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Symptomatic Subacute Subdural Haematoma Following Spontaneous Acute Subdural Haematoma

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Summary

Two patients with non-traumatic acute subdural haematoma were observed, initially without surgical intervention. Eleven days after the onset, each patient developed hemiparesis and an increase in severity of headache. Serial computed tomography scans demonstrated that the initial hyperdense haematomas became hypodense with a definite increase in volume. The term "symptomatic subacute subdural haematoma" was proposed to properly define this pathological process, which necessitated removal of the haematoma in the subacute stage. The mechanism of increase in the volume of the haematoma is discussed.

Keywords: Acute subdural haematoma; subacute subdural haematoma; chronic subdural haematoma.

Introduction

Subacute subdural haematoma (SDH) itself has rarely received attention, and has merely been recognized as a pathological condition either transforming into chronic SDH or occurring at a subacute stage after acute SDH [2, 4]. Recently the authors have encountered two patients who developed new symptomatology in the subacute stage after spontaneous acute SDH; this pathological process was considered to be properly defined as symptomatic subacute SDH. Details of this lesion are described, and the mechanism of the increase in the volume of the haematoma is discussed.

Case Reports

Case 1. This 72-year-old man who had a ten-year history of hypertension developed sudden onset of headache and vomiting during dinner on April 22, 1987. His recent medical history was unremarkable with neither cerebrovascular disease, head trauma nor alcohol intake. Although no disturbance of consciousness or other associated neurological symptoms were present, he attended the Department of Neurosurgery, Tokyo Metropolitan Fuchu Hospital, in the early morning on August 23, complaining of persistent headache.

Examination: On arrival physical examination showed no abnormal findings apart from a moderate degree of hypertension. Although the patient complained of pain all over his head, neurological evaluation including funduscopic examination failed to reveal any abnormalities. Computed tomography (CT) scans without intravenous injection of contrast medium (Fig. 1) revealed a high density lesion covering the entire cerebral hemisphere on the left side, indicating acute SDH. A mild degree of mass effect was noted. Cerebral angiography did not show any aneurysm, arteriovenous malformation or other findings. Results of laboratory examinations including blood routine, blood chemistry and coagulation studies were all within normal limits.

Clinical course: Because of the lack of objective neurological findings, he was initially treated conservatively. His headache was gradually alleviated after the use of intravenous administration of glycerol and peroral analgesics. On May 3, eleven days after the onset of symptoms, however, he was found to have an unsteady gait, urinary incontinence and disorientation. Hemiparesis on the right side was evident. CT scans (Fig. 2) showed an increase in the volume of the subdural haematoma on the left side, of lower density than the cerebral parenchyma. Distortion of the midline structures was also increased. On the following day, upon percutaneous subdural tapping a dark-brown, liquid haematoma gushed out. The subdural space was irrigated with saline. His subsequent clinical course was uneventful. Although a small volume of subdural fluid still was visible on CT scans, he was discharged from this hospital without neurological deficits on May 18. His final follow-up examination three months later confirmed complete resolution of symptoms. No further CT evaluation was performed.

Case 2. This 36-year-old man with an unremarkable past history developed pain on the occipital region when he awoke at 7.30 a.m. on July 31, 1987. He did not have any recent episode of head trauma. Since no relief of pain followed, he visited a nearby hospital on August 3, and was diagnosed as having acute SDH. On that day, he was referred to this hospital.

Examination: Physical and neurological examination showed him to be alert and oriented. No retinal haemorrhages were present. CT scans without contrast injection (Fig. 3) demonstrated a small volume of a high density lesion over the left cerebral hemisphere, indicating acute SDH. Cerebral angiography failed to reveal any abnormal features. No abnormalities in routine blood counts, blood chemistry and coagulation studies were discovered.

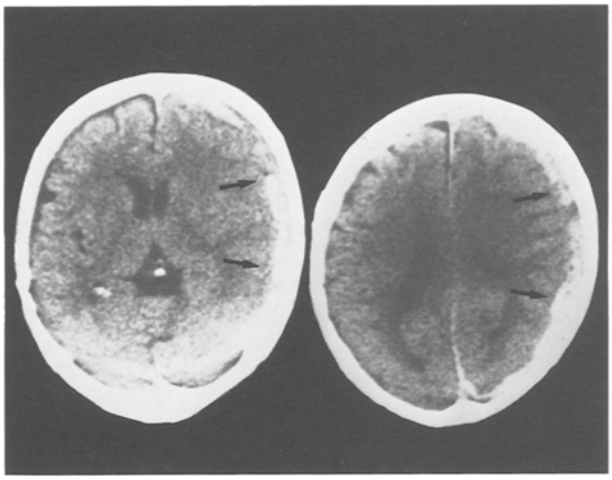

Fig. 1. Computed tomography scans at the time of admission in case 1, showing a high density lesion covering the entire cerebral hemisphere on the left side (arrows)

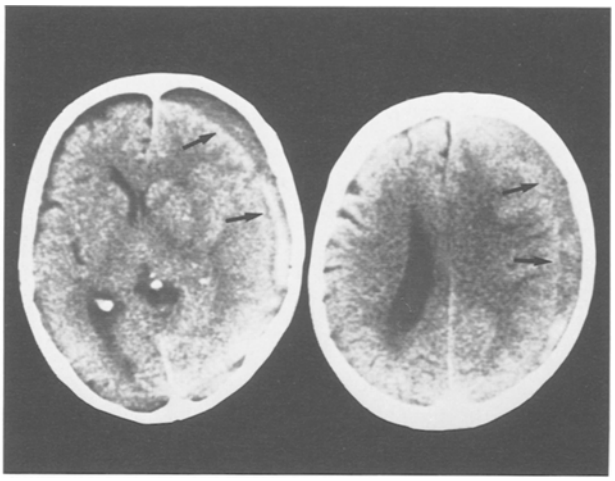

Fig. 2. Computed tomography scans eleven days after the onset in case 1, disclosing enlargement of subdural haematoma with low density (arrows). Note the midline shift

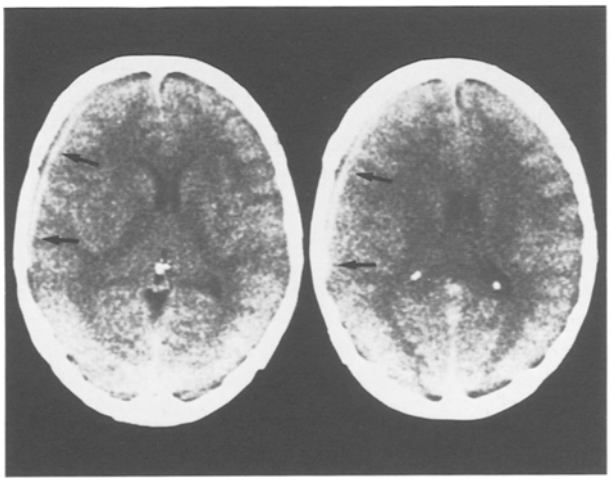

Fig. 3. Computed tomography scans 3 days following the onset in case 2, revealing a thin, high density mass over the right cerebral cortex (arrows)

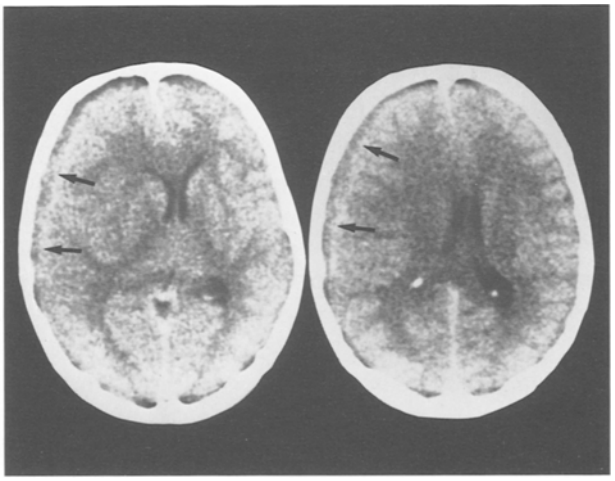

Fig. 4. Computed tomography eleven days after the onset in case 2, demonstrating an increase in the volume of the haematoma associated with decreased attenuation (arrows)

Clinical course: He was admitted for neurological observation. The headache was alleviated by administration of peroral analgesics, and, though without complete resolution of the headache, he left this hospital on August 6. Eleven days after the onset of the headache (on Augst 11), however, he suffered an acute increase in the severity of his headache, and was readmitted to this hospital. Although there were no objective findings on neurological examination, CT scans (Fig. 4) disclosed an increase in the volume of subdural haematoma, which had a lower density than that on the initial scans. A midline shift was also shown. Percutaneous subdural tapping yielded liquid haematoma with chocolate-like colour, which was irrigated with saline. He was discharged from this hospital on August 30, without reaccumulation of subdural fluid on CT. The further follow-up with CT scans on September 12 revealed minimal fluid collection over the left frontal region. At that time, no symptoms were noted.

Discussion

Clinical characteristics in the present two patients include development of new symptoms eleven days after the onset of nontraumatic acute subdural haematomas. The progressive enlargement of SDH on CT scans in the subacute stage was responsible for these symptoms. This pathological process may be referred to as symptomatic subacute SDH. Subactue SDH has not been fully recognized as being a definite clinical entity[2, 5]; the mechanism of increase in the volume of the haematoma is worth discussing. Based on serial CT scans, the progressive enlargement of SDH in the present patients is accompanied by decreased attenuation

of the haematoma. Although analysis of the haematoma content or investigation of the capsule was not obtained, upon percutaneous subdural tapping[1], the macroscopic appearance of the haematoma content did not suggest subsequent bleeding. It has been known that cerebrospinal fluid (CSP) contributes minimally to an increase in the volume of chronic SDH[11]. However, in the subacute stage during the haemolytic process, CSF influx into the subdural space may be caused by the colloid-osmotic pressure difference between the liquid haematoma and CSF[13], or through the residual tearing of the arachnoid membrane. In addition, opacification of the subdural space by metrizamide CT cisternography during the early stage of chronic SDH has been documented[8]. Subdural fluid collection compresses the underlying brain, possibly followed by venous obstruction or shifts of the brain. This could be proposed as another mechanism, by which brain swelling and further collection of fluid are formed, causing additional symptoms at the subacute stage.

After the advent of CT scans, post-traumatic subdural fluid collection has been known to contribute to the development of chronic SDH[12]. There has been, however, only a few reports documenting the occurrence of chronic SDH originating from acute SDH[6, 7, 10, 14]. In the present two patients, the SDH resolved after subdural tapping in the subacute stage when new symptoms developed; however, if they were not treated at the time because of minimal symptoms, the transformation into chronic SDH could ensue from the subacute SDH. This developmental process of chronic SDH can be facilitated by participation of CSF[9] which is drawn into the haematoma cavity during the subacute stage.

To summarize, acute SDH without surgical treatment is liquified and may progressively enlarge, so that new symptoms develop in the subacute stage. Furthermore, this pathological process may contribute to the subsequent development of chronic SDH.

References

1. Aoki N (1984) Subdural tapping and irrigation for the treatment of chronic subdural hematoma in adults. Neurosurgery 14: 545–548
2. Cooper PR (1985) Traumatic intradural hematomas. In: Wilkins RH, Rengachary SS (eds) Neurosurgery, vol 2. McGraw-Hill Book Company, New York, p 1659
3. Ito H, Yamamoto S, Komai T, Mizukoshi H (1976) Role of local hyperfibrinolysis in the etiology of chronic subdural hematoma. J Neurosurg 45: 26–31
4. Loew F, Wüstner S, 1961: Diagnose, Behandlung und Prognose der traumatischen Hämatomie des Schädelinneren. Springer, Berlin Göttingen Heidelberg
5. Loew F (1982) Management of chronic subdural haematomas and hygromas. In: Krayenbühl H, Brihaye J, Loew F, Logue V, Mingrino S, Pertuiset B, Symon L, Troupp H, Yaşargil MG (eds) Advances and technical standards in neurosurgery, vol. 9. Springer, Wien New York, pp 113–131
6. Taguchi Y, Nakamura N, Sato J, Hasegawa Y (1982) Pathogenesis of chronic subdural hematoma. Sequential study with computerized tomography. Neurol Med Chir (Tokyo) 22: 276–282 (in Japanese)
7. Takahashi K, Takizawa T, Sato S, Sano A, Takahashi K, Murakami Y, Ota K (1988) Two cases of acute idiopathic subdural hematoma with delayed intracranial hypertension. No Shinkei Geka 16: 1383–387 (in Japanese)
8. Takahashi Y, Mikami J, Ueda M, Ito K, Sato H, Matsuoka T, Takeda S, Ohkawara S (1984) Analysis of chronic subdural hematoma based on CT (Part III). Clinical stage classification based on CT findings. Neurol Med Chir (Tokyo) 24: 607–614 (in Japanese)
9. Watanabe S, Shimada H, Ishii S (1972) Production of clinical form of chronic subdural hematoma in experimental animals. J Neurosurg 37: 552–561
10. Watanabe T, Watanabe T, Sasaki T, Murata S, Yamada H (1980) Chronic subdural hematoma formed from acute subdural clot. In comparison with the type occurring subsquent to the traumatic subdural hygroma. Neurotraumatology 3: 159–164 (in Japanese)
11. Weir B (1971) The osmolarity of subdural hematoma fluid. J Neurosurg 34: 528–533
12. Yamada H, Watanabe T, Murata S, Shibui S, Nihei H, Kohno T, Itoh T (1980) Developmental process of chronic subdural collections of fluid based on CT scan findings. Surg Neurol 13: 441–448
13. Yamamoto S, Ito H, Mizukoshi H, Yoshida S (1973) Hemorrhage from the outer membrane of chronic subdural hematoma. No Shinkei Geka 2: 239–242 (in Japanese)
14. Yoshimasu N, Tamura A, Wakai S, Yoshida T (1981) Evolution from acute subdural hemorrhage to chronic subdural hematoma. Observation of subdural hematoma by computerized tomography. No Shinkei Geka 9: 1025–1031 (in Japanese)

Addendum. Since acceptance of this paper another publication reporting on 4 cases of subacute subdural haematoma has been published: (Morinaga K, Matsumoto Y, Omiya N, Mikami J, Ueda M, Sato H, Inoue Y, Okawara S, Takahashi Y, Fujisawa Y [1990] Subacute subdural hematoma. Report of 4 cases and a review of the literature. Brain Nerve 42: 131–136 [in Japanese])

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