ABSTRACT

Usually, acute subdural hematomas (ASDHs) result from head trauma and require urgent surgical treatment. However, there have been many reports of rapid spontaneous resolution of ASDHs since 1986. Recently, we experienced a case of a massive ASDH that resolved spontaneously within 1.5 days. A 76-year-old man was admitted to a local hospital after a head injury. According to his clinical records, his initial neurologic status was good (Glasgow Coma Scale score of 14). However, his head computer tomography (CT) scan demonstrated a massive ASDH to the right, with a significant midline shift. Based on his neurological status and general condition, surgery was not considered, and the patient was closely monitored in the intensive care unit. The next day, the patient was transferred to our hospital as requested by his family, after which his neurological state stabilized, and the customary follow-up brain CT was performed. It was about 32 hours after the patient’s head injury, and it revealed an unexpected finding, near-total resolution of the ASDH. Herein, we review previously reported similar cases and relevant mechanisms of rapid resolution of the ASDH. We believe that neurosurgeons should comprehensively assess the patient’s condition and CT findings and provide appropriate treatment, especially when surgical intervention is unnecessary.

Keywords: Acute subdural hematoma; Head trauma

INTRODUCTION

In most cases, acute subdural hematoma (ASDH) is caused by head injury and needs an urgent surgical intervention. In very small proportion of ASDH, we could observe spontaneous subgroup also. Regardless of trauma-origin or spontaneously developed one, we could experience evolution of initially non-operated ASDH to chronic subdural hematoma or its spontaneous resorption. Those cases are patients in the category of benign clinical course so emergent surgery is not needed or patients’ family refuses an aggressive management because of poor prognosis they heard from doctors. Son et al. reported 136 spontaneous resolution cases (77%) among 177 initially non-operated ASDH patients, and their patients required 1-4 weeks for resolution of ASDH. In spontaneous resolution group, there are 2 different courses, slow and rapid. “Slow resolution” cases are not uncommon as shown by Son et al., but “rapid resolution” cases are relatively rare clinical entity. We present a case of traumatic ASDH which initially showed significant amount and midline shift but very benign
Spontaneous ASDH Resolution by Conservative Care

clinical appearance, and showed rapid near total spontaneous resolution within 1.5 day. And we reviewed literatures to study this unexpected peculiar phenomenon.

CASE REPORT

A 76-year-old man who developed a headache after an unprovoked assault was delivered to nearby local hospital. Then he was near alert, and Glasgow Coma Scale scores 14 (E4V5M5). But the initial brain computed tomography (CT) taken at that hospital showed large amount of ASDH in the right convexity with midline shift about 17 mm without definite parenchymal contusions (FIGURE 1). As the patient’s benign condition, emergent surgical intervention was not considered, and the brain CT was repeated after 3 hours later. This CT showed a slight decrease of ASDH amount, and mixed low-densities within the hematoma and spreading of hemorrhagic density to midline structure (FIGURE 2). His neurologic status was stable, so he was admitted to an intensive care unit for close observation. A day later, at the request of his family, the patient was transferred to our hospital. A brain CT was taken after transfer at our hospital which is performed 32 hours after the accident and it showed a marked resolution of ASDH and midline shift, and negligible hemorrhagic densities adjacent to the falx and tentorium (FIGURE 3). With conservative management, the neurological symptoms were

FIGURE 1. First CT scan. The initial CT scan demonstrates an acute subdural hematoma of 20 mm in thickness with a severe midline shift of 13 mm in the left convexity.
CT: computed tomography.

FIGURE 2. Second CT scan. A follow-up brain CT taken 3 hours after admission shows a low-density band in the right frontal area.
CT: computed tomography.
alleviated within 5 days following the trauma. In post-traumatic 13th day MRI, thin sheet-like remnant SDH is noted in both sides of hemispheres (FIGURE 4). About 3 weeks after admission, he was discharged without any neurological deficits. CT taken at out-patient department, post-traumatic 26th day, revealed near total resolution (FIGURE 5).
DISCUSSION

An ASDH is usually caused by head trauma, and if not treated quickly, it could result in considerable mortality and morbidity. Emergent surgical intervention is known to improve the prognosis, and thus, rapid surgery is a preferred treatment modality. However, many researchers have reported cases of rapid spontaneous resolution of ASDH within hours or days, so made surgical procedures unnecessary. Kato et al.\(^\text{11}\) reported their own 2 cases and summarized other 26 cases reported previously by 15 separate articles since mid-1980s. They encompass clinical reports from 1985 to 2001. Wen et al.\(^\text{20}\) reported a case in 2009 and they summarized 19 other authors’ case reports from 1989 to 2008. Among 19 cases, partial overlapping was identified with cases in Kato’s report. After then sporadic case reports were continued until recent years.\(^\text{1,2,7,9,11,14,16,17,19}\) Öğrenci et al.\(^\text{25}\) summarized total 12 pediatric cases from 1986 to 2015. Fujimoto et al.\(^\text{7}\) analyzed 18 patients of rapid spontaneous resolution of ASDH between 2006 and 2012. They postulated that a low-density band sign, and the use of antplatelet agents prior to head trauma are 2 predictable factors for the rapid resolution of ASDH.

According to all above literatures, predictable factors for rapid resolution were low-density band, coagulopathy, premorbid antplatelet agent ingestion and presence of additional intracranial hemorrhage. And Brooke et al.\(^\text{3}\) reported their 29 cases and insisted 2 other predictable factors which were previously unrecognized; lower rates of comorbidities and prehospital anticoagulation.

The phenomenon of rapidly resolving ASDH is still debated, but there are 2 major hypotheses for mechanism. 1) Dilution and wash out of subdural hematoma by cerebrospinal fluid (CSF) flow driven into subdural space from subarachnoid space through torn arachnoid membrane. So called “low-density band” between ASDH and skull seen in CT scan suggests such CSF collection.\(^\text{1,2,7,10,17,19,20}\) 2) Redistribution of hematoma to another space; contralateral intracranial space, intradiploic space and/or subgaleal space through the skull fracture, spinal subdural space, by raised pressure caused by brain swelling, other intracranial hemorrhages, and/ or subdural hematoma itself.\(^\text{1,3,6,7,12,13,19,20}\) It has been claimed also that brain atrophy leads to widening of subarachnoid space, which facilitates the removal of ASDH by aiding the redistribution of the hematoma.\(^\text{1,5,6,7,12,13,19,20}\) Lee et al.\(^\text{25}\) suggest that the expanded subarachnoid space due to brain atrophy can be a favorable factor which could affect the dilution and redistribution of the hematoma. In addition, coagulopathy or premorbid ingestion of antplatelet agent or anticoagulant is another ancillary factor contributes to rapid resolution.\(^\text{3,4,7,11}\)

In our case, relatively prominent senile atrophy was noted in the left cerebral cortex in first CT scan, and the patient did not have coagulopathy and did not take any antplatelet agent. A low-density band between the ASDH and the inner table of the skull was observed in the second follow-up CT. Furthermore, 32 hours later, third follow-up CT images showed that the main hematoma volume was surprisingly reduced and small remnant hematoma is redistributed to another subdural and/or subarachnoid space. Therefore it can be assumed that senile cerebral atrophy in initial CT and low-density band in second CT are 2 predictable factors for rapid resolution of ASDH in our patient.

In addition, we think that the mechanism operating in rapid resolution of ASDH is dependent on initial neurological status; in benign clinical course, there is no intracranial hypertension therefore main mechanism would be “CSF dilution washout” theory, contrarily in initially poor neurological cases, “redistribution” of hematoma by intracranial high pressure would be
main mechanism. Mixed operation of 2 mechanisms could be working together of course in case by case.

CONCLUSION

This case is peculiar in 2 points. First, neurological status and clinical course were very benign despite significant amount of hematoma and midline shift. Second, rapid spontaneous resolution within 32 hours was revealed by customary CT follow-up taken after transfer from other hospital, so it was an unexpected surprising incidental finding. Because the patient had no premorbid factors for rapid spontaneous resolution (e.g., ingestion of antiplatelet or anticoagulant, and other coagulopathy), senile brain atrophy and low-density band on CT were presumed as favorable predictable factors for rapid resolution through both mechanisms; CSF dilution/wash out and redistribution.

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