



MIDDLE MENINGEAL ARTERY EMBOLIZATION FOR CHRONIC SUBDURAL HEMATOMA AFTER RANDOMIZED TRIALS: MEMBRANE-TARGETED THERAPY, RECURRENCE-RISK STRATIFICATION, AND INTEGRATED NEUROSURGICAL-ENDOVASCULAR CARE

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Abstract

Background. Chronic subdural hematoma is increasingly understood as an angiogenic inflammatory disease of the dural border-cell compartment rather than a passive residual collection. Surgical drainage relieves mass effect rapidly but does not directly interrupt vascularized membrane biology, creating a rationale for middle meningeal artery embolization.

Materials and methods. Randomized trials, consensus statements, prospective cohorts, anatomical studies, and technical investigations published through July 2026 were synthesized. Evidence was organized according to urgency, hematoma phenotype, recurrence risk, surgical strategy, embolic agent, distal penetration, dangerous anastomoses, antithrombotic requirements, radiological resolution, and functional outcome.

Results. EMBOLISE reduced recurrence or progression requiring reoperation after surgery from 11.3% to 4.1%. STEM reduced a broader 180-day treatment-failure composite from 36% to 16% across surgical and nonsurgical strata. MAGIC-MT and EMPROTECT did not meet their primary efficacy endpoints, demonstrating that endpoint definition, population, timing, and embolic strategy materially affect trial results. MEMBRANE reported a reduction in six-month residual or recurrent hematoma or surgery from 22.1% to 11.6% with n-butyl cyanoacrylate. MMA embolization acts gradually and should not replace urgent decompression in patients with severe mass effect or neurological deterioration.

Conclusion. The strongest evidence supports adjunctive embolization in selected nonemergent patients at risk of recurrence. The proposed framework integrates membrane activity, expansion risk, morphology, brain re-expansion, recurrence predictors, arterial anatomy, neurological urgency, embolic strategy, and follow-up to distinguish patients requiring immediate drainage, combined treatment, or embolization-centered management.

Keywords: chronic subdural hematoma; middle meningeal artery embolization; burr-hole drainage; hematoma recurrence; n-butyl cyanoacrylate; ethylene-vinyl alcohol copolymer; neomembrane; endovascular neurosurgery; antithrombotic therapy; patient selection.

ЭМБОЛИЗАЦИЯ СРЕДНЕЙ МЕНИНГЕАЛЬНОЙ АРТЕРИИ ПРИ ХРОНИЧЕСКОЙ СУБДУРАЛЬНОЙ ГЕМАТОМЕ ПОСЛЕ РАНДОМИЗИРОВАННЫХ ИССЛЕДОВАНИЙ: ТЕРАПИЯ, НАПРАВЛЕННАЯ НА МЕМБРАНУ, СТРАТИФИКАЦИЯ РИСКА РЕЦИДИВА И ИНТЕГРИРОВАННАЯ НЕЙРОХИРУРГИЧЕСКАЯ-ЭНДОВАСКУЛЯРНАЯ ПОМОЩЬ

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Аннотация

Актуальность. Хроническая субдуральная гематома всё чаще рассматривается как ангиогенно-воспалительное заболевание пограничного клеточного слоя твёрдой мозговой оболочки, а не как пассивное остаточное скопление крови. Хирургическое дренирование быстро устраняет масс-эффект, но непосредственно не прекращает патологию васкуляризованной мембраны, что обосновывает эмболизацию средней менингеальной артерии.

Материалы и методы. Синтезированы рандомизированные исследования, консенсусные документы, проспективные когорты, анатомические и технические работы, опубликованные до июля 2026 года. Данные систематизированы по срочности, фенотипу гематомы, риску рецидива, хирургической стратегии, эмболизирующему агенту, дистальному проникновению, опасным анастомозам, антитромботической терапии, рентгенологическому разрешению и функциональному исходу.

Результаты. EMBOLISE снизило частоту рецидива или прогрессирования, потребовавших повторной операции, с 11,3% до 4,1%. STEM уменьшило более широкий комбинированный показатель неудачи лечения за 180 дней с 36% до 16% в хирургической и нехирургической стратах. MAGIC-MT и EMPROTECT не достигли первичных конечных точек, показывая влияние определения исхода, популяции, сроков и техники. MEMBRANE снизило шестимесячную частоту остаточной или повторной гематомы либо операции с 22,1% до 11,6% при применении n-бутилцианоакрилата. Эмболизация действует постепенно и не заменяет срочную декомпрессию при выраженном масс-эффекте или ухудшении.

Заключение. Наиболее убедительные данные поддерживают дополнительную эмболизацию у отобранных неэкстренных пациентов с риском рецидива. Модель объединяет активность мембраны, риск увеличения, морфологию, расправление мозга, предикторы рецидива, артериальную анатомию, неврологическую срочность, эмболизационную стратегию и наблюдение. Она предназначена для различения пациентов, которым необходимы немедленное дренирование, хирургия с последующей эмболизацией, первичная эндоваскулярная стратегия или наблюдение с последовательной компьютерно-томографической оценкой толщины, объёма, смещения срединных структур и клинической динамики во времени.

Ключевые слова: хроническая субдуральная гематома; эмболизация средней менингеальной артерии; дренирование через фрезевые отверстия; рецидив гематомы; n-бутилцианоакрилат; этиленвинилспиртовой сополимер; неомембрана; эндоваскулярная нейрохирургия; антитромботическая терапия; отбор пациентов.

RANDOMIZATSIYALANGAN TADQIQOTLARDAN KEYIN SURUNKALI SUBDURAL GEMATOMADA O'RTA MENINGEAL ARTERIYANI EMBOLIZATSIYA QILISH: MEMBRANAGA YO'NALTIRILGAN DAVOLASH, RETSIDIV XAVFINI STRATIFIKATSIYA QILISH VA INTEGRATSIYALASHGAN NEYROXIRURGIK-ENDOVASKULYAR YORDAM

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Annotatsiya

Dolzarblik. Surunkali subdural gematoma tobora ko'proq qattiq miya pardasining chegaraviy hujayra qatlamiga oid angiogen va yallig'lanish kasalligi sifatida qaralmoqda, oddiy qoldiq qon to'plami sifatida emas. Jarrohlik drenaji massa ta'sirini tez kamaytiradi, biroq tomirlangan membrana patologiyasini to'g'ridan-to'g'ri to'xtatmaydi, bu o'rta meningeal arteriyani embolizatsiya qilish uchun asos yaratadi.

Material va usullar. 2026-yil iyuligacha chop etilgan randomizatsiyalangan sinovlar, konsensus hujjatlari, prospektiv kohortalar, anatomik va texnik tadqiqotlar sintez qilindi. Dalillar shoshilinchlik, gematoma fenotipi, retsidiv xavfi, jarrohlik strategiyasi, embolik modda, distal

penetratsiya, xavfli anastomozlar, antitrombotik ehtiyoj, radiologik rezolyutsiya va funksional natija bo'yicha tizimlashtirildi.

Natijalar. EMBOLISE operatsiyadan keyin qayta operatsiyani talab qilgan retsidiv yoki progressiyani 11,3% dan 4,1% gacha kamaytirdi. STEM jarrohlik va nojarrohlik qatlamlarida 180 kunlik davolash muvaffaqiyatsizligi ko'rsatkichini 36% dan 16% gacha tushirdi. MAGIC-MT va EMPROTECT asosiy samaradorlik yakunlariga erishmadi, bu natija ta'rifi, populyatsiya, vaqt va texnikaning ahamiyatini ko'rsatdi. MEMBRANE n-butyl-sianoakrilat bilan olti oylik qoldiq yoki qaytalangan gematoma yoxud operatsiya ko'rsatkichini 22,1% dan 11,6% gacha kamaytirdi. Embolizatsiya asta ta'sir qiladi va og'ir massa ta'siri yoki nevrologik yomonlashishda shoshilinch dekompressiyani almashtirmaydi.

Xulosa. Eng kuchli dalillar retsidiv xavfi mavjud tanlangan, favqulodda bo'lmagan bemorlarda qo'shimcha embolizatsiyani qo'llab-quvvatlaydi. Model membrana faolligi, kengayish xavfi, morfologiya, miya qayta yozilishi, retsidiv prediktorlari, arterial anatomiya, nevrologik shoshilinchlik, embolizatsiya strategiyasi va kuzatuvni birlashtiradi. U bemorlarni zudlik bilan drenaj qilish, jarrohlikdan keyingi embolizatsiya, birlamchi endovaskulyar davolash yoki gematoma qalinligi, hajmi, o'rta chiziqli siljishi va klinik dinamikani ketma-ket KT bilan kuzatish guruhlariga ajratishga xizmat qiladi.

Kalit so'zlar: surunkali subdural gematoma; o'rta meningeal arteriya embolizatsiyasi; freza teshiklari orqali drenaj; gematoma retsidivi; n-butyl-sianoakrilat; etilen-vinil-spirt kopolimeri; neomembrana; endovaskulyar neyroxirurgiya; antitrombotik davolash; bemor tanlash.

Introduction. Chronic subdural hematoma is among the most common neurosurgical disorders in older adults. Its incidence is increasing because populations are aging, falls remain common, and a growing proportion of patients receive anticoagulant or antiplatelet therapy for atrial fibrillation, venous thromboembolism, coronary artery disease, prosthetic valves, and cerebrovascular disease. Patients may present with headache, gait instability, cognitive decline, focal weakness, dysphasia, seizures, urinary dysfunction, somnolence, or progressive loss of independence [1, 2, 3]. The term chronic subdural hematoma can create the misleading impression of an inert liquefied blood collection persisting after trauma. Contemporary pathological and imaging evidence instead supports an active disease process involving disruption of the dural border-cell layer, inflammatory organization, fibrinolysis, angiogenesis, formation of internal and external neomembranes, and repeated microhemorrhage from fragile capillaries [2, 3, 18]. Minor trauma or spontaneous tearing of bridging veins may initiate the subdural collection. Blood products activate inflammation within the dural border-cell compartment. Fibroblasts, inflammatory cells, macrophages, collagen, fibrin, and newly formed capillaries create a vascularized outer membrane attached to the dura. The inner membrane develops adjacent to the arachnoid and is generally less vascular [7, 14, 19]. The neocapillaries of the outer membrane are structurally immature. They contain thin walls, wide interendothelial gaps, incomplete basement membrane, and limited smooth-muscle support. Recurrent plasma leakage and microbleeding enlarge the hematoma. Local hyperfibrinolysis prevents stable clot organization, while inflammatory mediators increase vascular permeability and perpetuate the cycle [5, 11, 16, 20]. Surgical evacuation through one or two burr holes remains an effective method of rapidly relieving mass effect. The liquefied hematoma is irrigated or drained, and a subdural or subperiosteal drain reduces early recurrence. Surgery, however, does not directly eliminate the vascular supply of the outer membrane. Residual membranes may continue to exude fluid or bleed after apparently successful decompression [2, 3, 10, 18]. Recurrence after surgery is clinically important because affected patients are frequently elderly, frail, anticoagulated, and vulnerable to anesthesia, infection, seizures, delirium, immobilization, and repeated hospitalization. A second operation also exposes the patient to additional interruption of antithrombotic therapy and delays neurological rehabilitation. Middle meningeal artery embolization was introduced as a membrane-directed treatment. The MMA supplies most of the dura over the cerebral convexity and contributes to the pathological vascular network of the external membrane. Endovascular occlusion of its distal branches reduces blood flow to the membrane, promotes vessel regression, limits recurrent exudation, and permits gradual

resorption of the subdural collection [2, 6, 18, 27]. The mechanism differs fundamentally from surgical drainage. Surgery treats the intracranial mass effect immediately. Embolization treats the biological source of persistence and recurrence but usually requires weeks or months for substantial radiological resolution. The two procedures are therefore complementary rather than interchangeable [1, 3, 10, 13]. These findings encouraged rapid clinical adoption but were vulnerable to selection bias, nonstandardized definitions, variable imaging follow-up, differences in embolic agents, and preferential treatment of stable patients [6, 13, 28].

Between 2024 and 2026, several major randomized trials transformed the evidence base. EMBOLISE evaluated adjunctive MMA embolization in patients already selected for surgical evacuation. MAGIC-MT enrolled patients assigned to surgical or nonsurgical management. STEM similarly incorporated both standard-treatment strata and used a broad composite definition of treatment failure. EMPROTECT studied postoperative embolization in patients at high risk of recurrence. MEMBRANE evaluated n-butyl cyanoacrylate with either surgical or nonsurgical standard treatment [4, 7, 14, 19]. EMBOLISE showed that recurrence or progression requiring reoperation within 90 days occurred in 4.1% of patients treated with surgery plus embolization and 11.3% treated with surgery alone. STEM reported a 180-day primary treatment-failure outcome in 16% of the embolization group and 36% of controls. By contrast, MAGIC-MT and EMPROTECT produced directionally favorable but statistically nonsignificant primary efficacy results. MEMBRANE subsequently reported a reduction from 22.1% to 11.6% in its six-month composite endpoint. These trials establish that MMA embolization can reduce treatment failure in selected populations, but they do not justify universal treatment of every chronic subdural hematoma. Differences among trials emphasize that clinical urgency, surgical indication, hematoma morphology, endpoint definition, embolic material, distal penetration, recurrence risk, and follow-up duration modify the observed effect.

Materials and Methods. This manuscript was developed as a structured integrative review with an original clinical and technical synthesis. Randomized controlled trials, contemporary consensus statements, prospective cohorts, anatomical studies, embolic-agent comparisons, imaging investigations, and clinically relevant technical series published through July 2026 were evaluated. The principal evidence base included EMBOLISE, MAGIC-MT, STEM, EMPROTECT, MEMBRANE, earlier randomized and prospective studies of postoperative embolization, surgical-drain trials, dexamethasone trials, atorvastatin studies, and major observational MMA embolization cohorts [6, 11, 12, 20]. Evidence was classified into nine domains.

The first was neurological urgency, including consciousness, focal deficit, seizures, progressive deterioration, and signs of cerebral herniation. The second was hematoma phenotype, including thickness, volume, midline shift, density, septations, bilateral disease, acute components, and brain expansion. The third domain was recurrence risk, including advanced age, anticoagulation, antiplatelet therapy, bilateral hematoma, large residual cavity, internal architecture, previous recurrence, liver disease, thrombocytopenia, and cerebral atrophy. The fourth was treatment strategy, including urgent surgical drainage, elective drainage with adjunctive embolization, embolization as primary therapy, and observation. The fifth domain was endovascular anatomy, including MMA origin, frontal and parietal branches, recurrent meningeal–ophthalmic connections, petrosal branches, facial-nerve supply, anastomoses with the internal carotid artery, occipital artery, ascending pharyngeal artery, and contralateral dural circulation. The sixth domain was embolic technology, including polyvinyl-alcohol particles, calibrated microspheres, n-BCA, ethylene-vinyl alcohol copolymers, PHIL, coils, and combination strategies. The seventh was procedural performance, including distal penetration, branch coverage, angiographic stasis, radiation, contrast, access route, and complications. The eighth domain was longitudinal outcome, including recurrence, progression, rescue surgery, hematoma thickness, volume reduction, complete resolution, functional outcome, cognition, hospitalization, and mortality. The ninth was systems implementation, including timing relative to surgery, antithrombotic management, outpatient follow-up, cost, endovascular capacity, and multidisciplinary workflow. No original patient cohort was generated and no independent quantitative meta-analysis was conducted. Numerical results were interpreted according to each trial’s population, embolic agent, endpoint, follow-up duration,

and statistical design. The tables represent an original synthesis and contain no fabricated patient-level data.

Results. The dura and arachnoid are normally connected by a thin layer of flattened border cells with limited extracellular collagen and weak intercellular adhesion. Trauma, intracranial hypotension, cerebral atrophy, or shearing can separate this layer and create a potential subdural space [10, 13, 23]. The initial collection triggers deposition of fibrin and recruitment of inflammatory cells. The outer membrane develops adjacent to the dura and becomes progressively vascularized. Histological studies demonstrate sinusoidal capillaries with fragile endothelial junctions, inflammatory infiltration, hemosiderin-laden macrophages, fibroblasts, collagen fibers, and evidence of recurrent hemorrhage [5, 11, 16, 20]. Vascular endothelial growth factor, angiopoietins, interleukins, matrix metalloproteinases, tissue plasminogen activator, thrombomodulin, and other inflammatory and fibrinolytic mediators are elevated within the hematoma fluid and membrane. These factors promote angiogenesis, vascular permeability, matrix remodeling, and failure of stable coagulation [4–8]. The MMA does not directly supply the subdural fluid. It supplies the dura and external neomembrane. Embolization reduces arterial inflow to the capillary bed, producing ischemic regression and reduced exudative activity. The subsequent reduction in hematoma volume depends on resorption, restoration of pressure balance, and gradual brain re-expansion. The biological response is therefore delayed. A patient with declining consciousness, severe hemiparesis, marked midline shift, or herniation physiology requires immediate decompression. Embolization cannot remove the mass quickly enough to substitute for surgery [2, 6, 18, 27].

Standard Surgical Treatment. Burr-hole evacuation with closed-system drainage remains the standard rapid treatment for symptomatic cSDH producing clinically meaningful mass effect. One or two burr holes may be used. The hematoma is irrigated or allowed to drain, and a subdural or subperiosteal drain is maintained for a defined period. Drain placement reduces recurrence compared with evacuation without drainage. Subperiosteal or subgaleal drains may decrease the risk of cortical injury while providing recurrence outcomes comparable with subdural drains in selected settings [22–26]. Craniotomy is reserved for organized, calcified, multiloculated, recurrent, or acute-on-chronic hematomas that cannot be treated adequately through burr holes. Membranectomy may be required when thick internal membranes prevent brain expansion, but extensive membrane removal increases the risk of cortical and venous injury. Twist-drill drainage and subdural evacuating-port systems can be used under local anesthesia in selected patients. They reduce operative burden but may be insufficient for thick, septated, or partially organized collections. Surgery treats mass effect immediately but recurrence remains possible. Predictors include bilateral disease, large residual thickness, poor brain re-expansion, mixed-density or hyperdense components, persistent midline shift, antithrombotic therapy, advanced atrophy, and previous recurrence [7–11].

Randomized Evidence. EMBOLISE enrolled patients with symptomatic subacute or chronic subdural hematoma who had already been selected for surgical evacuation. Patients were randomized to surgery plus Onyx MMA embolization or surgery alone. Recurrence or progression resulting in repeat surgery within 90 days occurred in 4.1% of the combined-treatment group and 11.3% of the surgery-only group. The relative risk was 0.36. Functional deterioration occurred in 11.9% and 9.8%, respectively, demonstrating that reduced reoperation did not automatically translate into a superior short-term functional endpoint [16–18]. The trial provides strong evidence for adjunctive treatment in surgical candidates but does not establish stand-alone efficacy. Patients requiring emergency decompression were not treated with embolization instead of surgery.

MAGIC-MT was conducted in China and enrolled patients with symptomatic nonacute subdural hematoma and mass effect. The treating surgeon selected burr-hole drainage or nonsurgical management before randomization to MMA embolization with liquid embolic material or usual care. Approximately 78% underwent burr-hole drainage. Symptomatic recurrence or progression within 90 days occurred in 6.7% of the embolization group and 9.9% of controls, a difference that did not reach statistical significance. Serious adverse events were less frequent with embolization, occurring in 6.7% versus 11.6% [17–20]. The neutral primary result may reflect a lower control-

event rate, heterogeneous surgical and nonsurgical populations, early follow-up, exclusion of patients requiring craniotomy, procedural factors, or insufficient statistical separation.

STEM randomized 310 patients to standard treatment with or without embolization using Squid. Surgical or nonsurgical management was selected before randomization. The primary composite treatment-failure endpoint at 180 days included residual or recurrent hematoma, reoperation or rescue surgery, and major neurological events. It occurred in 16% of the embolization group and 36% of controls, corresponding to an odds ratio of 0.36. Thirty-day death or major disabling stroke occurred in approximately 3% of both groups [18–21]. The broad endpoint captures radiological and clinical failure but makes direct comparison with EMBOLISE difficult because EMBOLISE focused on repeat surgery.

EMPROTECT enrolled 342 patients who underwent surgery for a first high-risk or recurrent cSDH. Participants were randomized to postoperative embolization with calibrated microparticles or standard postoperative care. Six-month recurrence occurred in 14.8% of the embolization group and 21.0% of controls. The adjusted odds ratio was 0.64, but the difference was not statistically significant. Repeat surgery was required in 4.3% and 8.3%, respectively [19–22]. The direction of effect was favorable, but the study did not meet its primary endpoint. Potential explanations include particle penetration, selected high-risk population, sample size, control-event rate, and differences between radiological recurrence and clinically important reoperation.

MEMBRANE randomized 376 patients with symptomatic cSDH to n-BCA MMA embolization plus standard care or standard care alone. Surgical or nonsurgical treatment was determined before randomization. The six-month primary effectiveness endpoint consisted of residual or recurrent hematoma greater than 10 mm or a required surgical procedure. Events occurred in 11.6% of evaluable participants receiving embolization plus standard care and 22.1% receiving standard care alone. The common odds ratio was 0.53. Surgery during follow-up was required in 3.8% and 10.0%, respectively [20–22]. The safety profile was broadly comparable. A transient facial-nerve palsy was adjudicated as device related. The trial strengthens evidence for liquid embolization but used a one-sided primary hypothesis test and had missing primary-endpoint data, which should be considered during interpretation.

Table 1. Major Randomized Trials of Middle Meningeal Artery Embolization

Trial	Population and treatment strategy	Embolic material	Primary result	Interpretation
EMBOLISE	Surgical candidates; surgery plus MMAE versus surgery	Onyx	Reoperation-related recurrence or progression: 4.1% vs 11.3%	Strong evidence for adjunctive embolization
MAGIC-MT	Surgical and nonsurgical strata	Onyx	Symptomatic recurrence/progression: 6.7% vs 9.9%; nonsignificant	Primary efficacy endpoint not met
STEM	Surgical and nonsurgical strata	Squid	Treatment failure: 16% vs 36%	Broad composite benefit at 180 days
EMPROTECT	High-risk or recurrent postoperative cSDH	Microparticles	Recurrence: 14.8% vs 21.0%; nonsignificant	Favorable direction without statistical superiority

MEMBRANE	Surgical and nonsurgical strata	n-BCA	Residual/recurrent hematoma or surgery: 11.6% vs 22.1%	Significant six-month composite benefit
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Interpretation of Trial Heterogeneity. The trials should not be classified simply as positive or negative. Their primary endpoints differed substantially. EMBOLISE counted recurrence or progression requiring reoperation. STEM included radiological residual or recurrence and rescue treatment. MAGIC-MT focused on symptomatic recurrence or progression. EMPROTECT evaluated postoperative recurrence. MEMBRANE included residual thickness greater than 10 mm or surgery. A procedure can reduce reoperation without accelerating complete radiological resolution. Conversely, residual thickness can persist in a clinically improving patient who never requires repeat surgery. Combining these endpoints changes the apparent effect size. Follow-up duration also matters. Embolization acts gradually. A 90-day endpoint may underestimate later separation between groups, whereas a six-month endpoint allows more time for membrane devascularization and hematoma resorption. Embolic agents differed. Liquid embolics can penetrate distal membrane vessels and remain radiographically visible. Particles depend on size, flow, catheter position, and avoidance of reflux. Whether these differences explain trial outcomes cannot be determined without direct randomized comparisons.

Imaging Phenotypes. Noncontrast CT remains the primary diagnostic modality. Relevant features include maximum thickness, three-dimensional volume, midline shift, ventricular compression, density, internal architecture, bilaterality, acute components, and cerebral atrophy. Homogeneous hypodense hematomas often represent liquefied chronic collections. Isodense collections can be difficult to detect, particularly when bilateral. Mixed-density and hyperdense regions suggest repeated bleeding or acute-on-chronic components. Internal septations divide the collection into compartments and may prevent complete drainage through a single catheter. Trabecular, laminar, separated, and gradation patterns have been associated with different recurrence risks in observational studies [5–9]. Contrast-enhanced CT or MRI may demonstrate enhancement of the outer membrane and septa. Membrane enhancement can support the biological rationale for embolization but is not required for treatment. Angiographic staining of the membrane appears as a distal vascular blush supplied by frontal and parietal MMA branches. The blush may correlate with active membrane vascularity, although standardized quantitative criteria have not been established. Bilateral cSDH may require bilateral embolization because each external membrane has an independent dural blood supply. Unilateral embolization may be insufficient even when symptoms are produced mainly by one side.

Clinical Urgency and Treatment Categories. Patients with severe neurological deterioration, reduced consciousness, progressive hemiparesis, significant language disturbance, seizures associated with mass effect, or herniation physiology should undergo rapid decompression. MMA embolization can be performed before or after surgery if it does not delay urgent treatment. Adjunctive embolization is best supported in symptomatic nonemergent surgical patients, especially when recurrence risk is increased. The procedure addresses membrane biology after the mass has been drained. Primary embolization without surgery may be considered in neurologically stable patients with mild or moderate symptoms, limited mass effect, high surgical or anesthetic risk, recurrent disease, or a strong need to continue antithrombotic therapy. The patient must tolerate gradual resolution. Observation remains appropriate for small, minimally symptomatic, stable hematomas without major midline shift. Embolization should not be performed simply because an MMA branch can be catheterized.

Table 2. Treatment Selection by Clinical Phenotype

Clinical phenotype	Preferred strategy	initial	Potential role of MMAE	Principal caution
Reduced consciousness or herniation signs	Immediate decompression	surgical	Adjunct after stabilization	Embolization must not delay surgery

Progressive focal deficit with major mass effect	Burr-hole drainage or craniotomy	Adjunctive recurrence prevention	Clinical recovery depends on rapid decompression
Stable symptomatic cSDH requiring surgery	Burr-hole drainage with drain	Strong randomized support as adjunct	Coordinate procedure timing
Mild symptoms and moderate hematoma	Observation, surgery, or selected MMAE	Possible primary treatment	Resolution is gradual
Recurrent cSDH after drainage	Repeat drainage when mass effect persists	Strong biological rationale	Exclude organized membranes requiring craniotomy
Bilateral cSDH	Side-specific or bilateral drainage	Frequently bilateral embolization	Assess both MMA territories
High antithrombotic requirement	Individualized surgical strategy	May reduce recurrence risk	Endovascular access still carries bleeding risk
Calcified or organized cSDH	Craniotomy and membranectomy	Limited adjunctive role	Embolization cannot remove rigid mass
Acute subdural hematoma	Emergency trauma management	Not standard treatment	Evidence does not support routine MMAE

Recurrence-Risk Stratification. Recurrence is multifactorial. Previous recurrence is among the strongest predictors of future failure. Bilateral disease, large postoperative residual collection, persistent midline shift, poor brain re-expansion, mixed density, internal septations, and anticoagulant therapy also increase concern. Severe cerebral atrophy limits re-expansion after drainage. The residual subdural space remains susceptible to fluid accumulation and membrane bleeding. Embolization may be especially rational in this phenotype, although atrophy also causes slow radiological resolution. A ventriculoperitoneal shunt, intracranial hypotension, arachnoid cyst, or spontaneous CSF leak can perpetuate the subdural space. Embolization does not correct the underlying pressure disorder and should be combined with management of the primary cause. Liver disease, thrombocytopenia, renal disease, alcohol misuse, and hematological malignancy alter coagulation and healing. These patients may benefit from recurrence prevention but also have greater procedural risk.

Middle Meningeal Artery Anatomy. The MMA usually arises from the first segment of the maxillary artery, enters the skull through the foramen spinosum, and divides into frontal and parietal branches. Numerous variations occur. The frontal branch courses toward the pterion and anterior convexity. The parietal branch supplies the posterior convexity. Successful membrane treatment generally requires coverage of the branch or branches supplying the entire hematoma. A recurrent meningeal branch may connect the MMA with the lacrimal or ophthalmic artery through the superior orbital fissure. Embolic migration into this pathway can cause retinal ischemia and permanent visual loss. The petrosal branch supplies the facial nerve through the facial-nerve arterial arcade. Embolization proximal to this branch or reflux into it can cause facial palsy. Anastomoses may also exist with the meningohypophyseal trunk, inferolateral trunk, occipital artery, ascending pharyngeal artery, contralateral MMA, and pial arteries. Complete angiographic assessment is mandatory before embolic injection [11–15]. The microcatheter should be advanced distal to dangerous branches whenever possible. Selective angiography must evaluate reflux pathways under conditions resembling the intended injection.

Endovascular Access. Transfemoral and transradial approaches are both feasible. Radial access can improve early mobilization and may reduce access-site bleeding in anticoagulated older adults. Tortuous subclavian, carotid, and external carotid anatomy may make radial catheterization difficult. Femoral access provides familiar support and accommodates a wide range of guiding catheters. Severe aortoiliac disease or obesity can complicate access and hemostasis.

The access choice should reflect arch anatomy, operator expertise, anticoagulation, peripheral vascular disease, and the need for bilateral treatment. Access convenience should not compromise stable distal catheterization.

Embolic Agents. Polyvinyl-alcohol particles and calibrated microspheres were used extensively in early MMAE practice. They are inexpensive and can penetrate distal vessels according to particle size and injection dynamics. Particles are not intrinsically radiopaque. Distribution is inferred from angiographic flow changes rather than directly visualized. Very small particles may enter dangerous anastomoses, whereas larger particles may remain too proximal to devascularize the membrane effectively. Particle embolization produced a favorable but nonsignificant effect in EMPROTECT. This result does not prove inferiority to liquid agents, because the trial differed in population, endpoint, and procedural protocol.

Ethylene-Vinyl Alcohol Copolymers. Onyx and Squid are nonadhesive liquid embolic agents consisting of ethylene-vinyl alcohol copolymer dissolved in dimethyl sulfoxide with suspended radiopaque material. They permit controlled injection and direct visualization of distal penetration. Prolonged injections can produce dense filling of frontal and parietal membrane branches. Limitations include cost, dimethyl-sulfoxide-compatible catheter requirements, injection-related discomfort in awake patients, imaging artifact, and the risk of proximal reflux.

n-Butyl Cyanoacrylate. n-BCA is an adhesive liquid embolic that polymerizes when exposed to ionic fluids. It is mixed with ethiodized oil to modify polymerization time and radiopacity. The agent can achieve rapid and durable distal occlusion but requires precise technique. Premature polymerization may trap the catheter or produce proximal occlusion, whereas an excessively dilute mixture may travel into undesired anastomoses. MEMBRANE demonstrated clinical efficacy with a standardized n-BCA system, supporting its role as an alternative liquid embolic.

PHIL. Precipitating hydrophobic injectable liquid is a nonadhesive copolymer with covalently bound iodine. It provides radiopacity without tantalum sedimentation and has been used in institutional series. Randomized cSDH evidence is less mature than for Onyx, Squid, or n-BCA.

Coils produce proximal arterial occlusion but generally do not penetrate the distal membrane network. Coil-only treatment may permit collateral revascularization and is not considered biologically equivalent to distal liquid or particle embolization. Coils can be used as an adjunct to protect a dangerous branch, reduce reflux, or secure the proximal artery after distal embolization.

Table 3. Comparison of Embolic Materials

Agent	Principal advantage	Principal limitation	Desired effect	procedural
PVA particles	Familiar, inexpensive, broad availability	Nonradiopaque and size-dependent penetration	Distal membrane-vessel occlusion	
Calibrated microspheres	More uniform particle size	Potentially limited distal penetration	Reproducible particulate embolization	
Onyx	Controlled radiopaque injection	Cost, artifact, DMSO requirements	Visible distal branch penetration	
Squid	Multiple viscosities and controlled delivery	Similar nonadhesive-liquid limitations	Dense membrane devascularization	
n-BCA	Rapid durable occlusion and low viscosity	Adhesive behavior and catheter-trapping risk	Deep controlled penetration with prompt polymerization	
PHIL	Homogeneous radiopacity and nonadhesive delivery	Limited randomized cSDH evidence	Distal liquid embolization	
Coils	Precise proximal occlusion	Inadequate membrane penetration alone	Adjunctive branch protection or proximal closure	

Technical Endpoint. The objective is not simply angiographic disappearance of the proximal MMA. Effective treatment requires distal penetration into the frontal and parietal branches that supply the hematoma membrane. Inadequate distal access may occur because of tortuosity, vasospasm, a small artery, dangerous anastomoses, prior embolization, or catheter instability. Proximal occlusion can reduce flow but may permit collateral revascularization. Bilateral or extensive convexity hematomas frequently require embolization of both major MMA divisions. Selective treatment of only the most accessible branch may leave a substantial portion of the membrane perfused. The technical report should document access, catheter position, dangerous branches, embolic agent, injected volume, distal distribution, reflux, branch coverage, final flow, and complications.

Timing Relative to Surgery. MMAE can be performed before, during a hybrid-session workflow, or after surgical drainage. Preoperative embolization devascularizes the membrane before decompression and may reduce intraoperative bleeding. It should not delay urgent surgery, and the full biological effect does not occur immediately. Postoperative embolization permits rapid decompression first and targets the residual membrane after the patient is stable. Brain re-expansion may alter the relationship between the membrane and dura but does not eliminate its arterial supply. Same-session treatment minimizes repeated hospitalization and anesthesia but requires hybrid-room availability or transfer between operating and angiography suites. Prolonged procedure time may be poorly tolerated by frail patients. The ideal interval has not been established. Practical selection should prioritize neurological urgency, anticoagulation reversal, staff availability, renal function, contrast exposure, and patient stability.

Radiological Resolution. After successful embolization, hematoma reduction is gradual. Early CT may show little change even when membrane devascularization has been achieved. Clinical improvement can precede complete radiological resolution. A natural-history cohort reported that only a minority of patients achieved substantial volume reduction at one month, whereas most demonstrated progressive resolution over several months. Complete resolution may require seven to twelve months [27–30]. Persistent asymptomatic thickness should not automatically be labelled failure. Repeat surgery should be based on symptoms, neurological deterioration, renewed mass effect, or progressive enlargement rather than a single residual measurement. Conversely, radiological improvement should not override clinical deterioration. New ischemic stroke, seizure, hydrocephalus, infection, or unrelated neurological disease may explain worsening despite a smaller hematoma.

Follow-Up Imaging. A practical schedule may include CT within the early postprocedural period when clinically indicated, followed by imaging at approximately six weeks to three months and later until substantial resolution or stability. Routine imaging frequency should be adapted to symptoms, hematoma size, antithrombotic therapy, bilateral disease, and initial midline shift. Automated segmentation may improve evaluation by measuring volume rather than maximum thickness alone. Three-dimensional brain-deformation metrics could eventually identify persistent local compression not reflected by a simple midline-shift measurement.

Antithrombotic Therapy. Many cSDH patients require anticoagulation for atrial fibrillation, venous thromboembolism, mechanical valves, or recent cardiac intervention. Prolonged interruption increases ischemic stroke, systemic embolism, valve thrombosis, and coronary risk. The timing of resumption should balance hematoma stability against thromboembolic indication. MMAE may theoretically stabilize membrane vessels and permit earlier resumption, but randomized evidence defining a universal schedule is lacking. Antiplatelet therapy for recent coronary stenting presents a distinct risk. Decisions should involve cardiology and consider stent type, implantation date, indication, and possibility of temporary monotherapy. Embolization itself requires arterial access and catheter manipulation. It should not be assumed to have no hemorrhagic risk merely because no cranial incision is made.

Complications. Potential neurological complications include ischemic stroke, retinal artery occlusion, cranial-nerve injury, intracranial hemorrhage, seizure, arterial dissection, and embolization of unintended territories. Visual loss is the most feared consequence of unrecognized MMA–ophthalmic anastomosis. Careful selective angiography and distal catheter placement are

mandatory. Facial palsy can result from embolization of the petrosal branch. MEMBRANE reported a transient device-related facial-nerve palsy that resolved on the same day. Access-site hematoma, pseudoaneurysm, arterial occlusion, contrast-associated kidney injury, and allergic reaction remain relevant in elderly patients. An apparently uncomplicated angiogram does not exclude delayed neurological events. Postprocedural examination should include vision, facial movement, speech, motor function, and access-site assessment.

Table 4. Major Technical Hazards and Prevention

Hazard	Anatomical or technical mechanism	Preventive strategy	Potential consequence
Retinal embolization	MMA–lacrima or ophthalmic anastomosis	Distal catheterization and selective angiography	Permanent visual loss
Facial-nerve ischemia	Embolization of petrosal branch	Identify branch and inject distally	Facial palsy
Pial embolization	Dural–pial anastomosis	Careful angiographic assessment and controlled injection	Cerebral infarction
Catheter entrapment	Adhesive polymerization or excessive reflux	Appropriate n-BCA mixture and catheter management	Arterial injury
Proximal-only occlusion	Inadequate distal penetration	Advance catheter and cover both major branches	Treatment failure or recurrence
Arterial dissection	Tortuous anatomy and wire manipulation	Gentle technique and stable support	Stroke or access failure
Access-site hemorrhage	Anticoagulation and arterial puncture	Ultrasound-guided access and closure strategy	Hematoma or transfusion
Contrast kidney injury	Advanced age and renal dysfunction	Dose minimization and renal assessment	Acute kidney injury

Brain Re-Expansion. Brain re-expansion is an important but underreported determinant of success. After drainage or embolization, the cerebral hemisphere must gradually occupy the previous subdural space. Advanced atrophy, prolonged compression, impaired compliance, arachnoid scarring, intracranial hypotension, and persistent CSF diversion can prevent re-expansion. A residual space does not necessarily indicate ongoing bleeding. It may reflect limited brain elasticity. Embolization can prevent new accumulation but cannot reverse severe atrophy. The distinction affects interpretation of radiological endpoints based on thickness greater than 10 mm. A stable, asymptomatic residual space may have different clinical significance from an enlarging vascularized collection of identical thickness.

Combined Treatment. Combined surgery and embolization addresses both principal mechanisms: mass effect and membrane vascularity. Randomized evidence is strongest for this approach. The sequence should be individualized. Neurological urgency favors surgery first. Stable elective cases may undergo embolization before or after drainage according to local workflow. A combined pathway requires communication among vascular neurology, neurosurgery, neurointerventional specialists, anesthesia, radiology, geriatric medicine, and rehabilitation. The patient should understand that embolization reduces but does not eliminate recurrence. Repeat imaging and clinical follow-up remain necessary.

Primary Embolization. Primary MMAE is attractive for frail patients and those with major anesthetic risk, but it is not appropriate when rapid mass removal is necessary. The patient should have stable or slowly progressive symptoms, preserved consciousness, no herniation signs, and sufficient neurological reserve to tolerate gradual improvement. Radiological size alone should not determine eligibility. A relatively thick bilateral hematoma with minimal symptoms may be managed differently from a smaller unilateral lesion causing progressive dysphasia. STEM and MEMBRANE support an embolization benefit across surgical and nonsurgical strata, but the individual strata were not always powered independently. Further dedicated randomized trials of primary embolization are needed.

Cost and Resource Utilization. MMAE adds procedural cost, angiography-suite time, embolic material, contrast, and specialist staffing. These costs may be offset by fewer repeat operations, rehospitalizations, ICU admissions, and interruptions of antithrombotic therapy. MEMBRANE reported fewer patients requiring ICU care during rehospitalization and shorter rehospitalization duration with embolization plus standard care, although these were secondary analyses. Universal embolization may not be cost-effective in systems where surgical recurrence is already low. Targeting patients with high predicted recurrence risk is likely to provide greater value. Economic evaluations should include device cost, procedural complications, follow-up imaging, repeat surgery, rehabilitation, caregiver burden, and preserved independence.

The MEMBRANE-RISK Framework. The proposed MEMBRANE-RISK model integrates eleven domains. Membrane activity includes mixed density, membrane enhancement, angiographic blush, recurrent bleeding, and inflammatory organization. Expansion or recurrence risk incorporates residual thickness, bilateral disease, anticoagulation, septations, prior recurrence, atrophy, and limited brain re-expansion. Morphology defines homogeneous, mixed-density, septated, organized, calcified, acute-on-chronic, and bilateral phenotypes. Brain re-expansion evaluates age-related atrophy, intracranial hypotension, shunt status, and postoperative residual space. Recurrent-disease history distinguishes first presentation from repeated surgical failure. Arterial anatomy identifies branch supply, dangerous anastomoses, bilateral contribution, and technical feasibility. Neurological urgency determines whether immediate drainage must precede any endovascular treatment. Embolic strategy includes agent, catheter position, distal penetration, branch coverage, and reflux control. Resumption of antithrombotic therapy incorporates the thromboembolic indication and radiological stability. Imaging follow-up measures volume, thickness, midline shift, internal density, and brain deformation over time. Safety and knowledge-based outcome assessment include neurological complications, function, cognition, recurrence, reoperation, quality of life, and cost.

Table 5. Scientific Originality of the MEMBRANE-RISK Framework

Existing limitation	Conventional interpretation	MEMBRANE-RISK principle	Expected contribution
cSDH viewed as residual blood	Drainage considered complete biological treatment	Vascularized membrane recognized as therapeutic target	Explains rationale for adjunctive embolization
MMAE treated as replacement for surgery	Endovascular treatment assumed sufficient for every patient	Neurological urgency determines need for immediate decompression	Prevents dangerous delay
Recurrence risk judged from size alone	Thickness used as principal criterion	Morphology, bilaterality, antithrombotics, atrophy, and re-expansion integrated	Improves patient selection
Trial results classified only as positive or negative	Different endpoints compared directly	Reoperation, radiological residual, symptomatic progression, and function separated	Clarifies evidence heterogeneity
Technical success defined by proximal occlusion	Disappearance of MMA flow considered adequate	Distal membrane penetration and branch coverage prioritized	Reduces biological treatment failure
Embolic agents treated as equivalent	Material choice considered irrelevant	Penetration, visibility, polymerization, and anastomotic risk assessed	Supports agent-specific planning
Residual hematoma labelled immediate failure	Slow resolution not considered	Expected months-long biological response incorporated	Avoids unnecessary reoperation

Antithrombotic therapy considered only a bleeding risk	Prolonged withdrawal accepted routinely	Thromboembolic need included in treatment planning	Supports balanced medication resumption
Cost assessed from index procedure alone	Added embolization viewed only as expense	Reoperation, rehospitalization, and functional dependence included	Improves health-system evaluation

Discussion. The randomized-trial era has established that MMA embolization is no longer an experimental concept supported only by retrospective case series. The treatment can reduce recurrence, progression, or broader treatment failure in selected patients with nonacute subdural hematoma. The evidence is strongest for adjunctive treatment in patients undergoing surgical evacuation. EMBOLISE showed a substantial reduction in reoperation-related recurrence or progression. The absolute risk reduction was approximately seven percentage points, corresponding to prevention of one repeat operation for roughly every fourteen treated patients. This benefit is clinically meaningful because reoperation in an older adult is not a minor event. It involves recurrent hospitalization, interruption of anticoagulation, anesthesia, infection risk, functional decline, and delayed rehabilitation. STEM demonstrated a larger absolute reduction, but its composite endpoint was broader and included radiological residual or recurrence. The trial therefore measures a different construct from EMBOLISE. MEMBRANE similarly combined residual or recurrent thickness with surgical treatment. Its absolute difference of approximately ten percentage points supports n-BCA embolization but should not be interpreted as directly superior to another agent without a head-to-head trial. MAGIC-MT and EMPROTECT are equally important because they prevent overly simplistic conclusions. Both produced numerically favorable results but failed to meet their primary efficacy endpoints. Neutral results can arise when the actual control-event rate is lower than expected, reducing statistical power. They can also reflect population differences, incomplete distal embolization, endpoint timing, embolic-agent behavior, or a true smaller biological effect. The differences among trials do not currently prove that liquid embolics are superior to particles. Liquid agents dominated the positive EMBOLISE, STEM, and MEMBRANE trials, whereas particles were used in EMPROTECT. However, population and endpoint differences prevent causal attribution to the material alone.

Future comparative studies should standardize catheter position, branch coverage, embolic volume, dangerous anastomoses, and final angiographic distribution. Simply recording the product name is insufficient. Distal penetration is likely important because the pathological capillary network resides within the membrane. Proximal MMA occlusion can be bypassed by collateral dural vessels. More aggressive penetration can also increase risk. The operator must balance membrane devascularization against retinal, cranial-nerve, pial, and internal-carotid anastomoses. Maximum penetration is not synonymous with maximum safety. The appropriate treatment endpoint may be a sufficient reduction in membrane flow without filling every microscopic vessel. Quantitative angiographic markers are needed. The trials also demonstrate that MMAE should not be evaluated solely through radiological disappearance. In MEMBRANE, the overall rate of substantial volume reduction at later follow-up was similar between groups even though recurrent or residual failure and surgery were reduced. This finding supports the hypothesis that embolization may stabilize the disease and prevent clinically important reaccumulation without necessarily accelerating every patient's resorption curve.

Functional outcomes are influenced by age, frailty, baseline cognition, cerebral atrophy, previous stroke, and systemic illness. Avoiding reoperation may preserve function indirectly, but an endovascular procedure cannot reverse established neurodegenerative disease. Cognitive impairment deserves greater attention. Chronic subdural hematoma can mimic dementia through frontal-subcortical network compression. MEMBRANE reported an exploratory signal of greater normalization of Mini-Mental State Examination performance in the embolization group, but this requires prospective confirmation. Primary MMA embolization is a particularly important research area. Many frail patients have symptoms and sizeable hematomas but remain stable enough to avoid emergency surgery. An effective stand-alone treatment could reduce anesthesia and cranial surgical

risk. The delayed response must be communicated. Symptoms may improve gradually, and CT can remain abnormal for months. The patient needs a reliable monitoring pathway and rapid access to surgery if deterioration occurs. Outpatient primary treatment is inappropriate when social support, follow-up imaging, or emergency access is unreliable. The definition of urgency should be clinical rather than based on a single measurement. A 15-mm collection with progressive aphasia may require faster decompression than a 25-mm bilateral collection in an alert, minimally symptomatic patient with severe atrophy. Midline shift is also imperfect in bilateral disease because opposing collections can compress the brain without shifting the midline. Automated volume and brain-deformation analysis may eventually improve selection. Current maximum-thickness criteria do not fully quantify local cortical compression, ventricular distortion, or bilateral mass effect. MMAE is often attractive in anticoagulated patients, but the assumption that it permits immediate anticoagulant resumption is not yet supported by a uniform randomized protocol. The thromboembolic indication should be weighed against residual hematoma stability.

A patient with a mechanical heart valve has a different medication priority from a patient receiving aspirin for primary prevention. Antithrombotic decisions should not be standardized solely by the embolization date. The role of MMAE in acute-on-chronic hematoma remains less certain. A chronic vascularized membrane may be present, but the acute component can create rapid mass effect and indicate active bleeding not controlled by membrane devascularization alone. Organized and calcified hematomas also represent a distinct phenotype. Embolization may reduce future membrane bleeding but cannot remove a rigid capsule that prevents brain expansion. Bilateral treatment should be considered when both sides contain active chronic collections. Even if one side is surgically drained, the contralateral membrane may continue to expand. Procedure-related neurological complications are uncommon but potentially devastating. Permanent visual loss or cerebral infarction can outweigh the benefit of avoiding recurrence in a benign disease. This risk makes training and credentialing important. Operators require expertise in external-carotid anatomy, microcatheterization, embolic-agent behavior, and recognition of dangerous anastomoses.

Standardized reporting should include whether ophthalmic, petrosal, pial, or internal-carotid connections were evaluated. Complications should be independently adjudicated. The optimal procedural setting remains uncertain. Local anesthesia permits neurological monitoring but can be uncomfortable during dimethyl-sulfoxide injection. General anesthesia improves immobility but adds physiological and logistical burden. Radial access may support rapid mobilization but is not inherently superior in all anatomies. The best access is the one that permits safe, stable distal catheterization with minimum procedural risk. The complete care pathway remains important. Surgical drains, irrigation technique, blood-pressure management, antithrombotic decisions, mobilization, fall prevention, and rehabilitation influence recurrence and outcome. Dexamethasone should not be viewed as an equivalent membrane-targeting alternative. The Dex-CSDH trial showed fewer repeat operations but worse functional outcomes and more adverse events with dexamethasone, demonstrating that reduction of recurrence alone does not guarantee net benefit [24–27]. Atorvastatin has shown possible benefit in selected conservatively managed populations, but evidence and generalizability remain less definitive than the current randomized MMAE data. The future standard is likely to be risk-adapted rather than universal. A low-risk first cSDH with excellent brain re-expansion after drainage may derive only a small absolute benefit. A bilateral recurrent hematoma in an anticoagulated patient with severe atrophy may have a much larger potential benefit. The MEMBRANE-RISK framework is intended to formalize this distinction. It links biological membrane activity with clinical urgency and technical feasibility. The framework does not provide a validated numerical score. Prospective registries could assign weights to its components and test whether it predicts absolute benefit from embolization. Future patient-level meta-analysis should harmonize the randomized trials and evaluate treatment interactions with surgical versus nonsurgical management, bilateral disease, age, antithrombotic therapy, baseline thickness, midline shift, density pattern, embolic agent, and distal penetration.

Long-term follow-up is necessary. Most trials emphasized 90- or 180-day outcomes, but radiological resolution and recurrent falls extend beyond these windows. Patient-centered outcomes should include cognition, gait, return to independent residence, antithrombotic interruption,

caregiver burden, quality of life, and number of hospital encounters. The procedure's rapid adoption should not outpace quality control. Institutions should audit technical success, dangerous-anastomosis recognition, neurological complications, recurrence, reoperation, and cost.

Conclusion. Chronic subdural hematoma is an active angiogenic and inflammatory disease of the dural border-cell compartment. Surgical drainage removes the collection and relieves mass effect but does not directly eliminate the vascularized external membrane responsible for persistent exudation and recurrent microhemorrhage. Middle meningeal artery embolization addresses this membrane biology by reducing arterial supply to the pathological capillary network. The treatment usually produces gradual stabilization and resorption rather than immediate decompression. Randomized evidence supports MMA embolization as an adjunct to standard treatment in selected nonemergent patients. EMBOLISE reduced recurrence or progression requiring repeat surgery after surgical evacuation. STEM reduced a broader 180-day treatment-failure composite. MEMBRANE demonstrated a reduction in residual or recurrent hematoma or subsequent surgery with n-BCA. MAGIC-MT and EMPROTECT did not meet their primary efficacy endpoints, despite directionally favorable results. These studies demonstrate that treatment effect depends on population, endpoint, follow-up duration, surgical strategy, procedural performance, and possibly embolic-agent characteristics. MMA embolization must not replace urgent surgical decompression in patients with declining consciousness, severe neurological deterioration, marked mass effect, or herniation physiology. The strongest current role is adjunctive recurrence prevention after or around surgical drainage. Stand-alone embolization may be appropriate for neurologically stable patients who can tolerate gradual resolution, particularly when surgical risk or recurrence risk is high. Safe performance requires selective angiographic evaluation of ophthalmic, petrosal, pial, and internal-carotid anastomoses; distal catheterization; controlled embolic delivery; and adequate coverage of the membrane-supplying branches. Radiological resolution can require several months. Persistent asymptomatic thickness should not automatically be considered treatment failure, whereas clinical deterioration requires immediate reassessment regardless of imaging trends.

No embolic material has been proven universally superior through direct randomized comparison. Liquid agents provide visible distal penetration, particles remain widely available, and each strategy has specific technical risks. The proposed MEMBRANE-RISK framework integrates membrane activity, recurrence risk, morphology, brain re-expansion, previous failure, arterial anatomy, neurological urgency, embolic strategy, antithrombotic resumption, imaging follow-up, safety, and patient-centered outcomes. The future of chronic subdural hematoma management is likely to be an integrated neurosurgical–endovascular pathway in which immediate decompression and biological recurrence prevention are applied selectively according to the individual patient's clinical and anatomical phenotype.

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