CASE REPORT

Bilateral Post-Traumatic Hygromas in a Patient with Frontotemporal Dementia: A Case Report

Mweushi Mphande[1], Kabelele Sipalo[1], Clays Siwale[1], Gerald Musa[2,3], Keith Simfukwe[3], Elijah Katambo[1], Ali Ilunga[1], Kachinga Sichizya[1]

[1]*Department of Neurosurgery, University Teaching Hospital, Lusaka, Zambia.*
[2]*Department of Surgery, Neurosurgery Division, Livingstone University Teaching Hospital, Livingstone, Zambia.*
[3]*Department of Neurological Diseases and Neurosurgery, Peoples Friendship University of Russia (RUDN), Moscow, Russia*
[4]*Department of Surgery, Neurosurgery Division, Maina Soko Military Medical Hospital, Lusaka, Zambia*

ABSTRACT

Background: Frontotemporal dementia (FTD) is a neurodegenerative disorder characterized by behavioural, speech, and movement disturbances, typically affecting individuals with a mean age of 58 years. We present a rare case of FTD in a patient with Alzheimer's disease who experienced worsening symptoms due to bilateral subdural hygromas.

Case Description: A 50-year-old male with a history of head trauma six weeks prior presented with an inability to speak or walk. He had a five-year history of progressive frontal and temporal lobe syndromes and a convulsive disorder with generalized tonic-clonic seizures. Previously diagnosed with Alzheimer's disease and managed at a psychiatric institution, he had been lost to follow-up for two years. Brain computed tomography (CT) revealed a "knife-blade" appearance of cortical atrophy, bilateral subdural hygromas with mass effect, ventricular enlargement without periventricular lucency, and preserved gyral-sulcal differentiation. The patient underwent bilateral burr hole drainage of the subdural collections. Due to resource limitations, controlled drainage was not feasible. However, seven days postoperatively, he showed significant improvement in Glasgow Coma Scale and motor function. Follow-up magnetic resonance imaging (MRI) one month later demonstrated complete brain expansion.

Conclusion: FTD is characterized by progressive cortical atrophy, leading to an increased subarachnoid space, making patients susceptible to subdural hygromas even after minor head trauma. In Alzheimer's patients, structural causes of worsening dementia symptoms (e.g., subdural collections) should always be excluded through imaging.

INTRODUCTION

Frontotemporal dementia (FTD) refers to a group of neurodegenerative disorders affecting the frontal and temporal lobes.[1,2] Brain atrophy is a hallmark of FTD, with affected regions correlating with the patient's clinical symptoms.[3] Patients with FTD may

Corresponding author:
Gerald Musa,
Email: gerryMD@outlook.com
ORCID: 0000-0001-8710-8652

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develop aphasia, seizures, and significant changes in personality and social interactions.[4] FTD typically affects individuals between 40 and 60 years of age but is often misdiagnosed as Alzheimer's disease (AD), which usually presents later in life.[2,5,6] FTD accounts for 10% to 20% of dementia cases[4] and follows an aggressive disease course, with an average life expectancy of less than 10 years from symptom onset.[7] Progressive motor decline often leads to bedridden complications, such as pneumonia, which further worsen prognosis. Neuroimaging typically reveals cortical atrophy and hippocampal aplasia, predominantly affecting the anterior segment, in contrast to the generalized hippocampal atrophy seen in Alzheimer's disease. The marked cerebral atrophy seen in FTD reduces brain volume and tensile support within the cranial vault, predisposing patients to the development of subdural collections such as hygromas and hematomas, even with minor or unnoticed trauma.[5,7,9] These secondary pathologies can exacerbate existing neurological deficits, leading to a rapid and sometimes misleading deterioration in cognitive and motor function.[3,9,10]

We present a unique case of an adult patient previously diagnosed with Alzheimer's disease who developed clinical features suggestive of frontotemporal dementia, further complicated by bilateral subdural hygromas, leading to a worsening neurological decline.

CASE DESCRIPTION

A 50-year-old male farmer with no history of chronic illness or psychiatric disorder began to exhibit behavioural and cognitive changes over a period of five years. Initially, he developed apathy, impulsivity, and lost interest in work that he had previously taken pride in. Over time, he became increasingly withdrawn and had difficulty with memory and communication. These symptoms progressively worsened, particularly after he experienced an episode of transient loss of consciousness. He was no longer able to care for himself, which led to separation from his family and eventual relocation to live with his brother.

Approximately two years prior to his presentation at our facility, the patient was found wandering aimlessly and was admitted to a psychiatric hospital. A diagnosis of Alzheimer's disease was made based on his disorientation, memory impairment, and altered behaviour. He remained institutionalized for nearly a year. One year before presentation, he sustained a moderate head injury after being hit by a motor vehicle while walking. Following this incident, he experienced seizures and progressive limb weakness. His family, now reengaged in his care, noted further decline and sought medical attention, leading to his referral to our hospital.

Upon admission, the patient appeared chronically ill and emaciated. His Glasgow Coma Scale (GCS) was 11/15 (E3, V2, M6). He exhibited global aphasia and quadriparesis, with the left side more severely affected: motor power was 4/5 in the right upper limb, 1/5 in the left upper limb, 2/5 in the right lower limb, and 1/5 in the left lower limb. Pupillary responses were symmetrical and reactive to light. He had no signs of respiratory distress, jaundice, or cyanosis. Due to his impaired swallowing, a nasogastric tube was inserted for feeding.

A non-contrast CT scan of the brain revealed bilateral subdural collections, more prominent on the right side, causing significant compression of severely atrophic cerebral hemispheres. The scan also demonstrated sulcal effacement, ventricular enlargement, and a "knife-blade" appearance of the gyri, all suggestive of advanced frontotemporal atrophy (Figure 1).

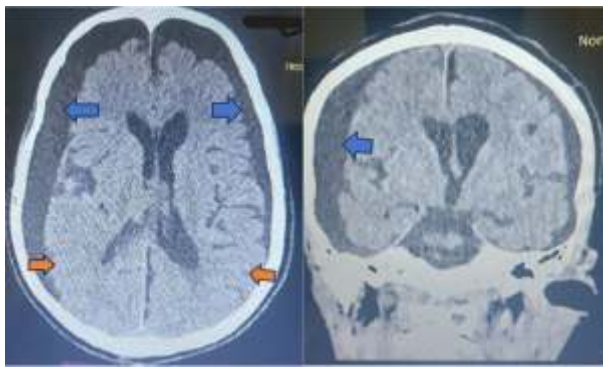

Figure 1: Brain CT at admission showing bilateral subdural collections (blue arrows), right more than left, with severe cortical atrophy and effacement of gyri

Surgical management was initiated, and two 1.5 – 2cm burr-holes were made bilaterally in the frontal and parietal regions. On opening the dura, clear subdural fluid under relatively high pressure was encountered, and samples were collected for analysis. There was no evidence of hematoma intraoperatively. Subdural drains were placed bilaterally (Figure 2). Due to the absence of pressure-controlled drains, ordinary 5mm drains were inserted and collection bags were placed level with the head to avoid rapid over-drainage on day 1 and lowered on day 2.

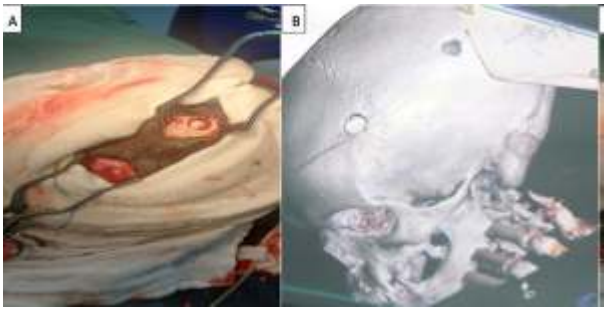

Figure 2: Intraoperative and postoperative images showing burr-hole placements used for hygroma drainage.

Postoperatively, the patient showed steady improvement. By the second day after surgery, the drains had become inactive and were removed. He tolerated the procedure well and showed progressive neurological recovery without any immediate complications. Eight days later, he was discharged with a GCS of 12/15 (E4, V2, M6). At that time, motor strength had improved to 4/5 in the right upper limb, 2/5 in the left upper limb, 3/5 in the right lower limb, and 2/5 in the left lower limb. He was able to take soft, ground food orally. A repeat CT scan prior to discharge demonstrated near-complete resolution of the left-sided subdural collection, residual collection on the right, and signs of partial brain re-expansion (Figure 3).

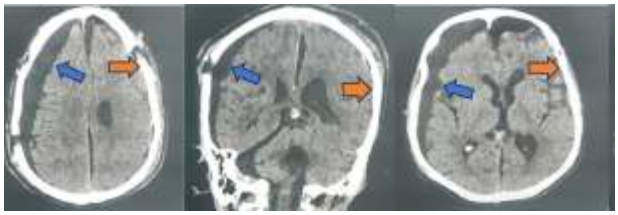

Figure 3: Postoperative CT scan showing reduced subdural collection on the left (orange arrow) with persistent right-sided hygroma (blue arrow), and improved sulcal visibility.

At the three-week follow-up, his condition had improved further. He was alert with a GCS of 14/15 (E4, V4, M6), showed better responsiveness, and had improved motor function: 4/5 in the right upper limb, 3/5 in the left upper limb, 4/5 in the right lower limb, and 3/5 in the left lower limb. However, apathy and expressive aphasia persisted. An MRI performed at this visit revealed complete resolution of the subdural collections. It also demonstrated bilateral frontal and temporal lobe atrophy, ventricular enlargement, and frontal hippocampal aplasia—findings that were highly consistent with frontotemporal dementia (Figure 4).

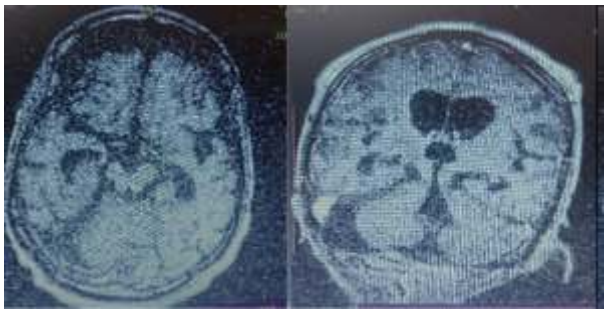

Figure 4: MRI at 3-week follow-up showing total resolution of subdural collections with marked bilateral frontal and temporal lobe atrophy and ventricular dilation.

Based on his clinical presentation and radiological findings, the initial diagnosis of Alzheimer's disease was revised to behavioural variant frontotemporal dementia (bvFTD), which was likely responsible for his early behavioural changes and progressive cognitive decline (**Table 1**). It should be noted that

no brain imaging had been performed for the 5 years prior. This was attributed to several factors including limited access to advanced imaging like CT and MRI and high associated costs leading to delay in diagnosis and timely management. The subdural collections were interpreted as secondary hygromas, likely exacerbated by his head injury and underlying brain atrophy. Spontaneous intracranial hypotension was considered but deemed unlikely due to the absence of orthostatic headache, diffuse meningeal enhancement, or spinal CSF leak. The subdural fluid analysis revealed normal albumin levels, further excluding the possibility of a resolving hematoma (Virchow's hygroma).

Table: Timeline of Disease Progression, Imaging, Intervention, and Follow-up

Timeline	Clinical Event / Progression	Imaging Findings	Intervention	Follow-Up / Outcome
~5 years before admission	Onset of behavioural and cognitive changes: apathy, impulsivity, social withdrawal, memory, and communication difficulties	None	None	Gradual decline in function; patient unable to care for self
~2 years before admission	Found wandering; admitted to psychiatric hospital; diagnosed with Alzheimer's disease	None	Institutional psychiatric care	Hospitalized for ~1 year
~1 year before admission	Sustained moderate head injury; developed seizures and progressive limb weakness	None	None	Continued decline in neurological function
At presentation	Chronically ill, emaciated; GCS 11/15; global aphasia; quadriparesis (L>R); required nasogastric feeding	CT Brain: Bilateral subdural collections (R > L), severe atrophy, sulcal effacement, ventricular enlargement	Bilateral burr-hole drainage; subdural drains inserted	Early postoperative improvement noted
Post-op Day 2	Better consciousness; initial motor recovery	Drains became inactive	Subdural drains removed	Continued neurological improvement

Timeline	Clinical Event / Progression	Imaging Findings	Intervention	Follow-Up / Outcome
Post-op Day 8 (Discharge)	GCS 12/15; oral intake resumed; improved motor power (best 4/5)	CT Brain: Resolution of left-sided collection, residual right hygroma, partial brain re-expansion	Discharged home	No complications; ongoing recovery
3-week follow-up	GCS 14/15; further motor improvement; apathy and expressive aphasia persisted	MRI Brain: Resolved hygromas; bilateral frontal and temporal atrophy; frontal hippocampal aplasia	Diagnosis revised to behavioural variant frontotemporal dementia (bvFTD); supportive care plan initiated	Continued follow-up for neurodegenerative progression

DISCUSSION

Frontotemporal dementia (FTD) is a progressive neurodegenerative condition marked by selective atrophy of the frontal and temporal lobes, resulting in profound behavioural, cognitive, and language impairments.[2,3,5] It is a leading cause of early-onset dementia, often affecting individuals between the ages of 45 and 65 years. Neuroimaging findings characteristically reveal anterior-predominant cortical atrophy, which correlates with the patient's clinical presentation and helps differentiate FTD from Alzheimer's disease (AD), where hippocampal and parietal atrophy are more typical.[5,6,7]

In addition to its cognitive and behavioural manifestations, the cerebral atrophy seen in FTD has structural implications that are often underrecognized.[2] Severe cortical volume loss reduces intracranial support and increases the subdural space, predisposing to the development of subdural hygromas or hematomas—even in the absence of overt trauma.[3,6,7,11] The fragility of stretched bridging veins in an atrophied brain, combined with even minor mechanical forces, can lead to cerebrospinal fluid (CSF) leakage into the subdural space.[8] These secondary complications may present as insidious or acute neurological deterioration and can be misattributed to progression of the underlying neurodegenerative disease.[7]

Our case underscores this important, and often overlooked, clinical intersection. The patient had initially been diagnosed with AD but developed prominent behavioural and language disturbances, consistent with a frontotemporal pattern of disease. Notably, imaging revealed bilateral subdural hygromas superimposed on significant cortical atrophy. The collections were exerting mass effect, particularly in the right hemisphere, contributing to neurological decline including seizures, quadriparesis, and decreased level of consciousness.

Surgical evacuation via burr holes resulted in neurological improvement, with gradual motor recovery and cognitive stabilization.[7,12] This suggests that, in patients with underlying FTD, subdural collections may act as reversible contributors to worsening symptoms, and should be actively investigated—particularly in the context of

abrupt clinical deterioration.[4,8] However, drainage must be performed cautiously. Rapid drainage of subdural collections in patients with marked cerebral atrophy carries significant risks due to the widened subdural space and increased vulnerability of bridging veins.[13] Abrupt decompression can result in brain shift, leading to tearing of these veins and potentially causing acute subdural hematoma, intracerebral haemorrhage, or even transtentorial herniation. In atrophied brains, where cerebral re-expansion is often delayed or incomplete, this risk is further amplified.[14,15] Therefore, a gradual and controlled evacuation of the collection is essential. Positioning drainage systems at the level of the head during the initial postoperative period helps to mitigate rapid pressure differentials. Importantly, the use of pressure-controlled drainage systems is recommended, as they provide a safer, regulated outflow of subdural fluid, minimizing the likelihood of complications associated with over-drainage.[14,16]

MRI follow-up confirmed symmetrical frontotemporal atrophy, supporting the diagnosis of FTD and ruling out other causes such as spontaneous intracranial hypotension (SIH), which typically presents with diffuse pachymeningeal enhancement and CSF hypovolemia—features absent in our patient.

Few cases in the literature have described the coexistence of FTD and subdural hygromas.[7] The rarity may reflect underdiagnosis or misattribution of symptoms to the natural course of FTD. Given the increased intracranial compliance in atrophied brains, sudden decompression following hygroma drainage should be approached with caution. Controlled drainage, ideally using valved systems, may help mitigate risks of hyperperfusion injury and re-expansion complications.[12]

Our case illustrates a critical diagnostic lesson: In patients with FTD—particularly those with advanced atrophy—new-onset neurological deficits should prompt neuroimaging to exclude subdural collections. While FTD itself follows a relentlessly progressive course, coexisting hygromas represent a potentially treatable cause of neurological decline. Early identification and appropriate intervention can result in significant functional recovery and improved quality of life, even in a neurodegenerative context.[10, 17]

Unfortunately, patient perspective of the management and recovery process was not recorded.

CONCLUSION

This case highlights that the profound cortical atrophy seen in FTD not only drives its characteristic clinical manifestations but also predisposes to the development of subdural hygromas, which may mimic or aggravate disease progression. In any FTD patient presenting with sudden or atypical neurological worsening, subdural collections should be actively excluded through imaging. Recognizing and managing this potentially reversible complication is essential in the comprehensive care of patients with frontotemporal dementia.

Key Takeaways

1. Severe cortical atrophy in FTD increases the risk of developing subdural hygromas, even after minor or unrecognized trauma, due to loss of intracranial support and bridging vein vulnerability.
2. Neurological deterioration in patients with presumed Alzheimer's or FTD should prompt imaging to exclude reversible causes such as subdural hygromas, which can mimic disease progression.
3. Timely surgical evacuation of symptomatic hygromas in FTD patients can lead to significant functional improvement, emphasizing the importance of considering structural complications in dementia care.

Declarations

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Informed consent: Informed consent was obtained from the caregiver.

Ethical approval was not sought as all the procedures were part of the standard patient care and no identifying information was revealed.

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