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Review Article

Updates in Chronic Subdural Hematoma: From Epidemiology, Pathogenesis, and Diagnosis to Treatment

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Abstract

Chronic subdural hematoma (CSDH) is a common disease in neurosurgery, with epidemiological characteristics showing an overall annual incidence of 1.7–20.6 per 100,000 people and a higher prevalence in the elderly. However, despite the increased disease burden, there have been limited breakthroughs in treatment options over the past 20 years. A significant gap exists in our understanding of the exact pathophysiological mechanism of CSDH, leading to a lack of specific clinical treatment options based on a clear pathological mechanism. Current research suggests that the development of CSDH involves dual mechanisms of trauma and inflammation, and that these pathologic processes together promote pathological changes such as angiogenesis, inflammatory response, and neovascularization. Therapies for CSDH encompass both surgical (e.g., twist-drill drainage, burr-hole drainage, craniotomy) and non-surgical approaches (e.g., clinical observation, medication, intracranial pressure monitoring, anticoagulation). Meanwhile, middle meningeal artery embolization, as an emerging minimally invasive interventional technique, has shown good prospects for clinical application. This review aims to bridge the gap between current treatment options and the need for effective strategies by providing a comprehensive summary of the epidemiological trends, pathophysiological advances, and optimization of therapeutic strategies for CSDH.

Introduction

Chronic subdural hematoma (CSDH) is one of the most common disorders in neurosurgery.[1] The incidence of CSDH in the population ranges from 1.7 to 20.6 per 100,000 people per year, with a higher prevalence in the elderly.[2] The incidence of CSDH is projected to increase with the global ageing of the population and the increased use of antiplatelet and anticoagulant drugs.[2] However, despite the increased disease burden, there have been limited breakthroughs in treatment options over the last 20 years.[3] As the exact pathophysiological mechanism of chronic subdural hematoma has not been fully elucidated, there is still a lack of specific clinical treatment options based on a clear pathological mechanism. The treatment of CSDH includes surgical and non-surgical approaches. Surgical therapies include twist-drill drainage, burr-hole drainage, and cranial hematoma removal.[4] Non-surgical treatments include clinical observation, medication, intracranial pressure testing, and anticoagulation.[1] Meanwhile, middle meningeal artery embolization (MMAE) and some emerging pharmacological treatments are being extensively studied.[5] Among these, MMAE, as a minimally invasive interventional technique, has attracted much attention in recent years and may provide new treatment options for patients at high risk of recurrence. Identifying the gap in current treatment options and the need for more effective strategies, this review aims to provide a comprehensive summary of the epidemiological trends, pathophysiological advances, and optimization of therapeutic strategies for CSDH, with a focus on the clinical evidence and application prospects of MMAE.

The epidemiology of CSDH

CSDH is the second most prevalent neurosurgical disease, occurring primarily in the elderly, with a prevalence of 127.1 per 100,000 people over the age of 80.[6] As the world ages and the use of antiplatelet and anticoagulant drugs increases, the incidence of CSDH is rising year by year.[7] Another important factor is the increased risk of ground-level falls and other age-related trauma in the elderly patient population.[8] It is projected that by 2030, the incidence of CSDH will surpass the incidence of cranial tumors

Keywords: Chronic subdural hematoma; Epidemiology; Etiology; Pathogenesis; Diagnosis; Treatment.

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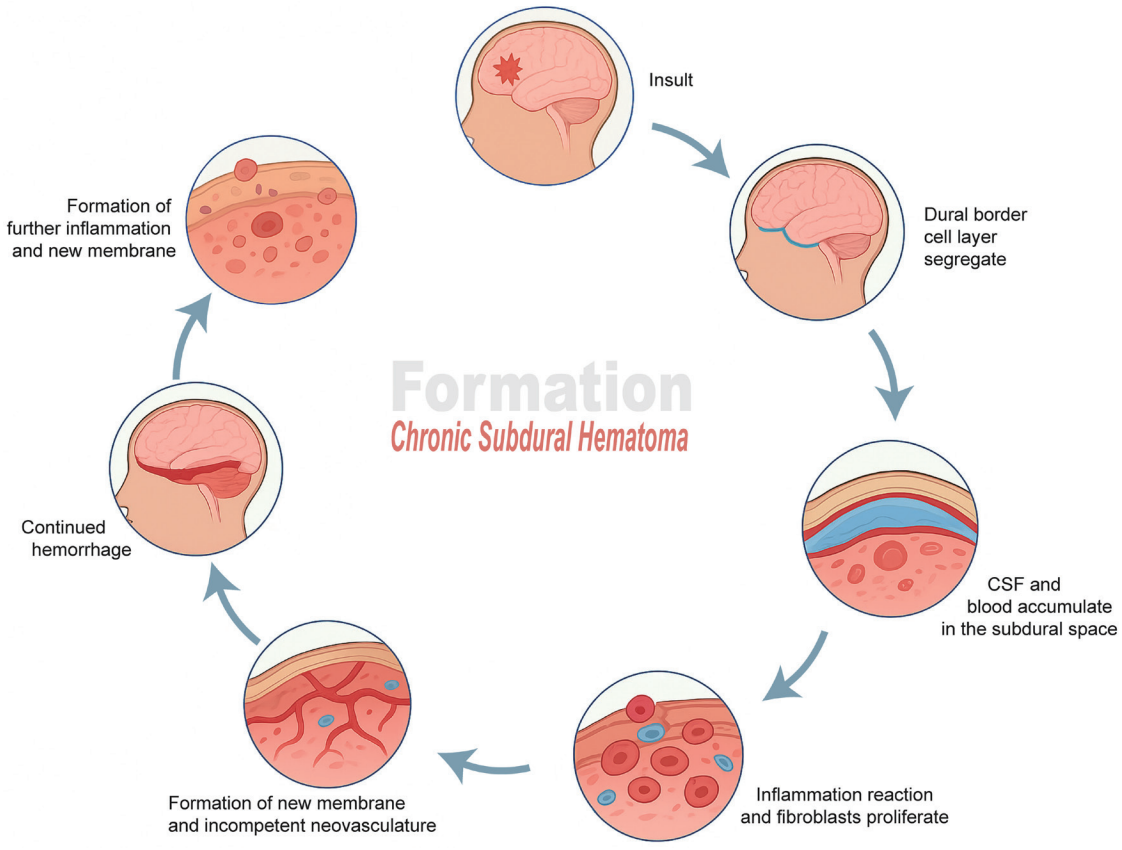

Fig. 1. Pathophysiology of chronic subdural hematoma formation. Initially, an insult cause separation of the dural border cell layer. CSF and blood accumulate in the subdural space. Inflammatory cells migrate to this area to repair the dural border cell layer, whereupon the inflammation reaction, together with fibroblasts proliferate, promotes the formation of a new membrane and incompetent neovasculature, leading to persistent haemorrhage. This, generates further inflammation and new membrane formation. CSF, cerebrospinal fluid.

(14/100,000) to become the most common condition in cranial surgery.[3] However, although CSDH is a curable neurosurgical disease, there is no single standard treatment for CSDH because the pathophysiological mechanisms of the disease are still unclear. As a result, recurrence remains a significant concern, occurring in one out of ten surgically treated patients.[9] Factors affecting the prognosis of CSDH are still being investigated, including age, use of postoperative anticoagulant/antiplatelet medications, and surgical approach, among others.[10] Various factors have been implicated in the recurrence of CSDH. These include the presence of a compartment within the hematoma preoperatively, poor brain re-expansion after surgery, alcohol abuse, use of antiplatelet/anticoagulant medication, male gender, and poor liver function.[11] The factors affecting CSDH recurrence and the relative importance of each factor vary across different studies.[12]

The etiology and pathogenesis of CSDH

The pathophysiology of CSDH formation and development is complex and includes trauma, inflammation, angiogenesis, coagulation disorders, and anticoagulation/antiplatelet therapy.[13] Yang *et al*.[14] found that skull density is significantly correlated with CSDH progression. Previous studies have demonstrated that angiogenesis, inflammation, and neovascularization play a key role in CSDH formation.[15] Vascular endothelial growth factor, a signaling protein present at extremely high levels in CSDH, can promote angiogenesis.[16,17] Due to cranial trauma, changes in intracranial pressure, craniocerebral disproportion, and medical factors, the bridging veins bleed as they cross the marginal cell layer of the dura mater.[4] Subsequently, injury to the dural border cell layer leads to the extravasation of cerebrospinal fluid and blood into the subdural space.[3] However, recent studies have shown that the theory that rupture of the bridging vein is the origin of the subdural hematoma needs further testing. Biomechanical studies suggest that the bridging vein requires a great deal of force to rupture, so the origin of hemorrhage in non-traumatic CSDH cannot be explained.[12] Schmolling *et al.*[18] found that a slow venous hemorrhage should cause symptomatic collection within a few days. However, the onset of symptoms in post-traumatic CSDH takes an average of four to seven weeks, and autopsies of CSDH rarely reveal a disrupted bridging vein. Recent studies have shown that inflammation plays a major role in CSDH. The physiological inflammatory response involves the overproduction of pro-inflammatory mediators, including fibrinogen degradation products, platelet-activating factor, interleukin (IL) 6, IL-8, bradykinin, and others.[2] Bounajem *et al*.[16] demonstrated a decrease in inflammatory markers (IL-8, tumor necrosis factor-α) and an increase inanti-inflammatory markers (IL-10, IL-13, IL-1) in response to subsiding CSDH hematoma. This supports the idea that inflammation is closely related to CSDH. CSDH pathophysiology is depicted in Figure 1.

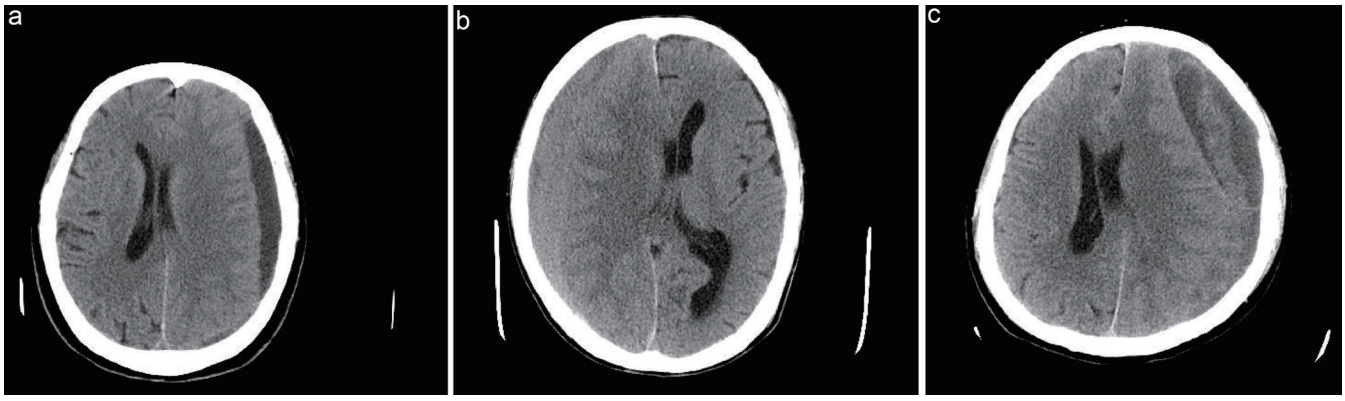

Fig. 2. Computed tomography images of chronic subdural hematoma. Typical computed tomography images of chronic subdural hematoma with hypodensity (a), isodensity (b), or mixed density (c).

The radiographic images of CSDH

Brain computed tomography (CT) has become the examination of choice for patients with CSDH and is usually used for diagnosis and follow-up.[3,19] CSDH typically presents as hypointense, isointense, or mixed-density lesions on CT, which are important for selecting the appropriate treatment (Fig. 2).[20]

Although CT is the standard diagnostic tool for CSDH, it is often difficult to differentiate the nature of the hematoma and almost impossible to recognize small isodense hematomas.[21,22] Compared with CT, magnetic resonance imaging (MRI) can more accurately identify hematomas and their internal structures.[22] Meanwhile, MRI can predict the risk of postoperative recurrence by observing structural changes in the reticular appearance of CSDH.[23] MRI images of unilateral versus bilateral hematomas are shown in Figure 3.

The treatment of CSDH

Treatment of CSDH includes surgical and non-surgical approaches. Surgical treatments include twist-drill craniotomy (TDC), burr-hole craniostomy (BHC), and craniotomy.[4] However, the recurrence rate after surgical treatment has been high, which may be because subdural drainage (SDD) alone does not fully address the underlying pathological factors.[16] With the in-depth study of CSDH pathophysiology, it has been found that CSDH can be treated using MMAE, which is therefore being widely investigated as an emerging therapeutic modality. Meanwhile, endoscopic-assisted treatment of CSDH is becoming increasingly widespread. Non-surgical treatment mainly refers to pharmacological therapy.

Surgical treatment

Surgery is preferred for patients who are symptomatic or who show significant mass effect on imaging.[24] Surgery has a rapid and favorable therapeutic outcome; however, it has a high recurrence rate, and the optimal surgical approach remains controversial. Indications, modality and timing of surgery, anesthesia, and timing and location of drainage are all debated.[25] A summary of each surgical treatment is shown in Table 1.[25–38]

TDC

TDC is considered the least invasive procedure for the surgical treatment of CSDH.[26] Compared to other surgical procedures, it has the advantage of not requiring general anesthesia and can be

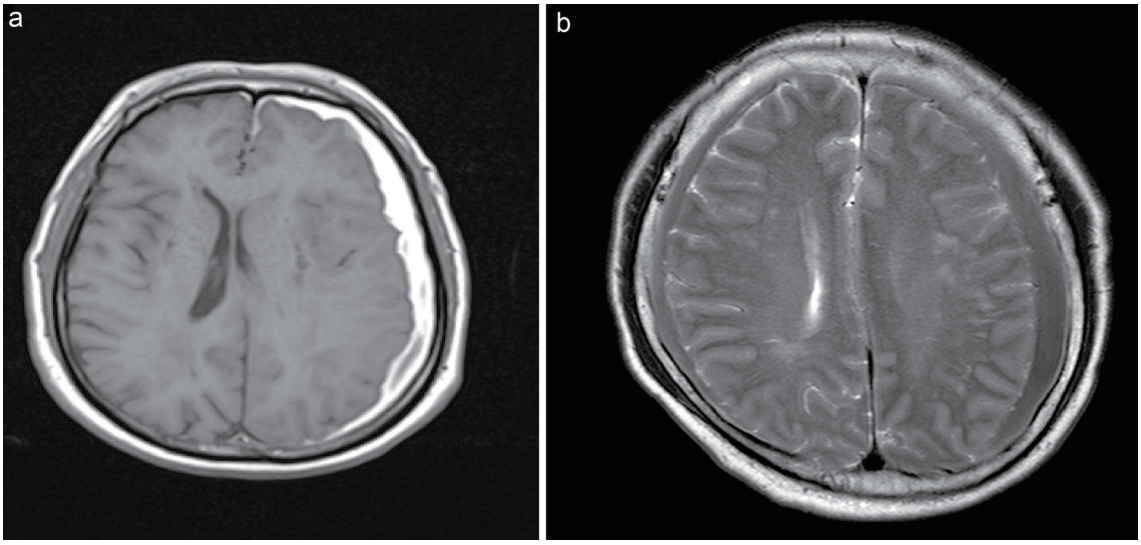

Fig. 3. Magnetic resonance images of chronic subdural hematoma. Unilateral (a) versus bilateral (b) hematomas.

Table 1. Summary of each surgical treatment

Subject	Recommendation	Controversies
Twist drill craniotomy	Least invasive procedure[26]; Not requiring general anesthesia[27]	Vascular damage, brain tissue[28]; Drainage dislocation[28]; Recurrence is the highest[29]
Burr hole craniostomy	Best cure rate[25]; Effective and safe in the elderly[30]	Number of holes drilled[31]; The choice of flushing solution[32]
Craniotomy	The hematoma removal efficiency is high[33,34]	Highest complication and mortality rates[35]
Endoscopic procedures	The entire hematoma cavity can be visualized[36]; Reduce the recurrence rate and shorten the drainage time[37]	Visual field blindness and operational errors[38]

performed rapidly at the bedside.[27] Therefore, it is the preferred surgical modality for elderly patients with multiple comorbidities who are at high risk from general anesthesia and require urgent decompression. Some studies have concluded that TDC is associated with significantly lower complication rates and shorter hospitalization than BHC,[39] while others have shown that TDC is comparable to BHC.[40] A single-center clinical study conducted by Wang *et al*.[41] included 219 patients with CSDH, and the results confirmed that TDC is safe and effective, with a low rate of postoperative recurrence. However, TDC carries a higher risk of vascular and brain tissue injury, hematoma formation, and drainage dislocation compared to BHC.[28] Another study indicates that the recurrence rate of TDC is the highest among surgical treatments.[29] Based on the evidence of its safety and efficacy, TDC should be prioritized in the selection of surgical protocols for CSDH.

BHC

Previous studies have shown that BHC is the preferred surgical treatment for CSDH,[33] as it not only offers the best cure rate,[25] but is also effective and safe in the elderly, and can be performed in patients aged >85 years.[30] With regard to positioning therapy, the sitting position preserves the best features of the classical position while providing additional comfort for both patient and surgeon. One study showed that even critically ill patients tolerate this position well, and no complications related to the sitting head position were found.[42] In addition, there is limited information available on the effect of postoperative supine versus elevated head position on clinical outcomes. Studies have shown no difference between supine and elevated head position in terms of recurrence rates, secondary interventions for recurrence, and somatic complications.[43,44] The main drainage positions are subgaleal/subperiosteal drainage (SGPD) and SDD.[45] A meta-analysis showed that SGPD was more effective in reducing recurrence rates compared to SDD.[45] Pranata *et al*.[46] also found that SGPD was associated with lower rates of CSDH recurrence but similar rates of mortality, seizures, postoperative bleeding, and infections compared with SDD. However, it has also been suggested that the choice of SDD versus SGPD has no significant effect on CSDH prognosis.[47] Saline (NS) and artificial cerebrospinal fluid are commonly used as irrigation fluids, with NS being the most frequently used. However, there is controversy regarding the choice and temperature of the flushing solution. A cohort study of 234 patients with CSDH found that artificial cerebrospinal fluid significantly reduced the rate of postoperative recurrence of CSDH compared to NS.[32] Meanwhile, Huang *et al*.[48] demonstrated that body-temperature flushing fluid reduced the postoperative recurrence rate of CSDH by 64% compared with room-temperature fluid. Many researchers believe that BHC is better than TDC, but the recurrence rate after BHC remains high, ranging from 11.4% to 21%, and increases with age.[30] There is also ongoing debate about aspects of BHC treatment, such as the number of holes drilled, whether or not to irrigate, anesthesia method, and drainage position.[25] Some researchers have reported that postoperative mortality and recurrence rates are significantly lower when BHC is performed under local anesthesia compared with general anesthesia.[49] Sale *et al*.[31] have shown no difference between single versus double drilling in the treatment of CSDH, while Salihi concluded that the efficacy of double drilling was superior to that of single drilling.[50] More large-sample, multi-center clinical studies are needed to further validate these findings.

Craniotomy

Before the advent of conventional imaging, craniotomy was the primary surgical approach for CSDH. Although craniotomy is not the procedure of choice for CSDH, it is still commonly used for recurrent CSDH, incomplete hematoma liquefaction, or hematomas with internal septa and membranes, and has demonstrated feasible, safe, and effective results.[33,34] However, craniotomy also has the highest complication and mortality rates of all surgical treatments.[34] Raghavan *et al*.[35] found that craniotomy was superior to BHC in terms of treatment outcomes; however, mortality rates were significantly higher. Therefore, to reduce the mortality rate, the indications for craniotomy should be carefully evaluated and good postoperative care provided.

Endoscopic procedures

The entire hematoma cavity can be visualized using neuroendoscopy, allowing complete removal of the hematoma and trabecular structures, as well as placement of drains under visual guidance.[36] Thus, achieving a good therapeutic effect. Wu *et al*. found that neuro-endoscopic-assisted drilling and drainage led to good therapeutic results, reducing the recurrence rate of CSDH and shortening postoperative drainage time compared with conventional drilling and drainage.[37] Guo *et al*.[51] also found that endoscopic-assisted treatment resulted in a significant reduction in recurrence and complication rates in 441 patients with CSDH. However, some researchers have suggested that endoscopic-assisted treatment of CSDH does not significantly improve recurrence rates, mortality rates, or neurological outcomes compared with other therapies.[37] It has also been suggested that endoscopic treatment may lead to prolonged procedure times and cortical injury due to limited visual field and operational errors.[52,53] Therefore, not all patients are suitable for endoscopic surgery. Overall, endoscopic-assisted treatment of CSDH remains an effective, feasible, and promising treatment modality.

MMAE

Mechanisms and indications

Histological studies have found that MMA branches are directly

Table 2. Summary of each drug treatment

	Potential pathway	Clinical evidence	Potential drug interactions
Atorvastatin	Anti-inflammation, Anti-angiogenesis	Retrospective study[66]; Meta-analysis[67]; RCT[68]	Leads to vascular endothelial cell dysfunction combined with dexamethasone[69]
Corticosteroids	Anti-inflammatory, antifibrinolytic, and antiangiogenic	Meta-analysis[70,71]; Sample analysis[72]	Improve the recurrence of CSDH when combined with atorvastatin[72]
TXA	Anti-fibrinolytic, anti-inflammatory	Prospective observational study[73]; meta-analysis[74,75]	Reduce bleeding; Risk when combined with clopidogrel[76]
ACEI	Anti-angiogenesis	Retrospective study[77]; meta-analysis[78]	Reducing the efficacy of ACEI when combined with Aspirin[79]
Herbal medicine	Unknown	RCT[80] meta-analysis[81]	Unknown

ACEI, angiotensin converting enzyme inhibitors; CSDH, chronic subdural hematoma; RCT, randomized controlled trial; TXA, tranexamic acid.

connected to CSDH, so repeated microbleeding from immature neovascularization of the MMA may be responsible for CSDH growth and recurrence.[54] MMAE has been suggested to inhibit hematoma membrane neovascularization, thereby preventing the recurrence of CSDH.[55] Salem *et al*.[56] also found that MMA diameter was the only significant predictor of clinical and imaging treatment failure. All these findings suggest the involvement of the MMA in the mechanism of CSDH formation. Therefore, MMAE currently appears to be a promising treatment for CSDH. In addition, MMAE can be performed in the awake state and is particularly suitable for high-risk groups such as the elderly, patients with coagulation disorders, and those receiving anticoagulation therapy.[13]

Surgical methods

The MMAE surgical approach is divided into primary MMAE (MMAE as initial treatment) and adjuvant MMAE (MMAE with surgical debridement).[57] Primary MMAE may better reduce the recurrence rate of CSDH and decrease hematoma volume. However, it has been shown that primary MMAE has a failure rate of 8.9%.[58] Adjuvant MMAE, combined with either surgical procedure, may better reduce the recurrence rate of CSDH and improve prognosis. A retrospective study showed no significant difference between primary and adjuvant MMAE in terms of the need for surgical rescue, complications, or mortality; however, the need for surgical rescue was significantly lower in primary MMAE than in surgical treatment alone.[57] This also affirms the effective role of MMAE in CSDH. Regarding the choice of embolization materials, including polyvinyl alcohol, N-butyl-2-cyanoacrylate, Onyx, spring coils, and gelatin, no difference in outcomes was found.[4] A multicenter pilot trial showed no significant difference in outcomes between liquid and granular agents.[59]

Treatment effects

A single-center study has shown that MMAE alone is a safe and effective minimally invasive technique for patients with CSDH who do not require immediate surgical clearance.[60] Regarding efficacy, the recurrence rate of CSDH treated with MMAE is 4.3%, which is significantly lower than that of various surgical treatments.[18] Henry *et al*.[61] demonstrated that MMAE can reduce the recurrence rate of CSDH by up to 80%. However, although MMAE is effective and has a low recurrence rate, the indications, optimal patient population, and other factors are unclear because MMAE is still under investigation. In the acute phase of CSDH, MMAE does not reduce the mass effect or relieve symptoms and dysfunction.[54] Additionally, MMAE does not reduce clinical symptoms or neurological dysfunction caused by the subdural collection.[62] Therefore, MMAE can only be used as an adjunctive treatment to conventional surgical or non-surgical therapies and cannot completely replace surgery.[3] Some adverse effects of MMAE have also been reported. It has been claimed that MMAE may cause intracranial infections in CSDH patients with underlying comorbidities and risk factors.[63] There are also case reports of complete and permanent facial palsy due to occlusion of the middle meningeal artery at the level of the foramen spinosum following MMAE.[64]

Future prospects

Clinical data and long-term efficacy studies have shown that MMAE significantly reduces the risk of recurrence.[65] MMAE is a safe and effective treatment with a low recurrence rate, either as a primary stand-alone treatment or as postoperative prophylaxis for recurrent CSDH. However, there is currently considerable controversy regarding MMAE, including the timing of the procedure, primary versus adjuvant MMAE, choice of embolization material, unilateral versus bilateral prophylactic embolization, and the populations that would benefit most. Overall, however, MMAE remains a promising treatment modality for CSDH.

Non-surgical treatment

Surgery is the treatment of choice for CSDH. However, for patients with mild symptoms, advanced age, long-term anticoagulant/antiplatelet therapy, or pregnancy, non-surgical treatment is preferred.[3] Non-surgical treatments mainly include clinical observation, medication, intracranial pressure testing, and anticoagulation.[1] The most important of these is pharmacological therapy. However, since the pathophysiology of CSDH is still unclear, no single drug has shown significant efficacy. In addition, the effectiveness of conservative treatment is unknown. A summary of each drug treatment is shown in Table 2.[66–81]

Atorvastatin

Atorvastatin reduces inflammation in the vessel wall and promotes endothelial progenitor cells for vessel wall repair, both of which are closely related to the pathophysiology of CSDH,[82] thus reducing hematoma volume and improving the clinical prognosis of patients. Animal experiments have shown that atorvastatin can eliminate SDH and improve neurological function in rats through its anti-inflammatory effect.[83] Therefore, atorvastatin is now widely used in conservative treatment and postoperative adjuvant therapy

of CSDH.[68] He *et al*.[67] demonstrated that atorvastatin can effectively reduce the recurrence rate of CSDH and improve neurological function. In addition, manual cervical lymphatic drainage, developed by Gao's team, promotes hematoma resorption in patients with CSDH and improves the efficacy of atorvastatin-based conservative treatment.[84] However, Xu *et al*.[66] found that postoperative administration of atorvastatin did not reduce the recurrence rate of CSDH but only reduced hematoma volume and improved neurological function. There is also controversy regarding combination therapy. Jiang *et al*.[68] conducted a clinical randomized controlled study on 240 patients with CSDH and showed that the combination of low-dose atorvastatin and low-dose dexamethasone was superior to low-dose atorvastatin alone. However, it has been suggested that atorvastatin combined with dexamethasone may lead to vascular endothelial cell dysfunction.[69] Additionally, atorvastatin has been reported to be more effective in older patients with larger hematomas,[82] although this finding is not supported by robust clinical data. Therefore, further studies are needed on the dosage of atorvastatin, its use in combination with other drugs, and the optimal patient population. Overall, however, atorvastatin may help reduce CSDH recurrence, especially in conservatively treated patients.

Corticosteroids

Corticosteroids are used in the treatment of CSDH due to their anti-inflammatory, antifibrinolytic, and antiangiogenic activities.[76] Among them, dexamethasone has become the preferred choice for conservative or adjunctive surgical treatment of CSDH due to its potent anti-inflammatory and antivascular activity and long half-life.[76] A multicenter, open-label, controlled, noninferiority trial showed that dexamethasone treatment was not noninferior to burr-hole drainage.[85] However, a large number of recent clinical studies have shown that dexamethasone has poor efficacy and more complications than surgical and other treatment modalities. Dexamethasone is not recommended for the treatment of CSDH.[86,87] One randomized controlled trial showed that at six months, dexamethasone treatment resulted in fewer favorable outcomes and more adverse events than placebo.[86] It has been shown, however, that dexamethasone in combination with atorvastatin is the best intervention to improve CSDH recurrence and enhances the efficacy of atorvastatin.[70,72] Meanwhile, Shi *et al*.[71] found that corticosteroid-assisted surgery reduced the risk of CSDH recurrence but did not improve all-cause mortality or functional outcomes. The clinical use of dexamethasone in CSDH remains controversial, and high-quality evidence supporting its efficacy and safety is lacking. Large-sample, multicenter randomized controlled trials are needed to provide a more reliable evidence-based foundation for the rational use of dexamethasone in CSDH treatment.

Tranexamic acid (TXA)

TXA is a synthetic derivative of the amino acid lysine, which inhibits fibrinolysis by blocking the lysine-binding site on fibrinogen, thus helping to reduce bleeding.[88] In addition, TXA inhibits fibrinolytic and inflammatory mechanisms,[89] which may prevent early CSDH progression and postoperative recurrence. Previous studies have found tranexamic acid effective in reducing CSDH recurrence rates without increasing complications.[73,74] This suggests that TXA has considerable potential in CSDH treatment. However, TXA also has downsides. Some studies have found that TXA increases the recurrence rate of CSDH.[75] Medium to high doses of TXA may induce neurological complications (seizures, transient ischemic attacks, and paresthesia) in adults and children.[90] As CSDH patients are mostly elderly with a high incidence of thrombotic events, the risks of bleeding and thrombosis should be carefully evaluated when applying antifibrinolytic drugs such as TXA. Multicenter, prospective randomized controlled studies are needed to further validate the safety and efficacy of these drugs and guide their rational clinical use.

Angiotensin converting enzyme (ACE) inhibitors

ACE inhibitors have been shown in previous studies to be anti-angiogenic, reduce the volume of CSDH hematomas, and decrease recurrence rates.[77] Weigel's cohort study of 310 patients demonstrated that ACE inhibitors can significantly reduce the recurrence rate of CSDH.[77] However, Bartek *et al*. showed no correlation between ACE inhibitors and CSDH recurrence.[91,92] More recent studies suggest that ACE inhibitors can increase vascular endothelial growth factor levels through bradykinin upregulation, enhancing subdural angiogenesis, increasing hematoma volume, and raising recurrence rates.[2] Another study also showed that ACE inhibitors are strongly associated with increased hematoma size and recurrence rates, especially in patients over 80 years old.[93] Based on current clinical evidence, ACE inhibitors should not be continued as a routine treatment option for CSDH, particularly in elderly patients.

Herbal medicine

Herbal medicines have been extensively studied for CSDH treatment.[80,94] Kwon *et al*.[81] found that herbal medicines have potential in treating CSDH and preventing recurrence without significant side effects. A prospective study in 2024 showed that the Japanese herbs Goreisan and Saireito were effective in reducing CSDH recurrence without complications. Liu *et al*.[95] found that Xiaoyukang capsule has anti-inflammatory and anti-angiogenic effects and can reduce the size of hematomas and membranes in CSDH model rats. However, there is a lack of strong clinical evidence supporting the efficacy of herbal medicines in CSDH. Current studies are limited to a few specific drugs. Further clinical and basic research is needed in the future.

Future directions

The complex pathophysiology of CSDH encompasses trauma, inflammation, angiogenesis, and additional factors, presenting a rich area for therapeutic exploration. Ongoing research is actively investigating a variety of treatment modalities that target distinct aspects of this pathophysiology. Anticipated advancements in medical technology and materials, coupled with findings from relevant clinical trials, are expected to yield safer and more efficacious treatment strategies for CSDH patients, thereby enhancing their quality of life and prognosis. Nevertheless, there is a recognized need for further clinical and basic research into the pathophysiological mechanisms of CSDH, as well as the accumulation of practical experience, to refine treatment guidelines and improve patient outcomes. While surgical intervention remains the most effective treatment method, efforts to reduce postoperative recurrence and complication rates are essential. Recent years have seen the emergence of promising therapeutic strategies such as MMAE, neuroendoscopic-assisted hematoma removal, and novel pharmacological therapies, offering innovative options for CSDH management. The ongoing debate regarding the safety and efficacy of pharmacological agents underscores the necessity for continued research in this area.

Conclusions

CSDH is a challenging neurological condition with a global incidence of 1.7–20.6 per 100,000 people. Its pathophysiological progression involves a combination of trauma, inflammation, and angiogenesis. Currently, surgical intervention remains the most effective treatment method available, but its postoperative recurrence rate and complication rate need improvement. Emerging therapeutic strategies have demonstrated preliminary efficacy and provided new options for the clinical management of CSDH. Among them, MMAE, as an emerging treatment, has shown good results in terms of efficacy and postoperative recurrence rate with few side effects, positioning it as a particularly promising treatment option. It is worth noting that, compared with other neurological diseases, basic research on CSDH is still lagging behind. Future efforts should focus on further elucidating the pathophysiological mechanisms and disease progression of CSDH to facilitate the development and implementation of more precise and effective therapeutic strategies.

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Conflict of interest

One of the authors, YY, has been an editorial board member of *Neurosurgical Subspecialties* since January 2025. The authors have no other conflict of interests to note.

Author contributions

Conceptualization, resources, and writing-original draft preparation (XZ, JC, TY, KF, YM, YY); methodology, image processing, and literature summarization (XZ); writing-review and editing (YM, YY); project administration and supervision (YM, YY). All authors have made significant contributions to this study and have approved the final manuscript.

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