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Ossified Chronic Subdural Hematoma with Armored Brain

Ossifiye Kronik Subdural Hematom ile Zırhlı Beyin

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ABSTRACT

Ossified chronic subdural hematoma is a very rarely seen entity. We present here a 22-year-old male who had presented with severe headache consequent to brain compression caused by bifronto-parieto-temporal ossified subdural hematoma. We evaluated our method and surgical intervention in the light of the literature. The question whether the ossified membrane should be excised or not excised in these cases is a matter of controversy. We think that an ossified membrane causing an armored brain appearance should be excised in symptomatic, young patients with prominent cerebral compression. During this dissection, the relatively thickened arachnoid mater provides a safe border.

KEY WORDS: Armored brain, Ossified chronic subdural hematoma, Surgical treatment, Hematoma

ÖZ

Ossifiye kronik subdural hematom çok nadir görülür. 22 yaşında, şiddeti artan baş ağrısı olan, bifrontoparietotemporal ossifiye subdural hematom nedeniyle beyni komprese, atrofik görünümlü bir erkek hastayı sunduk. Yönetimimizi ve uyguladığımız cerrahi tedaviyi literatür ışığında değerlendirdik. Bu olgularda ossifiye membranın eksize edilip edilmemesi tartışmalıdır. Semptomatik, genç hastalarda ve serebral kompresyonun belirgin olduğu olgularda zırhlı beyin görünümüne neden olan ossifiye membranın eksize edilmesi gerektiğini düşünüyoruz. Bu disseksiyonda nispeten kalınlaşmış olan araknoid mater güvenli bir sınır sağlar.

ANAHTAR SÖZCÜKLER: Zırhlı beyin, Ossifiye kronik subdural hematom, Cerrahi tedavi, Hematom

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INTRODUCTION

Chronic subdural hematoma is a frequently encountered disorder in neurosurgery practice (8). However, calcified and ossified chronic subdural hematoma is very rarely seen (2,7,10). Therefore, there are no common or absolute opinions regarding the mechanism of pathogenesis and the clinical method. It should be discussed whether the hematoma should be surgically drained and whether the calcified wall should be excised or not. Patients should be evaluated thoroughly for age, symptoms, neurological signs, and degree of cerebral compression on cranial imaging, before a decision for a surgical intervention is made.

CASE REPORT

A 22-year-old male patient presented with the complaint of headache that had been persisting for one and a half years, with increasing severity. The patient had a history of head injury about two years ago. He apparently fell backwards and hit his head on the floor. He was not on any medication at the time being that would otherwise interfere with blood coagulation. Neurological examination was normal except for papilledema. The computerized cranial tomography revealed a hypodense fluid collection with calcified bifronto-parieto-temporal borders. The brain was observed to have been pushed into the occipital region (Figure 1). On cranial MRI, a subdural fluid collection was observed in the bilateral anterior fronto-parieto-temporal regions, reaching about 6 cm in thickness at its widest part, which was hypointense in T1 and hyperintense in T2 and FLAIR sequences. This fluid collection was observed to be surrounded by diffuse hypointense areas due to calcifications on both the inside and the outside. The frontal lobes had an atrophic appearance bilaterally. A bi-coronal approach was used for craniotomy. After the bone was lifted and the dura mater was opened, the ossified thick and hard capsule was seen (Figure 2). When the capsule was opened, the fluid, which was greasy in appearance, and not viscous but freely flowing was drained. At that time, it was interesting to observe calcified vascular structures going through the ossified outer capsule extending to the inner capsule. After the fluid was completely drained, the ossified inner capsule surrounding the cerebral cortex was exposed. Since the capsule was thick and continuous, it was blocking the expansion of the brain like an armor. The inner capsule, which had the appearance of an eggshell, was excised by meticulous dissection from the arachnoid mater by microsurgery (Figure 3). The arachnoid membrane between the ossified inner capsule and the brain parenchyma, which was relatively thickened, was observed to have formed a reliable border for dissection. The patient's complaint of headache disappeared completely. On the follow-up computerized brain tomography on the tenth postoperative day, the compression on the brain was observed to be decreased comparatively and the brain that had been compressed into the occipital

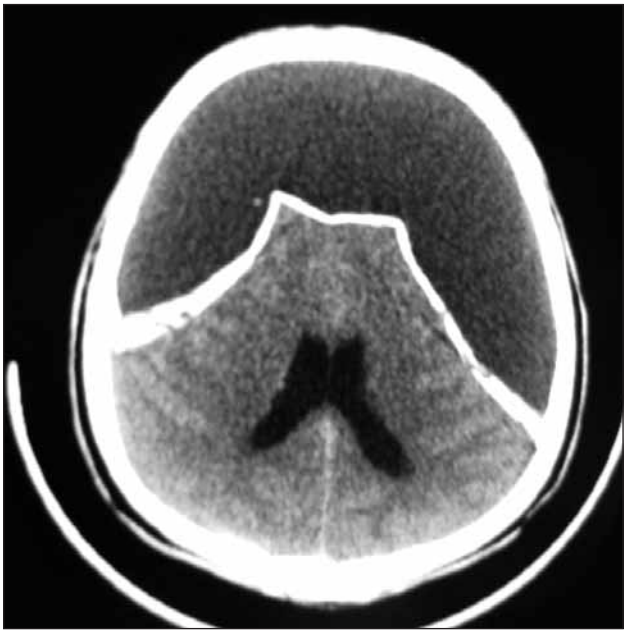

Figure 1: *Preoperative cranial CT imaging. It shows a hypodense fluid collection with bifronto-parieto-temporal limits calcified and occipital compression of the brain.*

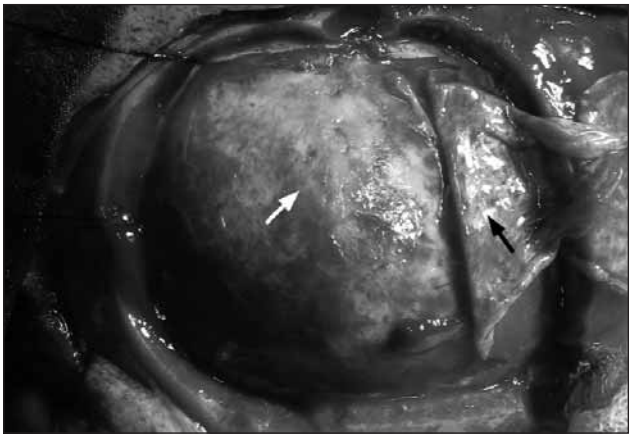

Figure 2: *After the bone is removed and the dura mater is opened (black arrow), the thick and hard outer capsule in the ossified structure (white arrow) is observed.*

region had begun to expand (Figure 4). The patient is now under follow-up for recurrence. No problems were noted during the regular follow-up. We preferred cranial MRI for the postoperative second month follow-up. The cerebral re-expansion was better seen in this MRI examination (Figure 5).

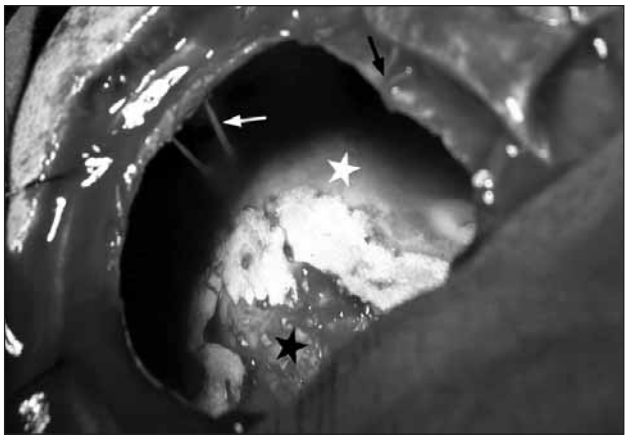

Figure 3: *After drainage of the hematoma, the inner capsule which appears like an egg shell, compressing the brain (white star), and the thickened arachnoid mater and the cerebral cortex (black star) are observed. Calcified vessels were observed going through the ossified outer membrane (black arrow) extending to the inner membrane (white arrow).*

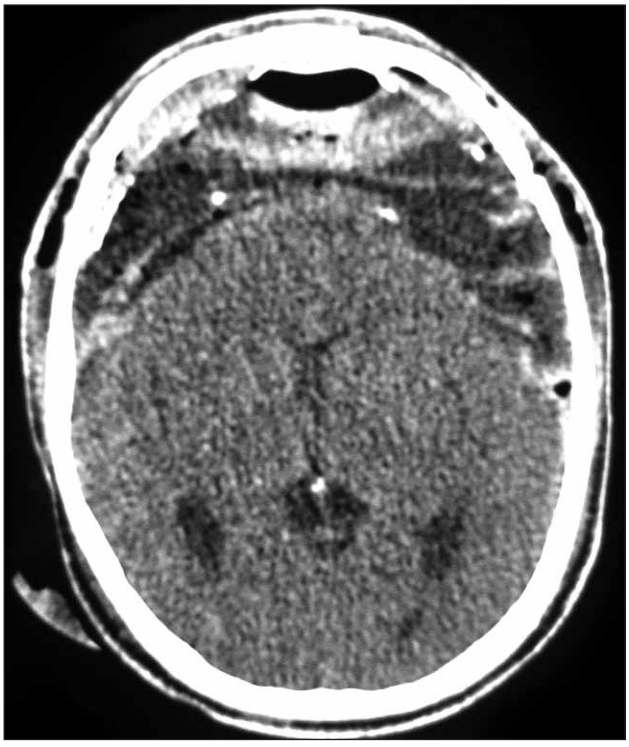

Figure 4: *On the postoperative tenth day follow-up with computerized brain tomography, it is observed that the compression on the brain decreased comparatively and the brain repressed to the occipital region began to expand.*

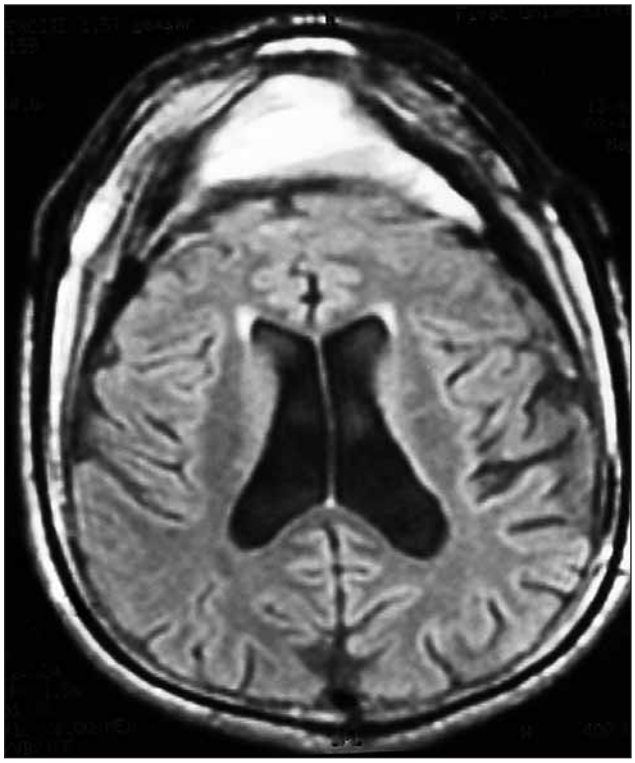

Figure 5: *The cerebral re-expansion was seen on MRI examination on the postoperative second month.*

DISCUSSION

Chronic subdural hematoma is usually one of the complications of minor head trauma. Disorders such as coagulopathy (therapeutic anticoagulant use, sepsis, liver insufficiency), intracranial hypotension (secondary to overdrainage resulting in bridge veins traction in shunted patients), chronic alcoholism, vascular malformations, and primary and metastatic tumors may also play roles in the etiology of chronic subdural hematoma (8). Calcification or ossification is observed at a rate of 0.8% to 10% in patients with chronic subdural hematoma (4,9). Calcified chronic subdural hematomas are frequently observed as a late complication of head injuries or as a result of postmeningitic subdural effusion. It may also be observed as a long-term complication in patients who have undergone a shunt operation due to hydrocephalus, although very rarely (10).

The pathogenesis of calcification in chronic subdural hematomas is not completely clear. However, many authors have suggested that local, metabolic, and vascular factors play a role in the development of calcification and ossification. Afra reported that the circulation in the subdural space with absorption and vascular thrombosis may be

responsible for the development of calcification (1). In the literature, there are also cases with bilateral chronic subdural hematomas developing unilateral calcification. This suggests that a local factor may play a role in the development of calcification. In addition, it has been stated that metabolic predisposition may be responsible (6). Development of calcification following chronic subdural hematoma generally takes about six months. Ossification develops a few years after calcification. It is thought that ossification is the result of this process (7,10).

In calcified chronic subdural hematoma, the symptoms and findings show differences. Asymptomatic cases and those with increased intracranial pressure have been reported in the literature. Lethargy, confusion, amnesia, hemiparesis, gait disturbance, seizures and mental retardation may be observed, in addition to headache as the most frequent symptom (2,4,7,10). Since a slow progression had been observed, neurological deficits may not have accompanied the remarkable hematoma and severely-compressed brain as in our case. Calcification of chronic subdural hematomas may be observed more frequently in children and young adults than older people (4). Since young people have a greater tendency to be affected by increased intracranial pressure, headache is more frequently observed (8). As a matter of fact, headache was the only complaint in our young patient.

When the calcified wall covers the brain surface, this is defined as an 'armored brain' (2,5). This calcified section is reported to possess tight adhesions with the dura mater and the brain surface, although thin. Surgical excision of the calcification is not routinely performed because of the risk of damaging the underlying cortex. It is recommended that patients who are asymptomatic, old, and without acute or progressive changes in their neurological condition are followed-up. Surgical treatment is reported to be necessary in symptomatic and young patients (4,7,9,10). Despite the absence of symptoms, surgical excision for the calcification over the cortex is recommended in cases where prominent cerebral compression is observed, in order to provide cerebral re-expansion and inhibit probably developing brain atrophy (4,10). In our case, it was also observed that the calcified inner membrane surrounded the brain like an armor and inhibited brain expansion in spite of hematoma drainage. We think that if the inner layer is thicker and compressing the brain seriously, fluid drainage by itself may be insufficient for improving the symptoms and helping the re-expansion of the brain. We therefore suggest the excision of the inner layer in these cases. Careful dissection has been reported to be necessary for inner capsule excision in these cases because of dense adhesions on the brain surface (7,10). In the present case, the relatively thickened arachnoid membrane contributed significantly to our performing the dissection without brain parenchyme injury.

One of the most frequent complications that may be observed after chronic subdural hematoma operations is recurrent hemorrhage. It is thought that insufficient brain expansion following hematoma drainage, developing following prolonged compression in recurrent hemorrhage is the basic factor. However, since ossified subdural hemorrhages are rather rare, there is insufficient information regarding the recurrence rate in the literature. In chronic subdural hemorrhage cases, recurrent hemorrhage and residual subdural fluid collection should be differentiated from each other. Disappearance of residual fluid may sometimes last for weeks or even months (3). Therefore, unless there is presence of clinical deterioration, no intervention should be carried out regarding the residues in the control CT (8). In our patient, who was attending regular follow-up and without complaints or clinical findings, no procedures were carried for the residual images in the CT and the MRI. A gradual decrease in residual subdural fluid was observed. Should recurrent hemorrhage, insufficient brain expansion and probable accompanying complaints and clinical findings be seen on further follow-ups, a subdural shunt will be planned.

CONCLUSION

A hematoma membrane may be calcified or even ossified long after the occurrence of a chronic subdural hematoma. In these cases, the calcified capsule may inhibit brain expansion like an armor. The hematoma should be particularly drained and the calcified capsule should be excised in cases where the inner layer of the capsule is thicker and the prominent cerebral compression is observed, The relatively thickened arachnoid membrane lying between the calcified capsule and the brain parenchyme composes a reliable border for dissection.

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