 doi: 10.2176/jns-nmc.2022-0007

NMC Case Report Journal 9, 151-155, 2022

Chronic Subdural Hematoma after Craniotomy with Preoperative Embolization of Middle Meningeal Artery: A Case Report

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Abstract

Endovascular embolization of the middle meningeal artery (MMA) has been reported as an effective method for treating chronic subdural hematoma (CSDH); however, its preventive effect on CSDH following craniotomy is unknown. We present a case in which MMA embolization was ineffective in preventing CSDH following craniotomy. A 56-year-old man who complained of diplopia was diagnosed with sphenoid ridge meningioma with a 3-cm diameter. MMA embolization prior to the operation and total surgical removal of the tumor were performed. Two months postoperatively, the patient complained of headache and hemiparesis of the left side. CSDH with a 15-mm thickness and a midline shift was observed. MMA embolization before inflammation may not play a role in preventing CSDH development because MMA embolization is considered effective in CSDH because it is associated with the blood supply of neovessels that are newly formed due to inflammation. Therefore, MMA embolization might not be effective in preventing the occurrence of CSDH following craniotomy.

Keywords: middle meningeal artery, chronic subdural hematoma, embolization, craniotomy

Introduction

Endovascular embolization of the middle meningeal artery (MMA) has been reported to be an effective method for recurrent or refractory chronic subdural hematoma (CSDH).[1-7] Surgical drainage is the first-line treatment for CSDH; however, some recent reports have suggested the efficacy of MMA embolization.[8-10] Although CSDH may also develop following neurosurgery,[11-17] there are no reports discussing the preventive effects of CSDH postoperatively. We present a case in which MMA embolization was ineffective in preventing CSDH after the removal of a meningioma.

Case Report

A 56-year-old man who had previously undergone coronary stent placement and continued taking aspirin (100 mg) presented with a complaint of diplopia. Magnetic resonance imaging revealed a tumor with a 3-cm diameter on the right sphenoid ridge (Fig. 1A). The selective angiogram of the right MMA depicted tumor stains from the anterior convexity branch (Fig. 1B). Endovascular embolization of the right anterior convexity branch of the MMA was performed using tris-acryl gelatin microspheres (TAGM) (Embosphere, Nippon Kayaku, Tokyo, Japan), platinum coils of Barricade Coil (Balt USA LLC, Irvine, California, USA), ED COIL (Kaneka Medix Corporation, Osaka, Japan), and SMART COIL (Penumbra Inc., Alameda, California, USA) (Fig. 1C). Total surgical removal of the tumor was performed 1 day after embolization. We were under the impression that there was less bleeding from the dura intraoperatively than other craniotomies due to the prior MMA embolization. After the removal of the tumor, the tumor tissue was scraped off from the dura at the attachment and then coagulated and cauterized without dural

Received January 14, 2022; Accepted March 22, 2022

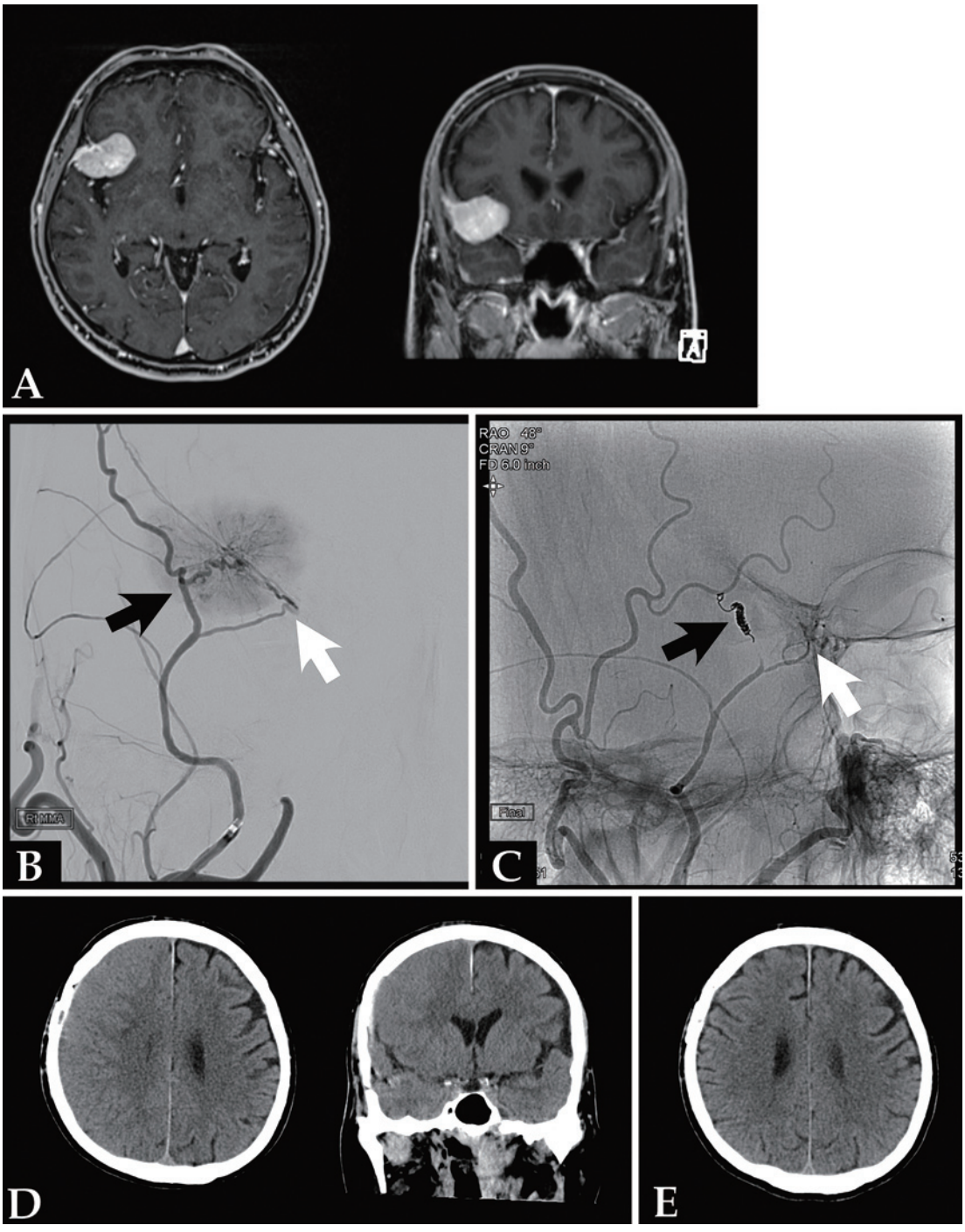

Fig. 1

A: Brain magnetic resonance imaging of contrast-enhanced T1-weighted image showing right sphenoid ridge meningioma. B and C: Selective angiogram of the right middle meningeal artery before and after embolization. B: Preoperative. Anterior convexity branches (white and black arrows) depict the vascular supply of the tumor. C: Postoperative. Embolization by acrylic copolymer (white arrow) and detachable coils (black arrow) succeeded in interrupting the blood supply to the tumor. D: Brain computed tomography (CT) showing right chronic subdural hematoma. E: Postoperative CT showing no subdural hematoma recurrence.

excision. Arachnoid plasty was not performed. Aspirin was discontinued 5 days preoperatively in consultation with a cardiologist and resumed 1 day postoperatively. The patient was discharged without neurological deficits. The pathological findings indicated an atypical meningioma of World Health Organization grade II. Two months postoperatively, the patient started experiencing a slight headache and left hemiparesis. A computed tomography (CT)

scan revealed a right CSDH with a 15-mm thickness and a midline shift (Fig. 1D). Burr hole irrigation was performed, and the headache and paresis immediately disappeared. No hematoma has been observed to date on the follow-up CT of 3 months postoperatively (Fig. 1E).

Discussion

Previous research has identified that CSDH is an angiogenic and inflammatory disease. After an inciting event, such as trauma or spontaneous tearing of bridging veins, an inflammatory response occurs, which involves migration of inflammatory cells derived from the dural border cells, followed by fibroblasts along the dura outermembrane development.[18,19] Vascular endothelial growth factor is released, leading to neovascularization of the membrane.[20] It is theorized that the continuous exudation and oozing from these neovessels within the subdural membrane leads to the generation and growth of CSDH over time.[21] Pathological analysis of the membranes revealed giant capillaries, inflammatory cell infiltration, neovasculature, and anastomosis with the MMA branches.[22] These capillary networks are considered the cause of the findings of "cotton-wool like stains" seen with superselective angiograms of MMA,[1,3,6,23] suggesting that MMA embolization eliminates the source of rebleeding in the neovasculature of the membrane and could allow the hematoma to be resorbed and the brain to expand over time. In the present case, MMA embolization was performed for the meningioma, resulting in a situation similar to that following endovascular treatment for CSDH. However, no local inflammation or angiogenesis occurred because CSDH did not exist at the time of MMA embolization. MMA embolization is considered effective in CSDH as it is associated with the blood supply of neovessels that are newly formed due to inflammation. MMA embolization before inflammation may not play a role in preventing CSDH development. There is still no consensus on the proper timing of MMA embolization for CSDH. Premature MMA embolization that does not involve MMA to hematoma may not contribute to the suppression of CSDH development. Thus, it is important to understand that embolization of the MMA, which is involved in neovascularization, is effective in treating CSDH, but MMA embolization itself may have no preventive effect.

On the other hand, it should be noted that the present case is not a common CSDH occurring after trauma but after craniotomy. It has been reported that CSDH develops following craniotomy, with an incidence of 0.3%-0.8%.[9,15,16] The cause of CSDH after craniotomy is not well understood owing to its low prevalence; however, cerebrospinal fluid from the cistern may be one cause. The incidence of postoperative CSDH is highest in aneurysm clipping surgery.[9,15,16] Aneurysm surgery requires opening of the arachnoid membrane, and cerebrospinal fluid flows into the subdural space through this artificial arachnoid tear. It is speculated that cerebrospinal fluid leakage causes subdural effusion, followed by inflammation and angiogenesis, resulting in CSDH.[10,17] In the present case, the operation was not aneurysm clipping, but it was required to open the arachnoid for the removal of the sphenoid ridge meningioma. This indicates that the conditions were conducive for CSDH to occur following craniotomy. The pathogenesis of CSDH after craniotomy and after trauma may be different; therefore, arachnoid plasty might be useful in preventing CSDH occurrence in cases where wide opening of the cistern is required for tumor removal.[17]

As a limitation of this report, the dural blood flow was not evaluated *via* angiography or three-dimensional CT angiography at the onset of CSDH. This prevents a detailed study of the possibility of reconstitution of blood flow from anastomotic vessels or recanalization of embolized MMA.

It has been established that there are anatomical anastomoses in the dura.[24] Therefore, it is possible that the posterior convexity branch of the MMA or the accessory meningeal artery, which had not been embolized, supplied blood to the dura. Alternatively, it may have complemented dural blood flow from the recurrent meningeal artery and the meningolacrimal artery, which are potential anastomoses between the internal carotid artery and the MMA. In particular, a previous report suggested that the posterior convexity branch of the MMA is involved in CSDH in some cases.[25] Thus, the unembolized posterior convexity branch artery of the MMA may have contributed to CSDH development. In addition, meningiomas may have markedly developed dural blood supply from MMA due to stimulation of angiogenesis, including vascular endothelial growth factor.[26] Although this abnormal increase in signaling has been eliminated after tumor resection, the dural vessels that developed preoperatively may have contributed to the dural blood flow reconstruction. It is also necessary to consider the possibility of recanalizing MMA after embolization. In the present case, we used TAGM, which are embolic particles used for tumor embolization, and platinum coils. Embolic particles commonly used for MMA embolization include polyvinyl alcohol (PVA) and TAGM, the latter being more uniform in particle size and having a consistent cross-sectional diameter, and are capable of embolizing distal tissue. Unexpected proximal arterial occlusion by large particles is responsible for the immediate revascularization of the tumor mass by collateral meningeal vessels bypassing the occlusion site[27]; therefore, TAGM may exert a higher embolic effect.

In previous studies, PVA was frequently used for embolization for CSDH,[4,8,10] and there are few studies using TAGM.[28] Although there are no reports of direct comparison between the two embolic particles, the effectiveness of embolization is high in both, but at least, TAGM seems to have a higher embolization effect.

In previous reports, MMA embolization for CSDH was

well in control at 2 months after the embolization, whereas in the present case, the patient developed CSDH at that time, although MMA embolization was performed. We speculated that neovascularization of the dural blood flow due to the unembolized vessels plays a major role, considering that the intraoperative findings of tumor resection did not indicate embolization failure. Nevertheless, it is not beyond speculation because the present case did not evaluate the dural blood flow at the CSDH onset. Therefore, further studies are required to evaluate the dural condition at the onset of CSDH.

In addition, we did not perform a histopathological examination of the dura in the present case. Therefore, future studies should consider performing dural biopsy for the evaluation of tissue inflammation and vascular structure, which may lead to new findings.

Conclusion

Here, we present a case of CSDH following MMA embolization. Although the embolization of the MMA may be effective in treating CSDH, it may not prevent the occurrence of CSDH after craniotomy.

Acknowledgments

We would like to thank Editage (www.editage.com) for English language editing.

Ethics Approval

The study was approved by the National Hospital Organization Kyushu Medical Center Research Ethics Board (#19C069). The patient has consented to the submission of the case report for journal publication.

Conflicts of Interest Disclosure

The authors report no conflict of interest concerning the materials or method used in this study or the findings specified in this paper.

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