

BEST PRACTICE PAPER

Best practice in the use of middle meningeal artery embolisation for chronic subdural haematoma

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Abstract

Chronic subdural haematoma (cSDH) is a common neurosurgical disorder in older people and projected to become the most common cranial neurosurgical pathology by 2030. Incidence of cSDH has risen in recent years as a consequence of improved access to brain imaging, and the increased prescribing of antithrombotic medication for both primary and secondary prevention of vascular disease. Chronic SDH typically presents with an insidious onset of broad-ranging symptoms including impaired cognition, gait, balance and mobility, often with headache. It progresses to more fulminant symptoms of hemiplegia, dysphasia and eventually coma. Although the established standard of care for clearly symptomatic cSDH is burr hole trephination, Middle Meningeal Artery Embolisation (MMAE) has emerged as a minimally invasive treatment option for some patients with cSDH. This is a rapidly evolving field: recently published randomised control trials have provided an evidence-base supporting the use of MMAE not only as an adjunct to burr hole trephination, but also in patients in whom trephination is contraindicated, or in patients with mild symptomatology who are not considered appropriate for immediate burr hole trephination. This article provides practical, real-world guidance on current best practice based on our experience and the published evidence available to date. We use case studies and treatment algorithms from the UK's highest volume MMAE centre to illustrate collaborative care pathways for patients with cSDH between neurosurgery, interventional neuroradiology and trauma geriatricians.

Keywords: chronic subdural haematoma; middle meningeal artery embolisation; older adults; frailty; neurosurgery; older people

Key Points

- Chronic subdural haematoma is a common neurosurgical disorder in older people.
- Middle Meningeal Artery Embolisation has emerged as a minimally invasive treatment option for some patients with chronic subdural haematoma
- Recently published randomised control trials have provided an evidence-base supporting the use of Middle Meningeal Artery Embolisation.
- Middle Meningeal Artery Embolisation should now be considered as adjunct to burr hole trephination to reduce the risk of recurrence.
- Middle Meningeal Artery Embolisation should also be considered in mildly symptomatic patients not undergoing surgery who are at high risk of recurrence.

Introduction

Chronic subdural haematoma (cSDH) is a common disorder of older people and projected to become the most common cranial neurosurgical pathology by 2030 [1]. Incidence of cSDH has risen in recent years as a consequence of improved access to brain imaging, and the increased prescribing of antithrombotic medication for both primary and secondary prevention of vascular disease [2].

In practical terms, diagnosis is achieved radiologically, where cSDH is characterised by the accumulation of non-acute blood degradation products in the subdural space. These appear isodense, hypodense or of mixed density on computed tomography (CT) imaging [3]. This heterogeneity in radiological findings reflects the process of blood degradation over time, where affected individuals may or may not report a history of head trauma [4].

It is important for geriatricians to fully appreciate that *chronic* SDH is an entity which should be considered clearly distinct from *acute* SDH (aSDH). Although these two pathologies share a common aetiology of haemorrhage into the subdural space, the two conditions differ very significantly in their presentation and management. Patients with symptomatic acute subdural haematomas typically present early following significant and recognised head trauma. As a result of higher energy head injuries, aSDH is often associated with concurrent traumatic brain injury. Symptoms prompting head imaging typically include visible external injuries to the head and neck, acutely altered conscious level and/or focal neurological deficits, such as hemiplegia or dysphasia [5].

However, it is essential for the geriatrician to recognise that surgical clot evacuation in aSDH cannot be reliably achieved through burr hole irrigation, due to the viscosity of acute haematoma. Surgical decompression of aSDH must therefore involve either craniotomy or decompressive hemicraniotomy with subsequent cranioplasty [6]. These procedures represent considerably more major surgery and are poorly tolerated by older people living with multimorbidity and frailty. Outcomes from surgery for aSDH in adults aged >65 have been shown to be very poor, with inpatient mortality >40% and good neurological functional outcome limited to around 10% of patients [7]. Survivors are therefore typically left with longstanding severe neurological disability. In the UK, few frail and multimorbid older adults typically undergo emergency surgery for aSDH.

However, in direct contrast, patients with cSDH have a different presentation and trajectory with surgical care. Chronic SDH typically presents with an insidious onset of broad-ranging symptoms including impaired cognition, gait, balance and mobility, often accompanied by headache. These symptoms can progress to more fulminant hemiplegia, dysphasia and eventually coma [8]. Diagnosis of cSDH can therefore be challenging in the early stages of disease, given the non-specific nature of early symptoms and potential

for confounding causes due to intercurrent acute medical illness.

Surgical treatment of symptomatic cSDH is via burr hole trephination with subdural or subgaleal drain placement [9]. Such surgery presents a modest physiological strain on the patient and is usually well-tolerated. Observational data indicate that patients with cSDH who undergo burr hole trephination display superior survival and functional outcomes compared to patients managed conservatively [10], although unadjusted case selection undoubtedly complicates the interpretation of such data. Moreover, in some patients, cSDH appears to be a sentinel health condition and some observational data indicate that 1-year mortality may be as high as 30% [11]. Nonetheless, other observational studies also indicate that age alone might not be associated with adverse outcomes following surgery [10]. This observation is consistent with the wider modern perioperative understanding that frailty, rather than age alone, is the key factor determining risk in the older adult.

Inflammatory pathophysiology—Basis for middle meningeal artery embolisation

Understanding of the natural history of cSDH has improved in recent years, and it has been observed that there are high rates of cSDH recurrence, even in patients managed surgically after initial diagnosis [12]. Failure of initial conservative treatment and progression to rescue surgery has been reported to be as high as 30% in one recent study [13].

Evolving understanding of the pathophysiology behind cSDH aids in explaining these high rates of recurrence in patients managed conservatively and also, to a lesser degree, surgically. It is now understood that bleeding into the subdural space triggers an inflammatory reaction, which is then perpetuated by a cytokine cascade resulting in the expression of vascular endothelial growth factors [14]. New vessels are formed in a haematoma membrane, which are prone to repeated microhaemorrhage into the subdural space. This drives further cyclical inflammation and neovascularisation. Oncotic forces drive the accumulation of fluid into the protein-rich space, which ultimately exerts progressive mass effect on the underlying brain tissue. Although factors contributing to this cycle of progression are not fully understood, patients taking antithrombotic medication are known to be at higher risk of recurrence or progression [15]. Intuitively it seems plausible that this effect may be mediated through repetitive microhaemorrhage.

This notion that cSDH is driven by an inflammatory pathology has prompted research into novel adjuvant medical therapies, which aim to attenuate inflammation. Various studies have investigated whether statins, angiotensin-converting inhibitors (ACEi) and corticosteroids can influence recurrence rate. At present, there is insufficient data to recommend statins and ACEi [16, 17], though randomised control trial data indicate corticosteroid use may even be harmful [18].

Middle meningeal artery embolisation technique

Middle meningeal artery embolisation (MMAE) has therefore emerged as a minimally invasive intervention which aims to embolise the hypervascular dural membrane capsule. This procedure aims to eliminate repeated microhaemorrhage and to attenuate the cyclical inflammatory process.

MMAE can be performed via a radial or femoral arterial approach under local or general anaesthesia. The selection of access point and mode of anaesthesia depends on aortic arch anatomy, operator preference and other patient factors such as cardiorespiratory fitness and *in-procedure* compliance. A pre-procedure arch-to-vertex CT angiogram can be helpful in deciding mode of access. Either unilateral or bilateral embolisation can be performed during a single sitting.

The procedure commences with control angiography via the common or internal carotid artery to establish the vascular anatomy and exclude anatomical variants. This is followed by positioning of a guide catheter in the distal external carotid artery, from which catheterisation of the MMA branches is achieved with a microcatheter. Our own experience suggests that each of the major branches of the MMA should be embolized to ensure success [19]. Liquid embolic agents such as Squid (Balt) and Onyx (Medtronic) are commonly used. Theoretically, liquid embolic agents allow distal penetration of vascular membranes. Other embolic agents are likely to result in more proximal arterial occlusion. Notably, *N*-butyl-2-cyanoacrylate, polyvinyl alcohol particles and platinum coils have also been shown to be effective in observational studies [20]. Currently there is no definitive evidence proving superiority of one technique over another.

Procedural complications are generally accepted to be infrequent but most commonly relate to the arterial access site (puncture site haematoma, pseudoaneurysm and limb ischaemia). Rarer complications include embolic stroke or ischaemic injury to cranial nerves. This can occur through inadvertent embolization of the petrous branch of the MMA causing facial nerve weakness. Alternatively, visual disturbance or blindness can arise if care is not taken to avoid orbital collateral pathways [21]. These risks are, however, low. Randomised control trial data have demonstrated stroke and facial paralysis risks of 1% and 0.3%, respectively [22, 23], whilst observational data report a 0.1% risk of blindness [24].

What is the effect of MMAE?

Numerous observational data exist reporting the efficacy of MMAE in cSDH. Several meta-analyses have shown that MMAE reduces both cSDH recurrence and reoperation rates when used as an adjunct to surgery ($n = 2783$ patient undergoing MMAE), and also when used as standalone therapy ($n = 156$ standalone MMAE) [21, 25]. These observational data have been augmented by publication in late 2024 of EMBOLISE, STEM and MAGIC-MT, the first three randomised control trials (RCT) evaluating use of MMAE in cSDH [22, 23, 26].

The EMBOLISE RCT recruited 400 patients and demonstrated that MMAE resulted in a 3-fold reduction in haematoma recurrence requiring reoperation when compared to surgery alone. This was accompanied by a 2% rate of serious adverse events. The STEM RCT recruited 310 patients and showed that adjunctive MMAE reduced composite treatment failure (defined as recurrent or residual chronic subdural hematoma on the target side measuring greater than 10 mm at 180 days after the intervention; reoperation or surgical rescue within 180 days after the intervention; or major disabling stroke, myocardial infarction, or death from neurologic causes within 180 days after the intervention). Treatment failure was observed to fall from 36% in the standard treatment group, to 16% in the embolisation group. Although the trial was not powered for the evaluation of subgroup outcomes, the benefit of adjunctive MMAE primarily appeared to be driven by the effect of MMAE in patients receiving non-surgical treatment (who were initially considered insufficiently symptomatic by their treating neurosurgeon to warrant surgery, prior to randomisation). Safety data revealed no increased rate of major adverse events or deaths in the embolisation group. The third randomised control trial (MAGIC-MT, $n = 722$ patients recruited) revealed that adjunctive MMAE reduced symptomatic SDH recurrence within 90 days from 9.9% in the standard care group to 6.7% in the embolisation group, with a lower incidence of serious adverse events in the embolisation group.

Existing guidance

The UK's National Institute of Health and Care Excellence (NICE) published guidance in 2023 advising that there was at that time insufficient data to support the use of MMAE outside of a research context [27]. Subsequently, use of MMAE in the UK has been varied. The 'Improving Care in Elderly Neurosurgery Initiative' (ICENI) guideline for cSDH management adopted a similar stance, and also concluded that there was insufficient evidence at the time of guideline development (literature search in 2022–23) to support the routine use of middle meningeal artery embolisation in the treatment of cSDH outside of a research context [28].

Moreover, due to the number of RCTs that are currently recruiting patients in the evaluation of MMAE, there had been some reluctance in the cross-specialty community to adopt guidance prior to publication of randomised data. However with increasing volume of observational data supporting MMAE, this position became untenable and in late 2024, an international, multi-society consensus statement on the use of MMAE in cSDH was published [29].

MMAE was recommended in three contexts:

1. *As 'stand-alone' treatment in de novo cSDH requiring intervention, but where surgery is prevented due to either coagulopathy or in those on antithrombotics in whom the risk of suspension is considered unacceptably high.*

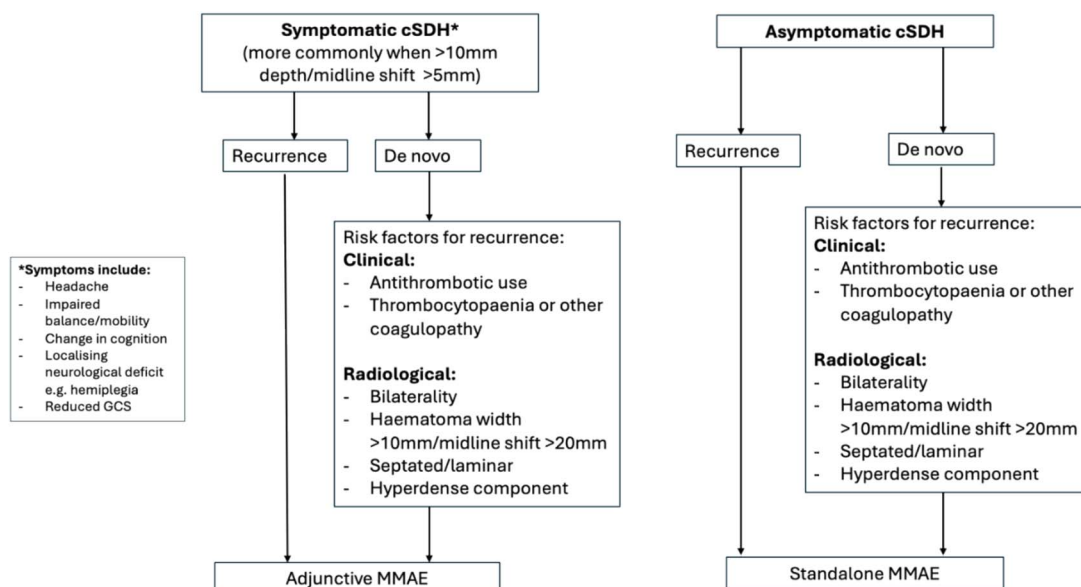


Figure 1. Proposed treatment algorithm for use of MMAE in management of cSDH.

2. As 'stand-alone' treatment in recurrent cSDH requiring intervention, but where surgery is prevented due to either coagulopathy or in those on antithrombotics in whom the risk of suspension is considered unacceptably high.
3. As an 'adjunct to surgery' in recurrent cSDH.

However, consensus could not be achieved by the working group on the utility of MMAE as an adjunct to conventional management with surgery in *de novo* disease. Subsequent to the publication of this guideline in 2024, the first three RCTS referenced above were published, arguably rendering prior guidance obsolete.

Current best practice

Case selection

Although systematic review and meta-analysis of pooled RCT data has not yet been published, we consider that the published observational and randomised data support the principle that MMAE is a safe and effective treatment to promote resorption and prevent recurrence of cSDH.

For the avoidance of doubt, note should be made that MMAE should not be considered an alternative to conventional burr hole trephination in patients for whom this is indicated. However, we believe that MMAE has applications to reduce the risk of progression or recurrence in all patients considered to be at high-risk of cSDH recurrence.

Risk factors predictive of cSDH recurrence are not yet fully understood. Broadly speaking, associations have been made in the observational literature, which can be divided into clinical and radiological risk factors. Clinical risk factors include: the use of clopidogrel or anticoagulation; co-existent coagulopathy, including chronic liver disease [15, 30, 31]. Radiological risk factors for recurrence include bilaterality, haematoma width > 10 mm, septation, and haematoma

density [32–34]. Furthermore, the recurrence risk after repeated surgery is approximately double the recurrence risk following *de novo* surgery (~10% risk in *de novo* surgery and 20% in recurrent cSDH surgery) [34].

With this in mind, we consider that MMAE should be contemplated in any case of recurrent cSDH, as well as in *de novo* cSDH in patients displaying risk factors for recurrence. In symptomatic patients with large volume collection, we consider that the evidence supports the use of MMAE as an adjunct to conventional burr hole trephination. In patients who are minimally symptomatic or genuinely asymptomatic and for whom surgery is not indicated, we believe the evidence supports consideration of MMAE as standalone therapy.

We summarise this approach in a proposed treatment algorithm for treatment of cSDH reflecting the most recently published evidence and previous recommendations (Figure 1). In addition, we provide two case vignettes in boxes 1 and 2, detailing real life examples of when we have used MMAE as both an adjunct to burr hole surgery and as standalone therapy.

Operational factors

Chronic subdural haematomas primarily occur in older adults with multimorbidity. Therefore, a multi-disciplinary approach including neurosurgeons, interventional radiologists and geriatricians is recommended [28].

Our practice involves trauma geriatrician review of all patients admitted to our centre with cSDH aged >65 years. This helps facilitate functional and cognitive assessments, surgical optimisation and shared decision making. Multi-disciplinary discussion then takes place between neurosurgery, interventional neuroradiology and geriatric trauma teams regarding the appropriateness of MMAE.

Patients requiring burr hole trephination are admitted under neurosurgery, although their care is shared with the geriatric trauma team. Although some units favour contemporaneous MMAE with burr hole trephination under the same anaesthetic, we have found it difficult to logistically facilitate this approach given the operational pressures that co-exist between neurosurgery and interventional neuro-radiology. Therefore, most patients in our centre undergo independent procedures. No evidence currently exists to mandate MMAE within a defined timeframe after diagnosis. However, we hypothesise that early MMAE is likely to attenuate re-bleeding risk more promptly. MMAE is therefore undertaken at the first available opportunity. Patients receiving standalone MMAE without the need for burr hole trephination (e.g. those who are minimally symptomatic or unfit for surgery), are admitted under geriatric medicine. Moreover, we have piloted day-case MMAE for selected patients who are minimally symptomatic. We hypothesise that the costs of MMAE in this subgroup may be offset by avoiding index admission as well as through the prevention of future care episodes [35].

Conclusion

The most recently published trial evidence indicates that MMAE is an effective, low-risk technique that can be used as both an adjunctive and stand-alone treatment in the management of patients with cSDH. However, further research is needed to report long-term safety data and to evaluate the economic impact of MMAE. Data from registries (or equivalent sources) will be required to determine whether trial outcomes are replicated in real-world clinical practice.

Nonetheless, we anticipate that the care of patients with cSDH who are at high-risk of recurrence will be transformed by the publication of randomised trials which have rendered previous guidance obsolete. However, investment in hardware and equipment is required to allow a growing neurointerventional workforce to deliver MMAE within the NHS. Future care pathways will require close collaboration between neurosurgery, interventional neuroradiology and geriatric medicine to ensure that patients with cSDH are able to access timely and cost-efficient care.

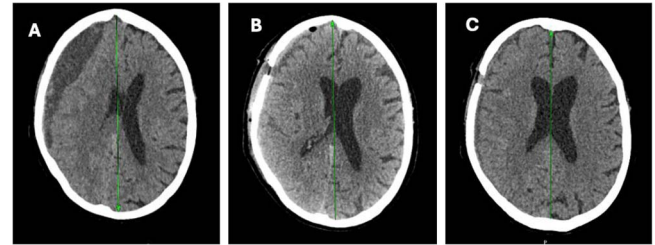
Case 1: Adjunctive MMAE

A male aged >80 presented to hospital with persistent headaches, unsteadiness and deterioration in short-term memory, 1 month after a fall at home. He had a history of ischaemic heart disease (IHD) and previous coronary artery bypass grafting, for which he was prescribed aspirin.

On admission, a CT head scan demonstrated right-sided acute-on-cSDH with features of midline shift and early herniation (Image A). The patient underwent burr hole trephination and repeat CT head 2 days thereafter. Repeat imaging demonstrated reduced volume and mass effect exerted by the subdural collection (Image B). Considering the need

to reinstate antiplatelets for IHD, the patient underwent right sided MMAE. The procedure was uncomplicated, and he was discharged home the following day. Aspirin was recommenced at 1-week post-MMAE. Interval non-contrast CT head at 3 weeks demonstrated complete resolution of mass effect and midline shift, with further improvement of right hemispheric subdural collection (Image C). Montreal Cognitive Assessment score increased from 23/30 (76%) pre-procedure to 25/30 at 24 h after and 20/22 (conducted by telephone, 91%) at 30 days.

The patient reported resolution of symptoms and return to independent activities of daily living.



Written informed consent for publication of their clinical details and clinical images was obtained from the patient.

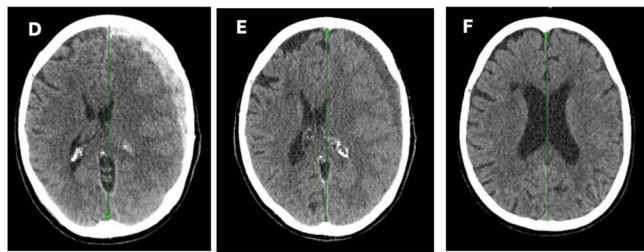
Case 2: Primary MMAE

A female aged >80 presented following a fall with an occipital head injury and new blurred vision. CT head demonstrated bilateral traumatic SDHs with mass effect and midline shift of structures towards the right (Image D). Three months prior to admission, the patient had undergone percutaneous coronary intervention for a myocardial infarction and was prescribed dual antiplatelet therapy (DAPT).

The patient was admitted for conservative management of aSDH; DAPT was held. During admission, the patient's confusion and headaches worsened, however repeat CT head scans showed stable intracranial appearances. The decision was made to restart single antiplatelet therapy (clopidogrel) after 2 weeks. She was discharged to her daughter's house due to concerns around safety returning to her own home.

Two days following discharge, the patient represented with increasing headaches and right sided sensory disturbance. The clopidogrel was held and a repeat CT head scan demonstrated a mild increase in size of the left SDH with ongoing mass effect without acute bleeding (Image E). A neurosurgical decision was made that her symptoms did not justify the risks of surgery, and she underwent primary standalone left sided MMAE. Aspirin was started 24 h following MMAE, and clopidogrel at 72 h.

Montreal Cognitive Assessment (MOCA) score increased from 13/30 (43%) pre-MMAE, to 21/30 (70%) at 24 h, and 18/22 (82% conducted by telephone) at 30 days. The patient's symptoms improved, and she returned to independent living. Repeat CT head scan at 120 days demonstrated almost complete resolution of SDH (Image F).



Written informed consent for publication of their clinical details and clinical images was obtained from the patient.

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