RESEARCH REPORT

G-6-PD deficiency and Sickle cell anaemia in Badhiys Muslims of Purnia District (Bihar)

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Manuscript details:

Received: 03.10.2015
Revised: 02.11.2015
Accepted: 10.12.2015
Published : 30.12.2015

Editor: Dr. Arvind Chavhan

Cite this article as:
Sanjeeva Kumar, Md. Jahangeer and Pandey BN (2015) G-6 PD deficiency and Sickle cell anaemia in Badhiys Muslims of Purnia District (Bihar). *International J. of Life Sciences*, 3(4): 395-398.

ABSTRACT

G-6 PD deficiency and sickle cell anaemia were studied in Badhia Muslims of Purnia distrct (Bihar) which are migrated from West Bengal. In a sample size of 509, no case of G-6 PD deficiency and sickle cell anaemia were observed. Once upon a time, Purnia district was popularly known as Kala Pani due to its bad climate and malaria was endemic in this area. However, now a day the climate has improved and the area is not endemic to malaria. It needs further investigation to know if there is any correlation between decreasing trends of malaria with G-6-PD deficiency, and sickle cell anaemia

Keywords:G-6-PD deficiency, sickle cell anaemia, Badhiya Muslims, malarial endemicity and Purnia district

INTRODUCTION

Hereditary hemolytic disorders like hemoglobinopathies, thalassemia syndrome and glucose-6-phosphate dehydrogenase (G-6-PD) enzyme deficiency are important genetic and public health problems in India. The sickle cell anemia especially affects 60-70 million people all over the world. The victims include the growing children, adolescent girls, pregnant women and a large chunk of ignorant people. Inherited disorders of hemoglobin cause high degree of hemolytic anemia, clinical jaundice, painful crisis, frequent infections, splenomegaly, growth retardation, etc. and are responsible for high infant morbidity and mortality, maternal mortality and fetal wastage in India. Although both hemoglobinopathies and G-6-PD deficiency are prevalent in malaria endemic areas but to the best of our knowledge, no study has ever reported combined conditions in a single individual from India. The present study highlights G-6-PD deficiency and sickle cell anaemia in a randomly conducted study in Badhiya Muslims of Purnia district (Bihar), India.

MATERIALS AND METHODS

Blood samples (509) were collected in test-tubes containing an anticoagulant, Acid Citrate Dextrose (ACD) solution from individuals from different villages of Purnia district.

The samples include unrelated individuals. The G-6-PD deficiency was detected with brilliant crystal blue dye test of Motulsky & Campbell-Kranel (1961). Testing of sickling was done on the spot, by using freshly prepared solution of sodium-meta-bisulphate ($Na_2S_2O_3$) in the manner described by Daland and Castle (1948). A two percent solution of salt was prepared in sterile distilled water immediately before the tests. A small droplet of fresh blood from the finger tip was mixed with a drop of solution on a clear microscope slide and the mixture immediately covered with a sealed cover glass. The first observation was made after fifteen minutes in which sickling could be seen in most of the positive cases, but the final reading was taken after half-an-hour under the dry objective of the microscope. Longer time was avoided to avoid false sickling. All the positive cases were re-tested in order to ensure the reliability of the results.

RESULTS AND DISCUSSION

G-6-PD deficiency was mainly found in populations originating from tropical areas of the world. In India, G6PD deficiency was first reported in 1963 by Baxi *et al*. and the prevalence rate varies. The frequency of G-6-PD deficiency is 4.5% (varies from complete absence to 27.1%) among Indian population and it is quite high among the Scheduled tribes as compared to the other ethnic groups (Bhasin *et al*., 1994). The frequency is higher among the tribals than the caste populations. The frequencies of G-6PD deficiency among Indian population as a whole ranges from complete absence to 27% (Bhasin and Walter 2001). 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 (Gupte *et al*., 2005) and 27.1% for the Angami Nagas, a tribal group in Northeastern India Seth and Seth, 1971). However, in the present study no case of G-6PD deficiency was found. It has been found that the distribution of G-6PD deficiency is not closely related to that of malaria (Bhasin *et al*., 1994). Saha *et al*., (1990) observed a very low frequency of G6PD among Naga, Hamar, Lepcha and Adi populations in the region of high malarial endemicity.

Anaemia is a condition in which the red corpuscles are reduced or the amount of haemoglobin is lessened. Sickle cell disease (SCD) is an autosomal recessive genetically transmitted hemo-globinopathy responsible for considerable morbidity and mortality. The sickle gene is an example of balanced polymorphism. Heterozygotes have a selective advantage and are protected against *Plasmodium falciparum* malaria while there is an increased premature death rate of homozygotes (Stuart and Nagel, 2004). It is prevalent in many parts of India including Central India, where the prevalence in different communities has ranged from 9.4-22.2% (Shukla and Solanki, 1985). Dunlop and Mazumder reported the first case of sickle cell hemoglobin in India.

Haemoglobinopathies are concerned with the abnormality in the protein molecule of red blood cells. This abnormality is due to defective synthesis of globin chains or its structure. A genetic defect that results in abnormal structure of one of the globin of the haemoglobin molecule is termed as haemoglobinopathy. These inherited genetic diseases of haemoglobin are controlled by a single gene and are transmitted from generation to the next. In India the presence of Sickle cell gene (HbS) was first detected in Nilgiri Hills of southern part (Lehman and Cutbush, 1952). Sickle cell is most common pathological

haemoglobin variant worldwide (Weatherall *et al*., 2006). The Indian subcontinent is a rich reservoir of sickle cell anaemia (SCA), thalassaemia (β- thal) and various abnormal haemoglobins. In India, HbS gene ('HbS' for HbAS carrier) is mostly confined to tribes in central and south India and the frequency ranges from 5 to 35 per cent (Bhatia and Rao, 1987) and in most of the Indian populations, castes and tribes the high incidence of various abnormal haemoglobins has been reported . In Central India study on sickle cell anaemia has been carried out mostly on tribal groups and very few on castes and other populations (Agarwal, 2005). WHO (2006) has reported an estimate of about 20-25 million homozygous individuals for sickle cell disease worldwide of which 5-10 million are in India (Serjeant,2006).

In India, the trait occurs most commonly among the tribal peoples in central India (southeastern Gujarat, Maharastra, Madhya Pradesh, Chhattisgarh, western Odisha) with a smaller focus in the south of the country (northern Tamil Nadu and Kerala), and trait frequencies as high as 40% have been described in some groups (Serjent, 2013). Among the Indian populations the frequency of sickle cell trait is 3.1% (Varies from complete absence to 41.0%). It is present in high frequency among the Scheduled tribes (5.4%) as compared to other ethnic groups (Bhasin *et al*, 1992, 1994). The frequency of HbS in Brahmins is 4.17%, in Kalar 5.41%, in Rajput 2.04%, in Muslims 3.73% in Maratha 2.08% in Bania 9.09% while in Teli it is 3.65% of Central India Urade (2012). Shah *et al*., (2012) have reported the allele frequency of sickle cell gene, i.e., 16.96% and 8.6% respectively in Moghul and Naga populations of Manipur. The earlier researchers have shown a complete absence of gene HbS in Muslims (Hakim *et al*., 1972; Saha *et al*., 1976; VijayKumar *et al*., 1987; Gorakshakar *et al*., 1987). But Urade has reported presence of HbS gene in Muslims (3.73%). In the present study of Badhiya Muslims no case of sickle cell was found. However, this finding needs verification by enlarging study area as well as sample size as once upon a time, malaria was prevalent in this area and there is abundance of swampy area in this region. This trait is reported to be present in scheduled castes and communities, who are living in close proximity with tribal populations. The trait has been transmitted among these groups due to admixture with tribal groups (Bhasin *et al*., 1994). The sickle cell trait is either absent (Bihari group of Indo-european family) or present in very low frequency (0.010) among Munda group of austro-asiatic speakers.

Haemoglobin polymorphism with G-6PD deficiency is advantageous to the communities against lethal effect of malaria especially against *Plasmodium falciparum* at population level but their combination is harmful at the individual level because of low level of blood cells indices to cope with the routine human physiology, where malaria is endemic. There is unexpected association between haemoglobinopathies and G-6PD deficiency (Jacques *et al*., 2007; Balgir, 2010). However, in the present study no case of G-6PD deficiency and sickle cell trait was found. Though, once upon a time malaria was prevalent in this area. Thus there is a decrease tendency of G-6PD deficiency and sickle cell anaemia in the study area which is according to observation made by Pandey and Ranjana, 20013 and Pandey *et al*., 2014. However, sickle cell has not been observed in Muslim populations in India (Balgir, 2007)

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