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Letter to Editor

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# Prevalence of G6PD Deficiency in Iran

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## LETTER TO ETITOR

G6PD (Glucose-6-Phosphate Dehydrogenase) enzyme deficiency is the most common inherited enzyme deficiency so far reported.[1] This enzyme deficiency affects 400 million people worldwide.[2] Most cases of the disease are from tropical regions of Africa, the Middle East, tropical and subtropical regions of Asia and the Mediterranean margin, arise from the process of natural selection.[2-4] The majority of cases of this enzyme deficiency (about 50%) have been reported in male Kurdish Jews.[5] Up to now, more than 160 mutations associated with G6PD enzyme deficiency have been reported.[6] G6PDA$^-$ genotype is a specific variant caused by A376G and G202A mutations.[7] G202A mutation is responsible for 95% of the reported cases of G6PDA-variantin Africa. Moreover, G6PD-santamaria forms a class II variant with low prevalence caused by two simultaneous mutations in nucleotides 376 and 524 of the code in gexons of G6PD gene. This genotype was first identified in Costa Rica, and is also found with low prevalence in southern Italy.[8]

Large deletion, nonsense and frameshift mutations resulting in complete elimination of G6PD production are fundamentally incompatible with human life. Furthermore, no mutation has been reported in the coding region of the active enzyme site or in the promoter region. It is noteworthy that polymorphisms in non-coding sequences of G6PD are also involved in causing deficiency of G6PD enzyme. The exact mechanism of this enzyme deficiency remains unknown.[9]

Malarious regions are important because of the high incidence of G6PD enzyme deficiency. In the Middle East, there are many cases of G6PD enzyme deficiency in Iran, Oman and Saudi Arabia. Various cities and provinces of Iran have been studied for prevalence of the variants of G6PD enzyme deficiency. In Golestan Province, 69% of the variants were related to Mediterranean variant and 26.7% were the Chatham variant.[10] In a descriptive study conducted in Zanjan to determine the frequency of G6PD enzyme deficiency and review the type of defect in molecular level, it was shown that the prevalence of G6PD deficiency in Zanjan is lower than some other provinces. The incidence of malaria in Zanjan is low, and can be justified with low prevalence of enzyme deficiency in this city. In this survey, it was found that most people in this area have a Mediterranean-type mutation at position 563 of G6PD gene.[11] In other studies carried out in Tehran, the dominant (73.4%) incidence of Mediterranean-type mutation relative to other types of mutations was distinguished. Thus, the prevalence of Mediterranean-type mutation in Iran is similar to neighboring countries.[12]

The results of a number of studies indicate a high prevalence of G6PD deficiency in Iran, especially of Mediterranean and Chatham variants. The dominant variants in Cambodia and Myanmar are Viangchan and Mahidol variants, respectively. In Brazil and Mexico, G6PDA is considered as the dominant variant. The Mediterranean variant is more prevalent in India than the other variants.

High prevalence of G6PD deficiency in Iran demands higher attention on the part of healthcare system. In this regard, genetic counseling and health education are important to prevent the birth of newborns with this enzyme deficiency, particularly in families with a history of this disease. This high incidence undoubtedly suggests the importance of planning for early diagnosis and treatment of the patients. It seems that despite various diagnostic studies in recent years, many cases of G6PD enzyme deficiency have not been studied. In this context, the diagnosis for timely treatment to prevent the birth of affected newborns seems to be a priority. In the residents of regions with mixed ethnic background due to economic, political and cultural factors such as the provinces of South and South West of the country, there would be higher diversity of variants, and this will cause numerous problems for treatment of these patients. Molecular diagnostic methods for screening of asymptomatic women who are carriers of the disease may significantly contribute to prevention of the birth of affected newborns. Thus, the use of molecular diagnostic methods in areas where there is a high prevalence of the disease is recommended.[9]

In addition, it is important to pay attention to a number of points in controlling the complications of the disease. In cases of clinical symptoms associated with G6PD deficiency, the disease presence should be confirmed by quantitative assessment of the enzyme using a spectrophotometer. If it is necessary to implement a screening program in a population, semi-quantitative fluorescent blot test can be used before spectrophotometry. If G6PD deficiency is diagnosed, patients should avoid exposure to any oxidant drug or chemical. The patient should also be educated to take the necessary measures in the event of acute hemolysis. The use of diagnostic tests is recommended to identify and screen infants who are born in families with a history of disease, are related to a specific race or geographical region or show signs of acute hemolysis.

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