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Research Article

Epidemiological Study of Glucose-6-phosphate Dehydrogenase Deficiency in Scheduled Caste Population of India

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The aim of the present study was to determine the glucose-6-phostphate dehydrogenase (G6PD) deficiency in scheduled caste (SC) population of eastern Uttar Pradesh, India. After taking clearance certificate from the Institutional Ethics Committee, blood samples were collected from total 200 healthy individuals belonging to scheduled caste. G6PD deficiency analysis was done by methemoglobin test according to the method of Brewer et al. (1962). Out of 200 samples, 20 individuals were glucose-6-phosphate dehydrogenase deficient and 22 samples were heterozygous that is, carriers. The percentage of G6PD deficient (Gd+/+) and G6PD carrier (Gd+/Gd−) phenotypes were 10% and 11%, respectively. The frequency of mutant allele (Gd−) was observed 0.172. Early detection and prevention is the key strategy for successful management and control of this genetic disease.

1. Introduction

Glucose-6-phosphate dehydrogenase (G6PD) is a highly conserved housekeeping enzyme and rate-limiting enzyme of the pentose phosphate pathway in all cells [1]. The pentose phosphate pathway (PPP) converts glucose to ribose-5-phosphate, a precursor to RNA, DNA, ATP, CoA, NAD, and FAD. In addition, in mammalian cells G6PD provides reductive potential in the form of NADPH [2]. G6PD is a ubiquitous enzyme that must be quite ancient in evolution because it has been found in all organisms, from prokaryotes to yeasts, to protozoa, to plants, and animals [3, 4]. G6PD deficiency results from mutations in the G6PD gene and is well-known common cause of hemolytic anemia in human [5]. Most cells have a back-up system of other metabolic pathways that can generate the intracellular NADPH necessary, but red blood cells do not have the other NADPH producers. Therefore, G6PD deficiency becomes especially lethal in red blood cells, where any oxidative stress will result in hemolytic anemia. G6PD deficiency was first identified in American blacks in the course of studies of sensitivity to the hemolytic effect of primaquine [6]. Clinically, this deficiency affects as many as 400 million individuals worldwide [3] and predisposes affected individuals to neonatal jaundice, drug- or infection-mediated hemolytic crisis, favism, and, less commonly, to chronic nonspherocytic hemolytic anemia [7].

The G6PD gene is present on the long arm of the X chromosome (Xq28) and consists of 13 exons with a length of 18 kb [8]. The active form of G6PD enzyme is either a dimer or a tetramer of a single polypeptide subunit of about 59 kD [9]. G6PD deficiency is mainly found in populations originating from tropical and subtropical areas of the world and geographic distribution is similar to that of falciparum malaria. This deficiency is beneficial as it is known that red cells that are deficient in G6PD are resistant to *Plasmodium falciparum* invasion since the parasite require the enzyme for its normal survival in the host cell [10]. This deficiency offers a selective protection against *P. falciparum* malaria [11]. It has, however, been reported that some *P. falciparum* parasite strains have been able to synthesize their own G6PD enzyme thereby evading the immunity offered by G6PD deficiency in such individuals [12].

In India, G6PD deficiency was first reported in 1963 by Baxi et al. [13], and the prevalence rate varied from 0 to 27% in different caste, ethnic, and linguistic groups [14]. The frequency is higher among the tribals than the caste

Table 1: Distribution of the glucose-6-phosphate dehydrogenase enzyme phenotypes and their allele frequencies among SC samples.

	Normal (Gd+/Gd+)	Heterozygous (Gd+/Gd−)	Deficient (Gd−/Gd−)	Allele frequencies
Total samples	158	22	20	Gd+ = 0.828 Gd− = 0.172
Males	92	00	11	
Females	66	22	09	

populations [15]. Studies in the last few years also support the trend. Warli and Dhodia, tribal populations in Dadra and Nagar, Haveli have a frequency of 10.1% [16] and 13.5% [17], respectively, while Rajput, caste group from the same geographical region, has low frequency of 2.1% [18]. The prevalence of G6PD deficiency has been extensively studied in several population groups; however, there is no information about G6PD deficiency from different caste groups of Uttar Pradesh. Hence, the aim of the present study was to determine the frequency of glucose-6-phosphate deficiency among the scheduled caste population of Uttar Pradesh, India.

2. Materials and Method

Ethical clearance certificate was taken for the present study from the VBS Purvanchal University Ethics Committee, and the study conducted from October 2009 to April 2010. Blood samples were collected from the young healthy adults of scheduled caste population belonging to both sexes. After an informed consent, a brief clinical record including age, ethnic group, place of residence, and history of past illnesses including fever and episodes of recurrent jaundice was recorded. Four-milliliters cubed blood was collected from each subject in acid citrate dextrose (ACD) coated vials. Glucose-6-phosphate dehydrogenase deficiency analysis was done by methemoglobin test according to the method of Brewer et al. [19]. The test is based on methemoglobin production by addition of nitrites in test blood, and then process of methemoglobin reduction to oxyhemoglobin is triggered by methylene blue. Positive controls and negative controls were employed in each batch. Gene frequency was calculated by simple gene count method.

3. Result and Discussion

Total 200 scheduled caste samples were collected and analyzed for G6PD deficiency. In 200 SC samples, 103 samples are of males and 97 are of females. Out of 200 samples, 20 individuals are found to be G6PD deficient and 22 samples are heterozygous that is, carriers. The percentage of G6PD-deficient (Gd+/+) and G6PD carrier (Gd+/Gd−) phenotypes were 10% and 11%, respectively. The frequency of normal allele (Gd+) was 0.8283, and frequency of mutant allele (Gd−) was 0.172 (Table 1). In total 97 female samples, 9 are glucose-6-phosphate dehydrogenase deficient and 22 are carriers while in males 11 are glucose-6-phosphate dehydrogenase deficient. G6PD deficiency is an X-linked recessive trait, it is predominantly a disease of males. But in present study the percentage of G6PD deficiency in females and males is found to be 9.3% and 10.7%, respectively. The percentage of female deficient is quite high. The reason may be the small sample size.

Several reports regarding G6PD deficiency were published in several caste groups/tribes like Parsee [13], Muslim [20], Brahmin [21–23], Jat [22], Rajputs [18], Vatalya Prajapati [24], Nagas [25], Kharia [26], Bhuyan [26], Danguria tharu [27], Kabui [20], and so forth. The frequency is higher among the tribals than the caste populations [15]. According to Bhasin and Walter [28], the frequency of G6PD deficiency among Indian population as a whole ranges from complete absence to 27%. It is higher among the scheduled tribes as compared to other ethnic groups. G6PD-deficient allele frequency is comparatively higher in North and West Indian zones, whereas in South India it is uniformly low except in Andhra Pradesh and Tamil Nadu. Prevalence of G6PD deficiency is generally 0−10%, although some communities may have higher prevalence: 27.5% for the Vataliya Prajapati community in Western India [24] and 27.1% for the Angami Nagas, a tribal group in Northeastern India [25]. In present study, the G6PD deficiency in scheduled caste population is observed 10% which is comparable with the earlier reports in the same caste group. So it is evident from the observation of the present study that the G6PD deficiency is quite high in eastern Uttar Pradesh, and it is need of the hour to explore the different caste population of Eastern Uttar Pradesh population for G6PD deficiency, because UP population is genetically very less explored.

G6PD deficiency was mainly found in populations originating from tropical and subtropical areas of the world. The geographical distribution was similar to that of falciparum malaria, and suggested G6PD deficiency similar to the sickling trait owed its distribution to selection by this malaria organism [29]. The deficiency of G6PD is found in a belt extending from the Mediterranean area through Southwest Asia and India to Southeast Asia. Several epidemiological studies have identified pockets in Asia and the Middle East with prevalence of the disease as high as 62% in Kurdish Jews [30] and 31% in Northern Vietnam [31]. Frequency of G6PD deficiency varies worldwide among different ethnic groups ranging from 20 to 30% in Greece, 6% in Saudi Arabia, and 5.5% in South China [32]. In Africa, the prevalence of G6PD deficiency has been reported as high as 28.1% in Southwest Nigeria [33], 22.5% in Congo (Brazzaville) [34], 15.7% in Mali (Bamako) [35], 13.0% in Uganda [36], and 9.0−15.5% in Gabon [37]. Numerous reports have been published on this genetic disorder in various Asian countries like Indonesia [38], Thailand [39], Malaysia [40], Taiwan [41], Bangladesh [42], Pakistan [43, 44], China [45], and Myanmar [46]. In Southeast Asia, the prevalence of G6PD deficiency differs greatly by region and ethic group and variants are similarly

Table 2: Percentage of G6PD deficiency in different populations.

Author	Country/population	Percentage of G6PD deficiency
Iwai et al. [47]	Myanmar	10.8%
Hilmi et al. [50]	Iraq	6.1%
Usanga and Ameen [51]	Iran	11.6%
May et al. [33]	Nigeria	15.7%
Al-Riyami and Ebrahim [52]	Oman	2–29%
Usanga and Ameen [51]	Lebanon	2.1%
Bayoumi et al. [53]	UAE	11%
Hussein et al. [54]	Egypt	5.9%
Al-Arrayed [55]	Bahrain	18%
Atlay and Gumruk [56]	Turkey	0.5–20%
Usanga and Ameen [51]	Syria	30%
Segeja et al. [57]	Tanzania	4.43–11.32%
White et al. [58]	Yamen	6.2%
Davis et al. [36]	Uganda	13%
Alabdulaali et al. [59]	Saudi Arabia	1–39.8%
Usanga and Ameen [51]	Kuwait	5.5%
Usanga and Ameen [51]	Jordan	3.6%
Akhter et al. [42]	Bangladesh	3.33–20%
Nuchprayoon et al. [39]	Thailand	3–18%
Matsuoka et al. [60]	Vietnam	2.3%
Matsuoka et al. [61]	Cambodia	7%
Ali et al. [43]	Pakistan	1.07–3.17%
Chan and Todd [62]	South China	5.5%

diverse. For example, in Myanmar, Iwai et al. [47] found prevalence of G6PD deficiency as high as 10.8% for the Shan people, 7.3% in the dominant ethnic group the Burma, and absent in the Akha, despite a regional proximity to the Shan. Table 2 shows percentages of G6PD deficiency reported from several Mediterranean, African, and Asian countries. As evident from the table, the highest frequency of G6PD deficiency is in Mediterranean countries like Syria (30%) and Southwest Asia like Saudi Arabia (39.8%), but frequency is low in Southeast Asian countries like China (5.5%), Bangladesh (3.33–20%), Myanmar (10.8%), and Vietnam (2.3%).

Investigations of enzyme in different human populations have shown more than 300 variants, and most of them are relatively rare, but some have appreciable frequencies in certain localized populations [48, 49]. So far several different G6PD variants are reported from India, namely, G6PD* Mediterranean, G6PD* Orissa, G6PD* Andhra Pradesh, G6PD*Cutch, G6PD* Jammu, G6PD* Kerala-Kalyan, G6PD*Porbandar, G6PD*Chatham, G6PD* Insuli, G6PD*Coimbra, and G6PD*West Bengal have been reported from India.

Early detection and prevention is the key strategy to successful management and control of G6PD deficiency. Genetic counseling, prenatal diagnosis, health education, and public awareness can provide benefits by way of preventive genetics to the affected individuals and their families. It is emphasized that there is further need to evaluate the clinical and prognostic aspects of the G6PD enzyme deficiency among the population of Eastern Uttar Pradesh, which will yield some definite insights into this genetic health problem in Uttar Pradesh.

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