MIDDLE MENINGEAL ARTERY (MMA) EMBOLIZATION FOR THE TREATMENT OF SUBDURAL HEMATOMAS

What is a subdural hematoma (SDH)?

The brain is encased in a protective bony shell called the skull. Inside the skull, the brain is also surrounded by a smooth fibrous pouch called the dural membrane (aka: dural meninges; dural sac; dura mater). This pouch's blood supply consists of arteries and veins that are embedded within the dural membrane. Small connecting vessels connect the dural veins with other veins located on the surface of the underlying brain. These connecting structures are termed "bridging veins". Bridging veins can be damaged through many mechanisms that cause stretching and subsequent tearing of the vessels. When this occurs the veins leak blood into the space located between the surface of the brain and the inner surface of the dural membrane (Figures1,3). This blood collects to form what is termed a subdural hematoma (SDH). As the SDH enlarges, it begins to put pressure on the underlying brain which in turn can cause headaches and neurologic deficits which include weakness, sensory changes, confusion, lethargy, seizures and at times death. While SDH can develop from other types of vascular injury, tearing of bridging veins remains the most common etiology.

Types of SDH fall into four categories (Figure 2).

1. Acute SDH: These lesions consist of freshly coagulated blood that is firm to the touch.

2. Subacute SDH: These lesions consist of jelly-like partially degraded clotted blood which forms as the acute blood clot ages and breaks down.

3. Chronic SDH: These lesions consist of liquid that develops as the blood clot completely breaks down.

4. Mixed SDH: These lesions are mixtures of acute, subacute and chronic SDH.

Figure 1: Subdural hematoma

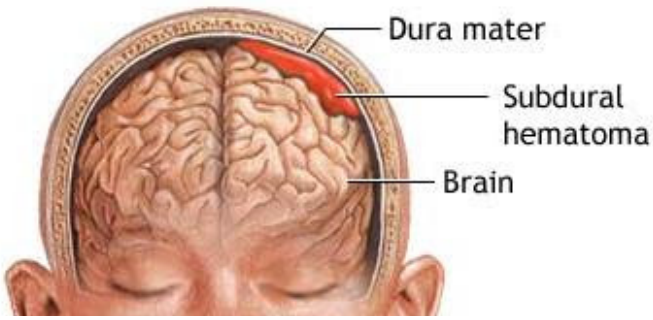
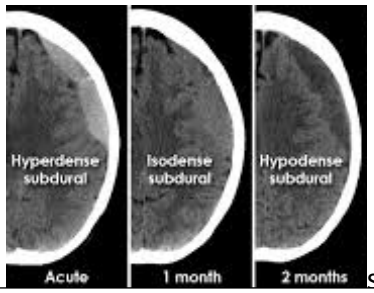


Figure 2: CT Scan Appearances of Acute, Subacute and Chronic SDH



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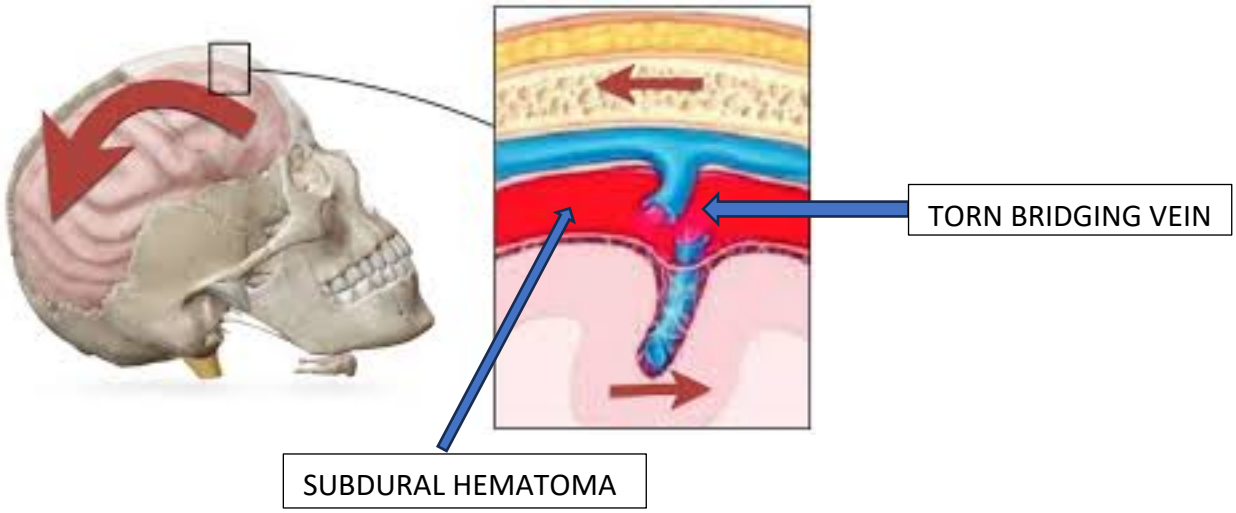
Acute. Subacute. Chronic

What causes SDH?

SDH most commonly develop after some form of head injury. The degree of injury can be severe or minor with some injuries being so mild as to not have been noticed by the patient. Any force that promotes tearing of a bridging vein can cause a SDH (Figure 3).

Figure 3: Mechanism of Injury



Who develops SDH?

Patients of any age can develop a SDH, but they become more common in the elderly. As individuals age, the brain normally loses volume and decreases in size due to tissue dehydration and tissue death (Figure 4). As the brain shrinks in size its surface moves further away from the overlying dura. This movement stretches the bridging veins (Figure 3) that connect the inner surface of the dura to veins located on the brain's outer surface. When veins are stretched in the

elderly, they become more susceptible to tearing and leakage of blood when even minor head trauma occurs (Figure 5).

Figure 4: Younger brain (A) vs Older brain (B)

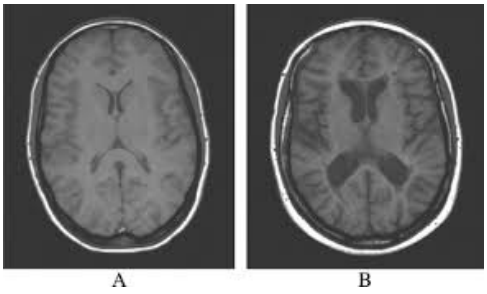
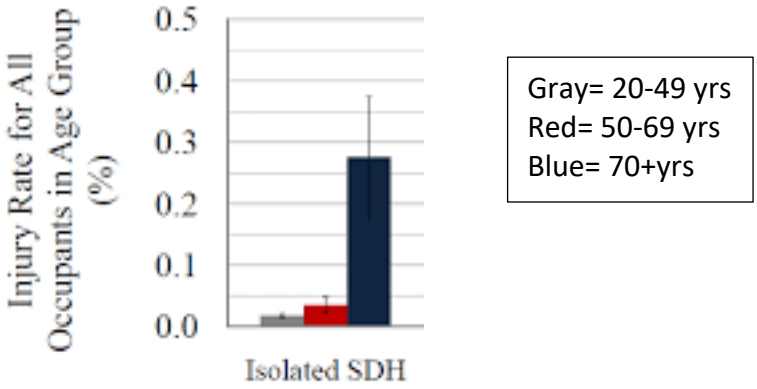


Figure 5: Frequency of SDH by age group



What risk factors increase the likelihood of developing a SDH?

Risk factors for SDH development include, but are not limited to:

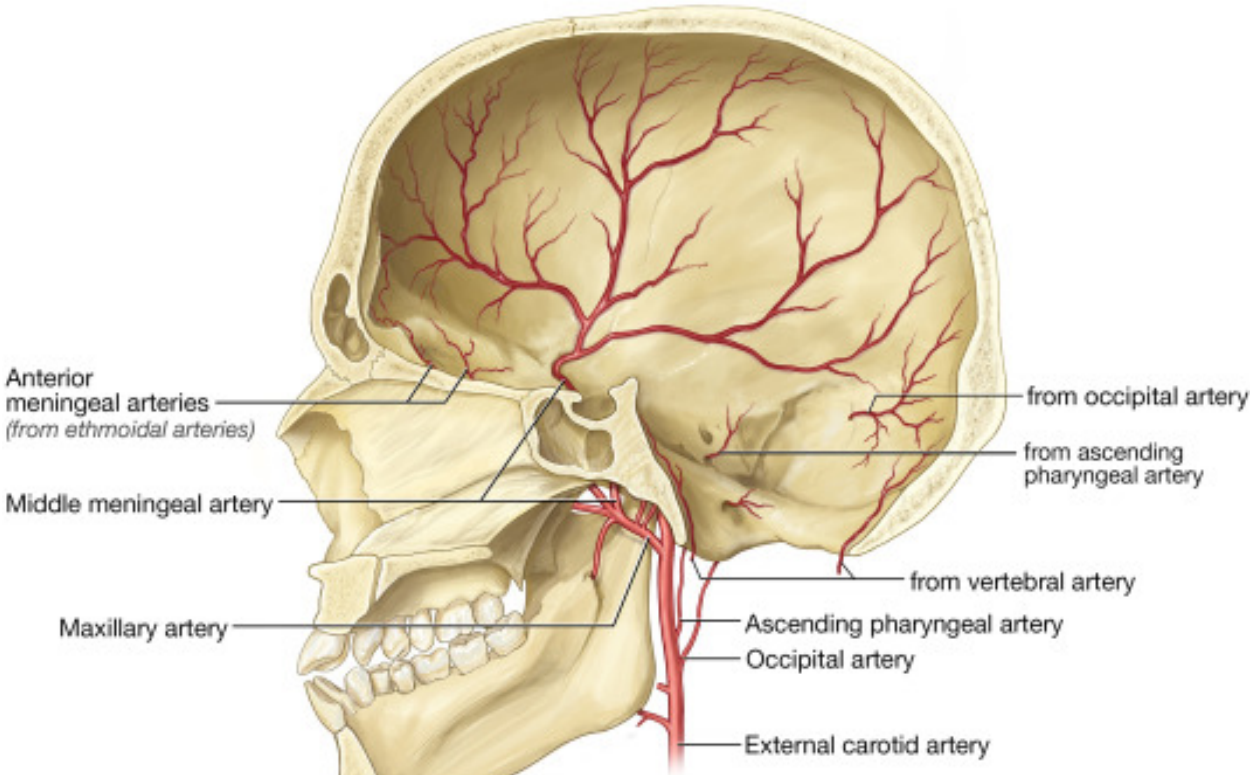
1. Head injury (minor or major)
2. Advancing age
3. Use of anticoagulant or antiplatelet medications
4. Thrombocytopenia (low platelet count)
5. Coagulopathy (abnormal ability of person to clot blood)

SDH Treatment Options:

SDH can be removed from the head using a variety of surgical procedures which involve opening the skull and dura and removing the hematoma followed by closing the dura, replacing the skull and closing the skin incision. In some of these patients SDH can recur. In other patients, open surgical SDH evacuation as described above is either not necessary or not safe. For both groups of individuals (surgical and non-surgical) it may be beneficial to seal off the arterial blood vessels located in the dura with the goal of halting further SDH enlargement and /or reducing the risk of SDH recurrence). In some cases, closure of the MMA may result in SDH shrinkage over time. The procedure that is used to seal off the dural (aka: meningeal) arteries is called "Embolization".

Embolization of the dural/meningeal arteries involves advancing a catheter from either an artery in the wrist or groin into a dural artery called the Middle Meningeal Artery (MMA) (Figure 6). Once the catheter is positioned within the MMA, a variety of materials can be injected into the MMA which cause the artery to close off thus blocking further blood supply to the dura. This elimination of the dural blood supply (middle meningeal artery embolization; MMAE) can halt further growth of the SDH and at times lead to shrinkage of the SDH over time.

Figure 6: Anatomic drawing showing the left MMA and its branches that are embedded within the dura (meninges) that line the inner surface of the skull and surround the brain



Middle Meningeal Artery Embolization (MMAE) combined with Surgical Evacuation of Chronic and Subacute SDH:

Middle meningeal artery embolization (MMAE) is often recommended for the treatment of chronic/subacute and on occasion acute SDH. The efficacy and safety of MMAE is best documented in case series where the procedure was used to treat chronic and subacute SDH. Multiple published studies have demonstrated that MMAE, particularly when used in addition to open surgical evacuation, significantly reduces the risk of hematoma recurrence and the need for reoperation. In the EMBOLISE Trial, MMAE plus surgery reduced the rate of hematoma recurrence or progression leading to repeat surgery at 90 days in 4.1%, compared to 11.3% with surgery alone. Serious adverse events related to embolization were rare (2.0%) and there was no significant increase in mortality or functional deterioration at 90 days compared to surgery alone. Functional outcomes were similar between groups, with 11.9% experiencing functional deterioration in the MMAE group versus 9.8% in the control group.

A GRADE-assessed meta-analysis of 1,544 patients confirmed the EMBOLISE Trial findings. This study found that MMAE plus surgery reduced recurrence rates to 8% versus 15.6% for standard care. Need for open surgical reoperation for recurrent SDH was 4.5% in the embolized group versus 12.7% in the non-embolized group. There was no significant increase in serious adverse events, stroke, or mortality.

The MAGIC-MT Trial found that MMAE did not significantly reduce the 90-day incidence of symptomatic recurrence or progression compared to usual care (6.7% vs. 9.9%). This study did, however, find a lower incidence of serious adverse events when comparing the embolized and non-embolized groups (6.7% vs. 11.6%).

In summary, multiple clinical studies have shown that MMAE performed along with open surgical removal of a SDH, reduces recurrence and reoperation rates without increasing severe complications or mortality. The overall complication rate for MMAE is low, typically below 2–3%, and includes rare events such as access site complications or non-target embolization. MMAE is best supported for chronic and subacute SDH, particularly in patients with moderate-sized hematomas, minimal mass effect, and high surgical risk. Large, symptomatic, or acute hematomas generally require surgical evacuation, though MMAE may be used before or after surgery to reduce the risk of recurrence.

Result for MMAE Embolization Without Surgical Evacuation of SDH (aka: Standalone MMAE):

Standalone MMAE is particularly useful when patients are too ill to undergo open surgical procedures or have SDH that are not yet large enough to warrant surgery. In a large US database study, standalone MMAE was associated with a significantly lower risk of the need for surgical rescue or death at 180 days (8.2% vs. 10.9%) and a lower risk of all-cause mortality (1.1% vs. 3.0%).

A 2025 meta-analysis published in World Neurosurgery looking at over 250,000 patients with chronic SDH found that patients undergoing MMAE embolization alone for treatment of chronic SDH had a lower likelihood of requiring subsequent open surgical evacuation and had a lower overall mortality rate (12.1 vs 15%)

Hematoma size and location may also influence management decisions. Large hematomas with significant mass effect or midline shift generally require prompt surgical evacuation. MMAE is less likely to be used as a primary therapy in these cases, though it may be considered adjunctively.[15][21] In patients with moderate-sized or smaller hematomas, particularly those who are asymptomatic or minimally symptomatic, MMAE may be considered as a standalone therapy, especially in those at high surgical risk. MMAE is particularly attractive in elderly or medically frail patients who are poor surgical candidates, as it is minimally invasive and associated with low rates of serious complications.

MMAE Embolization for Acute SDH:

The evidence for MMAE benefits when treating acute SDH is limited. There are no high-quality studies specifically evaluating MMAE when used alone to treat acute SDH. Surgical evacuation remains the standard of care for acute SDH with significant mass effect or neurological deterioration. MMAE may be considered in select cases of acute-on-chronic SDH or in patients with contraindications to surgery, but this remains an area for further research.

Timing of Embolization Relative to Surgery:

While perioperative MMAE performed before or after surgical evacuation has been shown to reduce recurrence rates compared to surgery alone, the timing of MMAE relative to surgery (pre- vs post-operative) does not appear to significantly affect outcomes.

Safety and Complication Rates:

Complication rates for MMAE are low across all major studies, with serious adverse events (such as stroke or cranial nerve palsy) being rare (less than 1-3%).

Long-Term Clinical and Radiographic Outcomes:

Long-term (≥1 year) outcomes of MMAE for SDH are characterized by a significant reduction in recurrence and reintervention rates, particularly when used along with open surgery. Recurrence rates remain below 5% for the MMAE plus surgery group at long-term follow-up, even in patients with significant comorbidities. Standalone MMAE also demonstrates favorable long-term outcomes, with recurrence rates of approximately 6.3% and retreatment rates of 9.6% at long-term follow-up. Over time, most hematomas achieve substantial volume reduction. Functional outcomes are generally comparable to those achieved with conventional management, with the added benefit of fewer recurrences and hospital readmissions. The safety profile of MMAE is favorable, with low rates of long-term complications.

Cost-Effectiveness:

MMAE is generally more costly upfront than conventional management strategies for SDH, but its ability to reduce recurrence, reoperation, and readmission rates can offset these costs, particularly in high-risk or recurrent cases. Economic modeling and real-world data suggest that MMAE is cost-effective for chronic and subacute SDH, especially as an addition to surgery or as a primary therapy in nonsurgical patients.

Summarized Information Regarding MMAE for SDH:

1. For chronic and subacute SDH with an indication for surgical evacuation, MMAE in addition to surgery should be considered to reduce recurrence and reoperation rates below 7%.

2. MMAE can be performed either before or after surgery, with no significant difference in outcomes.

3. For patients with nonsurgical or high-risk SDH (e.g., advanced age, coagulopathy, antithrombotic use), standalone MMAE is a reasonable alternative to conservative management, with a target recurrence rate of 6–7% and a significant reduction in all-cause mortality compared to conservative management.

4. For acute SDH, surgical evacuation remains the standard of care particularly in patients with significant mass effect or neurological deterioration. MMAE may be considered in select cases of acute-on-chronic SDH, in patients with contraindications to surgery or as a rescue therapy after failed surgical intervention, but these indications are not well supported by high-quality evidence.

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