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Case Report

Concomitant extradural, subdural, and intraparenchymal abscesses of the brain in a patient with cerebral melioidosis – A case report

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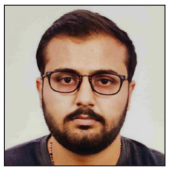

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ABSTRACT

Background: Extra axial abscess of the brain is a rare entity, moreover, extra-axial abscess concomitant with intraparenchymal purulent collections are scarcely reported in the literature. Etiology includes penetrating trauma, paranasal sinusitis, mastoiditis, craniospinal surgeries, and the rare spread of infectious agents through the hematogenous route.

Case description: We present a case of a young male with Burkholderia pseudomallei Central Nervous System (CNS) melioidosis, forming abscesses in extra-axial and intraparenchymal planes without contiguity.

Conclusion: This is to emphasize the importance of MR spectroscopy and other convenient methods in differentiating the etiology in cranial infections.

Keywords: Abscess, Burkholderia, Epidural, Intraparenchymal, Melioidosis, Subdural

INTRODUCTION

Purulent collections in the central nervous system (CNS) occur in the spaces between the inner table of the skull and dura; between the dura and arachnoid mater, known as epidural, and subdural spaces, respectively, collectively referred to as extra-axial abscesses. Furthermore, abscess formation can also occur intraparenchymal known as an intra-axial abscess. Developing countries have a higher incidence of abscesses, around 8%, of all CNS space-occupying lesions. Of the micro-organisms affecting the brain, Gram-negative bacilli account for 9–31% of the total infections.[10] Our case report features CNS melioidosis, caused by *Burkholderia pseudomallei*, a Gram-negative bacillus endemic to tropical countries.[2] The rising occurrence of melioidosis in India can be attributed to rising temperatures, humidity, and ultraviolet radiation; the increased prevalence of diabetes mellitus, and immunocompromised status in the population.[6] The bacillus is transmitted either through direct inoculation onto the abraded or lacerated skin through contaminated soil or water, or inhalation of droplets or bacilli-laden dust particles.[8]

CASE REPORT

A 24-year-old male patient presented to us with global aphasia and right-sided hemiplegia. Medical history was significant for intermittent headaches for 4 months. One month after

the onset of symptoms, the patient developed an evening rise in temperatures and abdominal pain. He sought treatment at another institution where he was evaluated with ultrasound abdomen that showed splenomegaly with multiple granulomas and the findings were later confirmed with computerized tomography (CT) abdomen which also revealed periportal, para-aortic, and aortic caval lymphadenitis-like features, for which he was treated presumptively as a mycobacterial tuberculosis infection. Ten days later, the symptoms worsened and the patient developed new-onset speech difficulty along with right-sided weakness. Consequently, the patient was evaluated with a magnetic resonance imaging (MRI) brain which was suggestive of left frontal thin subdural (measuring around 2*3*2 mm, volume 6 cubic mm) and a rim of thin epidural collection, and the patient was continued on anti-tubercular treatment (ATT) presuming CNS tuberculosis.

A month later, he presented to us when complaints progressed to aphasia and hemiplegia. Physical examination was significant for altered sensorium with a Glasgow Coma Scale of E3VAM5 and right-sided hemiplegia. The patient was reevaluated with a plain and contrast MRI of the brain. On T1, isointense rim with the hypointense collection was noted in the lesion [Figures 1a and 1b], findings confirmed in T2 sequence with fluid-fluid level, also T2 and MR flair sequence shows hyperintense extra-axial/subdural collection [Figures 1c and 1d]. MRI contrast scan featured conglomerate peripheral ring-enhancing lesions [Figures 1e and 1f] in the left frontal lobe (intraparenchymal) with moderate to significant perilesional edema causing mass effect over the frontal horn and body of the ipsilateral ventricle. Diffuse weighted imaging was suggestive of diffusion restriction within the wall of the lesion and in the dependent content and debris [Figure 1g]. Magnetic resonance (MR) spectroscopy showed significantly large lipid lactate peaks in the lesion [Figure 2a]. Findings favored infective etiology with cerebral abscess, subdural empyema, and dural thickening with an intradural abscess in the left frontal region with frontal cortical vein thrombosis. Radiological findings were confirmed intra-op, subsequently was compared with post op scan which showed complete abscess evacuation [Figure 2b].

The patient underwent left fronto-temporo-parietal decompressive craniectomy and abscess evacuation. After craniectomy, adherent purulent collection was noted on the inner table of the bone flap and dura, suggestive of osteomyelitis and epidural abscess [Figure 3a]. Durotomy was performed and thick pus was noted in the subdural plane [Figure 3b] which was evacuated. Subsequently, the abscess

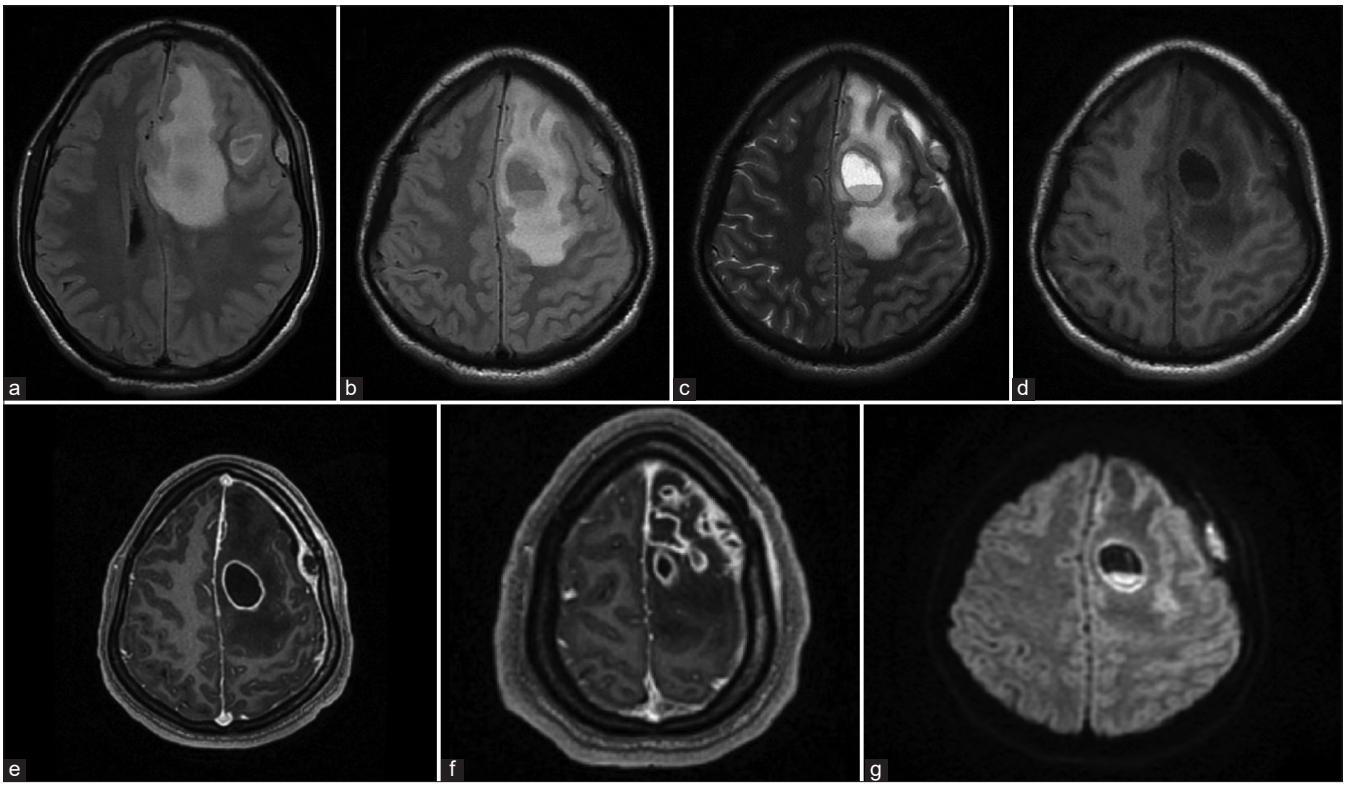

Figure 1: MRI findings (a) MRI T1 showing intraparenchymal abscess and edema causing mass effect, (b) MRI T1 fluid-fluid level in the abscess, (c) MRI T2 shows hyperintense collection in subdural and epidural space, (d) MR Flair shows hyperintense extra-axial collection, (e and f) MR Contrast showing multiple ring-enhancing lesions, sub dural, and epi dural collections, (g) Diffusion restriction noted along the capsule and contents of the abscess.

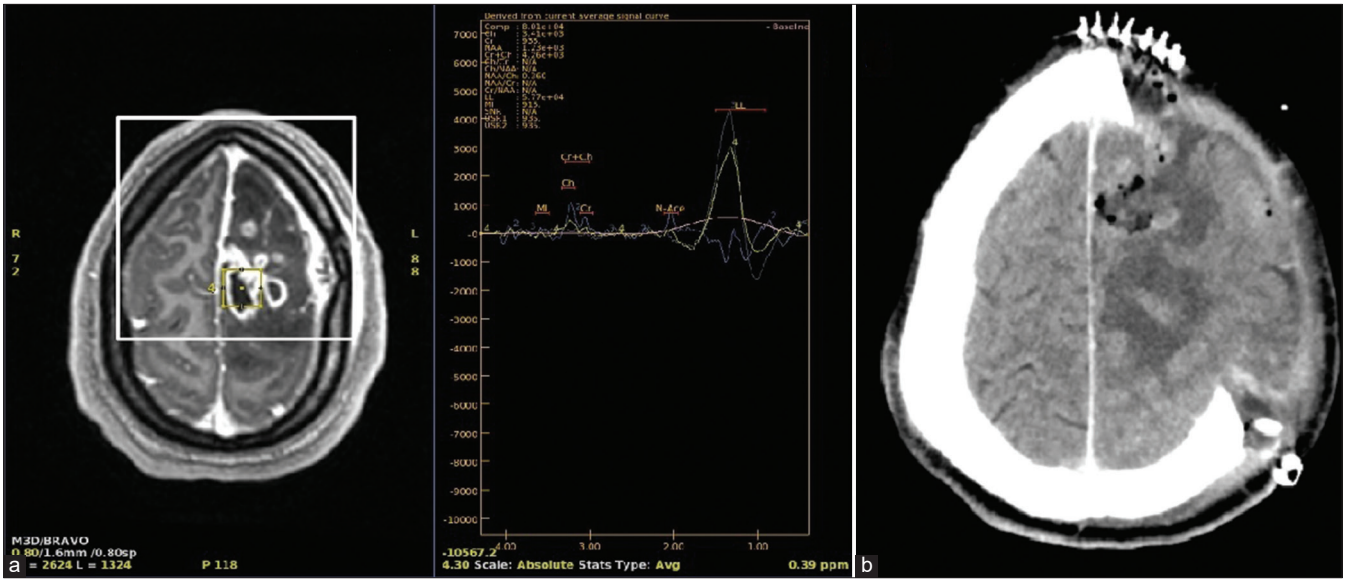

Figure 2: MRS findings (a) MR spectroscopy suggestive of lipid lactate peaks, (b) Postoperative contrast-enhanced CT suggestive of complete abscess evacuation.

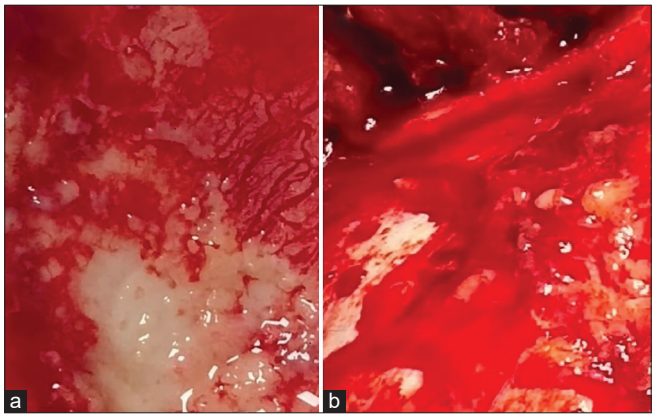

Figure 3: Intraoperative images (a) Purulent collection over the dura and (b) Purulent collection over the cerebral cortex, respectively.

was approached through a pial incision over the superior frontal gyrus at the point of tense thickened pia; clear differentiation was seen between the capsule and surrounding glial tissue. The capsule was incised with a stab, purulent contents were evacuated, the leftover capsular wall was excised, and hemostasis was achieved. The osteomyelitic bone and raised intracranial pressure features were indications for not replacing the bone flap. The histopathology and culture evaluation grew *B. pseudomallei*, subsequently, he was started on intravenous antibiotics and the postoperative contrast-enhanced CT showed complete abscess evacuation with a significant reduction of perilesional edema [Figure 2b]. Recovery was uneventful with aphasia resolution and right-sided motor power improvement. At 1-month follow-up, the patient had significant neurological improvement with no new deficits.

DISCUSSION

CNS infections are caused by wide variety of organisms including bacteria, parasites, fungi, and viruses. The most common route for cerebral fungal abscess is systemic foci spreading to CNS through hematogenous route, commonly isolated organisms being *Candida* and *Aspergillus* species. Radiological features are multifocal lesions, involvement of deep grey matter nuclei, signal heterogeneity on diffusion weighted (DW) imaging and higher apparent diffusion coefficient (ADC) values. Other features include meningitis, multiple infarcts, thrombosis, and aneurysm formation all attributable to vascular invasion by fungal elements.[10] Although viral etiology is rare, it has high mortality. Radiologically, there is negligible literature on viral cerebral abscess, as viral CNS infections has different traits of presentation including encephalitis (Herpes and Rabies virus), leukoencephalopathy (Human Immunodeficiency Virus), ventriculoencephalopathy (Cytomegalovirus), tissue necrosis, and hemorrhagic foci with sequelae of diffuse gliosis and cyst formation [malacia] with calcification (Coxsackie virus).[1]

Melioidosis is caused by a Gram-negative aerobic motile bacillus, *B. pseudomallei*, endemic to regions like Australia and parts of South-east Asia like India.[2] The clinical course may be acute, subacute, or chronic depending on the pathogen, location, and immune status of the host. The clinical manifestations are protean that range from pneumonia-like symptoms to transient bacteremia,[2] where the severity of the disease is attributed to risk factors such as diabetes, renal disease, and alcoholism.[6] Neurological manifestations are usually seen in only 1–1.5% of melioidosis infections.[12] We aim to describe the pathogenesis

of the unique presentation of a case of CNS melioidosis with abscesses occurring in all three planes. Isolated extra-axial purulent collections contribute 12% and intraparenchymal to 35% of overall CNS melioidosis infections.[12] A retrospective analysis performed by Kural *et al*. in 2019 revealed that the most likely etiologies for the intracranial infections were localized spread from sinusitis, previous cranial surgeries and lumbar procedures, nasopharyngeal and otogenic infections, and trauma.[7] Our patient did not have any of the conditions mentioned above indicating that the likely source of infection to the brain was hematological. A study performed by Wongwandee and Linasmita showed that the spread of infection through blood played a crucial role in disease pathogenesis.[12] The blood cultures in our patient at the time of presentation were negative for any bacterial growth implying that the infection was latent.[12]

The purulent collection of the subdural space and the intraparenchymal abscesses resulted in significant vasogenic edema in the left frontal lobe causing aphasia and right-sided weakness in our patient. *B. pseudomallei* crosses the blood-brain barrier using the transcellular, paracellular, or trojan horse methods.[12] The latter is essential to the pathogenesis of intraparenchymal lesions, as the bacteria transmits intracellularly in the monocytes.[12] The spread of infection to the subdural space could be the result of the extension of thrombophlebitis into the cerebral veins, containment of infection with the inflammatory response leads to abscess formation.[11]

The clinical signs of raised intracranial pressure such as altered sensorium, alongside radiological findings of vasogenic edema leading to mass effect and involvement of bone by epidural abscess warranted management with decompressive craniectomy over conventional craniotomy and abscess evacuation. Intraoperatively, following the retrieval of the bone flap, pyogenic material was noted over the dura and the inner table of the bone suggesting epidural empyema. The valveless nature of the diploic veins facilitated the retrograde spread of infection to the epidural space.[4]

Role of MR spectroscopy in infectious etiology: Cerebral Bacterial abscess usually show peaks of amino-acids, acetate, aspartate, and succinate peaks with marked decline of neuronal markers such as N-acetyl aspartate, creatine, and choline. Closest differential diagnosis to bacterial abscess is cystic glioma and cystic metastasis, radiologically they can be distinguished by ADC values and DWI sequence, where bacterial abscess features higher diffusion restriction on DWI and lower ADC values, whereas neoplastic etiology usually features moderate diffusion restriction and higher ADC values.[3]

Our case report also identifies the need for convenient methods to differentiate between TB and other bacterial abscesses. The radiological impression at the time of presentation seen in the patient was closely mimicking tuberculosis abscess prompting the initiation of ATT without a definitive diagnosis. At present, the most effective method to differentiate between tuberculous and bacterial abscesses is MR spectroscopy.[5] The presence of acetate and succinate with lipid peaks is seen in pyogenic abscesses whereas tuberculosis has lipid peaks without the metabolites.[5] Clinicians need to be mindful of the possibility of other bacterial abscesses and have diagnostic suspicion in disease-endemic areas to avert complications.

CONCLUSION

This is to emphasize the importance of MR spectroscopy and other convenient methods in differentiating the etiology in cranial infections.

Declaration of patient consent

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

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Nil.

Conflicts of interest

There are no conflicts of interest.

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