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Original Article

Middle Meningeal Artery Embolization for Chronic Subdural Hematoma: A Case Series

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BACKGROUND: Chronic subdural hematoma (CSDH) represents a common neurosurgical condition particularly in the elderly. Middle meningeal artery embolization (MMAE) has been proposed as a treatment option for this condition.

OBJECT: To demonstrate the feasibility of MMAE for the treatment of CSDH avoiding the risk of interrupting anticoagulation and reducing the perioperative risk in a Canadian health care institution.

METHODS: A retrospective chart review of patients receiving MMAE as primary treatment for CSDH is presented. Baseline demographics are collected (age, sex, and side of hematoma). Outcomes of interest include hematoma thickness pre-intervention and at follow up, procedural and post-operative complications, and length of hospital stay. Clinical outcomes (subdural hematoma resolution) are supplemented by computed tomography (CT) imaging.

RESULTS: Patients (N=4) underwent MMAE for the treatment of CSDH to avoid risk of cessation of anticoagulation and expected perioperative risk. Median age was 74 years. Two patients were on antithrombotic therapy at the time of intervention. Patient follow-up occurred at 1, 3, 6, and 9 months post-operatively. Resolution of symptoms and significant reduction of hematoma thickness was evident (up to 14 mm) at follow-up. Improvement in all cases was confirmed by CT and clinical evaluation. No patients suffered from complications or recurrence.

CONCLUSION: The MMAE is a safe and practical treatment for CSDH, even in this rather frail patient cohort with some on anticoagulation.

KEYWORDS: Anticoagulation, chronic subdural hematoma, embolization, therapeutic radiology.

INTRODUCTION

Chronic subdural collections represent a complex and myriad spectrum of pathology and may cause significant morbidity and mortality if left untreated.[1] The occurrence of CSDH is relatively common within the aging population (79.4 patients per 100,000 annually) and is one of the most common neurosurgical diagnoses in adults.[2] The incidence of CSDHs is expected to increase due to an aging population and use of anticoagulation and antiplatelet therapies.[3]

Chronic subdural hematomas are collections of fluid, blood, and blood degradation products between the dura matter and the arachnoid matter.[4] The inner facade of the dura matter is normally adherent to the underlying arachnoid, each of which forms a respective border layer. [4] The CSDH is often initiated by a head injury, usually minor in the elderly, damaging the dural border cell layer. Pathological and histological examinations of chronic subdural hematomas show the inner membrane layer consisting of proliferated dural border cells. The subsequent breakdown of blood products and inflammation accentuates the formation of membranes.[5] Neovascularization develops as a natural consequence of repetitive inflammation. The presence of these vessels has been readily demonstrated with pathological analysis of this layer as well as with angiographic studies.[6] Due to their fragility, these vessels are a source of hemorrhage within this space, as evidenced by Ito et al. in a study using tagged red blood cells demonstrating perpetual bleeding within this space.[7] Thus, perpetuating the cycle of hemorrhage and inflammation, this is exemplified by the persistence and pestilence of chronic subdural hematomas. This cyclical inflammation alludes to the possible mechanism by which steroids may have an effect. These membranes forming from the dural border layer derive their vascular supply from dural vasculature presenting another therapeutic target with arterial embolization.[8,9]

The MMAE can be implemented to reduce recurrence of CSDHs or as an alternative minimally invasive management strategy for CSDH.[10] In a meta-analysis of the available literature, Srivatsan et al. have shown a significantly lower recurrence rate for patients treated with MMAE, despite more patients on anticoagulation or antiplatelet therapy in the MMAE cohort. The recurrence rate after MMAE was found to be 2.1%, whereas the recurrence rate in the surgically treated group varied between 11-29% depending on the surgical intervention.[11]

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The purpose of this study was to demonstrate the feasibility of MMAE for the treatment of CSDH avoiding the risk of interrupting anticoagulation and reducing the perioperative risk in a Canadian health care institution.

METHODS

Patients (N=4) diagnosed with CSDH and treated with MMAE are presented. Two patients presented with bilateral lesions equaling a total of six subdural hematoma lesions. Institutional review board (IRB) approval was granted. Patient demographics were collected. Clinical outcomes were supplemented by follow up CT imaging. A total of seven middle meningeal artery (MMA) occlusions were performed. All procedures were performed with polyvinyl alcohol (PVA) particles to occlude the desired middle meningeal branches. All procedures were tolerated well with no peri-procedural or technical complications.

Technical note

All procedures were done under light conscious sedation and local anesthetic in the interventional radiology suite. Employing standard angiographic techniques and equipment, the femoral artery was cannulated, and a 6 French sheath inserted. The carotid artery was subsequently cannulated with an H1 catheter (Cook Medical, Bloomington, IN, USA). Imaging of the internal and external carotid anatomy and pathology was then evaluated looking for important anatomic variants and disease. Occlusion of external carotid supply with internal flow to the brain or ophthalmic supply from the MMA would change the risk benefit ratio in this otherwise straightforward procedure. Usual subtraction and road mapping techniques allowed a Renegade microcatheter (Boston Scientific, Marlborough, MA, USA) and Whisper guidewire (Abbott Cardiovascular, Ann Arbor, MI, USA) to be advanced into the MMA. The MMA and its respective branches were then injected, and the anatomy defined **(Fig. 1-Left).** Care was taken to elucidate any intradural supply from the meningeal vessels. The middle and posterior branches were then occluded using contour PVA particles (Boston Scientific, Marlborough, MA, USA) **(Fig. 1-Right).**

It is critical to avoid reflux into the non-target territories, and in addition if the procedure was prolonged 5000 units of unfractionated heparin was sometimes administered intra-arterially through the femoral sheath, to avoid catheter associated embolism. A meticulous inspection for any external to internal anastomoses is also of utmost importance. Ophthalmic artery may derive blood supply or contain collaterals from middle meningeal vessels. Another variant to be avoided is a petrosal branch supplying the petrous segment of the facial nerve[12,13].

RESULTS

Four patients underwent MMAE for the treatment of CSDH to avoid the risk of cessation of anticoagulation and the expected perioperative risk. All 4 patients were males and the median age was 74 years. Two patients were on antithrombotic therapy at the time of intervention. Patient follow-up occurred at 1, 3, 6, and 9 months post-operatively. Resolution of symptoms and significant reduction of hematoma thickness was evident (up to 14 mm) at follow-up. Improvement in all cases was confirmed by CT and clinical evaluation. No patients suffered from complications or recurrence.

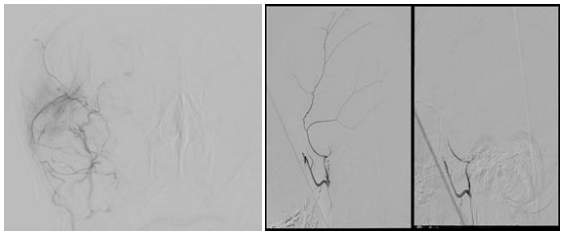

Fig 1 Left: Distal external carotid artery injection, demonstrating the maxillary artery as well as the middle meningeal artery. Shown here is an intracranial blush of contrast overlying the convexity, commonly seen with CSDH, demonstrating the dural arterial supply of these collections. Right: First image shows pre-embolization injection of the MMA showing both distal branches. Second image shows postembolization run showing complete occlusion of both branches.

Case 1

A 77-year-old male, with a history of rheumatic fever and a previous Bentall procedure, on warfarin for a mechanical aortic valve presented with a severe headache and a normal neurological exam. A CT head obtained at the time of presentation demonstrated bilateral chronic subdural hematomas **(Fig. 2-Left)**. As a consequence of his anticoagulation and contraindication to its withdrawal, successful bilateral MMAE was performed. There was no cessation of anticoagulation during his hospital stay or at discharge from hospital. He was seen subsequently for follow-up to 7 months, complete resolution of the chronic subdural hematomas on last follow-up imaging is shown **(Fig. 2-Right).** Hematoma thickness pre-intervention was 8mm on both sides and was successfully reduced to 0 mm on both sides.

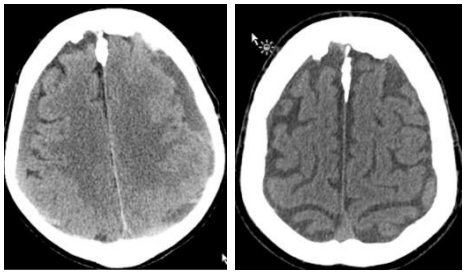

Fig 2 Left: Non-contrast CT head on initial presentation, demonstrating bilateral CSDH with minimal sulcal effacement and compression. Right: Non-contrast CT head at last follow up, demonstrating complete resolution.

Case 2

A 72-year-old male with significant cardiac history including coronary artery disease treated previously with a coronary artery bypass graft (CABG), and atrial

fibrillation for which he is on warfarin. He sustained a fall and a CT head was performed which showed bilateral chronic subdural hematomas with extensive septations. At that time, he was asymptomatic, the collections were treated conservatively, and the warfarin was withheld. However, he presented a month later to hospital with a cough and shortness of breath. An electrocardiogram (ECG) showed an ST-elevation myocardial infarction (STEMI), he was seen by cardiology and considering his late presentation and bilateral subdural collections was not given a thrombolytic agent. He was started on dual antiplatelet therapy in the form of Aspirin and Plavix. He was admitted into the coronary care unit and a CT head was done which showed similar bilateral subdural hematomas **(Fig. 3-Left)**.

A decision was made to proceed with bilateral MMAE after an informed discussion with the patient and his family. The procedure was successful with both anterior and posterior branch of the MMA embolized bilaterally. After the procedure he developed slurred speech and an urgent CT stroke protocol including a plain CT head and an aortic arch to circle of Willis angiogram was done. This revealed slight increase in the size of the collections, which has been described previously after embolization in addition to contrast stagnation. His symptoms quickly resolved by the time the scan was done. Bilateral pulmonary emboli were found on the CT angiogram and considered a likely cause of these transient neurologic symptoms by paradoxical embolism. As such he was started on Heparin and transitioned to a novel anticoagulant. He continued to progress well during his hospitalization and was asymptomatic with regard to the bilateral subdural hematomas. The dual antiplatelet therapy was stopped prior to his discharge home. Patient was seen for follow-up at 1, 3, and 9 months with a reduction of hematoma thickness to 1mm on both sides from 13mm on the right side and 14 mm on the left side pre-operatively. He continued to be asymptomatic with serial CT head scans showing complete resolution of his collections **(Fig. 3-Right)**. His anticoagulation was not discontinued throughout this period.

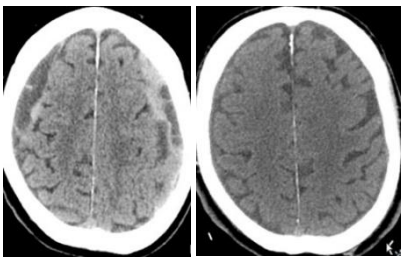

Fig 3 Left: Initial non-contrast CT head demonstrating bilateral subdural collections with significant septations. Right: Non-contrast CT head at 9 months follow up, with complete resolution of the previous bilateral CSDH associated with significant septations.

Case 3

A 67-year-old male presented with headache for 4 months after a history of a fall. He was neurologically intact and a CT head was performed which showed a left sided subdural hematoma thickness of 10 mm **(Fig. 4-Left).** Initially he was started on steroids and showed rapid resolution of his symptoms. Considering the current improvement, he continued on a four-week course of steroids with continued resolution of his headaches and a repeat CT which showed improvement. Follow up CT scans showed progression of the size of the subdural, though he remained neurologically intact during follow up visits. Considering the asymptomatic nature of the subdural and the persistence of the lesion, bilateral MMAE was proposed. The procedure was performed with no complications and he tolerated the procedure well. He remained neurologically intact and asymptomatic after the procedure. A follow up CT head was obtained 1-, 6-, and 9-months post procedure showing significant decrease in the size of his collection (0 mm) nearing resolution **(Fig. 4-Right).**

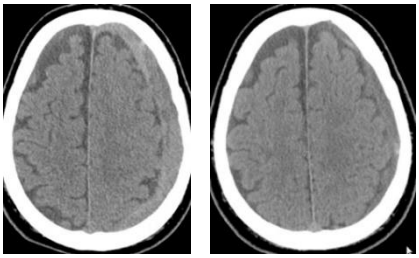

Fig 4 Left: Non-contrast CT head after 4 weeks of steroid therapy, demonstrating persistence of the left CSDH. Right: Non contrast CT head done at 6 months follow up demonstrating near resolution of the left CSDH.

Case 4

A 92-year-old male who was frail with a significant history of chronic obstructive pulmonary disease (COPD) presented with functional decline and left sided weakness. The CT head showed a large right chronic subdural hematoma with elaborate septations **(Fig. 5-Left)**. Considering his overall surgical risk, he was treated with a course of steroids and showed some gradual improvement. A subsequent right MMAE was done as a more definitive long-term treatment. The procedure was successful with no complications. He showed improvement in his left sided weakness and cognition. A follow up CT head was done immediately after the procedure showing early reduction in the size of the hematoma **(Fig. 5-Right)**. Unfortunately, he passed away prior to the 6 month follow up scan from other systemic conditions not related to the procedure or the chronic subdural hematoma.

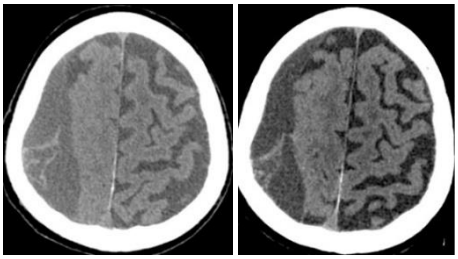

Fig 5 Left: Initial non-contrast CT head at presentation, showing the large right sided CSDH with significant sulcal effacement. Right: Immediate follow up non-contrast CT head, showing early reduction in the size of the CSDH with mild improvement of the sulcal effacement.

DISCUSSION

Our case series presents fragile patients on anticoagulants (N=4) diagnosed with CSDH and treated with MMAE. The MMAE has been introduced within the armamentarium as a treatment for chronic subdural hematomas. The merits of this intervention are the minimally invasive nature of this procedure and the direct impact on membrane vascularization without the need for a craniotomy. Certain circumstances where mass effect is present should continue to be treated with evacuation through one of the traditional techniques. However, for persistent collections and patients on long term anticoagulation or with a high surgical risk, MMAE provides a novel and attractive treatment modality. The geriatric population is commonly afflicted, where multiple comorbidities, antiplatelet, and anticoagulants are customary.

The MMAE has a positive therapeutic effect on CSDH and is more effective than conventional treatment.[14] Conservative treatment with corticosteroids has also been implemented as a stand-alone treatment or in addition to surgical hematoma drainage.[15] Although no established guidelines for management of CSDH currently exist, if the hematoma is >10 mm thick and >5 mm midline shift, it is suggested that corticosteroid use alone is not warranted and surgical intervention is indicated. In fact, symptomatic CSDH patients showed less favorable outcomes compared to control group under the treatment of oral dexamethasone.[16] Repeating irrigation and drainage are still effective for recurrence of CSDH, however, with appropriate selection, this procedure is an important alternative particularly in the frail, anticoagulated or coagulopathy population.[15]

There is ample evidence that contradicts a simple traumatic etiology for chronic subdural hematoma and alludes to a more active inflammatory and angiogenic pathology. The subdural space is not a normal anatomical space and is absent in the disease-free state.[4] The chronicity, progressive nature, and recurrence of these lesions demonstrate an active pathological process occurring contrary to an isolated reparative process.[2] The development of these collections in the absence of trauma or radiological evidence of acute blood again suggests a more complex etiology than simple hemorrhage and healing. Recently detailed pathological examination of the membranes associated with chronic subdural collections has suggested novel treatment modalities, in this fragile patient population.

Multiple studies have examined the dural-arachnoid interface with electron microscopy and have found an adherent and intimate interface.[9] The dural border cells constitute the innermost layer of the dura, a thin layer of interspersed fibroblasts with a prominent extracellular matrix lacking collagen and with sparse intercellular adhesions. This is contrary to the collagen rich, thicker more superficial dura. This layer is adherent with the most external arachnoid layer, the arachnoid border cell layer. The arachnoid border cell layer in contrast consists of many intercellular adhesions and little extracellular matrix. These two layers are juxtaposed and adherent, but with few tight junctions connecting the two layers. However, both layers are heavily anchored in opposing directions. The arachnoid border cell layer fastened via arachnoid trabeculae to the pia, and the dural border cell layer affixed to the periosteal dura. This in turn creates a tight but frail junction between the dura and arachnoid. In careful histological examinations of the meninges, the lack of a true physiologic space between any dural and arachnoidal layers is vividly seen. These two surfaces naturally do not interact with an intermediate space, nor do they normally move relative to each other. This is in contrast to other mesothelial lined membranes in the body such as the pleura.[4]

This fragile conjoined layer is thus prone to shearing and separation from trivial injuries. Models where blood was injected within the dura-arachnoid interface showed reproducible separation of the dural border cell layer.[17] Also, pathological and histological examinations of chronic subdural hematomas show the inner membrane layer consisting of proliferated dural border cell layer. The familiar action of 'lifting the dura' by neurosurgeons is not a consequence of peeling two distinct layers, but rather separating the thin dural border cell layer. The historically described subdural space is only the consequence of separation of the dural border cell layer whether by hemorrhage or iatrogenic intent. Hence in chronic subdural hematomas, surrounded by the dural border cell layer which is of mesenchymal origin, the reactive and inflammatory responses seen are analogous to wound healing. Proliferation of fibroblasts, secretion of angiogenic substances, neovascularization, and fibrinolysis are evident. Subsequent inflammation, development of fragile neo-vasculature, rebleeding events, fibrinolysis and blood degradation products all create a vicious cycle with a resultant accumulation of the characteristic subdural fluid.[4,6] This self-perpetuating cycle, in a pathologically created space produces the tenacious entity of a chronic subdural hematoma; unlikely to spontaneously resolve and notoriously recurrent. Although contentious, this may be the mechanism by which steroids work, targeting the inflammatory reaction in this membrane.

The proliferated layer of dural border cells is biologically active and dependent on the newly formed frail vessels. These vessels are also thought to be the source of active hemorrhage within this space, as evidenced by Ito et al. in a study using tagged red blood cells demonstrating perpetual bleeding within this space.[7] Some authors have also shown the presence of exudate within this space, probably seepage from these abnormal vessels. The presence of these vessels has been readily demonstrated with pathological analysis of this layer as well as with angiographic studies.[6,13] This vascularization is derived from the overlying dura, which extends its vascularity from the meningeal vessels.[9]

The MMAE has been shown to be a safe and minimally invasive therapeutic modality for select cases of chronic subdural hematoma.[12,14] However, a careful interrogation of the dural arterial anatomy must be carried out in every case prior to occlusion of the target vessels. This includes careful inspection for anastomoses with the intradural arterial system. Specifically, as illustrated by previous authors, the two most common forms of intradural meningeal artery supply are the ophthalmic artery and cranial nerve VII through a petrous branch.[13,18]

This case series demonstrates that MMAE as a primary treatment for CSDH was effective in reducing hematoma thickness at our healthcare facility, despite maintenance of anticoagulation. Objective evidence of efficacy is seen in progressive accelerated resorption of these collections seen in successive CT scans, in the absence of any other intervention. Timeline of the effect of treatment seems to vary among our patients but was seen as early as 6 weeks.

CONCLUSION

The MMAE has been shown to be an effective treatment for chronic subdural hematoma by previous studies outlined above. This innovative approach to a common and troublesome pathology in a frail population is now available to vulnerable individuals and others with skills and equipment required for embolization in the territory of the external carotid artery.

List of abbreviations

CABG: Coronary artery bypass graft
COPD: Chronic obstructive pulmonary disease
CSDH: Chronic subdural hematoma
CT: Computed tomography
ECG: Electrocardiogram
IRB: Institutional review board
MMA: Middle meningeal artery
MMAE: Middle meningeal artery embolization
PVA: Polyvinyl alcohol
STEMI: ST-elevation myocardial infarction

Disclosure

The authors report no conflict of interest in the materials or methods used in this study or the findings specified in this paper.

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REFERENCES

1. Bond B, Bond K, Kattah J. Middle meningeal artery embolization for the treatment of chronic subdural hematoma: A systematic literature review. Neurology. 2020;94(15 Supplement):2552.
2. Fiorella D, Arthur AS. Middle meningeal artery embolization for the management of chronic subdural hematoma. J Neurointerv Surg. 2019;11(9):912-915.
3. Baechli H, Nordmann A, Bucher HC, Gratzl O. Demographics and prevalent risk factors of chronic subdural haematoma: Results of a large single-center cohort study. Neurosurg Rev. 2004;27(4):263-266.
4. Haines DE, Harkey HL, Al-Mefty O. The "subdural" space: A new look at an outdated concept. Neurosurgery. 1993;32(1):111-120.
5. Yamashima T, Yamamoto S. The origin of inner membranes in chronic subdural hematomas. Acta Neuropatho. 1985;67(3-4):219-225.
6. Yamashima T, Yamamoto S. How do vessels proliferate in the capsule of a chronic subdural hematoma? Neurosurgery. 1984;15(5):672-678.
7. Ito H, Yamamoto S, Saito K, Ikeda K, Hisada K. Quantitative estimation of hemorrhage in chronic subdural hematoma using the 51Cr erythrocyte labeling method. J Neurosurg. 1987;66(6):862-864.
8. Tanaka T, Kaimori M. Histological study of vascular structure between the dura mater and the outer membrane in chronic subdural hematoma in an adult. No Shinkei Geka. 1999;27(5):431-436.
9. Mack J, Squier W, Eastman JT. Anatomy and development of the meninges: Implications for subdural collections and CSF circulation. Pediatr Radiol. 2009;39(3):200-210.
10. Suzuki K, Sugita K, Akai T, Takahata T, Sonobe M, Takahashi S. Treatment of chronic subdural hematoma by closed-system drainage without irrigation. Surg Neurol. 1998;50(3):231-234.
11. Srivatsan A, Mohanty A, Nascimento FA, et al. Middle meningeal artery embolization for chronic subdural hematoma: Meta-analysis and systematic review. World Neurosurg. 2019; 122:613-619.
12. Hashimoto T, Ohashi T, Watanabe D, et al. Usefulness of embolization of the middle meningeal artery for refractory chronic subdural hematomas. Surg Neurol Int. 2013;4(1):104.
13. Link TW, Rapoport BI, Paine SM, Kamel H, Knopman J. Middle meningeal artery embolization for chronic subdural hematoma: Endovascular technique and radiographic findings. Interv Neuroradiol. 2018;24(4):455-462.
14. Ban, SP, Hwang G, Byoun HS, et al. Middle meningeal artery embolization for chronic subdural hematoma. Radiology. 2018;286(3):992-999.
15. Tempaku A, Yamauchi S, Ikeda H, et al. Usefulness of interventional embolization of the middle meningeal artery for recurrent chronic subdural hematoma: Five cases and a review of the literature. Interv Neuroradiol. 2015;21(3):366-371.
16. Hutchinson PJ, Edlmann E, Bulters D, et al. Trial of dexamethasone for chronic subdural hematoma. N Engl J Med. 2020;383(27):2616-2627.
17. Zwetnow NN, Orlin JR, Wu W, Tajsic N. Studies on supratentorial subdural bleeding using a porcine model. Acta Neurochirurgica. 1993;121(1-2):58-67.
18. Weigel R, Schmiedek P, Krauss JK. Outcome of contemporary surgery for chronic subdural haematoma: Evidence based review. J Neurol Neurosurg Psychiatry. 2003;74(7):937-943.