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Glucose-6-Phosphate Dehydrogenase Deficiency in Children with Cleft Lip/Palate Attending Multidisciplinary Cleft Clinic at a Teaching Hospital in Ghana

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Abstract

Background: Cleft lip and palate are among the most common orofacial congenital anomalies worldwide known to be caused by both genetic and environmental factors. Glucose-6-Phosphate Dehydrogenase (G6PD) deficiency is an X-linked disease that affects around 400 million people globally and leads to haemolytic crisis. The main complication of these cleft surgeries is bleeding which, in a G6PD deficient child, could be prolonged and could contribute to an increase in other complications among the affected children. **Aim:** The aim of the study was to determine the prevalence of G6PD deficiency and its association with haemoglobin levels among children with cleft lip and palate attending the multidisciplinary cleft clinic at the Komfo Anokye Teaching Hospital in Kumasi, Ghana. **Method:** A cross-sectional study of consecutively selected patients attending the multidisciplinary cleft clinic at the Komfo Anokye Teaching Hospital from February-May 2025 was conducted. Consented participants' socio-demographic and clinical information were collected. A venous blood sample (3 ml) was collected based on standard procedures. G6PD enzyme activity and complete blood count were measured using the Methemoglobin reduction test and a fully automated haematology analyzer respectively. Logistic regression and their odds ratio were used to determine the association between haemoglobin (Hb) level and G6PD deficiency. **Results:** A total of ninety-one (91) participants were enrolled for the study, comprising

51 (56.0%) males and 40 (44.0%) females. A higher proportion (27.5%) presented with Veau III (cleft lip and palate), 22.0% presented with Veau II (cleft palate) and 17.6% with Veau IV (bilateral cleft lip and palate). Almost 61% of the participants were anaemic with nearly 29% having mild and moderate anaemia respectively. About 44% in the 6 - 59-month-old group had mild anaemia whilst a quarter had moderate anaemia. 12.1% (95% CI = 6.2% - 20.6%) had Glucose-6-Phosphate Dehydrogenase (G6PD) deficiency. A higher proportion of males had G6PD deficiency compared to females (OR = 4.07; 95% CI = 0.83 - 20.04). There was no statistically significant association between G6PD deficiency and anaemia [OR = 1.17 (95% CI = 0.32 - 4.31)]. **Conclusion:** Glucose-6-phosphate dehydrogenase deficiency was found in 12.1% of children with cleft lip and palate. Association between G6PD deficiency and anaemia was not statistically significant.

Keywords

Cleft Lip and Palate Orofacial Congenital Anomalies, X-Linked Genetic Disease

1. Introduction

Glucose-6-Phosphate Dehydrogenase (G6PD) is the catalyst in the rate-limiting first step of the pentose phosphate pathway, which uses glucose-6-phosphate to convert nicotinamide adenine dinucleotide phosphate (NADP) into its reduced form, nicotinamide adenine dinucleotide phosphate hydrogen (NADPH). In red blood cells (RBCs), NADPH is critical in preventing damage to cellular structures caused by oxygen-free radicals [1].

The disorder has been classified into variants based upon the degree of deficiency and associated clinical symptoms. In many cases, a mutation occurs as a new (sporadic or de novo) mutation, which means that in these cases the gene mutation has occurred at the time of the formation of the egg or sperm for that child only, and no other family member will have the mutation. In cases with a family history, the G6PD gene mutation is inherited in an X-linked manner [2].

Therefore, males are usually affected more frequently than females as males only have one X-chromosome. The highest prevalence of G6PD is reported in Africa, southern Europe, the Middle East, southeast Asia, and the central and southern Pacific island. Worldwide, more than 400 million people (90% being males) are affected by G6PD deficiency, in regions that are, or have been, endemic for malaria and in populations originating from these regions [3]-[5].

In Ghana, Amoah *et al.* found that 10.6% of children under 12 years in the southern zone were G6PD deficient. Furthermore, in a multicenter study of children under 12 years including Gabon, Ghana, (middle belt) and Kenya, Nguetse *et al.*, had an overall 13% prevalence of G6PD deficiency [6] [7].

Cleft of the lip, palate, or both is one of the most common congenital abnor-

malities and has a birth prevalence rate ranging from 1/1000 to 2.69/1000 amongst different parts of the world.

The incidence of 1 in 763 live births found in a study in Kumasi, Ghana, indicates that cleft lip/palate is a common congenital anomaly. They result from a failed fusion of the frontonasal and maxillary prominences, which normally occurs between the 6th and 10th weeks of intrauterine life. Orofacial clefts (OCs) can appear as an isolated anomaly or as a part of a multiple congenital anomaly accompanied by other non-cleft malformations. Both genetic and environmental factors are known to contribute to these congenital malformations [8].

Clefts could be of the syndromic type and develop as part of Mendelian syndromes, or they could develop as part of the clinical phenotype associated with a chromosomal anomaly. Also, prenatal exposure to certain teratogens and environmental factors has been proved to be associated with the development of orofacial clefts [9].

Sickle cell disease (SCD) is also an inherited autosomal recessive disorder of the β-globin gene, characterized by clinical manifestations such as haemolytic anaemia and recurrent episodes of vascular occlusion [10]. In Africa, SCD is a major public health problem with over 200,000 babies born per year. In Ghana, approximately 15,000 (2%) of newborns are diagnosed with SCD annually [11].

Cleft lip/palate, G6PD deficiency, and SCD all have underlying genetic disorders and prevalence for all have been ascertained. Both G6PD deficiency and SCD are associated with haemolytic crisis and children born with cleft lip/palate who undergo surgeries as part of their treatment are at risk of delayed surgeries due to anaemia or have anaesthetic complications if status of the child is not known. There is a knowledge gap in the prevalence of G6PD deficiency among children born with cleft lip and palate hence, this study.

2. Materials and Methods

2.1. Study Design

A cross-sectional study was carried out among children with cleft lip and palate attending the weekly multidisciplinary cleft clinic at the Komfo Anokye Teaching Hospital (KATH), oral and maxillofacial surgery (OMFS) unit from February to May 2025.

2.2. Study Site/Area

The study was conducted at the Komfo Anokye Teaching Hospital (KATH), Oral and Maxillofacial Surgery (OMFS) unit. Approximately 860 children with cleft lip and palate are seen at the Out-Patient-Department (OPD) yearly and 100 - 120 cleft surgeries are performed yearly.

2.3. Inclusion and Exclusion Criteria

All children with cleft lip and palate attending the multidisciplinary cleft clinic weekly aged between neonates to twelve (12) years and those yet to have surgeries

or post-surgery were included in the study. Very sick children needing admission and those whose parents or guardians refused to take part in the study were excluded.

2.4. Sample Size Determination

The sample size determination was based on the total attendance of cleft cases reported at the clinic per year. The total cases per year is eight hundred and sixty (860). Using this number of cases (N), and acceptable margin of error (e) of 10%, the sample size of 90 was found adequate using Yamane formula ($n = N/1 + Ne^2$). The cost per participant was high and the timeline was short, so a 10% margin of error allows for a representative that remains within budgetary and logistical constraints.

Moreover, a 10% margin of error is still narrow enough to prove statistical significance and support a decision.

2.5. Sample Collection and Laboratory Analysis

Assent and informed consent from children 7 - 12 years and parents and guardians of children below 7 years of age were obtained respectively from consecutively selected patients. Demographic characteristics of both child and parents were recorded. Blood sample collection and analysis was done at the KATH laboratory. Three (3) ml of blood was taken from the cubital fossa of the children, using a 5 ml syringe and needle and kept in ethylenediaminetetraacetic acid (EDTA) tubes for analysis using the Methemoglobin reduction test. When test result maintained the brown colour of the positive control, then, G6PD deficiency was positive and negative when the test maintains a clear red colour as the negative test [12]. A complete blood count was done using part of the blood sample taken in the EDTA bottle using the fully automated hematology analyzer, that employs the volumetric impedance and light scatter technique to count blood cells [13].

Haemoglobin levels were age based as recommended by the Harriet Lane Handbook and World Health Organization (WHO) [14] [15].

2.6. Statistical Analysis

Data was analyzed in SPSS (version 25). Categorized variables were summarized as frequencies and proportions. Chi-squared test was used to analyze the differences in G6PD deficiency between sex and age. Logistic regression and their odds ratio were used to determine the association between G6PD deficiency and haemoglobin levels. A p-value less than 0.05 was considered statistically significant.

2.7. Ethical Consideration

Ethical clearance for the study was sought from the institutional review board of the KATH (Protocol ID: KATH IRB/AP/002/25.Written informed consent was obtained from the parents and guardians of participants who were below 7 years and assent (mostly verbal) was obtained from children 7 - 12 years recruited after an

explanation of the study. Refusal to participate did not interfere with their treatment. The cost of all investigations related to this study was borne by the principal investigator. Data obtained during the course of the study was kept confidential.

3. Results

A total of ninety-one (91) participants were enrolled for the study, comprising 51 (56.0%) males and 40 (44.0%) females with more than 50% being 6 - 59 months old. Nearly one-third had had neonatal jaundice once and 70% did not have neonatal jaundice. A higher proportion (27.5%) presented with Veau III (cleft lip and palate), 22.0% presented with Veau II (cleft palate) and 17.6% Veau IV (bilateral cleft lip and palate) (Table 1).

Table 1. Demographic and clinical characteristics of patients.

Variable	n (%)
Sex	
Male	51 (56.0)
Female	40 (44.0)
Age (in months)	
<6 months	23 (25.3)
6 - 59 months	48 (52.7)
5 - 11 years	20 (22.0)
Neonatal Jaundice	
Once	26 (28.6)
Twice	1 (1.1)
None	64 (70.3)
Type of Cleft	
Bilateral Tessier 2, 3, 11	1 (1.1)
Bilateral Tessier 4	1 (1.1)
Right Tessier 3	1 (1.1)
Bilateral Tessier 7	1 (1.1)
Right Tessier 7 and Veau II palate	1 (1.1)
Left unilateral complete cleft lip and alveolus	5 (5.5)
Left unilateral incomplete cleft lip	2 (2.2)
Left unilateral complete cleft lip	4 (4.4)
Left Veau III	14 (15.4)
Right unilateral complete cleft lip	3 (3.3)
Right unilateral complete cleft lip and alveolus	6 (6.6)
Right unilateral incomplete cleft lip	4 (4.4)
Right Veau III	11 (12.1)
Veau I	1 (1.1)
Veau II	20 (22.0)
Veau IV	16 (17.5)

There was no statistically significant difference in the cleft type between male and female participants. A total of 51% males had unilateral cleft lip while 52.5% females had unilateral cleft lip. Slightly more males had cleft palate alone 23.6% (Veau 1 and 11) and 22.5% females had cleft palate alone (Table 2).

Table 2. Cleft type between male and female participants.

	Sex, n (%)		
Cleft Type	**Male**	**Female**	**p-value**
Bilateral Tessier 2, 3, 11	1 (2.0)	0 (0.0)	
Bilateral Tessier 4	0 (0.0)	1 (2.5)	
Bilateral Tessier 7	0 (0.0)	1 (2.5)	
Left unilateral Incomplete cleft lip	0 (0.0)	2 (5.0)	
Left unilateral complete cleft lip	1 (2.0)	3 (7.5)	
Left unilateral complete cleft lip and alveolus	4 (7.8)	1 (2.5)	0.549
Left Veau III	9 (17.6)	5 (12.5)	
Rt Tessier 3	0 (0.0)	1 (2.5)	
Right Tessier 7 and Veau II palate	1 (2.0)	0 (0.0)	
Right unilateral complete cleft lip	1 (2.0)	2 (5.0)	
Right unilateral complete cleft lip and alveolus	3 (5.9)	3 (7.5)	
Rt unilateral incomplete cleft lip	2 (3.9)	2 (5.0)	
Right Veau III	6 (11.8)	5 (12.5)	
Veau I	1 (2.0)	0 (0.0)	
Veau II	11 (21.6)	9 (22.5)	
Veau IV	11 (21.6)	5 (12.5)	

Almost 61% of the participants were anaemic with nearly equal proportions having mild and moderate anaemia. About 44% in the 6 - 59 months old group had mild anaemia whilst a quarter had moderate anaemia. More than one-third of 5 - 11 years old had moderate anaemia whilst 15% had mild anaemia (Table 3).

Table 3. Age-based haemoglobin levels among children.

Child age (in months)	**Hb level g/dl, n (%)**			
	Normal	**Mild**	**Moderate**	**Severe**
<6 months	11 (47.8)	3 (13.0)	7 (30.4)	2 (8.7)
6 - 59 months	15 (31.2)	21 (43.8)	12 (25.0)	0 (0.0)
5 - 11 years	10 (50.0)	3 (15.0)	7 (35.0)	0 (0.0)
Total	**36 (39.6)**	**27 (29.7)**	**26 (28.6)**	**2 (2.2)**

Classified according to WHO classes of Anemia.

12.1% (95% CI = 6.2% - 20.6%) presented with Glucose-6-Phosphate Dehydrogenase (G6PD) deficiency. Although not statistically significant, a higher proportion of males were found to have G6PD deficiency compared to females (OR = 4.07; 95% CI = 0.83 - 20.04) (Table 4). A higher proportion of Children (<6 months) were found to have G6PD deficiency compared to the other age groups.

Table 4. Prevalence of G6PD deficiency according to sex and age among children.

	G6PD status, n (%)		
Variable	Deficiency	No Deficiency	p-value
Sex			
Male	9 (17.6)	42 (82.4)	0.066
Female	2 (5.0)	38 (95.0)	
Age			
<6 months	5 (21.7)	18 (78.3)	
6 - 59 months	5 (10.4)	43 (89.6)	0.214
5 - 11 years	1 (5.0)	19 (95.0)	

Approximately 64% with G6PD deficiency were anaemic (mild to moderate). Association between G6PD deficiency and anaemia was not statistically significant. [OR = 1.17 (95% CI = 0.32 - 4.31)] (Table 5).

Table 5. Association between G6PD deficiency and Haemoglobin (Hb) levels.

	Hb level g/dl, n (%)					
	Normal	**Mild**	**Moderate**	**Severe**	**OR (95% CI)**	**p-value**
G6PD status						
Deficiency	4 (36.4)	4 (36.4)	3 (27.3)	0 (0.0)	1.17 (0.32 - 4.31)	0.817
No deficiency[a]	32 (40.0)	23 (28.7)	23 (28.7)	2 (2.5)	[a]	

[a]Reference category for logistic regression.

4. Discussion

Cleft of the lip, palate, or both is one of the most common congenital abnormalities and has an incidence of 1 in 763 live birth in Kumasi, Ghana. The current study recorded more males (56%) than females (44%) which is comparable to other studies that noted more males than females [16].

There was no statistical difference in the prevalence of cleft lip and palate and cleft palate alone among both males and females which is in contrast to earlier studies that noted more females than males with cleft palate alone (CPO occurs more in females (57%) than in males (43%)). Left-sided clefts are twice as common as right-sided ones as stated by Azaria but in this present study, there was no

difference between the prevalence of right and left sided cleft lip and palate. This could be attributed to the number of participants (91) enrolled for this study as compared to other studies involving a minimum of 200 and above participants [17] [18].

Many participants (61%) in this study were found to be anaemic according to WHO age based haemoglobin levels. Again, up to 63% of the under-five (5) years participants in this current study were found to be anaemic (mild to moderate) and agrees with findings by Tadesse *et al.* [19]. The results of this study, indicates that children under five years still need a lot of attention from both nutritionist and paediatricians in the team particularly for these children with cleft lip and palate who already have issues with feeding.

Cleft lip/cleft palate (CL/CP) and craniofacial syndromes are often associated with special challenges with regards to the feeding and swallowing process in infancy, and beyond in certain circumstances. Problems vary depending on the degree of clefting and if a craniofacial syndrome is present. Severe feeding and swallowing issues have the potential to result in nutritional and/or respiratory compromise, creating significant stress for families and caretakers. There is the need for more education at the antenatal and post-natal clinics for the parents and guardians on how to avoid anaemia in the children, as well as nutrition and paediatric care for these children [20] [21].

The gene for G6PD is located on the X-chromosome, and G6PD deficiency is inherited in a sex-linked fashion, being fully expressed in hemizygous males and homozygous females, but in only a proportion of female heterozygotes due to X-inactivation effects. Unlike G6PD deficiency, the inheritance of CL/P has a multifactorial etiology, comprising both environmental and genetic factors. For families with a history of CL/P, the risk to subsequent children is dependent on familial involvement. If one child or one parent has CL/P, then there is a 4% risk to subsequent children. If two children have CL/P, the risk increases to 9%. If one child and a parent have CL/P, then the risk increases to 17%. For patients with syndromic etiologies for CL/P, such as Van der Woude syndrome, the risk to subsequent children follows Mendelian inheritance patterns increasing the risk to subsequent children being born with CL/P to 50% [22] [23].

G6PD deficiency is common globally, particularly in African populations (14% of males). Individuals with the deficiency are at risk of haemolytic anaemia which can be triggered by infections, certain foods, or medications. This study also found that 12.1% of the participants with cleft lip and palate had G6PD deficiency and also a seventeen (17) times risk of anaemia which agrees with T. Motshoge *et al.*, who found that G6PD A-form (G6PD deficiency), was associated with low RBC count and haemoglobin levels without a known cause or illness in their study of prevalence of G6PD deficiency and associated haematological parameters in children from Botswana. These children undergo surgeries at specific timelines and anaemia will not help wound healing leading to wound breakdown, fistulas and abnormal scarring and eventually, increase the cost of management. There is the

need to adequately prepare them before, during, and after surgery [22] [24].

The global prevalence of G6PD deficiency based on DNA analysis is estimated at 7.1%, with Africa estimated to have a prevalence of 24%, and a prevalence of 12.4% in Ghana. In this current study, 12.1% of the participants were found to have G6PD deficiency which is close to the national prevalence of 12.4%. The results emphasize the fact that children with cleft lip and palate can also have G6PD deficiency and should be part of the routine laboratory investigations to obtain baseline information, to prepare the children adequately before, during, and after surgery [25].

In general, there is insufficient evidence regarding medication use, including that of anaesthetic agents, in G6PD-deficient patients. One in vitro study revealed that isoflurane, sevoflurane, diazepam, and midazolam have an inhibitory effect on G6PD activity. However, sevoflurane and midazolam are controversially discussed in another review. Ketamine does not cause G6PD inhibition and can be used as an intravenous anaesthetic for G6PD deficient patients. Anaesthetic management should focus on avoiding the drugs implicated in haemolysis, and monitoring for and treating the haemolysis, should it occur [26]-[28].

The most effective management strategy is to prevent hemolysis by avoiding oxidative stressors. Therefore, management for pain and anxiety should include medications that are safe and have not been shown to cause hemolytic crises, such as benzodiazepines, codeine/codeine derivatives, propofol, fentanyl, and ketamine. Also, parents and guardians of the children with G6PD deficiency should be educated and as the children grow, they should be educated on their condition especially on the need to avoid Sulphur drugs, naphthalene etc. so as to avoid haemolytic crisis and hence anaemia [29].

5. Limitations

The sickle cell status which if present, could also lead to anaemia could not be ascertained for majority of the participants who had G6PD deficiency due to lack of funds and some of the participants not coming for follow up after their blood samples were taken. Also, wound outcomes were not done for those who had surgery during the study period and would be included in the next study.

6. Conclusion

Glucose-6-phosphate dehydrogenase deficiency was found in 12.1% of children with cleft lip and palate. Association between G6PD deficiency and anaemia was not statistically significant.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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