Case Report

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Development of a delayed chronic subdural haematoma 3 years after traumatic brain injury with urinary incontinence: a case report

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Introduction: The authors present a case of a delayed chronic subdural haematoma, a rare occurrence that manifested 3 years after a traumatic brain injury, accompanied by an unexpected symptom of urinary incontinence. Chronic subdural haematoma (CSDH) is a well-known condition characterised by the accumulation of old, liquefied blood under the dura mater, usually following minor head trauma. However, the atypical presentation of CSDH in a young patient without predisposing factors and the association with urinary incontinence challenge conventional understanding. This report explores the clinical manifestations, radiological findings, and management of this exceptional case, providing valuable insights into this unusual presentation.

Case presentation: In this report, the authors present the case of a 23-year-old male with an unremarkable medical history, devoid of prior neurological deficits, who presented with persistent headaches, memory impairment, left-right disorientation, slurred speech, and urinary incontinence, troubling him for the past month. The patient had a history of a traumatic brain injury from a road traffic accident 3 years earlier, initially devoid of concerning symptoms. Imaging revealed a large heterogeneous mass lesion in the left fronto-parietal lobe consistent with a chronic subdural haematoma. The patient underwent surgical evacuation and excision of the haematoma, leading to the successful resolution of symptoms.

Clinical discussion: Conventionally, chronic subdural haematoma is observed in elderly individuals following minor head trauma. However, this case challenges the traditional understanding by highlighting its delayed occurrence in a young patient without known predisposing factors. This case emphasises the need to consider delayed presentations even without immediate neurological deficits. The unexpected symptom of urinary incontinence underscores the necessity of comprehensive evaluations to understand the associated neurological effects of CSDH. A surgical approach was crucial for both diagnosis and treatment, underscoring the significance of prompt intervention in such atypical cases.

Conclusion: This exceptional case sheds light on a delayed chronic subdural haematoma occurring years after traumatic brain injury in a young patient without known risk factors. The presence of urinary incontinence as a symptom further amplifies the uniqueness of this case. Understanding and recognising atypical presentations of CSDH is vital for accurate diagnosis and timely intervention. This report underscores the importance of vigilance and an integrated approach to managing patients with subdural haematomas, particularly in unexpected demographics and circumstances, to ensure optimal outcomes and patient well-being.

Keywords: atypical symptoms, chronic subdural haematoma, delayed presentation, diagnostic challenges, surgical intervention, traumatic brain injury, urinary incontinence, young patient

Introduction

A subdural haematoma refers to a condition where blood accumulates beneath the dura mater, which is one of the protective layers surrounding the brain, often resulting from bleeding of the bridging veins situated between the brain's protective membranes[1,2]. Chronic subdural haematoma (CSDH) is a distinctive type, characterised by the gradual accumulation of aged, partially liquefied blood, forming a chronic subdural haematoma[3]. Typically, CSDH becomes evident ~3 weeks after a head injury[1,3]. This condition predominantly affects the elderly population and is frequently associated with neurological deficits, even following minor traumatic events[4]. Symptoms can vary widely, ranging from alterations in mental status, such as mild confusion to coma[5], and commonly presenting as post-traumatic headaches and weakness or paralysis on one side of the body, often interspersed with clear intervals. Healthcare practitioners use specific timeframes to categorise CSDH presentations, distinguishing them as acute (within 3 days of trauma), subacute (within 4–21 days), or chronic (after 21 days).

Several factors contribute to the risk of developing CSDH, including advanced age, a history of prolonged alcohol misuse, and prior traumatic brain injuries, which can lead to significant cerebral atrophy[6,7]. This cerebral atrophy subsequently heightens the susceptibility to subdural haematoma (SDH) following

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minor head injuries or whiplash, even in the absence of direct physical impact. Additionally, the use of anti-platelet medications, direct oral anticoagulants, or vitamin K antagonists further increases the vulnerability to SDH[8].

Although urinary incontinence may not seem directly related to CSDH, it is crucial to understand that CSDH can exert pressure on the brain[9]. This pressure can disrupt the brain's critical role in regulating bladder function, ultimately leading to urinary incontinence. While urinary incontinence is a symptom observed in various other medical conditions, its presence can serve as an indicator of a lesion within the cerebrum.

The management of urinary incontinence in patients with CSDH typically involves a comprehensive approach that addresses both the underlying haematoma and the associated neurological symptoms. Surgical drainage of the haematoma is a common intervention aimed at relieving pressure on the brain and alleviating neurological symptoms[10]. Furthermore, rehabilitation and physical therapy may be necessary to assist patients in regaining bladder control and restoring other functional abilities that may have been affected by the haematoma.

This case report presents a unique patient with delayed chronic SDH observed on brain computed tomography (CT), initially devoid of clinical abnormalities. The presented case is notable for its atypical presentation of a delayed CSDH in a young patient without predisposing factors, who did not report any deficits or alterations in the level of consciousness (ALOC) or fluctuations in the Glasgow Coma Scale (GCS) following the initial traumatic incident or in the subsequent years. This case highlights the importance of considering radiological imaging in all patients presenting with neurological symptoms, emphasising the necessity of prompt referral to further evaluate their condition.

The reporting of the following case adhered to the Surgical CAse REport (SCARE) guidelines[11].

HIGHLIGHTS

- A 23-year-old male presented with a delayed chronic subdural haematoma (CSDH) 3 years after a traumatic brain injury, challenging conventional understanding due to the absence of immediate neurological deficits.
- The patient exhibited symptoms uncommon for CSDH, including persistent headaches, urinary incontinence, memory impairment, and left-right disorientation without immediate paralysis or weakness.
- Surgical evacuation and excision of the haematoma were performed, leading to the successful resolution of symptoms and highlighting the importance of prompt intervention in atypical cases.
- The case challenged conventional understanding as it occurred in a young patient without known predisposing factors and underscored the need for considering delayed presentations even without immediate neurological deficits.

Case presentation

A 23-year-old male patient with no known comorbidities presented to our outpatient department (OPD) with a range of concerning symptoms. These included persistent headaches, left-right disorientation, acalculia (difficulty with arithmetic calculations), memory impairment, slurring of speech, and shameless urinary incontinence. These issues had been troubling him for the past month. According to the patient, he was in good health until three years ago when he was involved in a road traffic accident (RTA) due to a motorcycle slip. He was initially treated at a local hospital for a laceration on his left frontal skin. However, due to the unavailability of a CT scan at the time, no further imaging was performed. The patient was discharged home as he was clinically stable and did not exhibit any apparent injuries or fractures.

The patient's recent symptoms began with a diffuse and dull headache that was progressively worsening. Notably, this headache did not respond to over-the-counter painkillers and was aggravated by lying down and bending forward. Additionally, the headache was accompanied by memory impairment and shameless urinary incontinence. His previous medical history was insignificant.

Upon examination, the patient's vital signs and sub-vitals were found to be within normal limits. A mini-mental examination revealed that the patient was oriented to time, place, and person. However, he displayed signs of memory impairment, acalculia, left-right disorientation, and fluent but disordered speech. Other than these cerebral signs, there were no observable cranial nerve deficits, cerebellar signs, or motor and sensory abnormalities.

Initial baseline investigations, including a chest X-ray (CXR), complete blood count (CBC), urea and creatinine levels (UCEs), liver function tests (LFTs), prothrombin time (PT), activated partial thromboplastin time (APTT), and viral markers, all returned within normal ranges. The haematological and biochemical profile of the patient is shown in Table 1.

Table 1

Haematological and biochemical profile of the patient

Test	Result	Unit	Reference range
Blood urea nitrogen	9	mg/dl	6–20
Creatinine	1.1	mg/dl	Females: 0.6–1.1; Males: 0.9–1.3
Sodium	144	meq/l	136–146
Potassium	3.6	meq/l	3.5–5.1
Chloride	105	meq/l	98–106
Haemoglobin	12.8	gm/dl	Adult male 13-18; Adult female 11.5-16
RBC count	6.1	10^6 mil./µl	Adult male 4.5–5.8; Adult female 3.7–5.1; Cord blood 4.5
HCT	44.0	%	Cord blood 45–64; Birth 48–77; 1 weeks 41–64; 1 year 35- years 35–50; Adult male 45–58; Adult female 37–50
MCV	72.6	fl	Adult 76–96 fl
MCH	21.1	pg	Adult 28–32 Pg
MCHC	29.1	gm/dl	Adult 32–36 Gm/dl
Total leucocytes count	7.2	10^3/µl	Adult: 4.0–11.0/µl
Neutrophils	64	%	Adult 50–75%
Lymphocytes	23	%	Adult 20–50
Eosinophils	6	%	Adult 1–6
Monocytes	7	%	Adult 1–6
Basophils	0	%	Adult 0–1
Platelet count	238	10^3/µl	150–400/µl
RDW-SD	41.1	fl	—
RDW-CV	14.5	%	—

HCT, haematocrit; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; RBC, red blood cell; RDW-CV, red cell distribution width-coefficient of variation; RDW-SD, red cell distribution width-standard deviation.

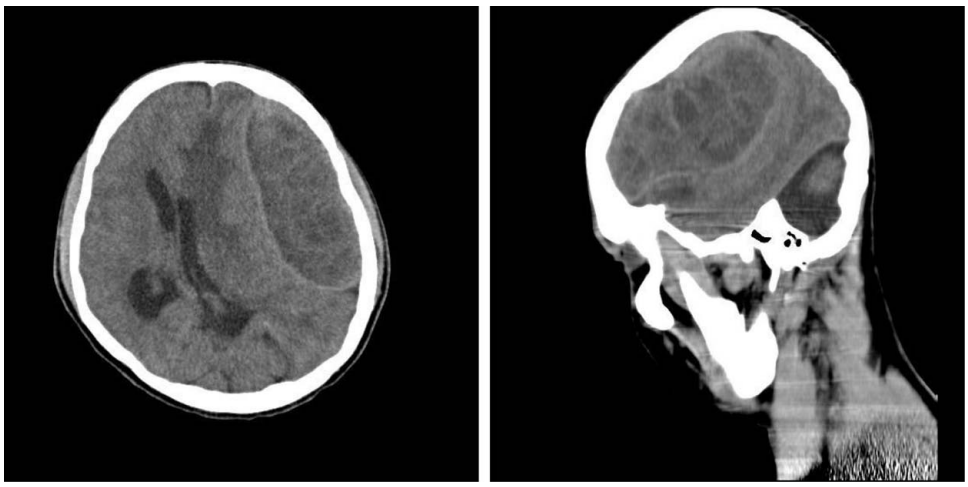

Figure 1. Computed tomography scan of brain—large heterogeneous mass lesion (9.7 × 4.6 × 6.5 cm) in left fronto-parietal lobes with hyperdense rim and internal septae, causing compression of adjacent brain. parenchyma, sulci, and gyri; effacement of left lateral ventricle; compensatory dilatation of right. lateral ventricle; significant sub-falcine herniation (1.8 cm); and bilateral cerebellar hemispheric hypoplasia.

A plain CT brain scan (Fig. 1) revealed a concerning finding: a large heterogeneous mass lesion with a hyperdense rim and internal septae in the left fronto-parietal lobe, causing mass effect and sub-falcine herniation. Subsequently, an MRI brain scan (Fig. 2) with contrast was conducted, which provided further insights. The MRI showed a large extraxial abnormal signal intensity area along the left fronto-parietal region, displaying heterogeneous hyperintense signals on T1-weighted images with a hypointense outer membrane and similarly heterogeneous hyperintense signals on T2-weighted/FLAIR images. Multiple internal hypointense septae and a hypointense outer membrane were also observed. Post-contrast sequences demonstrated enhancement of both the inner and outer membranes. Additionally, restricted diffusion was observed on DWI/ADC mapping, with the lesion measuring ~13.7 × 4.7 cm in anteroposterior (AP) and transverse (TR) dimensions and 6.1 cm in craniocaudal (CC) dimension.

The SWI sequence revealed areas of susceptibility adjacent to the dural surface and septae, as well as surrounding vasogenic oedema in the left frontal lobe. Susceptibility foci corresponding to contusions were also identified. Notably, the mass effect caused significant compression of adjacent brain parenchyma, leading to the effacement of sulci and gyri in the left front-parietal lobe, as well as the frontal horn and body of the left lateral ventricle. Additionally, there was significant sub-falcine herniation with a midline shift measuring ~1.9 cm towards the right side. Furthermore, the mass effect caused medial displacement of the left anterior cerebral artery, M4 segment of the left middle cerebral artery, and left posterior cerebral artery. Interestingly, there was asymmetry with hypoplasia of both cerebellar hemispheres, along with prominent vascular spaces identified at the right basal ganglia region. Magnetic resonance angiography (MRA) demonstrated patent vessels, and magnetic resonance venography (MRV) showed no filling defects within the sinuses.

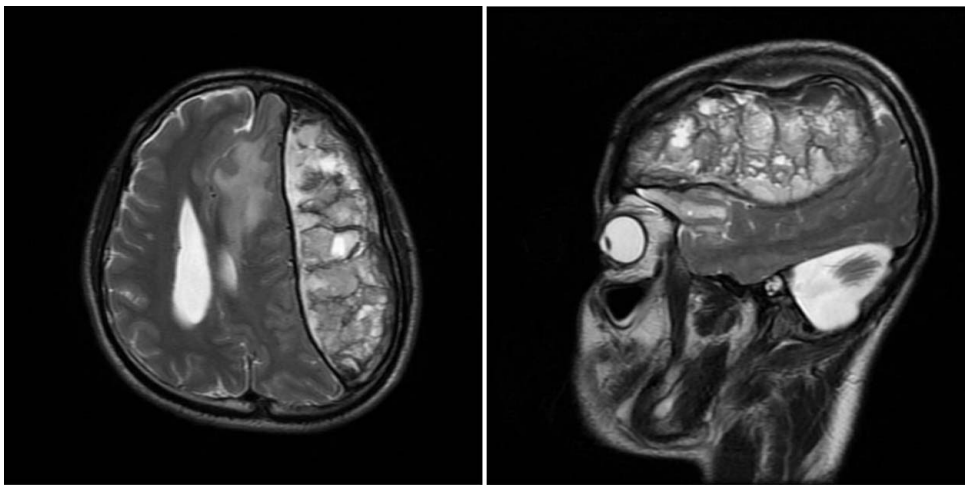

Figure 2. MRI scan of brain—chronic subdural haematoma, haemorrhagic contusions at left frontal lobe with surrounding vasogenic oedema, and absence of cerebral venous sinus filling defect on magnetic resonance venography.

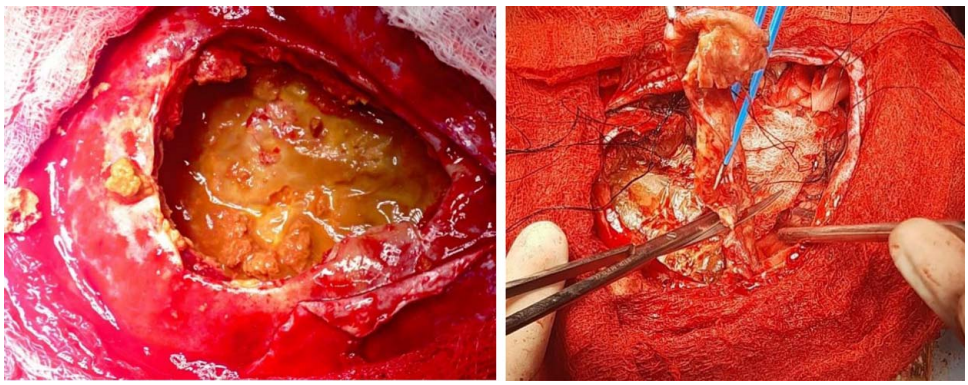

Figure 3. Intraoperative surgical site.

In the surgical procedure, a meticulous and complex approach was taken to address the brain lesion. A reverse question mark incision on the left side, guided by preoperative MRI findings, provided access to the targeted area. A fronto-temporo-parietal craniotomy was executed, allowing the surgical team to remove a portion of the skull to reach the brain. The dura mater, a protective membrane surrounding the brain, was opened in a C-shaped manner with the base oriented towards the middle fossa. Notably, the dura was found to be avascular, and devoid of blood vessels. Upon opening the lesion capsule, foul-smelling and dirty contents were found (Fig. 3). Biopsies and samples of these contents were meticulously collected for histopathology and culture analysis. The procedure also involved an internal debulking process, aided by the observation that the lesion was extra-axial and easily separated from the surrounding brain while maintaining the arachnoidal plane. A gross total resection was successfully achieved. To ensure a secure closure, the dura was meticulously closed in a water-tight manner, and the wound was closed in layers. Additionally, an autologous subdural (ASD) procedure was performed intraoperatively, indicating a comprehensive surgical approach aimed at addressing this complex brain lesion. The postoperative CT of the patient is shown in Figure 4.

The final biopsy and histopathological report were consistent with haematoma formation. Upon gross examination, the specimen consisted of a single dura-covered tissue piece measuring $5.5 \times 4 \times 1$ cm. The cut surface appeared light brown to dark brown and firm. Microscopic examination revealed a thick fibrous wall containing fibrin and haemorrhage. There was no evidence of significant inflammation, granulomata, or neoplastic processes. The diagnosis rendered is a posterior fossa SOL with features compatible with haematoma formation. No evidence of any neoplastic process was identified.

Discussion

Neurological deficits represent a hallmark of CSDH. Hemiparesis, characterised by weakness or paralysis on one side of the body, is a prevalent neurological deficit in CSDH, occurring in up to 58% of cases. Additional presentations encompass focal neurological deficits, weakness or paralysis in a single limb (monoplegia), seizures, altered mental status, or extra-pyramidal symptoms, including movement disorders. However, our case diverges from the typical pattern, as the patient, despite having a large CSDH, did not display any post-trauma paralysis or

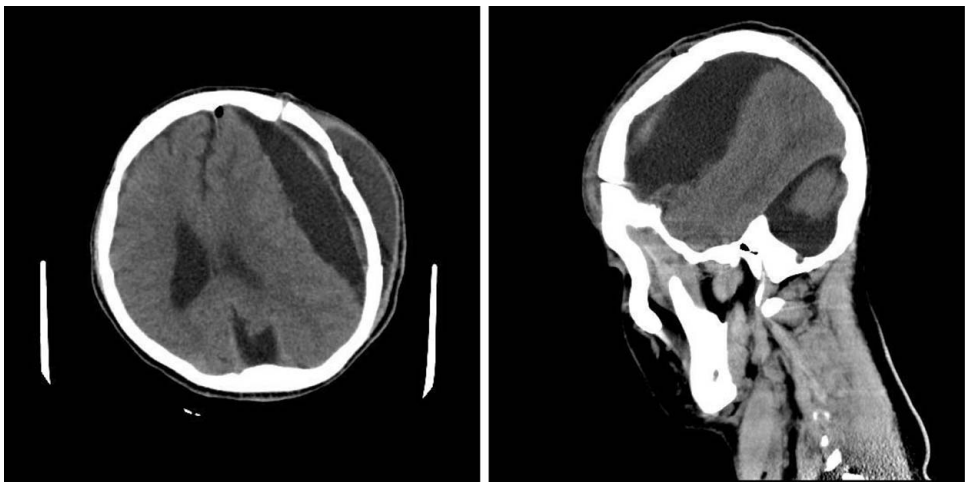

Figure 4. Postoperative computed tomography scan of brain—craniotomy defect with pneumocephalus and subgaleal collection (2.3 cm) in left fronto-parietal bone, crescentric hypodensity indicating subdural collection (2.3 cm) with compression effects on adjacent brain parenchyma, partial effacement of left lateral ventricle, and 1.4 cm midline shift towards the left side.

weakness, indicating an unusual absence of immediate neurological deficits. Symptoms only manifested after 3 years, persisting for a month, primarily as headaches, urinary incontinence, and left/right disorientation or slurring of speech, which are atypical for CSDH.

The mechanism underlying CSDH generally involves minor head trauma, causing bleeding from bridging veins within the brain into the meninges[12]. Over time, the accumulated blood partially liquefies and encapsulates, forming a chronic subdural haematoma. If left unevacuated, it can undergo calcification or ossification[13]. However, in this case, the absence of calcification or ossification after 3 years suggests that the haematoma did not result from immediate post-trauma, as calcification typically begins around 6 months, leading to calcified CSDH, occasionally progressing to ossification[13].

CSDH commonly affects elderly individuals, presenting with altered mental status, monoplegia, headache, and seizures[3]. In contrast, our case is unique, involving a 23-year-old patient without known comorbidities, no alcohol use, and no blood thinners or anti-platelet drugs. Factors such as advanced age, chronic alcohol abuse, and prior traumatic brain injury (TBI) predispose individuals to significant cerebral atrophy, heightening the risk of SDH from trivial head injury or whiplash without direct physical impact[3,5]. Most documented cases involve delayed traumatic intraparenchymal or extradural haematomas. However, the occurrence of delayed acute SDH or delayed chronic SDH in patients devoid of coagulation disorders or risk factors is infrequent and poorly understood. Notably, our patient developed urinary incontinence, suggesting that the voiding symptoms stemming from the subdural haematoma might have resulted from compression of the descending corticospinal tracts[14]. The haematoma's location might have led to compression of the area responsible for detrusor innervation.

The management of urinary incontinence in patients with CSDH typically involves a comprehensive approach that addresses both the underlying haematoma and the associated neurological symptoms. Surgical drainage of the haematoma is a common intervention aimed at relieving pressure on the brain and alleviating neurological symptoms[10]. Furthermore, rehabilitation and physical therapy may be necessary to assist patients in regaining bladder control and restoring other functional abilities that may have been affected by the haematoma.

When evaluating patients with neurological symptoms, it is crucial to recognise that not all cases will conform to the typical patterns or expectations. This particular case underscores the need for a high index of suspicion and thorough diagnostic investigations, even in the absence of immediate neurological deficits or significant clinical abnormalities.

Given the unique nature of this case, it is recommended that healthcare practitioners maintain a vigilant approach to patients presenting with neurological symptoms, regardless of their age or apparent risk factors. Radiological imaging, such as CT or MRI of the brain, should be considered as an essential diagnostic tool to identify potential underlying pathologies, including subdural haematomas. Early recognition and appropriate referral for imaging studies can help prevent delayed diagnoses and ensure timely interventions.

This case challenges conventional understanding, highlighting the need for a more comprehensive grasp of atypical presentations and underlying mechanisms of CSDH. A multidimensional evaluation considering various factors is essential for precise diagnosis and effective management in such uncommon cases.

Conclusion

CSDH is well-reported post-trauma and in the elderly but it's rare occurring in young patients without any comorbid, years after trauma without any prior neurological deficits and calcified changes. Seizures and paralysis are common presentations in CSDH patients but urinary incontinence and disruption of high cerebral function is an uncommon presentation. An initial CT scan is crucial in determining the aetiology as well as the management. Although this phenomenon is uncommon, emergency physicians must be vigilant for this possibility in the face of persistent or delayed post-traumatic symptoms even if an initial CT scan is normal.

Ethical approval

This study was approved by the ethics committee of the institution.

Consent

Written informed consent was obtained from the patient for the publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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None.

Author contribution

M.S.K., H.R., and A.N. were involved in the study concept, the collection of the data, drafting, literature review, data validation, supervision, and editing of the manuscript. H.S.N. and N.R. were responsible for the literature review and revising the manuscript for important intellectual content.

Conflicts of interest disclosure

There are no conflicts of interest.

Research registration unique identifying number (UIN)

This is not an original research project involving human participants in an interventional or observational study but a case report; this registration was not required.

Guarantor

Nahid Raufi.

Data availability statement

All data underlying the results are available as part of the article and no additional source data are required.

Provenance and peer review

Not commissioned, externally peer-reviewed.

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