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Schizophrenia Secondary to COVID-19 and Tuberculosis: A Case Series of Post-Infection Schizophrenia

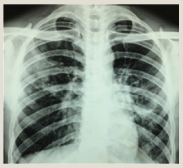
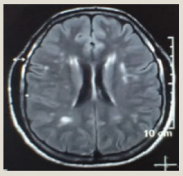
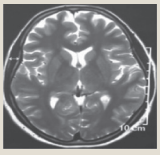
Dear Editor,

Etiopathogenesis of schizophrenia (SZ) is multifactorial; it includes genetic vulnerability and perinatal infection leading to abnormal neurodevelopment.[1] Multiple infectious agents have been implicated in the etiology of SZ. Maternal infection with Herpex Simplex Virus (HSV) 1&2, Toxoplasma gondii, Influenza virus, and Rubella are associated with an increased risk of development of psychosis in the offspring.[2] Also, childhood viral infections, especially with Mumps, Cytomegalovirus, and Coxsackie B5 virus, may cause the development of SZ or other non-affective psychosis in adult life.[2] In the adult, infection with HSV-1, Influenza, Borna disease, Coronavirus, Measles, Hepatitis C, Toxoplasmosis, and Lyme disease are associated with SZ and psychosis.[2]

Here we describe two cases of SZ presumed to be post-infection. Both cases were diagnosed by two psychiatrists using the *International Classification of Disease*—10th edition (ICD-10) Classification of Mental and Behavioural disorders—clinical description and diagnostic guidelines. All tests were done from accredited laboratories. Written informed consent was obtained from the patients for this publication.

TABLE 1.

Clinical and Investigation Profile of the Cases.

Cases	Demographic Profile	History of Present Illness	Mental Status Examination	Investigations
A	28 years, Male, Unmarried	Duration: 1.5 months. Acute onset. Unusual suspiciousness towards family and friends, muttering to self, cough, and mild fever.	Delusion of persecution, auditory hallucination of 2nd and 3rd person, thought broadcasting, restricted affect, poor judgment, and impaired insight.	Sputum for acid-fast bacilli 3+  Chest X-ray: Koch's focus in the left upper lobe of the lung.  MRI brain: white matter ischemic changes.
B	40 years, Male, Married	Duration: 2 months. Onset: gradual. Suspiciousness towards family members and co-workers about their harmful intent, neighbors following him, listening to derogatory voices, irritability, and decreased sleep. Two days before the onset of symptoms patient had a history of fever, running nose, and cough. He had no breathing difficulties. He was diagnosed to have mild COVID and advised for home isolation. His fever and respiratory symptoms subsided within ten days.	Delusion of persecution, delusion of reference, auditory hallucination of 2nd person, thought echo, fearful affect, judgement and insight poor.	RTPCR for COVID-19 was positive. SpO2 96% at room air.  MRI Brain: Normal

Demographic details, history of present illness (HOPI), mental status examination (MSE), and investigations of our cases are described in **Table 1**. Both of our cases had no family history or past history of mental illness and no history of using any psychoactive substance. Case A (**Table 1**) had a history of pulmonary tuberculosis one year back, for which he was successfully and adequately treated, but within one year, he developed intermittent mild fever, cough, and behavioral change like irritability, odd behavior, and decreased interaction with people. Initially, for the first few weeks of fever and cough, he took some homemade preparation and visited a local faith healer for behavioral change. When he did not improve, he visited a local general physician and was treated with Amoxycillin, Paracetamol, and Risperidone 2 mg for 1–2 weeks. As symptoms did not improve but rather worsened, he was referred to our institute with 1.5 months duration of symptoms of SZ (**Table 1**), cough, and intermittent mild fever in the background. On working up, we found a relapse of Pulmonary Tuberculosis. Case A responded to T. Olanzapine (15 mg), with symptom improvement in 4 weeks.

Case B (**Table 1**) attended us after his 2 weeks of home isolation. We initially started him on T. Risperidone 2 mg and followed up with him every 2 weeks. On the first follow-up visit, there was an improvement in his sleep and affective symptoms, but no improvement was seen in other psychotic symptoms. We increased the dose to 4 mg, and on the second visit, there was a weakening in his delusions and a reduction in the frequency of auditory hallucinations. On a further increase to 5 mg by the end of 6 weeks, all symptoms showed complete improvement.

On neurological examination, neither patients had focal neurological or meningeal signs. Both maintained the improvement with the same treatment on a further 2-month follow-up, with self-reported satisfactory psychosocial functioning.

These cases are unique in that most psychosis cases reported in association with tuberculosis are either due to neurotuberculosis[3] or drug-induced,[4] to our best knowledge. In Case A, there is no evidence of neurotuberculosis as well as he was not on any antitubercular drug at the time of diagnosis. COVID-19-associated psychosis is reported to be acute onset and brief in nature,[5] occurring weeks to months after the infection,[6] or drug-induced.[7] In Case B, psychosis was of gradual onset, persistent, and started concomitantly with COVID-19 in a drug-naive person.

Mycobacterium tuberculosis infection and SARS-Cov2 infection are both associated with a rise in proinflammatory cytokines.[5, 8] The inflammatory condition may also result in white matter ischemic changes in the brain, mediated by microglial activation and beta-amyloid accumulation,[9] which may be the reason for MRI brain

changes seen in Case A, or it may be an incidental finding. A high level of proinflammatory cytokines has been found in the body fluid of patients with SZ.[10] It is hypothesized that an inflammatory state may result in abnormal microglial triggering and release of cytokines that, in turn, create an imbalance in dopaminergic, serotonergic, and glutamate neurotransmission, precipitating psychosis in vulnerable individuals.[10] Similar pathogenesis might have been an underlying mechanism in our cases. Although the chance of coincidental independent pathology cannot be impossible in our cases, SZ started in close proximation with COVID-19 and tuberculosis, and given the past evidence of association of psychosis with similar infections, sharing of explainable common pathomechanism and scarcity of nullifying evidence regarding in-between association, argues for its secondary nature to COVID-19 and tuberculosis.[11]

From our above cases, we can opine that infectious causes should always be considered an etiological factor while evaluating patients with SZ, especially in an endemic region. COVID-19 and mycobacterium tuberculosis may also cause full-blown SZ in adults.

Declaration of Conflicting Interests

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