

Lessons from Dural Arteriovenous Fistula Angioarchitecture: A Case for Multi-Vessel Assessment in Middle Meningeal Artery Embolization for Chronic Subdural Hematoma

Ahmed Samy Sheta, MBBCh* and Mohamed Hassanin, MD

Department of Neurosurgery, MTI University, Cairo, Egypt.

*Correspondence:

Ahmed Samy Shetta MBBCh, Department of Neurosurgery, MTI university, Cairo, Egypt.

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ABSTRACT

Middle meningeal artery embolization (MMAE) now has randomized evidence from five completed trials, with pooled analyses increasingly favoring benefit. Yet no trial, and no predictor identified to date, explains which patients fail treatment through a vascular mechanism. We propose that a subset of chronic subdural hematoma (CSDH) patients has clinically significant membrane supply from arteries beyond the MMA — the accessory meningeal, ascending pharyngeal, and occipital arteries — the same anastomotic network that makes multi-vessel assessment standard practice in dural arteriovenous fistula (DAVF) treatment planning. We review the anatomic basis for this network, direct evidence from DAVF literature linking multiple feeders to treatment failure, and gaps in how CSDH trials have characterized MMA anatomy. We propose a multi-vessel angiographic assessment protocol, adapted from DAVF practice, to identify this subset prospectively.

Keywords

Chronic subdural hematoma, Middle meningeal artery embolization, Middle meningeal artery, Dural vascular anatomy, Meningeal collateral circulation, Accessory meningeal artery, Ascending pharyngeal artery, Occipital artery.

Introduction

Middle meningeal artery embolization (MMAE) has moved rapidly from case series to randomized evidence. Five trials — EMBOLISE, STEM, MAGIC-MT, EMPROTECT, and MEMBRANE — have now been completed, using three different embolic agents (Onyx, Squid, and TRUFILL n-BCA) across surgical and non-surgical populations [1-5]. Pooled analyses restricted to the original three trials found a weak, non-significant effect for MMAE as an adjunct to surgery (RR 0.60, 95% CI 0.19–1.88) [6]. A separate pooled analysis adding EMPROTECT reported a tighter, significant effect (RR 0.40, 95% CI 0.28–0.58) [4] and MEMBRANE, the most recently completed trial, was independently positive [5]. The overall evidence base is trending toward benefit. What remains

unresolved is not whether MMAE works on average, but why it fails in specific patients — a question none of the five trials, individually or pooled, were designed to answer.

The clearest existing clue comes from outside the trials. In a multicenter analysis of 636 embolizations, small MMA diameter (<1.5 mm) was the only factor independently associated with both clinical and radiographic failure [7]. If the dominant vessel is small, something else is likely supplying the territory, a question the study identifies without asking.

Even as the trial evidence has matured, the search for predictors of individual patient benefit has come up empty by every variable examined so far. A 2025 meta-analysis of EMBOLISE, STEM, MAGIC-MT, and EMPROTECT catalogs the risk factors already investigated for CSDH recurrence: antithrombotic and antiplatelet therapy, age, presenting GCS, coagulopathy, obesity, liver disease, prior stroke, bilateral hematomas, drain duration, hematoma septation and layering, hematoma density and width, post-operative

residual volume, pneumocephalus, degree of brain re-expansion, and even ABO blood type [4]. Non-MMA arterial contribution to the membrane is absent from this list. Tellingly, EMPROTECT — the one trial specifically designed to enroll patients at high risk of recurrence, the population in which a true effect should be most detectable — failed to reach significance, and the same meta-analysis attributes this to the embolic agent used rather than to any property of the patients selected [4]. The authors note directly that their analysis “relied on aggregate data from published trials rather than individual patient-level data, which precluded more detailed subgroup analyses” — the field has not yet had the data to ask the question this paper raises [8].

Anatomy: The Dura Is Not a Single-Vessel Bed

The MMA is the dominant, but never the only, arterial supply to the dura. The accessory meningeal artery contributes an adjacent territory; the ascending pharyngeal artery's neuromeningeal trunk supplies the clivus and adjacent dura; the occipital artery reaches the posterior dura through transosseous and mastoid branches; the ophthalmic artery contributes anterior falcine and ethmoidal branches to the anterior fossa dura [8]. These vessels anastomose freely with the MMA and with each other.

The clearest demonstration of this redundancy comes from angiographic series of dural arteriovenous fistulas in the parafalcine/superior sagittal sinus region — anatomically adjacent to where CSDH membranes typically form along the convexity. In a systematic review of 51 angiographically confirmed cases, the MMA was present as a feeder in 100% of cases, but bilateral MMA supply — the contralateral vessel crossing the midline to contribute — was present in 78.4% [9]. The superficial temporal artery contributed in 62.7%, and the occipital artery in 49.0% [9]. Even restricted to a single named vessel category, multiplicity, not singularity, is the norm in this vascular bed.

What DAVF Treatment Already Knows About This Weakness

Dural arteriovenous fistulas arise from and are sustained by this same dural network, and the field has already generated hard evidence on what happens when a feeder is missed. In a cohort of 51 patients with high-grade cranial DAVFs, recurrence occurred in 13.1% of cases after apparent complete radiographic occlusion — and every single recurrent case had multiple feeders present before embolization [10]. In a separate series of 71 spinal DAVF patients, multiple feeders were present in 73.7% of patients whose initial embolization was incomplete, compared to just 27% of those who achieved complete occlusion ($P < 0.001$) [11]. The relationship is consistent, direction-specific and statistically robust across two independent series in two different DAVF locations: more feeders predicts more failure. Multi-vessel angiographic assessment before treatment is standard DAVF practice for exactly this reason.

What the CSDH Trials Actually Did — and Did Not Do

None of the trials' angiographic protocols were built to answer this question, because none was looking for it. In EMBOLISE, pre-embolization angiography characterized the MMA and screened

specifically for collateral pathways to the cerebral and central retinal arteries — a safety check against non-target embolization into the orbit, not an assessment of whether the membrane's blood supply extended beyond MMA territory [2]. Where a dangerous anastomosis was identified, the protocol response was to embolize more proximally with coils rather than pursue distal penetration [2]. MAGIC-MT achieved combined main-trunk-and-distal occlusion in the large majority of cases,¹ but a separate nonacute-SDH protocol defined technical success as either distal branch embolization or main-trunk occlusion alone, treating the two as equivalent. In no trial was non-MMA contribution to the membrane measured, reported, or used to stratify outcome.

It is worth noting that a comparison of embolization completeness against trial-level outcome does not, by itself, resolve this question cleanly. MAGIC-MT achieved the most comprehensive technical embolization of the three original trials (combined trunk-and-distal occlusion in 92.2% of cases) yet produced the weakest result, while EMBOLISE, with roughly half its patients receiving distal branch treatment, produced the strongest [1,6]. This is more plausibly explained by differences in enrolled populations — MAGIC-MT's control arm event rate was 9.9%, against 36% in EMBOLISE's — than by technique itself. This confound is precisely why the question requires patient-level angiographic data within a controlled population, rather than inference across structurally different trials.

The Hypothesis

If a subset of CSDH patients has clinically significant membrane supply from the accessory meningeal, ascending pharyngeal, or occipital arteries — the same vessels that make DAVF treatment planning multi-vessel by default — that subset would be invisible in existing trial datasets. A proximal, safety-motivated embolization in exactly these patients would be expected to fail for a reason no trial was positioned to detect. The small-MMA-diameter finding³ is the closest the CSDH literature has come to this question, discovered without the underlying mechanism being named or tested.

Proposed Multi-Vessel Assessment Protocol

We propose a pre-procedural angiographic assessment, adapted directly from DAVF treatment planning, for patients being considered for MMAE: selective injection of the ipsilateral external carotid artery to characterize MMA caliber and course, with attention to filling of the membrane territory via accessory meningeal, ascending pharyngeal, and occipital branches; bilateral assessment given the demonstrated frequency of contralateral MMA contribution [8] and documentation of residual membrane filling after MMA embolization, before the procedure is considered complete, analogous to the post-embolization control run used in DAVF treatment to confirm elimination of shunt filling.

Discussion and Limitations

This is a hypothesis grounded in anatomic plausibility and an analogy to a related, better-characterized disease process — not a

demonstrated mechanism. No study has yet measured non-MMA contribution to the CSDH membrane directly, nor correlated it with embolization outcome. The proposed protocol requires prospective validation before it can inform patient selection. The cross-trial technique comparison presented here is explicitly acknowledged as confounded and is not offered as evidence of a causal relationship between embolization completeness and outcome; it is offered as evidence that the question cannot be answered without patient-level data, which does not yet exist.

Conclusion

Five randomized trials have established that MMAE reduces CSDH recurrence on average. None have explained why some patients fail. The dural vascular bed targeted by this procedure is, by extensive angiographic precedent in DAVF literature, a multi-vessel network in which the MMA is frequently not the sole contributor. We propose that systematic characterization of this anatomy, before and after embolization, is a testable next step toward identifying which patients truly benefit from MMA-directed treatment alone.

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