e-ISSN: 2807-7970
doi: 10.20473/aksona.v5i1.52439

AKSONA

Volume 5 Number 1, January 2025

Successful Management of Bilateral Subdural Hematoma: Combining Bilateral Middle Meningeal Arteries Embolization and Unilateral Burr Hole Craniostomy—A Case Report

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Article info

Article History:
Received Dec 15, 2023
Revised Jul 29, 2024
Accepted Sept 20, 2024
Published Jan 29, 2025

Keywords:
Burr hole craniostomy
Chronic subdural hematoma
Middle meningeal artery embolization

ABSTRACT

Introduction: Subdural hematoma (SDH) is frequently seen in the elderly population. Although anticoagulant use and traumatic brain injury are recognized risk factors, the exact cause of subdural hematomas in certain cases remains unknown. Furthermore, unilateral or bilateral hemispheres may be involved in SDH, which can occur acutely or gradually. The old age group's SDH profile, which includes several comorbidities, may complacte surgery, the gold standard for treatment. Embolization of the middle meningeal artery (MMA) may be a treatment option because it is safer and has a higher success rate in terms of improvement compared to complications. It can be done as either a standalone procedure or an adjuvant to surgery.
Case: An elderly male patient experienced acute left-sided weakness that worsened over three days. Imaging revealed bilateral SDH, with symptomatic chronic SDH on the right side, asymptomatic acute SDH on the left side, and midline shift. Bilateral MMA embolization was done, followed by burr hole craniostomy, which resulted in clinical and radiological improvement throughout a three-month post-intervention period.
Conclusion: MMA embolization can improve clinical outcomes and prevent recurrent bleeding, which makes it a potential treatment option for severe symptomatic SDH, especially in patients with comorbidities.

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INTRODUCTION

A subdural hematoma (SDH) is a blood collection in the subdural space. Chronic subdural hematomas are prevalent in the elderly population, with 1.7 to 20.6 cases per 100,000 individuals annually, with a median age in the seventh decade.[1,2,3] It serves as a predictor of four-year mortality and five-year functional dependence following its diagnosis. Additionally, the condition may recur, even with effective treatment. [1,2,3]

Nonspecific symptoms, which can manifest as asymptomatic or with a gradual and rapid onset, pose numerous challenges in managing SDH, as does the treatment approach. A subdural hematoma can develop acutely, chronically, or bilaterally, causing nonspecific symptoms such as headaches, gait instability, cognitive impairment, focal neurological deficits, and decreased consciousness. Additionally, chronic SDH lacks a standardized management approach. Generally, conservative management is employed for asymptomatic cases and surgical management for symptomatic cases.[2,3] However, postsurgical recurrence rates of SDH have been reported to hit 30%.[1,2,3] As for management, some aspects are up for debate. These include when surgery is needed, when to begin intervention, the surgical techniques used, the number and location of burr holes, irrigation, the placement and length of drainage implants, and the decision made about membranectomy and middle meningeal artery (MMA) embolization.[3]

A novel approach to minimize postsurgical recurrence and manage bleeding in chronic SDH is MMA embolization. One of the main places where SDH patients bleed is through the middle meningeal artery. MMA embolization is expected to stop the cycle of recurrent bleeding and hyperfibrinolysis, especially in chronic SDH. This technique can be performed as a standalone procedure, as a presurgical adjuvant, or as postsurgical bleeding prophylaxis with a high degree of safety.[2,3] This approach serves as a valuable alternative for conventional surgical procedures, providing enhanced clinical outcomes and a reduced risk of recurrence.

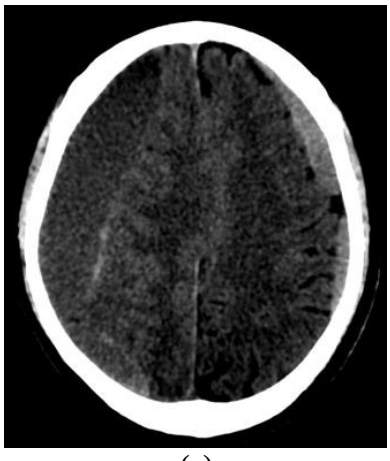
This case report describes the successful management of an elderly male with bilateral different-age SDH, who underwent bilateral MMA embolization followed by burr hole craniostomy on the symptomatic side. The intervention led to a significant clinical improvement, including the loss of weakness on the left side. During the three-month follow-up period, radiological findings showed a decrease in SDH volume and midline shift. In Indonesia, reports of this technique have been infrequent to date. Further studies are warranted to validate and expand upon these findings, with the aim of optimizing treatment approaches for patients with bilateral SDH.

CASE

The emergency room received a 70-year-old male patient who had developed acute left-sided weakness that progressively worsened during the past three days. The patient and family reported no history of head trauma, headaches, or memory loss. Prior to the incident, the patient was capable of doing daily activities independently. He had hypertension with an average measurement of 140/90 mmHg and was regularly treated with antihypertensive medications. No history of diabetes, stroke, heart disease, or previous use of antithrombotic drugs was present.

On physical examination, his blood pressure was 150/90 mmHg, his pulse was regular at 100 beats per minute, his respiration rate was 20 breaths per minute with a regular pattern, and his temperature was 36.6°C. The patient was alert and oriented with a Glasgow Coma Scale score of Eye 4, Motor 6, and Verbal 5, with mild cognitive impairment and left-sided hemiparesis. Non-contrast brain computed tomography (CT) showed acute left frontotemporoparietal SDH and chronic right frontotemporoparietal SDH with a midline shift to the left.

The patient got both non-pharmacological and pharmacological treatment, as well as bilateral MMA embolization and a burr hole craniostomy to remove a hematoma from the right temporal region. The procedure was performed in the acute phase, duringthe first week of treatment. A catheter was inserted up to the external carotid artery, and then a microcatheter was advanced to the maxillary artery to reach the middle meningeal artery (MMA). The embolic material of N-butyl cyanoacrylate (NCBA) was introduced at the middle segment of MMA until it was obliterated.



(a)

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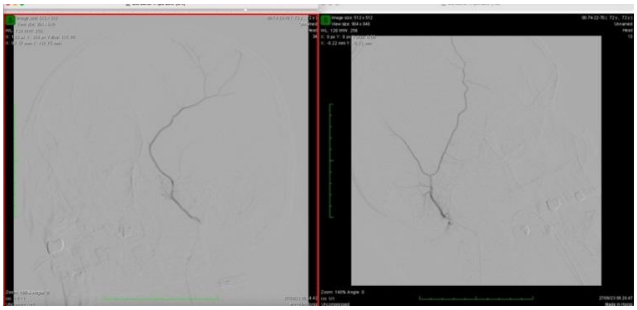
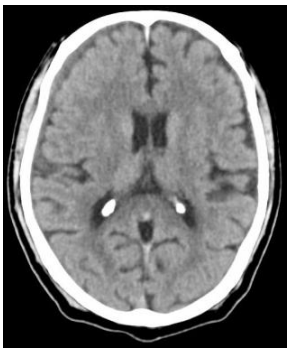


(b) (c)



(d)

Figure 1. (a) the first CT scan shows acute left frontotemporoparietal subdural hematoma and chronic right frontotemporal subdural hematoma with midline shift; (b) obliteration of the left middle meningeal artery; (c) obliteration of the right middle meningeal artery; (d) the CT scan evaluation three months later shows improvement in bilateral subdural hematoma.

The three-month postsurgical follow-up revealed improvements in the patient's condition and imaging, with residual symptoms consisting of left-sided weakness that had improved.

DISCUSSION

The management of symptomatic bilateral SDH remains challenging. Although surgical evacuation remains the gold standard, the decision must be made based on which side to evacuate (both sides or only the symptomatic side), the patient's comorbidities, and the risk of recurrent subdural bleeding.

This case pertains to an elderly male who has bilateral SDH, characterized by symptomatic chronic right-sided SDH and asymptomatic acute left-sided SDH, both of which had a sudden clinical onset. The clinical condition fits with reports that SDH may not always present gradually with symptoms like cognitive impairment, repetitive falls, delirium, Parkinsonism, or transient neurological deficits, but can sometimes appear suddenly as acute weakness or seizures. In this case, bilateral SDH is thought to be spontaneous, because there is no history of head trauma, anticoagulant usage, or chronic worsening symptoms such as headaches and cognitive impairment.[2,3]

The pathogenesis of acute and chronic SDH is different. Acute SDH happens when bridging veins rupture, causing an accumulation of blood and its components in the subdural space. In the meantime, people with chronic SDH not only have repeated ruptures in bridging veins, but they also experience the activation of a chronic inflammatory response that triggers the infiltration of proangiogenic cells. This condition leads to the formation of an inflammatory neovascular membrane in the subdural space. The membrane is made up of endothelial cells with many gap junctions and thin-walled capillaries devoid of smooth muscle. This lets fluids keep flowing through the inflammatory environment. Higher levels of vascular endothelial growth factor (VEGF) and inflammatory cytokines cause more immature blood vessels to grow on the walls of the SDH. This makes gap junctions and vascular permeability worse. Additionally, there is a decrease in anti-inflammatory agents and factors supporting vascular repair, such as regulatory T cells and endothelial progenitor cells. These interactions contribute to the pathology of immature angiogenesis, endothelial injury, and vascular leakage. Microbleeding and the accumulation of excessive fibrinolysis products trigger recurrent SDH. The inflammatory neovascular membrane, which is a pathognomonic finding in SDH, gets most of its blood from the middle meningeal artery.[2,4,5]

If symptomatic bleeding on the right side is evacuated, it could make the acute bleeding on the left side wors. Also, evacuation of bilateral bleeding poses significant risks in older people with unknown bleeding triggers, making management of this case challenging. Currently, there are no specific guidelines that provide high-quality evidence for the management of SDH.[1] When deciding how to treat chronic SDH, consider the following factors: (1) figure out if the SDH is asymptomatic, mildly symptomatic, or severely symptomatic; (2) determine whether the surgical risk is high or low; and (3) evaluate other factors such as hematoma size and mass effect, as well as the patient's comorbidities and overall condition (Figure 2, Table 1).[2,6] The gold standard for managing SDH is surgical hematoma evacuation,[2,3] but this tends to be contraindicated in the elderly with complex comorbidities due to its high risk of recurrence.[1] Surgery is usually the best option for people who have neurological deficits supported by brain imaging that last or get worse for at least two weeks, a hematoma expansion, or difficulty taking their medicine (Table 1).[3,4] Embolization of the MMA is an innovative alternative to surgery, particularly for patients at high surgical risk. Individuals with severe symptomatic chronic SDH can use this procedure either alone or before surgery to reduce the size of the hematoma. Furthermore, due to its minimally invasive nature compared to surgery, it may be the preferred option for

reducing SDH size in asymptomatic or mildly symptomatic cases.

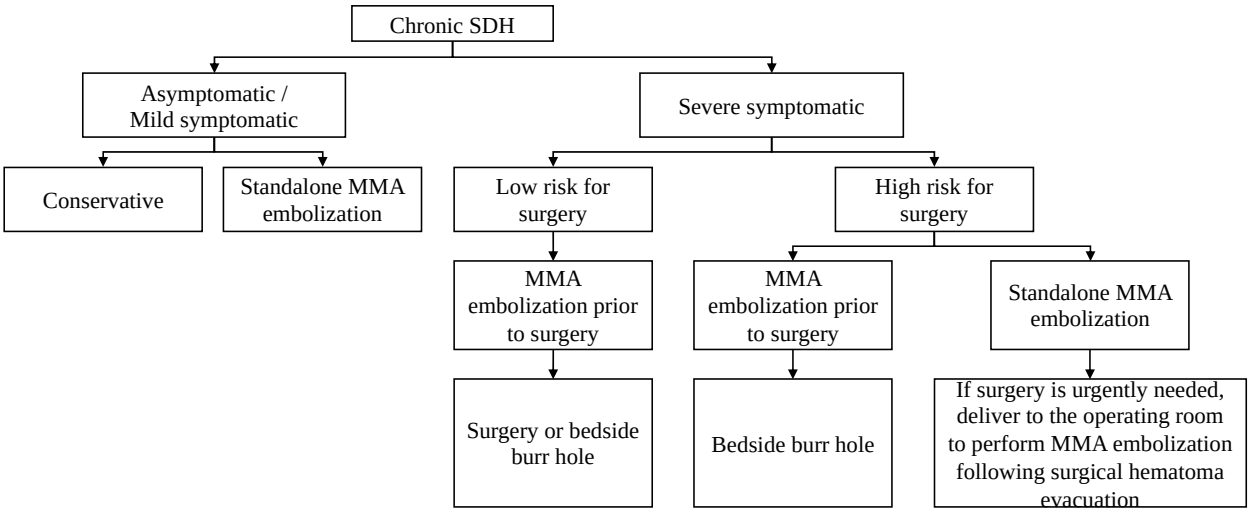


Figure 2. Proposed algorithm for the management of chronic subdural hematoma.[2]
CSDH: chronic subdural hematoma; MMA: middle meningeal artery.

Table 1. Indication and contraindication of pharmacologic medication[4,6]

Indication for standalone pharmacological therapy	Contraindication of standalone pharmacological therapy
1. Stable vital signs with an MGS-GCS score of 0-2; 2. Midline shift on brain imaging less than 1 cm and no indication for emergency surgical management; 3. Multiple organ failure, coagulation disorders, or not a candidate for or refusing surgery; 4. Surgery has been performed as a preventive measure against recurrence.	1. MGSC-GCS score of 3-4; 2. Brain imaging consistent with severe brain compression and midline shift greater than 1 cm; 3. Signs of brain herniation such as decreased consciousness, nausea, and vomiting; 4. Drug allergy or contraindication to the use of medication.

Note: MGS-GCS: Markwalder's grading scale and Glasgow Coma Scale.
MGS-GCS grade 0 indicates GCS 15 without any symptoms or neurological deficits. MGS-GCS grade 1 indicates GCS 15 without neurological deficits but with symptoms such as headaches or unsteady gait. MGS-GCS grade 2 indicates GCS 13-14 with symptoms of disorientation or drowsiness or varying focal neurological deficits (hemiparesis). MGS-GCS grade 3 indicates GCS 9-12 with sopor and a good response to pain or severe focal neurological deficits (hemiplegia). MGS-GCS grade 4 indicates GCS <9, coma without motor response to painful stimuli, decortication, or decerebration.

Conservative management is the current recommendation in cases where surgery is contraindicated or the patient is asymptomatic or mildly symptomatic with SDH. This can be anything from observation to the administration of atorvastatin 20 mg/day for a at lease 8 weeks, with or without short-term low-dose dexamethasone 2.25 mg/day for 1–2 weeks, which should be tapered off within 4 weeks in cases of recurrent or refractory SDH.[1,4] Tranexamic acid can also be considered. Atorvastatin is reported as the best therapy to reduce hematoma volume in chronic SDH, and dexamethasone is reported as the best therapy to reduce SDH recurrence.[1] These pharmacological therapies aid in the absorption of bleeding in chronic SDH. Symptomatic therapy may also be recommended. Some examples are hyperosmolar agents that can help control high intracranial pressure (strong recommendation, moderate-quality evidence), nonsteroidal anti-inflammatory drugs for headaches, antiepileptics like valproic acid in cases with seizures (strong recommendation, moderate-quality evidence), antidepressants like olanzapine for mood disorders (strong recommendation, weak-quality evidence), and insomnia management (strong recommendation, moderate-quality evidence). Prophylactic antiepileptic use is not recommended (weak recommendation, weak-quality evidence), but it can be considered based on the benefits and risks in the elderly and alcohol-dependent populations.[4]

In cases of symptomatic SDH, physicians may recommend surgical management in addition to symptomatic management of increased intracranial pressure. Surgical management options include twist drill craniostomy, burr hole craniostomy, or craniotomy for hematoma evacuation with or without surgical drain. The type of surgery used depends on the SDH profile. For SDH with most fluid hematomas, a twist-drill craniostomy is used. For CSDH with complex hematoma and a clear neovascular

membrane, a craniotomy is used.[2] Burr hole craniostomy is considered the first-line operative therapy with the best repair-to-complication ratio (Grade A recommendation), followed by the placement of a subgaleal or subdural drain for 24–48 hours (Grade A recommendation).[3] However, any operative technique carries a risk of SDH recurrence of 3–30%,[2,7] and a risk of acute SDH in 5% of cases post-burr hole craniostomy.[2] SDH recurrence leads to an increased frequency of hospitalization, surgical risk, and a decrease in the quality of life. Having more SDH after surgery is linked to a higher risk of complications, including bilateral SDH, presurgical hematoma, larger midline shift, larger postsurgical air and subdural cavity volume, mixed density of perioperative hematoma with low and high-density components (separated type), comorbid hypertension, diabetes, leukemia, liver disease, chronic kidney disease, seizures, and anticoagulant use.[7]

This case concerns a symptomatic SDH patient with an MGS-GCS of 2. The midline shift is still less than 10 mm, but the thickness of the symptomatic SDH diameter suggests surgical intervention. However, advanced age and bilateral SDH with different ages complicate intrasurgery risk and postsurgical SDH recurrence. Therefore, bilateral MMA embolization is considered before operative intervention. Burr hole craniostomy is the current choice of surgery based on the latest systematic review.[3]

Sila *et al*. reported that MMA embolization alone can treat SDH that is less than 18mm wide, has a midline shift of less than 5 mm, and doesn't have any acute or subacute hematomas. On the other hand, surgery may be better for SDH that is more than 18 mm wide, has a midline shift of more than 5 mm, and doesn't have any membranous segmentation.[8]

Currently, there are no recommendations on the appropriate timing of surgical intervention in SDH. The severity of SDH and other factors, like anticoagulant usag, determine the timing of the procedure (Grade B recommendation).[3] Bilateral MMA embolization and burr hole craniostomy in this case were done in the acute phase to avoid the elevated intracranial pressure from increasing. Aside from that, preoperative bilateral MMA embolization may significantly reduce the volume in SDH and the midline shift after the surgical removal of SDH.[9]

MMA embolization can be used to manage recurrent bleeding and inflammation. It works by blocking the main artery in the neovascular membrane of chronic SDH. It can be used on its own or along with surgery (Grade B recommendation).[2,3,10] This approach, as reported in meta-analyses, lowers the recurrence rate of SDH and has a low risk of postsurgical stroke complication because it only involves the external carotid circulation. It is reported 92% of cases of chronic SDH are absorbed 6 months after surgery, and only 4–8% of chronic SDH cases require further surgical intervention after MMA embolization.[2,11] Whether embolization is performed as a single procedure,[12] or as an adjuvant procedure, the recurrence rate of chronic SDH is lower, ranging from 2.1–4.8% in various studies.[3,13] A randomized control study, which involved 17 embolization and 19 control groups of chronic SDH, revealed that the embolization group significantly outperformed the control group at 3 months, with a modified Rankin Scale (score 0–1) of 100%, compared to 53% in the control group.[14]

MMA embolization for chronic SDH is performed by inserting the catheter into the external carotid artery, followed by inserting a microcatheter into the MMA. The embolic agent is then inserted as distally as possible, marked by the "wedged" catheter position to ensure that it enters the neovascular membrane. It is inserted into the anterior and posterior branches of the MMA.[2] It is important not to inject the embolic agent too quickly, to keep the microcatheter as far away from the ophthalmic collateral as possible, and to inject the embolic agent maximally before reaching the petrosal branch (proximal branch of the MMA). This will help prevent venous thrombosis, ophthalmic artery occlusion, or facial nerve injury. The embolic agent won't be able to get into the superior sagittal sinus, the anastomosis of the MMA-ophthalmic artery, or the petrosal branch of the MMA.[2,15] Bilateral MMA embolization can also be performed in cases of bilateral CSDH in a single or staged procedure depending on the patient's condition.[2] As long as flow restriction to the SDH was achieved, the embolization could be done at the proximal, distal, or combined MMA. It wouldn't make a significant difference in the outcome.[16,17]

A meta-analysis found that using Onyx for embolization had the lowest rate of SDH recurrence and reoperation. On the other hand, using polyvinyl alcohol and coils had the best clinical outcome.18 N-butyl cyanoacrylate was also reported to be safe and effective.[19]

The patient's hemiparesis got better after MMA embolization and a unilateral symptomatic side burr hole craniostomy. Imaging tests three months after surgery showed that the SDH was absorbed. This procedure, which is minimally invasive, is a viable option for patients with SDH, particularly those who are predominantly elderly and at an increased risk of bleeding.[20]

This case report provided successful experience with the MMA embolization approach as an option to manage SDH. Further study is required to strengthen the evidence of this approach.

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CONCLUSION

The management of bilateral SDH remains a challenge. Since the discovery of MMA embolization, this technique has shown promise in reducing bleeding risk prior to surgical hematoma evacuation. It is also reported to be a safe neurointervention approach compared with the other operative approaches. The treatment of bilateral spontaneous SDH with surgery, endovascular therapy, or both requires further research. The goal is to identify potential confounding factors that could influence the outcome of the clinician's chosen treatment options.

Acknowledgement
We thanked the patient in this case for the consent to publish this article.

Conflict of Interest
The authors have no conflicts of interest to disclose.

Funding
This study was self-funded by the author.

Author Contributions
GWS provided data collection and produce the manuscript. PMJL and FSU reviewed the manuscript.

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