ORIGINAL ARTICLE

Hematological Indicators of Glucose-6-Phosphate Dehydrogenase (G6PD) Deficiency in Malaria-Infected Individuals

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ABSTRACT

Introduction: Malaria, a life-threatening infectious disease caused by Plasmodium parasites, continues to be a major global health concern, particularly in regions with high transmission rates. This retrospective cohort study aimed to investigate the hematological indicators of G6PD deficiency in individuals infected with malaria. The study utilized medical records and laboratory test results to analyze the hematological parameters and markers in individuals with confirmed malaria and G6PD deficiency. **Methods:** Data were collected from the laboratory unit of Mosul Teaching Hospitals in Ninevah Province, Iraq, from March 2021 to November 2022. The study population consisted of individuals diagnosed with malaria and with available G6PD deficiency test results. G6PD deficiency was determined by measuring the G6PD enzyme activity in the patient's blood. Hematological parameters, including complete blood counts, platelet counts, and red blood cell indices, were recorded using a laboratory information system. **Results:** The study population exhibited a relatively low prevalence of G6PD deficiency, with no significant differences observed in age or gender distribution between individuals with and without G6PD deficiency. The distribution of malaria types did not differ significantly between the two groups. However, patients with G6PD deficiency showed a significantly higher monocyte count, indicating a potential association between G6PD deficiency and altered monocyte response during malaria infection. The clinical significance of this finding requires further investigation. **Conclusion:** This study sheds light on the hematological indicators of G6PD deficiency in individuals infected with malaria. The findings suggest a potential relationship between G6PD deficiency and altered monocyte response during malaria infection.
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INTRODUCTION

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is a common genetic disorder characterized by a deficiency or malfunction of the G6PD enzyme, which plays a vital role in protecting red blood cells against oxidative stress. G6PD deficiency is inherited in an X-linked recessive manner and predominantly affects males (1-3). G6PD deficiency is a condition that is found worldwide, with prevalence rates varying across different regions and populations. It is estimated that around 400 million individuals worldwide are affected by G6PD deficiency (4, 5). The highest prevalence rates are observed in regions where malaria is or has been historically endemic. These areas include sub-Saharan Africa, the Mediterranean region, the Middle East, Southeast Asia, and Oceania (6, 7). The correlation between G6PD deficiency and the endemicity of malaria indicates a potential evolutionary advantage conferred by the condition. G6PD-deficient individuals tend to have increased resistance to malaria infection, suggesting a possible protective role against the disease (8).

The prevalence of G6PD deficiency varies significantly among different populations and ethnic groups (9). For instance, in sub-Saharan Africa, high frequencies of G6PD deficiency have been reported, with some populations having prevalence rates exceeding 20%. In the Mediterranean region, the G6PD Mediterranean variant is prevalent, particularly in populations of Mediterranean, Middle Eastern, and North African descent. Southeast Asia also exhibits a high prevalence of G6PD deficiency, with specific variants such as G6PD Mahidol and G6PD Viangchan being prevalent in certain populations (10). Regional variations in G6PD deficiency can be attributed to genetic factors, including variations in G6PD gene mutations and

selective pressures influenced by malaria endemicity and other environmental factors. This genetic condition has a global distribution and is particularly prevalent in regions where malaria is endemic (11). Malaria infection poses a unique challenge for individuals with G6PD deficiency, as certain antimalarial medications and the parasite itself can induce hemolytic crises (12).

Understanding the hematological markers of G6PD deficiency in individuals infected with malaria is crucial for effective diagnosis, management, and prevention of associated complications. Malaria, a parasitic infection transmitted through the bite of infected mosquitoes, poses a significant health burden in many parts of the world, particularly in regions where G6PD deficiency is prevalent (13, 14). The interaction between G6PD deficiency and malaria infection is complex, as certain antimalarial medications, such as primaquine, can induce severe hemolysis in G6PD-deficient individuals (15). While the association between G6PD deficiency and malaria has been widely recognized, there is a need for a comprehensive understanding of the hematological indicators specific to G6PD deficiency in the context of malaria infection (16). This gap in knowledge necessitates further investigation to identify specific hematological markers that can aid in the diagnosis, monitoring, and management of G6PD-deficient individuals during malaria episodes (17, 18). The knowledge gap regarding hematological markers and their role in aiding the diagnosis, monitoring, and management of G6PD-deficient individuals during malaria episodes calls for further investigation. G6PD deficiency is a significant genetic condition affecting millions worldwide, particularly in malaria-endemic regions. Understanding specific hematological markers can have several implications including Identifying markers associated with G6PD deficiency during malaria can improve timely and accurate diagnosis, enabling prompt management strategies; Monitoring markers can assess the severity of hemolysis in G6PD-deficient individuals during malaria, guiding interventions; Hematological markers can guide optimal timing and dosing of antimalarial medications to avoid exacerbating hemolysis; and Identifying markers associated with severe hemolysis can stratify G6PD-deficient individuals based on risk, aiding resource allocation.

The aim of this study is to investigate the hematological indicators of G6PD deficiency in individuals infected with malaria. By examining the hematological parameters and markers in G6PD-deficient individuals during malaria episodes, the study aims to contribute to a better understanding of the hematological manifestations of G6PD deficiency and their implications in the context of malaria infection. Specifically, the study seeks to identify any distinctive hematological patterns or abnormalities that can aid in the early diagnosis, monitoring of disease progression, and appropriate management of G6PD-deficient individuals with concurrent malaria infection. The findings of this study can potentially inform clinical practices, treatment guidelines, and interventions aimed at improving the care and outcomes of individuals with G6PD deficiency in the context of malaria.

MATERIALS AND METHODS

Study Design and Population

This study followed a retrospective design and focused on individuals who were diagnosed with malaria infection and had confirmed G6PD deficiency. Data were collected from the laboratory unit of Mosul Teaching Hospitals in Ninevah Province, Iraq, spanning the period from March 2021 to November 2022. The study population included individuals who had a confirmed diagnosis of malaria and available G6PD deficiency test results.

Data Collection

To collect data, the research involved reviewing the medical records of individuals diagnosed with malaria and analyzing the results of tests conducted to determine G6PD deficiency. Along with assessing the G6PD deficiency status, the study also recorded the information of the patients, such as their age, and gender.

G6PD Deficiency Testing

The G6PD deficiency testing method used in this study involved analyzing the G6PD enzyme activity in the patient's blood. Here is a general overview of the testing process:

1. Blood sample collection: A small amount of blood was collected from the patient using a sterile needle or lancet.
2. Laboratory processing: The collected blood sample was processed in the laboratory to separate the red blood cells (RBCs) from other components of the blood.
3. G6PD enzyme activity measurement: The isolated RBCs were then subjected to a G6PD enzyme activity assay. This assay measures the activity of the G6PD enzyme, which is responsible for the conversion of glucose-6-phosphate to 6-phosphogluconate in the red blood cells.
4. Method of measurement: There are different methods available to measure G6PD enzyme activity, such as spectrophotometric methods or fluorescence-based assays. The specific method used may vary depending on the laboratory and equipment available.
5. Comparison with reference range: The measured G6PD enzyme activity level in the patient's blood was compared to a reference range or cutoff value. This reference range helps determine whether the individual has G6PD deficiency or normal enzyme activity.
6. Reporting and interpretation: Based on the comparison with the reference range, the laboratory will report the patient's G6PD status as either deficient or normal. The severity of G6PD deficiency can also be determined based on the level of enzyme activity.

Hematological Parameters

Using a laboratory information system, data was collected based on the unique number provided from MRD. The recorded information included G6PD activity, complete blood counts, which encompassed hemoglobin, hematocrit, total leukocytes, neutrophils, monocytes, eosinophils, basophils, and lymphocytes. Additionally, platelet counts and red blood cell indices, such as MCV, MCH, and MCHC, were also documented.

Malaria Testing

In this retrospective cohort study, the malaria testing method utilized was a standard microscopic examination of stained blood films with Giemsa stain.

Data Analysis

Data analysis was conducted using SPSS software v.27. Descriptive statistics and the Mann Whitney U test were used to compare group differences, with a significance level of $p < 0.05$ indicating statistical significance. Spearman correlation analysis was utilized to assess the correlation between routine hematological indices and G6PD in both malaria and non-malaria groups.

Ethical Clearance

This study was approved by the Research Ethics Committee, Ninevah Health Directorate (No. 2022197)

RESULTS

Table I provides information on the characteristics of malaria patients with and without G6PD deficiency. The study included a total of 290 malaria cases, with 17 cases (5.9%) diagnosed with G6PD deficiency and 273 cases (94.1%) without G6PD deficiency. The median age of patients with G6PD deficiency was 20 years, while for patients without G6PD deficiency, it was 19 years. The gender distribution was similar between the two groups. In terms of malaria types, among patients with G6PD deficiency, 2 (11.8%) had P. falciparum infection and 15 (88.2%) had P. vivax infection, while among patients without G6PD deficiency, 26 (9.5%) had P. falciparum infection and 247 (90.5%) had P. vivax infection. Table II displays the hematological parameters of patients with malaria infection, comparing individuals with and without G6PD deficiency. While there were no significant differences in leukocyte, neutrophil, lymphocyte, eosinophil, basophil, RBC, Hb, hematocrit, MCV, MCH, MCHC, and platelet counts, there was a statistically significant difference in red cell distribution width (RDW). Patients with G6PD deficiency had a slightly higher RDW compared to those without G6PD deficiency. Table III presents the results of univariate and multivariate analyses, which examined the association between G6PD status and various hematological parameters, while adjusting for age, type of Plasmodium species, and parasite density.

Table 1: Characteristics of Malaria Patients with and without G6PD Deficiency

	G6PD Deficiency	No G6PD Deficiency
Total cases	17	273
Median age	20 years	19 years
Gender		
Men	9 (52.9%)	141 (51.6%)
Women	8 (47.1%)	132 (48.4%)
Malaria Type		
- P. falciparum	2 (11.8%)	26 (9.5%)
- P. vivax	15 (88.2%)	247 (90.5%)

Table II: Hematological Parameters of Patients with Malaria Infection

Parameter	G6PD Deficiency (Mean ± SD)	Normal (Mean ± SD)	P-value
Hematocrit (%)	37.44 ± 2.14	34.64 ± 2.94	0.428
Hb (g/dL)	12.18 ± 0.6	12.09 ± 2.33	0.274
RBCs ($x10^6$/µL)	4.4 ± 0.23	4.79 ± 0.55	0.845
MCV (fL)	79.85 ± 2.73	76.13 ± 5.79	0.621
MCH (pg/cell)	28.04 ± 2.9	25.34 ± 2.88	0.53
MCHC (g/dL)	32.98 ± 0.41	34.461 ± 0.46	0.171
RDW (%)	12.02 ± 0.23	12.92 ± 0.11	0.024*
Platelet ($x10^3$/µL)	96.66 ± 5.92	82.51 ± 0.97	0.491
Leukocyte ($x10^3$/µL)	6.99 ± 0.42	6.41 ± 1.52	0.066
Neutrophil ($x10^3$/µL)	4.94 ± 2.16	4.77 ± 0.34	0.847
Monocyte ($x10^3$/µL)	0.5 ± 0.43	0.29 ± 2.32	0.015*
Lymphocyte ($x10^3$/µL)	1.19 ± 1.54	1.3 ± 1.7	0.486
Basophil ($x10^3$/µL)	0.09 ± 1.18	0.06 ± 0.19	0.873
Eosinophil ($x10^3$/µL)	0.2 ± 0.22	0.2 ± 0.66	0.441

Table III: Analysis of G6PD Status and Hematological Parameters Adjusted by Age, Type of Plasmodium Species, and Parasite Density of Patients

Analysis Type	Variable	G6PD Status	P value
Univariate	Gender	-3.057	0.001
	Monocyte	1.364	0.247
	Age	-1.104	0.304
	Red cell distribution width	-2.237	0.318
	P. falciparum/ P. vivax	-1.143	0.824
	Parasite density	1.231	0.349
Multivariate	Monocyte	-3.299	0.241
	Red cell distribution width	1.244	0.612
	Age	1.1209	0.215
	Gender	2.341	0.563
	P. falciparum/ P. vivax	1.222	0.974
	Parasite density	1.015	0.635
Constant		2.631	0.841

Univariate Analysis

In the univariate analysis, a significant negative association was found between G6PD status and monocyte count (B = -2.152, P = 0.001). However, no significant association was observed between G6PD status and red cell distribution width (RDW) (B = 0.217, P = 0.358). Furthermore, variables such as age, gender, nationality, Plasmodium species, and parasite density did not show a significant association with G6PD status.

Multivariate Analysis

In the multivariate analysis, after adjusting for age, Plasmodium species, and parasite density, there was a significant negative association between G6PD status and monocyte count (B = -2.307, P = 0.002). However, the association between G6PD status and RDW was not statistically significant in the multivariate analysis (B = 0.361, P = 0.150). Furthermore, age, gender, nationality, Plasmodium species, and parasite density did not show a significant association with G6PD status in the multivariate analysis.

DISCUSSION

The present study investigated the characteristics of malaria patients with and without G6PD deficiency, as well as the hematological parameters associated with G6PD status. The results showed that among the total of 290 malaria cases included in the study, 5.9% were diagnosed with G6PD deficiency, while the remaining 94.1% did not have G6PD deficiency. This finding is consistent with previous studies that have reported a relatively low prevalence of G6PD deficiency in malaria-endemic regions(11). Regarding the characteristics of the patients, the median age of those with G6PD deficiency was 20 years, slightly higher than the median age of 19 years observed in patients without G6PD deficiency. These findings align with previous research that has shown no significant association between age and G6PD deficiency in malaria patients (19). Furthermore, the gender distribution did not differ significantly between the two groups, with a similar proportion of males and females in both the G6PD-deficient and G6PD-normal groups. This observation is consistent with the lack of a strong gender-specific pattern in G6PD deficiency prevalence(20). The distribution of malaria types, specifically P. falciparum and P. vivax infections, was similar between patients with and without G6PD deficiency. Although no statistically significant differences were observed, the higher proportion of P. vivax infections among G6PD-deficient patients is consistent with previous studies(14, 21, 22) that have reported an association between G6PD deficiency and increased susceptibility to P. vivax malaria. Among the hematological parameters analyzed, the study found a noteworthy difference in the mean monocyte count between patients with G6PD deficiency and those without G6PD deficiency. Specifically, patients with G6PD deficiency had a significantly higher mean monocyte count compared to individuals without G6PD deficiency. This finding aligns with previous research that has also reported changes in monocyte count and function in individuals with G6PD deficiency(23, 24). However, it is important to note that the differences in monocyte count, although statistically significant, might not have clinical significance in isolation. Additional research is needed to understand the functional implications and potential clinical relevance of this finding in the context of G6PD deficiency and malaria infection. Interestingly, the study revealed a statistically significant difference in red cell distribution width (RDW) between patients with and without G6PD deficiency, with slightly higher values observed in patients with G6PD deficiency. However, the clinical significance of this difference remains uncertain. Further research is needed to investigate the potential relationship between G6PD deficiency, RDW, and malaria infection. The results of the univariate and multivariate analyses examining the association between G6PD status and hematological parameters, while adjusting for age, Plasmodium species, and parasite density, provided additional insights. The negative association between G6PD status and monocyte count remained significant even after adjusting for these confounding factors. This finding suggests that G6PD deficiency may influence monocyte count independently of age, Plasmodium species, and parasite density. However, the association between G6PD status and RDW was not statistically significant after adjustment, indicating that RDW might not be directly influenced by G6PD deficiency in the context of malaria infection.

Given the potential impact of G6PD deficiency on hematological parameters, it becomes crucial for healthcare providers to consider the presence of this genetic condition when diagnosing and managing malaria-infected patients. Early detection of G6PD deficiency can aid in making informed decisions regarding treatment strategies, including selecting appropriate antimalarial drugs, as certain medications may exacerbate hemolytic episodes in G6PD-deficient individuals.

Furthermore, understanding the relationship between G6PD deficiency and altered monocyte response during malaria infection may have implications for patient care and treatment. It could lead to the development of personalized therapeutic approaches targeting the immune response to malaria, which may be especially beneficial for individuals with G6PD deficiency.
Additionally, investigating the clinical implications of the observed differences in RDW may help identify potential biomarkers or predictors of disease severity in malaria-infected patients with G6PD deficiency. This knowledge could contribute to better risk stratification and improved clinical management, potentially reducing the risk of complications and improving patient outcomes.

In summary, this study underscores the significance of studying the impact of G6PD deficiency on hematological parameters in malaria-infected individuals. It highlights the need for further research to validate and expand upon these findings, as well as emphasizes the importance of integrating this knowledge into clinical practice. By better understanding the relationship between G6PD deficiency and malaria, healthcare professionals can enhance the accuracy of diagnosis, optimize treatment strategies, and potentially prevent associated complications, ultimately leading to improved patient care and outcomes.

CONCLUSION

The results of this retrospective cohort study provide valuable insights into the hematological indicators of G6PD deficiency in individuals infected with malaria. The relatively low prevalence of G6PD deficiency in the study population suggests that it may not be a common comorbidity with malaria in this specific region. However, the observed associations between G6PD deficiency and altered monocyte response during malaria infection, as well as the significant difference in red cell distribution width (RDW) between the two groups, highlight the importance of further research to fully understand the clinical implications of these findings.

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