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"Chemical meningitis emerged as a consequence of the epidermoid cyst excision in the left medial temporal region – A Case report

Dr. Dharmikkumar Kamleshkumar Velani (M.B.B.S., M.S.(General Surgery), M.CH. Neurosurgery, 1 Year Resident, Department of Neurosurgery, Smt. NHL Municipal Medical College & S.V.P. Hospital, Ahmedabad, Gujrat, India.)

Dr Jaimin Rajeshkumar Modh (M.B.B.S., M.S. (General Surgery), M.Ch Neurosurgery, 2 Year Resident, Department of Neurosurgery, Smt. NHL Municipal Medical College & S.V.P. Hospital, Ahmedabad, Gujrat, India.)

Dr Arvind Verma (M.B.B.S., M.S. (General Surgery), M.Ch Neurosurgery, 2 Year Resident, Department of Neurosurgery, Smt. NHL Municipal Medical College & S.V.P. Hospital, Ahmedabad, Gujrat, India.)

Dr Krushi Soladhra (M.B.B.S., M.S.(General Surgery), M.CH. Neurosurgery, 1 Year Resident, Department of Neurosurgery, Smt. NHL Municipal Medical College & S.V.P. Hospital, Ahmedabad, Gujrat, India.)

ABSTRACT:

Chemical meningitis, a subtype of aseptic meningitis, as a complication of large left medial temporal epidermoid cyst excision is not a rare complication. Aseptic meningitis is characterized by noninfective serous inflammation of the meninges. It can occurs when associated with epidermoid cysts due to rupture of cyst contents into subarachnoid space resulting in aseptic chemical meningitis. Chemical meningitis is believed to occur due to an exaggerated inflammatory reaction, characterized by the release of several cytokines such as interleukin-10 (IL-10), interleukin-1β (IL-1β), and tumor necrosis factor-α (TNF-α). This response is triggered by the presence of keratin debris released into the subarachnoid spaces during the surgical extraction of epidermoid cyst contents. Consequently, these inflammatory processes can lead to the development of meningitis symptoms and even hydrocephalus. Here We present a case of a 45-year-old female with a Chemical meningitis following left medial temporal epidermoid cyst excision . This uncommon diagnosis was further complicated by the rare occurrence of chemical meningitis as a post-operative complication.This review article conducts an in-depth analysis of the available research on intracranial epidermoid cysts, with a particular focus on exploring their imaging characteristics and the potential surgical complications that may arise. Additionally, we offer a comprehensive overview of the present understanding of the biochemical mechanisms responsible for the infrequent occurrence of chemical meningitis following surgical intervention.

Keywords: Aseptic meningitis, Chemical meningitis, Epidermoid cyst, Neurosurgery

INTRODUCTION

Epidermoid cysts are benign lesions that occur due to a disorder in neural tube closure during development, in which epithelial remnants are trapped inside neural tissue. These tumors are lined with a capsule made of keratinized squamous epithelium and have a central core of keratin, desquamated cells, water, and cholesterol. The cysts grow due to a continual process of desquamation of epithelium and account for approximately 0.2–1.8% of all intracranial tumors, making cysts in the left medial temporal region is relatively rare.Complications of epidermoid cyst excision in left medial temporal region can include the manifestation of meningitis, hydrocephalus, cerebrospinal fluid (CSF) leakage which can indicate any or all the above. Chemical meningitis is a form of aseptic meningitis, not caused by a virus, and presents with similar symptoms as infectious meningitis, but is differentiated by a negative spinal fluid Gram stain, negative results of spinal fluid cultures, and failure of antibiotics to improve the patient's condition. Chemical meningitis as a complication of epidermoid cyst excision in left medial temporal region surgery is not a rare complication; however, development of such a severely protracted course following the surgical removal of a epidermoid cyst excision in left medial temporal region has not been described. Here, we present a patient case following the resection of a large left medial temporal region epidermoid cyst and course of aseptic meningitis .that was further complicated by wound revision, a prolonged comatose state, and a need for extended duration of steroid administration followed by a long course of neuro rehabilitation.

case report:

A 45-year-old woman underwent a resection for a large epidermoid cyst excision in left medial temporal region .[Figure 1]. The patient presented 4 days after surgery with minimal drainage from the surgical site, wound gap and severe headache. The wound was reapproximated and treated as a superficial wound infection with oral antibiotics. The patient was also treated with a 7 days course of dexamethasone for a presumed chemical meningitis. One month later, the patient returned with decrease conciousness , severe headche , nausea, vomiting, and multiple fever spikes . There was no wound dehiscence and following an additional short course steroid taper administration that the patient's headache and nausea improved. Two weeks later, she once again presented with worsening headache, nausea, but this time with episodes of confusion, fever, and chills. A repeat CT showed development of post operative changes [Figure 2]. CSF was obtained from a lumbar puncture which showed a csf sugar 46 mg/dl ,csf protein 38 mg/dl with csf wbc 100 cells/cubic millimeter with polymorph 70% and lymphocytes 30% and CSF cultures were negative.csf gene expert and csf cbnaat , csf fungus also negative.The patient continued to have intermittent fevers without leukocytosis, tachycardia, or worsening of presenting symptoms. Following a course of broad-spectrum antibiotics for the treatment of a possible occult bacterial meningitis, the patient continued to endorse symptoms of meningitis. As a result, with continued clinical symptoms and low concern for infectious etiology, the patient underwent further broad spectrum antibiotics..

Afterward, the patient remained in the hospital and continued to exhibit intermittent fevers. A repeat csf examination from lumbar puncture obtain concerning for worsening meningitis; however, repeat CSF Gram stain and culture remained negative. Over the coming days, the patient's symptoms improved and she was again started on a course of steroids for chemical meningitis and discharged home following 17 days of hospital admission. One-month later, the patient returned with a complaint of persistent postoperative pain and decrease responsiveness and alertness . She also endorsed blurry vision, and headaches while bending forward. On physical exam,left petrional craniotomy scar wound healthy and no CSF leak. The patient had a neck stifness and fever without any additional lower cranial nerve dysfunction. Ct brain was done showing post operative changes [Figure 4]. The patient was again admitted, and while the CSF profile continued to raise suspicions for an occult infectious process (WBC 158 cells/cubis milimeters lymphocytes 72% , protein 168 mg/dL , glucose 38 mg/mL, and normal neutrophils 2% that the Gram stain remained negative. Several hours into her admission, the following morning her clinical status continued to decline necessitating emergent intubation. Afterward, the patient was taken emergently to the operating room for left petrional craniotomy for reexploration . Intraoperatively, there was no gross purulence encountered. Tissue obtained shows cerebellar parenchyma with epidermoid cyst, chronic inflammation, necrosis, granulation tissue, and foreign body reaction with degenerating keratin debris . The patient required persistent ICU level care with continued decline into a comatose state. Ultimately, following multidisciplinary discussions between infectious disease, immunology, rheumatology, and neurology for atypical clinical course, the patient was started on high-dose prednisone (60 mg) for severe chemical meningitis. The patient's clinical examination slowly improved to the point of being able to follow simple one-step commands. Subsequent CSF analysis done on post op day 78 from first operation began showing improvement in profile WBC 8 cells/cubis milimeters , 74 mg/dL glucose, and 6 mg/dL protein and the leptomeningeal enhancement was no longer identified on further follow-up imaging. Throughout the remainder of her hospitalization, her GCS score continued to improve. she gone to a neurorehabilitation facility, where she completed a long course of therapy. The patient is now 6 month postoperative from her second operation.

DISCUSSION

Aseptic meningitis Typically following epidermoid resection, resultant postoperative aseptic meningitis is thought to arise from the release of breakdown products, mainly keratin and cholesterol, from either the spontaneous leakage or surgical release of epidermoid contents into subarachnoid spaces leading to the inflammatory reaction.[8] Due to this risk, care must be taken during surgical excision to avoid rupture to prevent leakage of cyst contents with the goal of safe and complete resection without damage to surrounding neurovascular structures.[7,10] The risk of aseptic meningitis is high and thought to be related to the amount of residual cyst postoperatively.[7] Characteristic symptoms of aseptic meningitis include headaches, vomiting, fevers, meningismus, and cognitive impairment.[6,9] Diagnostic signs are obtained through lumbar puncture which often demonstrates an elevated CSF white cell count, protein, reduced glucose, negative Gram stain and culture results (diagnosis can be confirmed after three consecutive negative results), as well as CT and MRI imaging with associated meningeal enhancement.[3,8,9] .Hydrocephalus can often be associated with aseptic meningitis. The occurrence of aseptic meningitis is the result of epidermoid cyst contents entering the subarachnoid space which results in a cascade of inflammatory changes.[2,8] These inflammatory changes can contribute to decreased CSF absorption.It is often associated with either a CSF infection and/or a CSF leak occurring more frequently in patients with larger tumors and in those with a prior history of cranial surgery. The treatment for patients with aseptic meningitis, with or without hydrocephalus, involves the initial use of antibiotic therapy until infectious meningitis etiologies are ruled out, at which point they should be discontinued and systemic steroid

treatment initiated. Inflammatory mediators The specific biochemical inflammatory cascade driven by the breakdown products such as keratin in epidermoid cysts that result in aseptic meningitis has not been thoroughly investigated. However, a study by Cuff et al. found interleukin-6 (IL-6) to be elevated in patients with either an infectious or noninfectious cause of meningitis and lower levels of IL-17 in noninfectious meningitis patients. IL-6 has been known to provide a variety of central nervous system (CNS) functions including neuroprotection and pathological inflammatory responses.TNF-α is itself a pro-inflammatory cytokine with important CNS functions that initiate a major cascade of other inflammatory cytokines, including IL-6.[21] This mechanism functions by causing the phosphorylation of nuclear factor kappa B which enters the cell nucleus and induces the transcription of responsive genes including the pro-inflammatory cytokines IL-6 and more TNF-α. Steroids are known to be responsible for the alteration of inflammatory gene transcription. In animal models, it has been shown that dexamethasone reduces concentrations of IL1β and TNF-α, especially in bacterial meningitis.[5] Steroids have also been demonstrated to activate gene expression of IL-10, which function as an anti-inflammatory cytokine.[1] Supratherapeutic steroid administration was paramount to the recovery in the above-presented case and we suspect its effects specifically in decreasing TNF-α and prolonging IL10 contributed to the patient's recovery. However, further investigation into the optimal steroid dosing, duration, versus need for empiric treatment or prophylactic use is needed to better identify difficult/protracted episodes of aseptic and chemical meningitis to provide better diagnosis and treatment regimens for this rare presentation of postoperative aseptic meningitis.

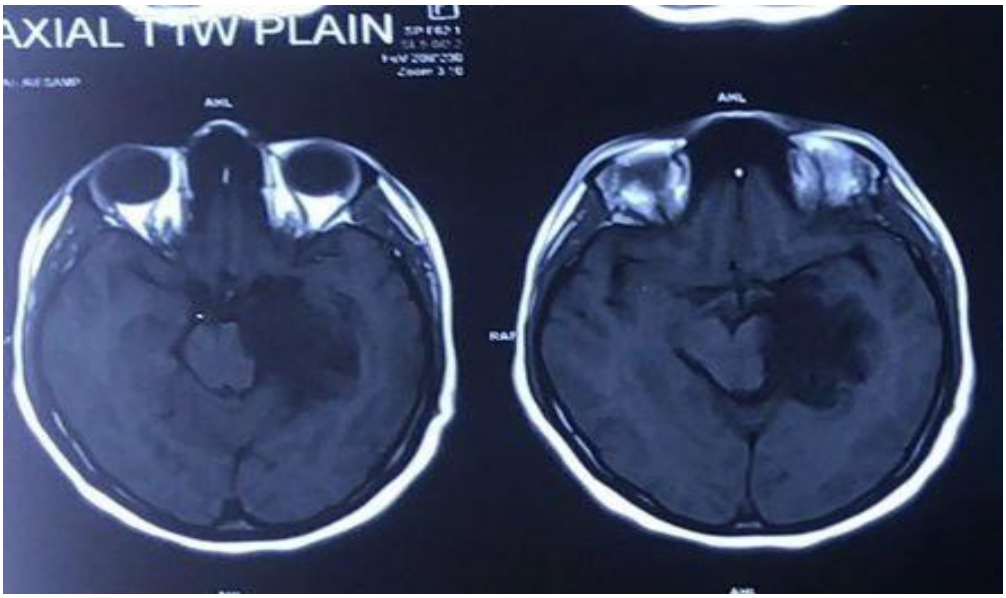

Figure 1: pre op image of MRI T1W plain shows lesion appear hypointense

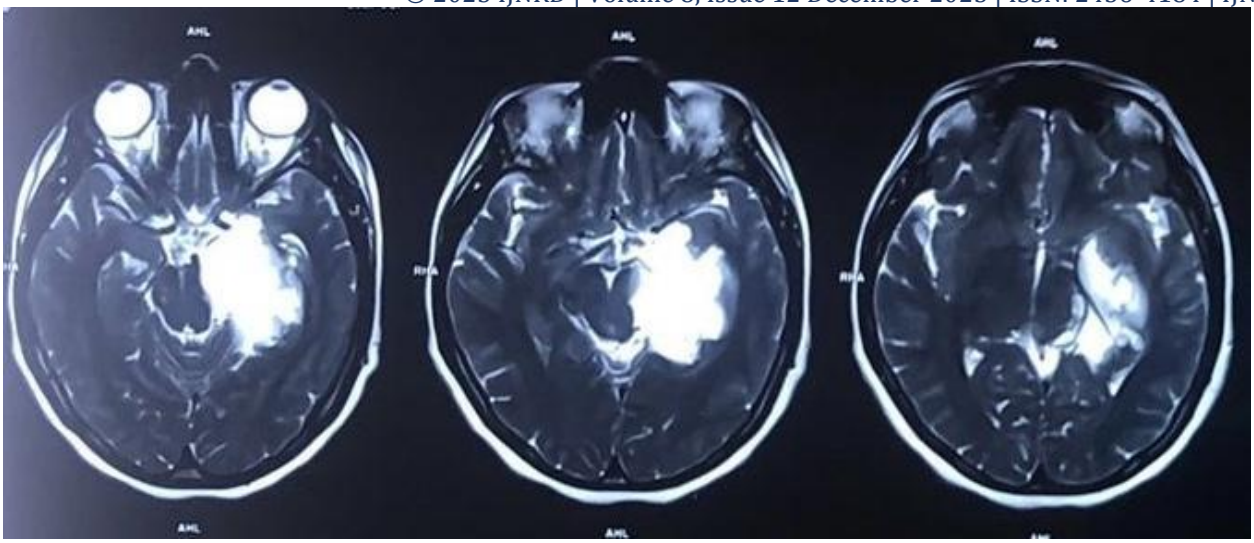

Figure 2: pre op image of MRI T2W shows illdefined nonenhancing hyperintense extraaxial lesion in left medial temporal region

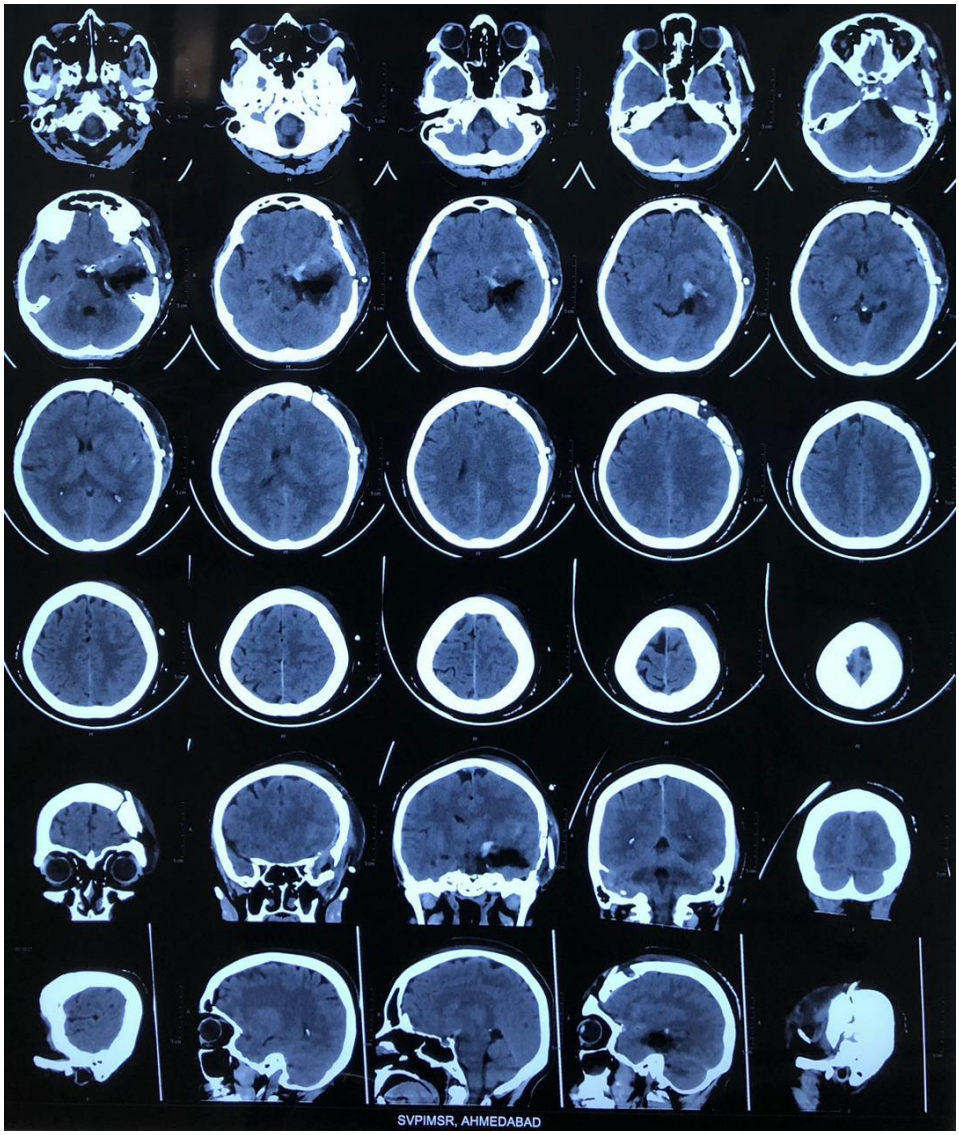

Figure 3 : post op image of ct brain (post operative day 2)

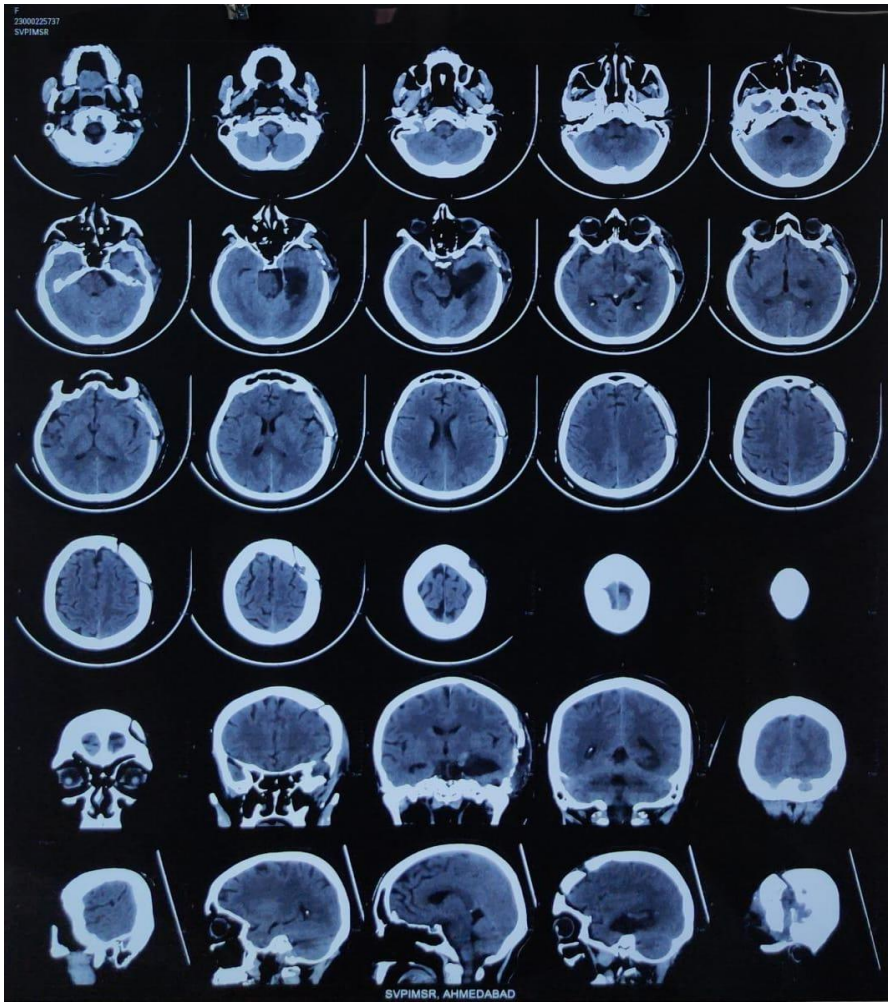

Figure 4 : post op image of ct brain (post operative day 47)

CONCLUSION ::

This case revolves around a patient who experienced aseptic meningitis, a condition not caused by infection, following the surgical removal of an epidermoid cyst in the left medial temporal region. The meningitis was particularly severe, necessitating an extended period of critical care spanning several months. While aseptic meningitis following resection of epidermoid cysts is not a rare phenomenon, the clinical course and severity of the inflammatory reaction in this patient is quite unique. The patient's clinical progression was attributed to a hyperreactive inflammatory reaction triggered by residual keratin debris remaining after the surgery. This reaction manifested as a delayed and severe episode of inflammation affecting the entire brain. Throughout the extensive clinical evaluation, no infectious cause was ever identified.The purpose of reporting this case is to inform neurosurgeons about the potential for this

uncommon yet severe scenario, given the absence of established criteria for identifying at-risk patients. The patient is now self-sufficient and lives on their own.

Declaration of patient consent:

Patient's consent not required as patient's identity is not disclosed or compromised.

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Conflicts of interest : There are no conflicts of interest.

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