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Symptomatic Calcified Subdural Hematomas

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Key Words

Calcified subdural hematoma · Raised intracranial pressure · Craniotomy · Congenital hydrocephalus · Shunt surgery · Good outcome

Abstract

Two unique cases of chronic calcified subdural hematomas are reported in children as a long-term complication of a ventriculoperitoneal shunt. Both the patients had undergone shunt procedures in infancy for congenital hydrocephalus. In one patient, the cause of the hydrocephalus was aqueduct stenosis, while in the second patient, a lumbar meningomyelocele was associated with hydrocephalus. In both these patients, a ventriculoperitoneal shunt was done in infancy. In one of them, following the shunt surgery, a bilateral subdural collection was noticed which required burr hole evacuation. Both the patients remained asymptomatic for 9 years, when they presented to our center with acute raised intracranial pressure and contralateral hemiparesis. Both the patients had a relatively short history and had altered sensorium at admission. Surprisingly, in both the patients, the CT scan showed significant mass effect producing calcified subdural hematomas. The shunt systems were

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Accessible online at: www.karger.com/journals/pne found to be working well at surgery. Craniotomy and excision of the calcified subdural hematomas was undertaken. Postoperatively, the patients showed satisfactory recovery, and at discharge the patients were doing well. At the follow-up at the outpatient clinic, the patients were asymptomatic.

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Introduction

Calcified subdural hematoma is an uncommon condition [1-5], and can result from postmeningitic subdural effusion in chronic subdural hematoma, and less frequently as a long-term complication of a ventriculoperitoneal shunt. Sometimes, calcified subdural hematomas are detected on routine follow-up CT scans in asymptomatic patients. A variety of presentations have been reported in the literature. We present 2 unusual cases with calcified subdural hematomas presenting long after the ventriculoperitoneal shunt procedure for congenital hydrocephalus. The cases are discussed in the light of the available literature, and the possible mechanisms for their problems are hypothesized.

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Fig. 1. CT brain scan showing a large calcified subdural hematoma in the right temporoparietal region and a smaller hematoma in the left temporoparietal region.

Case Reports

Case 1

A 10-year-old boy was admitted to our neurosurgical ward with a history of headache with vomiting for a week and progressive deterioration of the conscious state for 3 days. The patient was a known case of congenital hydrocephalus secondary to aqueduct stenosis, in whom ventriculoperitoneal medium-pressure shunting had been performed 9 years ago. Three months following the shunt, the patient was readmitted with bilateral subdural hematomas for which burr hole evacuation was carried out. Thereafter, the patient remained asymptomatic and was repeatedly followed up at the outpatient clinic.

At admission, this time, he was drowsy, disoriented, and the fundi revealed moderate papilledema. There was a mild left hemiparesis. The biochemical parameters were unremarkable. Contrastenhanced CT scan revealed a bilateral chronic subdural hematoma. On the right side, the hematoma was biconvex with calcified margins and with areas of calcification in its contents (fig. 1). On the left side, it revealed a concavoconvex shape and the inner concave margin showed a thick calcification. There was a significant shift of the midline from right to left. There was evidence of thinning of the bone on the right side, overlying the hematoma. On the basis of the CT findings, a diagnosis of bilateral calcified subdural hematoma with mass effect was made.

In view of the clinical condition of the patient, an emergency surgical procedure was undertaken. On the right side, as the mass effect was significant, a large osteoplastic bone flap was made. The dura was relatively thin. The outer membrane was excised. The cavity contained a paste-like soft, yellowish material which could be sucked out easily. The inner membrane was thick and calcified and fixed to the brain at places. With difficulty, partial excision of the inner membrane could be done. Postoperatively, the patient had a good recovery. A repeat CT scan prior to discharge revealed a reduction of the mass effect and the expansion of the brain on the right side (fig. 2). The patient was discharged on the 7th postoperative day. He was



Fig. 2. Postoperative CT brain scan showing significant relief in the compressive effect of the partially excised calcified subdural hematoma.

regularly followed up at the outpatient clinic. Histological examination of the tissue revealed fibrous tissue with focal calcification consistent with calcified subdural hematoma. At the last follow-up 1 year later, the patient was asymptomatic.

Case 2

A 9-year-old boy was admitted to our unit with a history of repeated vomiting and altered sensorium for 2 days. The patient was operated for a lumbar meningomyelocele at the age of 1 day. He also underwent ventriculoperitoneal medium-pressure shunting at the age of 6 months for progressive hydrocephalus. The patient was regularly follwed up at the outpatient clinic and remained asymptomatic.

At admission, this time, the patient was drowsy and the fundi revealed papilledema. He also had spastic hemiparesis on the left side (power 4/5). A contrast-enhanced CT scan showed a large rightsided chronic subdural hematoma with calcification in both the inner and outer membrane. The contents of the hematoma also showed areas of calcification (fig. 3). There was a significant compression of the right lateral ventricle, and the left lateral ventricle was dilated. There was a mild shift of the midline to the left. A right-sided large osteoplastic craniotomy was performed as an emergency procedure. The subdural hematoma was evacuated, and the calcified membrane was totally excised microsurgically. The brain surface appeared normal. Good brain pulsation and expansion of the brain was observed at the end of the surgery. Postoperatively, the patient improved rapidly. A repeat CT scan prior to discharge revealed good expansion of the brain, and there was no shift of the midline (fig. 4). However, the ventricles were moderately enlarged with bone flap bulging outwards. As the patient had a good recovery and was asymptomatic, the shunt was not revised, and he was discharged on the 10th postoperative day. Histological examination of the tissue revealed fibrous tissue with focal calcification consistent with calcified subdural hematoma. He was regularly followed up; 6 months following the surgery, he was asymptomatic, and a repeat CT scan showed no hydrocephalus.

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Fig. 3. CT brain scan showing a large compressive calcified subdural hematoma in the right frontoparietal region and a thin subdural hematoma in the left parietal region.

Discussion

Chronic subdural hematoma is a common condition; surprisingly, however, calcification in a chronic subdural hematoma is rare [1-5], and until recently, has been published as single case reports [6, 7]. The exact incidence of calcification is not known, though in 1973, Harwood-Nash [8] reported a 10% incidence in the chronic subdural hematoma detected by skull film in children. Calcification and ossification in chronic subdural hematoma have long been reported [1, 3, 9], and severe forms of calcification in the subdural hematoma have been reported as ossified chronic subdural hematoma [6, 9, 10]. It has also been reported in subdural empyema [11, 12].

Calcification can occur in unilateral or bilateral chronic subdural hematomas [5, 7, 12, 13]. Both our patients had a bilateral chronic subdural hematoma; however, calcification was noticed bilaterally in the first case, while in the second case it was limited to one side only.

Calcification is reported in chronic subdural hematoma of various etiologies. Several authors have reported calcification in traumatic subdural hematoma [1–3, 8, 10, 14, 15], which is relatively commoner than other causes of subdural hematoma. Rarely, calcifications is noticed in subdural hematomas or subdural effusion secondary to ventricular shunting procedures [13, 16–20]. Shunting procedures could include ventriculoatrial [19], ventriculocaval [18], or ventriculoperitoneal shunts [16, 17]. Calcification can occur in subdural hematomas with normal



Fig. 4. Postoperative CT brain scan showing a complete excision of the right frontoparietal calcified subdural hematoma with expansion of the cerebral parenchyma and decompression of the cerebral ventricles.

ventricular pressure [20] or even in patients with arrested hydrocephalus [17]. In both our patients, the ventriculoperitoneal shunts were done a long time back (9 years). Hence, it is likely that the subdural hematomas on which the calcification set in were asymptomatic. Calcification is also reported in subdural effusions due to pyogenic meningitis in infants and children [13, 21–23]. Ludwig et al. [13], in 1983, reported 5 patients with calcified subdural hematoma, among them, postmeningitic and postshunt subdural hematoma accounted for 2 each. Two patients presented with postshunt calcified subdural hematoma; calcification occurred 3 and 8 years after the shunting. One patient had bilateral calcified subdural, while the other patient had unilateral calcified subdural hematoma.

Calcified subdural hematoma is more often reported in children [3, 6, 8, 13, 22, 24]. Rarely, calcified subdural hematoma is reported in the elderly [2, 4, 7]. Debois and Lombaert [2], in 1980, reported a calcified subdural hematoma in a 70-year-old man who had had a head injury 35 years prior to the presentation. Both our patients were children under 10 years of age in whom shunt surgery was performed 9 years prior to their presentation. The above point highlights the long duration a hematoma might require for the slow deposition of calcium. Similar long duration was reported by Ludwig et al. [13]; however, the usual interval between hemorrhage and the development of calcification varies between 6 months and many years [1, 2, 8, 9].

Clinical presentation of calcified subdural hematoma varies widely, from asymptomatic [13, 14, 25, 26] in some to acute raised intracranial pressure in others [6, 13]. Ide et al. [14] in 1993, reported 3 cases in whom the calcified chronic subdural hematoma was without symptoms. One among the 5 calcified subdural hematomas reported by Ludwig et al. [13] was an 8-year-old girl who had had a ventricular shunt for hydrocephalus in her first year of life. She was asymptomatic, and on routine checkup bilateral calcified subdural hematomas were noticed. The remaining 4 patients were symptomatic. One patient each had a gait disturbance, chronic headache and deteriorating vision. Two patients had a history of seizures. Iplikcioglu et al. [10] reported a 13-year-old girl with epileptic seizures and mental retardation. Both our patients had a short history of raised intracranial pressure and presented with altered sensorium. In one patient, the history was of 1 week duration, while in the second patient, the history was of only 2 days. Sometimes the patient may be asymptomatic and present only after a history of injury or seizure which can precipitate the problem [13]. Our patients were asymptomatic, and there were no presenting factors like trauma, seizure or fever. Pathogenesis of the raised intracranial pressure in our patients remains unexplained. Turgut et al. [8], in 1997, reported a similar problem in a child who presented with acute raised intracranial pressure. Investigation revealed a huge ossified hemispheric subdural hematoma.

Pathogenesis and mechanism of the slow calcification by and large remains unclear [3, 10, 13, 20, 26]. As early as 1943, Boyd and Merrel [1] had suggested the possibility of metabolic factors leading to the calcification. In 1961, Afa [9] suggested several factors like poor circulation and absorption in the subdural space and vascular thrombosis. McLaurin and McLaurin [3], in 1966, suggested the possibility of local factors, as their patient with bilateral chronic subdural hematoma had unilateral calcification. In chronic subdural hematoma, dense collagen deposits occur on the membrane, forming a fibrotic capsule. At a very late stage, this fibrotic capsule calcifies due to the progressive mineralization [14, 27]. Calcification or ossification may be found in both the visceral and parietal layers of the capsule and in the hematoma. These findings were recorded in our patients.

Radiological investigations were initially limited to skull X-ray [1, 9, 16, 18, 20] prior to the CT scan era. With the availability of the CT scan, the diagnosis has now become easy, and various types of CT scan appearances have been described [1, 10, 12, 13, 24, 25]. Debois and Lombaert [2], in 1980, were the first to report a calcified

chronic subdural hematoma on a CT scan. A typical picture has been reported, termed 'armored brains' [1, 9, 13, 26, 28] when the entire hemisphere is involved. The classical CT scan picture by Ludwig et al. [13] has shown that generally the postshunt calcified subdural hematomas are less extensive. Management is largely individualized. Patients with seizures require careful monitoring of the anticonvulsant therapy. Successful surgical removal has been reported only sporadically [9, 10, 12, 13, 16, 25]. Generally, the technical difficulties of complete excision of these hematomas have been highlighted [2, 9, 13]. In our first case, total excision was not possible; luckily, however, in the second case, a total excision was possible. McLaurin and McLaurin [3], reported their results in 6 children operated for calcified subdural hematoma. They did not notice any improvement in their IQ following the surgery. Iplikcioglu et al. [10] reported a 13-year-old boy who had a partial improvement in the neurological deficit; however, he became seizure free following the surgery when maintained on antiepileptic drugs. Improvement in epileptic fits has been reported postoperatively by many authors [9, 10]. Both our patients improved to a normal state following the surgery and remained asymptomatic.

Conclusion

Calcified chronic subdural hematoma 'armored brain' is a rare complication of shunt surgery. Not even 20 cases have been reported. We present 2 cases, presenting with acute raised intracranial pressure necessitating surgical intervention. The patients improved rapidly postoperatively and have remained asymptomatic. Calcified subdural hematoma, though rare, must be borne in mind, and interval CT scan would be appropriate to detect and follow up such patients.

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