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MIDDLE MENINGEAL ARTERY EMBOLIZATION FOR CHRONIC SUBDURAL HEMATOMA: PATIENT SELECTION, EMBOLIC STRATEGY, PROCEDURAL TIMING, AND INTEGRATION WITH SURGICAL DRAINAGE

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Abstract

Background. Chronic subdural hematoma is increasingly prevalent among older adults and frequently recurs after burr-hole evacuation because surgery removes the collection but does not directly eliminate the vascularized inflammatory membranes that sustain exudation and repeated microhemorrhage. Middle meningeal artery embolization targets this pathological supply and may complement, defer, or replace surgery in selected patients.

Materials and methods. A structured narrative review integrated randomized trials, prospective cohorts, meta-analyses, technical studies, guidelines, and reports available through July 2026. Evidence was organized by pathophysiology, clinical phenotype, imaging architecture, operative indication, embolic agent, procedural timing, angiographic endpoint, safety, recurrence, functional recovery, and cost-effectiveness. A prospective multicenter comparative study is proposed for symptomatic chronic subdural hematoma managed surgically or nonoperatively.

Results. EMBOLISE, STEM, and EMMA-Can demonstrated lower treatment failure or symptomatic recurrence with adjunctive embolization, whereas MAGIC-MT and EMPROTECT produced neutral primary outcomes despite directionally favorable estimates or fewer serious adverse events. Divergent findings may reflect differences in recurrence definitions, embolic materials, timing, surgical pathways, patient risk, and follow-up. The proposed primary endpoint is utility-weighted treatment failure at 180 days, integrating symptomatic recurrence, rescue surgery, neurological deterioration, disabling stroke, and death. Secondary endpoints include hematoma-volume resolution, midline-shift correction, functional independence, cognition, frailty, antithrombotic resumption, radiation exposure, cranial-nerve injury, and cost.

Conclusion. Middle meningeal artery embolization is an evidence-supported adjunct for selected patients but not a uniform substitute for decompression. Standardized endpoints, anatomical safety assessment, agent-specific trials, and independent long-term validation are required before routine use across diverse neurosurgical and neurointerventional healthcare systems worldwide in practice.

Keywords: chronic subdural hematoma, middle meningeal artery embolization, burr-hole drainage, recurrence, liquid embolic agent, microparticles, neurointervention, elderly patients, antithrombotic therapy.

ЭМБОЛИЗАЦИЯ СРЕДНЕЙ МЕНИНГЕАЛЬНОЙ АРТЕРИИ ПРИ ХРОНИЧЕСКОЙ СУБДУРАЛЬНОЙ ГЕМАТОМЕ: ОТБОР ПАЦИЕНТОВ, ВЫБОР ЭМБОЛИЗИРУЮЩЕЙ СТРАТЕГИИ, СРОКИ ВМЕШАТЕЛЬСТВА И ИНТЕГРАЦИЯ С ХИРУРГИЧЕСКИМ ДРЕНИРОВАНИЕМ

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Аннотация

Актуальность. Хроническая субдуральная гематома все чаще встречается у пожилых пациентов и нередко рецидивирует после дренирования через фрезевые отверстия, поскольку операция удаляет содержимое, но непосредственно не устраняет васкуляризованные воспалительные мембраны, поддерживающие экссудацию и повторные микрокровоизлияния. Эмболизация средней менингеальной артерии воздействует на патологическое кровоснабжение и у отдельных пациентов может дополнять, отсрочивать либо заменять хирургическое лечение.

Материалы и методы. Проведен структурированный нарративный обзор рандомизированных исследований, проспективных когорт, метаанализов, технических работ, рекомендаций и публикаций, доступных до июля 2026 года. Доказательства систематизированы по патофизиологии, клиническому фенотипу, визуализационной архитектуре, показаниям к операции, эмболизирующему материалу, срокам вмешательства, ангиографической конечной точке, безопасности, рецидивам, функциональному восстановлению и экономической эффективности. Предложено проспективное многоцентровое сравнительное исследование симптомной хронической субдуральной гематомы при хирургической или консервативной тактике.

Результаты. EMBOLISE, STEM и EMMA-Can показали снижение неэффективности лечения либо симптомного рецидива при дополнительной эмболизации, тогда как MAGIC-MT и EMPROTECT имели нейтральные первичные результаты, несмотря на направленно благоприятные оценки или уменьшение серьезных нежелательных явлений. Различия могут объясняться определениями рецидива, материалами, сроками, хирургическими алгоритмами, исходным риском и продолжительностью наблюдения. Первичной конечной точкой предлагается взвешенная по клинической полезности неэффективность лечения через 180 дней, объединяющая симптомный рецидив, спасительную операцию, неврологическое ухудшение, инвалидизирующий инсульт и смерть. Вторичные показатели включают уменьшение объема гематомы, коррекцию срединного смещения, функциональную независимость, когнитивные функции, хрупкость, возобновление антитромботической терапии, лучевую нагрузку, повреждение черепных нервов и стоимость.

Заключение. Эмболизация является доказательно обоснованным дополнением у отобранных пациентов, но не универсальной заменой декомпрессии. Необходимы стандартизированные исходы, анатомическая оценка безопасности, исследования отдельных агентов и независимая длительная валидация до широкого внедрения метода в различных нейрохирургических и нейроинтервенционных системах здравоохранения мира повсеместно.

Ключевые слова: хроническая субдуральная гематома, эмболизация средней менингеальной артерии, дренирование через фрезевые отверстия, рецидив, жидкий эмболизат, микрочастицы, нейроинтервенция, пожилые пациенты, антитромботическая терапия.

SURUNKALI SUBDURAL GEMATOMADA O‘RTA MENINGEAL ARTERIYA EMBOLIZATSIYASI: BEMORLARNI TANLASH, EMBOLIZATSION STRATEGIYA, MUOLAJA MUDDATI VA JARROHLIK DRENAJI BILAN INTEGRATSIYA

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Annotatsiya

Dolzarblik. Surunkali subdural gematoma keksa bemorlarda tobora ko‘proq uchramoqda va frez teshiklari orqali drenajdan keyin tez-tez qaytalanadi, chunki operatsiya to‘plamni chiqaradi, ammo ekssudatsiya va takroriy mikrogemorragiyalarni saqlab turuvchi qon tomirlangan yallig‘lanish membranalarini bevosita bartaraf etmaydi. O‘rta meningeal arteriya embolizatsiyasi

patologik qon ta’minotini nishonga oladi va tanlangan bemorlarda jarrohlikni to‘ldirishi, kechiktirishi yoki almashtirishi mumkin.

Material va usullar. 2026-yil iyuligacha mavjud randomizatsiyalangan tadqiqotlar, prospektiv kohortlar, metatahlillar, texnik ishlar, tavsiyalar va hisobotlar asosida strukturaviy narrativ sharh bajarildi. Dalillar patofiziologiya, klinik fenotip, tasviriy arxitektura, operatsiya ko‘rsatmasi, embolizatsion material, muolaja vaqti, angiografik yakun, xavfsizlik, qaytalanish, funksional tiklanish va iqtisodiy samaradorlik bo‘yicha tizimlashtirildi. Jarrohlik yoki konservativ boshqariladigan simptomatik surunkali subdural gematoma uchun prospektiv ko‘p markazli qiyosiy tadqiqot taklif etildi.

Natijalar. EMBOLISE, STEM va EMMA-Can qo‘shimcha embolizatsiyada davolash muvaffaqiyatsizligi yoki simptomatik qaytalanish kamayganini ko‘rsatdi, MAGIC-MT va EMPROTECT esa yo‘nalish jihatidan qulay baholar yoki jiddiy nojo‘ya hodisalarning kamayishiga qaramay neytral birlamchi natijalar berdi. Tafovutlar qaytalanish ta’riflari, materiallar, vaqt, jarrohlik yo‘llari, boshlang‘ich xavf va kuzatuv davomiyligi bilan izohlanishi mumkin. Taklif etilgan birlamchi yakuniy nuqta simptomatik qaytalanish, qutqaruvchi operatsiya, nevrologik yomonlashuv, nogiron qiluvchi insult va o‘limni birlashtiruvchi 180 kunlik klinik foyda bo‘yicha vaznlantirilgan davolash muvaffaqiyatsizligidir. Ikkilamchi ko‘rsatkichlar gematoma hajmi, o‘rta chiziq siljishi, funksional mustaqillik, kognitsiya, mo‘rtlik, antitrombotik davoni qayta boshlash, nurlanish, bosh miya nervlari shikasti va xarajatni qamrab oladi.

Xulosa. O‘rta meningeal arteriya embolizatsiyasi tanlangan bemorlarda dalillarga asoslangan qo‘shimcha usuldir, ammo dekompressiyaning universal o‘rnini bosmaydi. Standart yakunlar, anatomik xavfsizlik bahosi, agentlarga xos tadqiqotlar va mustaqil uzoq muddatli validatsiya zarur turli neyroxirurgik va neyrointervensional sog‘liqni saqlash tizimlariga keng joriy etishdan oldin.

Kalit so‘zlar: surunkali subdural gematoma, o‘rta meningeal arteriya embolizatsiyasi, frez teshigi orqali drenaj, qaytalanish, suyuq embolizatsion agent, mikrozarralar, neyrointervensiya, keksa bemorlar, antitrombotik terapiya.

Introduction. Chronic subdural hematoma is an encapsulated collection of liquefied blood, fibrin-degradation products, inflammatory mediators, and cellular debris located between the dura mater and arachnoid membrane. Although minor head trauma commonly initiates the process, the clinical disorder is not simply a persistent traumatic hemorrhage. It evolves into an inflammatory and angiogenic disease characterized by formation of inner and outer neomembranes, excessive fibrinolysis, recurrent capillary leakage, and repeated microhemorrhage [6, 8, 10]. The condition predominantly affects older adults, particularly those with cerebral atrophy, frailty, recurrent falls, alcohol misuse, coagulation disorders, renal or hepatic disease, and exposure to antiplatelet or anticoagulant medication. The increasing age of the population and expanding use of antithrombotic therapy have made chronic subdural hematoma one of the fastest-growing components of contemporary cranial neurosurgical practice [8, 11, 17]. Symptoms are heterogeneous and often evolve over days or weeks. Patients may present with headache, gait instability, falls, psychomotor slowing, confusion, memory deterioration, aphasia, hemiparesis, urinary incontinence, seizure, reduced consciousness, or nonspecific functional decline. In older adults, these manifestations can be mistaken for ischemic stroke, dementia, medication toxicity, infection, normal-pressure hydrocephalus, or generalized frailty. Computed tomography usually demonstrates a crescentic extra-axial collection that may be hypodense, isodense, hyperdense, or mixed in attenuation. Septations, membranes, sedimentation levels, calcification, acute-on-chronic components, bilateral collections, and variable mass effect reflect different stages of organization and recurrent bleeding. Magnetic resonance imaging can provide additional information about membrane architecture, internal loculations, blood-product age, diffusion restriction, and alternative diagnoses.

Surgical evacuation remains the principal treatment for patients with neurological deterioration, substantial mass effect, reduced consciousness, progressive focal deficit, or a large symptomatic collection. Burr-hole evacuation with placement of a postoperative subdural or subperiosteal drain is the most widely used operation. Twist-drill drainage, craniotomy with

membranectomy, endoscopic evacuation, and bedside drainage may be selected in specific anatomical or physiological circumstances [6, 8, 9]. Surgery rapidly decompresses the cerebral hemisphere and can produce immediate neurological improvement. Nevertheless, it treats the mechanical consequence more directly than the underlying biological process. The vascularized outer membrane remains attached to the dura and may continue to exude fluid or bleed after evacuation. Residual subdural space, incomplete cerebral re-expansion, persistent inflammation, anticoagulation, bilateral disease, internal septations, and postoperative pneumocephalus can further promote recurrence [6, 9, 17]. Middle meningeal artery embolization emerged from the observation that the outer membrane is supplied predominantly by distal dural branches of the middle meningeal artery. Angiography may demonstrate enlargement of the frontal and parietal branches, diffuse membrane blush, abnormal capillary networks, and distal arteriovenous shunting. Embolization seeks to penetrate these pathological vessels and reduce repeated leakage, inflammation, and membrane viability [12, 13, 19]. The technique was initially reported for refractory or repeatedly recurrent chronic subdural hematoma. Subsequent retrospective series and meta-analyses suggested lower recurrence rates than historical surgical cohorts, stimulating rapid adoption before randomized evidence was available [12, 13, 14]. Several treatment roles have been proposed. Embolization may be performed as an adjunct before or after surgical drainage, as a stand-alone treatment in neurologically stable patients, as rescue therapy after recurrence, or prophylactically in patients considered to have a high recurrence risk. These strategies should not be considered equivalent because their clinical objectives differ.

In the adjunctive setting, surgery provides immediate decompression while embolization addresses the vascularized membrane. In the stand-alone setting, embolization depends on gradual resorption of the existing collection and therefore cannot provide immediate relief of severe mass effect. In recurrent disease, the goal is to interrupt the biological mechanism that persisted despite prior drainage. The randomized evidence published from late 2024 through 2026 has substantially changed the field. EMBOLISE, STEM, and EMMA-Can demonstrated clinically important reductions in treatment failure or symptomatic recurrence in selected populations. MAGIC-MT and EMPROTECT did not meet their primary efficacy endpoints, although the direction and safety profile of their results remained informative [1, 2, 3, 4, 5]. The discordance between trials does not support a simple conclusion that embolization is universally effective or ineffective. Trial populations, surgical pathways, embolic agents, timing, anatomical eligibility, definitions of recurrence, follow-up intervals, and composite outcomes differed. The central contemporary question is therefore which patients benefit, with which embolic material, at what stage of treatment, and at what acceptable procedural risk. The present article synthesizes the pathophysiological rationale, randomized evidence, technical considerations, safety profile, patient-selection variables, and implementation challenges of middle meningeal artery embolization for chronic subdural hematoma. It also proposes a multicenter comparative study designed to separate biological efficacy from treatment-pathway effects and to integrate recurrence prevention with neurological recovery, frailty, cognition, antithrombotic management, and cost.

Materials and Methods. A structured narrative review was conducted using randomized controlled trials, prospective interventional studies, blinded outcome studies, systematic reviews, technical investigations, surgical trials, clinical-practice recommendations, and relevant reports available through July 2026. The principal randomized evidence comprised EMBOLISE, MAGIC-MT, STEM, EMPROTECT, and EMMA-Can. Initial randomized findings from G-MEMBRANE and registered ongoing studies were considered when they addressed unresolved questions regarding timing, embolic material, stand-alone treatment, or surgical integration [1, 2, 3, 4, 5]. Evidence was categorized into pathophysiology, clinical presentation, radiological phenotype, operative urgency, recurrence risk, arterial anatomy, embolic agent, timing, angiographic endpoint, safety, functional outcome, radiological resolution, antithrombotic resumption, and economic sustainability. The clinical population included adults with symptomatic subacute or chronic subdural hematoma managed by surgical evacuation, conservative observation, or embolization. Acute traumatic subdural hematoma requiring emergency craniotomy was excluded because embolization cannot replace immediate decompression in an acutely deteriorating patient. The

proposed original study is a prospective, multicenter, randomized, stratified comparative trial. Participants would be assigned to one of two clinical strata before randomization.

The surgical stratum would include patients with neurological symptoms or radiological mass effect requiring burr-hole evacuation. Participants would be randomized to standardized surgery with drainage alone or standardized surgery plus middle meningeal artery embolization. The nonoperative stratum would include neurologically stable patients with symptomatic but nonurgent chronic subdural hematoma for whom immediate decompression is not required. These participants would be randomized to embolization plus best medical management or best medical management alone, with predefined rescue-surgery criteria. Randomization would be stratified according to unilateral versus bilateral disease, first episode versus recurrence, antithrombotic therapy, baseline frailty, hematoma architecture, and embolic material. Bilateral disease would be analyzed at the patient level, with each hematoma characterized separately. The proposed primary endpoint is utility-weighted treatment failure at 180 days. Treatment failure would include symptomatic ipsilateral recurrence, progressive hematoma requiring rescue surgery, clinically significant neurological deterioration, disabling stroke, severe procedure-related cranial neuropathy, or death. Events would receive different utility weights according to their clinical impact. Secondary endpoints would include modified Rankin Scale, Markwalder grading, Glasgow Outcome Scale–Extended, Montreal Cognitive Assessment, gait function, frailty, living independence, hematoma volume, maximum thickness, midline shift, membrane enhancement, time to radiological resolution, time to antithrombotic resumption, number of hospital days, readmission, rehabilitation utilization, radiation dose, procedure duration, and cost. All clinical outcomes would be adjudicated by a blinded independent committee. A central imaging laboratory would assess baseline architecture, postoperative residual collection, embolization penetration, radiological recurrence, new hemorrhage, ischemic injury, and serial resolution.

Results. The biological development of chronic subdural hematoma involves an interaction among mechanical separation of the dural border layer, inflammatory signaling, angiogenesis, fibrinolysis, osmotic gradients, and recurrent microhemorrhage. The outer membrane contains fibroblasts, macrophages, inflammatory cells, fragile capillaries, and vascular channels with incomplete endothelial junctions and limited smooth-muscle support [8, 10, 17]. Inflammatory mediators and angiogenic factors increase vascular permeability. Local hyperfibrinolysis prevents stable clot formation and promotes repeated bleeding into the subdural cavity. This process explains why the collection can enlarge even in the absence of substantial recurrent trauma. The distal middle meningeal artery supplies the dural surface and pathological outer membrane. Angiographic studies demonstrate diffuse membrane staining, enlargement of frontal and parietal branches, and abnormal distal vascular networks. Successful embolization should extend beyond proximal arterial occlusion and penetrate the membrane-supplying capillary bed [12, 13, 20]. Proximal coil occlusion may reduce arterial inflow but can permit reconstitution through collateral dural circulation. Distal liquid or particulate penetration may theoretically produce more effective devascularization, although greater penetration increases the importance of recognizing dangerous anastomoses. The therapeutic mechanism is delayed rather than immediately decompressive. Embolization reduces recurrent leakage and allows physiological resorption, membrane involution, cerebral re-expansion, and reduction in the subdural space. Radiological improvement may continue over several months.

Evidence from EMBOLISE. EMBOLISE evaluated patients with symptomatic subacute or chronic subdural hematoma who had an indication for surgical evacuation. Participants were randomized to surgery plus embolization with a nonadhesive liquid agent or surgery alone. Recurrence or progression leading to repeat surgery occurred in 4.1% of the adjunctive-embolization group and 11.3% of the surgery-only group, producing a relative risk of 0.36. The trial therefore showed that embolization added to surgical evacuation reduced clinically consequential recurrence requiring another operation [1, 3, 5]. The strength of EMBOLISE lies in its clinically direct endpoint. Repeat surgery represents a meaningful burden involving recurrent hospitalization, anesthesia, surgical risk, interruption of rehabilitation, and delayed resumption of antithrombotic therapy. However, the result applies most directly to patients already selected for surgery. It does

not establish that embolization alone is sufficient for patients with major mass effect or progressive neurological deterioration.

Evidence from MAGIC-MT. MAGIC-MT enrolled patients with symptomatic nonacute subdural hematoma in China. The majority underwent burr-hole drainage, while a smaller subgroup received nonsurgical management. Participants were assigned to usual care with or without embolization using a liquid material. The 90-day incidence of symptomatic recurrence or progression was similar between the groups, so the primary efficacy endpoint was neutral. Embolization was, however, associated with fewer serious adverse events [1, 2, 3]. Several aspects may have influenced this result. The trial combined surgical and nonsurgical pathways, and embolization was generally performed before burr-hole drainage in surgically managed patients. Surgical timing after embolization may limit the duration available for distal membrane devascularization before decompression alters local hemodynamics. The population, baseline recurrence risk, usual-care pathway, and event definition also differed from those of EMBOLISE. A low control-event rate can reduce statistical power even when a biological treatment effect exists.

Evidence from STEM. STEM included patients receiving either surgical or nonsurgical standard treatment. The primary efficacy outcome was a composite incorporating residual or recurrent hematoma thicker than 10 mm, rescue surgery, reoperation, major disabling stroke, myocardial infarction, or neurological death within 180 days. A primary-outcome event occurred in 16% of analyzed patients in the embolization group and 36% in the control group. The corresponding odds ratio was 0.36, indicating a substantial reduction in treatment failure without a detected increase in disabling stroke or short-term death [1, 2, 3]. The STEM endpoint captured both radiological persistence and major clinical events. This increased event sensitivity but complicates comparison with trials using symptomatic recurrence or repeat surgery alone. A residual collection larger than 10 mm does not necessarily represent clinical failure if the patient is improving and the hematoma is progressively resolving. Conversely, a smaller collection can remain clinically important when it produces focal deficit in a vulnerable patient.

Evidence from EMPROTECT. EMPROTECT studied patients who had undergone surgery for recurrent chronic subdural hematoma or for a first episode considered at high risk of recurrence. Participants were randomized to postoperative embolization with 300–500 μm trisacryl gelatin microspheres or standard medical care. Recurrence at six months occurred in 14.8% of evaluable patients in the embolization group and 21.0% in the control group. The adjusted difference did not reach statistical significance. Repeat surgery occurred in 4.3% and 8.3%, respectively, also without statistical significance [1, 4, 5]. The point estimates were directionally compatible with benefit, but the confidence intervals included no effect. The neutral result may reflect insufficient power, a smaller effect than anticipated, the use of particles rather than liquid embolic material, or differences in distal penetration. EMPROTECT highlights that the intervention should not be treated as a single uniform procedure. Agent properties, particle diameter, reflux control, microcatheter position, membrane penetration, and anatomical collateralization may modify clinical efficacy.

Evidence from EMMA-Can. EMMA-Can randomized adults with unilateral symptomatic chronic subdural hematoma undergoing surgical drainage at Canadian centers. Embolization with Onyx-18 was performed within 72 hours after drainage. Symptomatic recurrence detected on computed tomography at 90 days occurred in 4% of the embolization group and 28% of the surgery-alone group. The trial therefore provided additional evidence that postoperative liquid embolization can substantially reduce symptomatic recurrence after drainage [1, 3, 5]. The magnitude of the effect was larger than that observed in several earlier studies. This may relate to the unilateral symptomatic population, postoperative timing, liquid embolic agent, endpoint definition, or control-group event rate. The finding reinforces the need to identify whether post-drainage embolization has biological or procedural advantages over preoperative embolization. Immediate surgery decompresses the brain, while subsequent embolization is performed after stabilization and may allow more deliberate angiographic assessment.

Table 1. Principal randomized trials of middle meningeal artery embolization

Trial	Clinical pathway	Embolic strategy	Primary outcome	Main result	Interpretation

EMBOLISE	Patients requiring surgical evacuation	Adjunctive liquid embolization plus surgery	Recurrence or progression leading to repeat surgery	4.1% versus 11.3%	Reduced clinically important reoperation
MAGIC-MT	Predominantly burr-hole drainage, with nonsurgical subgroup	Liquid embolization plus usual care	Symptomatic recurrence or progression at 90 days	No significant difference	Neutral efficacy result; fewer serious adverse events
STEM	Surgical and nonsurgical standard treatment	Adjunctive liquid embolization	Composite treatment failure at 180 days	16% versus 36%	Reduced composite treatment failure
EMPROTECT	Postoperative patients at high recurrence risk	300–500 µm microparticles	Recurrence at six months	14.8% versus 21.0%; nonsignificant	Directionally favorable but neutral
EMMA-Can	Unilateral symptomatic hematoma requiring drainage	Postoperative Onyx-18 within 72 hours	Symptomatic CT-confirmed recurrence at 90 days	4% versus 28%	Strong reduction in symptomatic recurrence

Interpreting divergent trial outcomes. The randomized trials differ in several clinically important dimensions. EMBOLISE and EMMA-Can focused on adjunctive treatment after selection for surgical evacuation. STEM included both surgical and nonsurgical standard care. MAGIC-MT combined pathways in a large Chinese population. EMPROTECT specifically selected postoperative patients at increased recurrence risk but used microparticles. Definitions of failure ranged from symptomatic recurrence requiring repeat surgery to composite endpoints incorporating radiological residual thickness, rescue treatment, stroke, myocardial infarction, and death. Event rates therefore cannot be compared directly. Timing varied from preoperative to postoperative embolization. Preoperative treatment may theoretically reduce intraoperative membrane bleeding, but the immediate transition to surgery may limit the duration of ischemic membrane remodeling. Postoperative treatment separates the decompressive and biological components but exposes the patient to two procedures and may delay discharge. Embolic materials also differed. Nonadhesive liquid agents can penetrate distal branches and produce visible membrane casting. Microparticles depend on suspension, flow, particle size, and distal vascular resistance. n-Butyl cyanoacrylate is adhesive and can create durable distal occlusion but requires precise control because premature polymerization or reflux can limit penetration. The trials also enrolled different recurrence-risk populations. A patient with bilateral mixed-density hematomas, severe cerebral atrophy, anticoagulation, residual postoperative cavity, and prior recurrence has greater potential absolute benefit than a younger patient with a first homogeneous unilateral collection and rapid cerebral re-expansion.

Patient selection. Immediate surgery remains indicated when chronic subdural hematoma causes substantial neurological deterioration, declining consciousness, progressive hemiparesis, severe aphasia, significant mass effect, threatened herniation, or acute hemorrhagic expansion. Embolization should not delay decompression in these circumstances. Adjunctive embolization is most rational when the patient has a meaningful recurrence risk and can safely undergo angiography. Potential high-risk features include recurrent hematoma, bilateral disease, anticoagulant or antiplatelet requirement, persistent postoperative subdural space, advanced age, severe cerebral atrophy, mixed-density architecture, internal membranes, liver or renal disease, thrombocytopenia,

and previous treatment failure [15, 17, 18]. Stand-alone embolization may be considered for neurologically stable patients with mild or moderate symptoms, limited midline shift, high surgical or anesthetic risk, repeated recurrence, or a strong desire to avoid operative drainage. These patients require close clinical and radiological monitoring because resolution is gradual. A large but minimally symptomatic hematoma should not automatically be assigned to stand-alone embolization. Clinical deterioration can occur before resorption, particularly in patients with limited intracranial reserve. Frailty should be incorporated explicitly. A technically minor recurrence can produce major loss of independence in an older adult with marginal mobility, cognitive impairment, and limited caregiver support. Conversely, procedural burden and contrast exposure may outweigh a modest recurrence reduction in a patient with severe terminal comorbidity.

Table 2. Proposed personalized selection framework

Clinical domain	Features favoring adjunctive embolization	Features favoring surgery alone or observation
Neurological status	Stable after decompression; recurrent or high-risk disease	Emergency deterioration requiring immediate treatment
Recurrence history	Previous ipsilateral or bilateral recurrence	First low-risk episode with good cerebral re-expansion
Imaging architecture	Mixed density, membranes, bilateral collections, persistent cavity	Small homogeneous collection resolving rapidly
Antithrombotic need	Early resumption required for high thromboembolic risk	No continuing antithrombotic indication
Surgical risk	High risk of repeated anesthesia or reoperation	Low operative risk and low recurrence probability
Vascular anatomy	Safe distal MMA access without dangerous collateral supply	Prominent ophthalmic or cranial-nerve anastomoses
Expected benefit	High baseline recurrence risk and preserved life expectancy	Limited survival or minimal clinical consequence of recurrence
Follow-up capacity	Reliable serial imaging and neurological monitoring	Inability to access urgent rescue treatment

Embolic-agent selection. No embolic agent has been proven universally superior. Material choice is influenced by operator experience, vascular anatomy, microcatheter position, reimbursement, regulatory approval, and trial evidence. Nonadhesive liquid agents such as ethylene-vinyl alcohol copolymer formulations permit gradual injection and fluoroscopic visualization. The operator can observe distal penetration and proximal reflux. Limitations include cost, dimethyl-sulfoxide-compatible microcatheter requirements, injection time, radiopaque artifact, and the possibility of reflux into dangerous branches. Squid has similar nonadhesive properties and was used in STEM. Different viscosities permit adaptation to vessel caliber and desired distal penetration. Comparative clinical evidence between liquid formulations remains limited.

n-Butyl cyanoacrylate polymerizes rapidly on contact with ionic fluid. Dilution with iodized oil modifies polymerization time and radiopacity. The agent is inexpensive in some healthcare systems and can produce durable distal penetration. However, catheter adhesion, premature proximal occlusion, and uncontrolled distal migration are important technical concerns.

Microparticles can reach membrane capillaries when delivered from a sufficiently distal microcatheter position. Particle size influences penetration. Smaller particles may penetrate more distally but theoretically increase the risk of entry into occult anastomoses. Larger particles may be safer but produce less complete devascularization. Coils may be used to secure a proximal branch or protect against reflux, but coil-only embolization may not treat the distal membrane network adequately. Collateral reconstitution through accessory meningeal, superficial temporal, occipital, deep temporal, or contralateral dural branches may contribute to treatment failure. Future trials should compare embolic strategies directly rather than assuming class equivalence. Agent-specific outcomes should include distal membrane penetration, complete frontal and parietal branch treatment, reflux, collateral recruitment, radiation dose, procedure time, and recurrence.

Vascular anatomy and procedural safety. The middle meningeal artery most commonly arises from the internal maxillary artery and enters the cranial cavity through the foramen spinosum. It divides into frontal and parietal branches that course within the dura. The orbital or recurrent meningeal branch may communicate with the lacrimal or ophthalmic circulation. Embolization through an unrecognized anastomosis can cause retinal ischemia and visual loss. Connections with petrosal branches supplying the facial nerve, cavernous branches supplying cranial nerves, and branches related to the internal carotid or vertebral circulation must also be excluded [19, 20, 21]. Digital-subtraction angiography should evaluate external carotid, middle meningeal, accessory meningeal, and relevant internal carotid anatomy. Selective microcatheter angiography may reveal dangerous connections not evident on proximal injection. The microcatheter should be advanced beyond branches associated with the orbit, facial nerve, or skull-base cranial nerves whenever possible. Embolic injection should be stopped when reflux approaches a hazardous branch, resistance increases, or unexpected collateral filling appears. Three-dimensional rotational angiography may be useful in complex anatomy or treatment failure. Recent anatomical reports indicate that deep or middle temporal arterial supply can maintain membrane vascularization when the middle meningeal artery is incompletely treated, emphasizing the importance of collateral assessment. Potential complications include ischemic stroke, retinal artery occlusion, cranial-nerve palsy, skin or scalp necrosis, arterial dissection, vessel perforation, intracranial hemorrhage, contrast-induced kidney injury, access-site hematoma, pseudoaneurysm, and allergic reaction.

Although serious complications are uncommon in experienced centers, low event rates require large registries and long-term surveillance. A procedure performed prophylactically must meet a particularly high safety threshold because many control patients would not otherwise recur.

Timing relative to surgery. Preoperative embolization may be performed during the same hospitalization before burr-hole drainage. Theoretical advantages include early interruption of membrane supply and a single coordinated episode of care. Disadvantages include potential delay of decompression and uncertainty about whether immediate surgery alters the embolization effect. Postoperative embolization permits urgent drainage first. It may be performed during the same anesthesia session, within 24–72 hours, or later during the admission. EMMA-Can supports postoperative embolization within 72 hours, while EMBOLISE also provides evidence for integration with surgical treatment [1, 3, 5]. Single-session hybrid treatment can reduce repeated transfer and anesthesia. A hybrid operating room permits angiography and burr-hole evacuation during the same episode. However, workflow complexity, contrast administration, radiation exposure, sterile-field transitions, and staffing must be considered. Staged treatment may be preferable in medically fragile patients, allowing neurological recovery and reassessment before embolization. The risk is that discharge or clinical instability may prevent completion of the second procedure. Optimal timing remains uncertain, and observational evidence suggests that baseline hematoma volume and treatment sequence may influence radiological resolution. This question requires prospective randomization rather than retrospective comparison.

Radiological response. Reduction in hematoma thickness is gradual after embolization. Early computed tomography may show little change despite successful membrane devascularization. This should not be classified automatically as treatment failure if the patient remains neurologically stable. Radiological follow-up should record volume, maximum thickness, midline shift, density, internal membranes, acute components, cerebral re-expansion, and contralateral disease. Volumetric analysis is preferable to a single linear measurement because irregular bilateral collections are not adequately represented by maximum width. Treatment failure should prioritize symptoms and rescue intervention rather than residual imaging alone. A persistent 11-mm collection at 180 days may be clinically inconsequential if it is progressively shrinking and the patient is independent. In contrast, an 8-mm collection associated with new aphasia or falls may be clinically significant. The EMBOLISE imaging analysis indicates that serial changes in volume, thickness, and midline shift can provide a more complete description of treatment response than recurrence alone.

Antithrombotic therapy. Chronic subdural hematoma frequently occurs in patients receiving anticoagulation for atrial fibrillation, mechanical heart valves, venous thromboembolism,

or cardiac devices, and antiplatelet therapy for coronary or cerebrovascular disease. Interrupting antithrombotic medication reduces hemorrhagic risk but increases the probability of ischemic stroke, myocardial infarction, valve thrombosis, or recurrent venous thromboembolism. The decision to resume therapy is therefore a competing-risk problem. Embolization may theoretically permit earlier resumption by reducing membrane vascularity. This is clinically attractive but not yet established through adequately powered randomized trials. Resumption timing should be individualized according to thromboembolic indication, surgical hemostasis, residual hematoma, renal function, fall risk, and follow-up imaging. Future studies should treat time to safe antithrombotic resumption as a major endpoint rather than a secondary descriptive variable. Avoiding recurrent hematoma while delaying necessary anticoagulation for several months may not produce a favorable net clinical outcome.

Functional and cognitive outcomes. Most embolization trials emphasize recurrence, reoperation, or radiological progression. These outcomes are important but do not capture the full burden of chronic subdural hematoma. Older patients may experience persistent gait dysfunction, impaired attention, executive slowing, reduced confidence, recurrent falls, loss of driving ability, and inability to return to independent living even after radiological resolution. A recurrence prevented on computed tomography may not translate into improved quality of life if frailty and cognitive impairment remain unaddressed. Assessment should include modified Rankin Scale, functional independence, gait speed, falls, Montreal Cognitive Assessment, delirium, depression, frailty, living location, caregiver burden, and return to baseline activity. Rehabilitation and geriatric co-management are important components of treatment. Embolization should be integrated into a pathway that includes medication review, fall prevention, nutritional assessment, cognitive screening, and safe antithrombotic management.

Table 3. Proposed multicenter comparative trial

Component	Proposed specification
Population	Adults with symptomatic chronic subdural hematoma
Clinical strata	Surgical decompression required versus neurologically stable nonoperative management
Surgical intervention	Standardized burr-hole drainage with protocolized drain placement
Endovascular intervention	Bilateral or unilateral MMA embolization according to hematoma distribution
Comparator	Identical standard treatment without embolization
Primary endpoint	Utility-weighted treatment failure at 180 days
Clinical failure components	Symptomatic recurrence, rescue surgery, deterioration, disabling stroke, cranial neuropathy, death
Radiological endpoints	Volume, thickness, midline shift, density, membranes, time to resolution
Functional endpoints	mRS, cognition, gait, frailty, independence, living location
Medication endpoint	Time to clinically appropriate antithrombotic resumption
Procedural endpoints	Agent, penetration, reflux, radiation, contrast, duration, angiographic complications
Economic endpoints	Hospital days, readmissions, repeat surgery, rehabilitation, total treatment cost
Blinding	Independent blinded clinical and imaging adjudication
Originality	Simultaneous evaluation of pathway, timing, agent, biological response, function, and cost

Discussion. Middle meningeal artery embolization has moved rapidly from salvage therapy for recurrent chronic subdural hematoma to a procedure supported by multiple randomized trials. The evidence is strongest for its use as an adjunct to surgical drainage in selected patients. EMBOLISE demonstrated a reduction in recurrence or progression requiring repeat surgery. STEM demonstrated a reduction in a broader treatment-failure composite. EMMA-Can showed a large decrease in symptomatic recurrence after postoperative embolization. These findings establish that

the biological membrane can be treated in a manner that changes clinically relevant outcomes [1, 3, 5]. MAGIC-MT and EMPROTECT provide an equally important caution. Not every trial, population, material, or pathway produces statistically significant benefit. The procedure should therefore not be expanded indiscriminately to all patients with chronic subdural hematoma [2, 4, 5]. The evidence favors a patient-specific rather than disease-wide approach. The absolute benefit of embolization depends on baseline recurrence risk. A reduction from 28% to 4%, as observed in EMMA-Can, has major clinical value. A reduction from a very low baseline risk may not justify additional arterial catheterization, radiation, contrast, cost, and rare neurological complications. Risk prediction models could improve selection, but most available recurrence models have limited external validation. Variables such as age, bilateral disease, antithrombotic exposure, mixed density, internal membranes, cerebral atrophy, postoperative residual space, and previous recurrence are plausible but interact in complex ways.

Artificial-intelligence volumetry and membrane characterization may eventually improve prediction. Automated three-dimensional analysis can quantify hematoma volume, local brain deformation, subdural air, and cerebral re-expansion more accurately than a single linear measurement. Biological biomarkers may also be useful. Inflammatory mediators, angiogenic proteins, fibrinolytic markers, and membrane vascularity could identify patients in whom the middle meningeal supply remains particularly active. Such approaches remain investigational. The optimal embolic agent is unresolved. The positive liquid-agent trials and neutral particle trial may suggest greater effectiveness from distal liquid penetration, but cross-trial comparison cannot establish causality. Patient populations and endpoint definitions differed. A randomized head-to-head agent trial should standardize clinical indication, timing, microcatheter position, and angiographic endpoint. It should compare liquid agents, particles, and possibly n-butyl cyanoacrylate, while monitoring visual, cranial-nerve, ischemic, and skin complications.

The angiographic endpoint also requires standardization. Proximal disappearance of the middle meningeal artery is not equivalent to distal membrane devascularization. A procedural grading system could include penetration of frontal and parietal branches, reduction of membrane blush, collateral supply, reflux, and residual hazardous branches. Bilateral disease presents another uncertainty. Embolization may be performed bilaterally when collections are bilateral or when contralateral membrane supply contributes to recurrence. Bilateral treatment increases procedural time and contrast exposure but may reduce contralateral progression. The presence of acute blood within a chronic collection requires careful interpretation. Embolization does not treat a major active arterial subdural hemorrhage requiring urgent surgery. It may still be useful after stabilization when a chronic vascularized membrane underlies the acute-on-chronic event. Internal septations can limit the effectiveness of simple burr-hole drainage. Craniotomy, endoscopic membranectomy, or multiple drainage trajectories may be necessary for immediate decompression. Embolization can be considered an adjunct but should not replace adequate mechanical evacuation. Stand-alone embolization is appealing for frail or anticoagulated patients, but its delayed mechanism must be respected. A patient with severe mass effect may worsen while awaiting resorption. Randomized evidence for stand-alone treatment remains less mature than evidence for adjunctive treatment. Clinical pathways should define rescue criteria explicitly. Increasing headache, new focal deficit, reduced consciousness, progressive midline shift, or hematoma expansion should trigger urgent reassessment and possible surgical evacuation.

Follow-up schedules should be individualized. Routine computed tomography at approximately one month and three months may be reasonable, with earlier imaging for neurological change. Excessive surveillance increases radiation and cost without necessarily changing management. The role of contrast-enhanced MRI requires further study. Membrane enhancement and vascularity may provide biomarkers of biological activity and treatment response, but MRI is less practical than CT for routine follow-up in many older patients. Cost-effectiveness remains uncertain. Embolization adds an expensive procedure, device, angiography-suite time, contrast, and professional resources. These costs may be offset by fewer repeat surgeries, readmissions, rehabilitation interruptions, and delayed antithrombotic therapy. Universal embolization is unlikely to be economically efficient in all healthcare systems. Selective use in patients with high recurrence

risk is more likely to produce favorable value. Current economic analyses suggest that cost reduction and improved selection are central to sustainable implementation [22, 23, 24]. Workforce and institutional organization will influence adoption. The procedure may be performed by endovascular neurosurgeons, interventional neuroradiologists, or appropriately trained neurointerventional specialists. Multidisciplinary protocols should define referral, timing, anatomical review, and postoperative responsibility. Low-volume centers may lack immediate embolization access. Transfer solely for prophylactic intervention should be justified by expected absolute benefit. Regional pathways and delayed outpatient embolization may be alternatives for stable patients.

The procedure's rapid growth also raises the possibility of indication creep. Patients with small asymptomatic collections or low recurrence risk may be offered treatment because the technique is minimally invasive. Randomized evidence should define practice rather than enthusiasm, reimbursement, or device availability. Shared decision-making is essential. Patients and families should understand that embolization reduces recurrence risk but does not guarantee immediate hematoma resolution, eliminate the possibility of surgery, or restore neurological function independently of rehabilitation. Consent should include rare but serious risks such as stroke, visual loss, cranial-nerve injury, hemorrhage, arterial damage, contrast reaction, and kidney injury. The possibility of subsequent rescue surgery must be discussed. The proposed study addresses limitations of existing trials by separating surgical and nonsurgical populations, standardizing recurrence definitions, incorporating functional and cognitive outcomes, recording embolic-agent performance, and measuring antithrombotic resumption. The utility-weighted primary endpoint avoids treating all failures as equivalent. A radiological residual collection, repeat burr-hole procedure, disabling stroke, and death should not contribute equally to a composite.

Central imaging adjudication is necessary because local decisions to reoperate can vary according to surgeon preference, hospital capacity, and patient frailty. Blinded review can distinguish true radiological recurrence from slow expected resolution. Longer follow-up is also required. Most current trials report 90- or 180-day outcomes. Delayed recurrence, contralateral hematoma, antithrombotic complications, cranial neuropathy, and functional recovery may emerge over one year. Several limitations of this review should be acknowledged. The randomized trials used different clinical definitions, materials, surgical techniques, and outcome measures. Direct comparisons across studies are therefore hypothesis-generating rather than definitive. Some trials were industry supported, while others were academically funded. Funding source does not determine validity, but transparent disclosure, independent adjudication, and replication remain important. The available evidence is dominated by specialized centers with experienced operators. Outcomes may differ during broad implementation in lower-volume hospitals. Despite these limitations, the evidence has crossed an important threshold. Middle meningeal artery embolization can no longer be regarded solely as an experimental rescue procedure. It is an evidence-supported adjunct that should be incorporated selectively into multidisciplinary chronic subdural hematoma care.

Conclusion. Middle meningeal artery embolization targets the vascularized inflammatory membrane that drives persistence and recurrence of chronic subdural hematoma. This mechanism complements surgical drainage, which rapidly removes the collection but does not directly eliminate its pathological blood supply. Randomized trials demonstrate that adjunctive embolization can reduce repeat surgery, symptomatic recurrence, radiological persistence, or composite treatment failure in selected populations. EMBOLISE, STEM, and EMMA-Can provide the strongest positive evidence, whereas MAGIC-MT and EMPROTECT emphasize heterogeneity across pathways and embolic strategies. The procedure should not delay emergency decompression in patients with neurological deterioration or major mass effect. Its clearest current role is as an adjunct to surgical drainage in patients with meaningful recurrence risk and safe vascular anatomy. Stand-alone embolization remains a potential option for neurologically stable patients who do not require immediate decompression, but evidence is less definitive and careful rescue planning is necessary. Future research should standardize recurrence definitions, compare embolic agents directly, determine optimal preoperative or postoperative timing, quantify distal membrane penetration, and evaluate antithrombotic resumption, cognition, frailty, independence, cost, and long-term safety.

Middle meningeal artery embolization should be viewed neither as a universal replacement for burr-hole drainage nor as a niche experimental technique. It represents a pathophysiologically targeted component of personalized chronic subdural hematoma management.

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