CASE REPORT

Bali Medical Journal (*Bali MedJ*) 2021, Volume 10, Number 2: 673-676
P-ISSN.2089-1180, E-ISSN: 2302-2914

Bali Medical Journal
www.BaliMedicalJournal.org
Published by Bali Medical Journal

Meningoencephalitis due to SARS-CoV-2 and tuberculosis co-infection: a case report from Indonesia

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Received: 2021-02-02
Accepted: 2021-07-20
Published: 2021-07-31

ABSTRACT

Introduction: The novel coronavirus which firstly detected in December 2019 in Wuhan, China, has been known to cause neurological dysfunction either by directly or indirectly infecting the brain.
Case: We are reporting a case of meningoencephalitis due to co-infection of M. tuberculosis and SARS-CoV-2 in a hospital in Indonesia. A 26-year-old gentleman working as a courier in a Sars-Cov-2 red zone without adequate protection complained frequent headaches since a month to admission. M. tuberculosis was detected on very low level by GenXpert® and rapid test for SARS-CoV-2 was nonreactive. Repeated GenXpert® showed detected M. tuberculosis with undetected rifampicin resistance. Subsequent CSF SARS-CoV-2 PCR was positive although the oropharyngeal swab was negative.
Conclusion: The report of pulmonary co-infection of TB and SARS-COV-2 has been published, however, to our best knowledge there has been no report of neurological co-infection to date. We are reporting the report of CNS co-infection from our country.

Keywords: COVID-19, Meningoencephalitis, Tuberculosis, SARS-CoV-2.
Cite This Article: Retnaningsih., Purwaamijaya, B., Tugasworo, D., Andhitara, Y., Ardhini, R., Kurnianto, A., Afany, N., Bunyamin, J., Utami, F.S., Sogata, I.A., Hairuzaman. 2021. Meningoencephalitis due to SARS-CoV-2 and tuberculosis co-infection: a case report from Indonesia. *Bali Medical Journal* 10(2): 673-676. DOI: 10.15562/bmj.v10i2.2235

INTRODUCTION

The novel coronavirus which firstly detected in December 2019 in Wuhan, China, has been emerging as a global health emergency.[1] The virus has been known to cause neurological dysfunction either by directly infecting the brain or indirectly as a result of strong activation from systemic immunological reaction.[2] Human brain cells express the angiotensin-converting enzyme 2 (ACE2) protein in its surface, a protein involved in blood pressure regulation and a receptor for the virus to enter and infect the cells.[3] ACE2 is also found in the endothelial cells constructing the blood vessels. Infection of the endothelial cells allows the virus to transport from the respiratory system to blood and crosses the blood-brain barrier to the brain, in which the virus replicates and subsequently causing neurological dysfunction.[4,5] ACE2 expression is mainly found in both neuron and glial cells of the brainstem and regions responsible for cardiovascular function regulation, ie. subfornix, paraventricular nuclei, solitary tract nuclei, and rostral ventrolateral medulla regions.[6,7] We are reporting a case of meningoencephalitis due to co-infection of M. tuberculosis and SARS-CoV-2 in a hospital in Indonesia.

CASE REPORT

A 26-year-old gentleman working as a courier in a Sars-Cov-2 red zone without adequate protection complained frequent headache since a month to admission which resolves with over-the-counter drug and rest. Seven days prior to admission, he developed continuous fever, chilling, and shortness of breath, although decreased appetite and coughing were denied. Two days prior to admission his speech started to become disoriented and tended to be drowsy. Past history of tuberculosis, diabetes, hypertension, head injury, and blood transfusion were denied while alcohol consumption was moderate. He was initially admitted in a small hospital on 16th April 2020 for being disoriented and drowsy with initial GCS $E_3M_6V_4$, BP 110/60 mmHg, HR 86 bpm, RR 27 pm, temperature 38°C, and oxygen saturation 98%. As his condition deteriorated (GCS $E_3M_5V_4$), he was then transferred to a secondary-care hospital on the same day. Initial lab results showed leukocytosis (14,970/mm^3) with left shifting and increased absolute neutrophil counts (12,790) and he was admitted in the isolation ward. Chest X-ray can be seen on figure 1.

In day 4, he had further decreased consciousness (GCS $E_2M_4V_2$), developed hypotension (BP 80/60 mmHg), and appeared dyspneic. In day 5, he developed acute respiratory failure (RR 38 pm), pre-shock (BP 70/50 mmHg), and haematemesis which required prompt intubation, fluid resuscitation, and ICU transfer. Blood tests revealed respiratory alkalosis (pH 7.47, $PaCO_2$ 28 mmHg, PaO_2 67 mmHg, HCO_3 23