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Bilateral ossified subdural hematoma: Literature review

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Abstract

Calcified/ossified chronic subdural hematoma is a rare clinical and radiological entity. It can occur as a rare complication of shunt surgery, or it may occur spontaneously. Computed tomography imaging is a practical diagnostic method. The clinical presentation is essential in the decision of surgical intervention. This review aimed to discuss the limited number of reported bilateral calcified/ossified chronic subdural hematoma cases in the literature, which is an even more rare condition. We reviewed 16 case reports retrieved from PubMed as presenting a similar case.

Keywords: Bilateral calcified subdural hematoma, computed tomography, treatment

Introduction

Chronic subdural hematoma (CSDH) mainly develops due to cortical, bridge vein, sinus, or cortical vessel laceration in patients with weak arachnoid trabecula following minor trauma. Most patients do not even recall the exposed trauma [1]. Its incidence increases with aging, significantly above the age of 65. With the prolongation of life expectancy in developed and developing countries, the frequency of CSDH is getting higher. Other factors in the etiology include alcoholism, liver cirrhosis, chronic renal failure, and hematological disease, besides head trauma history. The increased use of anticoagulants and antiaggregant agents in the elderly population and coronary heart diseases in the recent decades have a promotive role for CSDH [2]. Calcified chronic subdural hematoma (CCSDH) is a rare disease that constitutes approximately 0.3-2.7% of all CSDH [3]. Von Rokitansky described the first CCSDH report in an autopsy performed in 1884. The first CCSDH operation was performed by Goldhahn on an 11-year-old child in 1930 [4]. We aimed to discuss the clinical features, underlying causes, and treatment strategies of bilateral calcified subdural hematoma (BCSDH) within the literature.

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Materials and Methods

We searched the PubMed database from the beginning of the database until December 2020. The keywords used for the search were: “Calcified subdural hematoma,” “Ossification of subdural hematoma,” and “armored brain.” Eighteen cases in the literature were identified. Two report were excluded due to failure in accessing the full text, abstract or any electronic record [5,6]. Also, we presented our case below. A total of 17 cases were summarized.

Exemplary case

A 70-year-old female patient diagnosed with bullous pemphigoid was hospitalized in the dermatology ward to initiate steroid therapy. Her medical history revealed ischemic stroke three years ago, chronic renal failure, and diabetes mellitus. In the neurological examination of the patient who did not have any neurological complaints, 4/5 motor strength (sequelae ?) was noticed in the right lower extremity. The cranial computerized tomography (CT) and magnetic resonance imaging (MRI) results were compatible with bilateral frontal ossified subdural hematoma (Figure 1). She claimed no trauma or shunt history. The neurosurgery department recommended outpatient follow-up as the patient was asymptomatic.

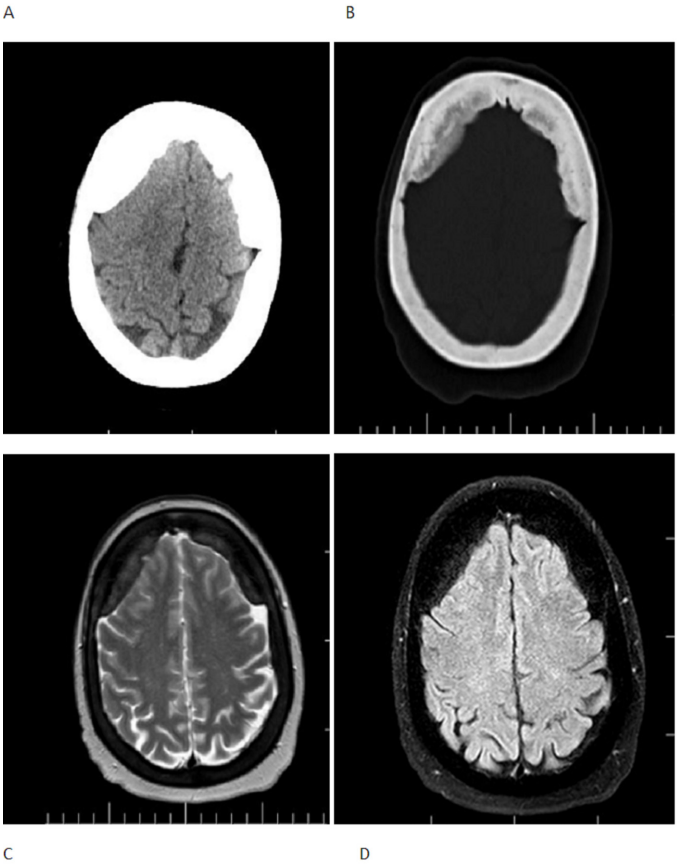

Figure 1. Computed tomography (A and B) and Magnetic rezonance image (C and D) done in showing bilateral calcified/ossified chronic subdural haematoma

Results

Age and sex distribution

The age of the patients ranged from 3 months to 86 years. In our literature review, a total of 17 cases were found, including our case. Five of the cases were female, and 12 were male. The summary of the case reports was presented in Table 1 [7-22].

Etiology

A total of 11 cases had a shunt history. Three had a ventriculoatrial (VA) shunt, and the remaining 8 had a ventriculoperitoneal (VP) shunt. Except for our case, one of the 3 cases had a history of skull fracture and subarachnoid hemorrhage due to birth trauma, another had a history of operation due to bilateral subdural hematoma, and the third case had a history of traffic accident. In patients with a history of shunt, the time to detect bleeding varies between 10 months and 40 years. In our case, there was a history of ischemic stroke 3 years ago.

Presenting symptom

The presenting symptoms include episodic headache, gait disturbance, altered level of consciousness, nausea, change of personality, painless gradual loss of vision, progressive right hemiparesis, seizure, vomiting, blurring of vision, papilledema. Some patients also have non-neurologic symptoms like abdominal pain, chills, fever, and polyuria which were related to shunt dysfunction [16].

Table 1. Cases of bilateral chronic subdural haematoma with calcified/ossified

	Author &publication year	Clinical features	Age (years)&Sex	Shunt history	Time between shunt and bilateral CSH development	History	C/S	Treatment
1	Mori N[7] et al., 1982	progressive right hemiparesis, seizure and mental physical retardation	5/M	-	-	birth injury caused by forceps delivery resulting in skull fracture and subarachnoid hemorrhage	C	craniotomy with an interval of 1 year
2	Sparado[8] et al., 1987	gait disturbance	11/M	VA	2,5 year		C	conservative
3	Al Whaibi[9]., 2003	asymptomatic	3month/M	VP (5 days after birth)	10 month		C	follow up
4	Dinç[10] et al., 2006	episodic headache	29/F			operation due to bilateral CSH 6 years ago	C	follow-up
5	Dimogerontas &Rovlias[11]; 2006	headache , vomiting, fever and deterioration of the conscious state	43/M	VA	40 year		C	follow-up (VA shunt was re-placed by VP shunt)
6	Dammers[12] et al., 2007	falls, headache, epileptic seizures, and a bilateral oculomotor paresis	67/M			meningitis at the age of 3 months, re-sulting in hydrocephalus and mental retardation	C	bilateral small craniotomy following left-sided twist drill craniostomy with controlled drainage
7	Papanikolaou[13] et al., 2008	symptoms of increased intracranial pressure	33/M	VA (2 months after birth)	33 year		C	follow up (VP shunt insertion)
8	Galldiks[14] et al., 2010	change of personality	86/M			hypertension, traffic accident 3 months ago	C	bilateral burr hole trepanation

9	Akhaddar[15] et al., 2011	episodic diffuse headache	7/M	VP	4 year		C	follow-up
10	Petraglia[16] et al., 2011	abdominal pain, nausea and chills	38/F	VP (in infancy)	38 year		C	follow-up (shunt revision)
11	Taha M[17], 2012	seizure	12/M	VP (at birth)	12 year	1	C	Follow up
12	Salunke[18] et al., 2013	history of headache and vomiting associated with blurring of vision for three days. papilledema	15/M	VP	11 year	-		bilateral burr holes and drainage of collection.
13	Garg[19] et al., 2013	polyuria and painless progressive visual diminution	24/M	VP	4 year		C	????
14	Goyal[20]et al., 2013	headache, seizure	15/F	-	-	-	C	Bifrontal craniotomy
15	Siddiqui[21] et al., 2016	progressive visual deterioration and polyuria	30/M	VP	11 year		O	bilateral craniotomies in a single sitting.
16	Viozzi[22] et al., 2017	increased frequency of epileptic seizures, progressive drowsiness, nausea, and vomiting	15/F	VP	15 year		O	follow up (shunt revision)
17	Present study	asymptomatic	70/F			hypertension, ischemic cerebrovascular accident	O	follow up

M- Male; F- Female; CSH- Chronic subdural haematoma; VA- ventriculoatrial shunt;
VP- ventriculoperitoneal shunt;
C/O: Calcified/Ossified

Treatment

The general approach in the treatment of these patients was follow-up for hematoma. The majority of patients did not undergo any neurosurgical intervention for hematoma, while only 4 cases were received a hematoma evacuation operation. Two out of 4 who had surgery had shunts, and the other two did not. However, the patients with shunts were received specific treatments targeting shunt dysfunction. Our completely asymptomatic case needed no neurosurgical intervention.

Discussion

Calcified CSDH (CCSDH) is an infrequent radiologic picture. It also appears as a long-term complication in patients who underwent ventricular shunt therapy for hydrocephalus [11,23,24]. The possible mechanism was based on the overdrainage of cerebrospinal fluid by ventricular shunt, reduced intracranial pressure, partial collapse of the brain, stretching of bridging veins due to widening of the subdural space and finally hemorrhage formation [15]. Some authors pointed at brain atrophy, microcirculation disorder, meningitis, encephalitis, and premature delivery as well [26]. The pathogenesis of calcified CSDH is not revealed clearly but the hemorrhage itself may be major contributor as progression from hyalinization to calcification [26,28]. Vascular factors like poor circulation and absorption in the subdural space and also thrombosis have been suggested as the underlying mechanism of calcification in recent years [25]. Other possible explanations for calcification in CSDH include active vascular proliferation in damaged brain tissue and metabolic factors [8,27]. The hematoma formation-calcification time interval is reported as a few years but can vary from 6 months to many years [29-31].

Bilateral calcified chronic subdural hematoma = Armored brain

Bilateral occurrence of CSDH is rarer, classically named “armored brain,” due to the classic CT appearance, results from extensive linear calcification lines [11]. The lines can extent in both the parietal and visceral capsules, or through the hematoma itself [15]. Physicians should be aware that the terms “calcified” and “ossified” are not the alternative for another. Ossification presents for the organized CSDH after hyalinization and calcification steps, respectively. Therefore, evidence of bone formation is expected in case of ossification. The literature offers only 2 cases with bilateral ossified subdural hematoma, which our case presents the third [21,22].

The presenting symptoms of CCSDH can vary in each case but headache is the most common. As the majority of cases are asymptomatic, they can suffer from the reflections of severe acute intracranial pressure as well [15,28,33]. The clinical signs of CCSDH are not very different form non-calcified CSDH and include headache, weakness, numbness, gait disturbance, dysphasia, seizures, memory impairment and altered level of consciousness [6,32]. The pictorial appearance on CT and MRI

studies provides the radiological diagnosis but in some cases, the exclusion of other calcified extra-axial lesions (calcified epidural hematoma, subdural empyema, meningioma, calcified arachnoid cyst, calcified convexity dura mater with acute epidural hematoma, and malignant tumors) cannot be easy performed. Therefore, pathological examination is essential for confirming the diagnosis [6]. The general approach is surgery for infants/young patients or patients with neurological symptoms, and non-surgical follow-up for asymptomatic patients with CCSDH which is still controversial [28,34-36]. Some authors advocated the unnecessity of surgery in elder population [33]. On the other hand, some justified surgery for relieving compression even in asymptomatic cases to prevent future brain damage [28]. Considering surgery in symptomatic cases as a result of mass effect of the lesion may be beneficial due to potential expanding pattern in some CCSDH examples. It is also a fact that the symptoms can improve after surgery [34]. But the occurrence of surgery-related complications is always on the table. For example, Moon et al. reported a case with acute subdural hematoma on the contralateral side after removal of CCSDH and suggested that perioperative parenchymal shifting proceeded by tethering venous structure damage as possible mechanism [36]. The regarding complication with same characteristics was described in a 16-year-old boy by Tatlı et al [37].

Other causes of the severe cerebral edema after surgery in the case could include NPPB (normal perfusion pressure breakthrough), seizure, and electrolyte disorder. NPPB is likely due to pressure autoregulation dysfunctionality of the vasculature on CCSDH side. The surgical removal of CCSDH may end up with tissue swelling or bleeding via NPPB theory [38,39]. Another report described a case who underwent surgery for CCSDH and developed bilateral tension pneumocephalus on the 3rd day following craniotomy. Their patient exhibited subsequent right-sided weakness on the upper extremity which showed complete recovery after frontal burr-hole intervention [40].

Conclusion

Although CSH is a common pathology in neurology and neurosurgery practices, calcified/ossified subdural hematoma is rare. In addition, BCCSDH is limited with 17 patients in the literature, including our case. The vast majority of them have a shunt history, and they show shunt dysfunction-related symptoms. Shunt dysfunction treatment constitutes the majority of the treatment.

Conflict of interests
The authors declare that they have no competing interests.

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