

Chronic Subdural Hematoma Associated with External Decompression for Acute Traumatic Intracranial Hematoma — report of two cases —

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ABSTRACT

A report is presented on two cases of chronic subdural hematoma which occurred after craniotomy, removal of the hematoma and external decompression for traumatic intracranial hematoma. As for the pathogenesis of chronic subdural hematoma of these two cases, these chronic subdural hematomas were considered to have originated from the subdural collection of mixed fluid of cerebrospinal fluid and blood caused by tearing of the arachnoid on head injury or craniotomy in the space between the brain and dural plasty. Opening and irrigating the hematoma cavity proved adequate as treatment at cranioplasty.

Chronic subdural hematoma after craniotomy is regarded to be an unusual condition. Recently the authors experienced two cases of chronic subdural hematoma which occurred during the postoperative period following removal of the traumatic intracranial hematoma, dural plasty with lyophilized human cadaver dura mater and external decompression. These two cases were of interest in considering the causative factors of chronic subdural hematoma after craniotomy.

CASE REPORTS

Case 1 : A 52-year-old female was admitted as an emergency case to Okamoto Hospital on February 29, 1984 because of disturbed consciousness after head injury received in a traffic accident. On admission, she was semicomatous, the pupils were anisocoric (left side was larger), and the eye balls were deviated to the right side. Bilateral decerebrate posture and Babinski reflexes were obvious. CT scan revealed a left

acute subdural hematoma. Left craniotomy, removal of the subdural hematoma, dural plasty with lyophilized human cadaver dura mater and external decompression were immediately performed. The movement of the right extremities improved soon after surgery. Her consciousness gradually improved and became clear on April 12, the 42th postoperative day. On March 10, the 10th postoperative day, the external decompressed area expanded softly. CT, taken on March 13, the 13th postoperative day, showed a thin layer of subdural effusion, which was found to be a chronic subdural hematoma on CT on April 13, the 44th postoperative day. Neurological symptoms due to chronic subdural hematoma were not observed. On May 23, the 84th postoperative day, irrigation of the chronic subdural hematoma cavity and cranioplasty were performed. Her postoperative course was uneventful and the chronic subdural hematoma disappeared (Fig.1). Histologically the outer

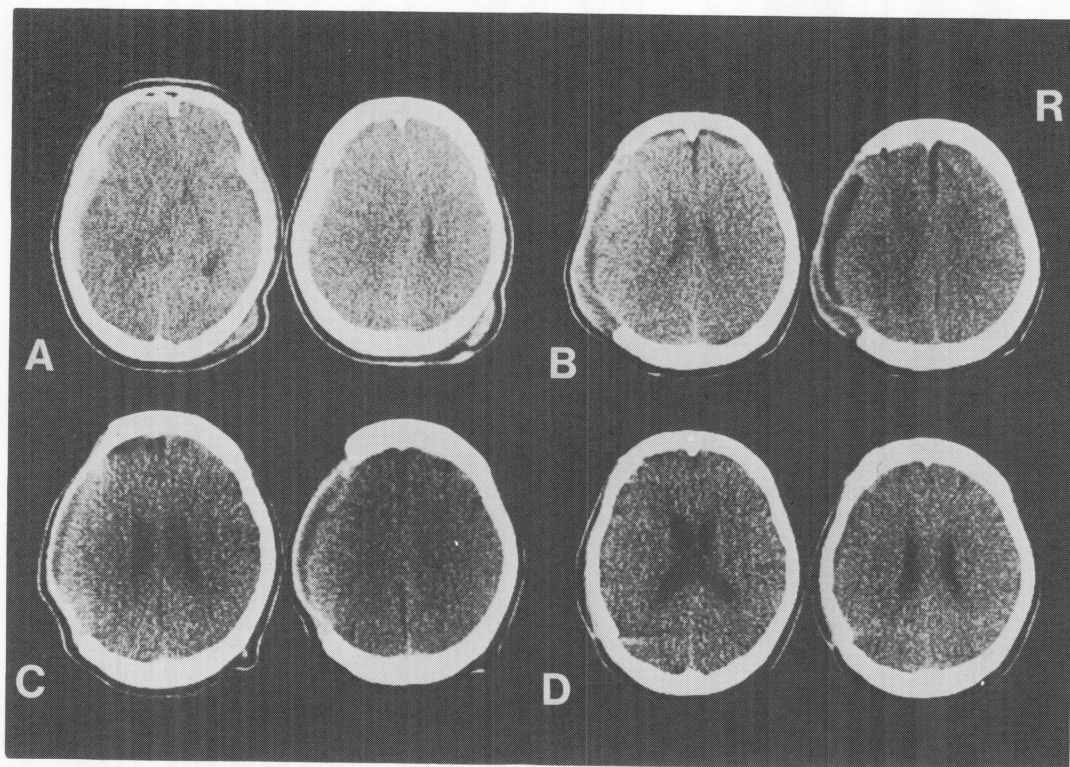


Fig. 1. Serial CT scans of Case 1

A: Before the removal of acute subdural hematoma: An acute subdural hematoma in the left frontotemporal region and midline shift are seen. The left lateral ventricle can not be visualized.

B: 13 days after surgery: Low density area is seen under the external decompressed area, which seems to be a subdural effusion.

C: 44 days after surgery: High density zone is seen in the base of the subdural effusion, which is considered to be a subdural hematoma.

D: 85 days after removal of hematoma and 1 day after cranioplasty: The subdural hematoma can not be observed.

membrane of the hematoma capsula showed typical feature of chronic subdural hematoma consisted of fibrous layer and sinusoidal channel layer.

Case 2 : A 18-year-old male was admitted as an emergency case to Okamoto Hospital on June 3, 1984 because of disturbed consciousness after head injury received in a traffic accident. On admission, he was semicomatous. Craniograms revealed a linear skull fracture across the right middle meningeal artery. There were no abnormal findings on CT. The patient was treated conservatively. On the following morning left hemiparesis became apparent and an acute epidural hematoma of the right side was found on CT. Craniotomy and removal of the hematoma were done immediately. Dural plasty with lyophilized human cadaver dura mater and external decompression were added. On the day

following the surgery, left hemiparesis disappeared. His consciousness was improved gradually and became clear on July 31, the 58th postoperative day. The decompressed area was soft and slightly expansive throughout the postoperative period. On June 13, the 9th postoperative day, CT revealed subdural effusion under the external decompressed area, which was found to have changed to chronic subdural hematoma on CT taken on September 10, the 97th postoperative day. Neurological symptoms due to chronic subdural hematoma were not observed. On September 27, cranioplasty and irrigation of the chronic subdural hematoma cavity were done. His postoperative course was uneventful, and chronic subdural hematoma was reduced markedly (Fig. 2). The histological specimen of the outer membrane of the hematoma capsula showed typical feature of chronic sub-

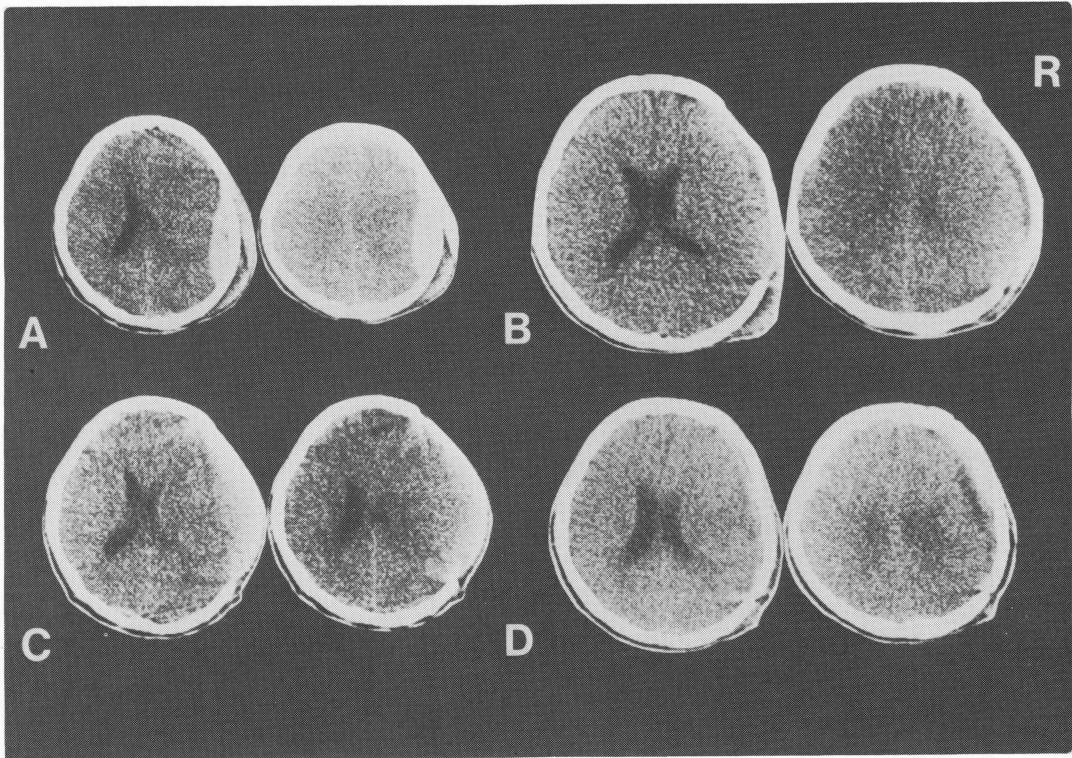


Fig. 2. Serial CT scans of Case 2

- A: Before the removal of acute epidural hematoma: An acute epidural hematoma in the right temporoparietal region and midline shift are observed. The right lateral ventricle can not be visualized.
 B: 7 days after surgery: Low density area with a high density zone at external decompressed area is seen.
 C: 97 days after surgery: The low density area has become highly dense implying that subdural effusion has changed into subdural hematoma.
 D: 145 days after removal of hematoma and 41 days after cranioplasty: The subdural hematoma is markedly reduced.

dural hematoma consisted of fibrous layer and sinusoidal channel layer (Fig. 3).

DISCUSSION

Several theories have been postulated on the pathogenesis of chronic subdural hematoma such as inflammation of dura mater (pachymeningitis haemorrhagica interna) and laceration of bridging vein^{8,12}. Reports of chronic subdural hematoma developing after craniotomy are, however, rare, the frequency being reported to be 1.5% by Komatsu et al⁵. They have concluded that hematoma or mixture of blood and cerebrospinal fluid in the subdural space is the cause of chronic subdural hematoma.

On the other hand, some cases of traumatic subdural effusion have been reported to change to chronic subdural hematoma during the follow-up period with CT. Tsai et al¹³ reported that

rebleeding into the traumatic subdural effusion due to minor head injury brought rise to chronic subdural hematoma. According to Yamada et al¹⁵, hemorrhage from the capsule of the traumatic subdural effusion was considered to be the cause of chronic subdural hematoma.

In both of our two cases of traumatic intracranial hematoma, craniotomy, removal of the hematoma, dural plasty and external decompression were performed. In the first case with an acute subdural hematoma, traumatic subarachnoid hemorrhage might have developed at head injury and cerebrospinal fluid might have mixed with blood. Furthermore it was considered that the cerebrospinal fluid space might be opened to the subdural space by tearing of the arachnoid membrane when the dura mater was opened at surgery. Postoperative brain edema was not so severe as could be expected that



Fig. 3. Photomicrograph of the outer membrane of the hematoma capsule in Case 2
Fibrous layer and sinusoidal channel layer are shown. (Hematoxylin eosin stain $\times 100$)

cerebrospinal fluid remained in the subdural space. Moreover, this thin bloody cerebrospinal fluid was considered to have mixed with blood again which flowed into the subdural space from the dural opening during surgery. According to Watanabe et al⁴, it is essential that the mixture of cerebrospinal fluid and blood remains in

the subdural space for the development of chronic subdural hematoma. In this case, the same condition was suspected, which reactively brought about the development of a capsule of chronic subdural hematoma. With the use of isotope labeled red cells Yamamoto et al^{4,16} showed that fresh hemorrhage into the chronic

subdural hematoma cavity occurred through sinusoidal channels in the outer membrane of the capsule of chronic subdural hematoma. Subdural effusion of this case was considered to have developed into chronic subdural hematoma by hemorrhage from sinusoidal channels in the outer membrane of the capsule. Furthermore, external decompression caused intracranial hypotension, which was suspected to be one of the factors of chronic subdural hematoma⁹.

The characteristic feature of the second case was that chronic subdural hematoma occurred in the epidural hematoma. As for the pathogenesis of chronic subdural hematoma, Apfelbaum et al²) and Labadie et al⁶) have denied the role of cerebrospinal fluid and stated that the presence of hematoma is essential. However, as in this case, cerebrospinal fluid might have played some role in the occurrence of chronic subdural hematoma, as Watanabe et al¹⁴) have emphasized, which originated from the collection of cerebrospinal fluid in the subdural space without any hematoma.

The authors assumed that in these two cases the following was the process of occurrence and growth of chronic subdural hematoma. First, intracranial hematoma was formed by head injury. Traumatic subarachnoid hemorrhage was assumed to have occurred especially in the acute subdural hematoma case, in which cerebrospinal fluid mixed with blood at head injury. At surgery, tearing of the arachnoid joined the subarachnoid space to the subdural space and blood flowed into the subdural space from the cut edge of dura mater or from the extradural operative field, causing the cerebrospinal fluid to mix with blood in the subdural space. Capsule was thought to have originated in reaction to collection of cerebrospinal fluid or mixture of cerebrospinal fluid and blood, and hemorrhage from the capsule was considered to have caused chronic subdural hematoma. External decompression by removing the skull flap at surgery made the intracranial pressure hypotensive, which might have assisted the occurrence of chronic subdural hematoma.

Many kinds of dural substitutes have been used in neurosurgery. Some, such as silastic dural substitute^{1,3,9}) and silicone-coated Dacron¹¹), have been reported to cause hematoma below them or enclosing them. Hemorrhage from new-

ly developed membrane which originated from reaction to the dural substitute has been described as a cause of hematoma. On the other hand, MacFarlane et al⁷), who used lyophilized human cadaver dura mater as a dural substitute, have reported such complications as liquorrhea, infection, and meningitis, but hematoma formation has never been observed. Thus, the use of lyophilized human cadaver dura mater was not considered to be a cause of chronic subdural hematoma.

Several methods of treatment for chronic subdural hematoma have been developed¹⁰). In both of these two cases, good results were obtained after opening and irrigating the hematoma cavity at cranioplasty. The following reasons may be given for satisfactory postoperative results. Irrigation of the hematoma cavity made the brain expand easily by relieving the brain of pressure from the hematoma, and cranioplasty balanced the volume of subdural space to the brain volume correcting the intracranial hypotension.

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