

Short Communication

Glucose-6-phosphate Dehydrogenase (G6PD) Deficiency and its Relation to Covid-19

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Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most common enzymopathy worldwide. It is prevalent in approximately 400 million people in East and Southeast Asia and central Africa [1, 2]. G6PD is an enzyme with X-linked recessive inheritance. Its level of activity varies among individuals and due to various drugs and foods. The level of enzyme activity decreases with aging. Clinically, men develop conditions, while women are carriers. G6PD deficiency is classified by the level of enzyme activity, and patients may suffer from mild/moderate G6PD deficiency. In the most severe type, the level of enzyme activity is below 10% [3]. It catalyzes the first and rate-limiting reaction step in the hexose monophosphate (HMP) pathway G6PD and provides the conversion of NADP to NADPH

(nicotinamide adenine dinucleotide phosphate). G6PD deficiency results in a lack of NADPH production. NADPH plays an important role in maintaining the integrity of the cell membrane in erythrocytes [4] and protects the erythrocytes against oxidative stress. Its deficiency may result in hemolysis of different severity and endothelial dysfunction in different tissues due to exposure to various factors. G6PD is also known to be essential in preventing opacification of the eye and for the development of cells of the lens. Studies on different age groups have found a strong correlation between G6PD deficiency in erythrocyte cells and cataract formation [5, 6]. One of the factors in the etiology of neonatal jaundice is G6PD deficiency [7]. G6PD deficiency has been found to vary from 1.5% to 35% in infants with jaundice [8].

G6PD activity changes depending on nutrition, hormones, and especially NADPH concentration [9]. The level of enzyme activity may also change due to various viral and bacterial causes. G6PD deficiency have been detected in patients with HIV, hepatitis viruses (A, B and E), and cytomegalovirus [10, 11]. It has been observed that some drugs activate and some inhibit the enzyme in some individuals with G6PD deficiency [12, 13].

Due to the Covid-19 pandemic, mortality rates have been increasing worldwide day by day. According to a meta-analysis, high levels of patient monitoring at ICU and mortality in men due to Covid-19 indicate the common enzyme deficiency with X-linked recessive inheritance [16]. Previous studies have suggested that oxidative stress contributes to the pathogenesis of severe SARS-CoV-2 infection [14], and researchers have found that cells with G6PD deficiency are more susceptible to infection [15]. Therefore, G6PD deficiency can cause vulnerability to SARS-CoV-2, while the treatment with HCQ, steroids, antibiotics, and antiemetics may also affect the level of G6PD enzyme activity positively or negatively. Drugs with negative effect will cause a decrease in NADPH levels, increasing the severity of oxidative stress, resulting in increased mortality and morbidity.

COVID-19 includes clinical presentations of various severities ranging from death due to severe acute respiratory failure to simple myalgia. There is no solid evidence on the cause of this broad range of clinical conditions. Based on the observations during the monitoring of patients in ICU that

1. A high number of individuals of the

same family had a severe condition and/or resulted in death;

2. Individuals or their first-degree relatives who had an indication for ICU hospitalization or resulted in mortality had a history of cataract;
3. Men have more severe clinical presentations and a higher mortality rate;
4. Individuals or their children admitted to ICU had a history of neonatal jaundice;
5. There is a partial coherence between the world map showing the regions of G6PD deficiency and the map showing mortality rates due to Covid-19 (Since the non-coherent countries are at low socioeconomic levels, reliability of their data should be considered);
6. Monitoring of the patients in ICU showed a moderate methemoglobinemia and hyperbilirubinemia in arterial blood gas; more studies analyzing the genetic basis of individual response to coronavirus infections are also warranted [17]. We believe that the patients with severe and complicated clinical presentations may have G6PD deficiency, and the patients with G6PD deficiency will have a more severe course of disease.

We are of the opinion that it will have a worldwide impact on mortality and morbidity to screen the patients with the above-mentioned clinical presentations for the history of G6PD deficiency and to determine their treatment and diet accordingly, as well as to carry out studies on it using multi-centered patient data.

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