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Thalassemic Child Presenting with Anosmia due to COVID-19

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To the Editor: Novel coronavirus (SARS-CoV-2) infection (COVID-19) has spread rapidly in Indonesia. It might also impact on patients with hemoglobin disorders, such as thalassemia [1]. Indonesia has one of the highest prevalence of thalassemia globally [2].

A 17-y-old girl with transfusion-dependent thalassemia (beta-thalassemia), presented with acute loss of sense of smell (anosmia) and diminished sense of taste (ageusia) since 8 d prior to admission, accompanied by 2 d history of sneezing. History inquiry revealed that she had contact with her father, who was positive for COVID-19. She was admitted to hospital due to pale appearance since 7 d prior to admission. Three days after admission, she also complained mild-moderate muscle aches, which were relieved by using Paracetamol. Lung examination showed normal result.

Laboratory studies showed low hemoglobin level (7.5 g/dl), normal white blood cell count ($9.4 \times 10^3/\mu\text{L}$), with 54.9% neutrophils and 34.2% lymphocytes, prothrombin time prolongation (PT 14.5 s; normal APTT 31.6 s), hyperuricemia (6.3 mg/dl), and slight increase in C-reactive protein level (6 mg/dl). Ferritin level was 980.51 ng/ml, accompanied by elevated transferrin saturation (88%). Oropharyngeal swab tested positive for SARS-CoV-2. Chest X-ray result was normal. Supportive and therapeutic care such as blood transfusion, antibiotic Azithromycin 500 mg/q24h and antiviral Oseltamivir 75 mg/q12h were given. Deferiprone has still been given, considering her good condition.

Anosmia and ageusia might become possible symptoms of COVID-19 in pediatric population with transfusion-dependent

thalassemia, as reported in adult cases [3]. Without full objective assessment and nasoendoscopy, precise pathomechanism is still hard to determine. The possible explanation is post-viral syndrome with direct infection of the olfactory mucous and impairment of olfactory sensory neurons [4].

Erythrocyte is strongly hypothesized for its involvement in COVID-19 pathophysiology, by which SARS-CoV-2 might attack beta chain of hemoglobin. Its turnover will be reduced, followed by cell lysis and development of hemolytic anemia [5]. This condition is interesting, because beta globin chain defect in beta-thalassemia might potentially interrupt the impact of SARS-CoV-2 infection. Therefore, beta-thalassemia patients would not have same risk of COVID-19 as other patients, considering its reduced severity. More studies are needed to have a better understanding of SARS-CoV-2 infection in pediatric population with beta-thalassemia.

Compliance with Ethical Standards

Conflict of Interest None.

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