Review Article

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Featured minimally invasive therapeutic approach for chronic subdural hematoma: Embolization of middle meningeal artery - A narrative review

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Abstract:

Chronic subdural hematoma (c-SDH) is a frequent and serious neurological disease. It develops due to hemorrhage to the subdural space, mainly caused by head trauma. The middle meningeal artery (MMA) plays a critical role in the supply of blood to c-SDH. The decision on the type of treatment for c-SDH depends mainly on clinical and imaging evaluation. In cases in which patients are critically ill, the hematoma must be evacuated immediately. For this purpose, surgery is generally accepted as the mainstay of treatment. Among surgical techniques, twist-drill craniotomy, burr-hole craniotomy, and craniotomy are the three most used. The recurrence rate of c-SDH after surgery is an important problem with a rate of up to 30%. The technical success classification embolization of MMA (EMMA) has emerged as an effective and safe option for the treatment of c-SDH, especially those that recur. EMMA is commonly used as an adjunct to surgery or less frequently alone. The technical success of EMMA has been a promising minimal invasive strategy as an alternative or adjunctive therapy to surgical methods. Polyvinyl alcohol is the most widely used among various embolizing agents, including n-butyl cyanoacrylate, coil, and gelatin sponge. EMMA has been shown to prevent the formation or recurrence of c-SDH by eliminating blood flow to the subdural space. Complication rates are low. The large-scale comparative prospective will ensure efficacy and safety. This article aims to highlight the current information about EMMA in patients with c-SDH.

Keywords:

Chronic subdural hematoma, effectivity, embolization of the middle meningeal artery, polyvinyl alcohol, recurrence, surgery

Introduction

Chronic subdural hematoma (c-SDH) is a frequent and serious neurological disease.[1] Its yearly incidence is about 10/100,000 and is estimated to increase steadily in the next years.[2] This disease mainly affects older people. It is among the most common pathologies in emergency neurosurgery practice and is associated with significant morbidity and mortality.[3]

Subdural hematoma develops primarily due to the rupture of the bridging veins and small cortical arteries caused by head trauma, bleeding into the subdural space that places the space between the dura and the arachnoid material [Figure 1]. Subsequently, recruitment develops consisting of encapsulated blood, blood degradation products, and fluids in the subdural space over weeks and months. It is crescent in gross pathology, and this is the characteristic finding of c-SDH. The use of antiplatelet or coagulopathy increases bleeding tendency.[4] Although c-SDH can arise spontaneously, it is most likely caused by trauma, as the structure

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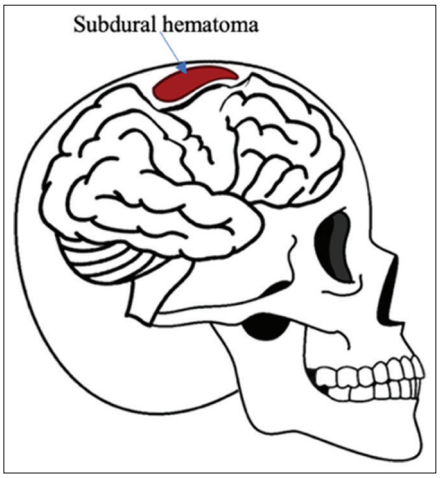

Figure 1: Schematic illustration of chronic subdural hematoma

of the subarachnoid layer is not very compact.[5] There are also other risk factors such as advanced age, male sex, intracranial hypotension, and cerebrovascular malformations.[6]

Injuries associated with c-SDH generally tend to be minor. The clinical forms of c-SDH range from asymptomatic to coma.[6] The standard treatment for symptomatic c-SDH is surgical drainage.[7] In c-SDH cases with an apparent compression effect, surgical treatment and irrigation are indicated.[4] Recurrence is an important problem in patients with c-SDH.[7,8] Diabetes, liver dysfunction, anticoagulant use, and postoperative air left in the subdural space are the factors that increase the risk of recurrence. There is no consensus against recurrence of c-SDH, although several strategies have been proposed.[9] Recently, embolization of middle meningeal artery (EMMA) has been a promising minimally invasive procedure that is often applied as an alternative or adjunctive therapy to surgery. EMMA has been shown to prevent the occurrence or recurrence of c-SDH by eliminating blood flow to the subdural space.[7,9]

This article aims to highlight EMMA, which has become prominent in patients with c-SDH in recent years, in light of current information.

Methodology

This narrative review article study was conducted by searching PubMed and Scholar Google. Based on the original full-text and the review articles on EMMA, the structure of the article was created and its writing was completed by selecting among the eligible ones.

Pathophysiology of chronic subdural hematoma

We now know that c-SDH generally develops as a result of trauma, particularly through blood leakage from the middle meningeal artery (MMA).[5,9] In immunology, it is also known that the primary purpose of the inflammatory response to any injury is to activate the immune system to repair it. However, long-term persistency of inflammation leads to pathology.[10] In this context, the key role of inflammation in c-SDH, which is primarily a trauma-related pathology, is not surprising.

Inflammatory cytokines such as interleukin-1 (IL-1), (IL-6), and (IL-8) play a key role in the formation of c-SDH, whereas anti-inflammatory cytokines such as IL-10 play a reciprocal role with inflammatory cytokines.[10] The initial injury caused by trauma to the dura border cell layer on the inner surface of the dura results in the release of inflammatory mediators and eventually recruitment of inflammatory and fibroblast cells in the subdural space. Activation of this cascade also induces the release of some vascular growth factors (including vascular endothelial growth factor [VEGF]), transforming growth factor-β1, and platelet-derived growth factor. When this initial damage is not repaired, the stimulated cycle of hyperfibrinolysis, inflammation, and angiogenesis results in subdural neomembrane development [Figure 2].[1] These neomembranes that form the capsule of the hematoma collection take weeks to months to develop and are immature forms.[4] Continued cytokine release further enhances angiogenesis and promotes blood flow to the subdural space.[9]

Not only inflammation but also some other factors take place in the formation of c-SDH. Of these, bleeding is an essential part in the formation of c-SDH. Coagulation and fibrinolysis, which cause the formation of fibrin/fibrinogen degradation products, are the two main biomolecular components of this process.[11]

Angiogenesis (neovascular formation) is another significant component in c-SDH formation. Most mediators such as angiopoietins, VEGF, and matrix metalloproteinases are involved in angiogenesis.[12,13]

Diagnostic workup

In the patient with suspected C-SDH, first the history, physical examination, and routine blood tests are evaluated.[14] Although magnetic resonance imaging gives more accurate results, computed tomography (CT) is preferred, especially in the initial evaluation because it is cheaper, more accessible, and faster.[15] A crescent shape that extends across the affected hemisphere is the best diagnostic signal [Figure 3].[4,14]

Therapeutic options in patients with chronic subdural hematoma

Deciding on the type of management for c-SDH is highly dependent on whether the patient is symptomatic or

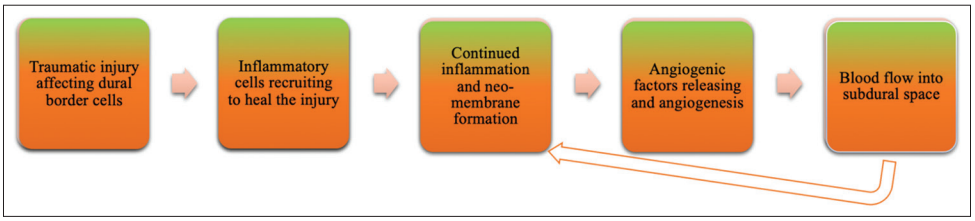

Figure 2: Summary of the pathophysiological processes of chronic subdural hematoma formation[10]

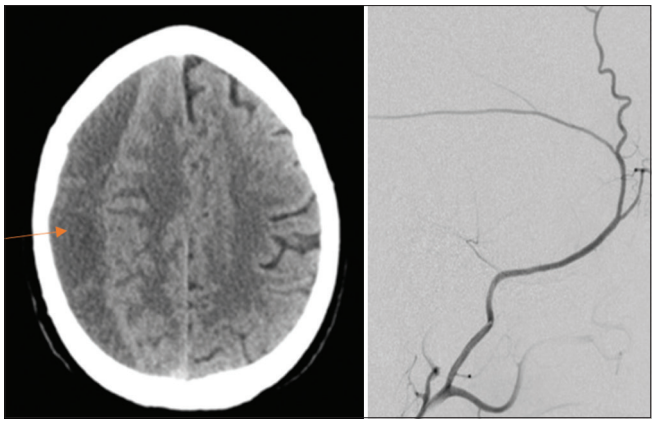

Figure 3: Representatives of computed tomography (left) of chronic subdural hematoma and angiogram of the middle meningeal artery (right)

asymptomatic and the size and compression effect of the hematoma on radiological evaluation. For asymptomatic cases, observation and follow-up are recommended. Spontaneous resolution may occur in cases with small hematomas.[16]

Pharmacological approaches such as an angiotensin-converting enzyme inhibitor or steroids remain the theoretical approach to the treatment of c-SDH. Clearly, more research is needed.[6] In critically ill patients, immediate evacuation of the hematoma is required to eliminate or reduce increased intracranial pressure. Surgery for this purpose is generally considered the mainstay of treatment.[10]

Although there is no evidence-based consensus, surgical treatment is indicated for c-SDH cases with significant mass effect (commonly for cases with >10 mm and >5 mm midline shift).[4,17,18] Among the surgical techniques, twist-drill craniotomy, burr-hole craniotomy (BHC), and craniotomy are the three most common used.[19]

After surgical intervention in c-SDH, perioperative complication rates have been reported that the rates of perioperative complication up to 32%.[20] Recurrence is also not uncommon. In fact, the recurrence rate after surgery can reach 30% depending on the surgical approach. Mortality rates were found to be similar when the three most common surgical techniques were compared.[21-26] Once surgical treatment has failed, recurrence is more common. This rate is estimated to reach 46%.[9]

The use of anticoagulants and antiplatelets plays a role in the development and recurrence of c-SDH.[27] For example, the risk of developing c-SDH is up to 42.5 times higher in warfarin use.[19] Therefore, the correct perioperative management of these drugs is extremely important. The use of these drugs should be weighed against the risk of recurrent bleeding and thromboembolic events.

Middle meningeal artery embolization in chronic subdural hematoma

Because of the high recurrence rate after surgical treatment, c-SDH remains a significant neurosurgical condition.[28] Various modalities have been investigated to overcome the challenge.[29] Unfortunately, the remaining options (such as peritoneal shunting, Ommaya reservoir application, or endoscopic drainage and debridement) are limited and have been proven effective.[4]

Recently, EMMA has shown that a minimally invasive strategy is an emerging effective and safe choice in c-SDH, especially recurrent ones.[30] It is used alone or adjunctive to surgery.[1,31] It is particularly useful when patients need to continue anticoagulant or antiplatelet therapy.[1]

The primary pathological mechanism for the formation of c-SDH is believed to be recurrent microhemorrhages from fragile new peripheral branches within the outer hematoma membrane of MMA. The essential concept of EMMA is to devascularize these immature capillary vessels and eventually prevent c-SDH recurrence.[28]

Why is the target of embolization of the middle meningeal artery?

MMA is the largest of the meningeal arteries. It is one of the branches of the maxillary artery. Through its branching arteries, blood is supplied to the dura mater and inner periosteum of the cranial bones.[4] After entering the dura mater, it is embedded in the groove of the inner skull face and follows a fixed route. This anatomical structure is suitable for being affected by common injuries during head trauma.[30] Therefore, MMA is a vital artery. It also provides blood flow to the c-SDH located in the middle anterior or middle posterior parts of cerebral convexity [Figure 4a].[4]

In terms of technical and clinical outcomes, middle meningeal artery embolization is the subject

Embolization has become common in patients with prolonged bleeding in various organs/tissues, including patients with c-SDH. Various liquid embolizing agents, such as polyvinyl alcohol (PVA), n-butyl cyanoacrylate (NBCA), and gelatin sponge, have been tested for EMMA in c-SDH patients, and significant success has been observed in preventing recurrence.[16] EMMA is performed by inserting and positioning a small guide catheter that supplies blood to the subdural hematoma in the MMA. Using image guidance, embolization material is injected by the guide catheter up to MMA occlusion.[32] Injecting particles or a special type of glue are released to stop bleeding that causes a subdural hematoma.[8] The procedure takes approximately 30 min and generally requires light sedation without general anesthesia.[32] Depending on the cause of the bleeding, it can be done alone or in combination with other surgical procedures.[1]

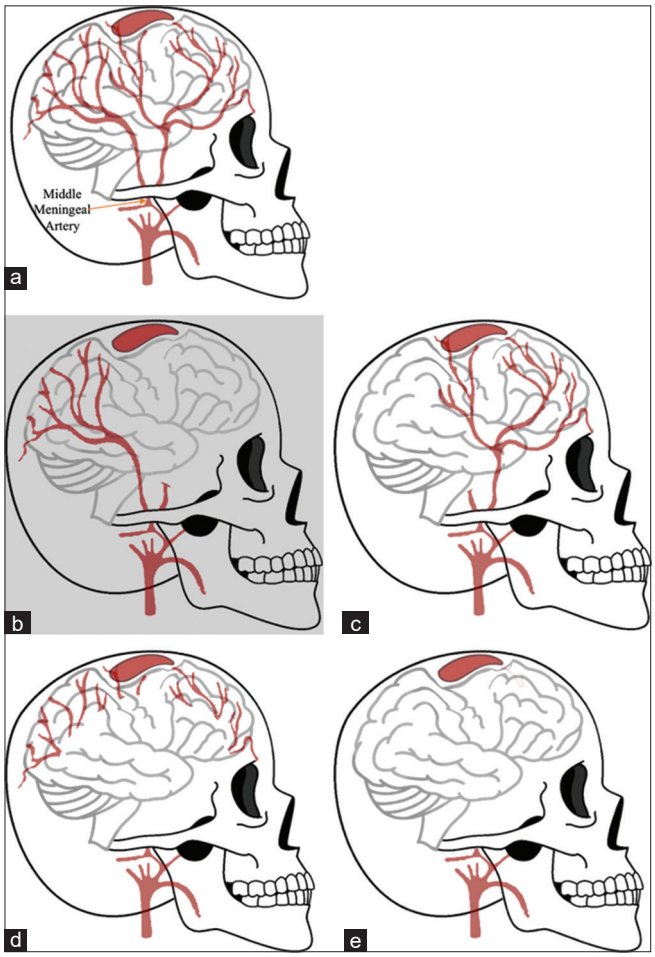

Figure 4: Middle meningeal artery (MMA) embolization technical success grading.[30] MMA: Middle meningeal artery. (a) Grade 0, No visible occlusion. (b) Grade 1. Anterior branch of MMA is occluded. (c) Grade 1. Posterior branch of MMA is occluded. (d) Grade 2. Distal ends of MMA are filled by the collaterals. (e) Grade 3. MMA is completely occluded

c-SDH arises from an active biological exudation process during the formation of loose vessels prone to spontaneous bleeding. Therefore, MMA occlusion not only prevents blood from being pumped into the subdural space but also affects this complex biology of the inner and outer membranes lining the c-SDH space.[4]

Currently, PVA is considered a promising permanent terminal embolization agent with good biocompatibility and is commercially available for use in interventional clinical practice.[33] Of these, Libro® Non-Adhesive Embolization [Figure 5] is an ethylene vinyl alcohol copolymer containing suspended micronized tantalum powder for radiopacity, manufactured by Invamed (Ankara/Turkey). The Libro® embolization procedure consists of MMA catheterization with access to the femoral or radial artery, followed by injection of 150–250 µm PVA.

Some clinical studies score their data on the basis of clinical neurological outcome. From these data, it appears that EMMA is an effective treatment option to prevent c-SDH recurrence and reducing hematoma volume.[29] Obviously, its technical success is also a major issue. In this context, Shankar and Kaderali proposed a grading system for EMMA from 0 to EMMA 3 in terms of technical success. The classification is based mainly on the extent of regional EMMA [Figure 4].[34]

EMMA Grade 0 indicates that there is no MMA embolization. The absence of embolization may be due to difficult access problems. EMMA Grade 1 refers to embolization of only the anterior or posterior branch of the MMA, again for technical reasons, such as access problems. EMMA Grade 2 indicates that most of the MMA region is embolized through the anterior and posterior branches. Grade 3 EMMA represents complete embolization of both the anterior and posterior branches of the MMA [Figure 4b-e]. EMMA Grade 3 is the ideal desired state.

Discussion

c-SDH, which is more frequently seen in older ages, is a common neurosurgical pathology. Although

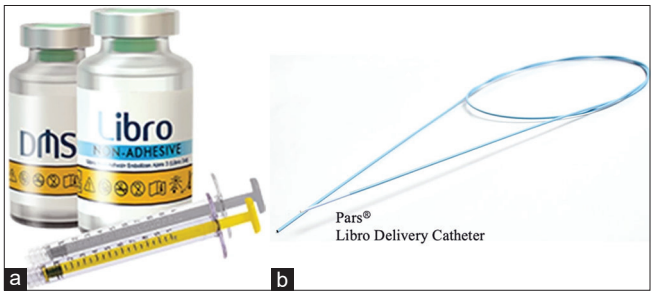

Figure 5: (a) Libro®: Non-adhesive liquid embolic agent for embolization of cerebral arteriovenous malformations. (b) Pars®: Delivery catheter for the Libro embolizing agent

evidence-based treatment selection can further improve patient outcomes, there are no established guidelines for its management and treatment practices vary.[35,36] Endovascular interventions, here EMMA, for c-SDH are partly new methods and are applied with different indications and techniques.[36] In general, c-SDH patients who are symptomatic, have a high surgical risk due to comorbid disease or advanced age, have recurrent/resistant hematomas, or are not sufficiently suitable for conservative treatment are considered suitable for EMMA. On the other hand, it should be noted that this approach may not be applicable in cases such as lack of suitable access routes for EMMA or patients at risk of allergy/hypersensitivity to the embolic agents used. Performing selective angiography before embolization may help guide the embolization process.[35]

In addition to the fluid particulates and liquids mentioned above, there are also various mechanical occlusive devices such as coils, plugs, stents, and balloons. In embolization application, it should be aimed to completely block the target vascular system to prevent recurrence.[37]

Liquid-based embolic commercial products are considered simple to use, potentially safe, and effective endovascular procedures. For example, NBCA can be used at low concentrations in more peripheral areas. However, care should be taken in terms of aberrant effects. NBCA can also be combined with agents such as trisacryl gelatin microspheres.[38]

Several commercial systems using PVA, such as Libro® Nonadhesive Embolization (Invamed, Ankara) and ONYX™ Liquid Embolic System (Medtronic), are available.[39] Coils, which are commonly used among mechanical plugs, have disadvantages such as incomplete closure, occlusion of nontarget vessels due to coil migration, and aneurysm rupture due to excessive radial force. Recently, plugs that can fill the vascular space without applying significant force to the vessel wall have become an important alternative to traditional coil embolization, thanks to their low radial force, and shape memory polymer structure.[40]

Although EMMA is often used as an adjunct to surgery, it is also used as the primary approach or rescue treatment for recurrent hematomas.[4] Studies conducted around the world mainly focus on the assumption that EMMA will reduce the rate of recurrence in high-risk patients and improve neurological outcomes by reducing the need for reinterventions, hospitalizations, and postoperative complications.[8] This ultimately means better results and lower costs.[41] In addition to being a minimally invasive method, EMMA is technically simple, relatively safe, and potentially eliminates the need for surgical drainage. It is stated to reduce the rate of recurrence by 20%.[34] However, information on the effect of embolization on the effect of recurrent hematomas is limited.[29]

Okuma *et al*. showed that EMMA can be effective when used as primary therapy in preventing recurrent c-SDH or rehospitalization in resistant case.[38] Shotar *et al*. reported that postoperative EMMA reduced the c-SDH recurrence rate from 14% to 4%.[42] Mino *et al*. observed the disappearance of common recurrent abnormal vascular spots around MMA observed after BHR with CT scanning.[43]

In a systematic review by Court *et al*., 190 patients with c-SDH who underwent EMMA were evaluated. Resolution occurred in 96.8% of these patients, 81.3% of whom were symptomatic. In most patients (83%), PVA particles were used, and no reported complications related to embolization procedures were reported.[44] Kan *et al*. reported that after EMMA, 70.8% of the patients had a reduction in hematoma diameter >50% and only 6.5% of the patients required surgery.[45] Orscelik *et al*. found similar results to those of Kan *et al*. in their study, both in terms of reduction in hematoma diameter and rescue treatment. In the study of Orscelik *et al*., it was also stated that recurrence rates were higher in patients with comorbid diseases such as cardiovascular disease, clotting problems, chronic alcoholism, and liver cirrhosis.[35]

Ban *et al*. observed a 1.4% treatment failure rate in their study in 541 patients who underwent EMMA. 27.5% of them were surgically removed.[46] Kim reported that one in 20 patients who underwent EMMA had recurrence, which later resolved spontaneously. On the other hand, the recurrence rate was found to be 33.3% in patients with BHC.[47]

In a meta-analysis study involving 902 patients, EMMA was found to have a lower probability of c-SDH recurrence (5% vs. 22%) and surgical rescue (4% vs. 16%) than conventional treatment. In-hospital complication rates were reported to be similar between the two groups. In this study, the combined rate of in-hospital complications was found to be 2% versus 5% and not statistically significant.[36] In the study by Catapano *et al*., it was reported that the rate of complete or near-complete resolution of hematoma was 92% and the average size of c-SDH decreased from 16.9 mm to 1.0 mm at 180-day follow-up after EMMA.[48] Scoville *et al*. reported up to 50% reduction in hematoma thickness at 180-day follow-up after EMMA. They stated that there was no statistically significant difference between particle and liquid embolizates, not only in terms of hematoma thickness but also in terms of complications.[49]

In several studies, it was assessed the functional outcomes of EMMA using the Modified Rankin Scale (mRS), and

they were reported a significant improvement in mRS score.[35,36,45,50]

Although c-SDH is mostly seen in older ages, EMMA is also of particular importance in pediatric cases. In this group, a history of ventriculoperitoneal shunt, blood coagulation disorder, and trauma are the main reasons responsible for the development of c-SDH. In a meta-analysis study, the EMMA success rate in the pediatric group was reported as 88.8%. It has been stated that the time to success varies between 2 and 9 months.[51]

Limitation of the study

This study has some limitations. Studies evaluating surgical and embolization methods used in the treatment of c-SDH to date in detail are limited. This study is also a traditional review type.

Conclusions

The available data reveal that EMMA could be a promising, effective, and safe treatment modality for c-SDH. It is effective in reducing the size of the hematoma and improving clinical outcomes. Complication rates are low, and hospital stay is short. It has an important alternative potential for a suitable indication. To further improve the outcomes of MMA embolization, larger scale comparative prospective studies and ultimately evidence-based patient selection are required.

Author contributions

RD designed, conceptualized, searched for data, wrote and finalized.

Ethics committee approval

This manuscript, which is a review article, does not include studies with human or animal participants. Therefore, the approval of the ethics committee is not required.

Patient consent

Not applicable.

Data availability statement

The author confirms that data supporting the findings of this study are available in the article.

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Nil.

Conflicts of interest

RD is the president of Invamed (Ankara, Turkey).

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