg, surgery, MMAE, or other) and manage peri-operative complications	Follow-up imaging and assessment as appropriate and management of complications (including recurrence)
Geriatrics	Assessment of frailty and comorbidity, co-existing diagnoses, and palliative care support when appropriate	Non-operative care, manage polypharmacy and antithrombotic medications, and identify delirium	Follow-up assessments including antithrombotic medication management, falls risks, cognitive function, and frailty assessments
Rehabilitation specialists	Assessment of baseline function and pre-optimisation before intervention	Postoperative rehabilitation and assessment of needs including occupational therapy, physiotherapy, and speech and language therapy, by dietitian review	Discharge planning and support and linkage to community rehabilitation
Additional specialists	Emergency care, radiologists, neurologists, stroke physicians, and general medical physicians	Anaesthetists and interventional radiologists	Neuropsychologists, dementia team, and falls team

All instances should involve shared decision making with patients and families. MMAE=middle meningeal artery embolisation.

Table 2: Multidisciplinary management of chronic subdural haematoma

159 conservatively managed CSDHs showed no need for intervention in 96 (60%) patients, with mean CSDH depth of 13 mm (compared with 14.8 mm in those crossing over to surgical treatment, at a mean of 19 days).⁹⁵ Another retrospective study on 289 CSDHs, with 199 conservatively managed (mean depth 11 mm vs 24 mm in 90 surgical cases), reported only 3% cross-over to surgery but a higher 1-year mortality in conservative cases (42% vs 12%; $p < 0.0001$).⁹⁶ These findings point to the importance of non-surgical CSDH as a marker of frailty itself and a potential prognostic marker to aid future care planning for older adults.

Follow-up imaging for small or asymptomatic CSDHs varies globally by individual patient and health-care environment, reflecting an absence of clear evidence on follow-up. Where national recommendations do exist, they highlight the necessity to counsel patients and their families regarding the risk of CSDH progression and the need for urgent assessment in the event of evolving neurological signs (eg, weakness) or symptoms.⁶⁶

Prognosis and multidisciplinary care

Outcomes in patients who have been operated on for CSDH are overwhelmingly favourable, particularly considering the age and frailty of many of the patients affected. Several trials have reported favourable outcomes (mRS scores 0–3) in around 90% of patients at 3–6 months.^{15,86,97} However, these patients have an excess mortality compared with healthy age-matched controls (hazard ratio 1.34, 95% CI 1.2–1.5), largely due to an increase in markers of frailty and a higher burden of comorbidities.¹ Pre-existing dementia (present in around 12%), appears to be a significant risk factor for increased mortality 1-year postoperatively (26% vs 15%; $p < 0.001$).¹⁰ A 2024 meta-analysis confirmed that frailty is a risk factor for poor outcome in CSDH, although studies are small and measures of frailty varied.⁹⁸ Consideration of selection

bias in interpreting randomised trial evidence is crucial, since the sickest and frailest patients might be excluded. Future research should address those with more risk factors for poor outcome or, where feasible, stratify results by such risk factors to understand differential benefits of interventions.

Multiple specialists are involved in the care of patients with CSDH and have different roles throughout the course of investigation and treatment (table 2). Regardless of the treatment strategy, patients with CSDH are likely to benefit from specialist input focused on the needs of older adults and early engagement from geriatric and rehabilitation specialists is essential to optimise outcomes and longer term care planning.^{66,67} Ultimately, shared decision making should be at the centre of CSDH care, particularly given the complexity of patient risk factors, needs, and varying treatment options. The current literature, as shown in this Review, still largely focuses on CSDH recurrence as an outcome, and greater focus on patient-reported measures, quality of life, cognitive outcomes, additional care needs, and caregiver burden are needed.

Conclusions and future directions

The broad and multiple treatment options discussed in this Review highlight the complexity of therapeutic strategies in CSDH. Interest in CSDH is increasing,⁹⁹ but practices vary widely depending on surgeon preference, patient factors, and health system context. Some countries have consensus guidelines to support management decisions and drive high-quality care, but the extent to which these are effectively implemented is not well reported.^{66,68}

CSDH poses a considerable research challenge given the heterogenous patient group with different clinical presentations and demographic factors, varied imaging findings, and a mixed natural history with some resolving without treatment and others progressing either

Search strategy and selection criteria

This Review is based on the clinical knowledge and research expertise of the authors, supported by a selective review of high quality, relevant studies published in the chronic subdural haematoma literature. We searched Ovid Medline and Embase for articles published between Jan 1, 2020, and July 13, 2026. We used the search terms: ("Chronic Subdural") AND ("haematom*" OR "hematom*" OR "haemorrhage" OR "hemorrhage"). We limited the search to English language studies. Additionally, we searched clinical trials databases (Cochrane Controlled Register of Trials, WHO International Clinical Trials Registry Platform, and ClinicalTrials.gov) for any relevant clinical trials registered since Jan 1, 2015. The PubMed search yielded 4209 articles. Of these, we selected studies that we perceived to be of high relevance and interest, methodologically sound, and representing progress in understanding in the field or relevant to future innovative research topics. Study selection was done by three authors (EE, BMD, and DJS) based on consensus, with BMD focusing specifically on middle meningeal artery embolisation.

gradually or rapidly. Many of the new therapies have emerged from a greater understanding about the pathophysiology of CSDH. Basic scientific work will continue to have an important role and identifying key biomarkers of disease activity might help risk-stratify patients and target treatments.

Robust evaluation of new therapies in well-designed trials is crucial, as illustrated by the steroid studies. MMAE requires careful consideration, economic analysis, and understanding of patient-reported outcomes before widespread adoption. Pharmacotherapeutic innovation and combined therapies as an alternative or adjunct to surgery also present an opportunity to improve patient outcomes. Given the multiplicity of research questions within one condition, efficient designs, such as platform studies, are likely to be valuable¹⁰⁰ in answering questions relating to anaesthesia type, management of anticoagulants, drain type, adjuvant therapies (including tranexamic acid), and specialist geriatric input. Clinical trial design needs to ensure broad patient inclusion, specifically frail and vulnerable older patients, who might require different approaches.

To enable true comparison and meta-analysis, high quality studies, registries, and trials should all report harmonised data and outcomes in people with CSDH across different interventions. An international consensus study on a core outcome data set will be published in late 2026 to help support this goal and improve the quality of CSDH research.⁴⁹

Priorities for future work will include identifying outcome measures relevant to patients, having clear definitions of outcomes, such as recurrence, and assimilating the data available into best practice guidelines

and clinical decision aids. Economic evaluation and relevance to low-income and middle-income countries, which remain under-represented in the data, will also be vital. As the CSDH population continues to grow, so do understanding of the condition and the treatment options, creating an exciting environment of innovative research opportunities with an aim to achieve individualised, holistic patient care.

Contributors

Conceptualisation, literature search, methodology, figures, and writing of original draft: EE. Conceptualisation, literature search, methodology, and writing of original draft: BMD and DS. Conceptualisation, review, and editing: CC, AC, IDB, MD-W, VH, AH, DH, AJJ, AK, DM, TM, and PW. Conceptualisation, methodology, supervision, review, and editing: PH.

Declaration of interests

MD-W is the co-founder and director of The Healthcare Improvement Studies Institute Labs. DM is supported by the TBI-REPORTER core programme grant; has received consulting fees from NeurotraumaSciences, Lantmannen AB, GlaxoSmithKline, PressuraNeuro, CSL Behring, and Invex; and has received speaker fees from Integra Neurosciences. TM has received travel reimbursement from Medtronic and Balt. DJS is funded by a National Institute for Health Care Research Advanced Fellowship and grant funding from the association of anaesthetists; is a council member for Neuroanaesthesia and Critical Care Society; and co-leads the Improving Care for Elderly Neurosurgery Initiative (ICENI). EE is a group member for the International Collaborative Research Initiative on Chronic Subdural Haematoma and ICENI. All other authors declare no competing interests.

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