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Middle meningeal artery embolization for chronic subdural hematoma-pathophysiology and radiological findings

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1. Introduction

Chronic subdural hematoma (cSDH) is a common neurological disorder with an increasing incidence rate and a deceptively poor prognosis.[1] Historically, the mainstay of treatment of symptomatic subdural hematomas has been surgical drainage. Perioperative mortality rates for patients with chronic SDH, however, can be as high as 14%. For an additional 20% of the surviving patients, chronic subdural hematomas result in significant disability.[1]

Recently, embolization of the middle meningeal artery (MMA) has emerged as a safe and effective therapy for chronic SDH that can be applied both as primary treatment and as an adjunct to surgery.[2] The mechanism of MMA embolization, however, is not well understood. The idea that embolization of an artery can lead to a resolution of a hematoma that has traditionally been thought to result from recurrent venous bleeding can seem paradoxical and is not universally accepted. Here we present two illustrative cases which provide visual evidence of the connection between middle meningeal artery neovascular branches and the membranes that form in chronic SDHs.

1.1. Case presentation

1.1.1. Illustrative case 1

A 67-year-old man with no remarkable past medical history presented to our emergency department (ED) with a chief complaint of holocranial headaches and progressive memory loss that had significantly worsened over the prior week. The patient's family recalled that he had hit his head several times against the roof of a golf cart a few weeks prior to his admission, but no falls or major traumatic events were reported. The patient was not on anti-platelet or anticoagulant medications. Examination on admission revealed no focal neurological deficits. A non-contrast CT scan of the brain demonstrated a large left frontoparietal subdural hematoma measuring 2.1 cm in maximal thickness (MT) and causing a 7.5 mm left to right midline shift (MLS) (Fig. 1A). Acute and chronic blood products were present, separated by membranes. Based on the CT scan findings and the patient's acute progressive memory loss, he was referred to neurointerventional surgery for selective embolization of the left MMA. During the injection of n-BCA glue into the anterior division of the left MMA, wispy, linear staining along the left hemisphere was observed (Fig. 1B–C). A follow-up CT scan demonstrated the presence of n-BCA glue within the membranes of the subdural hematoma, connected by bridging branches to the left MMA (Fig. 1D). There were no complications following the procedure. The patient clinically improved, with resolution of his headache and improvement in his memory function. A CT scan of the brain was performed 4 weeks later and demonstrated a 25% reduction in the MT of the SDH and a 40% decrease in the MLS (4.2 mm) (Fig. 1D). No areas of new hemorrhage were identified.

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Abbreviations

MMA -	middle meningeal artery
cSDH -	chronic subdural hematoma
SDH -	subdural hematoma
DBC -	dural border cell
MLS -	Midline shift
MT	Maximal thickness
n-BCA –	n Butyl Cyanoacrylate

1.1.2. Illustrative case 2

A 77- year-old man with no remarkable past medical history presented to the ED with an occipital headache that had worsened over the two prior weeks. He had no focal neurological signs. Five months prior to this admission, he had undergone surgery through two left fronto-parietal burr holes for a large heterogeneous subdural collection. The pre-embolization CT scan of the brain demonstrated a re-accumulated chronic subdural hematoma (Fig. 2 E). The patient underwent a left MMA embolization with n-BCA. A follow-up CT scan demonstrated the presence of the liquid embolic within the membranes of the hematoma (Fig. 2F–I).

2. Discussion

The classic theory of cSDH recurrence is rooted in the idea that SDH is the result of a venous bleed from ruptured bridging veins within the subdural space. The rebleeding of these veins, possibly by repeated trauma, was therefore classically attributed to be the cause of the recurrent nature of cSDH.[3]

A more updated view proposes that re-bleeding from newly formed, fragile arteries is the cause of growth of cSDHs. SDHs occur between the dura and arachnoid within an area known as the dural border cell layer (DBCL). Within hours of the initial venous bleed, the SDH coagulates and forms a clot. On CT, this clotted blood is seen as a hyperdense extra-axial collection that spreads over the affected hemisphere. An inflammatory response ensues and, over the next four to ten days, a membrane forms around the collection, composed of fibroblasts and collagen.[4] The DBCL cells have the ability to phagocytose cells and act to resorb the hemorrhage. The formation of the membrane, however, also gives rise to a fragile neovasculature. These fragile new vessels continuously exude blood into the subdural space.[4] The balance between the rate of hematoma phagocytosis and re-bleeding from the neovasculature determines whether a cSDH will resolve or continue to enlarge. Cutting off the blood supply to the neovasculature tips the balance toward resorption and hematoma resolution.

Our illustrative cases elegantly show that it is the MMA that supplies the neovasculature of the membranes of cSDHs, and that liquid embolic injection into the middle meningeal artery can target these membranes. The MMA, a branch of the maxillary artery, supplies blood to the meninges, and thus to the dura mater. Ex-vivo investigations have previously shown that branches of the middle meningeal arteries extend into the newly formed membranes of cSDHs. Through microcatheter injections of contrast into the middle meningeal artery and DYNA-CT

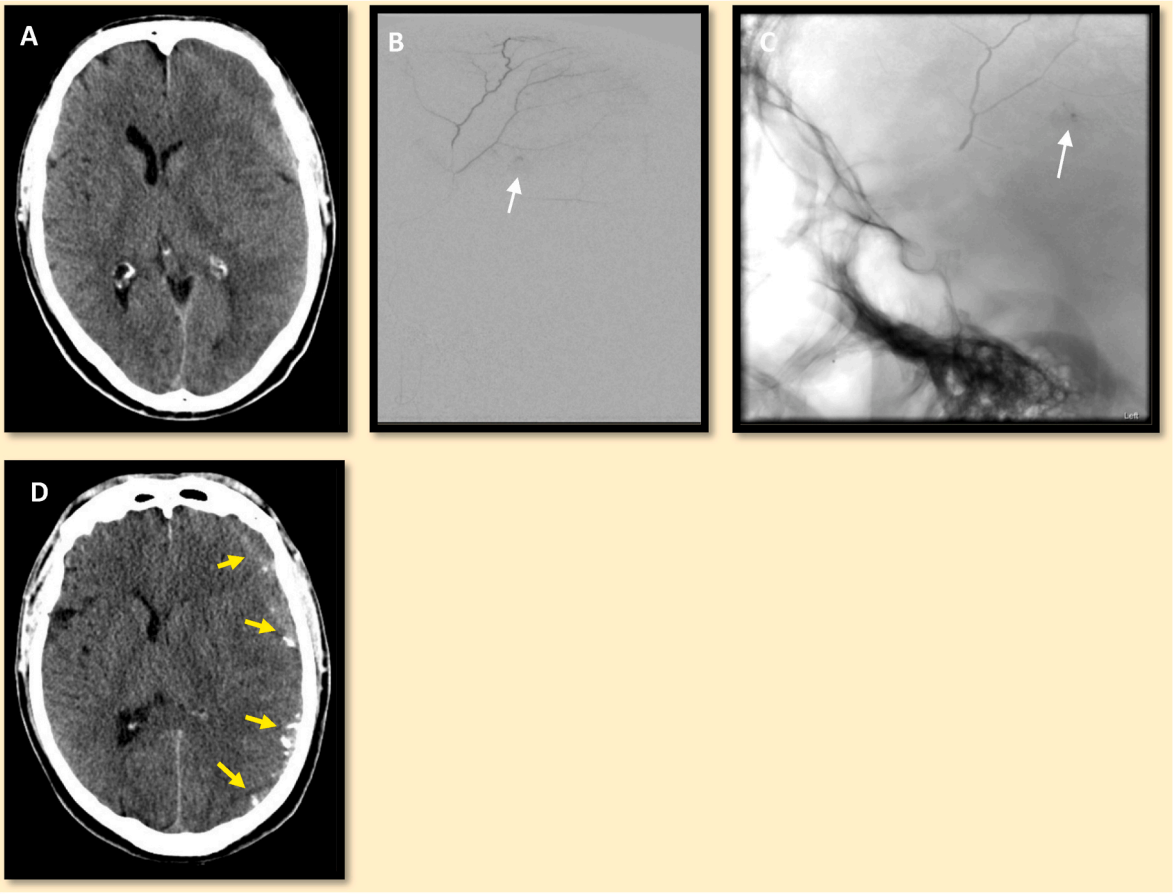

Fig. 1. A. Non-contrast CT brain demonstrating a chronic left subdural hematoma with different densities segregated into multiple compartments by membranes. B. Microinjection of contrast into the anterior division of the left MMA. White arrow points to the enhancement of the hematoma membrane. Post-embolization unsubtracted lateral view of the skull demonstrating n-BCA cast within the hematoma membrane. Post-procedure non-contrast CT scan showing n-BCA within the membranes of the subdural hematoma (yellow arrows).

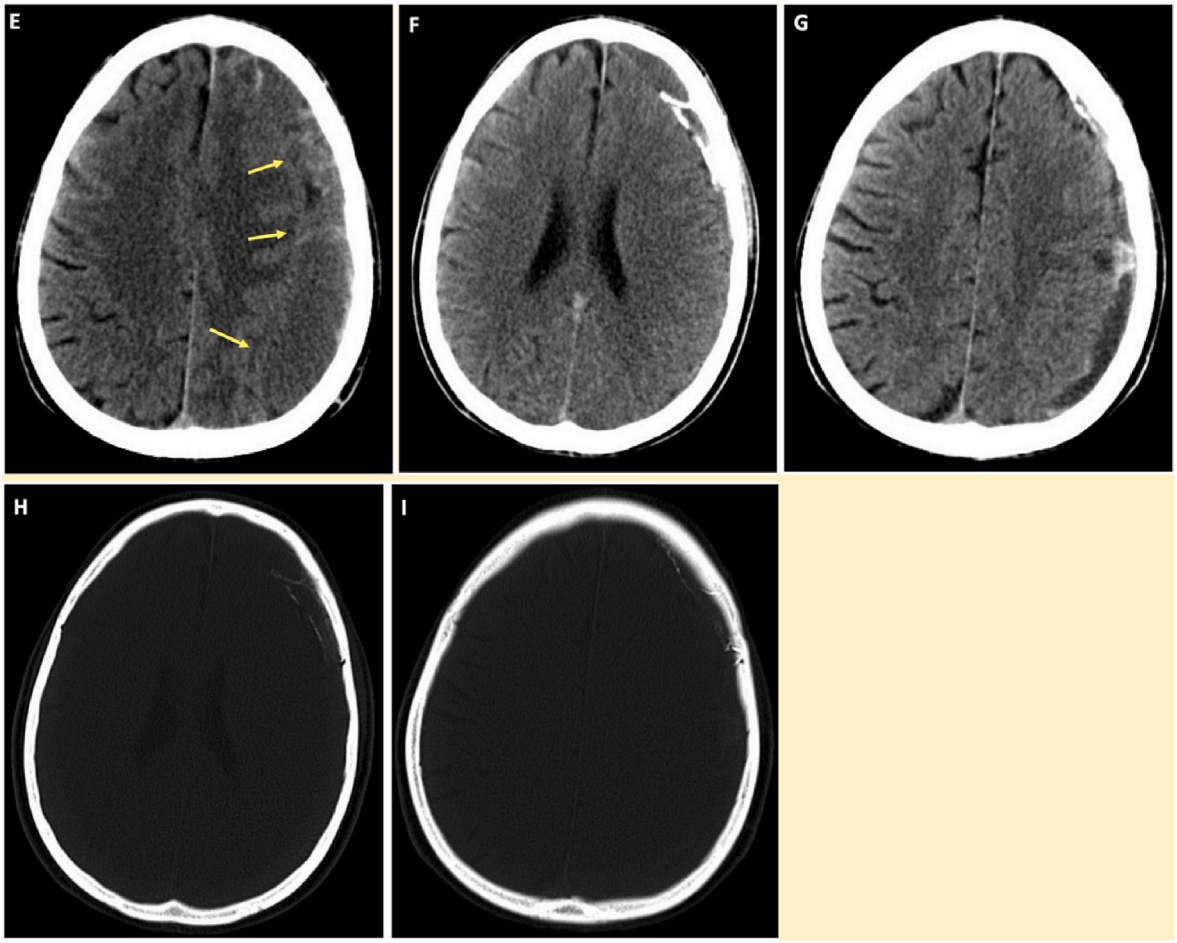

Fig. 2. E. Non-contrast CT brain demonstrating a chronic left subdural hematoma (yellow arrows) with a mixed attenuation that features multiple loculated compartments filled with different stages of evolution with 1.9 cm of maximal thickness and 8 mm of left-to-right MLS. Figure F–**G**. Post-procedure non-contrast CT head scan (brain window) showing n-BCA within the septations and membrane of the chronic subdural hematoma. Figure H–**I.** Post-procedure non-contrast CT head scan (bone window) demonstrating n-BCA in the septations.

imaging in patients with cSDH, Shapiro et al demonstrated patchy areas of hematoma membrane enhancement.[5] Our case demonstrates the in-vivo penetration of n-BCA glue into the membranes of the subdural hematoma. The casts of the liquid embolic injected into the anterior division branch of the left MMA can be traced on the post-procedure CT directly from the MMA into the membranes of the hematoma.

These cases directly show the connection between the branches of the middle meningeal artery and the neovascular channels that form in the membranes of cSDHs. Moreover, these cases also show the ability of liquid embolic agents to penetrate directly into the vessels of these membranes, reach the site of rebleeding, and shut down their blood supply.

Embolization of the MMA to treat cSDH is emerging as an effective and low risk procedure and gradually becoming integrated into clinical practice. Its main benefit is the prevention of recurrent bleeding and gradual hematoma resorption. For patients who present without significant focal neurological deficits and severe mass effect, as in our two illustrative cases, it can be used for primary treatment instead of surgery. However, in our experience, the complete resolution of a subdural hematoma after MMA embolization can take more than 12 weeks. Patients with large mass-effect hematomas and focal neurological deficits are not good candidates for primary embolization alone. Instead, they are considered for surgery alone or a combination of surgical evacuation and embolization. The literature indicates that surgery and embolization yields favorable results, demonstrating low recurrence and reoperation rates.[6,7] It is important for neurosurgeons to be aware of the mechanism of MMA embolization when performing surgical evacuation. Whenever possible, burr hole/craniotomy sites should be placed to avoid disruption of the main MMA trunks. This can keep these conduits open for possible subsequent MMA embolization and delivery of embolic material to the cSDH membranes.

3. Conclusion

In contrast to what is often presumed, cSDHs are not an indolent entity, and the morbidity and mortality related to them are not negligible. For years, surgical drainage was the mainstay of treatment. However, a thorough knowledge of the pathophysiology of cSDH demonstrates the important role arterial vessels play in the progressive expansion of these hematomas. These cases add to the growing body of literature demonstrating that embolization of the MMA is a safe and effective means of resolving cSDH, as it cuts off the blood supply to the membranes of the hematoma and prevents re-bleeding. As new research becomes available, questions such as who would be the best candidates for this treatment modality, radiological findings predictive of a better response, and a more comprehensive understanding of the pathophysiology of cSDH may be attained, leading to improvement in current therapeutic options as well as the development of new, innovative treatment strategies for this challenging disease.

CRediT authorship contribution statement

Raphael Bastianon Santiago: Conceptualization, Investigation, Methodology, Writing – original draft, Writing – review & editing. **Camille Jastrzebski:** Methodology, Writing – original draft. **Elias Dakwar:** Validation, Visualization. **Badih Adada:** Formal analysis, Methodology, Supervision, Visualization. **Hamid Borghei-Razavi:** Conceptualization, Investigation, Visualization. **Michal Obrzut:** Conceptualization, Supervision, Writing – original draft, Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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