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Development of Disseminated Tuberculosis in a Non-Immunocompromised Patient Following COVID-19 Infection: A Case Report

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Abstract

Background:
The COVID-19 pandemic has been associated with various complications and has altered patterns of disease presentation. COVID-19 is known to affect the immune system. We report a case of disseminated tuberculosis (TB) in a previously non-immunocompromised patient with a history of COVID-19 infection.

Case Presentation:
A 25-year-old woman with a history of mild COVID-19 infection three months earlier developed progressive back pain followed by worsening headache. She was eventually admitted with decreased level of consciousness, delirium, and focal neurological deficits. Further evaluation confirmed disseminated tuberculosis, including miliary pulmonary TB, TB meningitis, and TB spondylodiscitis. The patient showed gradual clinical and neurological improvement following anti-tuberculosis therapy.

Conclusions:
This case suggests a possible association between COVID-19 infection and subsequent development of disseminated tuberculosis, even in immunocompetent individuals.

Keywords: COVID-19; tuberculosis; disseminated TB; tuberculosis

Background

Tuberculosis is a contagious disease and remains one of the top ten causes of death worldwide, representing the leading cause of mortality from a single infectious agent. In 2019, approximately 10 million people developed TB, and 1.4 million deaths were reported globally [1].

Mycobacterium tuberculosis is transmitted via airborne droplets. After inhalation, droplet nuclei reach the bronchial tree and implant in the respiratory bronchioles or alveoli. The establishment of infection depends on both host immune responses and microbial factors [2,3]. Miliary tuberculosis is a form of disseminated TB resulting from lymphatic and hematogenous spread of the organism and is associated with high mortality if diagnosis and treatment are delayed [4]. In low-incidence countries, delayed recognition of miliary TB significantly increases mortality rates [5].

The COVID-19 pandemic has influenced the incidence and clinical presentation of many diseases. COVID-19 can alter immune function, and TB reactivation has been attributed, in part, to T-cell depletion following SARS-CoV-2 infection [6–8]. Herein, we describe a case of disseminated TB in a young, non-immunocompromised patient with a recent history of mild COVID-19 infection.

Case Presentation

A 25-year-old Iranian woman was admitted on June 2, 2021, with complaints of severe headache and decreased level of consciousness. She reported a two-month history of progressive headache, predominantly involving the frontal and parietal regions. During this period, she had multiple outpatient visits and was treated symptomatically with analgesics.

The patient had no known immunodeficiency or underlying medical conditions. She had experienced a mild COVID-19 infection three months prior, characterized by flu-like symptoms and transient anosmia and ageusia. There was no pulmonary involvement, and hospitalization was not required. Following recovery from COVID-19, she developed thoracic back pain, which persisted until admission.

Two weeks prior to hospitalization, her headache worsened significantly. On presentation, she complained of severe headache, nausea, vomiting, diplopia, back pain, urinary incontinence, and decreased consciousness. One day before admission, she developed acute lower-limb weakness and became unable to stand independently. She denied cough or respiratory symptoms and had no family history of tuberculosis. She reported an unintentional weight loss of 5 kg over the preceding month. She had moved from a rural area to an urban setting 22 years earlier.

On physical examination, the patient was lethargic, confused, and delirious. She was afebrile, with signs of meningeal irritation. Muscle strength in the lower limbs was reduced (4/5). Nystagmus was noted on ophthalmologic examination.

Brain magnetic resonance imaging (MRI) revealed diffuse leptomeningeal enhancement with mild hydrocephalus. A focal area of restricted diffusion without enhancement was observed in the splenium of the corpus callosum (Figure 1).

Spinal MRI demonstrated abnormal signal intensity and enhancement involving the T8–T9 intervertebral disc space, adjacent vertebral bodies, posterior elements, and a portion of the T10 vertebral body, along with prevertebral soft-tissue edema (Figure 2).

Chest computed tomography showed diffusely distributed pulmonary nodules consistent with miliary tuberculosis. Multiple lymphadenopathies with partial calcification were noted in the mediastinal and left axillary regions (Figure 3).

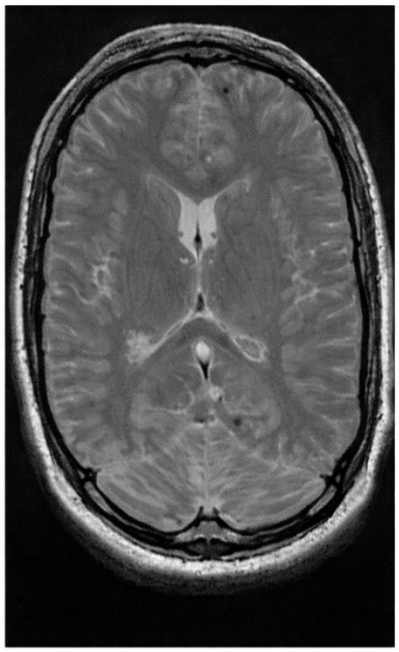

Figure 1. Brain MRI

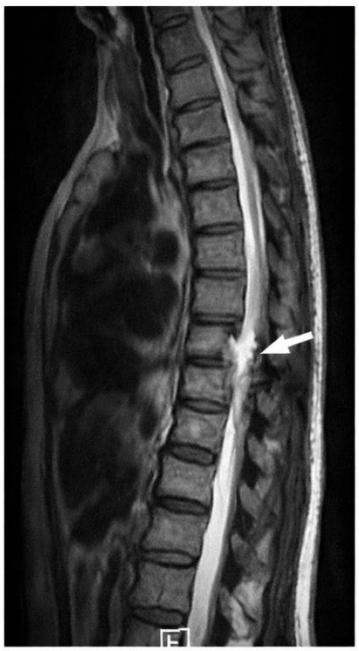

Figure 2. Spinal MRI

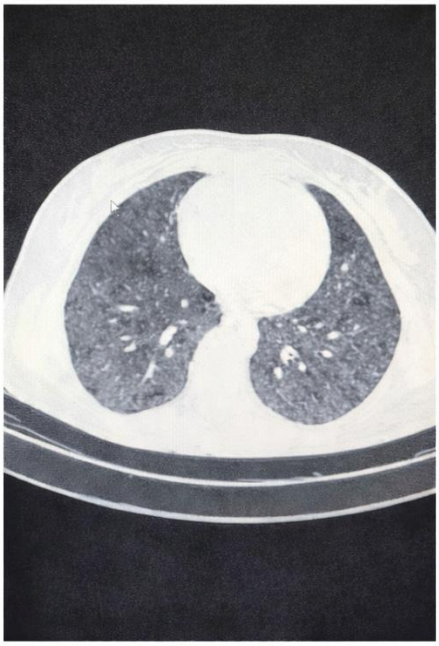

Figure 3. Chest CT

Cerebrospinal fluid (CSF) analysis revealed lymphocytic pleocytosis, elevated protein levels, and decreased glucose concentration (Table 1). Polymerase chain reaction testing of CSF detected Mycobacterium tuberculosis, with no evidence of rifampin resistance on Xpert MTB/RIF assay. PCR testing for CMV, EBV, HSV-1, HSV-2, and VZV was negative.

CSF Analysis:

Table 1. Analysis of Cerebrospinal Fluid (CSF)

Variable	Day 1	Day 8
WBC (cells/µL)	50	2
RBC (cells/µL)	3	2
Neutrophils (%)	40	–
Lymphocytes (%)	60	–
Glucose (mmol/L, CSF/Serum)	39/109	65/120
LDH (U/L)	149	68
Protein (g/L)	130	19.5

Laboratory investigations showed a white blood cell count of 5,900 cells/mm³ (90% neutrophils, 8% lymphocytes), C-reactive protein of 21 mg/dL, and erythrocyte sedimentation rate of 31 mm/h. HIV serology was negative. Liver function tests demonstrated mild elevations (AST 57 U/L, ALT 53 U/L, ALP 264 U/L). SARS-CoV-2 RT-PCR from nasopharyngeal and throat swabs was negative.

Table 2. Liver Function Tests During Treatment

Day of Treatment	1	3	5	7	9	11	13	15	17	19
AST (U/L)	57	65	68	76	111	137	135	128	125	84
ALT (U/L)	53	53	80	115	85	50	69	52	82	50

The patient was started on standard anti-tuberculosis therapy, including isoniazid (300 mg), rifampin (600 mg), pyrazinamide (1,200 mg), and ethambutol (825 mg) once daily. Due to significant neurological involvement, adjunctive corticosteroid therapy with prednisolone (80 mg/day) was initiated and gradually tapered over six weeks.

Follow-up CSF analysis on day eight of treatment showed marked improvement. Neurological status progressively improved, with resolution of delirium by the third week and recovery of lower-limb strength by the fourth week. Despite radiologic evidence of T8–T9 spondylodiscitis, there was no spinal cord compression, and surgical intervention was not required.

Transient elevation of liver enzymes occurred early during treatment but remained below five times the upper limit of normal and resolved without discontinuation of therapy (Table 2). The patient was discharged on day 18 in good general condition and was prescribed a 12-month course of anti-tuberculosis therapy with outpatient follow-up.

Discussion

This case describes disseminated tuberculosis involving the lungs, central nervous system, and spine in a young, previously healthy woman with no known immunodeficiency, occurring shortly after recovery from a mild COVID-19 infection. The unusual extent of disease and the absence of traditional risk factors raise the possibility of a link between recent SARS-CoV-2 infection and reactivation or progression of tuberculosis.

Effective control of *Mycobacterium tuberculosis* relies primarily on cell-mediated immunity, particularly $CD4^+$ T lymphocytes and macrophage activation [9]. Several studies have demonstrated that COVID-19 is associated with lymphopenia, functional exhaustion of T cells, and dysregulation of cytokine responses, even in patients with non-severe disease [7,10]. Although immune cell counts may normalize after clinical recovery, transient immune dysfunction may persist and predispose susceptible individuals to opportunistic or latent infections.

Tuberculosis reactivation following viral infections has been described previously, including after influenza and measles. Emerging evidence suggests that COVID-19 may similarly contribute to TB activation or dissemination [6–8]. Most reported cases involve patients with severe COVID-19 or additional comorbidities; however, cases following mild infection, as observed in our patient, have also been reported [6]. This suggests that even limited SARS-CoV-2 infection may be sufficient to alter host immunity in a way that facilitates TB progression.

The clinical presentation in this case was notable for predominant neurological symptoms, including prolonged headache, altered mental status, and focal neurological deficits. These features initially led to diagnostic delay, as early symptoms were treated symptomatically and

could have been misattributed to post-COVID manifestations. In TB-endemic regions, persistent constitutional or neurological symptoms following COVID-19 infection should prompt thorough evaluation for tuberculosis, particularly when symptoms progress or fail to resolve.

Radiologic findings were consistent with disseminated disease, including miliary pulmonary nodules, leptomeningeal enhancement, and thoracic spondylodiscitis. The diagnosis of TB meningitis was supported by CSF findings and confirmed by PCR. Early initiation of standard anti-tuberculosis therapy, combined with adjunctive corticosteroids, resulted in gradual neurological and clinical improvement, highlighting the importance of timely diagnosis and treatment in preventing irreversible complications.

Given the extent of extrapulmonary involvement, a prolonged duration of anti-tuberculosis therapy was selected in accordance with international guidelines [20]. The patient tolerated treatment well, with only transient liver enzyme elevation that resolved without interruption of therapy.

Conclusions

This case highlights the potential for disseminated tuberculosis to develop in immunocompetent individuals following COVID-19 infection. Recent SARS-CoV-2 infection may represent a contributing factor to immune dysregulation and TB reactivation, even in patients with mild disease. Clinicians should maintain a high index of suspicion for tuberculosis in patients presenting with persistent or unexplained symptoms after COVID-19, particularly in TB-endemic settings. Early recognition and prompt treatment are essential to improve outcomes in disseminated TB.

Consent for Publication
Written informed consent was obtained from the patient for publication of this case report and any accompanying images. The patient has provided permission to use medical images and clinical data for educational and scientific purposes.

Competing Interests
The authors declare that they have no competing interests.

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