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Middle meningeal artery embolization for chronic subdural hematoma

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ABSTRACT

Objectives To assess recurrence rates, procedural outcomes and patient outcomes following middle meningeal artery (MMA) embolization for the treatment of chronic subdural hematomas (cSDH).

Design Retrospective case series.

Setting Two tertiary neurosurgical referral centers in Sydney, Australia.

Participants 13 adult patients (mean age±SD, 68.5±9.5 years, 11 male) with 17 cSDHs (measuring 13.8±4.5 mm) undergoing MMA embolization alone (8/13) or with surgical evacuation (5/13) for cSDH. There were no exclusion criteria.

Interventions Embolization was performed via femoral access, using either liquid embolic, polyvinyl alcohol particles, coils, or a combination of agents. Embolization was done either as the sole treatment or with surgical evacuation.

Main outcome measures Primary outcomes were recurrence or increase in hematoma size requiring surgical evacuation. Secondary outcomes included procedural complications, hematoma size at follow-up, and patient clinical outcomes.

Results No procedural complications occurred. 12 patients were discharged home at baseline neurological function, and one was discharged to an aged care facility with significant disability. At follow-up (mean=8.7 weeks), combined embolization with surgical evacuation led to hematoma size reduction (14.3±2.6 mm to 5.7±6.5 mm, p<0.01), while embolization alone showed a stable hematoma size (13.3±5.7 mm to 10.0±8.8 mm, p=0.20). Recurrence or increase in hematoma size requiring surgical evacuation occurred in 2/13 (15.4%) patients, one of whom received only unilateral embolization, and the other received partial coiling due to the presence of dangerous collaterals.

Conclusions MMA embolization is a safe procedure that may reduce recurrence rates of cSDH when used as an adjunct to surgery or as a sole treatment. Possible reasons for treatment failure may include unilateral embolization, partial coiling, and absence of distal penetration of embolic agent. Large randomized control trials are currently in progress to assess the safety and efficacy of MMA embolization for this purpose.

INTRODUCTION

A subdural hematoma (SDH) is a collection of blood in the subdural space, the

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Middle meningeal artery (MMA) embolization for chronic subdural hematomas appears to be a safe and effective treatment when used alone or as an adjunct to surgery based on case series and small randomized controlled trials. Large randomized controlled trials are currently in progress to assess its safety and efficacy.
⇒ Reasons for treatment failure of MMA embolization are currently unknown.

WHAT THIS STUDY ADDS

⇒ This study is the largest Australian case series investigating the clinical and procedural outcomes of MMA embolization for the treatment of chronic subdural hematomas (cSDH).
⇒ The study confirms the safety of MMA embolization used as a sole treatment and in conjunction with surgery.
⇒ The study identifies and discusses potential reasons for treatment failure (which occurred in 2/13 of our patients), including presence of collateral supply of dural membranes, unilateral embolization, choice of embolic agent, and insufficient/absent distal penetration of embolic agent, among others.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE, OR POLICY

⇒ The study may influence local practice by encouraging consideration of MMA embolization for the treatment of cSDH, particularly in recurrent hematomas and high bleeding-risk or high surgical-risk patients.
⇒ The study may also influence local practice by suggesting factors that may optimize MMA embolization including bilateral MMA embolization, targeting both anterior and posterior MMA branches, and distal penetration of embolic agent.
⇒ The study may influence future research including investigating the optimal embolic agents and optimal treatment protocols, including comparisons between MMA embolization alone versus with surgery, and unilateral versus bilateral embolization.
⇒ The study raises the issue of no clear guidelines and indications for MMA embolization and may influence future local policy.

space between the dura and arachnoid mater. The most common cause of an acute SDH is trauma resulting in tearing of cortical bridging veins. Most acute SDHs resolve over time via resorption; however, a fraction of cases encapsulate and become a chronic SDH (cSDH), defined as being at least 3 weeks old. The overall incidence of cSDH is between 17.6 and 20.6 per 100 000 people,[1 2] being highest in the elderly. Risk factors for its development include advanced age, male sex, use of anti-coagulants, alcoholism, and coagulopathies. The incidence of cSDH is increasing due to an ageing population and the increased use of anti-coagulants in the community. cSDH may present with focal neurological signs including weakness and altered sensation secondary to the mass effect of the hematoma but can also present with non-specific, more insidious symptoms such as headaches, nausea, cognitive impairment, confusion, and falls. cSDH may also be asymptomatic, especially in the elderly with significant cerebral atrophy. cSDH has a significant morbidity and mortality, with the median patient survival being only 4.0 years after diagnosis.[3] cSDH has been named a sentinel health event, being not only a marker of prior ill health, but also a trigger for further deterioration.

Small and asymptomatic cSDHs may be managed conservatively with close monitoring of hematoma size and patient symptoms, as well as withholding anti-coagulant medications (which in itself carries significant risks). The standard management of cSDH requiring intervention is surgical evacuation (via burr hole or craniotomy) with or without a drain. Indications for surgical evacuation include symptomatic hematoma, maximal hematoma thickness >10 mm, and a midline shift >7 mm.[4] However, the recurrence rates post-evacuation are unfortunately high, estimated to be between 5% and 30%.[5] Surgical evacuation of a hematoma provides symptomatic relief by reducing the local mass effect; however, it does not target the cause of cSDH reaccumulation.

The traditional theory of the pathophysiology of cSDH recurrence involves the repetitive tearing of dural bridging veins between the brain and draining dural sinuses.[6] This theory, however, does not explain why accumulation of blood takes several weeks to occur and why blood often accumulates away from the site of bridging veins. The cSDH can also grow in size without acute hemorrhage, and with only minor, or even absent history of trauma, thus suggesting that repetitive venous bleeding is not the only source of cSDH growth. The new theory of cSDH formation proposed by Edlmann *et al* involves the injury of the border cell layer, a specialized dural connective tissue, whose repair triggers an inflammatory response, angiogenesis, fibrinolysis and surrounding membrane formation.[7] The new membrane blood vessels are fragile, resulting in recurring blood and fluid exudation, which further potentiates and propagates the cycle of inflammation and presence of chronic subdural hemorrhage. Histological studies have found small middle meningeal artery (MMA) branches anastomosing with the neovessels in the outer membranes of cSDHs, suggesting a possible mechanism for hematoma recurrence.[8]

The purpose of MMA embolization is to occlude the arterial supply of the dural membrane, aiming to prevent reaccumulation of the hematoma. The first reported case of a successful MMA embolization for the treatment of cSDH was in the year 2000.[9] The procedure gained widespread interest after a 2018 trial by Ban *et al* which proposed endovascular MMA embolization as an adjunct to surgery or as a sole treatment may be more effective than traditional surgical treatment for preventing cSDH recurrence.[10] Multiple large randomized controlled trials are currently in process to assess the effectiveness of MMA embolization for this purpose.[11–13] The aim of this study is to evaluate the early clinical experience and procedural outcomes of MMA embolization as either a sole treatment or in conjunction with surgical evacuation for the treatment of cSDH and to discuss possible reasons for treatment failure.

METHODS

The patient records from two large neurosurgical centres in Sydney, Australia, Westmead Hospital and Royal North Shore Hospital, were analyzed. Adult patients presenting with acute on chronic or chronic SDH and who underwent endovascular MMA embolization were included in the case series. The patients were identified via a data search of all operation reports between March 2019 and November 2022. cSDH was diagnosed based on the appearances on a non-contrast CT brain (crescent-shaped iso- or hypo-dense extra-axial collection) on presentation.

Patient characteristics of interest included sex, age, past medical history, use of anti-coagulant and/or anti-platelet agents, symptoms, and neurological signs (including Glasgow Coma Score; GCS) documented on presentation. Characteristics of interest of the SDH included side (left, right, or bilateral), etiology (traumatic or spontaneous), size on presentation (measured as maximal width of hematoma in mm on non-contrast CT brain), and whether surgical evacuation has previously occurred. Procedural information, including access site, whether the MMA embolization was done in conjunction with surgical evacuation or a stand-alone treatment, whether bilateral or unilateral embolization was performed, and embolic agent used, were obtained from operation reports and patient electronic medical records. The primary outcome was treatment failure, with failure defined as a recurrence or increase in size of the cSDH requiring surgical evacuation. Secondary outcomes included procedural complications, size of cSDH at follow-up (measured as maximal width of haematoma in mm on a non-contrast CT brain), neurological outcome (inferred from persisting deficits as documented in electronic medical records at follow-up), and functional outcome (inferred from discharge

destination). The patients were followed up at non-standardized time frames, as deemed appropriate by the treating team.

Data are expressed as a percentage or a frequency for discrete variables (including sex, comorbidities, and side and etiology of cSDH) and as a mean±SD for continuous variables (including age and size of hematoma). A comparison of means was performed using an independent t-test, and probability values (p) <0.05 were considered statistically significant. Statistical analyses were performed using an online calculator, GigaCalculator (www.gigacalculator.com).

As this study is a retrospective case series with no identifiable patient information, ethics approval was not sought.

RESULTS

Patient demographics

13 patients fulfilled the inclusion criteria (table 1). The mean age at presentation was 68.5±9.5 years (range 50–79 years). Two patients were female (mean age 63.0 years), and 11 were male (mean age 69.5 years). Five patients (38.4%) were on regular anti-coagulants (three warfarin, two apixaban), two patients (15.4%) were on a regular anti-platelet agent (one aspirin, one clopidogrel), and six patients (46.2%) were on neither agent at presentation. The most common presenting symptoms included headache (10/13; 76.9%), nausea (3/13; 23.1%), and confusion (2/13; 15.4%). The majority of patients (10/13; 76.9%) presented with a normal level of consciousness, while 3/13 (23.1%) presented with a reduced level of consciousness (GCS<15). Five patients (38.5%) had focal neurological signs on physical exam (including dysphasia, oculomotor nerve palsy, and limb weakness).

Common indications for MMA embolization in our population included re-accumulation of hematoma despite previous surgical evacuation in seven patients (four patients had previously undergone surgical evacuation once, three patients twice), the need to resume anti-coagulants for significant medical comorbidities (Factor V Leiden with previous deep vein thromboses, a pulmonary embolus, two ischemic strokes secondary to atrial fibrillation, and two mechanical heart valves) in six patients, and hoping to avoid surgical management in 1 patient with thrombocytopenia (platelets of 50×10^9/L on presentation) secondary to acute myeloid leukemia (AML).

Subdural hematoma (SDH) characteristics

13 patients presented with a total of 19 cSDH (table 1); six had bilateral SDHs, while seven had a unilateral SDH (three right-sided, four left-sided). Two hematomas were untreated as the ipsilateral MMA could not be embolized: one due to ipsilateral external carotid artery occlusion and the other due to being an ophthalmic origin MMA (high-risk variant). Of the 13 patients, seven had a traumatic cSDH, while six had a spontaneous cSDH. 6/13 patients presented with a primary cSDH, having not had previous surgical evacuation. Of those six

Table 1 Patient demographics, comorbidities, characteristics of subdural hematomas, and procedural factors

Patient demographics (n=13)	
Male	11 (84.6%)
Age (years)	68.5±9.5
Female (years)	63.0±7.0
Male (years)	69.5±9.6
Anti-coagulant use	5 (38.5%)
Anti-platelet use	2 (15.4%)
Comorbidities (n=13)	
Hypertension	6 (46.2%)
Hypercholesterolemia	6 (46.2%)
Ischemic heart disease	5 (38.5%)
Atrial fibrillation	4 (30.8%)
Type 2 diabetes mellitus	3 (23.1%)
Mechanical heart valves	2 (15.4%)
Thrombocytopenia	2 (15.4%)
Chronic kidney disease	1 (7.7%)
Liver disease	0
Chronic alcoholism	0
cSDH characteristics	
Etiology	
Traumatic	7/13 (53.8%)
Spontaneous	6/13 (46.2%)
Laterality	
Bilateral	6/13 (46.2%)
Unilateral	7/13 (53.8%)
Size on presentation (mm)	
All cSDH (n=17)	13.8 ± 4.5
MMA embolization alone (n=9)	13.3±5.7
MMA embolization + surgery (n=8)	14.3±2.6
Primary cSDH	6/13
MMA embolization as sole treatment	4/6
MMA embolization with surgical prophylaxis	2/6
Recurrent cSDH	7/13
One previous evacuation	4/7
Two previous evacuations	3/7
Embolization without further evacuation	4/7
Embolization with further evacuation	3/7
MMA embolization (n=13)	
Femoral access	13 (100%)
Onyx liquid embolic (sole agent)	6 (46.2%)
PVA particles (sole agent)	3 (23.1%)
Coils (sole agent)	0
Combination of agents	4 (30.1%)

.cSDH, chronic subdural hematoma; MMA, middle meningeal artery; PVA, polyvinyl alcohol.

patients, four patients received MMA embolization as a sole treatment, and two received surgical evacuation in conjunction with MMA embolization during that admission. 7/13 patients presented with a recurrence of a cSDH, with four patients having had one previous evacuation and three patients having had two previous surgical evacuations. Of those seven patients, three had received surgical evacuation with MMA embolization, while four had received MMA embolization alone during that admission. Thus, 8/13 patients received MMA embolization alone, and 5/13 patients received MMA embolization with surgical evacuation at admission. The mean maximum width of all cSDHs was 13.8±4.5 mm on presentation as measured on a non-contrast CT brain. The size of cSDHs that received MMA embolization as a sole treatment in this presentation (9/17) was 13.3±5.7 mm, while the size of cSDHs that received MMA embolization with surgical evacuation for symptomatic management in this presentation (8/17) was 14.3±2.6 mm.

Procedure

A total of 17 cSDH haematomas in 13 patients were treated with MMA embolizations (table 1). Femoral artery access was used in all cases. The most commonly used embolic agent was onyx liquid embolic (used as a sole agent in 6/13 patients); however, PVA particles (sole agent in 3/13 patients), coils, or a combination of agents were also used. All embolizations were technically successful. There were no documented procedural complications.

Radiological outcomes

12/13 patients were followed up in clinic with a progress non-contrast CT brain, with last follow-up occurring between 4 and 19 weeks post embolization (mean=8.7 weeks). 1/13 patient presented to the emergency department prior to their planned follow-up with symptoms secondary to a re-accumulation of the cSDH at 5 weeks post-MMA embolization. The mean size of the 17 treated cSDH on a non-contrast CT brain at follow-up was 8.0±8.1 mm. Of those, the eight hematomas receiving MMA embolization with surgical evacuation had a mean size of 5.7±6.5 mm on follow-up, and the nine hematomas receiving MMA embolization alone had a mean size of 10.0±8.8 mm on follow-up (table 2).

Clinical outcomes

2/13 (15.4%) patients had a recurrence/increase in size of the cSDH requiring surgical evacuation. Of those, one patient in their 50s with mild thrombocytopenia (platelets of 104×10^9/L on initial presentation) presented with a spontaneous left-sided cSDH and received unilateral MMA embolization with PVA particles in conjunction with surgery after a recurrence of their cSDH post-previous surgical evacuation. The second recurrence occurred in a patient in their 70s who presented with traumatic bilateral cSDH that had received bilateral MMA embolization with onyx liquid embolic and coils as a sole treatment, with no previous surgical evacuation. This patient received partial coiling due to the presence of dangerous ophthalmic collaterals, with an attempt at distal penetration with embolic agents being deemed too dangerous. Neither of the patients was on regular anti-coagulant or anti-platelet medications on admission or on discharge.

Table 2 Procedural, radiological, functional, and clinical outcomes

Procedural outcomes	
Technically successful embolization	13/13 (100%)
Procedural complications	0/13 (0%)
Discharge destination	
Home (or premorbid accommodation)	12/13 (92.3%)
Residential aged care facility	1/13 (7.7%)
Clinical outcomes	
Focal deficits	1/13 (7.7%)
Mortality	0/13 (0%)
Recurrence requiring evacuation	2/13 (15.4%)
Size of cSDH	
Mean last follow-up time (weeks)	8.7
Size (mm) at follow-up	
All treated cSDH (n=17)	8.0±8.1
cSDH - MMA embolization alone (n=9)	10.0±8.8
cSDH - surgery with embolization (n=8)	5.7±6.5

cSDH, chronic subdural haematoma; MMA, middle meningeal artery.

Functional outcomes

Of the 13 patients, 12 were discharged back home or to their previous accommodation (with one requiring new services at home), and one was discharged to a residential aged care facility (table 2). This patient had sustained an ischaemic stroke prior to MMA embolization while withholding regular anti-coagulation for atrial fibrillation and had persisting focal neurological deficits and required PEG feeds. 11 patients were at their neurological and functional baseline at follow-up, and one patient had seizures controlled with levetiracetam without other neurological deficits. No mortalities were recorded at follow-up.

DISCUSSION

Despite the increasing use of MMA embolization for the treatment of cSDH, there are still no clear guidelines for when it should be considered, and indications vary between centres. The indications for MMA embolization in our population included recurrence post-previous surgical evacuation, the need to commence or resume anti-coagulants for other medical comorbidities, and risk

factors for high recurrence rates (which include coagulopathies, hepatic impairment, and alcoholism). MMA embolization was used as a sole treatment in patients without focal neurological symptoms, and patients for whom surgical, anesthetic, and bleeding risks associated with surgical evacuation were deemed to be too high. MMA embolization however does not replace surgery in alleviating the mass effect in symptomatic cSDH and was therefore used as an adjunct to surgery in patients with focal neurological signs and symptoms.

Our population had a mean age of 68.5±9.5 years and included predominantly males (84.6%), representative of the typical demographics of cSDH patients.[1 2] We found the recurrence rates for a cSDH requiring surgical evacuation to be 15.4% (2/13); comparable to rates of cSDH recurrence following traditional (ie, surgical) treatment of cSDH (5%–30%)[5] and higher than the recurrence rates found in other studies. A 2021 meta-analysis of 20 cohort studies and case series including 1416 patients found a recurrence rate of 4.8% and the need for surgical rescue of 3.2% following MMA embolization, compared with a recurrence rate of 21.5% and need for surgical rescue of 16.4% following traditional surgical treatment.[14] The rate of complications was similar between the groups: 1.7% in the embolization group and 4.9% in the traditional surgical treatment group.[14] There were no procedural complications recorded in our population, in keeping with the low complications rates found by other studies.

The mean size of all 17 treated cSDH on a non-contrast CT brain at follow-up was 8.0±8.1 mm, significantly lower (p=0.01) than the 13.8±4.5 mm on presentation. Of those, cSDHs that received MMA embolization with surgical evacuation (8/17) had a significant reduction in size at follow-up (size on presentation=14.3 ± 2.6 mm, size on follow-up 5.7±6.5 mm, p<0.01), while cSDHs that received MMA embolization alone at admission (9/17) were stable in size (size on presentation=13.3 ± 5.7 mm, size on follow-up=10.0 ± 8.8 mm, p=0.20). This demonstrates that the significant reduction in the hematoma size was largely attributable to surgical evacuation rather than MMA embolization. The resorption of cSDHs is slow, with one study finding that complete or near complete resolution of a cSDH occurs in only 18% of patients at 30 days, 63% at 90 days, and 92% at 180 days after MMA embolization.[15] Hence, a longer follow-up time frame than our mean of 8.7 weeks is likely necessary to see significant haematoma resorption in the group receiving MMA embolization as a sole treatment. Hematoma resorption with MMA embolization alone is slow, and embolization should therefore be used in conjunction with surgical evacuation in symptomatic patients to relieve mass effect. A small randomized controlled trial of 46 cSDH patients managed with either surgical management alone or in conjunction with MMA embolization showed an equal recurrence rate (one patient in each group).[16] However, the group who underwent surgical evacuation with MMA embolization had a significantly higher degree of haematoma resorption at the 3-month follow-up than those post-surgery alone (mean difference 17.5 mL, p=0.015), likely by preventing further hemorrhage and fluid exudation.[16]

2/13 patients in our study had a recurrence of cSDH requiring further surgical evacuation. Potential reasons for failure of MMA embolization include collateral supply of dural membranes, unilateral embolization, choice of embolic agent, and insufficient/absent distal penetration of embolic agent, among other anatomical and technical factors. The supratentorial dura mater is predominantly supplied by the MMA; however, it receives collateral supply from the meningeal branches of the anterior ethmoidal, posterior ethmoidal, and ophthalmic artery, as well as the accessory meningeal, ascending pharyngeal and direct internal carotid artery branches. The typical MMA territory may also receive supply from the anterior meningeal, posterior meningeal, occipital, and temporal arteries that may contribute to cSDH membrane neovascularization and failure of treatment. Collateralization from the contralateral MMA has also been identified as a potential reason for treatment failure, suggesting bilateral embolization should be attempted even in cases of a unilateral hematoma.[17] Hubbard *et al* have demonstrated the formation of contralateral MMA collaterals resulted in hematoma recurrence following only ipsilateral MMA embolization and achieved treatment success in 2/2 patients by later embolizing the contralateral MMA.[17] Unilateral embolization therefore may have contributed to treatment failure in one of our patients. Sack *et al* suggest repeating angiography to assess for remaining collaterals post-treatment failure and suggest considering alternative embolic agents with the second treatment.[18] Chihara *et al* describe a case of a failed MMA embolization due to the presence of an organized hematoma that delayed healing and allowed time for the development of new collaterals, ultimately resulting in hematoma reaccumulation at 3 months, requiring a craniotomy with capsulectomy for definitive treatment.[19] An organized hematoma is diagnosed on MRI but not CT, suggesting that an MRI may be a useful investigation following an unsuccessful embolization.

There was significant variation in treatment regimens used in our cases, including the choice of embolic agent, whether unilateral or bilateral embolization was performed, and whether embolization was performed alone or in conjunction with surgical evacuation. The most commonly used embolic agent in our series was onyx liquid embolic (sole agent in 6/13 patients). The most commonly used embolic agent in other studies is PVA particles,[14] and the best embolic agent for the purpose of MMA embolization for cSDH is currently unknown. Distal penetration of embolic agent into both MMA branches (ie, targeting anterior and posterior branches) has also been shown to result in higher rates of hematoma resolution (22/29, 76%) than following single branch embolization without distal penetration (4/12, 33%).[20] Coiling does not provide distal penetration, making it a suboptimal embolic agent, and the need for partial coiling may explain the reason for treatment failure in our second

patient. Failure of MMA embolization had also been shown to occur more often when MMA embolization is performed as a sole treatment (8/90, 8.9%) than when used in conjunction with surgical evacuation (6/195, 3.9%), suggesting that the presence of a subdural collection contributes to hematoma re-accumulation.[21] Di Cristofori *et al* have suggested this may be due to the pro-inflammatory contents of the hematoma.

A significant number of patients included in this study were complex, with some having multiple significant medical comorbidities. Some patients underwent MMA embolization alone as they were deemed to not be surgical candidates due to factors including a patient with thrombocytopenia (platelets of 50×10^9/L) secondary to AML and the need for early anti-coagulation in 6 patients due to a recent ischemic infarct, pulmonary emboli, and mechanical heart valves. MMA embolization was performed successfully and safely without re-accumulation of the cSDH at follow-up in each of these seven complex cases. There is a general consensus among neurosurgeons that anti-coagulant and anti-platelet medications should be stopped, and anti-coagulation rapidly reversed, in patients presenting with a cSDH; however, no clear guidelines exist about the safe timing to restart such medications. MMA embolization may allow their resumption much earlier than with surgical management alone.

The small sample size is a major limitation of this study; however, it is the largest Australian case series on MMA embolization for the treatment of cSDH. Being a retrospective study, we relied on medical records to ascertain clinical information, with the quality of record-keeping being variable. We also had to use proxy markers of functional outcome based on documented neurological deficits, persisting symptoms and discharge destination. There was also a large amount of variation in the treatment techniques used between our patients likely reflecting clinical equipoise on optimal technique. Another limitation of the study was that follow-up timeframes were not consistent, with the last follow-up occurring anywhere from 4 to 19 weeks; hence, the hematoma size measured at follow-up was highly variable. A longer standardized last follow-up time frame (eg, 6 months), to ascertain if complete or near-complete resolution of cSDH has occurred, would have been optimal.

CONCLUSION

MMA embolization is a safe procedure with no complications reported in our population. We found hematoma recurrence requiring surgical evacuation following MMA embolization for cSDH to be 15.4% (2/13 patients). Potential reasons for treatment failure are broad and may include anatomical, patient, and technical factors. In our study, treatment failure occurred in a patient receiving unilateral embolization and a patient receiving partial coiling due to the presence of dangerous ophthalmic collaterals. Optimization of MMA embolization may therefore include bilateral MMA embolization, targeting both anterior and posterior MMA branches, and distal penetration of embolic agent. Large randomized control trials are currently in progress to assess the safety and efficacy of MMA embolization for the treatment of cSDH. Further research into the optimal embolic agents, treatment protocols, and investigations post-treatment failure are required.

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