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Case Reports & Case Series

Refractory chronic subdural hematoma supplied by contralateral middle meningeal artery: Case report

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ABSTRACT

The authors reported a case of refractory chronic subdural hematoma (CSDH) in a 56-year-old male after minor head trauma. Digital subtraction angiography (DSA) of the brain revealed contralateral middle meningeal artery (MMA) supply in the parafalcine hematoma.

1. Introduction

CSDH is one of the most common neurosurgical conditions, especially in the aging population. Although the surgical techniques are simple, recurrences remain one of the challenges in the treatment [1]. Here, we present a patient with refractory CSDH that was supplied by the contralateral middle meningeal artery.

2. Case report

This 56-year-old male without past medical history presented to our emergency department with progressive headache and consciousness disturbance. He had a minor head injury after a fall one week ago.

On neurological examination, the patient appeared somnolent but oriented. Right eye ptosis and left hemiparesis were noted. Brain computed tomography (CT) revealed right frontal-parietal-temporal and parafalcine subdural hemorrhage (Fig. 1). Single burr-hole drainage was performed, and follow-up brain CT scan showed resolution of SDH and mass effect.

Two days after removal of the subdural drain, the patient's symptoms reappeared. Brain CT scan disclosed recurrent SDH with apparent fluid level (Fig. 2). Subdural drainage via previous Burr hole was done again. However, recurrent headache happened three days after the drain tube was clamped for testing. Repeated brain CT scan confirmed reaccumulation of subdural hematoma.

Under the suspicion of a traumatic aneurysm, we obtained digital subtraction angiography (DSA) of the brain. It revealed a vascular stain which was mainly supplied by the left middle meningeal artery at the right parafalcine region. The DSA imaging resembled a meningioma (Fig. 3A, B). Brain magnetic resonance imaging (MRI) demonstrated a wide base and heterogeneously enhanced lesion that was attached to the anterior cerebral falx in the right parasagittal region (Fig. 4A, B).

Craniotomy crossing the midline was performed and bilateral MMA were coagulated diffusely. The parafalcine lesion was a hematoma encapsulated by outer and inner membranes, which were all resected completely. Pathological examination of these specimens all showed a hematoma with old and recent membranous fibrovascular tissue without any tumor cells. The patient was symptom-free after the craniotomy procedure. Post-operation 3 weeks brain CT scan and post-operation 6 weeks MR images follow up showed resolution of SDH. Brain CT was performed three weeks postoperatively and MR imaging was done six weeks postoperatively, and both showed resolution of SDH.

3. Discussion

A variety of the explanations for the pathogenesis of the formation of CSDH has been proposed in past decades [2–4]. Nevertheless, no consensus about the pathophysiology of CSDH has been reached. Nakaguchi et al. advocated three different phases of hematoma development based on the neuroimaging: homogenous stage (Stage 1), separated or multilayered stage (Stage 2), and trabecular stage (Stage 3) [4]. Tanaka et al. presented a chain of developing processes of CSDH. Development of a neomembrane at the inner surface of the dura mater was induced by inflammatory reaction to chemical mediators or various cytokines in the subdural fluid collection. Sinusoid channels formed in the neomembrane cause frequent minor bleeding. This process splits the neomembrane into outer and inner membranes to encapsulate the unclotted hematoma [3]. Edlmann et al. summarized the so-called CSDH cycle, noting the key process involved in

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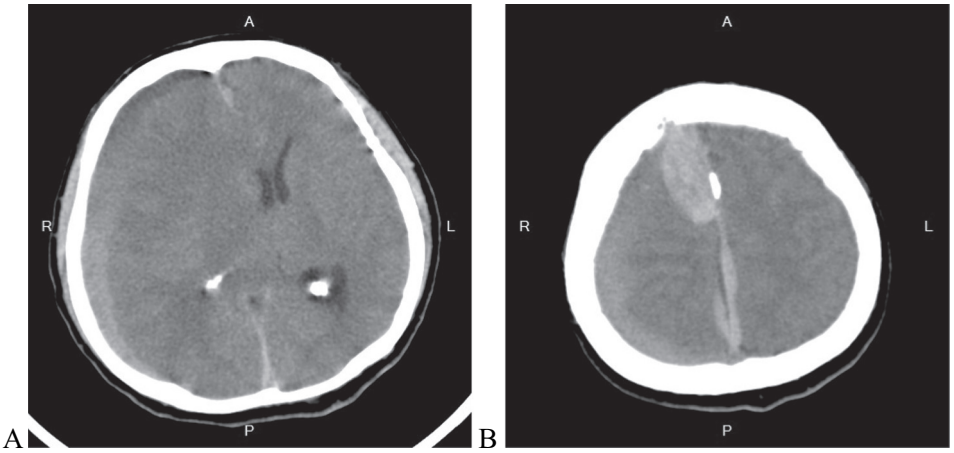

Fig. 1. Brain CT showed iso- to hyperdense frontal-temporal-parietal (A) and parafalcine (B) subdural hematoma.

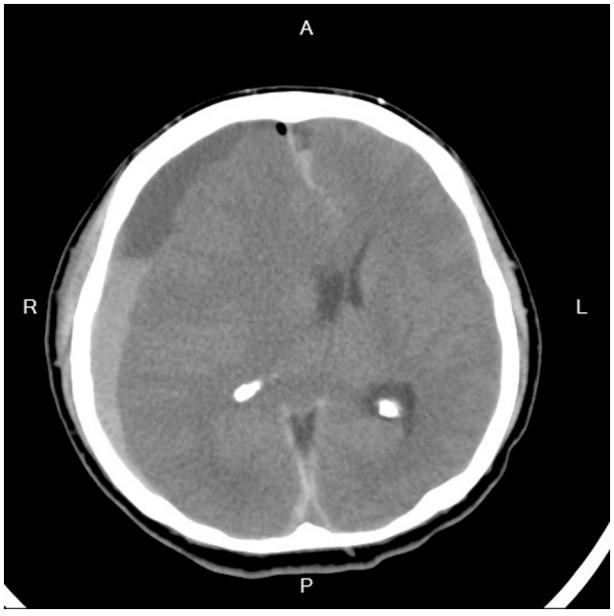

Fig. 2. Brain CT showed reappearance of subdural hematoma two days after the subdural drain removal.

CSDH development included angiogenesis, fibrinolysis and inflammation [2].

Angiography is rarely arranged for cases of CSDH. However, recent studies revealed that the MMA plays a role in refractory CSDF cases [5]. Mandai et al. discovered contrast material in the subdural hematoma cavity from the brain CT scan obtained immediately after MMA embolization [7]. Tanaka et al. found diffuse dilation of the MMA and scattered abnormal vascular networks, which seemed to be macrocapillaries in the outer membrane [6]. In the study of Takizawa et al., they found the diameters of the MMAs on the CSDH side were significantly larger than the diameters of the MMAs on the non-CSDH side [5]. The pathophysiology of the MMA enlargement was unclear but the MMA was suspected to be the contributing artery of the SDH. There are nine previous reports on MMA and recurrent CSDH [5,9]. However, no contralateral MMA enlargement or embolization has been documented. In our case, DSA showed contralateral MMA supply into the parafalcine SDH that caused recurrence. This phenomenon has not been reported in the current literature.

Single burr-hole surgery with irrigation and closed-system drainage is an effective initial treatment for symptomatic CSDH. Once the hematoma is removed, a good outcome is usually obtained. However, previous reports revealed that about 3.7% to 30% of patients have recurrence of CSDH [9]. In those with recurrent CSDH, different surgical methods such as repeated burr-hole trephination, membranectomy via craniotomy, implantation of reservoir or subdural-peritoneal shunt, and endoscopic surgery have been discussed [1]. A meta-analysis by Sahyouni et al. suggested that craniotomy with membranectomy yields a lower likelihood of CSDH recurrence and secondary intervention [9]. Embolization of MMA emerged as a new therapeutic modality. Most of these cases required additional post-embolization drainage [8,10]. Further randomized control trials are necessary to prove the efficacy of MMA embolization in recurrent CSDH.

Due to the uncertainty of the parafalcine lesion as well as the symptomatic midline shift that occurred in our patient, we did not take the risk of performing MMA embolization alone. Instead, we chose to perform a large craniotomy for the purpose of obtaining a pathological tissue specimen and decompression of the mass effect caused by SDH. We coagulated the contralateral and ipsilateral MMA on the dura during the process, believing that the MMA might bring blood influx through the microcapillaries into the hematoma cavity. The outer membrane is highly vascularized and exudation from macrocapillaries is critical in CSDH enlargement. The inner membrane is avascular and adherent to the underlying arachnoid over the cortical surface. Resection of the outer membrane may mitigate repeat bleeding and prevent recurrence while resection of the inner membrane allows the underlying brain to re-expand [9]. Hence, we also performed the outer and inner membranectomy during the procedure.

4. Conclusion

We believe that the MMA contributes to the mechanism of the formation of the inner and outer membranes of CSDH. We suggest performing an angiographic study in every case of refractory CSDH. Endovascular embolization of MMA may be attempted in those recurrent cases without symptoms. MMA coagulation and membranectomy through craniotomy is an effective treatment in refractory symptomatic cases.

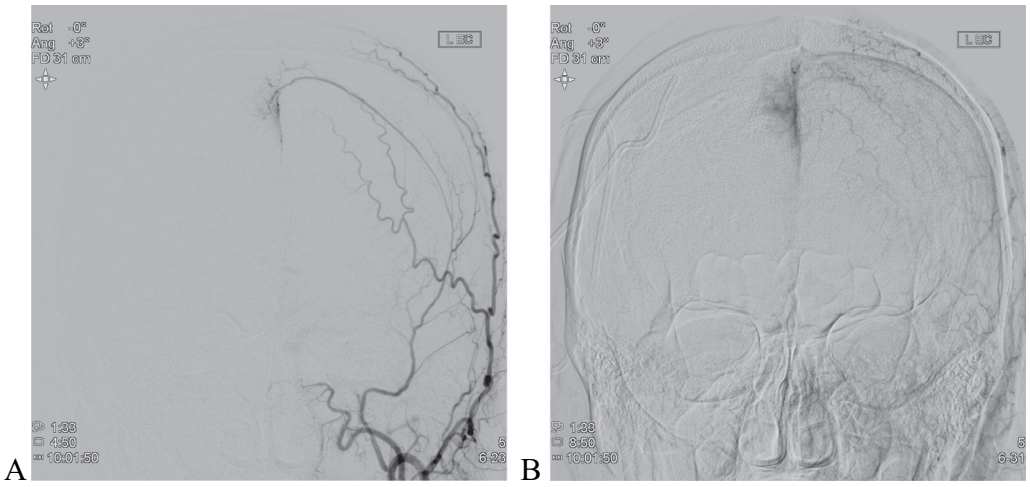

Fig. 3. DSA revealed contralateral middle meningeal artery supplying parafalcine lesion.

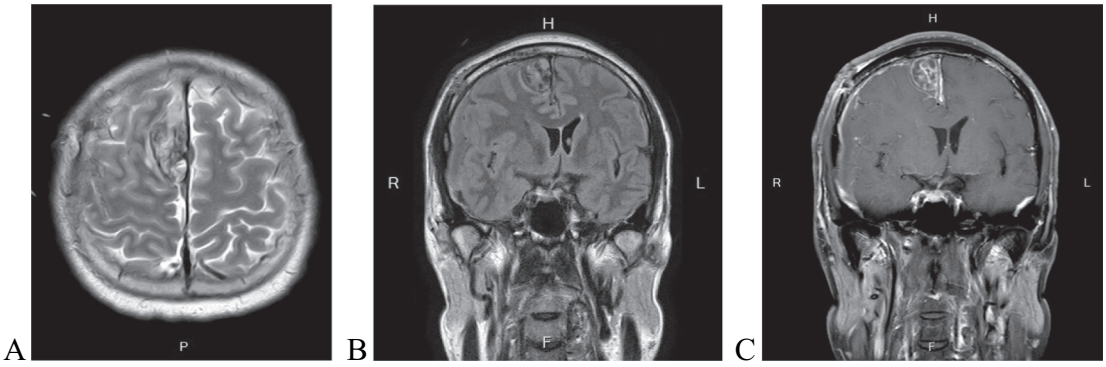

Fig. 4. Brain MRI. A) Axial view of T2 weighted image. B, C) Coronal view of T1 weighted image and T1 enhanced image.

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