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Neurobrucellosis, an Unusual Culprit in Sensoneural hearing loss and Acute Psychosis – A Riveting Clinical Case Exploration

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Abstract

Neurobrucellosis, an exceptional neurological complication stemming from Brucella infection, is renowned for its diverse clinical expressions. Delving into the extraordinary, this study unveils a captivating clinical case illuminating the atypical manifestation of Neurobrucellosis as acute psychosis. The patient, devoid of any precedent psychiatric history, displayed a sudden onset of psychotic symptoms, featuring agitated behavior, disjointed speech, restlessness, intermittent crying, and a 4-day bout of sleep disruption, necessitating a thorough diagnostic inquiry. The subsequent analysis of cerebrospinal fluid and serological tests revealed the presence of Brucella, conclusively establishing Neurobrucellosis as the underlying cause for the psychotic presentation. This case not only spotlights the critical consideration of Neurobrucellosis in the realm of acute psychosis but also underscores the vital need for a multidisciplinary approach to ensure precise diagnosis and timely intervention. Embark on this captivating journey through an intricate medical puzzle that challenges conventional diagnostic expectations.

Keywords: Neurobrucellosis, Meningoencephalitis**,** Malta fever.

Case report

In the emergency department, a 32-year-old male sought medical attention with a four-day history of aberrant behavior, irrelevant speech, and wandering tendencies. Accompanying symptoms included restlessness, impulsivity, episodic crying, and decreased sleep, with no reported psychiatric history, familial psychiatric disorders, or drug intake. Notably, the patient reported a seven-month history of decreased hearing, a concern that had not been previously addressed medically.

Upon examination, the patient exhibited afebrility, consciousness, and orientation to time, but not place or person. A Glasgow Coma Scale score of15/15 was noted, along with slurred and incoherent speech. Positive men in geal signs and acute psychosis features, including delusions, hallucinations, suspiciousness, and agitation, was observed. General physical examination, hemogram, kidney and liver function tests yielded unremarkable results. Contrast magnetic resonance imaging revealed bilateral temporal lobe hyper intensities with a ring-enhancing lesion in the right temporal subdural space, suggestive of encephalitis. While tubercular and viral meningoencephalitis was initially considered, the MRI

findings leaned more towards tubercular meningoencephalitis. (Figure 1 and 2).

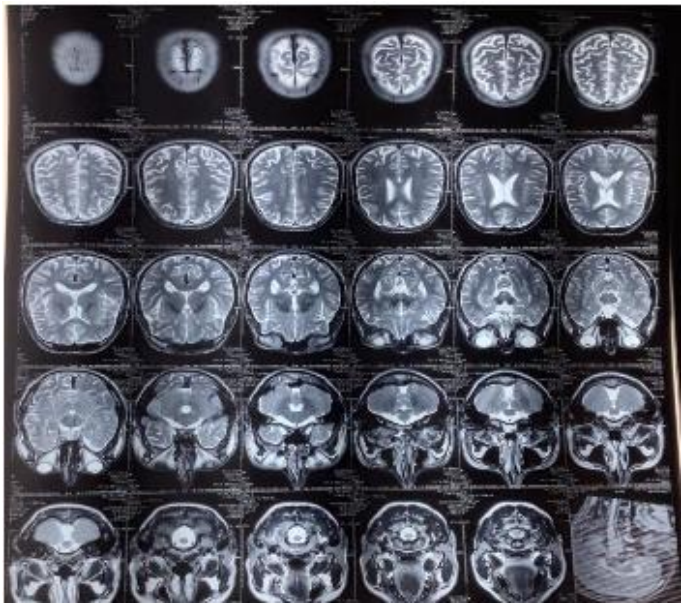

Figure 1

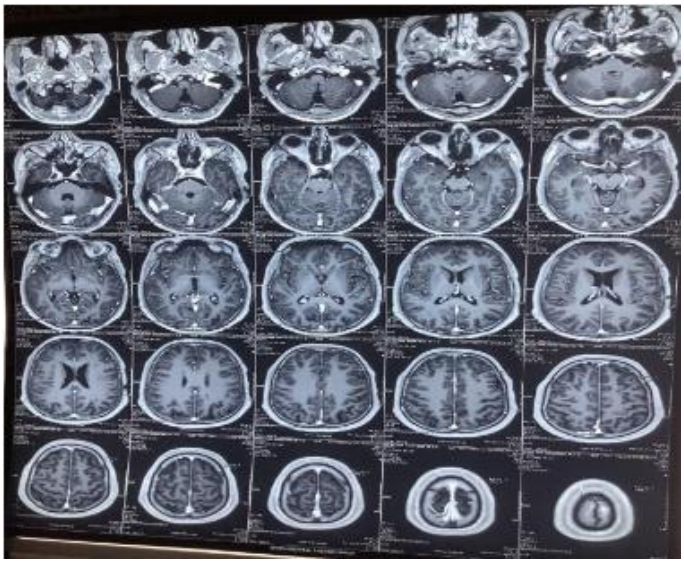

Figure 2

Subsequent cerebrospinal fluid (CSF) analysis unveiled reactive CSF with lymphocytic pleocytosis (210 cells, 80% lymphocytes), elevated protein (78 mg/dl), and decreased glucose (26 mg/dl). Despite negative results in Gram staining, fungal stain, acid-fast bacilli, and routine CSF cultures, adenine deaminase were elevated (13.50 IU/L). Surprisingly, tuberculosis PCR yielded negative results.

Further investigation into the patient's decreased hearing revealed a history of regular consumption of poorly cooked meat, particularly in the form of barbecues. This, coupled with evidence of moderate to severe sensor neural hearing loss as evidenced by pure tone audiogram (Figure 3), raised suspicion of Neurobrucellosis. Brucella antibody testing confirmed strong positivity for Brucella, with IgG and IgM levels in serum (EIA) measuring 18.51 and 15.79, respectively.

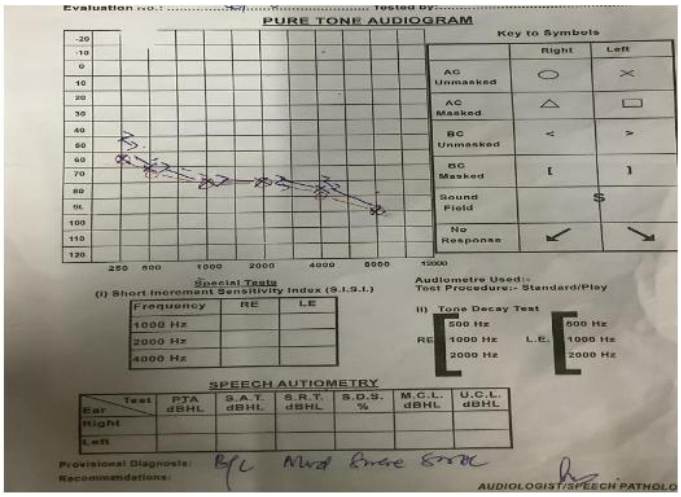

Figure 3: Pure tone audiogram demonstrating moderate to severe SNHL

Treatment commenced with injection ceftriaxone, oral doxycycline, and rifampicin. Psychiatric consultation initiated Olanzapine (10 mg once daily),resulting in the gradual resolution of psychotic symptoms within two weeks. Olanzapine was subsequently tapered off, and the patient demonstrated sustained improvement. This case underscores the intricate interplay between Neurobrucellosis and psychiatric manifestations, emphasizing the necessity of a comprehensive diagnostic and therapeutic approach in such complex presentations.

Discussion

Brucellosis is also known as “undulant fever,” “Mediterranean fever,” or “Malta fever” and is a zoonotic infection caused by the bacteria of genus Brucella. It is a gram-negative, intracellular, aerobic bacteria, of which

there are six species, with four causing brucellosis in humans (Brucella abortus, B. melitensis, B. suis, and B. canis)[1]. Human beings are usually dead-end hosts, and the main animal reservoirs are cattle, sheep, goats, and pigs. Human brucellosis is a multisystem disease involving the liver, spleen, bone marrow, lymph nodes, nervous, musculoskeletal, cardiovascular, gastrointestinal, and genitourinary systems[1].

The neurological presentation includes meningitis, meningoencephalitis, encephalitis, cranial neuropathies, intracranial hypertension, sinus thrombosis, radiculitis, peripheral neuropathy, myelitis, and psychiatric manifestations[2,3]. The disease can be insidious and present in many atypical forms, leading to delays in clinical recognition. Neurobrucellosis is most commonly diagnosed 2-12 months after symptom onset of symptoms[4]. As neurological complications can develop chronically, they are frequently misdiagnosed as other infections, such as tuberculosis.

The most common way to be infected is by eating or drinking unpasteurized or raw dairy products such as fresh milk, butter, and cheese[5]. Brucella can survive in these products for two weeks to three months. Other transmission routes include the ingestion of undercooked meat, contamination via wounds in the skin/mucous membranes through contact with infected animals, andinhalation[4]. Direct person-to-person spread of brucellosis is rare, as is transmission via sexual contact, tissue transplantation, or blood transfusion. This finds concordance with the findings seen in our case as he was eating barbeques frequently, which served as a source of infection .The nervous system can be involved at any stage of brucellosis. Both central and peripheral nervous systems can be affected by this condition. Acute or chronic meningitis are the most frequent nervous system complications of brucellosis.[6] The mechanisms of Neuropathophysiology in Neurobrucellosis remain unclear, however, three hypotheses exist a) a direct neuropathic effect, b) deleterious cytokine or end toxin release, and c) an inflammatory/immunologic host reaction to Brucella within the nervous system[7]. As brucellosis is usually a chronic infection, nervous system invasion can occur secondary to the persistence of intracellular microorganisms. Preexisting host immuno suppression is a significant risk factor, but the disease can also occur in healthy individuals. The development of Neurobrucellosis is also associated with age and prolonged time of infection[8].The patient in our case was a young healthy male with no associated Comorbity. The diagnostic criteria of Neurobrucellosis include (a) clinical findings compatible with Neurobrucellosis; (b) pleocytosis with predominant lymphocytes and elevated protein concentration in CSF; (c) positive results of either blood or bone marrow or CSF culture or positive serologic tests; (d)clinical improvement following antibiotic therapy against brucellosis; and (e)no other alternative diagnosis [6]. The patient had all of these: clinical findings and serologic evidence of brucellosis, lymphocytic pleocytosis with high protein in CSF, response to therapy, and no alternative explanation.

Sensor neural hearing loss although rare has long been reported in patients with Neurobrucellosis and if the disease is not treated early, permanent hearing loss can occur.[9] The finding can be overlooked if other features of Neurobrucellosis are not prominent. In our patient, the hearing loss was never evaluated properly and possible reasons were never dealt with earlier. Consequently, the patient had a residual hearing loss due to delay in diagnosis and treatment. Psychiatric symptoms are quite rare in Neurobrucellosis. The reported psychiatric manifestations usually seen in Brucella meningitis include depression, amnesia, agitation, personality

changes, euphoria, and psychosis among which depression is the most common.[10] Delirium may also occur inacute brucellosis. Our patient had psychosis of only 4-day duration that was controlled with a good dose of antipsychotic. Drugs may be needed in some cases to control acute psychosis, although the symptoms usually improve with treatment of brucellosis.[11]With aforesaid background we therefore draw aconclusion that the new-onset psychosis without fever with a history of hearing loss could suggest the possibility of chronic Neurobrucellosis.

Conclusion

In conclusion, this clinical case illuminates the complex and nuanced nature of Neurobrucellosis, highlighting its rare presentation as acute psychosis. The diagnostic journey encompassed a meticulous examination of neurological, psychiatric, and audiologically facets, ultimately converging on the unexpected yet definitive diagnosis of Neurobrucellosis. The patient's atypical clinical course, characterized by the absence of classical neurological symptoms and a conspicuous psychiatric presentation, underscores the importance of considering Neurobrucellosis in the differential diagnosis of acute psychosis, particularly when faced with puzzling clinical manifestations. Moreover, the case accentuates the interdisciplinary collaboration essential for unraveling such intricate medical puzzles. The integration of neurology, psychiatry and audiology expertise played a pivotal role in reaching a precise diagnosis and formulating an effective treatment strategy. The successful management, involving a combination of antimicrobial and psychotropic agents, further emphasizes the necessity of a comprehensive therapeutic approach tailored to the multifaceted nature of Neurobrucellosis. This case report contributes to the evolving understanding of the diverse clinical presentations of Neurobrucellosis and emphasizes the imperative for heightened clinical awareness, thorough investigation, and interdisciplinary cooperation in similar diagnostic challenges. As we navigate the intricate nexus between infectious diseases and psychiatric manifestations, this case serves as a compelling testament to the need for a holistic and collaborative approach in unraveling the complexities of Neurobrucellosis.

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