Traumatic Acute Subdural Hematoma Extending from the Posterior Cranial Fossa to the Cerebellopontine Angle

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Posterior cranial fossa subdural hematomas and extension of the subdural hematoma to the cerebellopontine angle is rarely seen and the concurrent development of acute peripheral facial palsy and the management strategy have not previously been reported in this pathology because of its rarity. We present this case to emphasize that minor head trauma may lead to a posterior cranial fossa hematoma extending to the cerebellopontine angle and cause peripheral facial palsy in patients using aspirin (acetylsalicylic acid). In addition, partial evacuation and waiting for the resorption of the hematoma may help to prevent damage to the 7th and 8th cranial nerves.

KEY WORDS: Antiplatelet agent · Cerebellopontine angle · Facial nerve · Head injury · Posterior fossa · Subdural hematoma.

INTRODUCTION

There is no exact algorithm for the surgical indication parameters in patients with acute posterior fossa subdural hematomas extending to the cerebellopontine angle due to the rarity of this pathology. The general concept is that an acute posterior fossa subdural hematoma is a neurosurgical emergency as it may cause brainstem compression and acute fourth ventricle obstruction, and finally result in sudden worsening of the clinical condition of the patient and death, and that it should therefore be evacuated promptly via the suboccipital approach whenever it is diagnosed in patients with the radiological signs of pressure effects on the fourth ventricle or brain stem or the surrounding cisterns, even when the patient has no neurological deficits. D’Avella et al. classified patients with posterior fossa subdural hematomas into two groups. Conscious patients with no signs of pressure effects on the brainstem, fourth ventricle or basal cisterns, and no subdural hematoma thicker than 1 cm can be followed conservatively. In contrast, emergency surgery should be performed for patients with mass effect on the brainstem, fourth ventricle or basal cisterns without considering their clinical condition. In this report, a case of posterior fossa subdural hematoma extending to the cerebellopontine angle in a patient who presented with ataxia, confusion and right peripheral facial palsy while on long-term use of an antiplatelet agent (aspirin) is described and possible surgical approaches are discussed based on the available literature and our experience in this case.

CASE REPORT

A 78-year-old woman presented to our hospital with minor occipital head trauma after falling down on a slippery floor while walking. Her clinical condition was stable and she admitted to the Emergency Department ward. Her Glasgow coma scale score was 12. Pupils were isocoric, but both reacted slowly to light. She was not able to walk due to ataxia. She also had ecchymosis of the left mastoid bone region. The patient developed right-sided peripheral facial paralysis at the Emergency Department during admittance. She had no signs of injury except the ecchymosis on the left mastoid region. Her medical history
revealed that she used aspirin 100 mg per day. Her blood chemistry and complete blood count showed no abnormality, but her epinephrine-induced thrombocyte aggregation test was 49% (normal level: 55-85%). A cranial CT scan and CT angiography performed immediately after admission showed an acute posterior fossa subdural hematoma extending to the cerebellopontine angle and also pressing the fourth ventricle with no vascular abnormality (Fig. 1, 2). A thrombocyte infusion was given, and the patient immediately underwent right suboccipital craniectomy due to her deteriorating clinical condition. The suboccipital bone was resected to the mastoid process (Fig. 3). The subdural hematoma was completely evacuated, but the cerebellopontine angle part of the hematoma was only partially evacuated. Her right-sided peripheral facial para-

Fig. 1. Unenhanced preoperative computed tomography scan showing a 17 mm-wide right hemispheric subdural hematoma extending to the right cerebellopontine angle and almost completely obstructing the fourth ventricle.

Fig. 2. Contrast enhanced three-dimensional computed tomography angiography showing no vascular abnormality at the posterior cranial fossa.

Figs. 4. Unenhanced computed tomography scan obtained 20 days after the surgery showing complete resolution of the residual hematoma.
lysis resolved completely in 5 days and the ataxia partly improved, but she was not able to walk without assistance during the postoperative period. The first postoperative follow-up cranial CT performed 12 hours after the operation showed no mass effect on the brainstem or the fourth ventricle, but there was a residual cerebellopontine angle hematoma (Fig. 3). The second postoperative follow-up cranial CT performed 20 days after the operation showed no hematoma in any part of the posterior fossa (Fig. 4). Her clinical condition was stable and she was able to walk without assistance three months after the surgery.

DISCUSSION

The management of an acute subdural hematoma of the posterior cranial fossa is more complicated than supratentorial cases. There is no exact treatment algorithm for a posterior cranial fossa subdural hematoma, especially if the subdural hematoma extends to the cerebellopontine angle at the posterior cranial fossa, due to the rarity of this pathology. The most important prognostic factor in patients with a posterior cranial fossa subdural hematoma is the clinical condition of the patient at the time of surgery. In terms of surgery, acute subdural hematomas of the posterior fossa are classically thought to be neurosurgical emergencies as they cause abrupt deterioration and death from brainstem compression. Prompt evacuation of the posterior cranial fossa hematoma through a suboccipital craniectomy is therefore a widely accepted method among neurosurgeons. Ashkenazi et al. reported that a conservative approach may also be advocated in posterior cranial subdural hematomas which are less than or equal to 1 cm in thickness with no pressure effect on the brain stem and fourth ventricle and no signs of neurological deficit. However, radiological signs of a mass effect on the posterior fossa such as fourth ventricle distortion and compression of the brainstem or distortion of the surrounding cisterns prompt surgical evacuation even in patients with no neurological deficit. D'Avella et al. reported that patients with a Glasgow coma scale score less than 8 have a high percentage of bad outcome, and these patients may have pressure effects at the posterior cranial fossa due to the subdural hematoma. They also noticed that patients with a Glasgow coma scale of 8 or more have more chance of recovery, and surgical evacuation should be performed if these patients have a subdural hematoma thickness over 1 cm. Our presented case had progressive neurological deficits such as ataxia and peripheral facial paralysis. In addition, cranial CT showed a mass effect and the hematoma extending to the right cerebellopontine angle (Fig. 1). We promptly operated on this patient while administering thrombocyte solution to neutralize the effects of aspirin on the thrombocytes. The obvious feature of our case was the extension of the posterior fossa subdural hematoma to the cerebellopontine angle. We performed right suboccipital craniectomy up to the mastoid bone. We evacuated the subdural hematoma, and also retracted the cerebellum to reach the cerebellopontine angle. We then aspirated the hematoma but did not try to complete its evacuation to prevent inadvertent injury to the 7th and 8th cranial nerves. The patient was in stable condition after the operation and the peripheral facial paralysis recovered fully but her ataxia continued in the early postoperative period. Follow-up cranial CT performed 12 hours after the surgery showed no signs of mass effect on the brainstem and fourth ventricle, but the hematoma persisted at the cerebellopontine angle (Fig. 3). The second follow-up cranial CT performed 20 days after the surgery showed no signs of residual hematoma or new hemorrhage in any part of the posterior cranial fossa (Fig. 4). In conclusion, our presented case had an uncommonly seen subdural hematoma of the posterior fossa extending to cerebellopontine angle and was successfully managed. Detailed information on the management of posterior fossa hematoma extending into cerebellopontine angle is lacking in the present literature. Ulivieri et al. advocated conservative management in their patient with a posterior fossa subdural hematoma following anticoagulant therapy. We believe that surgical evacuation of a subdural hematoma of the posterior cranial fossa extending into the cerebellopontine angle is the treatment of choice in patients with progressive neurological deficits; however, it is important to protect the cranial nerves during the evacuation of the cerebellopontine angle hematoma. Suboccipital craniectomy and evacuation of the subdural hematoma of the posterior fossa without complete evacuation of the cerebellopontine angle hematoma in our case provided considerable pressure relief for the posterior fossa.

CONCLUSION

The experience we gained from the presented case is that minor head trauma can lead to a posterior fossa subdural hematoma extending into the cerebellopontine angle and that complete evacuation of the cerebellopontine angle hematoma carries a risk of injury to the 7th and 8th cranial nerves. The best management strategy for these patients may therefore be partial evacuation of the cerebellopontine part of the subdural hematoma of the posterior fossa and waiting for spontaneous resolution of the residual hematoma.
References