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RESEARCH ARTICLE

WHEN ONE ISN'T ENOUGH: A CLINICAL CASE OF MULTIPLE TUBERCULOUS SUBDURAL EMPYEMAS

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Abstract

Introduction: Empyema is, by definition, an encapsulated purulent mass within the subdural space, distorting the underlying cortex and causing discrete neurological symptoms based on its position and volume. This condition is uncommon, particularly when it is situated interhemispherically. Diagnosis is chiefly based on imaging studies along with the infectious disease evaluation. Tuberculous empyema is the least frequently described form of neuro-meningeal tuberculosis. Prompt surgical intervention coupled with targeted long term antimicro bial therapy is required to ensure complete eradication of the pathogen while preventing potentially life-threatening complications.

case report: We report the case of a 76-year-old female patient who initially presented with intractable headaches accompanied by nausea and vomiting. Her condition worsened with the onset of dysarthria and right-sided facial weakness, followed by a generalized tonic-clonic seizure.A cerebral CT scan revealed multiple hypodense subdural collections with peripheral enhancement. Surgical drainage via burr hole evacuation was performed for the largest collection, and PCR testing confirmed Mycobacterium tuberculosis. Anti-tubercular therapy was initiated and maintained for 9 months, with favorable clinical and radiological outcomes.

Conclusion: Mycobacterium tuberculosis is a rare cause of subdural empyema, but it must not be overlooked due to the potentially life-threatening complications it can cause. Prompt and appropriate management is essential. While surgery plays a crucial role in pathogen identification, it is insufficient alone and must be combined with anti-tubercular therapy, which may extend up to 12 months

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Introduction:-

Empyema is, by definition, an encapsulated purulent mass within the subdural space, distorting the underlying cortex and causing discrete neurological symptoms based on its position and volume. This condition is uncommon, particularly when it is situated interhemispherically. Diagnosis is chiefly based on imaging studies along with the infectious disease evaluation. Tuberculous empyema is the least frequently described form of neuro-meningeal tuberculosis. Prompt surgical intervention coupled with targeted long-term antimicrobial therapy is required to ensure complete eradication of the pathogen while preventing potentially life-threatening complications.

Case report:-

A 76-year-old female treated for glaucoma presents with intractable headaches associated with nausea and vomiting of 5 days duration, which have been treated symptomatically. Her condition worsened with the onset of dysarthria and right-sided facial weakness, followed by a generalized tonic-clonic seizure. On admission, neurological examination revealed that the patient had a Glasgow Coma Scale score of 15, no focal deficits, and no clinical signs of menin- gism (kerning's was negative) . Her BP was 157/53 mm of Hg and her temperature was recorded at 39°C (102.2°F).white blood cell count was 18,300/mm3 with 70% lymphocytes, highC-reactive protein level 260mg/l, The erythrocyte sedimentation rate 100mm/hour, high procalcitonin level10ng/ml. Her liver function and renal function tests were within normal limits.

A cerebral CT scan performed upon admission revealed multiple hypodense subdural collectionsperipheral enhancement predominantly in the left frontal and interhemispheric regions, accompanied by mass effect on adjacent parenchyma.The patient was transferred to the ICU, where anticonvulsant therapy (levetiracetam) was initiated. Dosage was subsequently adjusted based on electroencephalogram (EEG) findings. Brain MRI corroborated the CT findings, confirming subdural empyemas in the bilateral frontal lobes, left parietal region, interhemispheric fissure, and extending to the cerebellar tentorium.Blood cultures and cytobacteriological urine analysis yielded negative results. A comprehensive oral and maxillofacial examination revealed no potential infectious foci of dental or otorhinolaryngological origin.

Dermatological assessment demonstrated no evidence of cutaneous pathology, including intertrigo or ulcerations, thereby excluding the skin as a probable portal of entry.The patient underwent neuro-navigation-guided abscess drainage through a burr hole targeting the left frontal subdural collection. This approach was chosen to access the collection responsible for the mass effect observed on CT scan, as well as the underlying epileptogenic activity detected on electroencephalography. Complete evacuation of the collection was achieved via aspiration after coagulation and dural opening, followed by thorough irrigation with normal saline). Cytobacteriological examination of the drained pus allowed the direct identification of Mycobacterium tuberculosis. In addition, PCR analysis using the GeneXpert test performed on the collected pus was also positive for M. tuberculosis.

Screening of systemic localizations was initiated via contrast-enhanced chest-abdomen-pelvis CT showed no evidence of No evidence of extracranial Mycobacterium tuberculosis involvement. An intensive antitubercular therapy was initiated according to the ERIP K4 protocol (Rifampicin 10 mg/kg/day, Isoniazid 5 mg/kg/day, Pyrazinamide 25 mg/kg/day, Ethambutol 15 mg/kg/day), maintaining standard weight-based dosing for a planned 9-month course.Following the initiation of treatment, the patient showed clinical improvement along with a progressive decline in inflammatory markers. At the one-month follow-up visit, a control brain CT scan revealed complete resolution of the empyema previously evacuated via burr hole, as well as a significant reduction in the size of the subdural collections located along the falx cerebri. The patient will continue to be monitored throughout the full course of anti-tuberculous therapy, with regular assessment of liver and kidney function, and systematic ENT consultations due to the potential toxicity of the treatment.

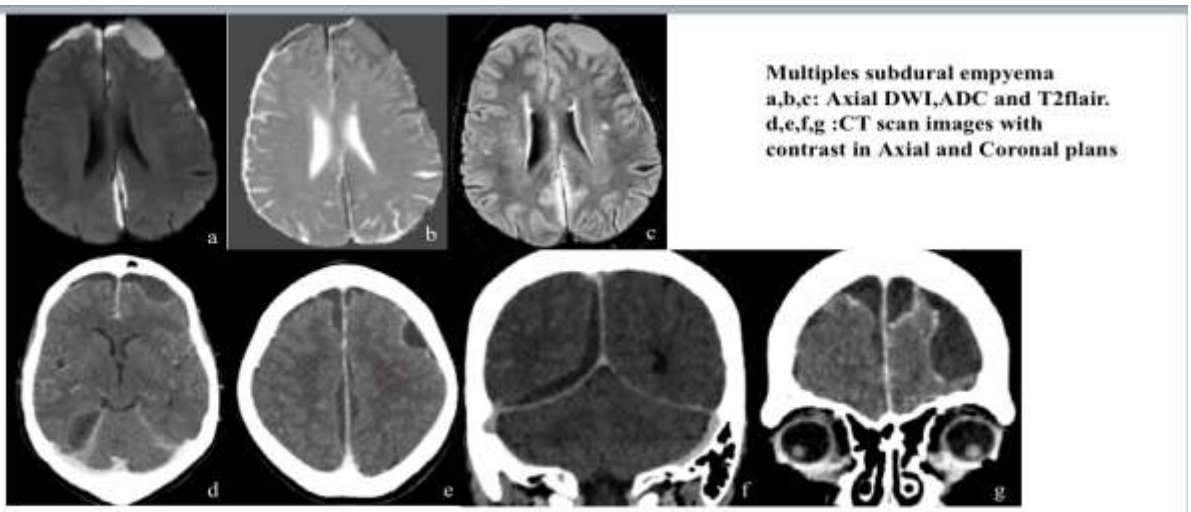

Figure 1

Figure 1 :
a, b, c. Axial MRI slices in DWI, ADC, and T2-FLAIR sequences showing multiple subdural collections in the right and left frontal regions as well as along the falx cerebri, appearing hyperintense on T2-FLAIR and DWI sequences, with corresponding restricted diffusion on the ADC map.
d, e, f, g. Contrast-enhanced brain CT scans revealing hypodense subdural detachments with peripheral enhancement in the bilateral frontal regions and along the falx cerebri.

Figure 2 :Intraoperative photograph of pus collected from the left frontal subdural space following burr hole trephination.

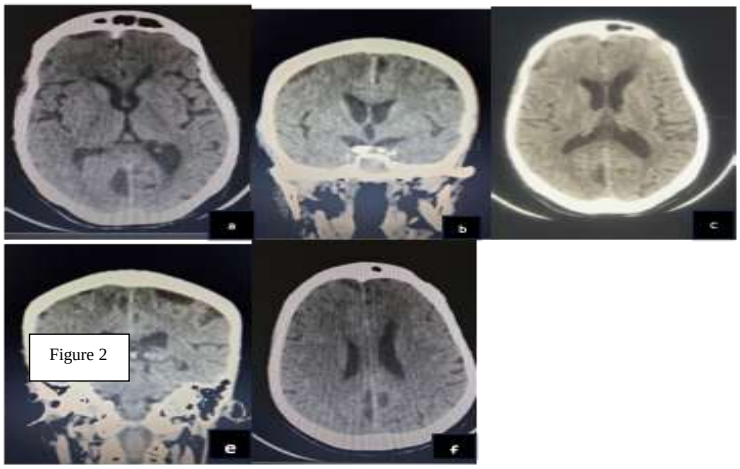

Figure 2

Figure 3 :
A,b,c,d,e,fContrast-enhanced axial and coronal brain CT scans at one month of treatment showing a reduction in thesize of the subdural collections.

Discussion:-

Empyema remainsa relatively rare condition that complicates an infection, most often in the ENT (ear, nose, and throat) area, that is untreated or poorly treated. It can occur in patients of all ages, with a higher frequency in individuals whose immune status is compromised. [1,3]The causative pathogen depends on the primary infection site,Haemophilus influenzae, Staphylococcus aureus, and Staphylococcus epidermidis are typically the most prevalent.[20]Tuberculous empyemas being exceptional should still not be overlooked in cases of severe intracranial suppurations.[9]The infection is spread as a result of a slow TB which spreads through the blood and causes infection to the cranial cavity.[7,9]Through this article, we aim to demonstrate that the etiological workup should not exclude testing for tuberculosis (TB), and that the treatment involves in certain cases, surgery combined with targeted and appropriate antibiotic therapy.

Clinical presentations vary significantly depending on the empyema's location and size [18]. While headaches, seizures, and focal neurological deficits represent the most commonly reported symptoms [18,20], our patient's initial manifestation followed this pattern with persistent cephalgia.Notably, classic infectious signs may be subtle or

absent during early stages. Many cases - including ours - demonstrate afebrile presentations without tachycardia or tachypnea, potentially delaying diagnosis.[18]

The standard etiological workup for suspected intracranial infection includes neuroimaging complemented by laboratory investigations. While non-contrast head CT provides initial assessment of subdural collections, contrast-enhanced brain MRI (1.5T or 3T) represents the gold standard for characterization, offering superior soft tissue resolution and multiplanar capability.[3,4]Essential laboratory studies should include complete blood count, inflammatory markers (CRP, ESR), and tuberculin testing. Lumbar puncture with cerebrospinal fluid analysis (including AFB smear, PCR, and culture) may be performed when mass effect is absent.[3]

The combination of thick peripheral enhancement with diffusion restriction shows 92% sensitivity and 88% specificity for bacterial empyema in recent meta-analyses.[16] When tuberculosis is suspected, the presence of basal meningeal enhancement or tuberculomas provides additional diagnostic clues.[17]MRI with gadolinium injection reveals a subdural collection with marked enhancement of the walls. Diffusion-weighted sequences, on the other hand, will show a hyperintense signal with restricted ADC within the collection. [3,2,4,16]These radiological features help differentiate empyema from its main radiological differential diagnoses, such as chronic subdural hematoma, which appears as hyperintense on T1, hypo- then hyperintense on T2, with signal changes in case of rebleeding. The diffusion and ADC signals are variable.[13,16,3]

Additionally, the wall enhancement is absent or moderate compared to that of empyema.As for hygroma, it appears as isointense on T1 and isointense on T2 (similar to CSF). There is no meningeal enhancement, and the ADC is also elevated[12,13,16] .Diffusion-weighted imaging (DWI), magnetization transfer (MT), and in vivo proton MR spectroscopy (PMRS).5–10 On DWI, low ADC has been found in fungal and tubercular abscesses similar to pyogenic abscesses.[9,11,10,15] Tuberculous brain abscesses exhibit significantly lower MT ratios compared with those of pyogenic abscesses, with no evidence of amino acids on in vivo PMRS –a spectral hallmark of the pyogenic abscess.[15,18,]Fungal lesions are known to show lipids (1.2–1.3 ppm), lactate (1.3 ppm), alanine (1.5 ppm), acetate (1.9 ppm), succinate (2.4 ppm), choline (3.2 ppm), and unidentified resonance at 3.8 ppm.[12,14,15]

MRI can help establish the etiological diagnosis in certain cases, such as in the context of empyema secondary to an ENT infection (e.g., acute frontal or sphenoid sinusitis)[9]. In such cases, an iso- to hypointense signal on T1, a hyperintense signal on T2, and gadolinium enhancement of the inflamed mucosa will be observed. [9] Diffusion-weighted imaging shows a hyperintense signal with low ADC, similar to the purulent material seen in empyema or abscesses.[2,9]In our patient, surgical intervention was proposed due to the significant increase in the size of the lesions, particularly the largest one, which caused a mass effect on the parenchyma. Evacuation was performed through a burr hole, with cytobacteriological samples taken; thorough lavage was carried out, and the follow-up CT scan showed complete collapse of the drained collection. The goal of surgical management is to relieve pressure and provide a targeted response to the identified pathogen

The choice of surgical procedure, particularly between a burr hole or a craniotomy, depends on the size and location of the empyema.[6,8,19]Craniotomy offers the advantage of wider exposure, especially in cases of large or multiple empyemas.[1,8,18,19,20] Evacuation by aspiration through a burr hole allows for quicker drainage, particularly in cases of hemodynamic instability, but carries a risk of recurrence, especially if intraoperative lavage is not effective and brain re-expansion occurs before complete evacuation.[5,6,8,18,19,20]A conservative (non-surgical) approach may be considered for parafalcine subdural empyema (SDE) in clinically stable patients without significant midline shift on imaging, given these lesions' typically indolent progression and the technical challenges associated with accessing the interhemispheric fissure.[8,20] However, surgical intervention should occur as early as possible, before the patient deteriorates[6,14,18]. Surgery also becomes imperative when the collection demonstrates progressive enlargement despite appropriate antibiotic therapy.[3,14,18]

Neurotuberculosis requires treatment with an appropriate therapeutic regimen, as the consequences of untreated cases are severe.[4,18] A key component of treatment is long-term anti-tuberculosis chemotherapy.[17,21] However, corticosteroid therapy, often used in the acute phase to manage cerebral edema, should be avoided.[5,21] This is because corticosteroids may hinder the penetration of anti-tuberculosis drugs into the abscess and disrupt the encapsulation process.[5,7] In most cases, the administration of anti-tuberculosis drugs results in the regression of tuberculous abscesses and empyemas.[7,10,21]

Conclusion:-

Intracranial empyema remains a rare but serious complication of infections, most often of ENT origin, requiring prompt diagnosis and management. Although tuberculous subdural empyema is exceptional, it must be considered in the differential diagnosis, especially in regions endemic for tuberculosis or in patients with atypical presentations. This case highlights the importance of maintaining a high index of suspicion and including tuberculosis in the etiological workup of intracranial collections, even in the absence of extracranial involvement.Magnetic resonance imaging, particularly with diffusion-weighted sequences and contrast enhancement, remains the cornerstone for accurate diagnosis and differentiation from other mimicking entities. Advanced imaging techniques, including magnetization transfer and proton MR spectroscopy, can further aid in identifying specific etiologies.Management requires a multidisciplinary approach, combining neurosurgical drainage — whether via burr hole or craniotomy — and targeted antimicrobial therapy. In the case of tuberculous empyema, prolonged anti-tuberculosis treatment is essential. Early intervention improves prognosis, while delayed diagnosis may lead to severe neurological sequelae or death.Through this case, we emphasize the need for vigilance regarding rare but treatable causes of intracranial empyema and advocate for individualized therapeutic strategies based on clinical, microbiological, and radiological findings.

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