Cerebrovascular Accident Complicating Pulmonary TB: a Rare Co-existence

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Introduction

The coexistence of pulmonary tuberculosis (TB) and cerebrovascular accident (CVA) presents a unique and seldom-discussed clinical conundrum. While pulmonary TB is a well-known infectious disease, its association with CVA remains relatively obscure. Given the low prevalence of TB in the United States, degree of suspicion is often low in patients presenting with non-classic symptoms, and even less with patients presenting with CNS symptoms

Case description

A previously healthy 25-year-old male presented to the Emergency Department with sudden onset right-sided numbness and weakness, alongside concurrent chest pain and cough. Notably, the patient disclosed being a recent immigrant from South America. A comprehensive neurological assessment and imaging studies were promptly initiated. Initial Computed Tomography Angiography (CTA) of head and neck, and Magnetic Resonance Imaging (MRI) brain were negative. The neurology consult determined that the patient was not a candidate for thrombolytics. He was started on dual antiplatelet therapy and high-intensity statins. Due to the persistent cough, a chest X-ray was performed, revealing abnormalities. Subsequently, a Computed Tomography Angiography (CTA) of the chest was conducted, which demonstrated bilateral pulmonary infiltrates and cavitation in the left upper lobe, suggestive of Tuberculosis (Figure 1). Acid-Fast Bacilli (AFB) smear and PCR (Polymerase Chain Reaction) were collected and turned positive for Mycobacterium TB Complex. Treatment with a combination of anti-TB medications was initiated, and the State Health Department was promptly notified. Patient was placed in isolation.

Despite treatment initiation, the patient's neurological condition deteriorated with worsening right-sided weakness, prompting repeat imaging. CT head demonstrated decreased attenuation in the left frontal subcortical white matter suggesting subacute infarcts, unusual for the patient's age (Figure 2). The echocardiogram ruled out a Patent Foremen Ovale (PFO). Patient was started on therapeutic Enoxaparin. Repeat MRI brain, showed diffuse abnormal signal in the left parietal lobe associated with cerebral edema, along with extensive enhancement involving the cortical gray matter and meninges throughout the high left parietal lobe (Figure 3). TB meningitis was suspected, prompting initiation of high-dose steroids and Levofloxacin. Cerebrospinal fluid analysis did not definitively confirm tuberculosis, though it could not be entirely ruled out. The MRI findings also suggest the possibility of an acute inflammatory response to the TB infection. Over the ensuing days, the patient exhibited gradual improvement in motor function.

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Figures

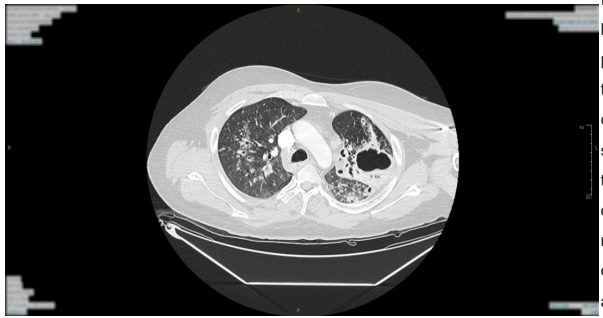

Figure 1

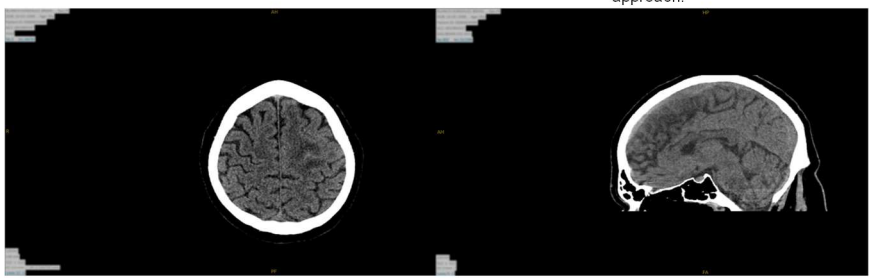

Figure 2

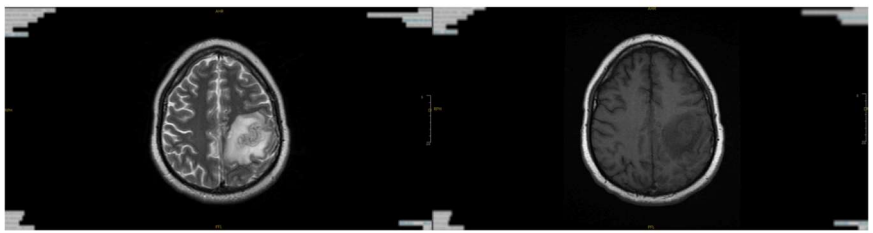

Figure 3

Discussion

Ischemic stroke as a presentation of pulmonary tuberculosis is exceptionally rare, with only a limited number of cases documented in the medical literature. The relationship between active pulmonary tuberculosis and ischemic stroke has not been established. A recent study revealed that most patients diagnosed with tuberculosis-related ischemic stroke (TBRIS) experience this complication within three months of being diagnosed with pulmonary tuberculosis. This timing suggests a correlation with the heightened inflammatory state characteristic of active pulmonary tuberculosis. TBRIS not only results in physical disability but also correlates with more severe outcomes in patients with pulmonary TB. Hence, understanding the pathogenesis of TBRIS is not just beneficial but imperative for both preventing its occurrence and improving the prognosis of affected individuals. Furthermore, our case underscores the importance of a comprehensive approach to differential diagnosis, highlighting the importance of epidemiology in clinical cases, for it can be used to understand the pathogenesis of diseases, improve diagnostic accuracy, help the patient to reduce risk factors and the physician to choose the correct therapeutic approach.

Conclusion

The convergence of pulmonary tuberculosis and ischemic stroke presents a complex and relatively rare clinical scenario. Understanding the pathogenesis of tuberculosis-related ischemic stroke is crucial for both prevention and improving patient prognosis

References

1. Wei Y, Tang S, Xie Z, et al. Pulmonary Tuberculosis-Related Ischemic Stroke: A Retrospective Case Control Study. *J Inflamm Res*. 2022;15:4239-4249. Published 2022 Jul 26. doi:10.2147/JIR.S368183
2. Briones-Claudett KH, Briones-Claudett MH, Briones Zamora KH, Nieves Velez RD, Martinez Alvarez ME, Grunauer Andrade M. Ischemic Stroke and Disseminated Tuberculosis in Intensive Care: A Case Report. *Am J Case Rep*. 2020;21:e920410. Published 2020 Jun 5. doi:10.12659/AJCR.920410
3. Hwang JH, Lee KM, Park JE, et al. Atypical Cerebral Manifestations of Disseminated *Mycobacterium tuberculosis*. *Front Neurol*. 2017;8:462. Published 2017 Sep 21. doi:10.3389/fneur.2017.00462
4. Koenig SP, Furin J. Update in Tuberculosis/Pulmonary Infections 2015. *Am J Respir Crit Care Med*. 2016;194(2):142-146. doi:10.1164/rccm.201601-0129UP
5. Zeng J, Zhang X, Hua W, et al. Diffuse large B-cell lymphoma with pulmonary and cerebral involvement: A case report. *Mol Clin Oncol*. 2018;9(3):264-268. doi:10.3892/mco.2018.1673
6. Gama L, Duarte J, Martins I, Santos E Silva A, Rita H. Tuberculosis: Cliché or Outsider?. *Cureus*. 2024;16(2):e53956. Published 2024 Feb 10. doi:10.7759/cureus.53956
7. Cimmino MA, Hazes JM. Introduction: Value of epidemiological research for clinical practice. *Best Pract Res Clin Rheumatol*. 2002;16(5):vii-xii. doi:10.1053/berh.2002.0277