Chronic Intracerebral Hematoma Associated with Ipsilateral Chronic Subdural Hematoma in a Head injured Patient

Case Report

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Abstract

Although some intracerebral hematomas have been known to increase in size chronically and produce delayed neurological deficits, the mechanism of hematoma expansion is not well understood. The authors present a case of a head injured patient with coexistence of a chronic intracerebral hematoma and an ipsilateral chronic subdural hematoma that expanded chronically showing clinical deterioration, in whom the simultaneous expansion of hematomas was observed sequentially from the beginning with neuroimaging studies. The will also discuss the mechanisms involved in hematoma enlargement.

Key words

Head injury, chronic intracerebral hematoma, chronic subdural hematoma

Introduction

Intracerebral hematomas (ICHs) that enlarge chronically and produce delayed neurological deficits have been known as chronic ICHs, or chronic encapsulated ICHs. Regardless of the pathoetiology, a thick capsule consisting of granulation tissue with rich neovascular channels has been reported in most patients. Due to the pathohistological similarity of this capsule to the outer membrane of chronic subdural hematomas (CSHs), some investigators consider that reported bleeding or exudation from the capillaries of the neovascularized capsule promotes expansion of ICHs. Coexistence of a chronic ICH and CSH may provide a much clearer understanding of their relationship, but, to our knowledge, there has been no report of such co-occurrence in a single patient. We herein report a case of a head injured patient having a chronic encapsulated ICH in association with an ipsilateral CSH, in whom the simultaneous expansion of hematomas was observed sequentially with neuroimaging studies, and discuss the mechanism of hematoma enlargement.

Case Report

A 60-year-old woman was struck by a car while she was taking a walk, and was brought to our emergency service by ambulance 30 minutes after traffic accident. A considerable amount of hemorrhage from the right external auditory canal was noted at the scene. She was deeply comatose on admission. Her blood pressure was 140/100 mmHg, pulse rate 99 beats/min, respiration regular 18/min. Her nostrils oral cavity was filled with blood. Bleeding from the right external ear appeared to be decreased but still continued. Her pupils were round and equal in size, and reacted promptly to light projections. Roving eye movement was seen and her extremities responded to escape from noxious stimuli without laterality. Routine laboratory blood examinations were normal in all. Her past medical
history was not contributory. After resuscitative treatment including an endotracheal intubation, imaging studies were carried out. Computerized tomography (CT) scans of the head revealed cerebral contusions in the left frontal base and the temporal pole, and diffuse thin subarachnoid hemorrhage as well (Figs. 1a, b). During CT examination, the patient’s blood pressure suddenly dropped down to 60 mmHg. And then, blood transfusion was performed. Since laboratory data (FDP: 44 μg/mL, Platelets: 67000/μL, Fibrinogen: 87 mg/dL) suggested disseminated intravascular coagulation (DIC), anti-DIC agents as well as intravenous dopamine and dobutamine infusions were required. A follow-up CT scan taken 24 hours after the initial study showed an ICH in the left temporal lobe in association with a thin subdural hematoma in the ipsilateral frontal lesion (Figs. 2a, b). Fortunately, her neurological condition remained unchanged. In the following week, the patient’s general condition was improved gradually with a conservative manner. She opened her eyes spontaneously and responded to simple verbal commands by the end of the second hospital week. However, the patient developed right hemiparesis four weeks after her admission and her consciousness level was deteriorated again. CT scan (Figs. 3a, b, and c) depicted that the left temporal ICH was developed to a large, heterogeneously high density mass with marked surrounding edema. This mass showed a ring-like enhancement after administration of contrast material. In addition, the left subdural hematoma was also evolved to a large, low density fluid collection. Considering magnetic resonance (MR) imaging examined two weeks after the insult (Figs. 4a, b), these hematomas appeared to grow gradually and could be considered as a co-occurrence of chronic encapsulated ICH and CSH. Left temporal craniotomy was formed. Dural opening disclosed a thin brownish capsule containing fluid in the frontal side of the operative field. The cavity was filled with brownish fluid. This condition was considered as common CSHs. A puncture of the temporal cortex allowed aspirating a small amount of dark brownish fluid similar to contents of CSHs. As a corticotomy was made, a firm capsule measuring approximately 3 mm in thickness was encountered at a depth of 5 mm from the cortex. Careful exploration of the inside of the capsule allowed us to see no abnormalities suggesting neoplasm or vascular malformations. The capsule was partly removed for
pathological examination, but mostly left unre-moved. Histopathological examination of the specimen showed neither vascular malformation nor neoplasm. The capsule of the temporal ICH consisted of abundant fibrous connective tissue with numerous congested thin-walled capillaries on the inside a reactive brain tissue on the outside (Fig. 5). Hemosiderin-laden macrophages and lymphocytes were also seen among the capillaries on the inside. Microscopical findings of the outer membrane of the encapsulated subdural hematoma were similar to those of the inner side of the ICH capsule. Postoperatively, the right hemiparesis was rapidly improved and overall neurological condition improved gradually. Follow-up neuroimaging studies showed gradual decreases in size of the ICH as well as the surrounding edema, and no reaccumulation of the left CSH. On her last visit 6 months after the insult, she showed no neurological abnormalities and returned to her previous lifestyle.

Discussion
ICHs usually resolve spontaneously, showing gradual sequential changes until they completely disappear 3 to 8 months later without producing further neurological deterioration. Since Yashon and Kosnik reported 12 patients with ICH showing gradual neurological deterioration and called for a recognition of chronic ICHs mostly treatable with surgery, a considerable number of patients with chronic ICH has been collected. Although vascular malformations are nominated as a leading cause of these hematomas, they may be attributed not only to vascular malformation, but also to hypertension, head injury, anticoagulation therapy, chemotherapy, or neoplasm. The mechanism of hematoma expansion is still unknown, but a thick capsule consisting of granulation tissue with rich neovascular channels has been reported in most patients. Some investigators considered that repeated bleeding or exudation from the capillaries of the neovascularized capsule may promote expansion of ICH, because of the pathological similarity of this capsule to the outer membrane of CSHs. While the exact mechanism of hematoma expansion in patients with CSH also remained unclear, two different evolving patterns of chronic subdural hematoma have been reported based on serial CT findings: 1) a gradual changing of low density subdural fluid collection (subdural hydroma or hy-
groma) to high density or iso-density subdural hematoma accompanied with volume expansion; and 2); a gradual changing of high density subdural clots to low density fluid collection in association with increase in size. Evolution of subdural hydromas to CSHs may be attributed to capsule formations followed by recurrent hemorrhages from the capsule with rich neovascularity occurring over 2 to 3 months\cite{10, 12}.

With regard to natural course of subdural clots, Taguchi, et al.\cite{11}, reported that serial CT findings showed gradual decrease in density with increasing in size of subdural clots in 23 out 52 patients with traumatic acute subdural hematomas who were treated conservatively without surgical intervention. Among 15 patients who underwent

Fig. 3. CT scan four weeks after admission showing an increase in size of the left intracerebral hematoma with a change in density (a). After administration of contrast material, a ring-enhancement is seen (c). Note that this intracerebral hematoma associated with marked surrounding edema. In addition, the right subdural hematoma had evolved a large, low-density fluid collection (b).
surgical evacuation because of neurological deterioration, a thick outer membrane having rich neovascularization could be found in the patients who were operated more than 19 days after onset.

This clinical evidence suggests that nearly a half of the patients having traumatic subdural hematoma may develop CSHs. Since hematoma contents consisted of mostly xanthochromic fluid and partly old clots, the mechanism of hematoma expansion was proposed not to recurrent hemorrhages from the capsule with rich neovascularity, but to increase in colloid osmotic pressure following the breakdown of red blood cells. Neuroimaging studies of our patient showed a gradual decrease in density associated with increase in size both in subdural and intracerebral hematomas. Therefore, the encapsulated subdural hematoma may be evolved by the mechanism described above. Although the role of associated DIC in our patient is not fully understood, it seems possible to consider that the initial ICH caused by a head injury encourages capsule formation with rich neovascularity and breakdown of the hematoma content causes increase in colloid osmotic pressure allowing increase in volume of the hematoma, similar to the mechanism of subdural hematoma expansion.

References


慢性脳内血腫と慢性硬膜下血腫への進展過程を観察した
外傷性脳内血腫と急性硬膜下血腫の合併例

抄録
慢性脳内血腫は一般の脳内血腫の自然経過と異なり、被膜を持って慢性期に增大し、臨床症状悪化をきたす。血腫被膜が病理学的に慢性硬膜下血腫に類似しているという理由から、慢性硬膜下血腫発生メカニズムの1つである被膜からの滲出や新たな出血が慢性化機序として想定されている。著者らは、70歳男性の頭部外傷患者において、CT上高吸収域として観察された左側頭葉脳内血腫と左急性硬膜下血腫が、約4週の臨床経過で、ともに慢性脳内血腫と慢性硬膜下血腫と診断できる画像所見を観察した事実を観察した。血腫被膜の病理所見は両者同様であったが、血腫内容は暗褐色の古い血腫であり、画像所見に一致した。通常の血腫除去、血腫腔洗浄によって神経症状は改善した。慢性硬膜下血腫発生機序として急性硬膜下血腫の慢性化が報告されているが、同時に存在した外傷性脳内血腫と急性硬膜下血腫の画像所見、病理所見、病理所見を総合すると、本例は急性血腫の慢性化で、慢性脳内血腫も同様のメカニズムが想定された。また、血腫増大機序として被膜からの滲出や新たな出血とは別に、血球破壊に伴う膠質浸透圧の上昇と考えられ、慢性脳内血腫の発生メカニズムは単一ではないことが示唆された。