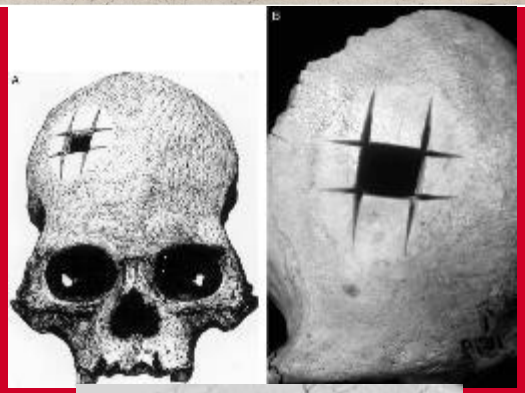
RUTGERS HEALTH

RWJBarnabas HEALTH



Middle Meningeal Artery MMA Embolization (MMAe) for Chronic Subdural Hematoma(cSDH)

Gaurav Gupta MD, FAANS

Associate Professor- Neurosurgery

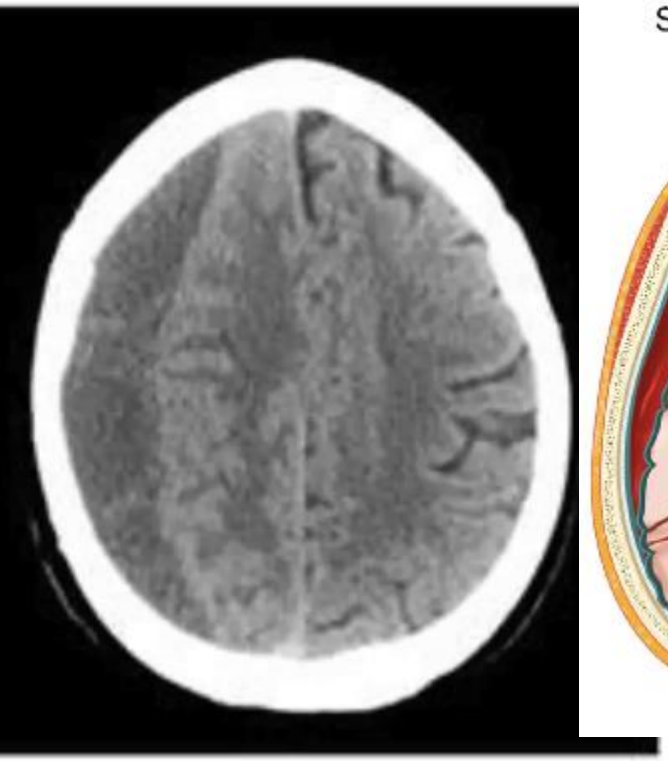
System Co-Director Cerebrovascular and Endovascular Neurosurgery
Rutgers- RWJ Barnabas Health care system

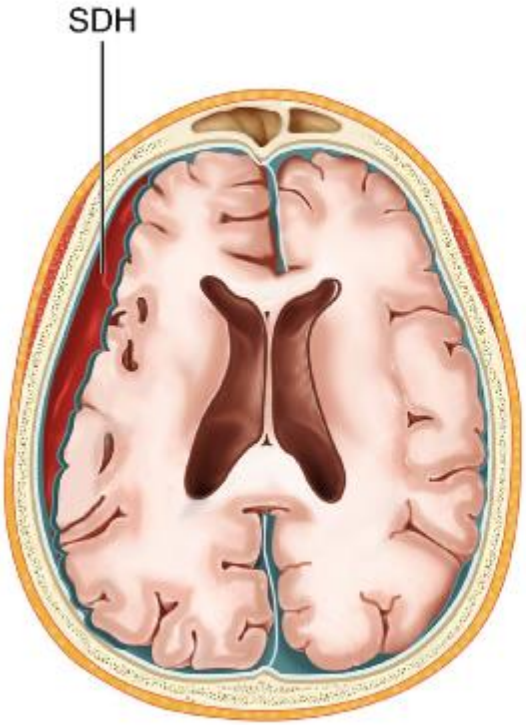
Director, Cerebrovascular and Endovascular Neurosurgery

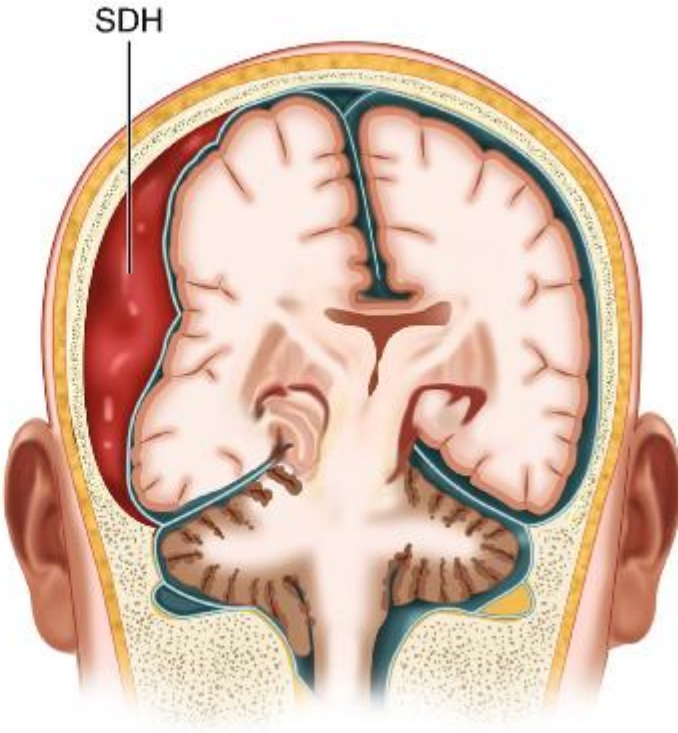
Rutgers Robert Wood Johnson Medical School and RWJ Univ Hospital

- Is MMA embolization effective at reducing reoperation rate?
- Is MMA embolization effective at reducing hematoma volume?
- Does choice of embolization agent matter?
- Does degree of embolic penetration matter?
- Is bilateral embolization necessary?

Subdural hematoma

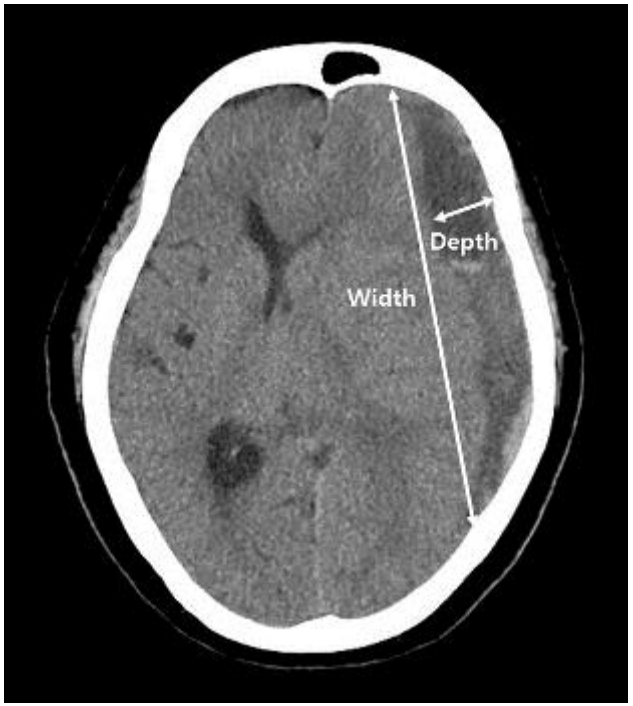


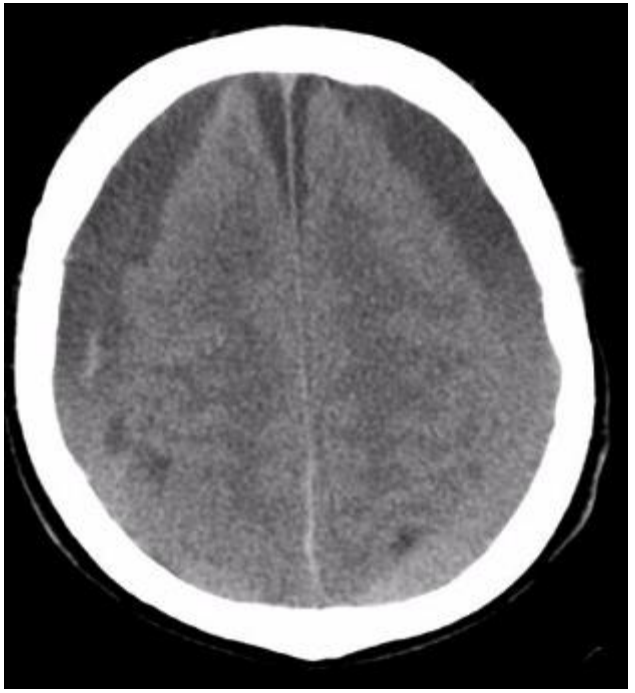




Turgut, M., Kalkan, E. (2021). Medicolegal Aspects of Subdural Hematoma. In: Turgut, M., Akhaddar, A., Hall, W.A., Turgut, A.T. (eds) Subdural Hematoma. Springer, Cham. https://doi.org/10.1007/978-3-030-79371-5_39

Unilateral or Bilateral or interhemispheric





- Concavo-convex peri-cerebral collection along cranial convexity
- Bilateral seen in 9-22% cases

M. Rauhala, T.M. Luoto, H. Huhtala, *et al.* The incidence of chronic subdural hematomas from 1990 to 2015 in a defined Finnish population J. Neurosurg, 1 (aop) (2019), pp. 1-11

K. MORI, M. MAEDA Surgical treatment of chronic subdural hematoma in 500 consecutive cases: clinical characteristics, surgical outcome, complications, and recurrence rate Neurol. Med.-Chir, 41 (8) (2001), pp. 371-381

Historical Vignette

3 centuries ago- Stroke

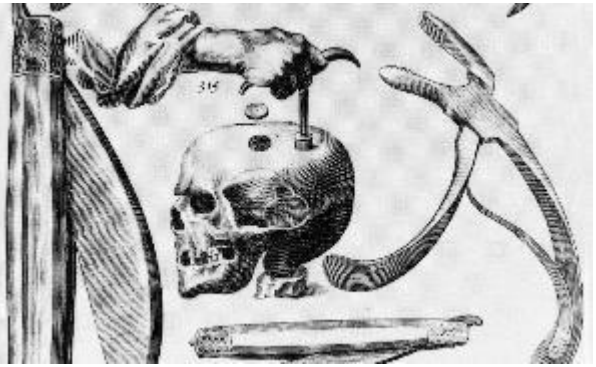
One century ago- inflammation

Early 20th century

- Traumatic tearing of bridging veins connecting cortex to dura.
- Osmotic pressure / effusion
- Coagulation and fibrinolytic systems activated, angiogenesis, inflammation , recurrent microbleeds, exudate.

(Virchow R. Das haematom der dura mater. Verh Phys Med Ges Wuerzburg. 1857;7:134–42).
Putnam TJ, Cushing H. Chronic subdural hematoma: its pathology, its relation to pachymeningitis hemorrhagica interna and its surgical treatment. Arch Surg. 1925;11:329–39. Schachenmayr W, Friede RL. The origin of subdural neomembranes. Fine structure of dura-arachnoid interface in man. Am J Pathol. 1978;92:53–68.
Shin DS, Hwang SC. Neurocritical management of traumatic acute subdural hematomas. Korean J Neurotrauma. 2020;16:113–25. https://doi.org/10.13004/kjnt.2020.16.e43.
Edlmann E, Giorgi-Coll S, Whitfield PC, Carpenter KLH, Hutchinson PJ. Pathophysiology of chronic subdural haematoma: inflammation, angiogenesis and implications for pharmacotherapy. J Neuroinflammation. 2017;14:108. https://doi.org/10.1186/s12974-017-0881-y.
Fu S, Li F, Bie L. Drug therapy for chronic subdural hematoma: bench to bedside. J Clin Neurosci. 2018;56:16–20. https://doi.org/10.1016/j.jocn.2017.07.034

Trephanation- borer or Auger (drill) – oldest neurosurgical procedure



- Trepanation / trephination for SDH- oldest neurosurgical surgery
- Hellenic Hippograte s (460-370 BC)

- Common in elderly population/ medically co-morbid population
- Average age 63 yrs
- 17.6 cases per 100,000 population
- 58.1 per 100000 per year for 65 yrs or older
- 130 per 100,000 per year for 80-89
- Incidence increases 3X in population > 80 years
- By 2030, chronic SDH is predicted to be the most common cranial neurosurgery procedure.

Balser D, Farooq S, Mehmood T, Reyes M, Samadani U. Actual and projected incidence rates for chronic subdural hematomas in United States Veterans Administration and civilian populations. J Neurosurg. 2015;123:1209–15. https://doi.org/10.3171/2014.9.JNS141550.
Neifert SN, Chaman EK, Hardigan T, Ladner TR, Feng R, Caridi JM, et al. Increases in subdural hematoma with an aging population-the future of American cerebrovascular disease. World Neurosurg. 2020;141:e166–74. https://doi.org/10.1016/j.wneu.2020.05.060.

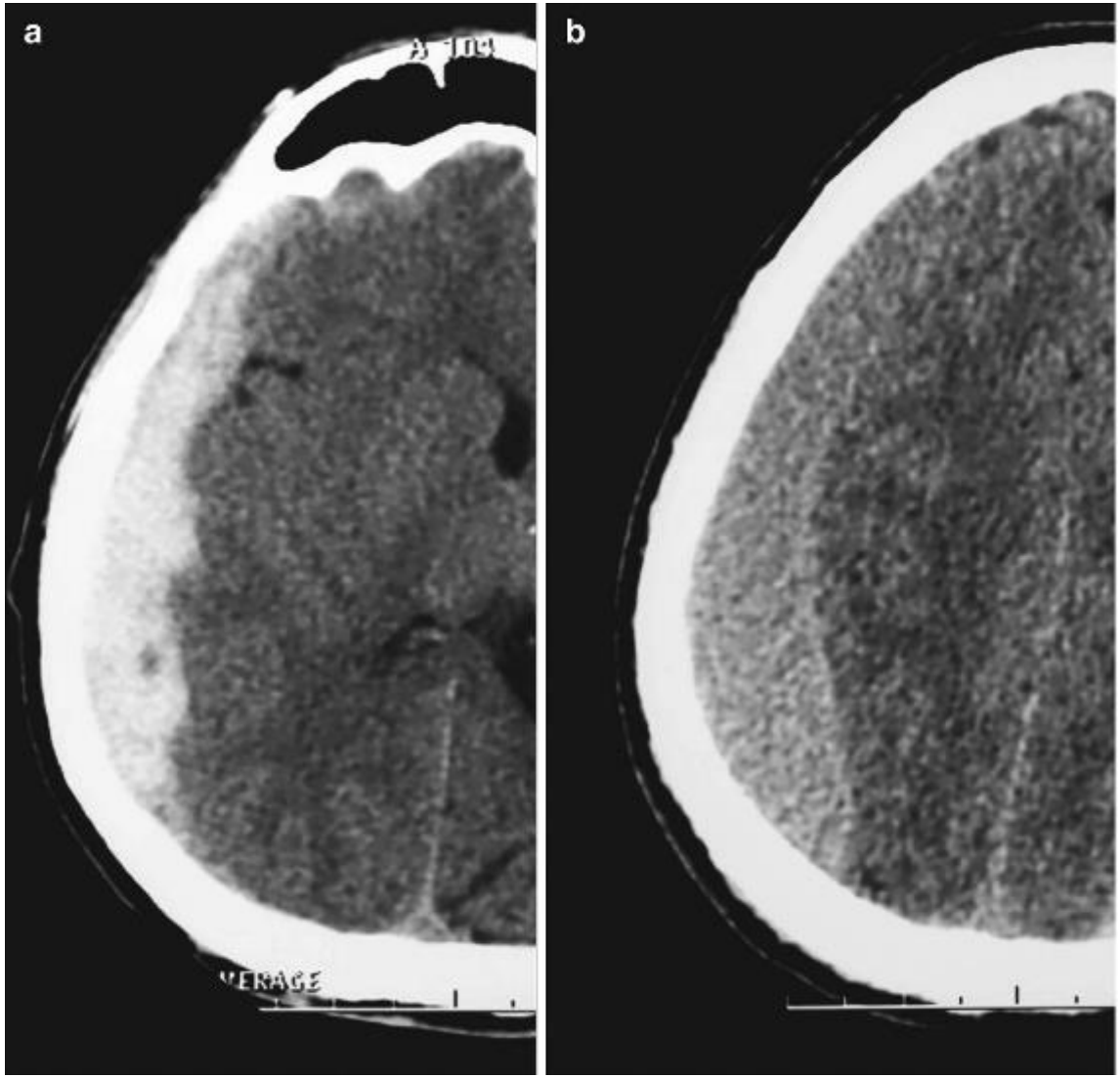
Risk factors

- Cranio-cerebral disproportion- Age related volume loss (also happens in every married man)
- Trauma - 50-80% have history of trauma and blood thinners.
- Coagulopathy (and therapeutic anticoagulation)
- CSF shunt
- Intra-cranial hypotension -LP shunts, Spinal fluid leak – bilateral
- Seizures
- Chronic alcoholism

Balser D, Farooq S, Mehmood T, Reyes M, Samadani U. Actual and projected incidence rates for chronic subdural hematomas in United States Veterans Administration and civilian populations. J Neurosurg. 2015;123:1209–15. https://doi.org/10.3171/2014.9.JNS141550.
Neifert SN, Chaman EK, Hardigan T, Ladner TR, Feng R, Caridi JM, et al. Increases in subdural hematoma with an aging population-the future of American cerebrovascular disease. World Neurosurg. 2020;141:e166–74. https://doi.org/10.1016/j.wneu.2020.05.060.

Acute SDH Vs Chronic SDH

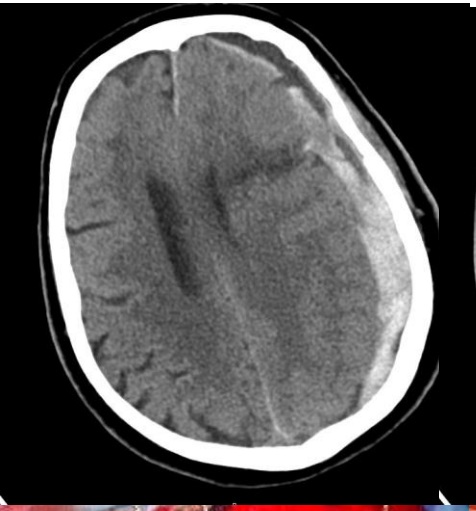
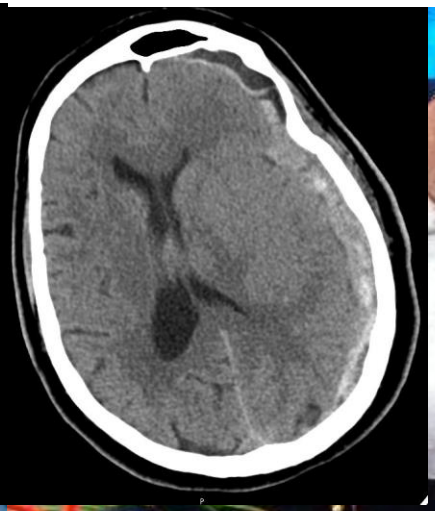
Acute SDH



Chronic SDH > 3 Weeks old

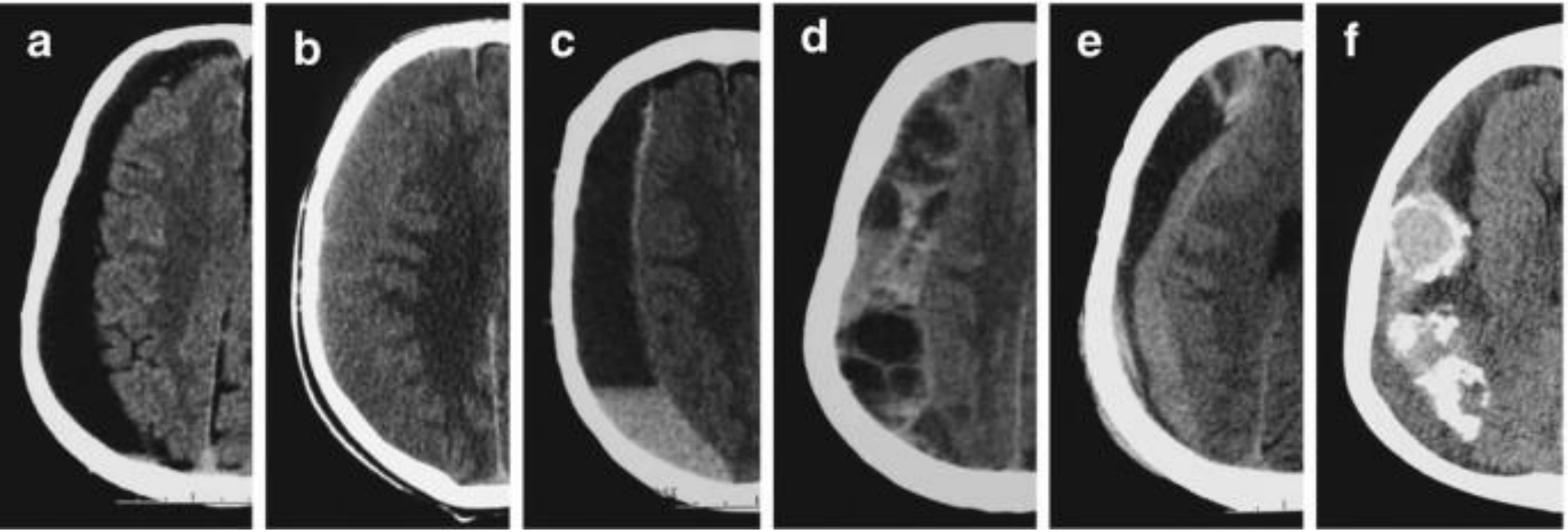
Akhaddar, A. (2021). Neuroimaging Differential Diagnosis (Imaging Mimicking Conditions) of Cranial Chronic Subdural Hematoma. In: Turgut, M., Akhaddar, A., Hall, W.A., Turgut, A.T. (eds) Subdural Hematoma. Springer, Cham. https://doi.org/10.1007/978-3-030-79371-5_26

Acute SDH







Low Density

Iso Dense

Acute on Chronic

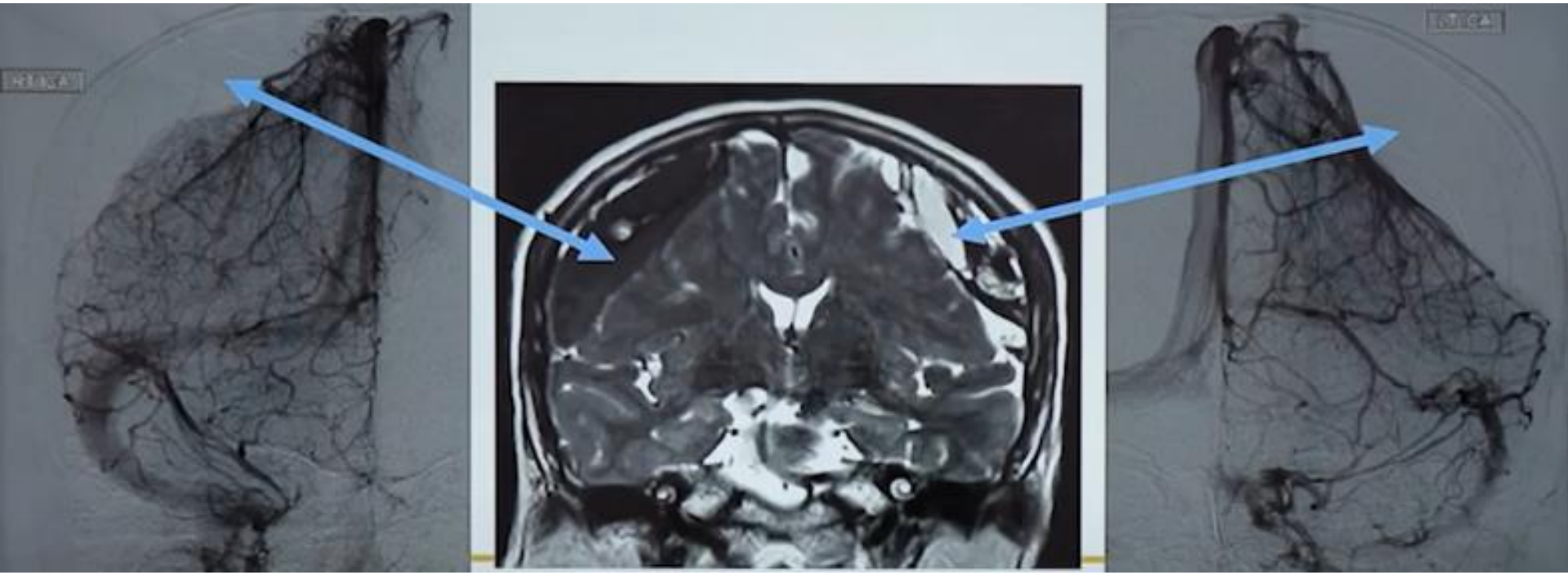
Mixed Density with loculations

Organized multi-compartment

Calcified SDH

Akhaddar, A. (2021). Neuroimaging Differential Diagnosis (Imaging Mimicking Conditions) of Cranial Chronic Subdural Hematoma. In: Turgut, M., Akhaddar, A., Hall, W.A., Turgut, A.T. (eds) Subdural Hematoma. Springer, Cham. https://doi.org/10.1007/978-3-030-79371-5_26

Mass effect on brain



MMA embolization" An emerging treatment technique for cSDH: Dr. J.Johnson, G. Colby- UCLA

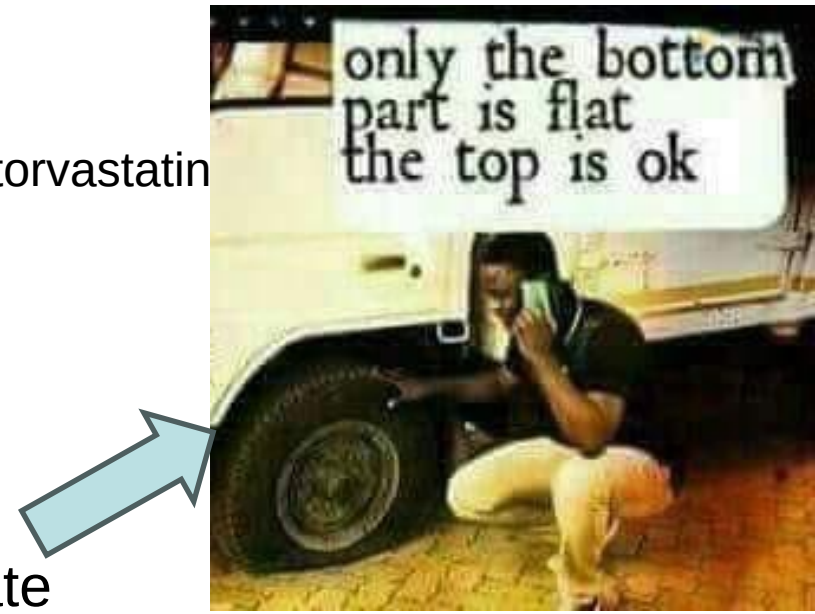
Clinical manifestations

- headache, confusion
- language difficulties,
- nausea/vomiting
- progressive mental deterioration
- gait disturbance/imbalance
- limb weakness- subtle drift –hemiparesis
- Seizures
- incontinence.
- Or acute symptoms with varying degrees of hemiplegia, seizures, or even coma

Akhaddar A, Karouache A, Boucetta M. Bilateral chronic subdural hematoma misdiagnosed as neuroleptic malignant syndrome. Emerg Med J. 2010;27:233. https://doi.org/10.1136/emj.2008.071001.

Treatment of cSDH

- Conservative mgmt-
  - Clot thickness < 5mm- can regress spontaneously
  - Minimal or no midline shift,
  - Neurologically intact
  - Meds- steroids, TXA, ACE inhibitors, Atorvastatin



- Surgical - Burr Holes
  Craniotomy

- Despite surgery, high recurrence rate
- Upto 35% Reacculation / symptomatic recurrence requiring re- treatment

Parlato C, Guarracino A, Moraci A (2000) Spontaneous resolution of chronic subdural hematoma. Surg Neurol. ;53(4):312-5; discussion 315-7. doi:10.1016/s0090-3019(00)00200-7
Kageyama H, Toyooka T, Tsuzuki N, Oka K (2013) Nonsurgical treatment of chronic subdural hematoma with tranexamic acid. J Neurosurg Aug 119(2):332-337. doi:10.3171/2013.3.JNS122162
Weigel R, Hohenstein A, Schlickum L, Weiss C, Schilling L (2007) Angiotensin converting enzyme inhibition for arterial hypertension reduces the risk of recurrence in patients with chronic subdural hematoma possibly by an antiangiogenic mechanism. Neurosurg Oct 61(4):788-792 discussion 792-3. doi:10.1227/01.NEU.0000298907.56012.E8

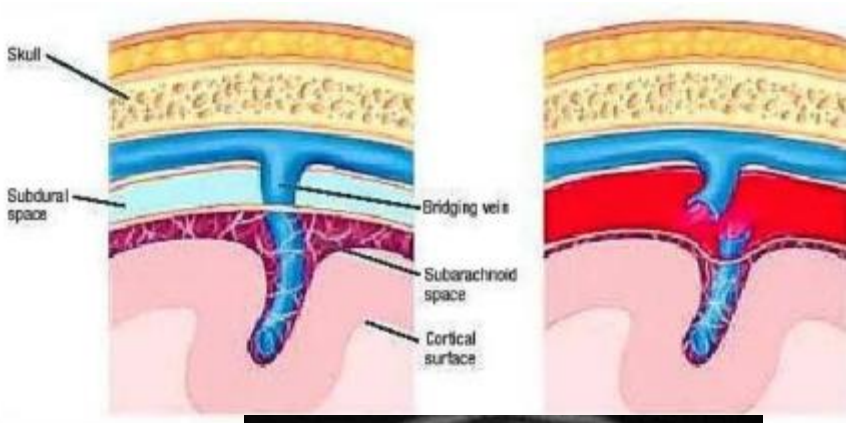
Clinical evolution

- 3 stages
  - 1. Initial traumatic event – day 0 often minor and sometimes unnoticed
  - 2. Latency phase – day 1-2 weeks asymptomatic
  - Chronic SDH – Clinical presentation phase

Pathophysiology

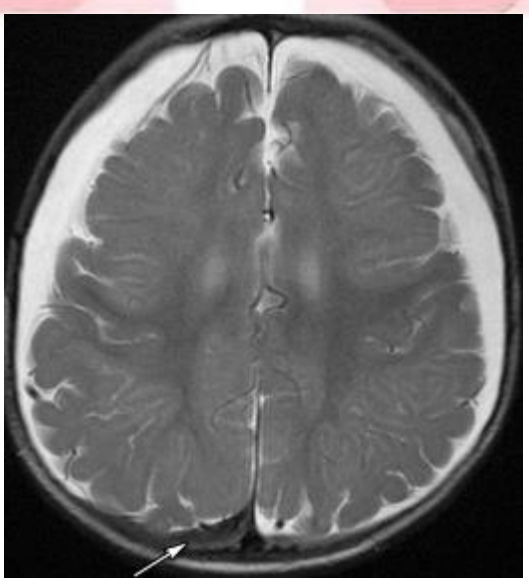
Trauma - tear in **Dural Border Cells of Dura** and avulsed subdural vein

CSF and blood leaks into Subdural space



Invokes an inflammatory response in Dural Border Cells(DBC)

Release pro-inflammatory cytokines (IL-2, IL-5,IL-6,IL-7,IL-8), angiogenetic factors
Overwhelm anti-inflammatory cytokines IL-10 and IL-13



Attracts inflammatory cells neurotrophils, eosinophils
Recruitment of fibroblasts

Shim YS, Park CO, Hyun DK, Park HC, Yoon SH (2007) What are the causative factors for a slow, progressive enlargement of a chronic subdural hematoma? Yonsei Med J Apr 30(2):210–217. doi:10.3349/ymj.2007.48.2.210
Edlmann E, Giorgi-Coll S, Whiteld PC, Carpenter KLH, Hutchinson PJ (2017) Pathophysiology of chronic subdural haematoma: inammation, angiogenesis and implications for pharmacotherapy. J Neuroinammation May 30(1):108. doi:10.1186/s12974-017-0881-y
Nakagawa and Conway 2004

Pathophysiology

Increased Local VEGF/VEGF-2 (an angiogenic factor)

Increased angiopoietin-2 (promotes cell death and disrupts vessels) increase in the outer membrane

↓

Surgical drainage decreases local VEGF

Neo vascularization in outer membrane

↓

Ingrowth of immature neo-capillaries, enzymatic fibrinolysis and liquefaction of clot in an attempt to repair by formatting granulation tissue

Sinusoidal capillaries (or macro-capillaries) are fragile and leaky compared with other types of capillaries (thin or absent basement membranes, a lack of smooth muscle cells and pericytes, and gaps)

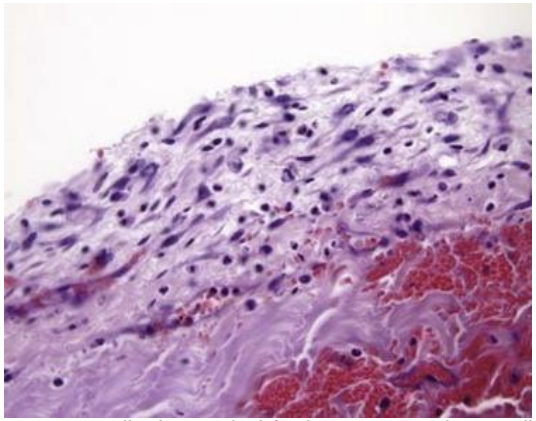
THESE CAPILLARIES ARE THE MAIN CAUSE OF REPEAR HEMORRHAGE

Fibrin degradation products reincorporated into new clots inhibit hemostasis

Shim YS, Park CO, Hyun DK, Park HC, Yoon SH (2007) What are the causative factors for a slow, progressive enlargement of a chronic subdural hematoma? Yonsei Med J Apr 30(2):210–217. doi:10.3349/ymj.2007.48.2.210
Edlmann E, Giorgi-Coll S, Whiteld PC, Carpenter KLH, Hutchinson PJ (2017) Pathophysiology of chronic subdural haematoma: inammation, angiogenesis and implications for pharmacotherapy. J Neuroinammation May 30(1):108. doi:10.1186/s12974-017-0881-y

The ‘membranes’ of cSDH

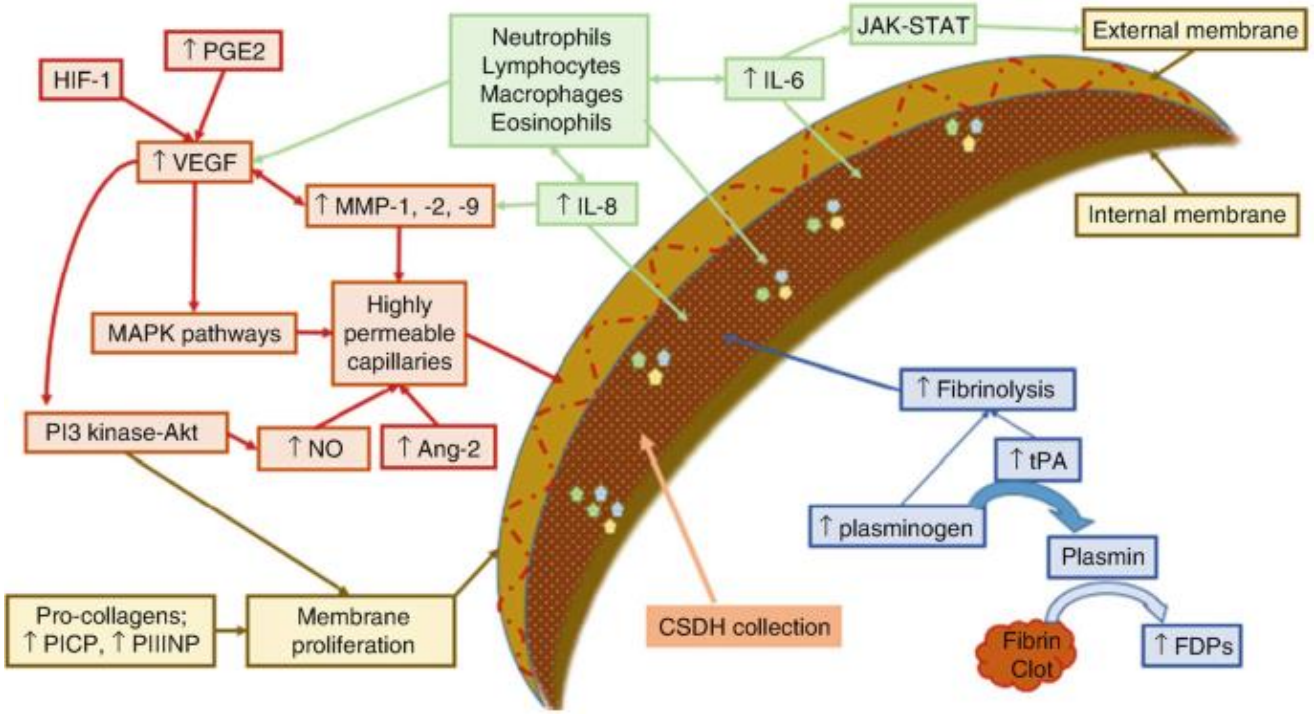
- Inner membranes
- Outer membranes
  - has abundant blood vessels, with giant capillaries having a large lumen similar to veins, but without smooth muscle cells.
  - These capillaries show abnormal permeability through the large gaps and sparse basal membrane
  - Permits direct spill of vascular contents in the extravascular space.
  - Has wide gaps, 0.4–1 μm, between adjacent endothelial cells,
  - facilitates transport of substances and migration of cells as they would from intercellular gaps of venules in inflamed tissue.



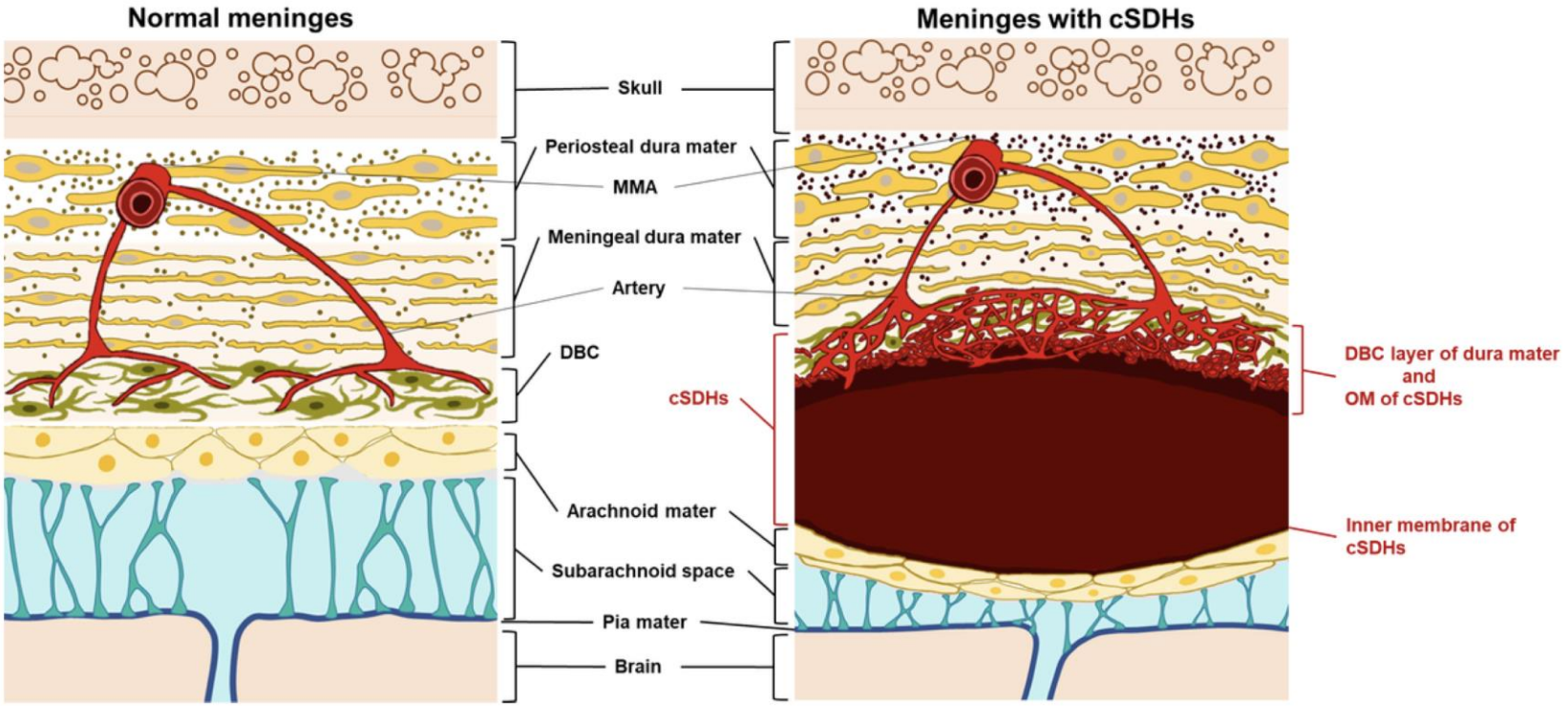
Histology of Outer Membrane 7 days post bleed showing proliferating fibroblasts and erythrocytes

Dana C. Holl, Victor Volovici, Clemens M.F. Dirven, Wilco C. Peul, Fop van Kooten, Korné Jellema, Niels A. van der Gaag, Ishita P. Miah, Kuan H. Kho, Heleen M. den Hertog, Hester F. Lingsma, Ruben Dammers, Pathophysiology and Nonsurgical Treatment of Chronic Subdural Hematoma: From Past to Present to Future, World Neurosurgery, Volume 116,2018,Pages 402-411.e2,

VEGF Angiogenic factor upregulated-causes formation of immature/leaky capillaries

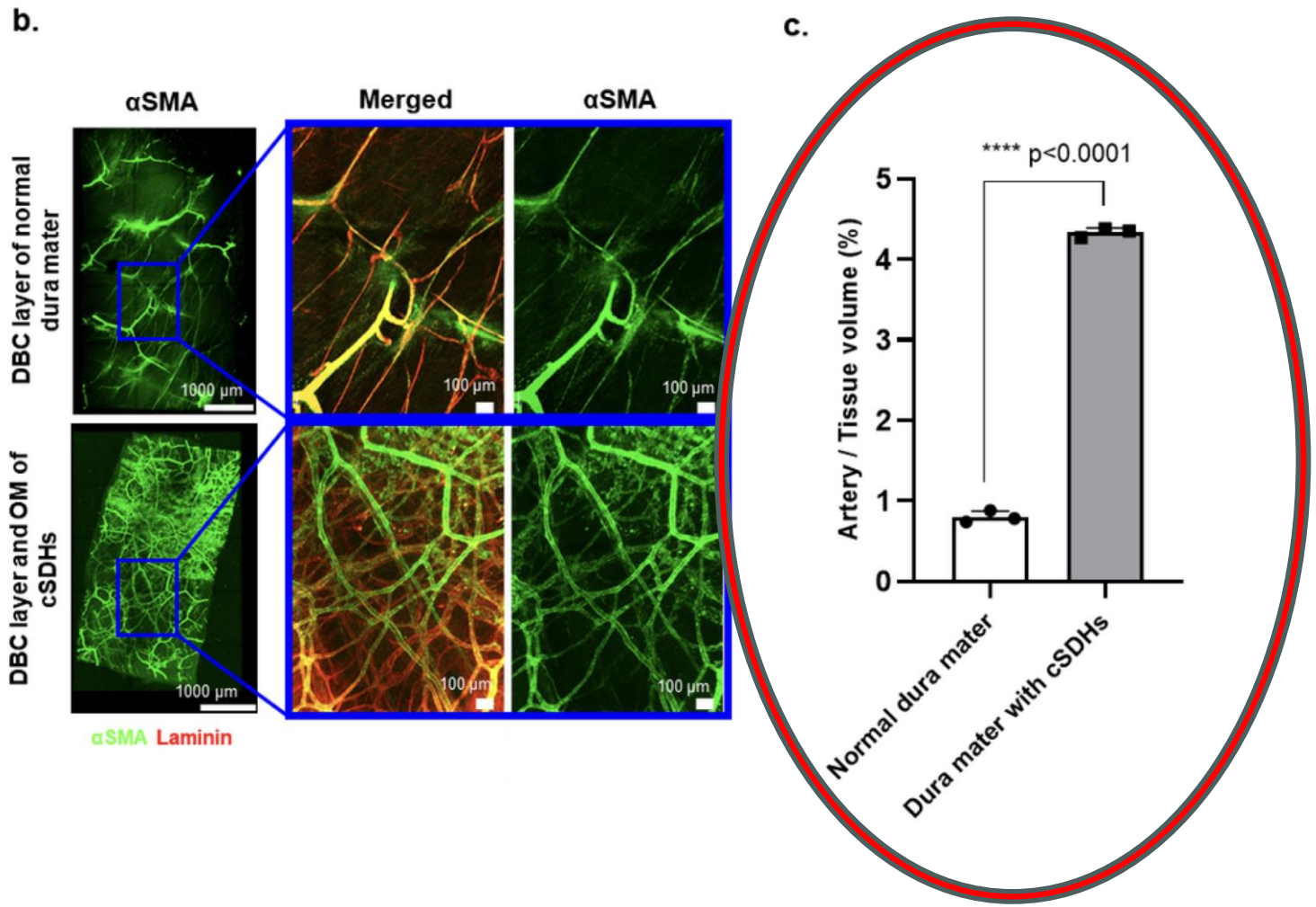


Inc. in leaky capillaries in cSDH membranes and Dural Border Cells DBC



Neovascularization in the outer membrane of chronic subdural hematomas from the dural border cell layer of the dura mater https://doi.org/10.21203/rs.3.rs-1732837/v1

Inc. in leaky capillaries in cSDH membranes



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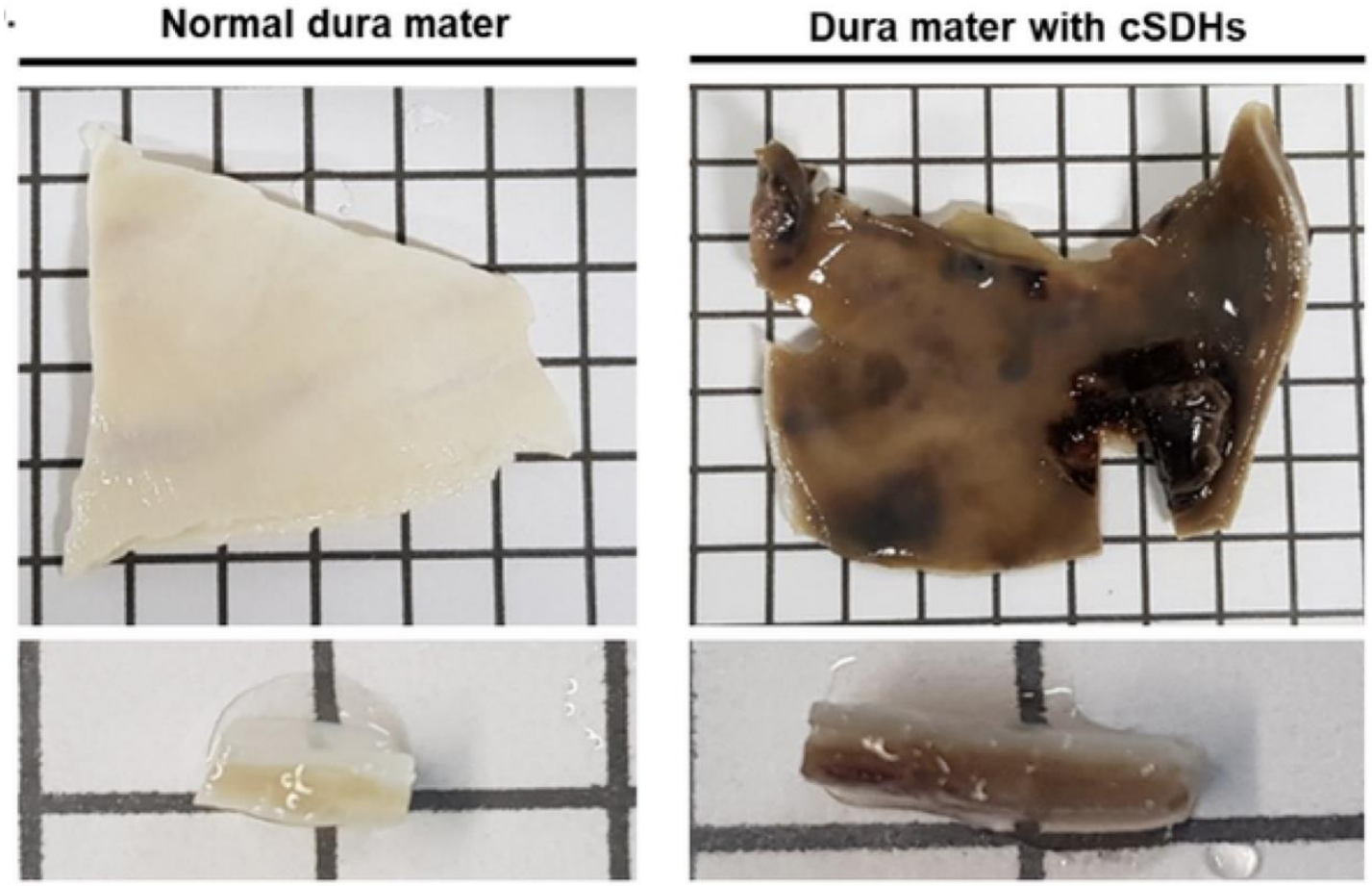
Gross pathology of dura with cSDH



Spontaneous absorption of cSDH Via meningeal lymphatics

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Published: 01 June 2015

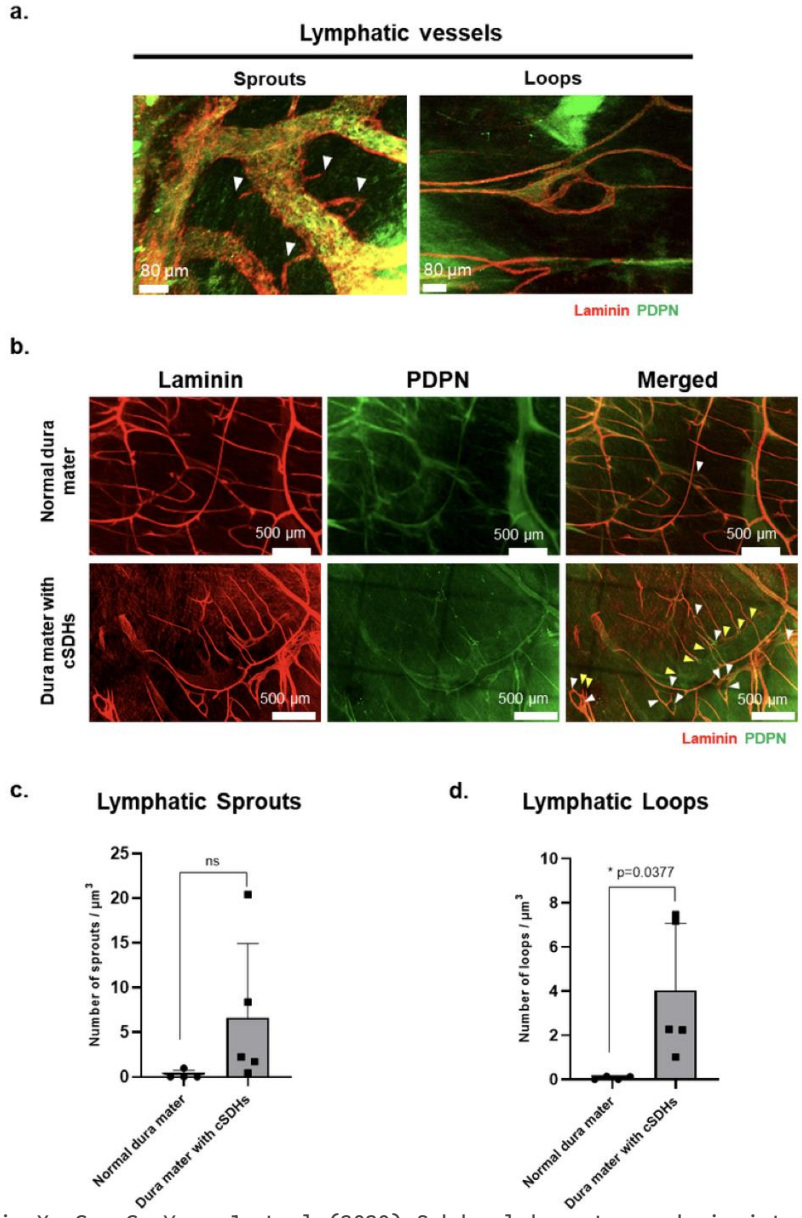
Structural and functional features of central nervous system lymphatic vessels

Antoine Louveau, Igor Smirnov, Timothy J. Keyes, Jacob D. Eccles, Sherin J. Rouhani, J. David Peske, Noel C. Derecki, David Castle, James W. Mandell, Kevin S. Lee, Tajie H. Harris & Jonathan Kipnis

Nature **523**, 337–341 (2015) | Cite this article

209k Accesses | **2575** Citations | **2175** Altmetric | Metrics

In searching for T-cell gateways into and out of the meninges, we discovered functional lymphatic vessels lining the dural sinuses. These structures express all of the molecular hallmarks of lymphatic endothelial cells, are able to carry both fluid and immune cells from the cerebrospinal fluid, and are connected to the deep cervical lymph nodes. The unique location of these vessels may have impeded their discovery to date, thereby contributing to the long-held concept of the absence of lymphatic vasculature in the central nervous system.



Increased in the number of lymphatic vessels in cSDH compared to controls

Liu X, Gao C, Yuan J et al (2020) Subdural haematomas drain into the extracranial lymphatic system through the meningeal lymphatic vessels. Acta Neuropathol Commun Feb 14(1):16. doi:10.1186/s40478-020-0888-y
Neovascularization in the outer membrane of chronic subdural hematomas from the dural border cell layer of the dura mater
https://doi.org/10.21203/rs.3.rs-1732837/v1

Spontaneous absorption of cSDH Via meningeal lymphatics

Subdural haematomas drain into the extracranial lymphatic system through the meningeal lymphatic vessels

Xuanhui Liu, Chuang Gao, Jiangyuan Yuan, Tangtang Xiang, Zhitao Gong, Hongliang Luo, Weiwei Jiang, Yiming Song, Jinhao Huang, Wei Quan, Dong Wang, Ye Tian, Xintong Ge, Ping Lei, Jianning Zhang ✉ & Rongcai Jiang ✉

Acta Neuropathologica Communications **8**, Article number: 16 (2020) | Cite this article

- Animal study showed that hematomas drained out of the subdural space through the meningeal lymphatic vessels and lymphatic dysfunction caused by **meningeal lymphatic ligation led to the attenuation of SDH resorption**

Liu X, Gao C, Yuan J et al (2020) Subdural haematomas drain into the extracranial lymphatic system through the meningeal lymphatic vessels. Acta Neuropathol Commun Feb 14(1):16. doi:10.1186/s40478-020-0888-y

Natural course of chronic SDH determined by the balance of plasma effusion and/or rebleeding from the neo-membranes on one hand and reabsorption of fluid on the other

Plasma effusion and/or rebleeding from the neo-membranes

reabsorption of fluid By membranes,

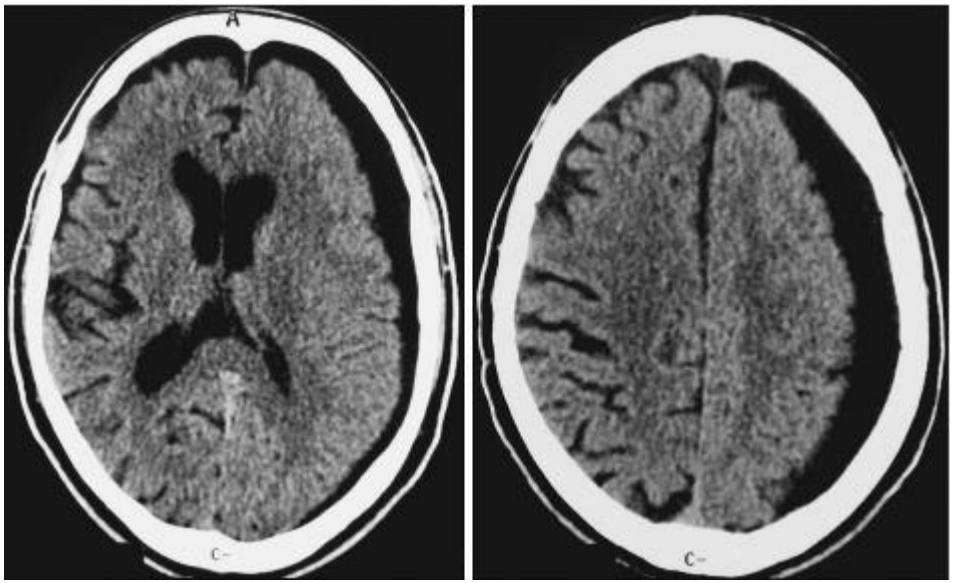
meningeal veins and

lymphatic system

The meningeal lymphatic system is known to be involved in passing immune cells and draining waste from the brain to the deep cervical lymph nodes

Louveau A, Smirnov I, Keyes TJ et al (2015) Structural and functional features of central nervous system lymphatic

Should we treat Subdural hygromas?



- Incidence of cSDH following hygroma as high as 58%
- Bilateral hygromas and male sex found to be predictors of conversion to cSDH

K.S. Lee, W.K. Bae, H.G. Bae, I.G. Yun The fate of traumatic subdural hygroma in serial computed tomographic scans J. Kor. Med. Sci, 15 (5) (2000), pp. 560-568
J.H. Ahn, H.S. Jun, J.H. Kim, J.K. Oh, J.H. Song, I.B. Chang

Analysis of risk factor for the development of chronic subdural hematoma in patients with traumatic subdural hygroma J. Kor. Neurosurg.Soc, 59 (6) (2016), p. 622

Akhaddar, A. (2021). Neuroimaging Differential Diagnosis (Imaging Mimicking Conditions) of Cranial Chronic Subdural Hematoma. In: Turgut, M., Akhaddar, A., Hall, W.A., Turgut, A.T. (eds) Subdural Hematoma. Springer, Cham. https://doi.org/10.1007/978-3-030-79371-5_26

Decision to treat

- Patients presenting symptoms
- Size of hematoma
- Associated mass effect
- Patients overall health co-morbidities

- Small cSDH- Manage conservatively
- Large lesions with mass effect/ midline shift or neuroligcal deterioration – surgery (crani of burr holes)

Management

- Seizure prophylaxis
- Reversal of anticoagulation (not needed in MMAe)
- Surgical Treatment (burr holes or craniotomy) if:

1. Symptomatic lesion
2. Mass effect (thickness(>1cm)
3. Midline shift >3-4mm
4. Progressive increase in size in serial CT scans

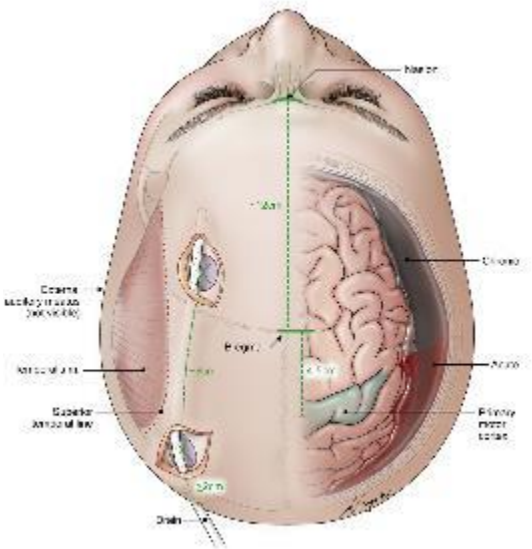
Surgical drainage decreases local VEGF

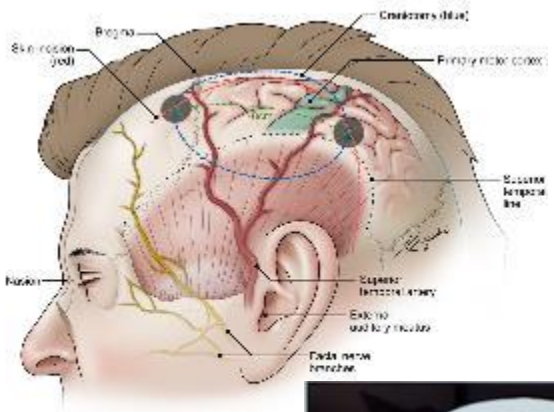
Dana C. Holl, Victor Volovici, Clemens M.F. Dirven, Wilco C. Peul, Fop van Kooten, Korné Jellema, Niels A. van der Gaag, Ishita P. Miah, Kuan H. Kho, Heleen M. den Hertog, Hester F. Lingsma, Ruben Dammers, Pathophysiology and Nonsurgical Treatment of Chronic Subdural Hematoma: From Past to Present to Future, World Neurosurgery, Volume 116,2018,Pages 402-411.e2,

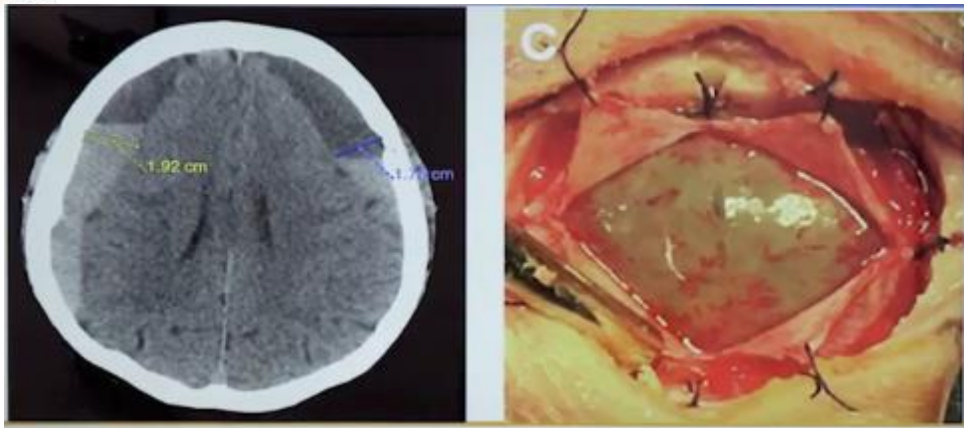
Shono T, Inamura T, Morioka T, Matsumoto K, Suzuki SO, Ikezaki K, et al. Vascular endothelial growth factor in chronic subdural haematomas. J Clin Neurosci. 2001;8(5):411–5.
https://doi.org/10.1016/j.bas.2021.100300

Surgical drainage of cSDH with burr holes

- Surgical drainage –
  - decreases subdural hematoma volume.
  - reduce the amount of VEGF in the hematoma cavity and therefore alleviate the adverse effects of increased stimulation







MMA embolization" An emerging treatment technique for cSDH: Dr. J.Johnson, G. Colby- UCLA

Shono T, Inamura T, Morioka T, Matsumoto K, Suzuki SO, Ikezaki K, et al. Vascular endothelial growth factor in chronic subdural haematomas. J Clin Neurosci. 2001;8(5):411–5.
https://doi.org/10.1016/j.bas.2021.100300

Surgical drainage of cSDH (Burr hole or Craniotomy)

- Problems-
  - Maximally invasive - psychologically terrifying
  - Doesn't address production of subdural fluid, therefore reoccurrence.
  - Reoccurence rates- 20-26% (symptomatic or asymptomatic)
  - Operation related Mortality 1.5%- 6% - acute SDH during or after surgery of cSDH
  - Other complications- infections, seizures, pneumocephalus
  - Blood thinners have to be held
  - seizures

Dana C. Holl, Victor Volovici, Clemens M.F. Dirven, Wilco C. Peul, Fop van Kooten, Korné Jellema, Niels A. van der Gaag, Ishita P. Miah, Kuan H. Kho, Heleen M. den Hertog, Hester F. Lingsma, Ruben Dammers, Pathophysiology and Nonsurgical Treatment of Chronic Subdural Hematoma: From Past to Present to Future, World Neurosurgery, Volume 116,2018,Pages 402-411.e2,

Shono T, Inamura T, Morioka T, Matsumoto K, Suzuki SO, Ikezaki K, et al. Vascular endothelial growth factor in chronic subdural haematomas. J Clin Neurosci. 2001;8(5):411–5.
https://doi.org/10.1016/j.bas.2021.100300

Middle Meningeal Artery Embolization

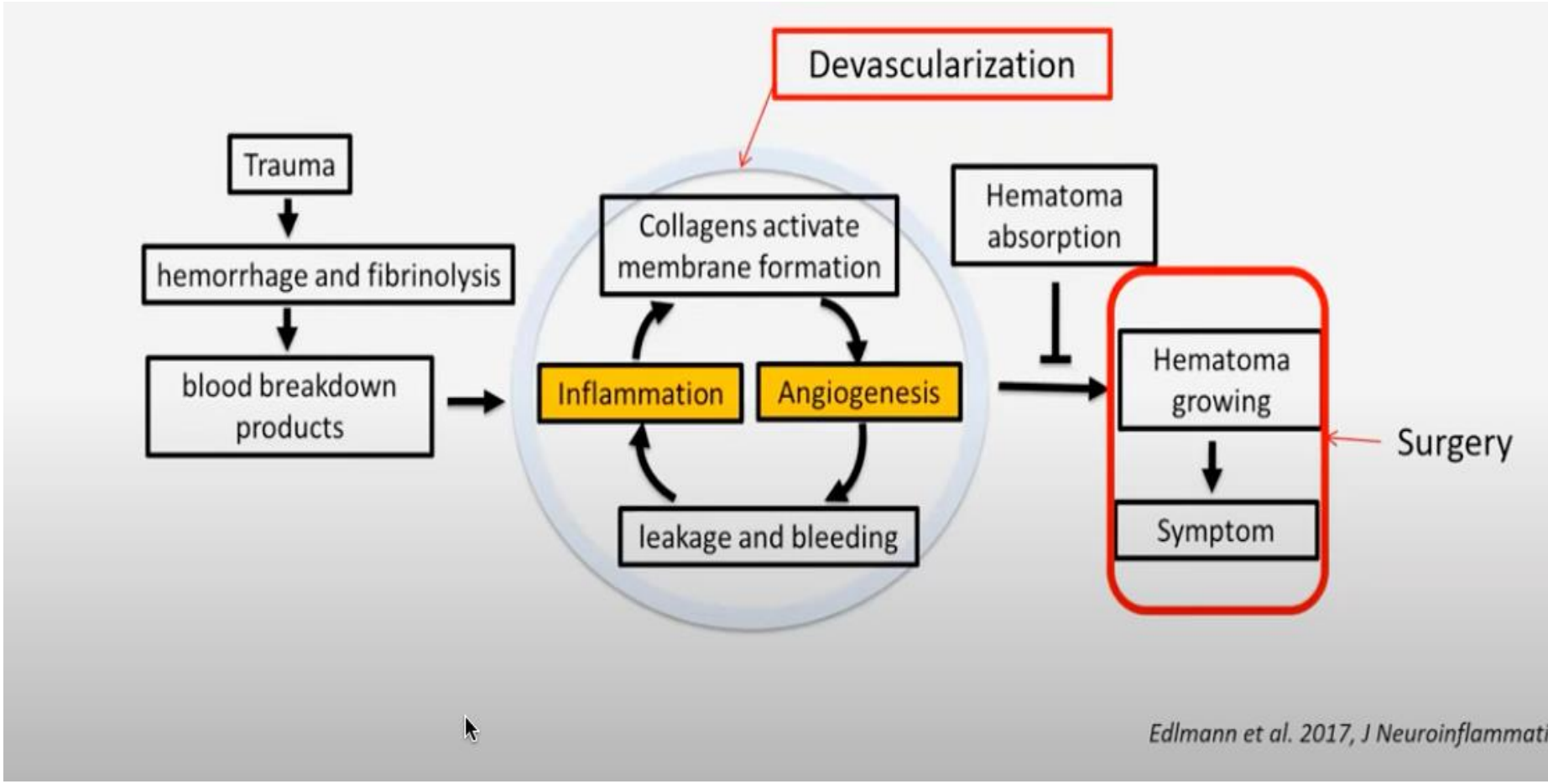
- Neovascularization supplied by branches on MMA.
- MMAe devascularizes dura
- The only procedure that addresses the ‘cause’
- Embolization helps decrease Inflammatory response of dural layer- promotes neo-vascularization / bleeding granulation tissue
- Stand alone or with burr holes
- I do both only if local mass effect from hematoma change of neurological exam

Ban SP, Hwang G, Byoun HS, et al. Middle meningeal artery embolization for chronic subdural hematoma. Radiology. 2018;286(3):992-999.
(25.) Kim E. Embolization therapy for refractory hemorrhage in patients with chronic subdural hematomas. World Neurosurg. 2017;101:520-527 (doi:10.1016/j.wneu.2017.02.070).

Advantages of MMA embo

- Treating the underlying cause: reduces recurrence
- Minimally invasive
- Mild sedation(MAC) can be used
- Can continue antiplatelet and anticoagulant therapy
- Rate of hematoma absorption appears to accelerate with MMAe

Rationale for MMA Embolization



MMA Embolization indications

- Primary MMA embolization- (no surgical evacuation) mildly symtomatic
- Rescue MMAe (recurrence after surgical evacuation)
- Adjunct MMAe (combined surgical and MMAe)
- Patients who cannot come off blood thinners / not candidates for surgery
- Asymptomatic patients with moderate size SDH

Case Reports > J Neurosurg. 2000 Oct;93(4):686-8. doi: 10.3171/jns.2000.93.4.0686.

Middle meningeal artery embolization for refractory chronic subdural hematoma. Case report

S Mandai [1], M Sakurai, Y Matsumoto

Affiliations + expand

PMID: 11014549 DOI: 10.3171/jns.2000.93.4.0686

Abstract

MMAe Surgical procedure

- General Anesthesia or conscious sedation
- Groin or Radial

- 5 or 6 French guide system
- Intermediate catheter- CAT 5,Phenom plus
- Scepter mini balloon / Balt Eclipse Balloon
- Marathon microcatheter / Prowler select plus
- Note physiological anastomosis / Collaterals
- Define danger areas
  - eg ophthalmic/ retinal collaterals- vision loss
  - Petrosal branch- Facial palsy

MMAe Surgical procedure

- Goal- devascularize both branches of MMA separately
- General consensus- penetrate as deep as possible and as many branches distally as possible
- Not clear if proximal occlusion is enough
- Try to treat atleast one dominant branch based on location of SDH
- Can do other branch proximal occlusion if cannot embolize other branch because of danger zone

MMAE Contra-indications

- Anatomical variance
- Danger zones- ophthalmic, petrosal
- MMA arising from ophthalmic artery
- MMA also giving perfusion to normal brain ie Moya Moya
- Inaccessibility of MMA-
  - Prior craniotomy with STA sacrifice
  - Carotid stent

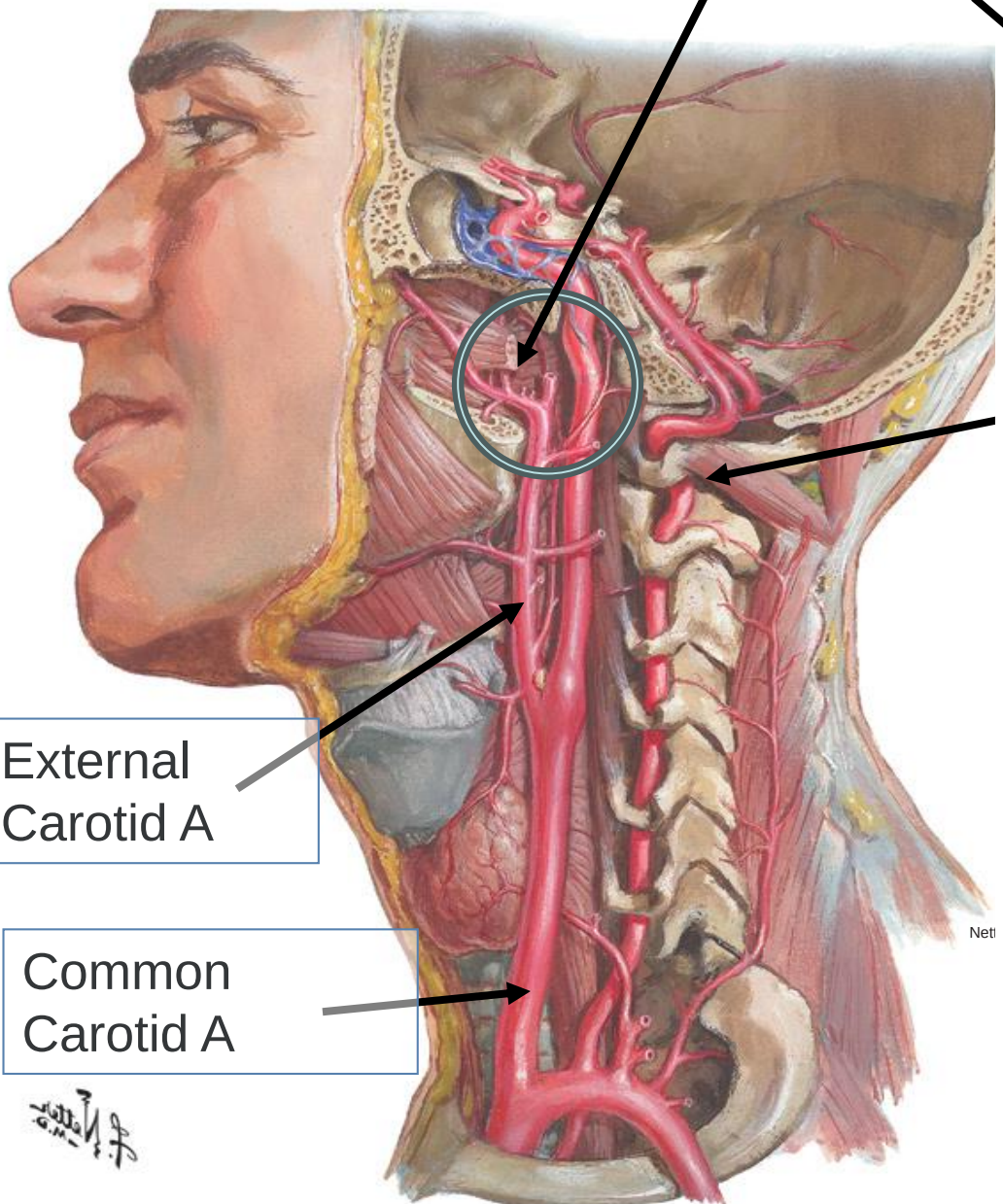
Choice of embolization agents- no consensus

- Liquid embolic agents- Onyx, n-BCA Glue
  - Advantage- relatively controlled with balloon, seen well
  - Disadvantage- can still penetrate unwanted collaterals
  - High Cost, DMSO painful, costly
  - Painful- general anesthesia

- PVA particles- (+/- coils)
  - Advantage- Deep Penetration, Low cost
  - Disadvantage- get clump at bifurcation, inadvertent reflux to collaterals, Recanalization.
  - Difficult to see

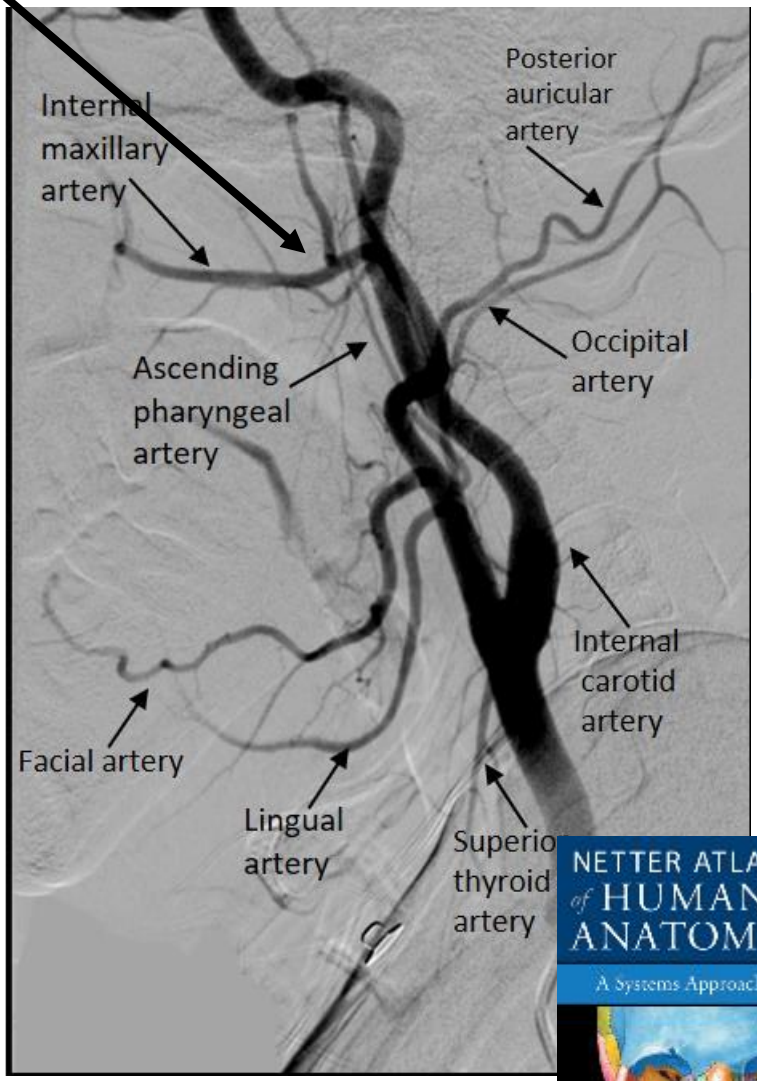
- Coils-
  - Advantage- may eliminate the risk of visual or facial nerve compromise. Fast,
  - Disadvantage- No deep penetration- recanalization by collaterals in dura

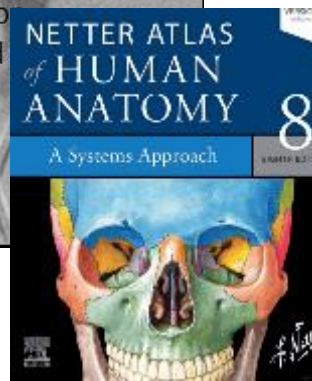
MMA- Branch of IMAX from ECA



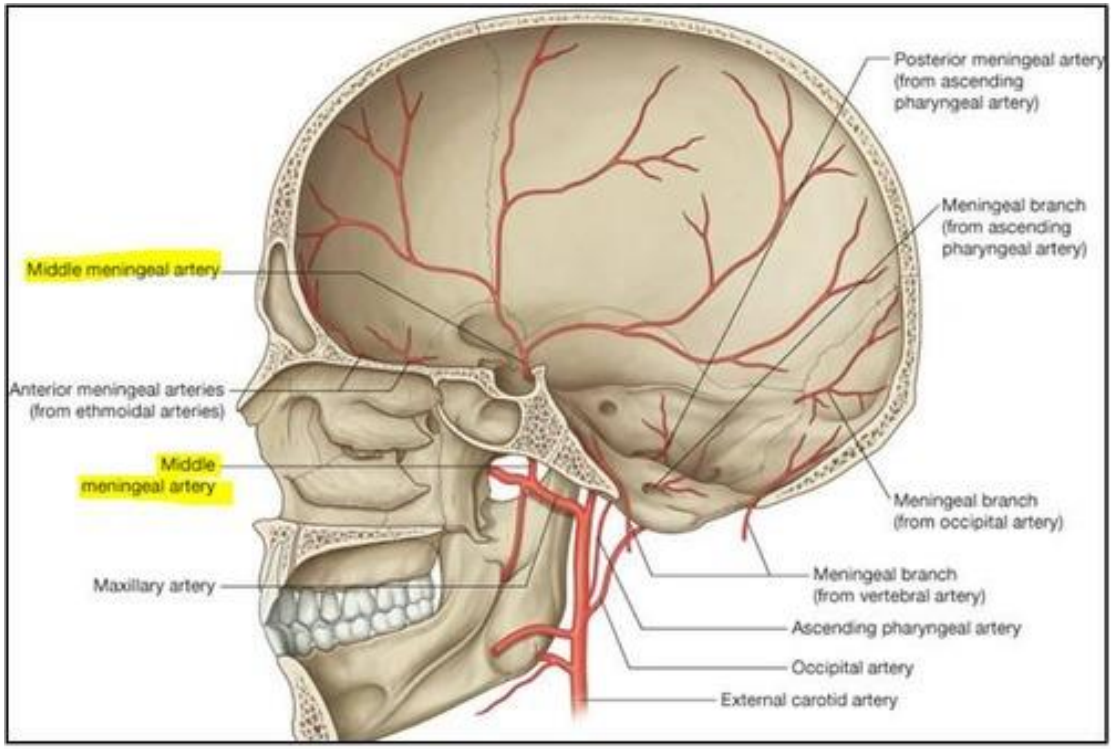
External Carotid A

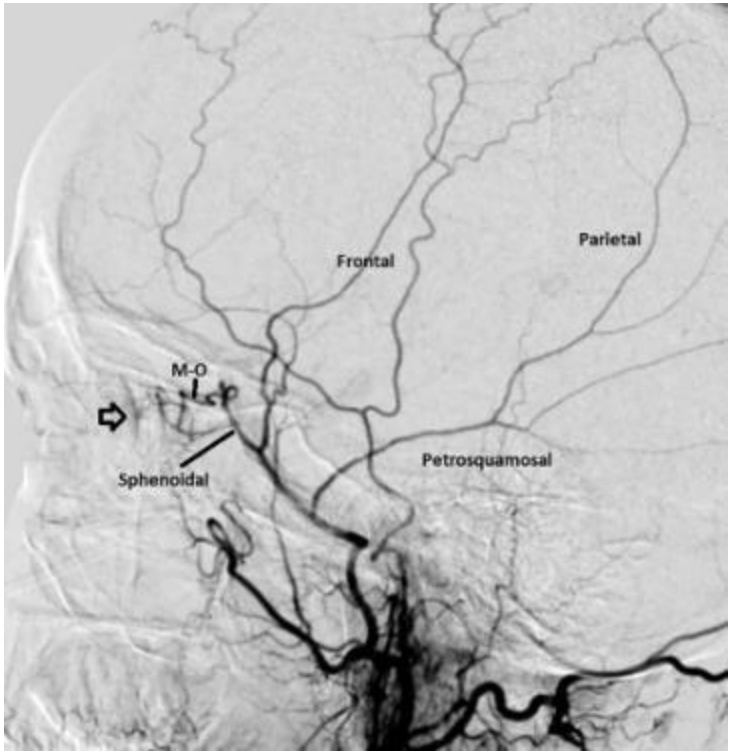
Common Carotid A





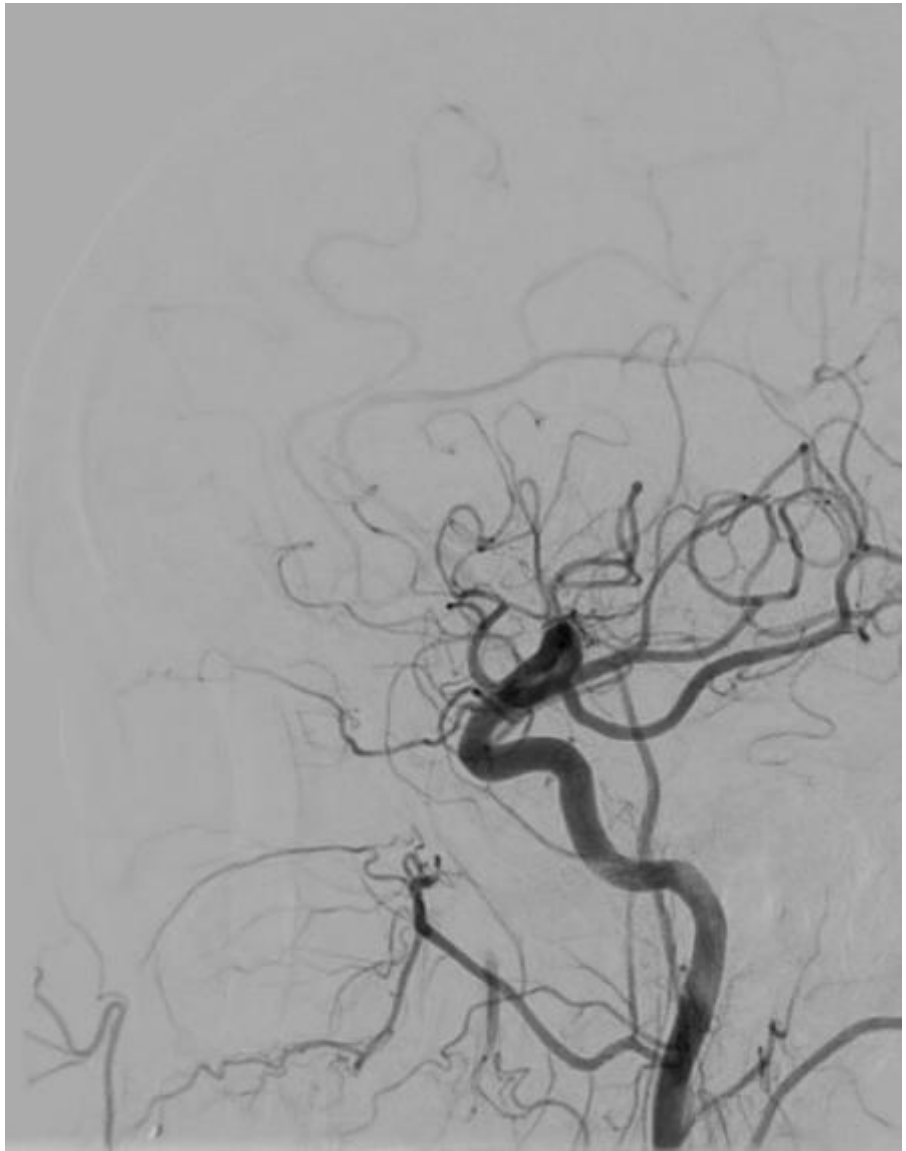
Anatomy of MMA
MMA can have variable anatomy





- Orbital/ Meningo-lacrimal branch: potential dangerous anastomoses with ophthalmic artery- The microcatheter tip must be navigated past this branch distally into the frontoparietal branch and avoiding reflux into the branch
- Petrosal branch: supplies the vasa nervorum of the cranial nerve VII within the petrous bone; navigate the microcatheter tip beyond this branch and avoid reflux into it

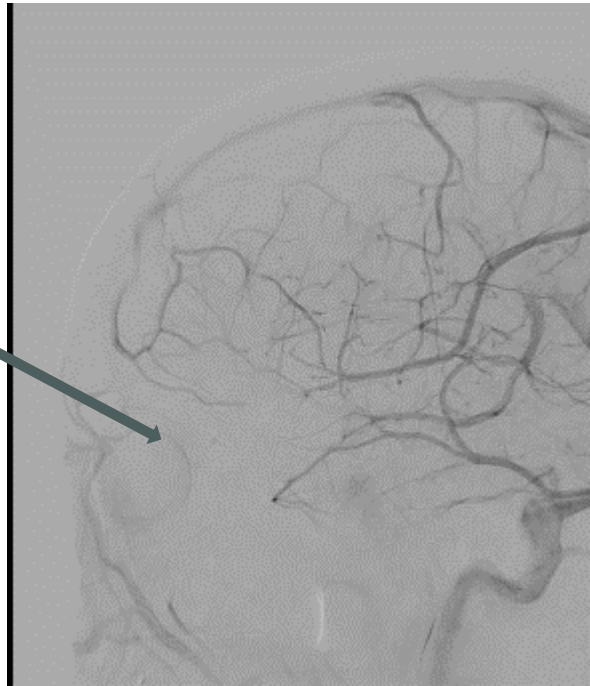
Common Carotid Cranial Run

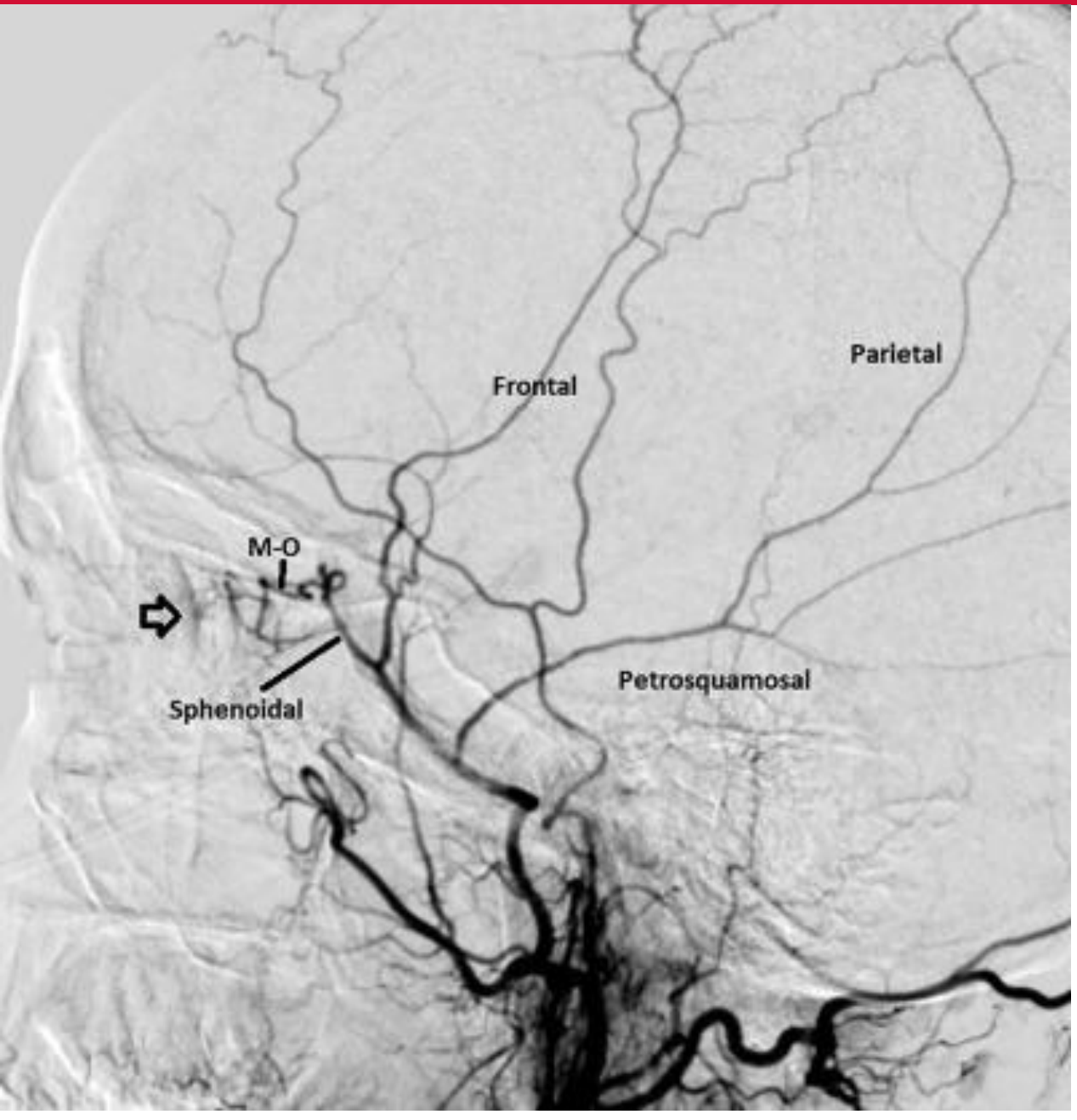


Establish baseline to compare pre and post MMAe

Identify areas of physiological anastomosis

Retinal /Choroidal Blush





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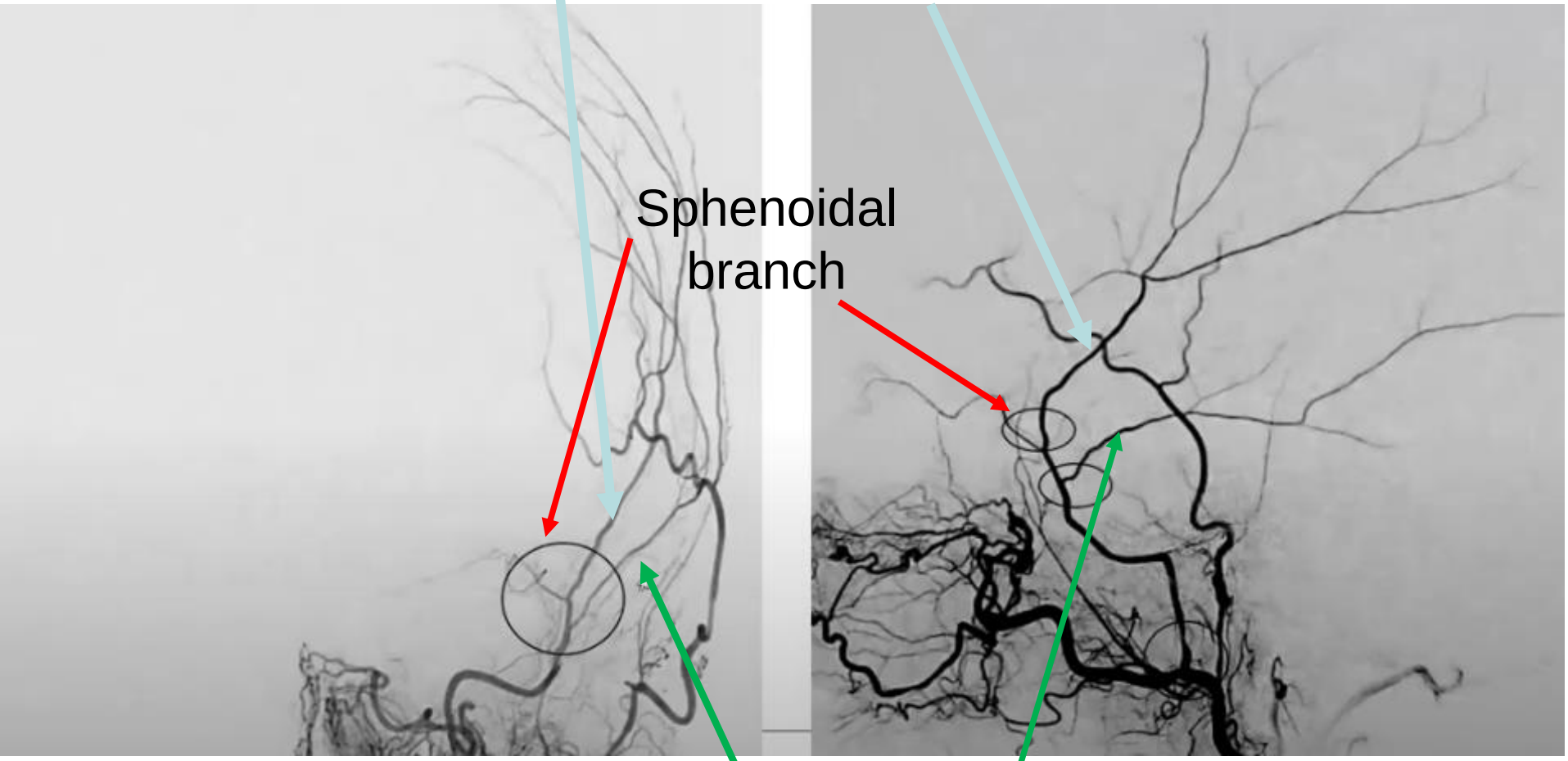
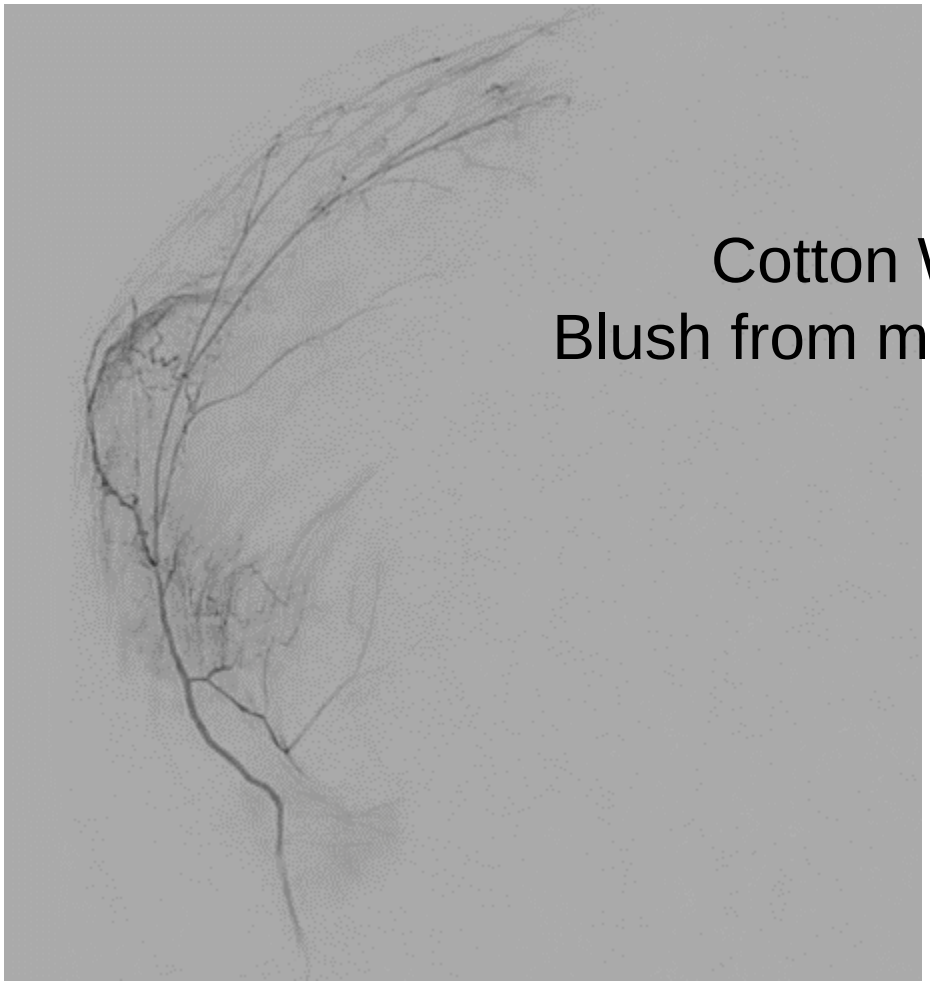
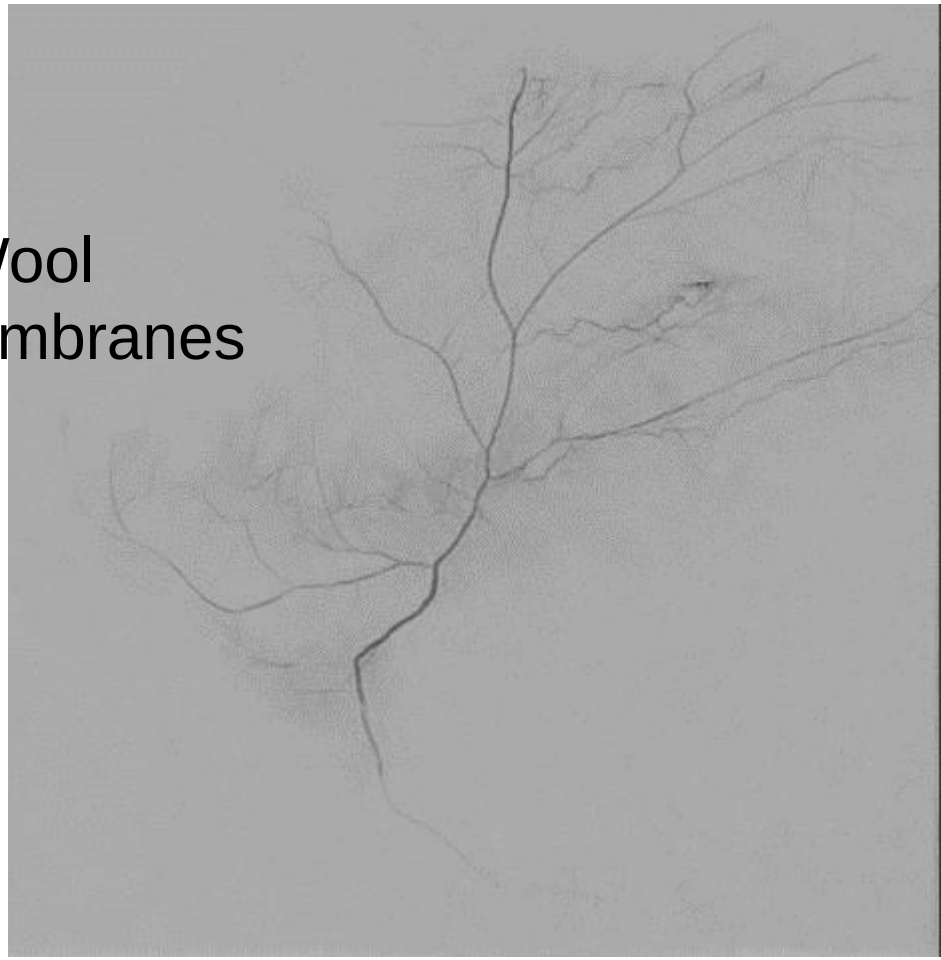
- Petrosal branch: supplies the vasa nervorum of the cranial nerve VII within the petrous bone; navigate the microcatheter tip beyond this branch and avoid reflux into it


Anterior frontal branch
Sphenoidal branch
Posterior parietal branch

MMA injection with contrast



Cotton Wool
Blush from membranes



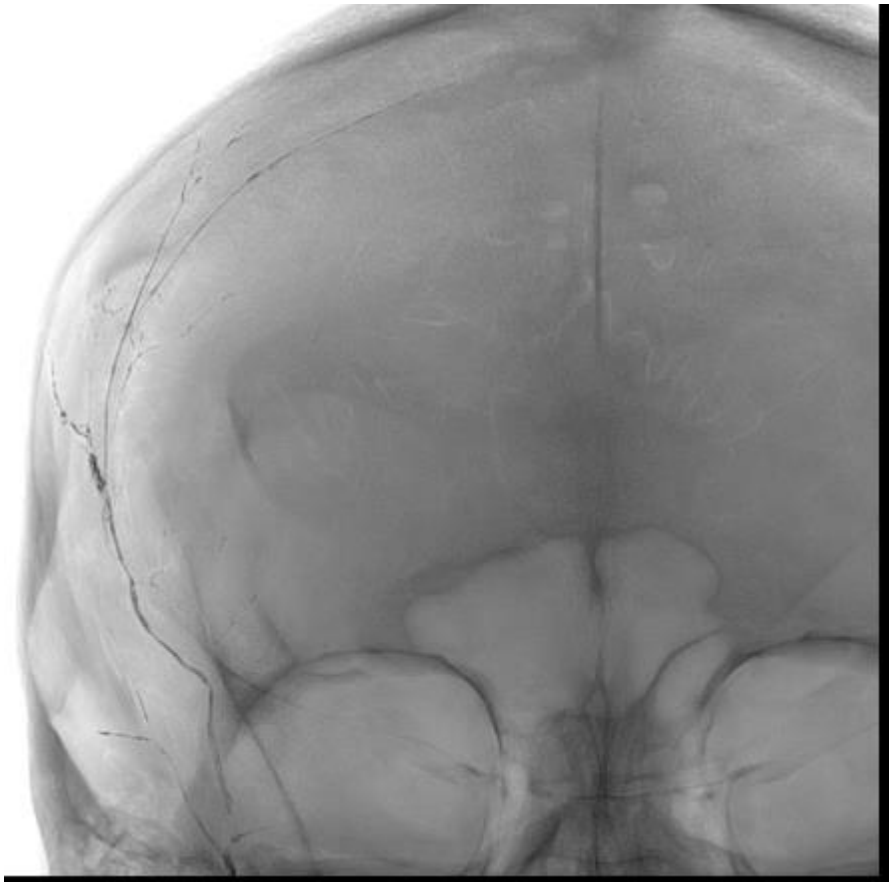
AP view

Lateral

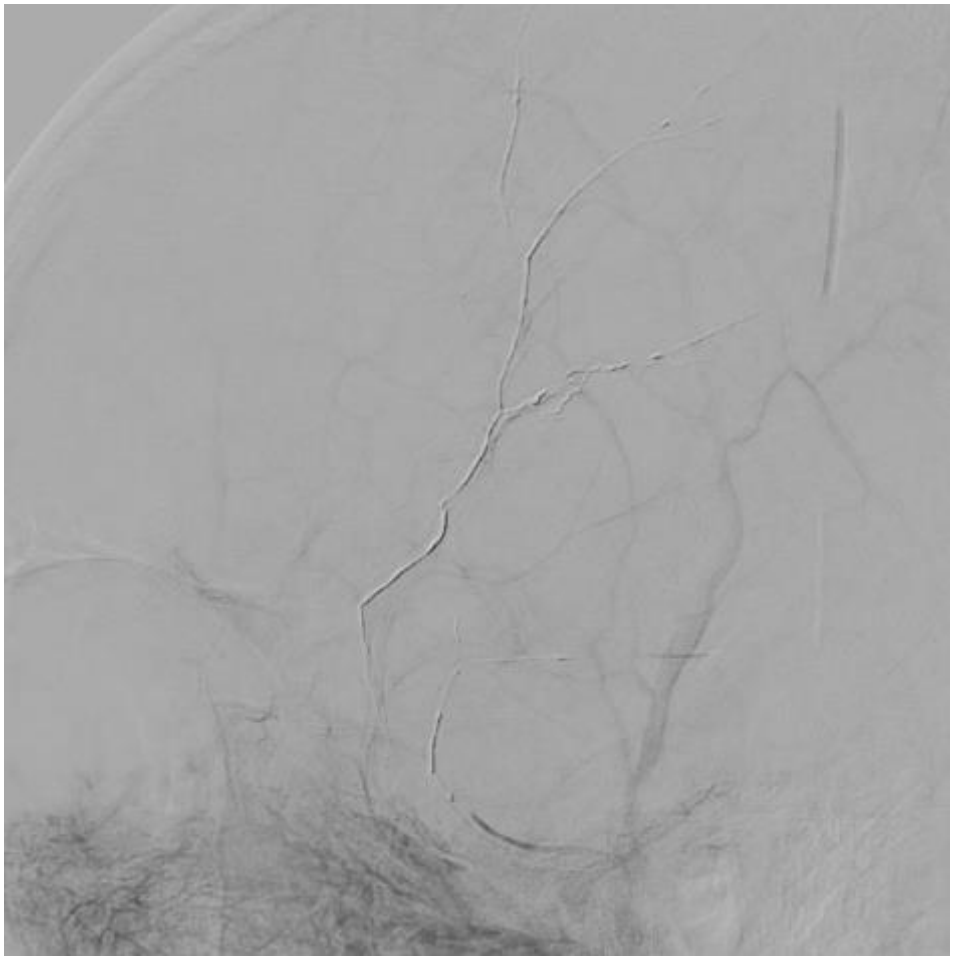
Patient with post SDH. Distal Parietal branch MMAe

Microcatheter distal to avoid

Post Embo Onyx cast

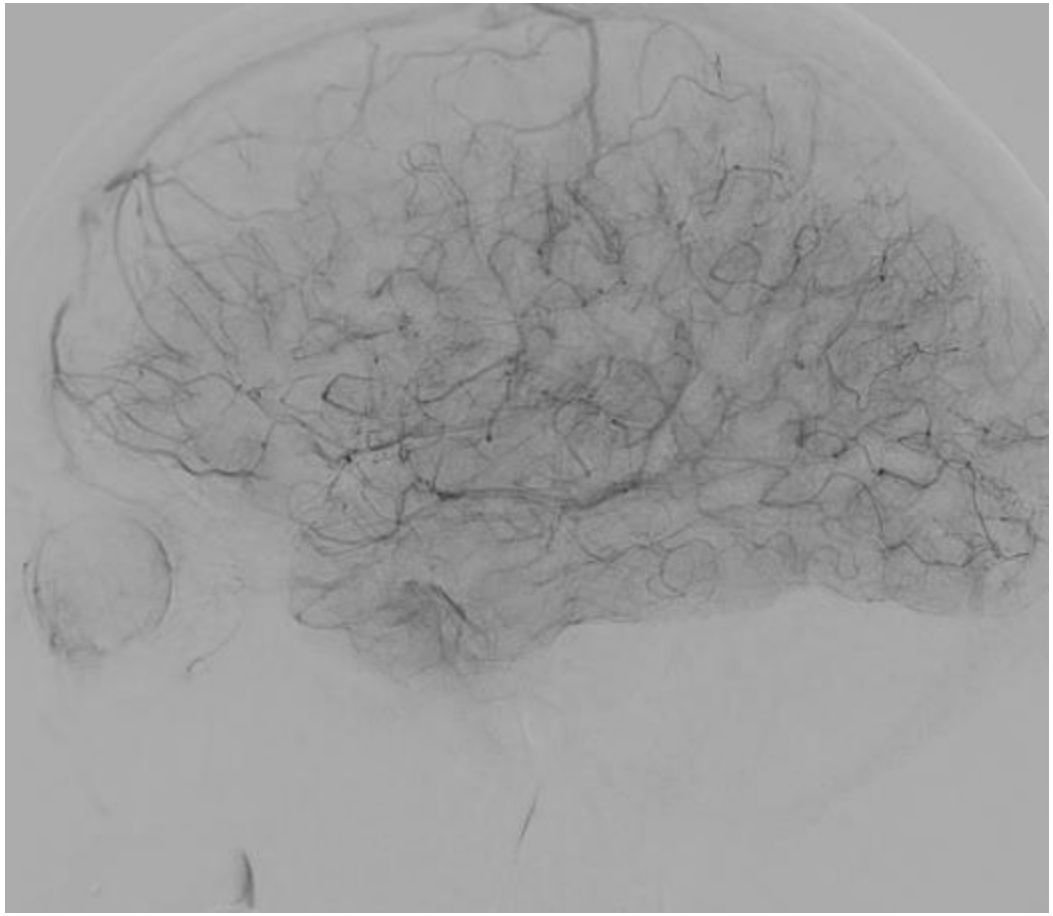


Post MMAe

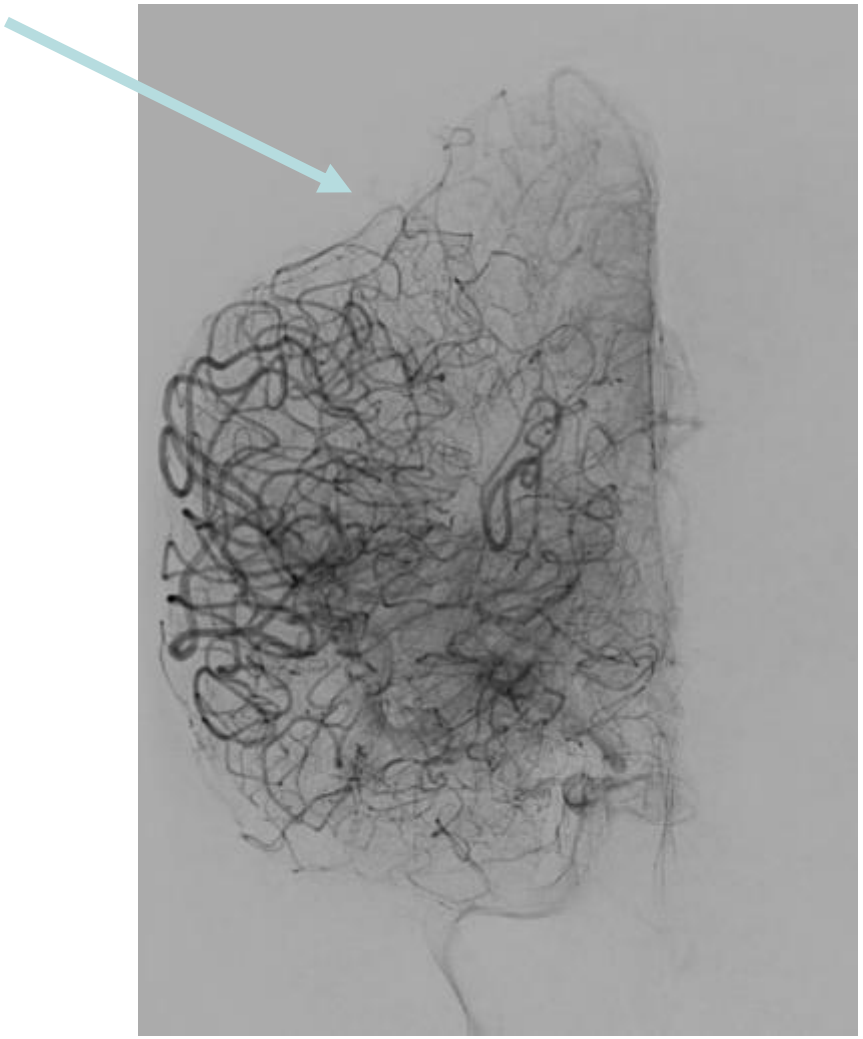


Stump of MMA. No more contrast blush for membranes

Post MMAe- Choroidal / Retinal blush



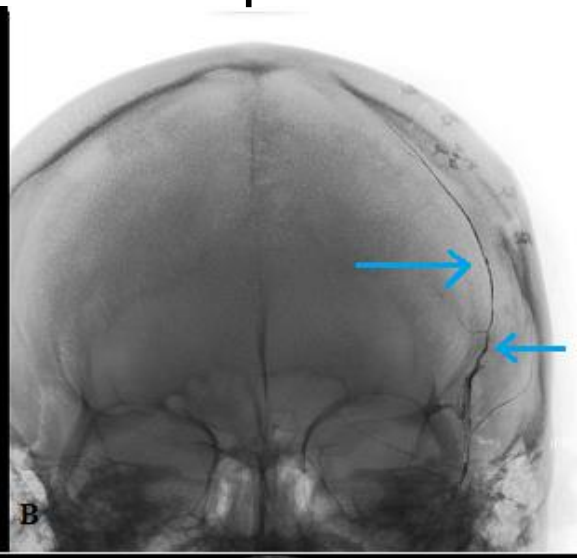
Mass effect on cerebral convexity



Right ICA run

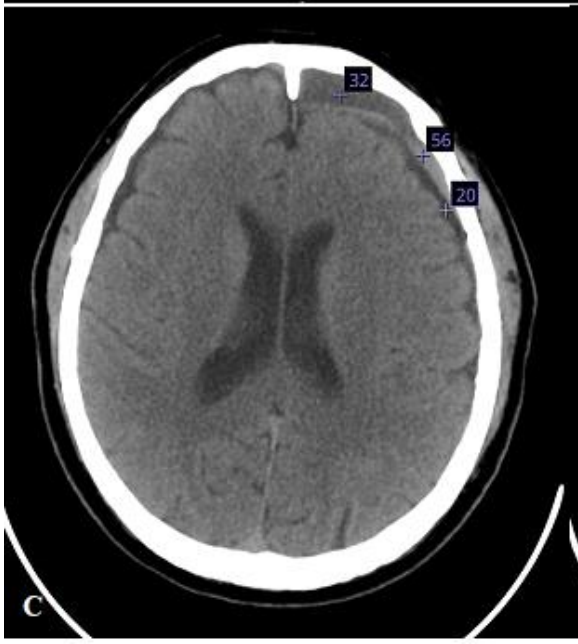
60 male cSDH recurrence post craniotomy

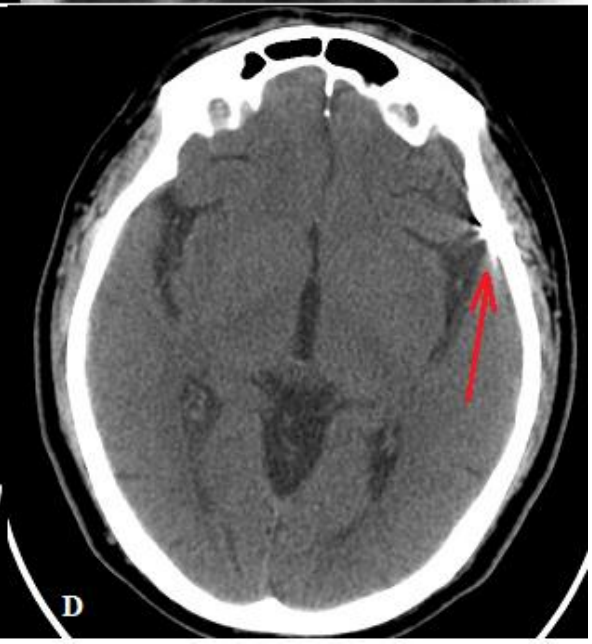
Preop



Onyx cast

1month post op

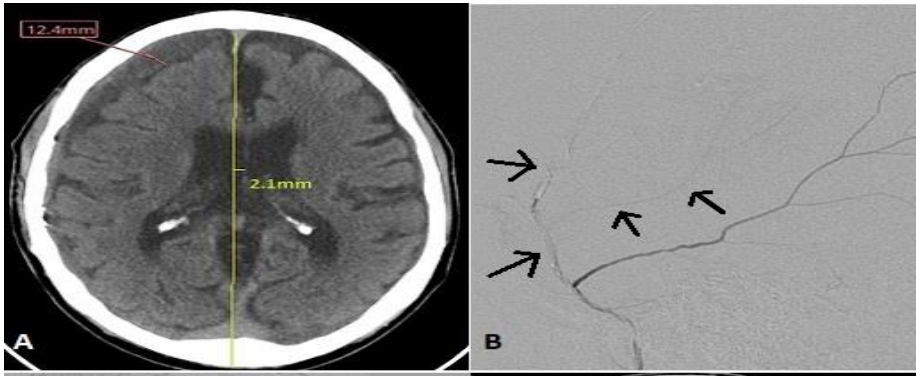




3 month postop

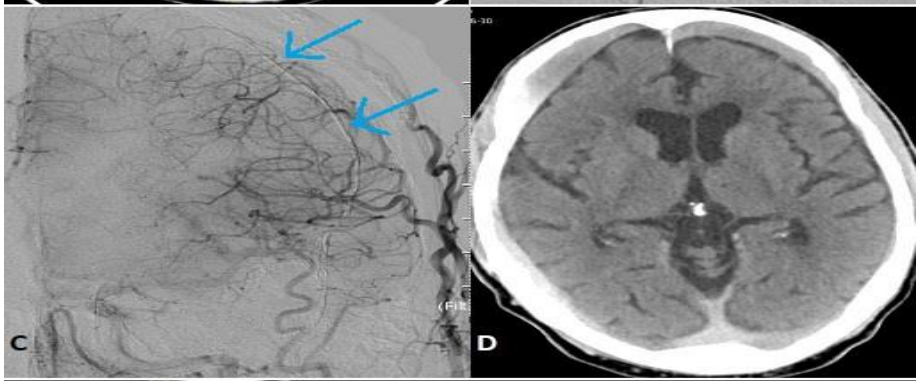
Does everyone have a good result?

Preop 14 mm



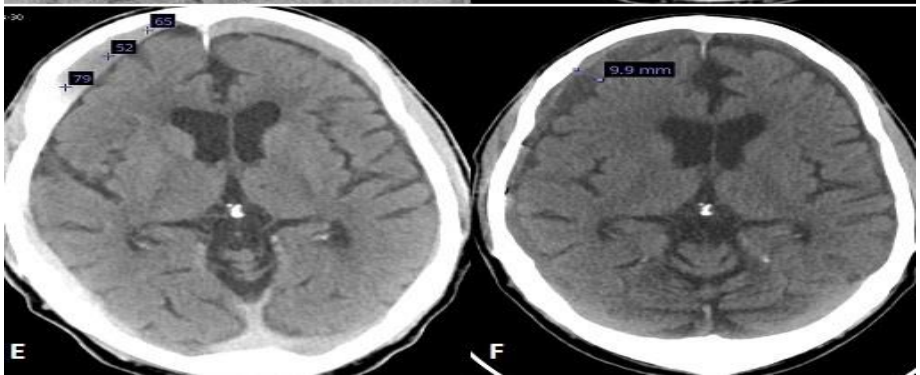
Intraop

Immediate post embo image with. onyx



24 Hrs postop

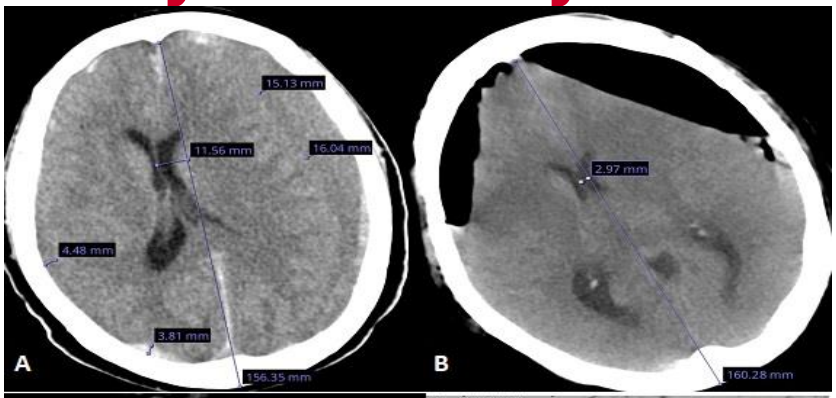
24 hrs postop



3month post op 9.9 mm

Recurrence-Proximal occlusion by Coils only by Dr. Roychowdhury

1st Preop

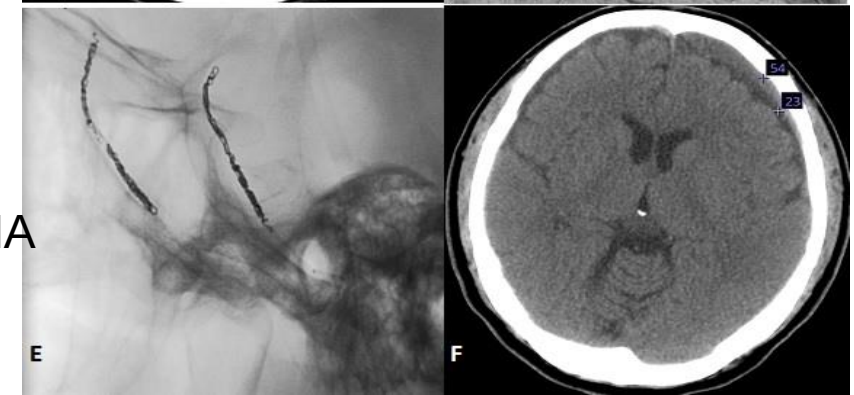


Post-surgical Evacuation

Recurrent cSDH

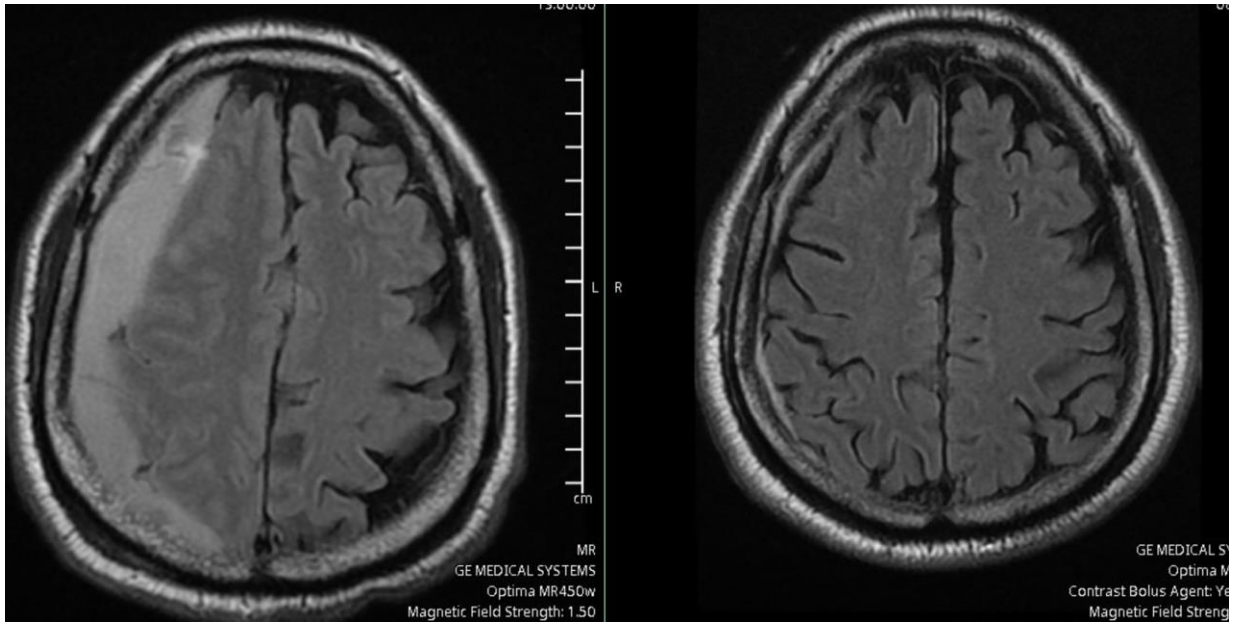
Intra-op Embolization image

Coils for bilateral MMA



3-month post op

Pre and 3 month post MRI
Burr Holes and MMAe



Follow up imaging post MMAe

- CT Head no contrast baseline study
- One month MRI brain with and without contrast
- 3 months MRI brain with and without

RESEARCH—HUMAN—CLINICAL STUDIES

Middle Meningeal Artery Embolization for Chronic Subdural Hematoma: A Multi-Center Experience of 154 Consecutive Embolizations

Kan, Peter MD, MPH, FRCS; Maragkos, Georgios A MD; Srivatsan, Aditya MS; Srinivasan, Visish MD; Johnson, Jeremiah MD; Burkhardt, Jan-Karl MD; Robinson, Timothy M MD; Salem, Mohamed M MD; Chen, Stephen MD; Riina, Howard A MD; Tanweer, Omar MD; Levy, Elad I MD; Spiotta, Alejandro M MD; Kasab, Sami Al MD; Lena, Jonathan MD; Gross, Bradley A MD; Cherian, Jacob MD; Cawley, C Michael MD; Howard, Brian M MD; Khalessi, Alexander A MD; Pandey, Aditya S MD; Ringer, Andrew J MD; Hanel, Ricardo MD, PhD; Ortiz, Rafael A MD; Langer, David MD; Kelly, Cory M MS; Jankowitz, Brian T MD; Ogilvy, Christopher S MD; Moore, Justin M MD, PhD; Levitt, Michael R MD; Binning, Mandy MD; Grandhi, Ramesh MD, MS; Siddiq, Farhan MD; Thomas, Ajith J MD

Author Information

Neurosurgery 88(2):p 268-277. February 2021. | *DOI:* 10.1093/neuros/nvaa379

138 patients- 154 embolizations
30% on anti-platelets 24% anti-coag
47% - general anesthesia
70%- PVA particles

15 underwent bilateral MMAe
Mean SDH thickness = 14mm

25% liquid emboli- (no difference in outcomes)

On follow up- median residual thickness = 4 mm

71% patients had > 50% improvement on imaging

32% improved clinically
Complications – 11%

7 % required further treatment
Mortality 4.4% (not related to procedure)

New devices and techniques
Review

Middle meningeal artery embolization for chronic subdural hematoma: a systematic review and meta-analysis

Natasha Ironside [1], Candice Nguyen [2], Quan Do [3], Beatrice Ugiliweneza [2], Ching-Jen Chen [1], Emily P Sieg [2], Robert F James [4], Dale Ding [2]

Correspondence to Dr Dale Ding, Department of Neurological Surgery, University of Louisville School of Medicine, Louisville, KY 40202, USA; daleding1234@gmail.com

Meta-analysis Comparing MMAe with conventional mgmt.

20 studies 1416 patients : 718 MMAe 698 Conventional

Results

- - MMAe associated with lower cSDH recurrence (OR=0.15 (95% CI 0.03 TO 0.75), P=0.02
- Lower surgical rescue(or= 0.21(0.07 to 0.58), p=0.003
- In-hospital complication rates comparable b/w both (OR=0.78(0.34 TO 1.76), P=0.55

AJNR
AMERICAN JOURNAL OF NEURORADIOLOGY

Research Article | Interventional

Embolic Agent Choice in Middle Meningeal Artery Embolization as Primary or Adjunct Treatment for Chronic Subdural Hematoma: A Systematic Review and Meta-analysis

J.C. Ku, A.A. Dmytriw, M.A. Essibayi, M.A. Banihashemi, J.E. Vranic, S. Ghozy, D. Altschul, R.W. Regenhardt, C.J. Stapleton, V.X.D. Yang and A.B. Patel

American Journal of Neuroradiology March 2023, 44 (3) 297-302; DOI: https://doi.org/10.3174/ajnr.A7796

DATA SYNTHESIS: Twenty-two studies were included with 382 patients with middle meningeal artery embolization and 1373 surgical patients. The rate of subdural hematoma recurrence was 4.1%. Fifty (4.2%) patients underwent a reoperation for a recurrent or residual subdural hematoma. Thirty-six (2.6%) experienced postoperative complications. The rates of good radiologic and clinical outcomes were 83.1% and 73.3%, respectively. Middle meningeal artery embolization was significantly associated with decreased odds of subdural hematoma reoperation (OR = 0.48; 95% CI, 23.4–99.1; *P* = .047) compared with surgery. The lowest rates of subdural hematoma radiologic recurrence, reoperation, and complications were observed among patients receiving embolization with Onyx, whereas good overall clinical outcome occurred most commonly with combined polyvinyl alcohol and coils.

LIMITATIONS: A limitation was the retrospective design of studies included.

CONCLUSIONS: Middle meningeal artery embolization is safe and effective, either as a primary or adjunctive treatment. Treatment using Onyx seems to yield lower rates of recurrence, rescue operation, and complications whereas particles and coils produce good overall clinical outcomes.

Meta-analysis

Onyx- Lowest rate of recurrence, reoperation and complications

PVA + Coils good too, but not as good as Onyx

What about n BCA glue as embo agent

> Neurosurgery. 2022 May 1;90(5):533-537. doi: 10.1227/neu.0000000000001882.

Middle Meningeal Artery Embolization for Chronic Subdural Hematoma Using N-Butyl Cyanoacrylate With D5W Push Technique

Shahram Majidi [1], Stavros Matsoukas, Reade A De Leacy, Peter F Morgenstern, Ria Soni, Hazem Shoirah, Benjamin I Rapoport, Tomoyoshi Shigematsu, Joshua B Bederson, Alejandro Berenstein, J Mocco, Johanna T Fifi, Christopher P Kellner

Affiliations + expand

PMID: 35225245 DOI: 10.1227/neu.0000000000001882

Results: A total of 61 patients were identified with a mean (±standard deviation) age of 62.5 ± 9 years. In 6 patients (10%), coil embolization of the origin of the frontal or posterior branch was performed because super-selective catheterization of the branch was unsuccessful because of tortuous anatomy. Complete obliteration of frontal and posterior branches was achieved in 100% of the cases. Recurrent SDH was seen in 3 patients (5%). No incidence of cranial nerve palsy, vision loss, or stroke occurred. One patient suffered a transient neurological deficit.

Conclusion: MMA embolization using diluted n-BCA with concomitant D5W injection is associated with a high degree of distal penetration and complete branch occlusion and minimal risk of cranial nerve palsy or other thromboembolic complications.

What about Primary, Rescue, adjunct MMAe

Trends and Outcomes of Primary, Rescue, and Adjunct Middle Meningeal Artery Embolization for Chronic Subdural Hematomas

Anna M. Nia[1], Visish M. Srinivasan[2], Farhan Siddiq[3], Ajith Thomas[4], Jan-Karl Burkhardt[5], Rishi R. Lall[1], Peter Kan[1]

BACKGROUND: Middle meningeal artery embolization (MMAE) is an effective minimally invasive treatment for chronic subdural hematomas (cSDHs). The authors investigated outcomes of primary, adjunct, and rescue MMAE and primary surgery for the treatment of cSDH using a large-scale national database.

METHODS: A retrospective study of all patients who underwent MMAE and/or surgery to treat cSDH was performed using the TriNetX Analytics Network. Primary MMAE was compared with adjunct and rescue MMAE and primary surgery. Primary outcomes included headache, facial weakness, mortality, and treatment failure, within 6 months.

RESULTS: A total of 4274 patients with cSDH met the inclusion criteria. Of these, 209 (4.9%) were treated with primary MMAE, 4050 (94.8%) were treated with primary surgery, 15 (0.35%) were treated using MMAE as an adjunct therapy, and 18 (0.42%) were treated using MMAE as a rescue following a failed surgical intervention. There were no significant differences in headache, facial weakness, and mortality between the groups. Patients who underwent primary MMAE had a significantly higher Charlson comorbidity index ($P < 0.0001$) than those who underwent primary surgery. The need for surgical rescue was not significantly different between primary MMAE, adjunct MMAE, and rescue MMAE ($P > 0.05$). Additionally, patients with primary surgery had significantly higher treatment failure than those with primary MMAE (odds ratio = 2.11, 95% confidence interval = 1.11–4.01, $P = 0.020$).

CONCLUSIONS: This analysis suggests no significant difference in the need for surgical rescue, complication, or mortality between primary MMAE, adjunct MMAE, and rescue MMAE. Additionally, primary MMAE is associated with a significantly lower need for surgical rescue than primary surgery.

INTRODUCTION

Chronic subdural hematoma (cSDH) is an increasingly common neurosurgical pathology with an estimated incidence of 10 cases per 100,000 persons in the United States.[1] cSDH is hypothesized to develop due to a prior traumatic acute subdural hemorrhage or subdural hygroma that forms at the dural border cell layer between the dura and arachnoid layer. Chronic inflammation with subsequent hyperfibrinolysis and release of angiogenic factors can lead to neovascularization and neomembrane formation.[2,3] Surgical evacuations of the cSDH are the standard of surgical management for symptomatic patients. Unfortunately, in elderly patients who undergo treatment with surgical intervention, the 1-year mortality rate is up to 32%.[4] Surgical intervention is additionally complicated by high recurrence and reoperation rates of up to 30%,[1] making

Secondary Headache (M Robbins, Section Editor) | Published: 08 July 2022

Recent Advances and Future Directions in Middle Meningeal Artery Embolization for Chronic Subdural Hematomas

Gary Kocharian ✉, Kyle B. Zappi, Joseph Carnevale, Justin Schwarz & Jared Knopman

Current Pain and Headache Reports **26**, 657–665 (2022) | Cite this article

Recent Findings

The latest data shows that MMA embolization is a safe procedure, with low complication rates and low recurrence rates. While cSDH managed with surgical evacuation can have a recurrence rate upwards of 30%, MMA embolization alone or as an adjunct to surgery decreases recurrence to less than 5% in most studies. MMA embolization can be especially useful in high-risk populations such as the elderly, patients on anti-platelet medication, and those with coagulopathies. It can also be done awake, done without general anesthesia, and is significantly less invasive than traditional surgical techniques.

Summary

In reviewing the literature on MMA embolization, it is clear that there are numerous retrospective studies and systematic reviews demonstrating its safety and efficacy, and some prospective dual-arm studies that present novel information. The numerous clinical trials that are currently underway should help to further establish MMA embolization as standard of care in the management of cSDH.

Industry-led clinical trials- ongoing

nBCA TRIAL: Cerenovus (Membrane)- Kellner & Rai

- Age: 18-90
- Mass effect with cSDH
- Pre randomization mRS <3
- CT performed within. 36 hours prior demonstrates no worsening of midline shift from baseline CT

Exclusion criteria:

- Acute SDH
- Prior ipsilateral craniotomy/ burr hole
- Bilateral cSDH
- GCS<9
- Underlying conditions like tumors, arachnoid cyst, IIH
- Carotid stent across ECA origin (ipsilateral) to hematoma
- Anastomosis between ophthalmic and MMA

Squid trial (STEM)- Balt- Fiorella & Arthur et al

Inclusion criteria:

- Age >30
  Pre morbid mRS 0-1
  cSDH >10mm in greatest thickness
  Mass effect (MLS or local cortical flattening)

EMBOLISE Trial-Medtronic Onyx

- Knopman & Davies et al

Morphological changes in chronic subdural hematomas following upfront middle meningeal artery embolization: sequence, timing, and association with outcomes

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RESULTS

Overall, 52 CSDHs in 45 patients treated with upfront MMAE were identified. Hematomas were followed for a mean of 92.9 days. Volume decreased by ≥ 50% in 79.6% of the CSDHs. The overall rescue surgery rate was 9.6%. A sequence of morphological changes after MMAE was identified. Hematomas that diverged from this sequence (5.4%) all progressed toward treatment failure and required rescue surgery. The CSDHs were categorized into early, intermediate, and late stages based on the baseline morphological appearance. Progression from early to intermediate and then to late stage took 12.7 and 30.0 days, respectively, on average. The volume of early/intermediate- and late-stage hematomas decreased by ≥ 50%, a mean of 78.2 and 47.6 days after MMAE, respectively. Early- and intermediate-stage hematomas showed a trend toward more favorable outcomes compared with late-stage hematomas. The density of homogeneous hypodense hematomas (HSDHs) transiently increased immediately after MMAE ($p < 0.001$). A marked decrease in density and volume 1 to 3 weeks after MMAE in HSDHs was detected, the lack of which indicated an eventual need for rescue surgery. In HSDHs, a baseline mean density of < 20 HU, and a lower density than baseline by 1 month post-MMAE were predictors of favorable outcomes. The baseline hematoma volume, axial thickness, midline shift, and loculation were not correlated with MMAE outcomes. Loculated, trabecular, and laminar hematomas, which are known to have unfavorable surgical outcomes, had MMAE outcomes similar to those of other "surgical" hematomas.

CONCLUSIONS

The current study was the first to describe the nature, sequence, and timing of morphological changes of CSDHs after MMAE treatment and has identified structural features that can predict treatment outcomes.

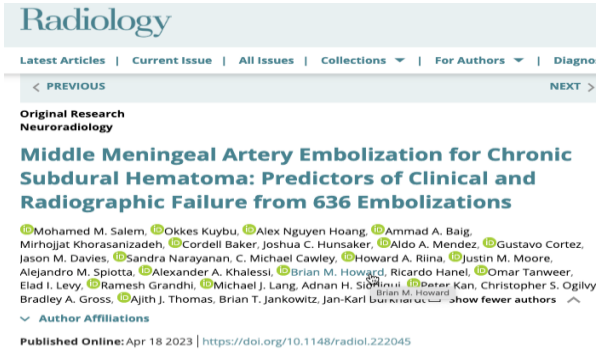
ABBREVIATIONS

Risk of recurrence with MMA Embo vs Surgical drainage alone

- Hematoma recurrence rate was significantly lower in the embolization group compared with conventional treatment group (2.1% vs. 27.7%; odds ratio = 0.087; 95% confidence interval, 0.026–0.292; $P < 0.001$; $I^2 = 0\%$);

- surgical complication rates were similar between groups (2.1% vs. 4.4%; odds ratio = 0.563; 95% confidence interval, 0.107–2.96; $P = 0.497$; $I^2 = 27.5\%$)

- Srivatsan et al. https://doi.org/10.1016/j.wneu.2018.11.167.



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Original Research
Neuroradiology

Middle Meningeal Artery Embolization for Chronic Subdural Hematoma: Predictors of Clinical and Radiographic Failure from 636 Embolizations

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Results

Overall, 530 patients (mean age, 71.9 years ± 12.8 [SD]; 386 men; 106 with bilateral lesions) underwent 636 MMAE procedures. At presentation, the median CSDH thickness was 15 mm and 31.3% (166 of 530) and 21.7% (115 of 530) of patients were receiving antiplatelet and anticoagulation medications, respectively. Clinical failure occurred in 36 of 530 patients (6.8%, over a median follow-up of 4.1 months) and radiographic failure occurred in 26.3% (137 of 522) of procedures. At multivariable analysis, independent predictors of clinical failure were pretreatment anticoagulation therapy (odds ratio [OR], 3.23; $P = .007$) and an MMA diameter less than 1.5 mm (OR, 2.52; $P = .027$), while liquid embolic agents were associated with nonfailure (OR, 0.32; $P = .011$). For radiographic failure, female sex (OR, 0.36; $P = .001$), concurrent surgical evacuation (OR, 0.43; $P = .009$), and a longer imaging follow-up time were associated with nonfailure. Conversely, MMA diameter less than 1.5 mm (OR, 1.7; $P = .044$), midline shift (OR, 1.1; $P = .02$), and superselective MMA catheterization (without targeting the main MMA trunk) (OR, 2; $P = .029$) were associated with radiographic failure. Sensitivity analyses retained these associations.

Conclusion

Multiple independent predictors of failure of MMAE treatment for chronic subdural hematomas were identified, with small diameter (<1.5 mm) being the only factor independently associated with both clinical and radiographic failures.

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