Case Report
Calcified chronic subdural hematoma (CCSH): report of two cases

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Abstract: Calcification of hematoma is seen rarely although chronic subdural hematoma (CSH) is a well-known. We present two cases with calcified subdural hematoma. 35 years old female who admitted to our hospital with a complaint of epilepsy and 58 years old male who was diagnosed as calcified subdural hematoma incidentally. We performed a wide craniotomy and duratomy including the whole of CCSH reaching dural arachnoid border and we dissected the calcified mass from arachnoid and invaginations to the brain in both of the patients. We did not observe deficit in both of our patients following operation and discharged them at the postoperative 3rd day with recommendation of antiepileptic for a month.

Keywords: Calcified chronic subdural hematoma, craniotomy, treatment

Introduction

Chronic subdural hematoma (CSH) develops due to cortical, bridge vein or sinus laceration or cortical vessel laceration in cases with weak arachnoid trabecula following minor trauma. Fibrin deposits accumulate on clot surface and adhere to dura. Fibroblasts arising from dura surround the clot and outer and inner capsule develops and separates dura and arachnoid. Capsule strengthens with connective tissue in a few months and hyalinises in a couple of years. Degradation of intracapsular blood and fibrin, increase in osmotic pressure, liquid flow through semi permeable capsule, intracapsular bleeding of fragile, highly permeable neo vascularization results with enlarged hematoma. Calcification of hematoma is quite rare [1-5].

We present two cases with calcified chronic subdural hematoma (CCSH).

Case report

Case 1: 35 years old female admitted to our hospital with epilepsy. She did not have a complaint up to this age. She did not have a history of trauma. Her neurologic examination was found to be normal (Table 1). Her Magnetic Resonance Image (MRI) revealed a concave CCSH 12×8×20 cm involving the left subdural area (Figure 1).

Case 2: 58 years old male have been undergoing checkup examination in every 6 months. Bilateral primary optic atrophy was detected at her last checkup examination. He was diagnosed as CCSH incidentally (Table 1). His cranial MRI revealed 10×5×2 cm CCSH at right subdural area (Figure 2).

We performed a wide craniotomy and duratomy including the whole of CCSH reaching dural arachnoid border and we dissected the calcified mass from arachnoid and invaginations to the brain in both of the patients. We paid attention not to injure arachnoid.

In both of the patients the masses was resected totally in form of small pieces by rongeur (Figure 3A-C). Following the washing up of the cortex we applied closed drainage for 1 day. We applied antibiotic and antiepileptic to both of the patients in time of admission to the hospital. We recommended antiepileptic and elastic bandage for 1 month and to cease antibiotic therapy at the 7th day.
Calcified chronic subdural hematoma

Neurological deficit did not develop and the patients were discharged at the 3rd day with recommendation of antiepileptic therapy.

Discussion

CSH develops following minor head trauma and as seen in our patients most of the cases do not remember trauma. Because the process is slow due to cortical adaptation neurologic deficit does not develop even when the thickness of hematoma reaches to 2 cm. Our first case was diagnosed following epilepsy and the second incidentally.

Epilepsy develops in 22% calcification occurs in 0.3-2.7% of the cases with CSH. Most of them settle at the parietal area and near superior sagittalsinus [1, 6-9].

It has elliptical shape with thick inner membrane, none liquefied, and has sinusoidal blood vessels and partially adhere to arachnoid and invaginates into the cortex. Therefore, we removed the mass totally in small pieces in order to preserve cortex. CCSH must be differentiated from calcified subdural empyema, calcified meningioma, calcified convexity dura, calcified arachnoid, histopathologic examination is required for definite diagnosis. In CSH calcification develops in 6 months, ossification occurs in a couple of years. Ossification of CSDH in our patients indicate that trauma occurred years ago and the patients forgot or disregarded the minor trauma [2, 3, 5, 7, 9].

Calcification of a tissue depends on the multiplication of extracellular Ca\(^{2+}\) and PO\(_{4}^{2-}\) amount when this multiplication 3.4×10\(^{-6}\) mol/liter Ca\(^{2+}\) and deposits as Ca\(^{2+}\) hydroxy phosphate occurs in all tissue, but calcification prevented by breakdown of phosphatase which exits in tissue in vivo.

If there is not a break down in calcium hydroxy phosphate it transforms in to calcium hydroxy apatite in 2-3 weeks and cannot be dissolved.

Table 1.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Gender</th>
<th>CP</th>
<th>NS</th>
<th>HHT</th>
<th>NP</th>
<th>MRI T1 Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>35</td>
<td>F</td>
<td>Epilepsy</td>
<td>Normal</td>
<td>No</td>
<td>No</td>
<td>Hyperintense left calcified chronic subdural hematoma</td>
</tr>
<tr>
<td>2</td>
<td>58</td>
<td>M</td>
<td>No</td>
<td>Bilateral primary optic atrophy</td>
<td>No</td>
<td>No</td>
<td>Hyperintense left calcified chronic subdural hematoma</td>
</tr>
</tbody>
</table>

CP (Clinical Presentation), NS (Neurological Signs), HHT (History of Head Trauma), NP (Number of Previous Operations).

Figure 1. T1-weighted axial magnetic resonance image shows hyperintense left calcified chronic subdural hematoma, 1st case.

Figure 2. T1-weighted sagittal magnetic resonance image shows hyperintense left calcified chronic subdural hematoma, 2nd case.
Calcified chronic subdural hematoma

Therefore local factors, metabolic predisposition plays an important role in calcification [3-5, 7, 9, 10].

Inadequate nourishment of the capsule and the content results with calcification due to tissue necrosis and absence of phosphatase enzyme. It is reported that hemorrhage and calcification at the subdural area mostly occurs due to over drainage following ventriculoperitoneal shunt operation for hydrocephalus [2, 4, 5, 7, 8].

We did not observe deficit in both of our patients following operation and discharged them at the postoperative 3rd day with recommendation of antiepileptic.

Disclosure of conflict of interest

None.

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References

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