

# Pseudotumoral Chronic Subdural Hematomas on Two Cases

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## Abstract

**Introduction:** Calcified forms with pseudo-tumor symptomatology of chronic subdural hematomas are rare. They are the result of slow bleeding over several years. The main etiology is related to the complications of the ventriculoperitoneal shunt (VP). The purpose of this study was to recall the peculiarities and physiopathology of its pseudotumoral hematomas through 2 observations. **Observation: Case 1:** 8-year-old patient with a history of ventriculoperitoneal shunt at 3 months of age for congenital hydrocephalus, was admitted for functional impotence of the left-side of the body of insidious onset spreading over 9 months in a chronic headache, blurred vision and generalized seizure. CT scan showed a heterogeneous subdural hematoma of the right frontoparietal with calcifications. The patient underwent an excision by morcellation of a yellowish, friable partly calcific mass. The postoperative history was marked by a total recovery of the neurological deficit. There was no recurrence at 6 months postoperatively. **Case 2:** 11-year-old adolescent, treated with VP shunt at 6 months of age for post-meningitic hydrocephalus, was admitted for helmet headache, dizziness, lightheadedness and apathy progressing for 3 years. CT scan showed hypodensity of right peri-hemisphere with calcified linings, exerting a mass effect on the medial structures. The patient was given a block excision of a calcific mass with blood content. The evolution was marked by the complete resolution of seizures and hemiparesis. There was no recurrence at 6 months postoperatively. **Conclusion:** Calcified subdural hematomas are rare and consecutive to the complications of VPS. The clinical signs are those of a benign brain tumor. Treatment is dominated by the difficulties of cerebral reexpression.

## Keywords

Calcified Subdural Hematomas, Pseudo, Tumor Symptomatology, Overdrainage

## 1. Introduction

Chronic subdural hematoma is a collection of aged blood in the subdural space. The tissue and calcified forms with pseudotumoral symptomatology are rare and are estimated between 0.3% and 2.7% of subdural hematomas [1] [2] [3] [4]. The actual mechanisms leading to this evolution are unclear; but they are most often witness of slow bleeding over several years and behave like true benign tumors [5] [6] [7] [8]. The etiologies are multiple, but the majority of reported cases follow the complications of ventriculoperitoneal (VP) shunt [4] [5] [9]. The purpose of this study was to recall the peculiarities and physiopathology of its pseudotumoral hematomas through 2 observations.

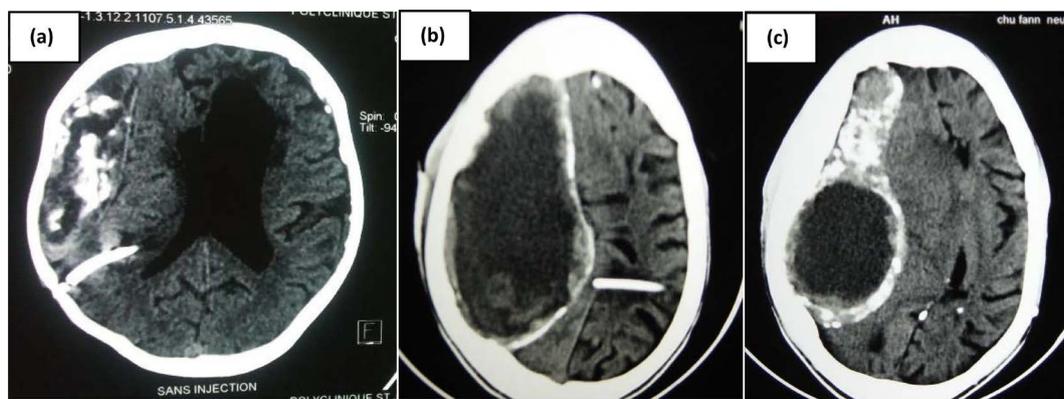
## 2. Observation

### 2.1. Case 1

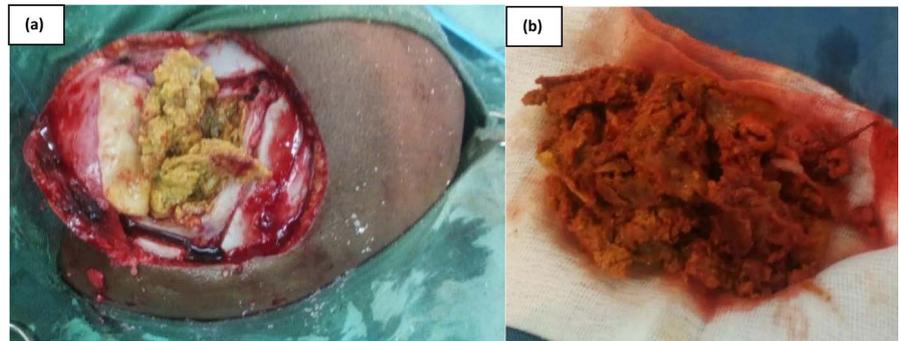
8-year-old patient with a history of ventriculoperitoneal shunt at 3 months of age for congenital hydrocephalus, who was admitted for functional impotence of the left-side of the body of insidious onset spreading over 9 months in a chronic headache, blurred vision and generalized seizure. No vomiting observed. Clinical examination noted a normal consciousness, left spastic hemiparesis at 4/5. Cerebral computed tomography showed a heterogeneous subdural hematoma of the right fronto-parietal with calcifications (**Figure 1(a)**). The patient underwent an excision by morcellation of a yellowish, friable partly calcific mass (**Figure 2**). We were able to perform an internal and external membranectomy. In addition, he was concomitantly given partial ligation of the distal catheter of VP shunt at the thoracic level. The postoperative history was marked by a total recovery of the neurological deficit. No recurrence of the hematoma at 6 months of follow-up. He still continues his anti-convulsant treatment.

### 2.2 Case 2

11-year-old adolescent, treated with VP shunt at 6 months of age for post-meningitic hydrocephalus, admitted for helmet headache, dizziness, lightheadedness



**Figure 1.** Tomodensitometric images of the heterogeneous subdural hematoma partly calcified of the first patient (a) and contralateral to the VP shunt for the other ((b) and (c)).



**Figure 2.** Macroscopic appearance of the intraoperative hematoma ((a), (b)) operative specimen. Yellowish mass, friable partly calcified.

and apathy progressing for 3 years. Generalized seizure observed. The examination revealed a predominantly right-sided static cerebellar and kinetic syndrome; a left spasmodic hemiparesis; and a papilledema in the fundus examination. Cerebral computed tomography showed hypodensity of right peri-hemisphere with calcified linings, exerting a mass effect on the medial structures (**Figure 1(b)**, **Figure 1(c)**). The patient was given a block excision of a calcific mass with blood content. The evolution was marked by the complete resolution of seizures and hemiparesis but with persistent cerebellar syndrome. No recurrence at 6 months of follow-up.

### 3. Discussion

Pseudotumoral chronic subdural hematomas are inherent in the duration of hematoma evolution, whatever its etiology [2] [6] [9] [10]. But the majority of cases reported are related to VP shunt complications [4] [5] [9] [11] [12]. The incidence would be higher if long-term systematic brain CT was performed in all patients. Overdrainage promotes the occurrence of subdural hematomas by intracranial hypotension [13] [14] [15]. Overdrainage is not permanent because it occurs only during the siphon effect of verticality, so it causes occasional minor bleeding. During normal drainage times, the optimal intracranial tension, helps stop bleeding. This alternation of overdrainage and normal drainage promotes a certain stability of the hematoma, which means that the patient can remain asymptomatic for a long time [16]. This stability of the bleeding volume over several months or even years is a favorable situation for the calcification of the hematoma and the pseudotumoral appearance of the symptomatology. Factors promoting this calcification are clot dehydration, low vascularization and thrombosis. Thus the pathogenesis of the calcification of these hematomas does not imply any particular consideration [8] [17]. Calcifications are due to parietalcalcium overload and cholesterol degeneration of certain elements in the central necrotic mass and in the vicinity of dead cells, which are probably remnants of macrophages [17]. The major therapeutic problem they pose is the difficulty of postoperative cerebral re-expansion favored by the inner shell and the loss of cerebral compliance, hence the high frequency of recurrence. Several techniques

have been developed to try to solve this problem such as the suturing of the inner membrane to the dura mater [18]. In both cases, to treat overdrainage and promote cerebral re-expansion to reduce recurrence, we proceed to partial ligation of the distal catheter.

#### 4. Conclusion

Calcified subdural hematomas are rare and result in slow, long-term bleeding. The clinic is that of a benign brain tumor. The etiologies are numerous but most often they are consecutive to the complications of the ventriculoperitoneal shunts. The difficulties of cerebral re-expression constitute the major therapeutic problem.

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