Letter to the Editor

Prevalence of glucose-6-phosphate dehydrogenase deficiency among schoolboys in Kermanshah, Islamic Republic of Iran

Sir

Glucose-6-phosphate dehydrogenase [G6PD] deficiency is a common X-linked recessive disorder affecting around 400 million people worldwide. Males are more affected than females and the condition is common in malaria-endemic regions [1,2]. The clinical manifestations of G6PD deficiency vary from no symptoms to acute haemolytic anaemia or severe chronic haemolytic anaemia [3].

This enzymopathy is very frequent in African, Mediterranean and Middle Eastern populations [1,4,5]. In Turkey, the frequency of G6PD deficiency among students was 1.2% [1], in Oman, the prevalence was 25% in males and 10% in females [5], while in Pakistan it was 1.8% [6] and in Iraq 6.1% [7]. A study of G6PD deficiency in male blood donors from different ethnic groups living in Kuwait revealed a wide range in the frequency of G6PD deficiency from 1% for Egyptians to 11.55% for Iranians [8]. The incidence of G6PD deficiency in Fars province, Islamic Republic of Iran, was estimated to be around 12% in males and 0.9% in females [9].

We studied 1000 randomly selected boys, aged 14–18 years, from 6 high schools in different areas of Kermanshah (a city in western Islamic Republic of Iran) to find the prevalence of G6PD deficiency. A questionnaire was prepared to ascertain the boys’ place of origin, ethnicity, and history of favism and anaemia. Whole blood samples were obtained from all individuals and enzyme activity was determined using the fluorescent spot test [10]. The screening test was classified and interpreted as absence of fluorescence (severe G6PD deficiency), weak fluorescence (partial G6PD deficiency) and bright fluorescence (sufficient G6PD activity). There were 53 out of 1000 subjects with G6PD deficiency, a frequency of 5.3%. All had severe G6PD deficiency. The frequency ranged from 2.2% to 9.0% among the schools.

Our study indicates a moderately high prevalence of G6PD deficiency in Kermanshah (5.3%), which suggests all newborns should be screened for G6PD deficiency to prevent neonatal jaundice and subsequent kernicterus.

References

3. Vullimy TJ et al. Diverse point mutations in the human glucose-6-phosphate dehydrogenase gene cause enzyme deficiency and mild or severe hemolytic


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