A Case of the Growing Epidural Hematoma Treated with the Embolization of the Middle Meningeal Artery

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Objective: We report a case of the growing acute epidural hematoma treated by embolization of the middle meningeal artery.

Case Presentations: An 18-year-old man was admitted with head trauma sustained in a traffic accident. Initial CT revealed a linear fracture and a small epidural hematoma in the left frontal region. As the hematoma was small and neurological deficits were absent, it was treated conservatively. A second CT assessment 3 h later showed that the hematoma had enlarged and external carotid angiography showed extravasation from the frontal branch of the middle meningeal artery. Embolization of the middle meningeal artery gradually decreased the hematoma.

Conclusion: Embolization of the middle meningeal artery effectively treated a small acute epidural hematoma.

Keywords ▶ epidural hematoma, middle meningeal artery, embolization

Introduction

In patients with acute epidural hematoma accompanied by injury of the middle meningeal artery or venous sinus, hematoma often enlarges, and craniotomy is often necessary. However, if the hematoma is small, an initial option may be observation.

We report a patient in whom a small acute epidural hematoma enlarged with time, but embolization of the middle meningeal artery rather than craniotomy was performed in consideration of the patient’s condition and volume of hematoma with a favorable outcome.

Case Presentation

The patient was an 18-year-old male with head trauma. There was no particular clinical history.

History of present illness: The patient had a traffic accident while riding a motorcycle, fell and hit the left frontal region of the head, and was transported by ambulance. He wore a helmet at the time of injury.

Condition on admission: The level of consciousness was JCSI-1, and no neurological deficits were noted.

Radiographic findings and clinical course: Head CT (Figs. 1A and 1B) showed a linear fracture in the left frontoparietal region and a small epidural hematoma immediately under the fracture site. No midline shift was noted. The patient was hospitalized for observation. Follow-up head CT (Fig. 1C) was performed after 3 h, indicating slight enlargement of the hematoma. There was no change in the level of consciousness, and no neurological abnormality had appeared. The volume of hematoma was 20 ml, the thickness was about 1.5 cm, and the mass effect was very slight. Because of the enlargement of the hematoma, we explained the necessity of craniotomy, but the patient refused the procedure. We, therefore, explained endovascular treatment although the condition was not its indication, and, after sufficiently explaining the procedure including its risk, performed cerebral angiography and endovascular treatment.

Findings on cerebral angiography (Fig. 2): Embolization of the anterior branch of the left middle meningeal artery was performed under local anesthesia. A 5 Fr guiding catheter was guided to the origin of the left external carotid artery. Left external carotid arteriography showed persistent extravasation from the anterior branch of the left middle meningeal...
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of the left middle meningeal artery completely disappeared from angiograms after embolization.

Course after treatment: Head CT performed after treatment (Fig. 3A) showed no enlargement of the hematoma,
which gradually decreased in size thereafter. The patient’s condition was stable, and he was discharged 10 days after treatment, capable of walking without assistance. On head CT taken about 3 weeks after treatment (Fig. 3B), the hematoma further decreased in size.

Discussion

Acute epidural hematoma occurs frequently in younger individuals with a peak incidence in the 10s–20s. Death and persistence of sequelae are reported to be observed in about 10% of all patients. Acute epidural hematoma is associated with linear fracture in most patients but with depressed fracture in some. Clinical symptoms are not parallel with the volume of hemorrhage and are reported to be correlated with the site and bleeding rate.

About 85% of epidural hematomas are caused by arterial hemorrhage (from the middle meningeal or posterior meningeal artery), and many of the rest are caused by hemorrhage from the middle meningeal vein or dural venous sinuses.

Generally, acute epidural hematoma is an indication for surgery except for small ones, and it is considered necessary to remove the hematoma, reduce the intracranial pressure, eliminate local compression signs, and quickly achieve hemostasis.

Even when conservative treatment is selected, strict monitoring is necessary in consideration of the possibility of enlargement of the hematoma. Various opinions have been presented concerning conservative treatment for acute epidural hematoma.

Sagher et al. objected to conservative treatment. If conservative treatment results in the loss of timing for surgery, the patient outcome is likely to be greatly affected. Since surgery for epidural hematoma can be usually performed very safely with satisfactory results, Sagher et al. maintained that surgery should be selected even for small hematomas. In consideration of the necessity of CT and monitors during the observation, they also suggested surgery to be economically advantageous.

According to Knuckey et al., craniotomy was necessary in 32% of the patients with asymptomatic small epidural hematomas but in 55% of those with a fracture line crossing the meningeal artery or venous sinus.

Bezircioglu et al. prospectively treated 80 patients with hematomas 30 cm³ or less in volume, 20 mm or less in thickness, a GCS score of 9 or higher, no neurological deficits, and hospitalized within 24 h after injury conservatively and followed them up. Symptoms were exacerbated, and surgery became necessary, in 5 (6.3%), and one of them (1.3%) died, but 4 showed a favorable outcome.

Concerning surgical indications for acute epidural hematoma, the third edition of the guidelines for the treatment and management of severe head injuries recommend surgery, in principle, when the hematoma is 1 cm–2 cm or greater in thickness, 20 ml–30 ml or greater in volume (15 ml–20 ml or greater for hematomas in the posterior cranial fossa), or complicated by another hematoma; (2) recommend emergency surgery when impending cerebral herniation is noted and neurological symptoms are progressively exacerbated, but (3) permit consideration of conservative treatment under strict monitoring when neurological symptoms are absent. Usually, there would be no objection to craniotomy when the condition is (2), but patients corresponding to (1) or (3) may occasionally show clear consciousness, mild clinical symptoms, and no neurological abnormalities.

Recently, patients who underwent endovascular treatment for epidural hematoma have been sporadically reported.
Endovascular treatment for epidural hematoma is primarily embolization of branches of the middle meningeal artery.

The risks of this treatment include unintended entry of the embolic material, e.g., embolic complications of the ocular artery.\textsuperscript{51} Attention to hemorrhagic complications due to the administration of heparin, which is commonly used in endovascular surgery,\textsuperscript{51} is also necessary although heparin was not used in the present case. Moreover, as hematoma remains even after hemostasis, local compression of the brain by hematoma may persist over a long period. Despite such demerits, endovascular treatment can be performed under local anesthesia and is less invasive than craniotomy.

Suzuki et al.\textsuperscript{6} reported endovascular surgery performed for epidural hematomas with no mass effect and bilateral lesions (epidural hematoma complicated by a contralateral subdural hematoma or brain contusion that required craniotomy). Yonaha et al.\textsuperscript{7} reported a patient with a very small acute epidural hematoma accompanied by a subgaleal hematoma treated by percutaneously aspirating the subgaleal hematoma after hemostasis by endovascular treatment. Ross et al.\textsuperscript{3} treated an epidural hematoma resistant to hemostatic procedures that appeared after surgery for an acute subdural hematoma by ultra-selective angiography followed by embolization for extravasation.

Since the patient that we encountered showed a small epidural hematoma on CT but had no neurological abnormalities including disturbance of consciousness at arrival, we judged that the mass effect of the hematoma is absent or very mild and selected observation. The hematoma was enlarged on head CT after 3 h to 20 ml in volume and 15 mm in thickness, the patient showed a Glasgow Coma Score of 14, and no neurological deficits were observed. While the mass effect remained mild, and no change was observed in neurological findings, the possibility of damage to the middle meningeal artery was considered to be high from the location of the fracture line, and the hematoma was judged likely to further enlarge. However, as the patient refused craniotomy at this point, we explained angiography and embolization of the middle meningeal artery as an alternative and performed the procedures. Since angiography showed persistence of bleeding from the middle meningeal artery, embolization of the vessel was performed in immediate succession. The clinical course thereafter was uneventful, and the hematoma gradually decreased in size without re-enlargement.

When the mass effect of epidural hematoma is mild as in this case or when craniotomy is impossible (due to causes including ethical or religious reasons that prevent general anesthesia), hemostasis by endovascular surgery is considered to be an alternative treatment.

\section*{Conclusion}

We encountered a patient with an enlarging acute epidural hematoma. The volume of hematoma was small, and the mass effect was mild, but, because of the tendency of enlargement, we performed embolization of the middle meningeal artery following angiography with a favorable outcome. Embolization of the middle meningeal artery is considered to be a treatment that deserves consideration against a small acute epidural hematoma.

\section*{Disclosure Statement}

The first author or any of the co-authors has no conflict of interest.

\section*{References}