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Abstract

Chronic subdural hematoma (SDH) is commonly seen in the aged population. It is hypothesized to occur due to damage to the dural border cells, resulting in an inflammation−proliferation reaction. Inadequate repair leads to the formation of an external layer of cells and fragile capillaries, which are vulnerable to damage, contributing to worsening of the condition. Conventionally, asymptomatic chronic SDH was managed by observation, while symptomatic cases by surgical evacuation. However, the recurrence rate of chronic SDH after surgical evacuation was high. The middle meningeal artery (MMA) provides blood supply to the dura mater and feeds the capillaries of the membranes covering the SDH. MMA embolization blocks blood flow to this system and promotes hematoma resolution. In this manuscript, we discuss the underlying pathophysiology and current management options for chronic SDH. We also discuss the existing literature on MMA embolization.

Keywords: Chronic, SDH, MMA, MMA embolization, Endovascular

1. Introduction

Chronic subdural hematoma (SDH) is characterized by an encapsulated organized blood collection in the subdural space between the dura matter and arachnoid matter. An overall increase in the elderly population has resulted in an increased incidence of chronic SDH. Over the past fifteen years, the incidence has roughly doubled from 8.2 to 17.6 cases per 100,000 years.[45,38] Apart from age, other well-established risk factors include male gender, dependency on antiplatelet or anticoagulant medication, and chronic alcoholism.[1] With an increase in the elderly population and polypharmacy, along with pervasive usage of imaging technology, more cases of chronic SDH are being diagnosed each year. It is predicted that by 2030, there will be at least 60,000 new cases of chronic SDH every year.[2]

The diagnosis of chronic SDH can present as a clinical challenge, with the early stage of the disease usually being insidious, while the later-stage symptoms can be hard to recognize.[47] These may include gait disturbance and falls (55.5%), mental deterioration (34.0%), limb weakness (34.0%), acute confusion (32.1%), headache (17.2%), drowsiness or coma (9.6%), speech impairment (5.7%), collapse (1.0%), and seizure (1.0%).[23] Motor dysfunction is observed as tremors or gait disturbances, and aphasia and paresthesia may also occur. Diseases like dementia, Alzheimer's disease, Parkinson's disease, normal pressure hydrocephalus, and other age-related diseases can disguise the illness, making it an even bigger diagnostic challenge, resulting in a delayed diagnosis.[36] In the later stages of the disease, patients may develop seizures or hemiparesis.[46]

Chronic SDH can have long-term adverse effects on health. One study showed that the mortality rate due to chronic SDH was 13%, and at least 20% of patients required prolonged healthcare assistance.[13] While the in-hospital mortality rate of chronic SDH is low, mortality rates as high as 30% can be seen six

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months to one year after the initial presentation. This shows that chronic SDH can play a role in triggering the 'end of life' period in the elderly.[10]

2. Alternate theories of underlying mechanisms

Traditionally, chronic SDH was believed to be an accumulation of a low-flow collection of venous origin in the subdural space, occurring due to a tear in the bridging veins on their route to the dural venous sinuses.[31] However, several other factors appear to play a central role. Venous injury would result in slow, but immediate extravasation of blood into the subdural space visualized on a head computerized tomography (CT) scan. In contrast, clinical observations show normal head CT scan obtained a few hours after trauma in patients who developed chronic SDH several weeks or months later.[11] The time frame of venous blood accumulation and symptoms is variable and does not correlate directly with the time of insult. In many cases, symptoms do not occur within days but usually develop after a period of 4–7 weeks following trauma.[14] On CT scans, chronic SDH is often viewed as a mass of mixed density with chronic and acute changes. It is inconsistent with venous bleeds that show up as an area of fresh hemorrhage[8] (Fig. 1).

Over the last few years, various pathophysiological mechanisms have been suggested. Chronic SDH is now considered a continuous process consisting of both inflammation and proliferation. In a detailed review, Edlmann et al. described the different steps involved in the pathogenesis of chronic SDH.[11] It has been deliberated that chronic SDH occurs not due to venous injury but due to injury to a highly specialized group of cells called the dural border cells. The dural border lies on the inner side of the dura mater. The dural border is made of special connective tissue cells. These cells, which when healthy, are capable of laying down fibro-cellular connective tissue. When damaged, they cause an inflammatory reaction, leading to fibrogenesis and angiogenesis.[29]

If inflammation fails to repair the dural border cells, a new dural layer forms, comprising of two layers. Of the two layers, the external layer is consistent with the dura and plays a role in the inflammatory process due to the presence of inflammatory cells, and a large number of fragile newly formed capillaries. The internal layer, next to the arachnoid mater, is not involved in inflammation.[4]

Injury to the new, fragile capillaries leads to bleeding and expansion of the chronic SDH. The bleeding is aggravated by a hyper-fibrinolytic state arising from raised levels of tissue plasminogen activator (released from the external membrane) and thrombomodulin (released from injured vessel walls).[34,20] The process of inflammation and bleeding continues to feed the hematoma until the hematoma is detected and appropriately managed.[17]

3. Reevaluating the standard approaches to management

The standard approach depends on the patient's symptoms and the size and mass effect of hematoma. Monitoring without intervention is recommended in asymptomatic cases. If the hematoma is small, spontaneous resolution may occur. Kim et al. studied 16 patients and showed that asymptomatic chronic SDH has a resolution rate of 81.3%.[22] However, even if asymptomatic, the hematoma size plays a role in treatment decisions: hematomas less than 10 mm in diameter and causing a midline shift of less than 5 mm, may be expected to resolve with conservative treatment.[12] Larger hematomas become symptomatic with time.

In symptomatic patients, decompression of the hematoma, usually by surgical evacuation, is recommended (Fig. 1). Different surgical approaches are available. The twist drill craniotomy can be performed at the bedside, and it consists of small burr holes (<10 mm). When the hematoma is large or a bedside approach is not feasible, the burr hole craniotomy is preferred. It consists of bigger burr holes (>10 mm) and is preferred by most surgeons. Craniotomy, which involves a larger bone window (>30 mm), is done in cases with an acute component, or in recurrent cases when multiple membranes prevent drainage through a simple burr hole. A subdural drainage system is usually necessary following all hematoma evacuation techniques postoperatively for a short period.[26] Unfortunately, surgical evacuation does not hinder the underlying process of inflammation and bleeding that is responsible for the hematoma. As a result, the risk of recurrence remains significant.

In a meta-analysis of over 5400 procedures by Ducret et al., the recurrence of chronic SDH was estimated to be 11.7% for burr hole craniotomies, 19.4% for open craniotomies, and 28.1% for twist drill craniotomies (Fig. 2).[9] These procedures also carry risks of complications. The same meta-analysis showed that the complication rates for these procedures were 9.3%, 3.9%, and 2.5%, respectively.

Possible complications include intracerebral hematomas, seizures, and tension pneumocephalus. Failure of cerebral re-expansion can also occur

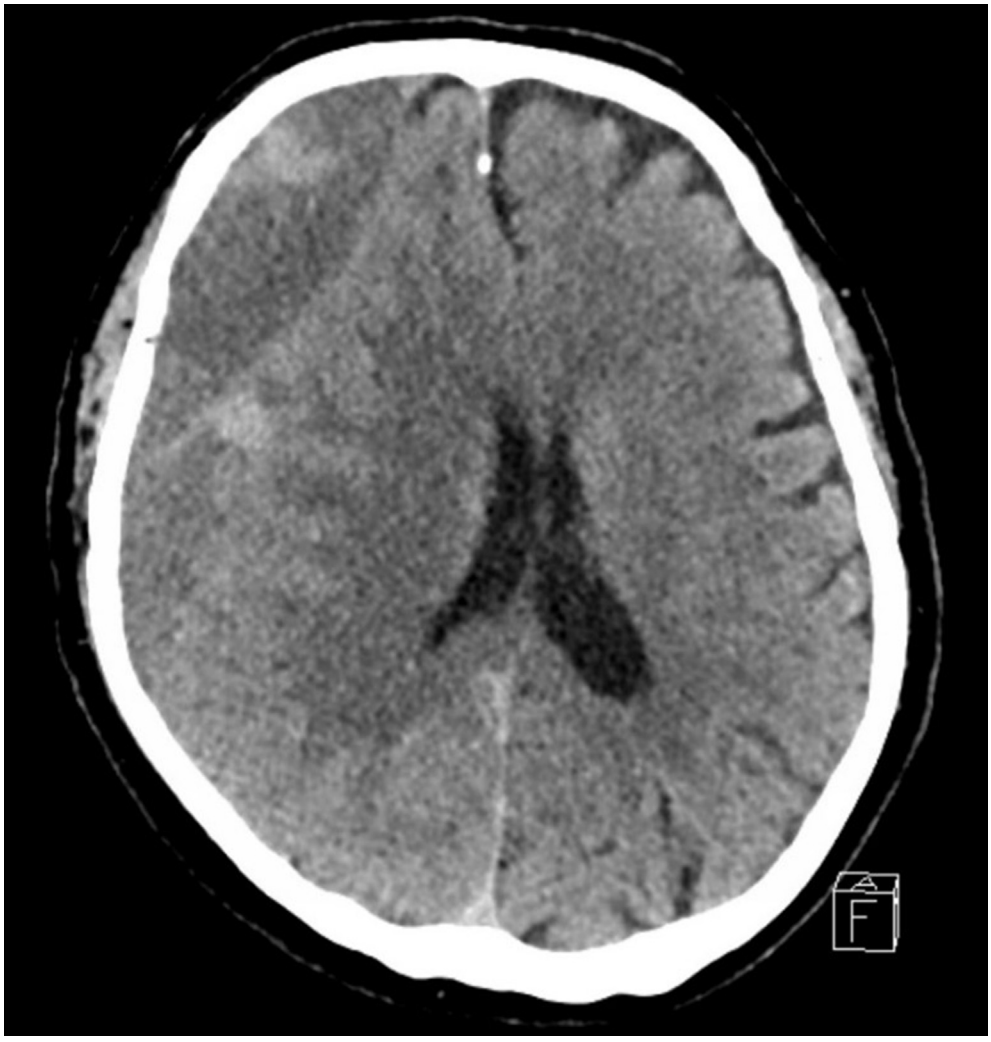

Fig. 1. Non-contrast CT head axial image showing mixed density of SDH overlying right cerebral hemisphere.

mainly due to the persistence of the dural membrane. Kung et al. established that the brain re-expanded only by about 41% on day 14 and by 60% on day 30 while re-expansion was lesser in larger, bilateral hematomas.[24] Compared to unilateral chronic SDH, bilateral chronic SDH resulted in poorer brain re-expansion leading to brain parenchymal shift, damage to blood vessels, post-operative pneumocephalus, and cerebrospinal fluid (CSF) accumulation in the hematoma cavity, with subsequently higher recurrence rates.[15,40,6]

In light of the more recent understanding of the pathogenesis of the disease, surgical procedures have been modified keeping in mind the role out of the outer dural membrane. In a meta-analysis of 17 clinical studies, Sahyouni et al. studied the effect of combined craniotomy and membranectomy in chronic SDH.[39] They found that the mean recurrence rate was 7.6%. The morbidity rate, defined as a major complication, disability, or poor health, was 6.9%, and the mortality rate was 3.7%. Even though the morbidity and mortality rates were comparable to the standard surgical techniques, recurrence rates were slightly lower with the addition of membranectomy.

In an attempt to avoid surgical complications, non-surgical modalities have been proposed. Among these, steroids have been postulated as a possible therapy as the belief is that their anti-inflammatory effects can decrease the size of the hematoma. They may also inhibit hematoma growth by inhibiting vascular endothelial growth factor (VEGF). Thotakura and Marabathina evaluated the effect of steroids in 26 patients with chronic SDH.[43] They found that 38.6% of patients did not show any improvement. An additional 19% showed initial improvement but developed recurrent symptoms after three weeks. The authors concluded that

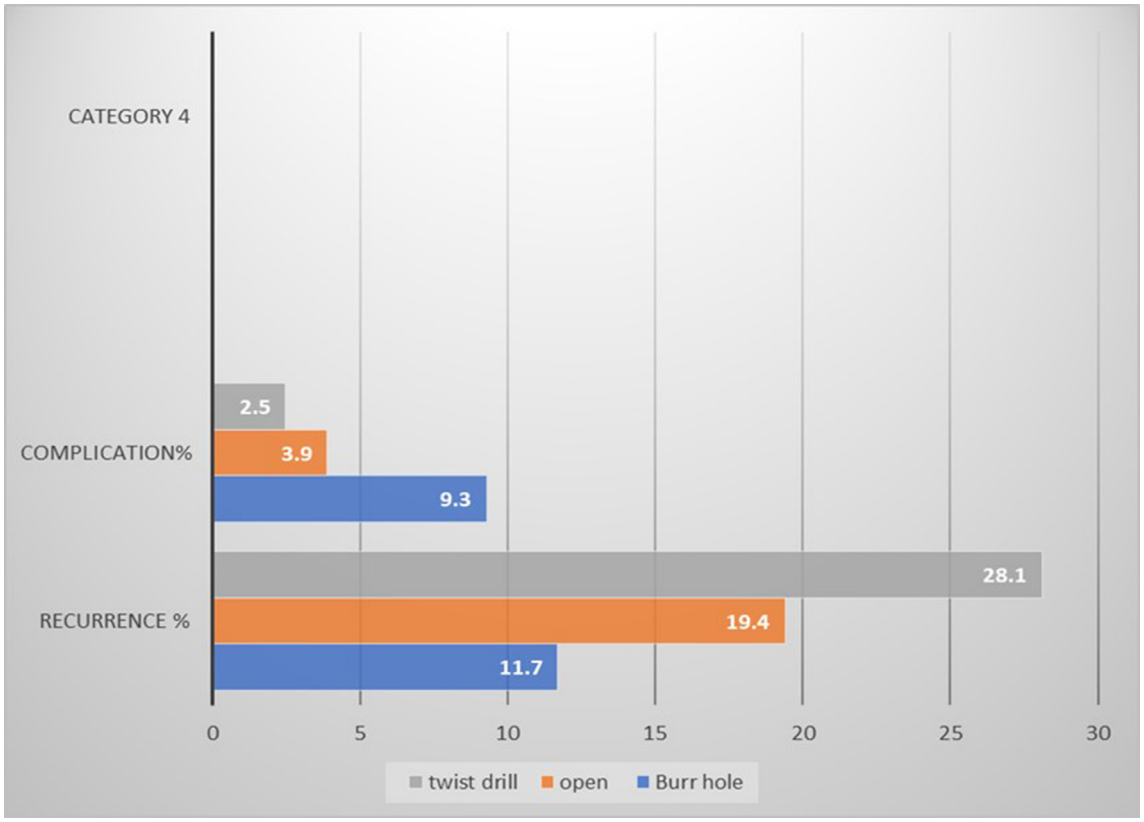

Fig. 2. Comparison of the complications and risk of recurrence in 3 different surgical techniques used for hematoma evacuation.[9]

steroids appeared to be more successful in patients with low-grade lesions.

Different treatment methods have also been tested but with little benefit. Atorvastatin can inhibit inflammation and promote vascular maturation. In one study, 25% of patients did not respond initially, while 16.7% of patients who initially responded required surgery later.[5] Another retrospective study evaluated the use of etizolam, a platelet-activating factor receptor antagonist. It reduced the need for surgery only in 46% of all patients.[18] One systematic review of all conservative management methods concluded that they could be employed only in small, unorganized lesions, where the neurological status was stable.[44]

4. The basic anatomy of MMA (middle meningeal artery)

The middle meningeal artery originates in the infratemporal fossa, from the first part of the internal maxillary artery. It enters the skull base at the middle meningeal fossa, through the foramen spinosum. It divides into four branches from anterior to posterior - the sphenoidal, frontal, parietal, and petro-squamosal branches (Fig. 3).

5. Prospect of an alternative approach

It is the middle meningeal artery (MMA) that is believed to feed the capillaries involved in the formation of chronic SDH. "Cotton wool-like staining'' of the distal vasculature was seen on digital subtraction angiography (DSA).[16] It also showed a buildup of contrast, suggesting the fragility of newly formed, immature blood vessels. Slow continuous contrast injections created the appearance of contrast outlining the hematoma. Contradictory to the strictly venous pathology proposed traditionally, other findings included direct arterial feeders.

Mino et al. evaluated four patients who presented with recurrent chronic SDH following single burr hole surgery.[33] They performed super-selective angiography of the MMA artery in these patients. They found diffuse abnormal vascular stains around the MMA, which, according to them, represented the macro-capillaries on the external membrane of the chronic SDH. Following MMA embolization, the

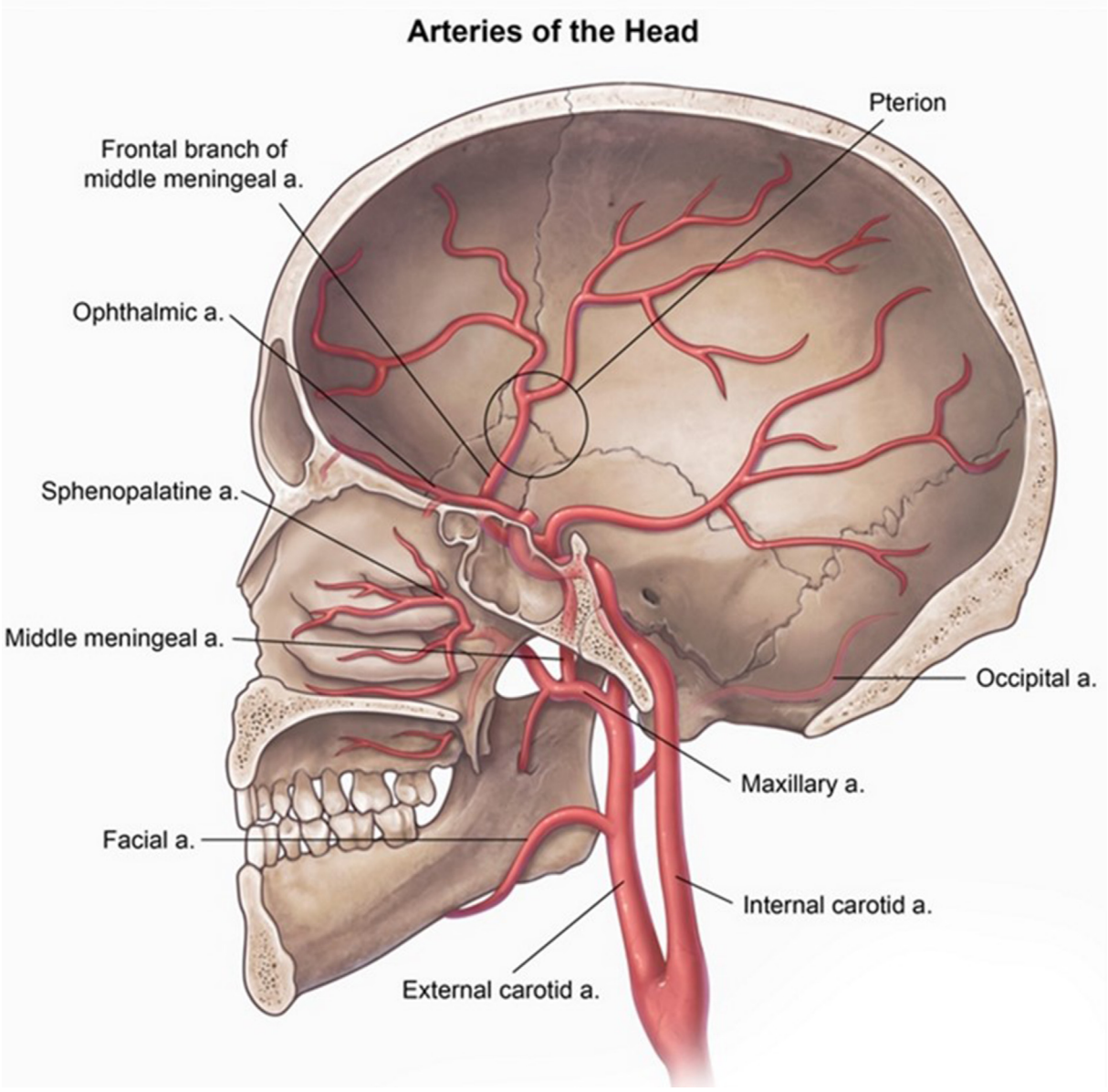

Fig. 3. The middle meningeal artery entering the cranial vault through the foramen spinosum and dividing into its respective branches.

network of abnormal vasculature around the MMA was no longer visible.

6. A lasting solution to a recurring problem

Initially, MMA embolization was used only when surgical treatment had failed. [35]Over time, surgeons have used MMA embolization combined with surgery for refractory cases that do not respond to surgery alone. In these reports, while the surgical evacuation relieves symptoms due to mass effect, MMA embolization was utilized to prevent recurrence by stopping a repeat of the chronic cycle of re-accumulation. Okuma et al. demonstrated that MMA embolization alone could also be effective in cases of refractory chronic SDH.[35] Seventeen patients who underwent this procedure were followed for over two years, with no signs of recurrence. Improvement in neurological disability scores was also seen, as measured by the modified Rankin scale (mRS), after MMA embolization. Similarly, Shotar et al. found that postsurgical embolization of the MMA reduced the recurrence rate of chronic SDH from 14% to 4%.[41]

7. Testing MMA embolization as the sole therapy

Recent studies show MMA embolization as the only single therapy, has a role in select number of cases. Link et al. prospectively followed 50 patients who underwent MMA embolization for chronic SDH. Of these, 42 patients underwent embolization as first-line treatment, and eight patients were treated as recurrent cases after surgical evacuation had failed.[27] Four patients (8.9%) developed recurrence, and surgical evacuation had to be performed, while 41 patients were stable and did not need further intervention. In 31 of these latter patients, imaging demonstrated a decrease in hematoma size greater than 50%. In the same study, MMA embolization was used prophylactically after surgical drainage in 10 patients. In a recent systematic review, Court et al. assessed 18 articles in which a total of 190 patients had undergone MMA embolization. The authors concluded that resolution occurred in 96.8% of all cases, with no post-procedure complications.[7] A multicenter trial conducted by Kan et al. involved 138 patients and 154 MMA embolization. The study employed endovascular therapy as first-line treatment in patients with either mild symptoms or midline shift per CT of under 5 mm, and as a secondary treatment in patients not meeting the above criteria, which made up 66% of the total patients. 70.8% of patients had a greater than 50% reduction in hematoma size, and only nine patients (6.5%) required surgery. Hence, MMA embolization can be considered a possibly safe and effective alternative to conventional surgery for specific patients.[19]

7.1. Is embolization superior to other management options?

As of now, three studies have conclusively proven that MMA is superior to other treatment modalities for both initial and recurrent management. Ban et al. compared conventional treatment modalities with MMA embolization in 541 patients.[3] Treatment failure occurred in only 1.4% of patients who underwent MMA embolization. However, in patients who underwent surgical removal, the failure rate was 27.5%.

Kim compared MMA embolization against burr hole craniotomy in patients with the prior surgical evacuation of chronic SDH.[21] They found that MMA embolization was successful in all except one case (3.8%), which eventually underwent spontaneous regression. On the other hand, the recurrence rate in patients who underwent craniotomy was 33.3%. Repeat craniotomy was performed in 20.8% of patients, with craniotomy needed in 12.5%. As a result, the author stated that MMA embolization must be considered the preferred treatment in recurrent chronic SDH.

In another comparative study, Matsumoto et al. reviewed various options for managing recurrent chronic SDH, defined as two or more recurrent episodes. Patients who underwent burr hole craniotomy alone had a recurrence rate of 25%.[32] No recurrence was seen in patients treated with MMA embolization or craniotomy with outer membranectomy. The authors recommended that MMA embolization should be considered for refractory patients with small hematomas. However, for larger organized hematomas, craniotomy with outer membranectomy may be a more suitable option.

Based on the above studies and six other single-arm studies, Srivatsan et al. performed a meta-analysis to assess the efficacy of MMA embolization.[42] They found that the odds of developing a recurrence after MMA embolization were 91.3% less than conventional methods. The overall recurrence rate was estimated to be 3.6%. Summary of the studies included in the review are listed in Table 1.

7.2. Technical aspects of MMA embolization

MMA embolization is a minimally invasive procedure and can be performed under moderate sedation or general endotracheal anesthesia. Arterial access is usually obtained through the common femoral artery or the radial artery, with the latter seen in more recent cases.[37] The diagnostic catheter is advanced to the common carotid artery (CCA). The vascular source of the SDH should correspond to the distribution of MMA. Under roadmap guidance, a microcatheter is advanced and carried selectively into the MMA.[28] Angiography of the MMA helps to determine the branches of MMA that supply the external membrane of chronic SDH.

Detailed knowledge of the anatomy of the external carotid artery is of vital importance. MMA supplies several cranial nerve nuclei, and its branches link the external carotid and internal carotid arteries through anastomoses. Therefore, it is imperative to be aware of its branches before an embolization is performed to prevent any serious adverse effects and disabilities.

The MMA anastomoses with the anterior falcine branch of the ophthalmic artery and the meningeal branches of the cavernous branch of the internal carotid artery (ICA). The cavernous branch (a variant balanced through anastomoses with the middle meningeal branch of the inferolateral trunk

Table 1. Summary of the studies included in the review.

Author	Year	Study design	Criteria	Aim	Patient population	Conclusions
Ban et al.[40]	2018	No-nrandomized clinical trial	**Inclusion criteria**: both genders, aged 20 years or older with chronic SDH **Exclusion criteria**: chronic SDHs with a focal location, thickness of 10 mm or less, no mass effect, underlying conditions, poor medical condition and with a life expectancy of less than 6 months	To evaluate the effect of MMA embolization on chronic SDH and compare the treatment outcomes of MMA embolization and conventional treatment	541 patients (391 male, 150 female) with chronic SDH aged 20 years or older	Embolization group had a lower treatment failure rate compared to the conventional treatment group ($p = .001$), surgical rescue was less frequent in the embolization group ($p = 0.005$); however, the treatment related complication rate was similar between the two groups ($p = 0.182$)
Kim et al.[41]	2017	Pilot Study	**Inclusion criteria**: 1) not requiring an emergent evacuation; 2) symptomatic recurrence within a short time after successful treatment; 3) elderly with brain atrophy; 4) enlarged ipsilateral MMA on magnetic resonance angiography (MRA); 5) under antiplatelet and anticoagulant therapy; 6) advanced combined medical diseases; 7) bleeding tendency or coagulopathy; and 8) hematomas with multilocular collections	To describe the safety and efficacy of MMA embolization in recurrent chronic SDHs	43 patients (30 male, 13 female) with one-time recurrence of chronic SDH, initially hospitalized for burr-hole surgery to evacuate unilateral or bilateral chronic SDHs	MMA embolization was curative with early brain re-expansion ($p = 0.003$) and a recurrence rate of 3.8% compared to 33.3% in the conventional treatment group ($p = 0.024$)
Matsumoto et al.[42]	2018	Literature Review	**Inclusion criteria**: ipsilateral hematoma identified on follow up computerized tomography (CT) and causing neurological deficits withing 3 months of the last operation -Refractory was identified as chronic SDH in patients presenting with recurrence on two or more occasions	To review the effective surgical procedures for refractory chronic SDH	14 (13 male, 1 female) patients with refractory chronic SDH presenting with two or more recurrences	The cure rate was 100% for both embolization of the middle meningeal artery with burr-hole irrigation and drainage and craniotomy, and 75% for burr-hole irrigation and drainage alone. No significant differences were identified. No further recurrences were recorded.

Okuma et al.[35]	2019	Case series	**Inclusion criteria**: intractable risk factors of chronic SDH included; 1) use of antiplatelet drugs or anticoagulants, blood coagulation disorder 2) hepatic dysfunction 3) hemodialysis 4) terminal malignancy 5) old age >80 years, cerebral atrophy, large preoperative hematoma volume (>150 mL) 6)niveau formation of hematoma 7) conditions after cerebrospinal fluid shunt 8) no placement of drain during surgery 9) postoperative residual air (>20%) 10) multiple recurrences	Reporting the usefulness of MMA embolization for intractable chronic SDH in patients with multiple intractable risk factors	17 (12 male, 5 female) consecutive patients with $\geq$2 intractable risk factors for chronic SDH	The modified Rankin Scale (mRS) score showed significant improvement at the time of discharge ($p < 0.05$) compared to score at the time of admission. MMA Embolization not associated with recurrent chronic SDH or rehospitalization in patients with intractable chronic SDH
Shotar et al.[36]	2020	Monocentric retrospective study	**Inclusion criteria:** operated by a single burr hole craniotomy for chronic SDH recurrence or a chronic SDH in the case of an independent risk factor defined as: (1) antiplatelet therapy or (2) full anticoagulation therapy or (3) coagulation disorder or (4) hepatopathy or (5) chronic alcoholism **Exclusion criteria:** eligible patients were excluded if they refused embolization procedure or were denied embolization by the attending physician because they presented in a moribund state, had contraindication to embolization procedure like severe renal dysfunction, life expectancy below 6 months.	To assess the impact on the recurrence rate of post-surgical embolization of chronic SDH in patients with a higher than average risk of recurrence	263 patients operated for a chronic SDH (191 male, 72 female)	Post-surgical embolization of the MMA reduced the recurrence rate of chronic SDH from 14% in the control group to 4% in the embolization group ($p = 0.002$)

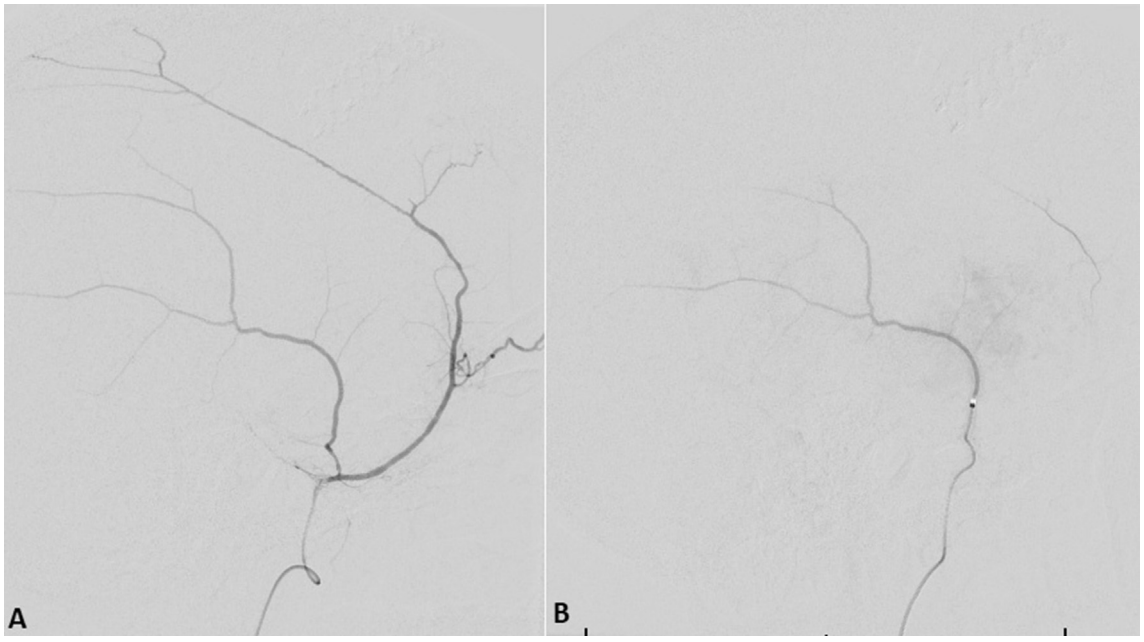

Fig. 4. Digistal subtraction angiography (DSA) images of endovascular middle meningeal artery (MMA) embolization. A: Before embolization. B: After embolization of frontal and parietal branches with polyvinyl alcohol (PVA) particles.

[ILT]) possibly supplying the Meckel cave and its associated nerves. Since the MMA can connect with the inferolateral trunk (ILT), it is also a possible "dangerous anastomosis" with the ICA. An extremely rare connection between the ILT and ICA is possible through a very long route of the sphenoid branch connection to the recurrent meningeal artery. Operators should also avoid the petrosal branch of the MMA, and if a prominent branch is identified, microcatheter is navigated past its take-off, and reflux is avoided as this branch may be the supply to vasa vasorum of cranial nerve VII within the petrous bone, as well as the greater petrosal nerve.

In addition, the microcatheter must be advanced to the sphenoid ridge before the anastomotic junctions to avoid involving the meningo-ophthalmic branches of the MMA.[25] If embolization is performed too close to the anastomoses, there is a risk of blindness or stroke. In case the vasculature is too small to navigate distally, the procedure may not be possible (Fig. 4).

7.3. Agents involved in MMA embolization

Studies conducted on MMA embolization have mostly used polyvinyl alcohol (PVA) particles suspended in opaque carrier solution.[30,3,28] However, some authors have also used coils for embolization.[48] Hashimoto et al. suggested that this approach may be feasible when there is a likelihood of anastomosis with the ophthalmic artery in the proximal part.[16] However, Fiorella and Arthur suggested that liquid embolic agents could be considered an alternative, as they would be faster and simpler to use.[12] They pointed out that PVA particles can only be viewed on the angiogram due to the opaque carrier, making distal penetration and reflux difficult to observe. However, since liquid agents are inherently radio-opaque, visualization would be easier and reflux better managed. Liquid agents like cyanoacrylates and sodium tetradecyl sulfates have been shown to produce better and more reliable results; however, they can cause inflammatory reactions and even tissue death. Therefore, these factors must be evaluated thoroughly before such agents are considered for MMA embolization. Recently, Rajah et al. have successfully used both non-adhesive (Onyx) and adhesive (cyanoacrylates) liquids for MMA embolization in the management of chronic SDH.[37]

8. Conclusions

MMA embolization is a promising treatment option for chronic SDH, in both primary and recurrent cases. Although a few comparative studies have proven its superiority, large-scale randomized controlled trials with long follow-up periods are essential to provide better evidence. Future research

should also focus on evaluating different agents that may be used for the embolization itself. Currently, most of the chronic SDH patients present in the ED for neurosurgical evaluation or are seen by primary care physicians. Select patients must be made aware of the option of endovascular therapy. The management of chronic SDH patients is complex and includes observation, medical therapy, endovascular and surgical options. Therefore, one must be cognizant of the need that successful implementation of this modality requires a whole-team approach, as the role of MMA embolization in treating chronic SDH will continue to evolve.

Authors' disclosures and conflict of interest

Hamza Hanif: Nothing to disclose.
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