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Review Article

Middle meningeal artery embolisation: The review of a new treatment for chronic subdural hematomas

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ABSTRACT

Background: This is a literature review aiming to provide an update about the recent findings related to the efficacy of middle meningeal artery embolization (MMAE) in the treatment of chronic subdural hematomas (cSDHs), comparison with conventional therapy and deduction of the current recommendations and indications.

Methods: The literature is reviewed using a search through the PubMed index using keywords. Studies are then screened, skimmed, and thoroughly read. 32 studies fulfilled the inclusion criteria and are included in the study.

Results: Five indications for the application of MMA embolization (MMAE) are deducted from the literature. The usage as a preventive measure after surgical treatment of symptomatic cSDHs in patients with a high risk of recurrence and the usage as a standalone procedure has been the most common reasons for indication of this procedure. Rates of failures for the aforementioned indications have been 6.8% and 3.8%, respectively.

Conclusion: The safety of MMAE as a procedure is regarded as a general theme in the literature and can be considered for future applications. Usage of this procedure in clinical trials with more patient segregation and timeframe assessment relative to surgical intervention are recommendations of this literature review.

Keywords: Burr hole, Chronic subdural hematomas, Elderly population, Inflammation, Middle meningeal artery embolization, Prevention, Recurrence

INTRODUCTION

Definition of subdural hematoma

Chronic subdural hematoma (cSDH) is defined as a liquefied hematoma in the subdural space.[68] A cSDH has a characteristic outer membrane that can be up to 10 mm in thickness.[75] It is usually linked to a recent incident of cranial trauma.[18] cSDH appears as a crescentic mass with hypodense to isodense consistency on cranial computed tomography.[75] It requires clinical

and radiological experience to discern the difference between cSDHs and hygromas.[1] A subdural hygroma contains cerebrospinal fluid (CSF) and bleeding could have occurred at some point.[68] Hence, it is termed xanthochromic. Dehiscence of the subdural space and the arachnoid space dictates a spectral variance of cSDHs from effusions to hygromas and eventually actual bleeding cSDHs.[48] The latter consists due to the acuity of the bleeding a separate spectrum of cSDHs.[48]

Anatomy and histology

cSDHs are encapsulated flattened structures that form within the virtual dural space [Figure 1]. In 19% of the case, cSDHs are bilateral.[56] The median fluid volume in cSDHs was 96 mL (range 33–270 mL) in 28 patients operated on in one series and 93 mL (range 20–170 mL) in another series of 19 patients with 22 cSDHs.[35] The aforementioned outer membrane of cSDHs closely resembles the granulation tissue that develops after injury to the dermis.[35] Neoangiogenesis from dural capillaries and the proliferation of marginal cells of the dural layer lead to the formation of the outer cSDH layer and promote bleeding.[44] Histologically, this membrane comprises fibroblasts, myofibroblasts, mast cells, and eosinophils. Richness in thin-walled sinusoidal blood vessels is evident along with smooth muscle cells and is reported.[7,44] With a diameter of 80 μm, endothelial fenestrations, gap junctions, and an incomplete basement membrane and pericyte layer, those vessels are susceptible to spontaneous bleeding or secondary to inflammatory and fibrinolytic activity in the cSDH fluid.[9] Evidence of erythropoiesis in the extracellular space as erythrocytes and in the cSDHs outer membranes as erythroblasts is reported.[7]

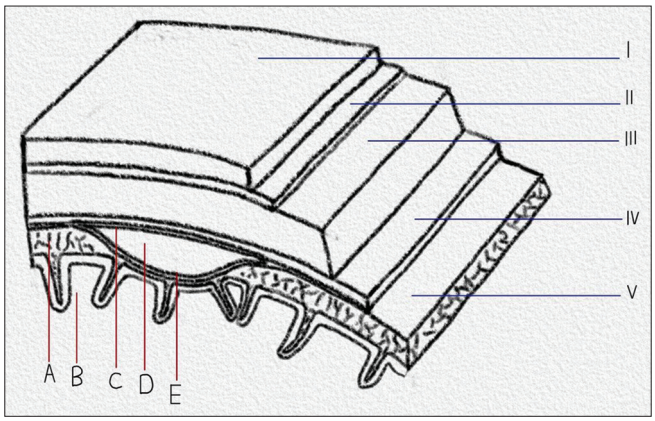

Figure 1: Drawn presentation showing the layers from the scalp to the cerebral cortex with a space-occupying subdural hematoma. Courtesy of the author Ali Msheik. (A) The arachnoid layer of the cerebral Dural membranes in coronal view, (B) The cerebral cortex, (C) The outer layer of the chronic subdural hematoma, (D): The content of the chronic subdural hematoma, (E) The inner layer of the chronic subdural hematoma, (i) Scalp, (ii) Periosteum layer of the skull, (iii): Skull bone, (iv) Dura matter, (v) Arachnoid layer of the Dural membranes in sagittal view.

Evolution

Being of embryonic mesodermal origin, tear of the cell layer of the meninges dictates a similar response to wound healing.[12] Hemorrhage promotes platelet activation and the subsequent steps of the coagulation cascade. Attempts to stage cSDHs according to levels of factors secreted and implemented in the coagulation cascade have failed due to the recurrent cycles of bleeding within a Csdh.[33] As one coagulation and fibrinolysis cycle is progressing, another cycle can start and variations within the same cSDH histologically and immunochemically can manifest.[33] When the initial bleeding starts, hemolysis pursues. The inner layer of the cSDH, which is the initial dural layer, is mainly avascular. The subsequent formation of granulation tissue, in the presence of platelet growth factors, allows the formation of a dural outer layer.[33] Neovascularization promotes vascular supply to this layer. Tissue plasminogen activator (tPA) leads to fibrinolysis through plasmin activation and hence can lead to rebleeding. The stage at which the cSDH is imaged and monitored belongs to a wide spectrum.[33] Differentials for a hypodense crescentic collection include subdural hygroma (discussed earlier) and subdural empyema.[38] Definitive diagnosis is mainly through magnetic resonance imaging and clinical history. A cSDH starts as an acute subdural hematoma.[72] Typically, on computed tomography (CT) scans, the latter is hyperdense.[65] If the cSDH started from hygromas, the latter appears a homogenous low-density collection of cranial CT scans.[65] This discrepancy in intensity relates to the presence and concentration of hemoglobin, which is hyperdense on CT scans, and signifies the acuity of the hematoma and being of a sanguineous consistency.[65] Degradation of hemoglobin with time delineates the progression of the cSDH. Hence, a cSDH turns less dense with time. Rebleeding shows as a cSDH with layers of different intensities 2 of 17.[65] Hence, it is laminar [Figure 2]. Complete fibrinolysis and resolution of the cSDH are termed trabecular cSDH.[65]

Correspondence of CT appearance with classification and prognosis

Postsurgical risk for recurrence and hematoma volume increase may be correlated to the CT scan features obtained before surgical intervention, that is, low risk is attributed to the homogenous appearance of cSDHs on CT scan.[79] Due to the chronicity of the SDH, ideally, it may appear as a hypodense. Radiologists[79] can miss bilateral cSDHs. However, failure of the convexity of the sulci to reach the inner table of the skull, and the medial displacement of the white matter-gray matter interface are indicative of the presence of cSDHs. The highest risk of progression of cSDHs is attributed to laminar

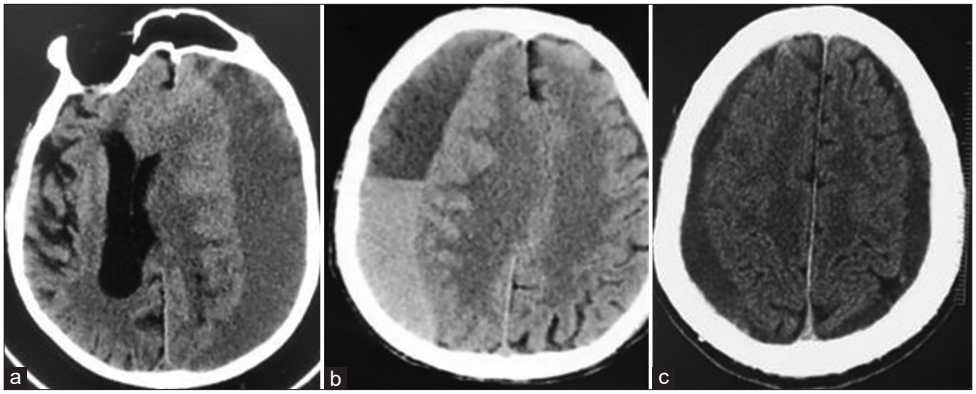

Figure 2: Axial cuts of cranial computed tomography scans showing different stages of chronic subdural hematomas (cSDHs). (a): Left hypodense cSDH, (b): Right laminar cSDH, (c): Bilateral hygroma. Courtesy of ZHUMC radiology department, Jnah, Beirut, Lebanon.

and separated cSDHs.[32] As previously aforementioned, the trabecular stage represents a mature stage of cSDHs and hence has a low risk of progression or re-bleeding.[32]

Causes of subdural hematoma

Bleeding

Tear of bridging veins as they traverse the dural cell layer is the most accepted cause of cSDHs.[8] Inertial brain injury secondary to direct trauma both major and minute is reported in two-thirds of patients and can provoke tearsin those fragile vessels.[8] Support of this theory is surgical findings describing veins with clotted ends recovered after the removal of the cSDHs outer layer.[8,15]

The transformation from hygroma

Formation of a cSDH as a progression from a subacute hygroma is another hypothesis advocated by Japanese neurosurgeons.[75] In the 1960s and 1970s, the latter presumed that trauma provoked dural cell layer tears, which led to CSF leak, which in terms promoted outer granulation tissue formation and a wound healing response.[52] Transformation into cSDHs was theorized to have been due to bleeding into the preformed hygromas due to subsequent tear of bridging vessels.[75] Support for this theory was attained by the prevalence of thin subdural neomembranes in 4% of consecutive autopsy specimens.[36] However, this does not hinder the possibility of unreported trauma in the medical history of these patients.[36]

Enlargement by osmotic and oncotic pressures

Indifference in osmolality and oncotic pressure between cSDH fluid, plasma, and CSF limits the credibility of the theory supporting the role of either in the enlargement of cSDHs.[58] On the other hand, the fact that a wound healing process along with the coagulation cascade is well-documented process in cSDHs development, an undeniable role of transudation and exudation must be considered in the evolution of cSDHs.[58]

Epidemiology of cSDH

The incidence of cSDHs is not well known and varies between operated and non-operated cases. Ranging from 1.3 to 5.3/100,000/year for operative cases, the incidence naturally increases to 8.2 up to 48/3 of 17 100,000/year.[71] Compared to the results of the Helsinki study in 1975, there is a rough 25 times increase in incidence.[71] This can be attributed to the advances in medical imaging, the increase in the aging population, and the increasing use of anticoagulants and antiplatelet medication.[66,71] This is further fortified by the fact that the mean age of diagnosis is about 75 years of age for males and 74 years of age for females. Of note, male predominance is widely reported in the literature with a ratio of 2:1 up to 5:1.[54] Factors associated with increased risk of cSDH are age, alcoholism, male sex, use of oral anticoagulants (OACs), neurological and systemic illnesses associated with brain atrophy, hemodialysis, and conditions associated with low intracranial pressure and dural tear.[10,31,34,61]

Treatments for cSDH

Treatment of cSDH requires prior assessment of patients on an individual basis. The symptomatology of patients is key in determining the urgency and the indication for surgical intervention. Typically, patients received at the ED for trauma or acute development of neurological compromise are diagnosed or incidentally found to have cSDH.[4] History of trauma, neurological compromise, the acuity of the symptoms, and the medical comorbidities associated with the condition of the patients of whom the elderly population is the vast majority govern the decision-making process.[19] Respiratory diseases, cardiac sufficiency, renal compromise, old age,

and the need for OACs and antiplatelet medication dictate the necessity for pre-surgical evaluation and preparation of patients to whom surgery is considered indicated.[4,19]

Correction of coagulopathy and thrombopathy

Reversal of coagulopathy instilled by OACs and antiplatelet medications, liver insufficiency, and other coagulation disease is essential for the prevention of further expansion of cSDHs and to allow control of blood loss after surgical evacuation of symptomatic cSDHs.[20] This can be done by utilization of fresh frozen plasma (FFP) for life-saving procedures and interruption of medication for few days before elective surgical evacuation.[20,37]

Adjuvant treatments

Steroids and anti-epileptic medications are two main medical therapies adjuvant to surgical evacuation used in managing symptomatic cSDHs. In 2021 lesser recurrence of cSDH under the treatment with steroids were reported.[78] However, no benefit of the steroid-based versus non-steroidal treatment in terms of mortality and treatment success was noted.[78] A significant increase in adverse side effects was correlated with steroid treatment, that is, hyperglycemia, hypertension, and immunocompromised status.[67] The reported incidence of seizures varies from 0.7% to 18.5% in patients with cSDHs.[22] The authors noted a lack of evidence of a significant reduction in the incidence of seizures in patients with cSDHs following the administration of anti-epileptic drugs.[22]

Conservative treatment

Conservative treatment is offered to patients whose morbid conditions hinder surgical intervention or to whom surgery will not add benefit compared to medical treatment. Patients with small asymptomatic cSDHs are managed conservatively.[63] However, no pre-set guidelines are applied. Mainly OACs and anti-platelet medication are stopped along with FFPs administration and correction of coagulopathies instilled by liver insufficiency and comorbid conditions.[20] Routine monitoring of the cSDH volume and evolution depends on the physician following the patient's condition. Some resources are pragmatic and resort to the repetition of a cranial CT scan as patients develop symptoms. Other physicians consider a 2-week routine CT scan as a regimen.[49]

Surgical intervention

Symptomatic patients are ideal for treatment with surgical evacuation with an 80% success rate. Accomplished with minimal surgical risk, usually patients recover fast postoperatively and show the reverse neurological compromise instilled by the expanded hematoma.[45]

Prognosis and complications

Complications of cSDH surgical evacuation range from the ones associated with any surgery, that is, infection, bleeding and possible tissue damage to more specific ones related to the nature of the operation, that is, continuous bleeding, re-bleeding, tension pneumocephalus, seizures, intra-parenchymal bleeding, stroke, and possible brain tissue damage.[25,56] In the literature, reports of recurrence define the latter by rebleeding into an evacuated cSDH in 2.3–33% of cases.[56] Factors promoting recurrence are summarized in the following: preoperative OAC and anti-platelets medication, intracranial air, poor intraoperative brain re-expansion, bilateral cSDHs, and post-operative midline shift persistence.[2,27,31,64,79]

Mortality due to cSDHs varies according to whether surgical intervention was held or not. Without surgical evacuation, mortality rises to 33%. However, it drops to 3% within 30 days of cSDHs evacuation.[6] Prognosis postoperatively depends on the preoperative morbid condition, the extent of neurological symptoms 4 of 17, and the age of the patient.

Future research areas

New approaches

Surgical evacuation and brain decompression are the main goals of the treatment of symptomatic cSDHs. Postoperative adjuvant therapies are understudied in the literature inclusive of corticosteroids, ACE inhibitors, and tranexamic acid.[26] Evidence of the lower recurrence rates is associated with corticosteroid administration. However, more clarification is foreseen for the other two modalities. Recently, an interventional approach was suggested. It is the embolization of the middle meningeal artery (MMA) as means to devascularize the outer layer of the cSDH to prevent further rebleeding and hence limit recurrence.[57]

Anatomy related to MMA embolization (MMAE)

It is believed that the MMA through its anterior and posterior branches, which supply the dural layers, can anastomose with the neovascular vessels. Hence, re-bleeding through these anastomoses can provoke the recurrence of a cSDH.[57] Embolization of MMA's branches related to the position of the cSDH is believed to limit the re-bleeding and hence prevent a recurrence.

Steps of MMAE

Embolization requires an endovascular approach through the femoral artery ipsilateral to the recurrent cSDH.[9] Guided progression through the femoral artery through the aorta through either the left or right carotid artery terminates in the MMA branching from the maxillary branch of

the external carotid artery. The desired part is embolized usually using n-butyl-2-cyanoacrylate. Stasis of the contrast thereafter indicates the success of the embolization.[14]

The aim of the literature review

This literature review aims to provide an update on the recent findings related to the efficacy of MMA embolization. Answers that this review will provide connectivity to the following inquiries:

1. Is MMA embolization an adjunctive technique besides surgical intervention?
2. Can MMA embolization replace surgery? In which patients?
3. What are the indications for MMAE?
4. How efficient is MMA embolization in the prevention of recurrence after surgical intervention and expansion of minimal cSDH not requiring surgical evacuation?
5. What does the failure of MMA embolization to prevent the recurrence of cSDH re-bleeding suggest?

MATERIALS AND METHODS

Primary revision: Identification

We reviewed the existing literature on PubMed until August 2022, in the English language. We performed the research with combinations of the keywords "MMA," "middle meningeal artery embolization," "embolization," "refractory cSDH," "chronic subdural hematoma recurrence", and "cSDH outer layer." All kinds of work were included. References of the relevant studies were considered as an additional source of articles. Pure literature reviews were not included. We focused on the cSDH and the embolization of MMA as means of treatment.

Secondary revision: Screening

The titles of the sequestered articles are screened. Causes of cSDH related to acute subdural and epidural hematomas and tumor-provoked cSDHs were excluded from the study. Studies inclusive of a pure methodological description of MMAE and MMA variable anatomy were excluded from the study.

Data sequestration: Inclusion and classification

As this literature review is the work of one author, data from the included studies were thoroughly read and analyzed. Referral to other recent literature reviews aided the guidance of the author and allowed further checking. The studies were classified according to the way MMAE was utilized. Hence, the patients were classified according to the indication of MMAE:

a. MMAE as a standalone therapy
b. MMAE to prevent recurrence in symptomatic cSDHs
c. MMAE in recurrent cSDHs without second surgery
d. MMAE in patients with recurrent cSDHs after the second surgery.

RESULTS

297 records were identified after a search using the combination of the aforementioned keywords on PubMed. 36 records were exempted because of being duplicate records. 213 records were excluded after screening the titles and abstracts. 16 records were excluded after reading the full text [Figure 3]. The exclusion was due to the following reasons: acute hematomas (1), CSF hypotension (2), tumor-associated cSDHs (3), and technical description of MMAE without patient follow-up (6).

32 records were finally reviewed [Figure 1].[44-79] A spreadsheet was established inclusive of the following details fragmenting each study. Among the 32 eligible studies, 20 were case series, 10 were case reports, one was a prospective clinical trial, and another was a retrospective study. Five of the case series and the aforementioned retrospective study presented each a comparison with a historical group of patients treated conventionally. Figure 4 shows the distribution of the studies during the revision period. More than two-thirds (79%) of the studies were published after 2019.

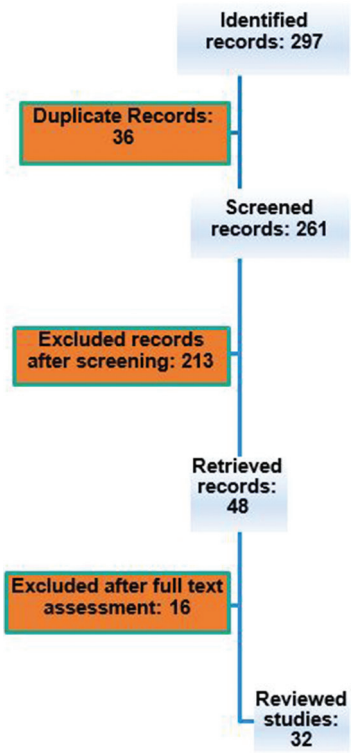

Figure 3: Diagram showing the step-wise refinement of articles in the literature review.

Totally, 973 patients were included and categorized into five groups. The distribution of the patients is evident in Figure 5. It is evident that more than half (exactly 53%) of the MMAE procedures were done without further surgical intervention. About one-third of the MMAE procedure was done to prevent recurrence after primary surgical intervention for symptomatic cSDHs. 82 cases of MMAE (8.4%) were done as treatment for the recurrence of cSDHs another surgical intervention. Only 35 MMAEs were done after a second surgery was done for recurrence. Arham *et al*. was the only author reporting MMAE application at the time of the first surgery as conjoint treatment with surgery.[3] Of note, none of the MMAE procedures were done concurrently with or before the surgical intervention. All procedures were done either solely or after surgical evacuation of the cSDHs was done.

MMAE as a standalone procedure

Being a standalone procedure is an application of MMAE reported in 14 of the reviewed articles published starting from the year 2018 [Table 1]. Before 2018, no report of any MMAE procedures as a standalone procedure was found in this review. However, indications dictating this application have varied through the reviewed articles. Below is enlisted for each article where MMAE has applied as a standalone procedure the corresponding indication(s). The patient's preference was the main indication for a standalone MMAE in 3 of the aforementioned articles.[54,57,62] Two studies proposed a standalone MMAE procedure for elderly patients on OAC and antiplatelet medication.[53,65] Entezami *et al*. added to this indicate the application of MMAE as a standalone procedure for COVID-19 patients.[54] Poor surgical candidacy with hematoma expansion and failed conservative treatment were reasons suggested to back up standalone MMAE in the studied population.[62] Of note, four studies did not include clarification concerning the indication of standalone MMAE.

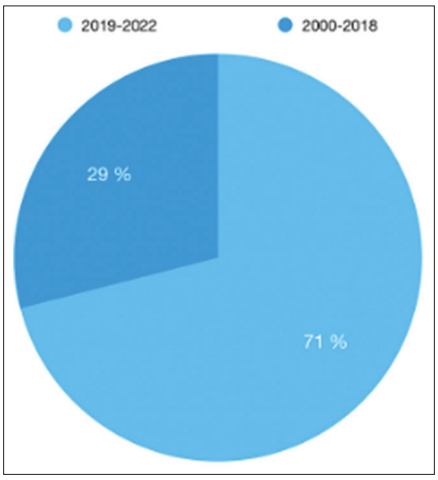

Figure 4: Graph showing the distribution of articles over the review period.

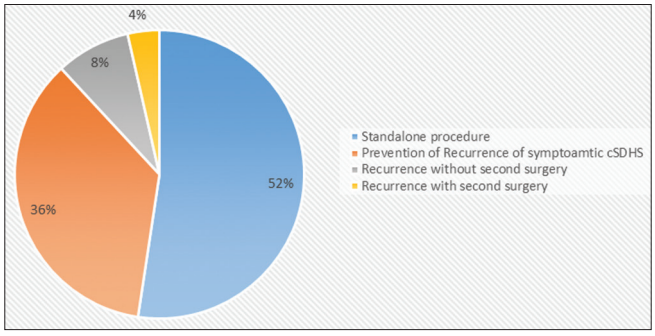

Figure 5: Graph showing the distribution of patients according to middle meningeal artery embolization indication.

MMAE as a prevention of recurrence for symptomatic cSDHs

Of the 773 patients included in the studies, 516 (66.75%) were subject to a standalone MMAE procedure with 3.8% failure rate if compared to the total MMAE procedures done [Table 2]. Relative to the standalone MMAE procedures, the failure rate drops to 0.77% (only 4 standalone MMAE procedures failed out of 516 done). Failure was defined as the need for rescue surgery or the expansion of the hematoma despite the MMAE procedure. As demonstrated in Table 3, 17 studies indicated MMAE as a prophylactic measure for the recurrence of symptomatic cSDHs. Out of 859 patients included in those studies, 321 (37.36%) were indicated to MMAE prophylactically. The decision to consider MMAE for recurrence depended on the definition of the risk at which a patient was considered prone to recurrence. The reasons varied according to each article and are summarized in Table 4. Recurrence risk of cSDHs after the surgical intervention was assessed in each study differently. Risk factors for recurrence are enlisted per study in the below table. Of note, the usage of OACs and antiplatelet medication was the most common risk factor. Hashimoto *et al*., Wang *et al*., and Okuma *et al*. have cited the history of trauma and old age. Alcoholism, liver disease, factor VI deficiency, ESRD, and the antiphospholipid syndrome were cited by Link *et al*., Shotar *et al*., Joyce *et al*., Okuma *et al*., and Ban *et al*.[5,23,28,39,40,53,59,60,73]

MMAE for recurrence of cSDHs

MMAE was applied for the recurrence of cSDHs either with a second surgery done or without any surgical intervention in 15 articles. Out of 556 patients included in those articles, 82 (14.74%) patients had MMAE procedures done without a second surgical intervention compared to 34 (6.29%) patients who received a second surgical intervention followed by an MMAE procedure. The only article reporting indicating MMAE for both reasons in the same study was Mino *et al*. in 2010.[43]

Table 1: Characteristics of MMAE applied as a standalone procedure in the reviewed articles.

Author	Year	Type of Study	MMAE standalone indication
Ban *et al.*	2018	Case series	Being asymptomatic
Link *et al.*	2018	Case series	Mild cSDHs white conservative treatment failing
Entezami *et al.*	2019	Case report	Preference of the patient
Entezami *et al.*	2019	Case series	Elderly patient with multiple complications
Waqas et *al.*	2019	Case series	Patients under OACs
Wang *et al.*	2019	Case report	Preference of the patient
Kan *et al.*	2020	Case series	Midline shift in mild cSDHs
Mureb *et al.*	2020	Case series	Hematoma expansion, failed conservative management, poor surgical candidacy, and patient preference
Yajima *et al.*	2020	Case series	High risk for recurrence
Gomez-Paz *et al.*	2020	Case series	-
Catapano *et al.*	2020	Case series	-
Joyce *et al.*	2020	Case series	-
Tiwari *et al.*	2021	Case series	cSDH asymptomatic
Nia *et al.*	2022	Retrospective	-

MMAE: Middle meningeal artery embolization, cSDHs: Chronic subdural hematomas, OACs: Oral anticoagulants

Table 2: Number of patients in articles reporting application MMAE as s a standalone procedure along with the number of failures per article.

Author	Year	Type of Study	Number of patients	Standalone	Failures
Ban *et al.*	2018	Case series	72	27	0
Link *et al.*	2018	Case series	49	32	0
Entezami *et al.*	2019	Case report	1	1	0
Entezami *et al.*	2019	Case series	5	5	0
Waqas *et al.*	2019	Case series	46	37	2
Wang *et al.*	2019	Case report	2	1	0
Kan *et al.*	2020	Case series	138	93	6
Mureb *et al.*	2020	Case series	8	8	2
Yajima *et al.*	2020	Case series	18	2	0
Gomez-Paz *et al.*	2020	Case series	23	23	0
Catapano *et al.*	2020	Case series	35	20	1
Joyce *et al.*	2020	Case series	121	51	9
Tiwari *et al.*	2021	Case series	13	7	0
Nia *et al.*	2022	Retrospective	242	209	0

MMAE: Middle meningeal artery embolization

MMAE for recurrence without surgery

Besides Mino *et al.*, of eight articles reporting MMAE procedures done for cSDHs recurrence without a second surgery, two were case reports, and six were case series [Table 5].[43] Out of 520 patients included in those articles, only 82 had an MMAE procedure for recurrence without surgery. The remaining patients were indicated for MMAE as either a standalone procedure or prophylactically after the first surgical intervention as by Link *et al.*, Waqas *et al.*, Catapano *et al.*, Joyce *et al.*, and Nia *et al.*[9,28,39,40,51,74] The failure rate was 2.23% (12 patients out of 520 patients. the number of failures attributed to MMAE procedures done for recurrence without surgery was not indicated in any article. Hence, the specific failure rate relative to this indication could not be calculated.

MMAE after second surgery for recurrence

Besides Mino *et al.*, only six articles reported applying MMAE procedures to patients for recurrence after the second surgery; two were case reports and four are case series [Table 6].[43] Except Hashimoto *et al.* who reported two MMAE procedures were applied prophylactically; all remaining five articles reported the application of MMAE procedures only after a second surgical intervention after a recurrence.[23] Hence, 34 out of 36 patients had an MMAE procedure after the second surgery. No failures were reported.

Failure of MMAE

MMAE procedures categorized as a failure were either a procedure that has resulted in non-resorption of cSDHs,

Table 3: Number of patients and failures of MMAE done for the prevention of recurrence after surgical evacuation.

Author	Year	Type of study	Number of patients	Prevent recurrence of symptomatic CSDHs	Failures
Hashimoto *et al.*	2013	Case series	5	2	0
Ban *et al.*	2018	Case series	72	45	0
Link *et al*	2018	Case series	49	10	0
Link *et al.*	2018	Case series	6	6	0
Okuma *et al.*	2019	Case series	17	17	0
Waqas *et al.*	2019	Case series	46	4	2
Wang *et al.*	2019	Case report	2	1	0
Piergallini *et al.*	2019	Case report	2	2	0
Kan *et al.*	2020	Case series	138	45	6
Shotar *et al.*	2020	Case series	89	89	4
Yokoya *et al.*	2020	Case report	2	2	0
Shotar *et al.*	2020	Case report	1	1	0
Catapano *et al*	2020	Case series	35	4	1
Joyce *et al.*	2020	Case series	121	53	9
Ng *et al.*	2020	Clinical trial	19	19	0
Tiwari *et al.*	2021	Case series	13	6	0
Nia *et al.*	2022	Retrospective	242	15	0

MMAE: Middle meningeal artery embolization, cSDHs: Chronic subdural hematomas

Table 4: Factors predicting recurrence of MMAE after surgical evacuation per article.

Author	Year	Predictive factors for cSDHs recurrence
Hashimoto *et al.*	2013	Old age, brain atrophy, alcoholic status, OACs
Ban *et al.*	2018	Trauma, smoking, alcoholism, liver disease, OACs, CVAs
Link *et al*	2018	GI bleed, history of SDH, epilepsy, ESRD, HTN
Link *et al*	2018	----
Okuma *et al.*	2019	OACs, liver disease, HD, old age, large cSDHs volume, no drain during surgery, postop residual air, previous recurrence
Waqas *et al.*	2019	OACs, trauma, CVAs
Wang *et al.*	2019	Head trauma
Piergallini *et al.*	2019	HTN, paroxysmal atrial fibrillation
Kan *et al.*	2020	OACs, prior cSDH evacuation
Shotar *et al.*	2020	OACs, thrombocytopenia, coagulation disorders, alcoholism
Yokoya *et al.*	2020	DM
Shotar *et al.*	2020	Alcoholism, Lewy body dementia
Catapano *et al.*	2020	Seizures, surgery failure, OACs
Joyce *et al.*	2020	OACs, factor VI deficiency, APS
Ng *et al.*	2020	Alcoholism, OACs
Tiwari *et al.*	2021	OACs
Nia *et al.*	2022	----

OACs: Oral anticoagulants, cSDH: Chronic subdural hematomas, SDH: subdural hematomas, GI: Gastrointestinal, ESRD: End-stage renal disease, HTN: Hypertension, CVA: Cerebrovascular accident, HD: Hypertension, APS: Autoimmune phospholipid syndrome, DM: Diabetes mellitus, MMAE: Middle meningeal artery embolization

increase in the hematoma volume, or recurrence of the hematoma after resorption. Out of the 997 MMAE procedures, 22 procedures failed and 19 procedures were complicated [Table 7]. Hence, the failure rate was 4.1%. Compared to Di Cristofori *et al.* in March 2022, this failure rate is low (5.5%). This could be attributed to the relative increase in the population size compared to the literature review by Di Cristofori *et al.*[13] The inclusion of the retrospective study done by Nia *et al.* published in August 2022 increased the population size from 727 to 973 without reports of additional failures in MMAE.[51] Reflecting on the population size of the included articles, the results of the failure rate were similar to the results deducted by Di Cristofori *et al.*[13] Reflection on the indication of MMAE application and its corresponding failure rate, a variance has been noted. This could be attributed to the overlap among MMAE indications for the failure count. Most of the studies did not refer to the failure rate per MMAE indication.

Table 5: Number of patients and failures for MMAE done for recurrence without secondary surgical evacuation.

Author	Year	Type of Study	Number of patients	For recurrence without second surgery	Failures
Masaki *et al.*	2010	Case series	4	3	0
Hirai *et al.*	2004	Case report	2	2	0
Kim	2017	Case series	20	20	0
Sirh *et al.*	2018	Case report	1	1	0
Link *et al.*	2018	Case series	49	7	0
Waqas *et al*	2019	Case series	46	5	2
Catapano *et al.*	2020	Case series	35	9	1
Joyce *et al.*	2020	Case series	121	17	9
Nia *et al.*	2022	Retrospective	242	18	0
			520	82	12

MMAE: Middle meningeal artery embolization

Table 6: Number of patients and failures of MMAE done after second surgical evacuation.

Author	Year	Type of study	Number of patients	For recurrence after second surgery	Failures
Mandai *et al.*	2000	Case report	1	1	0
Hashimoto *et al.*	2013	Case series	5	3	0
Chihara *et al.*	2014	Case report	1	1	0
Tempaku *et al.*	2015	Case series	5	5	0
Matsumoto *et al.*	2017	Case series	4	4	0
Nakagawa *et al.*	2019	Case series	20	20	0
			36	34	0

MMAE: Middle meningeal artery embolization

Table 7: Number of failures and complications per MMAE indication.

MMAE indication	Number of patients	Failures	Failure rate	Complications
Standalone	516	20	3.87%	5
Prophylactically for recurrence without second surgery	321	22	6.85%	
For recurrence without second surgery	82	12	10.25%	0
For recurrence after second surgery	35			0
At the time of first surgery	1	0	0%	5
	955	54		

MMAE: Middle meningeal artery embolization

Overall, the failure rate diminished with the inclusion of a large study published by Nia *et al.* in 2022.[51]

Complications of MMAE

Only five complications were reported in the reviewed articles and it was noticed that all complications happened with patients whose MMAE procedures were indicated as either a standalone or a prophylactic procedure. The complications were worsening cSDHs, two CVAs, a focal seizure, and an episode of aphasia.

Comparison with conventional treatment

In the literature review published by Di Cristofori *et al.* in March 2022, the author reported that five studies cited a comparison between patients to whom MMAE was applied versus patients treated conventionally.[13] The implementation of the results reported by Nia *et al.* in August 2022 along with the results of the article by Di Cristofori *et al.* is depicted in Table 8.[13,51] With a similar MMAE group population, Nia *et al.* reported much lower rates of failure (0.47% vs. 3.9%).[51] Although a similar result can be deducted concerning the rate of recurrence after conventional technique application, this can be attributed to the large population (4050) studied by Nia *et al.* relative to the 727 patients reported by Cristofori *et al.*[13,51]

DISCUSSION

Indications for MMA embolization through the literature

This review of literature unveiled the application of MMAE for the following indications: a standalone procedure, an

adjuvant treatment at the time of first surgery, for prevention of recurrence after first surgical evacuation, as a treatment after recurrence after first surgical evacuation without second surgery and as a treatment after recurrence after second surgical evacuation. Keen attention ought to discriminate the first indication versus the other four indications. MMAE as a standalone procedure manages cSDHs by vascular compromise. Hence, further progression of cSDHs by

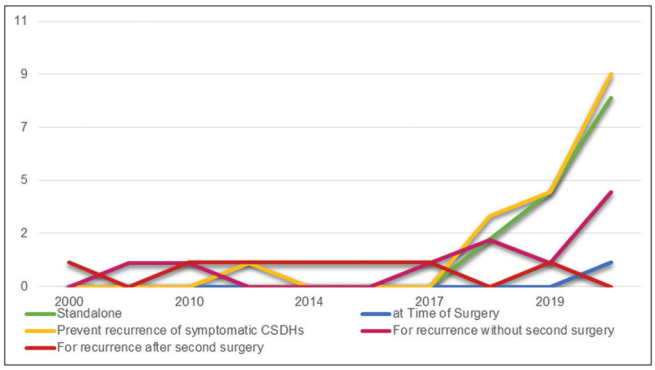

Figure 6: Variation of the indication of middle meningeal artery embolization during the revision period.

standalone MMAE is valid. The other four scenarios target the mass effect imparted by the hematoma volume by surgical evacuation and target the vascular supply of the dural cell layer by MMAE, yet each is at a different stage regarding the surgical intervention and a number of recurrences. Therefore, this justifies the higher rate of standalone MMAE procedures failure compared to the other four modalities reported by Di Cristofori *et al.*[13] (3.9% vs. 8.9%). Meanwhile in this review, the inclusion of the study by Nia *et al.* shifted the results in favor of standalone MMAE (3.87% vs. 6.25% and 10.25%). However, Nia *et al.* did not feature specific values regarding standalone MMAE procedures done for asymptomatic and symptomatic cSDHs patients.[51] Application of MMAE to patients with asymptomatic cSDHs imparts bias to the results. Similarly, the attribution of a higher success rate of the combined indications to the application of MMAE shadows 13 of 17 the obvious fact that some patients could have been cured by mere surgical evacuation. The combination with MMAE does not mandate additive benefits. Comparison with a control group for each of the four combined indications is key. Figure 6 shows the variation of the indication of MMAE throughout the period of this literature review.[44-78] It is

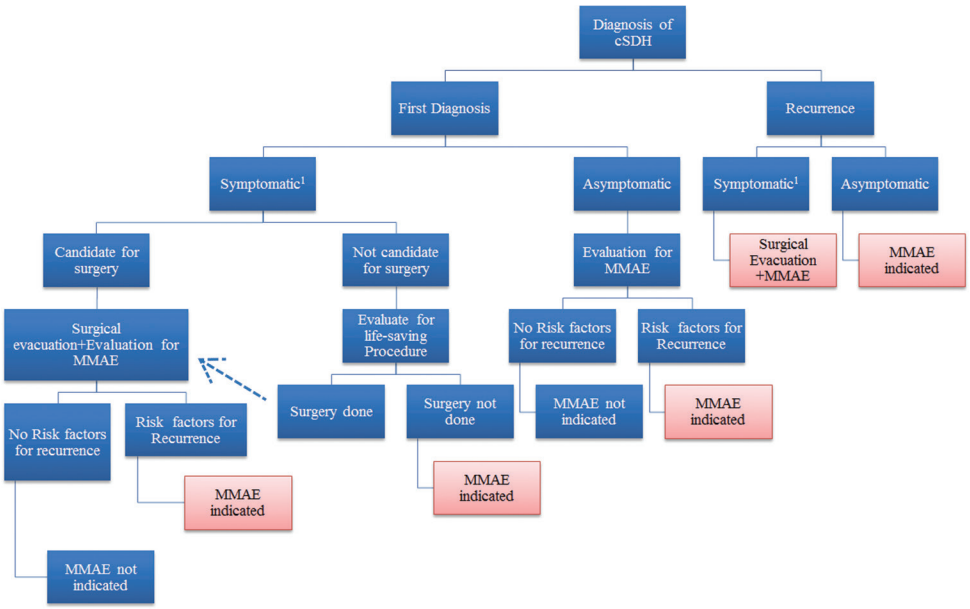

Figure 7: Algorithm and middle meningeal artery embolization indication advised by the authors. (1) Neurological deficit; Cerebral midline shift evident on imaging. Blue dashed arrow: Management continues similarly.

Table 8: Comparison of failure rate and recurrence rate between Nia *et al.* and Cristofori *et al.*

Study	MMAE group	Conventional technique group	Rate of failure of MMAE	Rate of recurrence after conventional technique
Cristofori *el al.*	205	727	3.90%	29.50%
Nia *et al.*	209	4050	0.47%	0.88%

MMAE: Middle meningeal artery embolization

evident that MMAE has been indicated recently as a primary option for therapy of cSDH as either a standalone procedure or adjuvant to primary surgical evacuation in the symptomatic patient. However, the eligibility of patients for MMAE remains not clearly defined. Some exclusion criteria reported by authors, that is, patients with midline shift are excluded by Ban *et al*.[5] are conversely considered as inclusion criteria by other authors for standalone MMAE i.e. Catapano *et al*.[9] Based on the findings, we advise the following algorithm for the management of cSDH cases [Figure 7].

Report of MMAE applications and results

The number of patients to whom MMAE was applied in the reviewed literature is conspicuous. Some articles reported the number of treated patients; others report the number of cSDHs yet not the number of patients. Bilateral cSDHs are reported as two cases and treatment of either has been reported by different modalities for different indications in the same individual. Limited is the report of segregation of patients according to whether the cSDHs is unilateral or bilateral. Risk factors of progression and recurrence, results of MMAE application, long-term outcomes, side effects, mortality, and morbidity need to be assessed for each of the two patient categories. This is not presented in the literature. Hence, results cannot be generalized. The heterogeneity of the data adds to the lack of parameters related to the number of patients within each category, the number of patients considered for each indication, the time between the surgical intervention and the MMAE procedure, and the type of MMAE material and technique specifications. Moreover, the categorization of patients according to morbid conditions, OACs administration and risk factor variance is missing in the articles, as a comparison among those groups is not derived.

Future considerations

Despite the aforementioned bias, discrimination between standalone MMAE and MMAE associated with surgical evacuation, regardless of the situation of either procedure, is paramount. Future application of MMAE associated with surgical evacuation is anticipated to provide better results versus standalone MMAE. This was noted previously in the results section. The mere fact that residual hematoma remains in the case of standalone MMAE constrains the efficacy of this application. This increases its failure rate as patients could fail to resorb the residual volume and re-bleeding could be a complication, especially in patients with OACs and anti-platelets intake. The necessity to surgically evacuate hematomas and consider MMAE later after surgical intervention appears as a better approach. Future consideration of studies addressing the adequate timeframe is highly recommended. This approach is anticipated to reduce the hospitalization period, and the costs of second surgical interventions, and promote a better prognosis with less morbidity and mortality, especially in the elderly population. Therefore, clinical trials inclusive of standalone MMAE applied to symptomatic yet poor surgical candidates and MMAE procedure applied at different timeframes to surgical candidates after surgical intervention versus conventional control groups are indispensable. The studies published by Nia *et al*. and Ng *et al*. are representative of the required studies.[49,51]

CONCLUSION

CSDH is a disease of the elderly who struggle with the eventual result of aging: comorbidities and chronic diseases. Treatment of this bleeding condition within the skull has been conventionally surgical. However, the recurrence of this condition and the risks implemented in the surgical evacuation of cSDHs renders less invasive procedures more appealing. Hence, the attention drives us toward MMAE. This literature review aimed to provide a walkthrough of the available data about MMAE application for the treatment of cSDHs. Anticipation of future applications of MMAE, the indications, and the foreseeable outcomes was speculated in light of the reported findings by articles over the past 22 years. As a result, application for the prevention of recurrence and as a primary modality of care has been the indication of choice. However, heterogeneity of the data limited this work and hindered the distinction between indication-specific MMAE failure rates. The safety of MMAE as a procedure is regarded as a general theme in the literature and can be considered for future application. We look forward to clinical trials with comparative themes to provide better population-specific deductions about this new technique.

Declaration of patient consent

Patient's consent not required as there are no patients in this study.

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Conflicts of interest

There are no conflicts of interest.

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