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Case Report

Armored brain as a late complication of CSF overshunting: A rare case report ☆

Bereket Girum Beyene, MD[a,*], *Michael Teklehaimanot Abera, MD*[b], *Tesfaye Abebe Zeleka, MD*[a], *Abdi Alemayehu Dhuguma, MD*[a,1]

[a] *Adama Comprehensive Specialized Hospital Medical College, Department of Radiology, Adama, Ethiopia*
[b] *Addis Ababa University, College of Health Sciences, Department of Radiology, Addis Ababa, Ethiopia*

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ABSTRACT

We hereby present a case of an 18-year-old patient who, following initial cerebrospinal fluid overshunting to relieve a congenital hydrocephalus for Dandy-Walker malformation, developed chronic calcified subdural hematoma or an armored brain as a late complication. Chronic calcified subdural hematoma or armored brain is generally rare, and it is even rarer after overshunting. In this report, we present a rare case of bilateral chronic subdural hematoma, also known as "armored brain", 18 years after a ventriculoperitoneal shunt was placed during infancy. We emphasize the appearance of the lesion on a noncontrast brain computed tomography.

Introduction

Chronic calcified subdural hematoma (CCSH), also known as armored brain, occurs when a subdural hematoma develops calcifications, a process that typically takes many years to develop [1–3]. It is usually linked to a hematoma that develops after a traumatic injury [2]. Cerebrospinal fluid (CSF) overshunting is a recognized cause of chronic subdural hematoma, but chronic evolution to calcification and clinical deterioration is rare [2,4]. Imaging studies are key to the diagnosis, and brain computed tomography (CT) is particularly advantageous as it can better outline calcifications over magnetic resonance imaging (MRI) [4,5].

Abbreviations: CCSH, chronic calcified subdural hematoma; CSF, Cerebrospinal fluid; CT, Computed tomography; MRI, Magnetic resonance imaging; VP, Ventriculoperitoneal.

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* Corresponding author.
E-mail address: berchgirum14@gmail.com (B.G. Beyene).
[1] First co-author.

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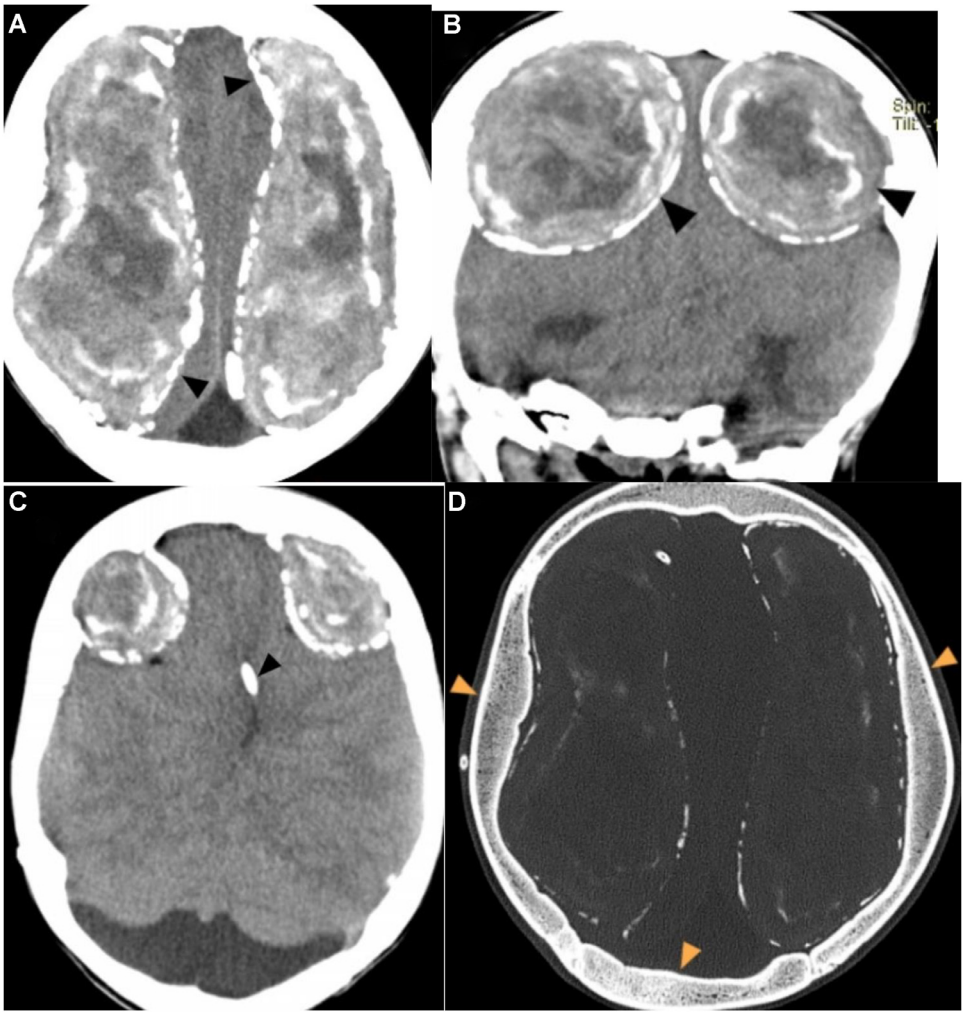

Fig. 1 – Noncontrast brain CT in axial (A) and coronal (B) sections show large bilateral calcified chronic subdural hematoma of the frontoparietal convexity (black arrowheads in A and B) and a bilateral trans tentorial herniation. A VP shunt catheter in a narrowed lateral ventricle (black arrowhead in C) and diffuse calvarial thickening (yellow arrowheads in D) are seen.

Clinical history

An 18-year-old boy who underwent ventriculoperitoneal (VP) shunt treatment in infancy presented with a chief complaint of a 1-month history of progressive headache and right side body weakness. He also reported a history of postural headaches, chronic nausea, and vomiting prior to the onset of his current symptoms. On a physical examination, he had decreased right upper and lower extremity power, each scoring 4/5. Otherwise, the rest of the examination was non-revealing. A complete blood count was normal. He underwent surgery early in life for congenital hydrocephalus due to Dandy-Walker malformation, and a VP shunt was placed.

A noncontrast head CT scan revealed a large bilateral chronic subdural hematoma with heavily calcified walls, causing bilateral downward transtentorial herniation (Figs. 1A and B). A VP shunt catheter is seen the narrowed lateral ventricles (Figs. 1C and 2), third ventricle is also slit-like and diffuse skull vault thickening is present (Fig. 1D), suggesting antecedent intracranial hypotension from CSF overshunting. There is also hypoplastic vermis, elevated tentorium and dilated 4th ventricle communicating with large posterior fossa cyst consistent with dandy walker malformation (Fig. 2). Based on the imaging findings, he was diagnosed with bilateral calcified chronic subdural hematoma complicated with transtentorial herniation secondary to overshunting + Dandy Walker malformation.

With this diagnosis, the patient was referred to another hospital for neurosurgical intervention.

Discussion

CCSH is extremely uncommon, with an incidence of about 0.3%-2.7% of all cases of chronic subdural hematomas [1]. Bilateral CCSH or “armored brain” has rarely been reported due to shunting for hydrocephalus [2,4]. Traumatic brain injury is the most important etiological factor for calcified or ossified

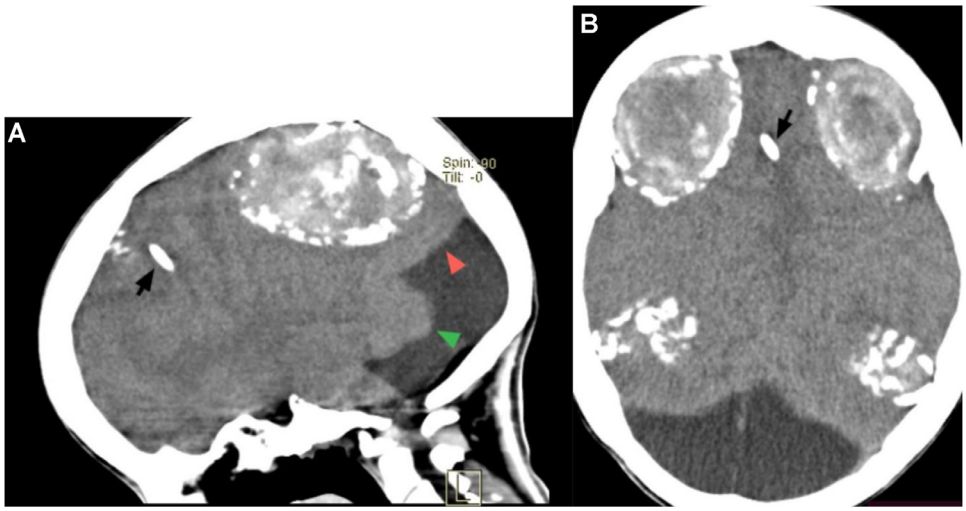

Fig. 2 – Noncontrast brain CT in sagittal (A) and axial (B) sections show vermian hypoplasia (green arrowhead in A), a midline cystic posterior fossa lesion communicating with dilated fourth ventricle, and elevated tentorium cerebelli (red arrowhead in A) suggestive of a Dandy-Walker malformation. A VP shut is seen anteriorly (black arrow in A and B).

chronic subdural hematoma, followed by postmeningitic subdural effusions [2]. It occurs more commonly in the younger age group because of lax adhesions between the dura and the calvarium [3].

The mechanism by which calcification occurs remains unclear, but a variety of local, vascular, and metabolic factors have been proposed [2,5]. Metabolic and Vascular factors where poor circulation and delayed resorption of the hematoma fluid within the subdural space are particularly important [2,5,6]. But a local factor is also likely to play a role, as calcification sometimes occurs unilaterally in bilateral hematomas [7,8]. The interval between the occurrence of the hematoma and the development of the calcification varies from 6 months to many years [9].

Clinical manifestations vary, but the majority of the patients are asymptomatic [2]. When symptoms arise, patients present with signs of increased intracranial pressure such as headache and hemiparesis, confusion, disorientation, papilledema, epileptic seizure, mental motor retardation, and decreased memory [2,4,8].

CT or MRI can diagnose most CCSHs and are considered reliable modalities [9]. Brain CT has classically been the imaging modality of choice and characteristically shows an extra-axial crescent-shaped hyperdense mass with thick hyperdense calcific margins that crosses suture lines. The condition has also been called ''Matrioska head'', a name derived from the famous Russian doll. These names reflect well the CT appearance, where another concentric skull appears to be inside the cranium [4,10]. The calcified hematoma may also show mass-effect midline shift and mixed density, a sign of recent hemorrhage [4].

Reports indicate that symptomatic and young patients require surgical treatment whereas asymptomatic elderlies are managed more conservatively [1,8]. There is a chance of damaging the underlying cortex during excision, which makes the surgery technically difficult because the calcified section tends to stick tightly to the dura mater and the brain surface [1,8,10].

In our case, the patient is symptomatic and exhibits signs of focal neurological deficit, warranting a neurosurgical referral.

In conclusion, in rare occasions, chronic calcified subdural hematoma or armored brain can be a complication of overshunting. This complication can occur a long time after surgery, as seen in our case. Noncontrast brain CT provides adequate details and is able to concurrently reveal imaging signs of overshunting. Treatment depends on age and clinical symptoms.

Patient consent

Written informed consent was obtained from the patient to publish this case report. Personal identifiers are not used in this paper.

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