

Disseminated Tuberculosis with Ischemic Cerebral Stroke in Sudanese Female: A Case Report

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1. Abstract

1.1. Background

Disseminated tuberculosis is a hematogenous diffusion of Mycobacterium tuberculosis impacting the lungs, central nervous system, and lymph nodes. Stroke in tuberculosis occurs in patients, especially in advanced episodes and extreme cases.

1.2. Case Presentation

We disclose the case of a 65-year-old female Sudanese patient who had never been exposed to tuberculosis or trauma, exhibiting a clinical progression marked by a productive cough, dyspnoea on mild exertion, fatigue, marked loss of weight, loss of appetite together with dysarthria of 2 months duration, and a decline in consciousness, shown by a Glasgow coma score of 10/15. A brain scan revealed indications of cerebral ischemia.

1.3. Conclusion

This case displayed a rare manifestation of disseminated tuberculosis in a critically ill patient. Disseminated tuberculosis must be regarded as a potential cause of abrupt focal neurological impairments in the elderly. Prompt diagnosis and suitable administration are crucial for limiting passing away.

2. Introduction

Tuberculosis (TB) remains a significant global public health issue. Approximately one-third of the worldwide population is infected with the TB bacillus [1,2]. Data from the World Health Organization (WHO) proves that tuberculosis is among the top ten global causes of death. In 2016, 10.4 million individuals contracted tuberculosis, resulting in 1.7 million fatalities [3]. Sudan is experiencing a worsening humanitarian crisis, with 7.8 million individuals confronting severe issues about mental and physical health, including 1.6 million internally displaced persons and 1.1 million refugees. Resources are limited, economic output declined by two-thirds from 2017 to 2018, and the nation's healthcare system is inadequately prepared to address increasing and overlooked demands. Compounding the situation, Sudan has significant progress to make in attaining the Sustainable Development Goals (SDGs). The WHO and the Sudan Health Observatory within the federal Ministry of Health identify the primary communicable illnesses contributing to morbidity as malaria, tuberculosis, schistosomiasis, pneumonia, and diarrheal disorders [4]. It is anticipated that tuberculosis contributed to 1% of total inpatient mortality in Sudan in 2017 [5]. Disseminated tuberculosis affecting the central nervous system is the most catastrophic and lethal variant of tuberculosis. This presentation accounts for roughly 1% of tuberculosis cases and 6–10% of disseminated forms in people with impaired immunity [6]. Consequently, TB affecting the brain poses a considerable challenge during diagnosis. Stroke, a vascular

epidemic in developed nations, is a significant public health trouble with global implications. It has recently been identified as the second leading cause of mortality worldwide [7]. However, the literature is yet to provide data on the correlation between ischemic stroke and disseminated TB, particularly as an early symptom in Sudanese patients. This study attempts to present a case of ischemic stroke as the early manifestation of disseminated tuberculosis in a Sudanese patient.

3. Case Presentation

A 65-year-old Sudanese woman from White Nile State, Sudan, presented with a history of persistent productive cough, dyspnea on light exertion, exhaustion, significant weight loss, and loss of appetite, as well as dysarthria that lasted for two months. The patient experienced left-sided weakness that worsened over a single day five days prior to admission, along with frequent episodes of partial seizures and headaches. The patient has a history of migraines, with no known diabetes mellitus, hypertension, or smoking status. She had no exposure to a tuberculosis patient or any trauma. Upon evaluation, the patient appears apathetic, stuporous, and pale, without signs of jaundice or cyanosis. Cachexia, xerosis, scant scalp hair, and distended jugular veins. No discernible lymphadenopathy. Vital signs: Pulse rate 58 beats/minute, regular, weak, synchronous, no respiratory failure delay, normal peripheral pulses. Blood pressure 90/60 (pulsus paradoxus). Respiratory rate: 28 cycles/minute; Temperature: 37.8 °C. Cardiovascular system (CVS) examination, Jugular vein pressure (JVP) distended to around 12 cm. Apex located at the distant 7th intercostal space along the anterior clavicular line: no discernible left parasternal heave or second heart sound. Muffled first and second heart sounds, with no murmurs or other sounds. Neurological examination: Patient with Glasgow Coma Scale score of 10/15, exhibiting dysarthria. Symmetrical facial features. Left-sided hemiplegia, spasticity, increased reflexes with delayed relaxation (ankle), and upward plantar response. The right side is normal. Respiratory assessment: Unremarkable. Abdominal scrutiny: Unremarkable. In the studies, the CBC indicates there is a white cell count of $8.4 \times 10^9/L$ (neutrophils $2.3 \times 10^9/L$, lymphocytes $5.2 \times 10^9/L$, monocytes $0.8 \times 10^9/L$, and eosinophils $0.1 \times 10^9/L$). Hemoglobin: 9.4 g/dL, Mean Corpuscular Volume: 85.4 fL, Mean Corpuscular Hemoglobin: 29.4 pg, Mean Corpuscular Hemoglobin Concentration: 34.2 g/dL, Platelet Count: $120 \times 10^9/L$, and Erythrocyte Sedimentation Rate: 95 mm/h. C-reactive protein level: 1.0 mg/l, Renal function tests showed that creatinine was: 0.9 mg/dL and blood Urea was: 15 mg/dL. Liver function test and enzymes provide the following information: Total bilirubin: 1.2 mg/dL, direct bilirubin: 0.3 mg/dL, total protein: 7.7 g/dL, albumin: 4.1 g/dL, globulin level: 3.6 g/dL, Alanine aminotransferase (ALT): 6 U/L, aspartate aminotransferase (AST): 29 U/L, alkaline phosphatase

(ALP): 36 U/L. Random blood sugar: 99 mg/dl. Urinalysis: Normal. Lipid profile: Normal. Thyroid function test results indicated: Thyroid-stimulating hormone (TSH) > 110 mIU/L (0.4-4.3), T3: 0.3 ng/dL (0.8-1.6), T4: < 0.5 µg/dL (4.9-11.0). Viral screening: negative. Chest X-ray indicates a grossly enlarged heart shadow flask shape [Figure 1]. No focal pulmonary lesion. Electrocardiogram: decreased voltage with sinus bradycardia (Figure 2). An echocardiogram (ECHO) revealed: satisfactory left ventricular systolic performance at rest and massive pericardial effusion with signs of tamponade necessitating immediate pericardiocentesis. Analysis of pericardial fluid disclosed: a straw-colored appearance, protein level of 5.0 g/dL (<0.5), albumin level of 2.9 g/dL, glucose level of 70 mg/dL, and the Ziehl-Neelsen stain (ZN) staining showed no acid-fast bacteria (AFB) present. Cytology: 700 cells per cubic millimeter, primarily lymphocytes, with no aberrant cells observed. Qualitative PCR for Mycobacterium TB: Positive. Magnetic resonance imaging (MRI) of the brain with contrast: findings indicate a right frontoparietal ring-enhancing cystic lesion measuring 3x2.8x2.3 cm, accompanied by perifocal edema indicating the presence of tuberculoma (Figure 3). Apathy, stupor, xerosis, and sparse hair. Left-sided hemiplegia. Sinus bradycardia, hypotension, distended jugular venous pressure, displaced apex, and muffled heart sounds are all reported findings. Lymphocytosis, thrombocytopenia with normocytic normochromic anemia. Elevated ESR. PCR positive for Mycobacterium tuberculosis. Ring-enhancing lesion in the frontoparietal region (Tuberculoma). Pericardial effusion (cardiac tamponade) and Hypothyroidism constitute the primary trouble. The plan commenced with urgent pericardiocentesis, during which 600 ml of straw-colored fluid was extracted. Dexamethasone injection 4 mg was administered every 6 hours, Tegretol CR tablets 400 mg were given once, anti-tuberculous medication was prescribed, and thyroxine tablets 25 mcg were administered once, along with a proton pump inhibitor. Upon discharge, the patient regained full consciousness (GCS 15/15), demonstrated orientation to place, time, and person, and had normal speech and memory. Her vital signs are within normal limits; her power is grade 3 in both upper and lower limbs. The heart sound became discernible. Her erythrocyte sedimentation rate is 40.

4. Discussion

Cerebral tuberculosis is the least common clinical manifestation of disseminated tuberculosis in emerging economies. Identifying and concluding a timely diagnosis is exceedingly challenging due to the absence of specific clinical characteristics. These challenges are inherently linked to increased morbidity and mortality among those who experience them, as was the case in this instance [8]. Our patient exhibited miliary TB, with cerebral infarction as the primary presentation. The unusual thing about this case is that there is bradycardia along with cardiac tamponade. This is because tamponade usually leads to tachycardia, which is the heart's way of trying to get back to normal cardiac output. Several tests, including an MRI that made it easier to see the tuberculoma lesion, thyroid function tests that confirmed hypothyroidism, and an analysis of

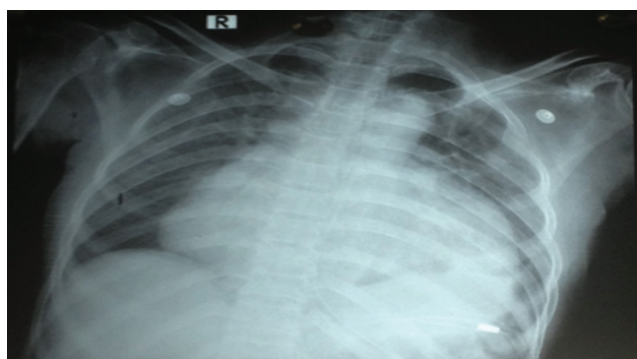


Figure 1: Chest X-ray showed cardiomegaly due to massive pericardial effusion.

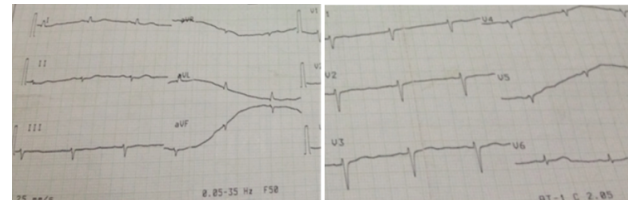


Figure 2: ECG showed low voltage ECG and sinus bradycardia.



Figure 3: MRI brain showed a right frontoparietal ring-enhancing cystic tumor measuring 3x2.8x2.3 cm (black arrow), accompanied by perifocal edema.

the pericardial fluid that showed higher protein and lower glucose levels, all supported the official diagnosis. The PCR analysis results for detecting Mycobacterium tuberculosis confirmed the presence of brain tuberculosis. While tuberculosis may be present upon admission, these cases generally do not progress till the consolidation phase of therapy [9]. CNS tuberculosis is correlated to boosted mortality rates, occurring in 1% to 5% of all tuberculosis patients [10]. CNS tuberculosis can be delineated as follows: 1) diffuse, shown by tuberculous meningitis; 2) localized, as seen in tuberculoma; 3) presenting as a tuberculous abscess; or 4) manifesting as extradural and intradural spinal infections. CNS tuberculosis may manifest non-specifically in the initial stages of the disease [11]. To our knowledge, there is no available data on disseminated tuberculosis with ischemic cerebral stroke in the Sudanese community, and no previous cases have been documented in our region.

5. Conclusion

We present a rare manifestation of disseminated tuberculosis with cerebral stroke in a critically ailing patient. This case highlights the necessity of acknowledging the diverse possible manifestations and clinical signs of Mycobacterium tuberculosis infection. Prompt diagnosis and suitable administration are crucial for minimizing fatality..

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