Original Article

G6PD Deficiency

Frequency of Glucose 6 Phosphate Dehydrogenase Deficiency in Patients with Plasmodium Vivax Malaria Presenting to a Tertiary Care Hospital

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ABSTRACT

Objective: To determine the frequency of glucose 6 phosphate dehydrogenase (G6PD) deficiency in patients with plasmodium vivax malaria presenting to the department of Medicine , Hayatabad Medical Complex, Peshawar.
Study Design: Descriptive / cross sectional study
Place and Duration of Study: This study was conducted at the Department of Medicine, Hayatabad Medical Complex, Peshawar from January 2016 to December 2016.
Materials and Methods: Patients with plasmodium vivax malaria were included and patients having evidence of acute hemolysis were excluded from the study. The G6PD level was measured in the Department of Pathology, HMC. The data collected was analyzed in SPSS latest version. Mean ± SD was calculated for continuous variable. Frequencies and percentages were calculated for categorical variables final results were presented in tables.
Results: A total of 870 patients were included. We found that 53(6.09%) subjects were deficient in G6PD. These cases were divided into non hospital and hospital groups. In Non-hospital group G6PD deficiency was 4.1% while13.3 % in the other group. Thirty three patients (63.3%) had anemia in hospital patients and 8 patients had severe anemia. None of the Non-hospital had severe anemia, while mild anemia was observed in 16(30.2%) patients in Non-hospital groups.
Conclusion: G6PD deficiency is predominantly found in male population. Moreover anemia is more common in G6PD deficient people than normal population.
Key Words: Glucose 6 phosphate dehydrogenase deficiency, plasmodium vivax, Malaria

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INTRODUCTION

The enzyme glucose 6phosphate dehydrogenase is found in almost all body tissue, and its function is to catalyze the initial step in the pentose phosphate pathway. Glucose 6phosphate dehydrogenase deficiency is a one of the commonest X-linked enzyme abnormality[1]. G6PD deficiency is endemic throughout tropical and subtropical regions of the globe[2-4]. This pathological problem which is endemic in tropical and subtropical countries, present wit hemolytic crisis and jaundice in the early days of a neonatal life[5, 6].

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Plasmodium vivax is equally endemic in most part of the world and it G6PD deficient are also affected as it is affecting individuals with normal G6PD[7-9]. Though G6PD deficient individuals are without symptoms most of the time, yet they can present with jaundice in early neonatal life and with acute hemolytic anemia especially when these people suffer from infection or the they take some drugs and broad beans (favism) which aggravate the process of red blood cells with the enzyme deficiency). Though G6PD deficiency is of several variants yet in many cases the problem is caused by a single amino acid substitution[10]. Premaquine has an important role in vivax malaria treatment protocol and its use is challenging in G6PD deficient patients[11,12,13].
This study was done to look for G6PD deficiency and to see presence of anemia in these patients with vivax malaria.

MATERIALS AND METHODS

This descriptive / cross sectional study was done in the Department of Medicine, Hayatabad Medical Complex, Peshawar from January 2016 to December 2016. The study was approved by research ethical committee HMC, Peshawar. All the patients having history of plasmodium vivax malaria detected in indoor or outdoor OPD visits were included in the study.

Patients with evidence of acute hemolysis were not included in the study as G6PD deficient patients can have normal levels during hemolysis. The G6PD level was measured in Hematology section of the Department of Pathology HMC as this laboratory is a reference lab and receives blood samples from various parts of the province.

2-3 ml of patient blood sample was transferred to an EDTA tube. Span Diagnostic Qualitative G6PD Test kit was used. Hemolysate was prepared form patient blood using 0.9% Normal Saline. The commercial kit was reconstituted using the supplied mineral oil. Hemolysate was mixed with the kit reagents and buffer. The mix was incubated for 1 hour at 37 degrees while being observed for color change at intervals of 5 min. A change in color form Blue to Red within the first 60 min signifies Normal G6PD levels. No Color change beyond 60 min was taken as evidence of low G6PD levels.

All the collected data was analyzed in SPSS latest version. Mean ± SD was calculated for continuous variable. Frequencies and percentages were calculated for categorical variables Final results were presented as tables.

RESULTS

A total of 870 patients fulfilling the inclusion criteria were included in the study. In our study we found patients age from age five years to thirty years. In Non-hospital group 439 were males while 243 were females out of 682 cases. In the hospital group 113 were males and 75 were females in total of 188 cases.

In total of 870 patients screened for G6PD deficiency 28 found deficient in the Non-hospital group; while 25 were deficient in hospital group. Out of these 68% from non-hospital group (19/28) were males while 32% (9/28) were females. In the hospital group 25(13.3%) patients (25/188) had G6PD deficiency. Amongst these 25 patients, 15 were male while 10 were female. The details have been given in table 1.

Table No.1: G6PD deficient patients in non-hospital and Hospital group.

Groups	Total subjects	Deficient	%
Non-hospital group	682	28	4.1
Males	439	19	68
Females	243	9	32
Hospital group	188	25	13.3
Males	113	15	60
Females	75	10	40
Grand Total	870	53	6.09

In 53 patients found G6PD deficient cases the red blood cell count and haemoglobin level was found to know about various level of anemia. We found that 33 patients (63.3%) had anemia. Eight (15.1%) had severe anemia, 9 (17.0%) were having moderate anemia and 16(30.2%) had mild anemia which is shown in table 2.

As far as presentation of the condition month-wise is concerned, Maximum were reported in March i.e. 97 followed by May, April, September and January; whereas minimum number found in November

Table No.2: Anemia status in 53 cases with G6PD deficiency

	ANEMIA			
	Severe	Moderate	Mild	Total
Non-hospital group	0 (0%)	1 (1.9%)	9 (17.0%)	10 (18.9%)
Hospital group	8 (15.1%)	8 (15.1%)	7 (13.2%)	23 (43.4%)
Total	8 (15.1%)	9 (17.0%)	16 (30.2%)	33 (63.3%)

Severe anemia =Hemoglobin <7.0g/dl.
Moderate anemia= Hemoglobin 7-10g/dl
Mild anemia = Hemoglobin 10-11g/dl

Table No.3: Presentation of G6PD deficiency I various months

Month	Total Subjects	G6pd	%
Jan	81	4	4.3
Feb	61	2	3.3
Mar	97	8	7.8
April	86	5	5.2
May	88	8	8.5
June	76	3	3.9
July	74	3	4.05
Aug	58	3	5.2
Sept	81	5	5.6
Oct	61	5	8.2
Nov	43	4	9.4
Dec	67	6	8.3

DISCUSSION

We did our this research work at KPK Tertiary level hospital to know the frequency of glucose 6 phosphate dehydrogenase deficiency, occurring as minimum as below 5% in central area to as high as 24%. Our study showed G6PD deficiency in tertiary hospitals is 6.09%. This frequency is above the other study conducted in tertiary level hospital[13].

We found in our study that frequency of G6PD deficiency in hospitalized group was much higher than the non-hospitalized ones; which was 13.3% and 4.1% respectively.

There was male predomince over female with a ration of almost 2:1 ratio. Results in other countries of the world are nearly same, as shown by Joshi et al[14] and Sanpavat et al[15] who conducted their native lands i.e. India and Thailand respectively.

In our study we found that incidence of anemia in patients with G6PD is quite high i.e. 67% which was different from other international studies which showed

lower frequency[15,16]. Asymptdomatic patient percentage was low in our studies i.e. 37 % while the asymptomatic patients [15, 16].

One of the core root cause responsible for this high incidence of anemia in our region is probably genetic polymorphism of G6PD.

In this study we found that the maximum number of cases were found in May followed by May, April, September and January; while the minimum number in November. This part of our study also coincides with other international studies [13,17].

In our study we found no causal relationship between bean ingestion and hemolytic anemia in the enzyme deficient cases and same is given by a Saudi study; while on international study showed contrary result by Warsy et al who found strong association of hemolysis in enzyme deficient patient with bean intake[18].

CONCLUSION

G6PD deficiency is predominantly found in male population. Moreover anemia is more common in G6PD deficient people than normal population

Recommendations: As both vivax malaria and Glucose 6 Phosphate deficiency are our common community problems but very less studies have been conducted. Therefore, various aspects of either condition should be considered for original studies to give maximum benefits to our community and create awareness in our public

Author's Contribution:

Concept & Design of Study:	Muhammad Bilal Khattak
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Data Analysis:	Arshia Munir
Revisiting Critically:	Shams Sulaiman
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