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LETTER TO EDITOR

Reply letter to: Rule out alternative entities before diagnosing post-COVID psychosis

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Dear editor,

Thank you for your valuable input and comments to our case report. In the letter to the editor entitled 'Rule out alternative entities before diagnosing post-COVID-19 psychosis'[1] addressed to our article entitled 'Covid-19, not your normal flu: A case report on Covid-19 psychosis and mania in a Malaysian Hospital',[2] the author has raised several points worthy of discussion.

In our case report, we considered various factors, particularly the administration of dexamethasone, for our patient during his COVID-19 admission, which could have led to psychosis. The incidence rate of corticosteroid-induced psychosis ranges widely from 1.8% to 62.0%. This is largely attributed to the multiple variabilities in clinical factors, but the most important risk factor is the dosage of oral prednisone prescribed.[3] According to the Boston Collaborative Drug Surveillance Program, 4.6% of patients receiving doses of more than 40 mg/day and 18.4% of those receiving doses of more than 80 mg/day present with psychiatric symptoms.[3] On this note, Mr K did not receive more than 24 mg/day of oral prednisone.[2] Nevertheless, even at lower doses, psychosis could precipitate in some patients.[3,4]

Dexamethasone has a mean terminal half-life of 4 h.[5] Although it is uncommon that a psychotic episode starts 7 days after dexamethasone is completely tapered off in patients with a normal renal profile, there is still a small possibility.[3] Other risk factors include female sex, previous diagnosis or family history of mental illness and prior incidence of corticosteroid-induced psychiatric effects, which were not evident in Mr K.[4] Taking all these into consideration, the dose and duration of dexamethasone treatment during COVID-19 infection could not have contributed to psychosis in our patient.

During the first 2 years of the COVID-19 pandemic, the incidence of psychosis increased significantly, which led the authors to consider it a complication of COVID-19. This is supported by the fact that several authors have presented similar cases of patients with no personal pathological psychiatric history who developed psychiatric disorders after COVID-19 infection. In a study conducted by Oxford University, COVID-19 psychiatric complications accounted for 18.1% of 62,000 hospitalised patients within 14–90 days of moderate or severe COVID-19 infection.[6] Although this emerging psychological phenomenon has been linked to the novel viral infection, I agree that further clinical studies should be conducted to exclude other possibilities. However, with our limited investigation, COVID-19 could not be ruled out as a cause of our patient's psychosis.

Our study provided limited information regarding further pharmacological therapy for COVID-19 in our patient. During his admission, Mr K received antibiotics and virostatics to cover for any occult infection, as meningoencephalitis initially ranked high on the list of suspected diseases.[2]

The specific magnetic resonance imaging modalities used were not included in his report and hence were not obtained for this study. Nevertheless, as previously mentioned in the case report, his bilateral frontal lacunar infarcts was not a likely contributing factor of psychosis.[2]

Regretfully, further mandatory investigations were not conducted. I agree that cerebrospinal fluid investigations should have included tests for cytokines or chemokines that may further confirm the suspicions of the neuroinvasive qualities of COVID-19. In addition, magnetic resonance venography, cerebrospinal fluid antibody evaluation and electroencephalography are warranted to rule out other alternative entities.[1]

In conclusion, I agree that our case report has its limitations, and the transient disorder should not be dismissed but deserves further laboratory investigations to rule out other likely causes before reaching a diagnosis of COVID-19 psychosis.

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Conflicts of interest
Nil

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