Health Reserch's Journal Volume 03 Number 02 April 2026

# A CASE REPORT OF 41-YEAR-OLD MALE WITH POST TRAUMATIC ENCEPHALOMALACIA

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## ABSTRACT

Encephalomalacia is the softening of brain tissue resulting from hemorrhagic contusions, infarctions, or diffuse axonal injury, which can develop months or even years after head trauma. Encephalomalacia may occur in adults, albeit rarely, following acquired brain injuries such as infection, trauma, or infarction. We report a 41-year-old male who presented with headache and memory loss, symptoms that developed one year after a motorcycle accident in which he sustained a head injury. Imaging revealed hypodense lesions in the left temporoparietal lobe, consistent with encephalomalacia and associated ventriculomegaly. This case highlights the significance of detailed clinical and radiological assessments in diagnosing encephalomalacia, underscoring the importance of early intervention and management to prevent long-term neurological complications.

## KEYWORDS

Encephalomalacia, Brain injuries, Craniocerebral trauma, Headache, Patient

## INTRODUCTION

The brain is the most essential and intricate organ, and encephalomalacia is the "softening of the brain." This condition occurs when brain tissue softens due to inflammation or bleeding caused by factors such as cerebral infarction, ischemia, infection, head trauma, or other injuries. Encephalomalacia is a type of brain injury linked to infections and is also a chronic condition that follows brain trauma. It is classified as a traumatic brain injury based on imaging findings. This condition can affect individuals of any age, including neonates and adults. Encephalomalacia is the loss of brain tissue in any area of the cerebral parenchyma, accompanied by adjacent gliosis (Ghandi et al., 2024).

Although research on encephalomalacia in humans is limited, encephalomalacia in adults can occur due to acquired brain injuries such as trauma, infection, or infarction. The symptoms and prognosis vary depending on factors such as the size, location, and number of lesions, as well as the presence of complications such as seizures, hydrocephalus, or infections (Harjpal and Quershi, 2023).

Encephalomalacia most commonly occurs as a result of hypoxia, resulting in irreversible neurological damage. Although hypoxic-ischemic brain injury is the most common cause of encephalomalacia, it can also occur due to other factors such as viral encephalitis, head trauma, or severe congenital metabolic disorders. In adults, cystic encephalomalacia can develop after acquired brain injury, including infection, trauma, or infarction (Chang et al., 2022).

## MATERIALS AND METHODS

This study applies a descriptive case report approach. The clinical evaluation began with anamnesis to explore the patient's main complaint of progressive headache, followed by detailed physical and neurological examinations. Further assessment was conducted using a head CT scan. The clinical and radiological data obtained were subsequently reviewed in relation to current literature, with the aim of determining the consistency of the case with existing evidence and clarifying its prognostic and recurrence implications.

## RESULTS

A 41-year-old male presented with a complaint of headache. Initially, he had a history of motorcycle accidents and hitting his head one year ago. The patient's family complained that the patient had difficulty remembering conversations and commands and had lost the ability to read and write. He denied experiencing seizures and motoric weakness. He didn't maintain regular communication with his family and the examiner, showing signs of cognitive or neurological impairment.

GCS 14 (E4M6V4), blood pressure 113/76 mmHg, heart rate 86x/minute, respiratory rate 20x/minute, temperature 36.5'c, and blood oxygen saturation 99%. The general physical examination showed difficulties with short-term and long-term memory. The patient couldn't remember his past meal or how to read and write. Laboratory examination of the blood revealed an elevated ALT.

Non-contrast head CT revealed a hypodense lesion in the left temporoparietal lobe, with a density of 8 HU, clear boundaries, and regular edges (figure 1a, 1b, and 1c)—no hyperdense lesion in the intracerebral or intracerebellar. There is visible widening of the temporal horn and posterior lateral ventricle (figure 1b). Midline in the middle, no deviation. Thickening of the bilateral maxillary sinuses, ethmoid, and sphenoid walls. Bulbus oculi and retrobulbar structures are within normal limits. The visualized bone system is intact. These findings are consistent with an encephalomalacia in the temporoparietal lobe, causing ventriculomegaly of the temporal horn and posterior lateral ventricle.

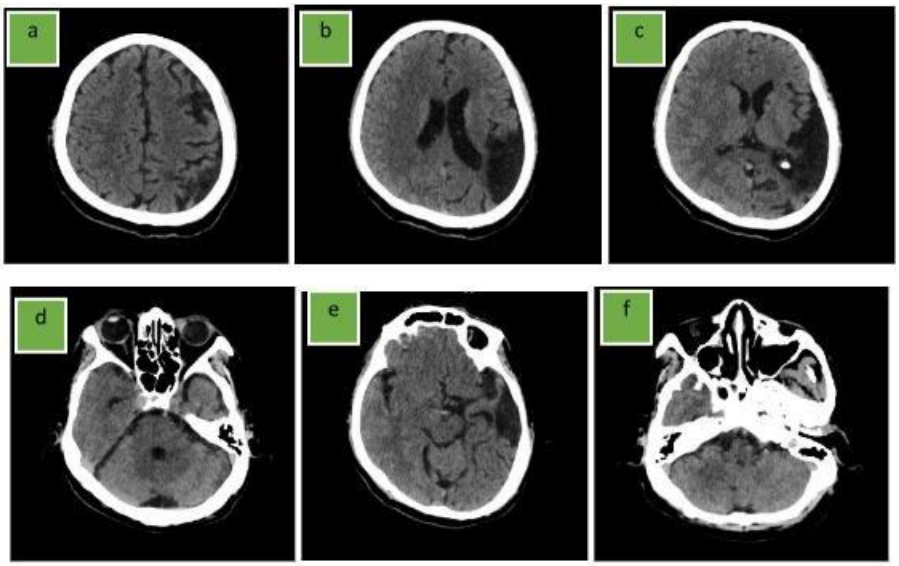


Figure 1. Head CT scan. a) Hypodense lesion in the left temporoparietal lobe; b) Hypodense areas in the parietal region with ventricular enlargement; c) Hypodense lesions near the ventricles; d) Temporal lobe hypodensity, e) Hypodense lesion; f) Thickening of the sinus wall. .

## DISCUSSION

This case involves a 41-year-old male who presented with a headache and memory loss, symptoms that developed one year after a head trauma. The patient did not seek medical attention immediately following the injury and only underwent a CT scan later when visiting the neurosurgery polyclinic. He was subsequently diagnosed with encephalomalacia. Encephalomalacia is the softening of brain tissue caused by hemorrhagic contusions, infarctions, or diffuse axonal injury, and it can develop months or even years following head trauma (Carmody, 2013). Encephalomalacia is rarely encountered in clinical practice. It is considered a rare case presentation due to its infrequent occurrence and is not typically seen in most patients. This condition, which involves the softening of brain tissue, tends to manifest in specific and less common circumstances, making its presentation quite unusual in most medical settings (Ghandi et al., 2024).

Encephalomalacia can affect individuals of any age group, from neonates to adults. It is characterized by the loss of cerebral parenchyma in any brain area, accompanied by adjacent gliosis, a reactive process involving glial cells. This condition can occur in various brain regions, depending on

the underlying cause, and is marked by the degeneration of brain tissue in the affected areas (Ghandi et al., 2024).

While there are limited published studies on encephalomalacia in humans, most focus primarily on newborns and children, with very few addressing adult cases. Adult cases are much rarer and are less commonly documented in the available literature (Harjpal and Quershi, 2023).

Encephalomalacia is primarily caused by hypoxic-ischemic brain injury, though it can also arise from factors like viral encephalitis, head trauma, or severe congenital metabolic disorders. In adults, while rare, cystic encephalomalacia can occur due to acquired parenchymal brain injury. This could include injuries from infections, trauma, or infarctions, which can damage and soften the brain tissue over time. Specific data on the percentage of encephalomalacia cases caused by head trauma is currently unavailable in medical literature (Chang et al., 2022).

Over time, hemorrhagic contusion and necrosis areas are gradually reabsorbed systematically, starting from the periphery and moving inward (Dogrul et al., 2020). Within the first day after the injury, neutrophils infiltrate the affected area. By day three, macrophages start clearing the necrotic tissue. Around one week after the injury, reactive astrocytes form around the damaged tissue, and there is an increase in capillary density. By one-month post-injury, the affected areas become highly cellular due to microglial and astrocytosis hyperplasia (Lawrence et al., 2023).

After 6 to 12 months, a contracted cystic cavity remains, surrounded by astrocytes and macrophages containing hemosiderin. The cortical gyri in the affected region undergo atrophy while adjacent cerebrospinal fluid (CSF) spaces expand. Additionally, compensatory atrophic enlargement of the nearby ventricle is commonly observed. In cases of severe traumatic brain injury, late-stage manifestations may include widespread degeneration of the subcortical white matter due to diffuse axonal injury. By approximately three months post-injury, Wallerian degeneration can result in secondary atrophy of the thalamus and brainstem, especially in extensive diffuse axonal injury (Carmody, 2013).

The size, location, and number of lesions influence the symptoms and prognosis of encephalomalacia. Other conditions, like seizures, hydrocephalus, or infection, can significantly affect the clinical outcome. Larger lesions, or those located in critical areas of the brain, may lead to more severe symptoms while developing secondary complications can further complicate the prognosis (Harjpal and Quershi, 2023).

This patient experienced intense, stabbing headaches and was unable to engage in conversations with family members. During a memory test, the patient struggled to recall basic information, such as what he had eaten earlier and even had difficulty remembering how to read and write. According to the patient's CT scan, the lesion was in the temporal-parietal lobe. The hippocampus and the temporal system, responsible for memory storage, are situated in this region. The hippocampus is located deep within the medial temporal lobe and plays a crucial role in forming and retrieving memories (Abuhasan et al., 2025).

The hippocampus plays a crucial role in the formation of declarative memories, which are memories that can be consciously retrieved and articulated. Declarative memory is categorized into two types: episodic and semantic. Episodic memory refers to recalling specific past events, including details about when and where they occurred. For instance, "I remember eating dinner at my friend's house last weekend." On the other hand, semantic memory involves recalling general knowledge and factual information about the world, such as "The Earth orbits the Sun" or "Water boils at 100 degrees Celsius." Research suggests that the hippocampus is crucial for episodic memory, whereas the medial temporal lobe plays a more significant role in semantic memory (Patel et al., 2025).

The location of the lesion in this patient provides a clear explanation for the memory loss he experienced. Since the lesion was situated in a brain region involved in memory processing, it directly impacted the patient's ability to recall information (Sandi, 2007). The specific area affected, likely within the temporal or parietal lobes, plays a crucial role in storing and retrieving short-term and longterm memories. Damage to this part of the brain can disrupt the neural circuits responsible for these functions, leading to difficulties in forming, recalling, or retaining memories. Therefore, the patient's memory loss can be understood as a direct consequence of the lesion's location and interference with the brain's memory systems (Duff et al., 2020).

Treatment for encephalomalacia typically focuses on managing symptoms and improving the patient's overall function through various therapeutic approaches (Ghandi et al., 2024). Medications

such as anti-inflammatory drugs help reduce inflammation in the brain, while neuroprotective agents may be used to prevent further neuronal damage and support healing. Anticonvulsants are commonly prescribed to control seizures, which can occur in patients with brain injuries (Sahid et al., 2023). Rehabilitation therapy also plays a critical role in the recovery process. Physical, occupational, and speech therapy are essential for enhancing functional outcomes, helping patients regain lost motor skills, improve speech, and boost cognitive function, ultimately contributing to a better quality of life and greater independence (Pekdemir et al., 2024).

In some instances, when surgical complications cause encephalomalacia or when other treatments prove ineffective, surgical intervention may be necessary. Procedures such as decompressive craniectomy may be performed to relieve increased intracranial pressure, and revascularization surgery may be used to restore blood flow to the affected areas of the brain, aiding in tissue repair and preventing further damage (Sahuquillo and Dennis, 2019). In addition to medical and surgical interventions, supportive care is crucial. A well-balanced diet is vital for promoting brain health and ensuring the body has the nutrients needed for healing. Psychological support, including counseling or therapy, is also essential to help patients cope with emotional and behavioral changes that may occur after brain injury. This support can assist patients and their families manage challenges related to memory loss, personality changes, and other cognitive impairments (Puri et al., 2023).

If left untreated, encephalomalacia can result in severe complications that significantly impact the patient's quality of life. Persistent neurological deficits, such as motor or sensory loss, may lead to long-term disability. The condition also increases the risk of seizures, which can further impair brain function and quality of life (Azouvi et al., 2009). Cognitive decline is another potential outcome, as damage to memory-related brain regions may result in memory, attention, and problem-solving difficulties. Behavioral changes, including personality shifts or emotional instability, may complicate the patient's social and personal interactions. Additionally, the development of secondary conditions such as hydrocephalus, where fluid accumulates in the brain, can occur, further exacerbating neurological impairment and requiring additional medical intervention. These complications highlight the importance of early diagnosis and treatment to prevent further deterioration (Fan et al., 2024).

The patient was prescribed a combination of citicoline, folic acid, paracetamol, a muscle relaxant, and acetazolamide. Citicoline is often used to support brain function and promote neural recovery, while folic acid aids in cellular repair and overall health. Paracetamol was given for pain management, and the muscle relaxant may have been prescribed to help alleviate any spasticity or discomfort. Acetazolamide, a diuretic, is often used to reduce intracranial pressure by promoting fluid balance (Jasielski et al., 2020).

The CT scan revealed enlargement of the temporal horn and posterior lateral ventricle, suggesting the presence of increased cerebrospinal fluid (CSF) in these regions. This finding raised concern about potential hydrocephalus, a condition where CSF accumulates in the brain. Due to this, a neurosurgical intervention was planned, which would be considered if there is a further increase in CSF accumulation, as this could require procedures like a ventriculoperitoneal shunt to relieve pressure and prevent further brain damage.

## CONCLUSIONS

This case illustrates the long-term impact of encephalomalacia following traumatic brain injury. A 41-year-old male developed severe memory loss and headaches one year after a motorcycle accident. His condition, diagnosed as encephalomalacia, resulted from brain tissue softening due to hemorrhagic contusion, infarction, or diffuse axonal injury. Though more common in neonates, encephalomalacia is rare in adults and often linked to trauma, infection, or infarction. The patient's symptoms were associated with damage to the temporal-parietal lobe, particularly the hippocampus, which is crucial for memory processing. CT imaging showed hypodense lesions and ventriculomegaly, raising concerns about hydrocephalus. Management included medication (citicoline, folic acid, paracetamol, muscle relaxant, and acetazolamide) to support neural recovery, reduce intracranial pressure, and rehabilitation therapy. Close monitoring for hydrocephalus was necessary, with possible neurosurgical intervention if symptoms worsened. Encephalomalacia can cause severe neurological deficits, cognitive decline, and behavioral changes, highlighting the importance of early diagnosis and intervention to prevent complications and improve patient outcomes.

**Acknowledgement**

The authors thank the Neurosurgery Department teams and the patient's family.

**Funding Source**

This study was fully financed by the reaserchers themselves, with no external funding or sponsorship involved. All expenses related to carrying out the research and preparing the manuscript were covered independently by the researchers, including costs for data collection, analysis, and report writing.

**Conflict of Interest**

The authors declare no conflict of interest in this report

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