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Research Article

Prevalence of Glucose-6-Phosphate Dehydrogenase in Children Attending Pediatric Clinic among Selected Hospitals in Abakaliki

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Abstract:

Glucose-6-phosphate dehydrogenase deficiency is prevalent worldwide and common in malaria-endemic regions, affecting over 400 million people globally. G6PD deficiency compromises erythrocyte protection against oxidative stress, potentially leading to haemolysis. This study evaluated the prevalence of G6PD deficiency among children attending paediatric clinics in selected hospitals in Abakaliki, Ebonyi State, Nigeria. This cross-sectional study sampled 100 subjects aged less than one year to 13 years with equal gender representation. Venepuncture and standard laboratory practices were employed for blood sample collection from consented children falling within the specified age range. Brewer methaemoglobin reduction method and thin blood film analysis were conducted for investigation. Statistical analysis, were performed using the Statistical Package for Social Science version 26 to compare G6PD activities across age groups and genders. Results indicated varying deficiency rates across age groups, with the highest prevalence observed in the 2-4-year-old group (9%). Males exhibited a higher deficiency rate (12%) compared to females (7%), culminating in a 19% overall prevalence among paediatric clinic attendees. While significant differences in G6PD activity were found between males and females (p = 0.00), no significance was noted across age groups (p=0.29). The study emphasises the importance of G6PD screening in children given the relatively high prevalence observed in Abakaliki.

Keywords: Prevalence, Gucose-6-phosphate dehydrogenase, Deficiency, Children, Paediatric

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Introduction

Glucose phosphate dehydrogenase causes red blood cells to break down prematurely (irreversible damage), resulting in acute and chronic haemolysis including neonatal hyperbiliribinaemia (1). Glucose-6-phosphate dehydrogenase was discovered by Alving and coworkers in 1956, when they investigated the unusual haemolytic reaction that occurred in black individuals following the administration of primaquine. Primaquine is an 8-aminoquinoline that is used for the treatment of malaria. Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most common enzyme deficiency in humans, affecting 400 million people worldwide (2). It has a high prevalence in Africa, Asia, and the Mediterranean. It is inherited as an X-linked recessive disorder. G6PD deficiency is polymorphic, with more than 300 variants (3). G6PD disorder most commonly affects people of African, Asian, Mediterranean, or Middle-Eastern descent. In Nigeria, the prevalence of G6PD deficiency ranges from 4% to 26% 8. A study by Akanni et al in Oshogbo, Nigeria among 86 jaundiced neonates indicated G6PD deficiencies of 19.5% and 47.7%, respectively (4). G6PD deficiency occurs most frequently in individuals of African descent, with frequency ranging from 3.6% to 28.0% 11. In Asia, the deficiency prevalence ranges from 6.0% to 15.8% (5). In India, it is 10.5%, and in the Middle East, the prevalence varies from 3% to 29% (6). In the United States, prevalence is highest among men of black and Mediterranean descent (10% to 14% 16) (7). Additionally,

the World Health Organisation has categorised G6PD deficiency based on degree of enzyme deficiency and degree of haemolysis caused, they include; class 1 variants, which are extremely deficient and are associated with non-spherocytic haemolytic anaemia. Class 11 variants have less than 10% of normal activity with residual activity and are usually not associated with non-spherocytic haemolytic anaemia. Class 111 variants normally have 10–60% normal activity (moderate–mild activity), Class 11 and 111 variants are related to periodic haemolysis triggered by oxidative stress in the form of infection, medications, or food. Class 1V variants have normal enzyme activity of about 60–150% (very mild or no deficiency), Class V variants have increased enzyme activity (> 200%) (1). The global distribution of G6PD deficiency is marked by considerable variability, with specific populations and geographic regions demonstrating higher prevalence rates. The prevalence of this condition is not only dictated by genetic determinants but also involvedly linked with various comorbidities, such as sickle cell disease, which can exacerbate the clinical impact of G6PD deficiency. In the paediatric context, understanding the prevalence of G6PD deficiency is of paramount importance. Early detection and effective management of this enzymatic deficiency become critical components of paediatric care, particularly in regions where prevalence rates may be elevated. Abakaliki, nestled in the southeast geopolitical zone of Nigeria, emerges as a unique locale with distinctive genetic and environmental factors that may contribute to the prevalence of G6PD deficiency among its paediatric population. G6PD deficiency has been recognised as a significant public health concern, influencing the morbidity and mortality of affected individuals, especially in areas where infectious diseases such as malaria are prevalent. The interaction between G6PD deficiency and malaria, characterized by oxidative stress, has been extensively studied, underscoring the importance of understanding the prevalence of this enzymatic disorder in populations where malaria is endemic. Abakaliki, as the focal point of this investigation, indicates attention due to its diverse demographic composition and healthcare landscape (4). The prevalence of genetic disorders often exhibits regional variations, and Abakaliki's unique genetic makeup, coupled with environmental factors, warrants a dedicated exploration of the prevalence of G6PD deficiency in its paediatric population. This study unfolds against the backdrop of three prominent healthcare facilities in Abakaliki, namely Mile 4 hospitals Abakaliki, the Federal Teaching Hospital Abakaliki, and Cape Town clinic Abakaliki. These institutions serve as crucial nodes in the healthcare network of the region, providing insights into the prevalence of G6PD deficiency among children seeking paediatric care. Significance of the Study: This research endeavours to contribute significantly to the existing body of knowledge on G6PD deficiency, specifically focusing on the paediatric population in Abakaliki. The significance of the study can be defined across multiple dimensions: to uncover insights into the clinical manifestations and impact of G6PD deficiency on paediatric patients, guiding clinicians in diagnosis and management, provide a foundation for targeted public health interventions, including genetic counselling and awareness programs, to mitigate the impact of G6PD deficiency, propel further research by elucidating potential genetic and environmental determinants of G6PD deficiency, paving the way for more nuanced investigations, inform healthcare policies tailored to the unique needs of the paediatric population in Abakaliki, aligning healthcare practices with the prevalence patterns of G6PD deficiency. As the study unfolds, it is anticipated that the findings will not only contribute to the local understanding of G6PD deficiency but will also offer broader implications for paediatric healthcare in regions with similar genetic and environmental contexts. This comprehensive exploration aims to bridge existing gaps in knowledge, fostering a holistic approach to the health and well-being of children affected by G6PD deficiency in Abakaliki. The primary objective of this research is to unravel the prevalence of G6PD deficiency in children attending paediatric clinics within selected hospitals in Abakaliki. This main goal is fortified by specific objectives, which include' conducting a comprehensive examination of the prevalence rates of G6PD deficiency among paediatric patients in the selected hospitals, investigating potential demographic variations in G6PD deficiency, including age and gender distribution among the affected children, investigate potential genetic and environmental factors influencing the prevalence of G6PD deficiency in the paediatric population of Abakaliki Ebonyi state.

Materials And Method

Study Area

The study area for this research comprises Abakaliki metropolis, situated in Ebonyi State, Nigeria. Ebonyi State is prominently positioned in the southeastern geopolitical zone of Nigeria, showcasing a rich cultural and geographical landscape. The state is characterized by its diverse population, and its capital, Abakaliki, serves as a hub of various economic, social, and healthcare activities. Abakaliki, being the epicentre of the study, is a bustling city that captures the unique blend of urban and regional dynamics. Its strategic location within the southeastern region makes it an ideal setting for research endeavors, providing insights into local health issues and trends. The metropolis is home to various healthcare facilities, contributing significantly to the overall health infrastructure of the state. Three key hospitals were targeted for the cross-sectional study, each playing a distinct role in the healthcare ecosystem of Abakaliki. The selected hospitals are Mile 4 hospitals Abakaliki, Federal Teaching Hospital Abakaliki, and Cape Town clinic Abakaliki. These healthcare institutions were chosen based on their prominence, accessibility, and the diversity of patient populations they serve. Mile 4 hospitals Abakaliki, founded by missionaries, for instance, is a cornerstone of healthcare delivery in the city, catering to a wide range of medical needs. The Federal Teaching Hospital Abakaliki, as a tertiary healthcare institution, adds a critical dimension to the study, providing insights into more complex medical cases and offering specialized services. Its affiliation with medical education also makes it a focal point for understanding health issues in a broader context. Cape Town Clinic Abakaliki, being a private healthcare facility, introduces an element of diversity into the study by encompassing the perspective of private healthcare provision. This inclusion is essential for capturing a comprehensive overview of the healthcare landscape in Abakaliki, considering both the public and private healthcare sectors. Ebonyi State, with its distinctive sociodemographic characteristics, provides a microcosm of the larger southeastern Nigerian population. The people of Ebonyi State are known for their rich cultural heritage, and their lifestyle and health practices contribute to the overall health profile of the region. The cross-sectional nature of the study allows for the collection of data at a specific point in time, offering a snapshot of the prevalence and characteristics of G6PD deficiency among children in the selected hospitals. By including diverse healthcare settings within Abakaliki metropolis, the research aims to generate findings that are reflective of the broader health scenario in the southeastern region of Nigeria.

Study Population

The study was conducted among 100 children comprising, 50 females and 50 males between the ages of 6 months to 13 years.

Eligibility criteria

The eligibility criteria for participant inclusion in this study were meticulously designed to ensure a comprehensive yet targeted examination of glucose-6-phosphate dehydrogenase (G6PD) deficiency among children. The study focused on children within the age range of 6 months to 13 years old, recognising this developmental span as crucial for understanding the prevalence and manifestations of G6PD deficiency in the paediatric population. To guarantee the relevance and accuracy of the findings, inclusion criteria were limited to children actively attending the designated healthcare facilities: Mile 4 hospitals Abakaliki, Federal Teaching Hospital Abakaliki, and Cape Town clinic Abakaliki. This strategic selection aimed to create a cohort that accurately represented the local population seeking healthcare services in the specified study area of Abakaliki metropolis, Ebonyi State. Parental involvement was a vital aspect of the study, and therefore, all parents of the participating children were actively counselled about the research objectives and procedures. Informed consent, obtained after thorough counselling, was a prerequisite for inclusion in the study. This ethical safeguard ensured that parents were well-informed about the study's purpose, potential benefits, and any associated risks, allowing them to make informed decisions regarding their child's participation. Conversely, exclusion criteria were established to maintain the study's focus and integrity. Children below 6 months and above 13 years were excluded from adhering strictly to the predefined age range. Additionally, children not receiving care at the specified healthcare facilities and those whose parents did not provide informed consent were excluded. This strict approach was implemented to uphold ethical standards, safeguard participant welfare, and

ensure that the study's outcomes accurately reflected the targeted population within the chosen healthcare facilities.

Sampling Method

The sampling method employed in this cross-sectional study was a simple random sampling technique. The subjects considered for inclusion were those attending Mile 4 hospitals Abakaliki, Federal Teaching Hospital Abakaliki, and Cape Town clinic Abakaliki who met the predefined inclusion criteria and provided informed consent. Through a random selection process, subjects were chosen until the required sample size of 100 children was attained. This method ensured a fair and unbiased representation of the paediatric population seeking healthcare services in the designated facilities, contributing to the generalizability of the study's findings. The use of simple random sampling added a layer of objectivity to participant selection, enhancing the study's reliability and providing a snapshot of the prevalence of glucose-6-phosphate dehydrogenase deficiency among children in the specified healthcare settings.

Sample Collection

Blood sample was collected by venepuncture technique. Blood sample were collected by a trained phlebotomist. 5 mL blood was obtained from each participant. After collection, blood samples were transferred into an ethylene diamine tetra acetic acid (EDTA) tube to prevent blood coagulation. Sample was used for brewer methaemoglobin reduction test and thin blood film.

Sample Analysis

The samples were labelled with unique identification codes.

Brewer Methaemoglobin Reduction Test.

Its principle is based on the conversion of haemoglobin (Hb) to methaemoglobin (Hi) by sodium nitrate. When no methylene blue is added, methaemoglobin persists. But incubation of the samples with methylene blue allows stimulation of the pentose phosphate pathway in subjects with normal G6PD levels. Methaemoglobin is reduced during the incubation period. In G6PD-deficient subjects, the block in the pentose phosphate pathway prevents reduction(8).

Thin Blood Film: Blood film was prepared using the push wedge method and stained using a Romanowsky stain (Leishman stain).

A drop of blood was gently touched onto one end of a clean, grease-free slide. A spreader was placed at a suitable angle in front of a blood vessel, and the blood was allowed to touch and spread along the edge of the spreader. The spreader was pushed along the slide, drawing the blood behind it, until the whole drop had been smeared. This was labelled and kept to air dry. This method permits the study of the morphology and density of the parasites and the condition of the blood corpuscles (9)

Statistical Analysis

Statistical Package for Social Science version 26 was used to analyze data. Frequency and percentage were obtained. Also, Kruskal-wallis test and Mann-Whitney test were used to compare difference in G6PD activities across age groups and sex at 0.05 level of significance. All results were presented in tables and charts.

Result

The study investigated the prevalence of Glucose-6-Phosphate Dehydrogenase in children attending pediatric clinics among selected hospitals. A total of 100 subjects (50 male subjects and 50 females) were involved in this study, demonstrating equal frequency (50 male, 50 female) and percentage distribution 50% male and 50% female)

Variable	Classification	Frequency (N =100)	Percentage (%)
Sex	Male	50	50%
	Female	50	50%
	Total	**100**	**100%**

Table 1: showing the Frequency Distribution of Socio-Demographics of the Study Subjects based sex distribution

Table 2 represents a frequency distribution for different age groups which are classified into distinct categories: ≤1 year, 2 - 4 years, 5 - 7 years, 8 - 10 years and 11 - 13 years. ≤1 year had frequency distribution of 7(7%), 2 - 4 years had frequency distribution of 32(32%), 5- 7 years had frequency distribution 21(21%), frequency of 27(27%) for age 8 - 10 years, and frequency of 13(13%) for age groups 11- 13 years. The total frequency sums up to 100. Additionally, the table reveals a complete distribution, with age group 2-4years constituting the largest proportion at 32%

Variable	Classification	Frequency (N =100)	Percentage (%)
Age (Years)	≤1year	7	7%
	2yrs - 4yrs	32	32%
	5yrs - 7yrs	21	21%
	8yrs - 10yrs	27	27%
	11yrs- 13yrs	13	13%
	Total	**100**	**100%**

Table 2: showing the Frequency Distribution of Socio-Demographics of the Study Subjects based age distribution.

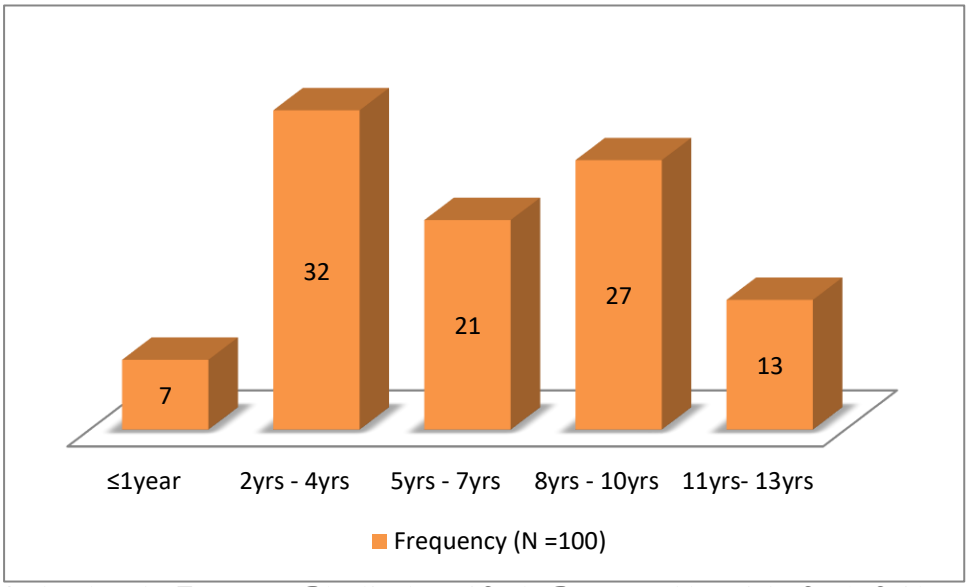

Figure 1: showing the Frequency Distribution of Socio-Demographics of the Study Subjects based age distribution

Table 3 indicates distinctive patterns in G6PD activity (deficient, intermediate and normal activity) across gender. 12 males were deficient exhibiting 24% of deficient G6PD activity, which have clinical implications. 5 males were intermediate exhibiting 10% of *intermediate G6PD* activity indicating level between deficiency and normalcy, potentially indicating a milder form of G6PD deficiency. 33 males were normal representing 66%, majority of males displayed normal G6PD activity. 7 females were deficient representing 14% of deficient G6PD activity. G6PD deficiency in females is generally milder due to the protective effect of the second X chromosome. 4 females were intermediate, small percentage of females (8%) fall within the intermediate G6PD activity range, indicating a potential range of enzyme activity that may warrant monitoring. 39 females were normal, majority of female's 78% exhibit normal G6PD activity. This aligns with the expected distribution, as G6PD deficiency is less common in females, and the majorities are expected to have normal enzyme activity. The combined results across genders reveal an overall prevalence of 19% for deficient G6PD activity, 9% for intermediate, and 72% for normal activity. This comprehensive analysis provides an in depth understanding of G6PD activity in the studied population, emphasizing gender-specific variations and potential clinical implications.

G6PD Activity	Male (%)	Female (%)	Total
Deficient	12 (24%)	7 (14%)	19 (19%)
Intermediate	5 (10%)	4 (8%)	9 (9%)
Normal	33 (66%)	39 (78%)	72 (72%)
Total	**50 (100%)**	**50 (100%)**	**100**

Table 3: Bar chart showing distribution of G6PD activity within male and female

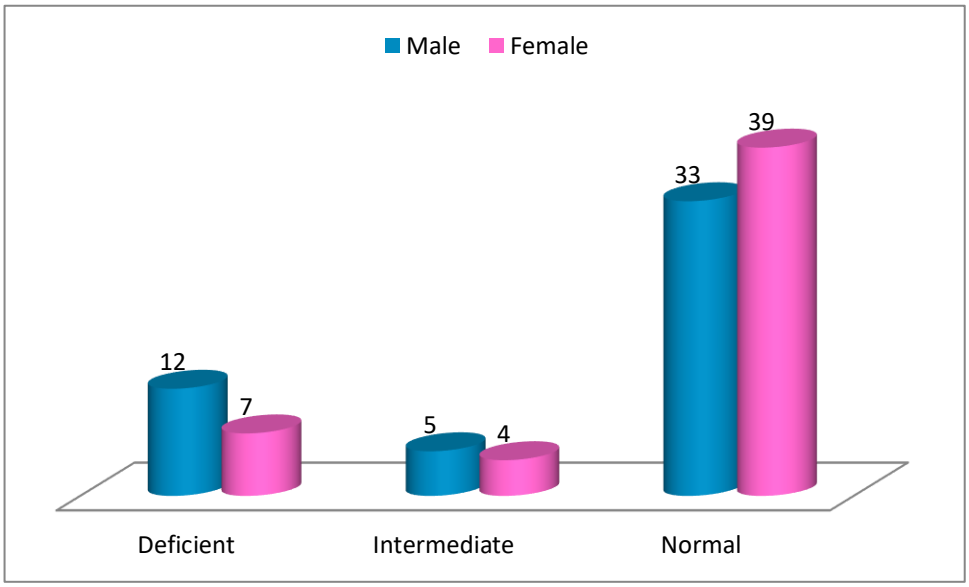

Figure 2: Bar chart showing distribution of G6PD activity within male and female

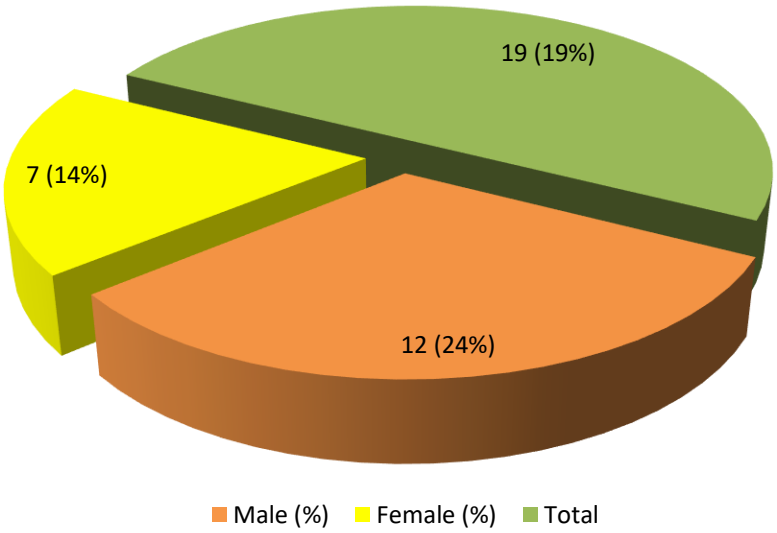

Figure 3: Pie Chart showing Prevalence of G6PD Deficiency among male and female.

Figure 3 shows the prevalence of G6PD deficiency among subjects with respect to sex, the prevalence of G6PD among subjects with respect to sex for male is 24%, the prevalence for female is 14%. G6PD deficiency is more in males than female with a ratio of 12:7, the total prevalence of G6PD deficiency among children attending pediatric clinics in selected hospital at Abakaliki is 19%.

Figure 4: Figure showing various result interpretations during analysis

The presence of a red color in the test tube indicates normal G6PD activity within the sample. Conversely, a brown color suggests a deficiency in the G6PD enzyme, indicating reduced enzymatic activity, typically found in hemizygous males or homozygous females. Meanwhile, a blue coloration suggests a partial defect in G6PD activity, often observed in heterozygous females, reflecting an intermediate level of enzymatic function.

Figure 5 provides G6PD activity across age groups, 4 out of 7 subjects within ≤1 year were normal majority of individuals in this age group exhibited 57% normal G6PD activity, signifying a relatively balanced enzyme function during the first year of life. 2 subjects were intermediate; a notable portion (28%) falls into the intermediate range. 1 subject was deficient, smaller percentage (14%) shows deficient G6PD activity. Also, among the 32 subjects within age group 2- 4 years 19 were normal. 59% of subjects in this age bracket exhibit normal G6PD activity. Among this group, 4 were intermediate a lower percentage (12%) falls into the intermediate category. 9 subjects within these group were deficient, a significant proportion (28%) shows deficient G6PD activity. Among 21 subjects within age Group 5 - 7 years 16 were normal, high percentage (76%) demonstrates normal G6PD activity in this age group, aligning with the expectation of stable enzyme function during middle childhood. 2 subjects were intermediate, a relatively small percentage (9%) falls into the intermediate range, suggesting a mild deviation from normal G6PD activity. 3 subjects within this age group were deficient, a moderate proportion percentage of 14% shows deficient G6PD activity, emphasizing the need for continued vigilance in managing potential risks. Among 27 subjects within age Group 8- 10 years 22 were normal, 81% showcases normal G6PD activity, indicating robust enzyme function during late childhood. 1 subject was intermediate, minimal percentage (3%) falls into the intermediate category, signifying a relatively low prevalence of milder G6PD deficiency. 4 subjects within this age group were deficient, 14% displays deficient G6PD activity. 11 out of 13 subjects were normal among age Group 11- 13 years, significant majority (84%) demonstrates normal G6PD activity in this age range, indicating a prevailing pattern of stable enzyme function during adolescence. No subjects exhibited intermediate G6PD activity in this age group. 2 subjects were deficient within the age group, a notable but relatively smaller proportion (15%) exhibits deficient G6PD activity.

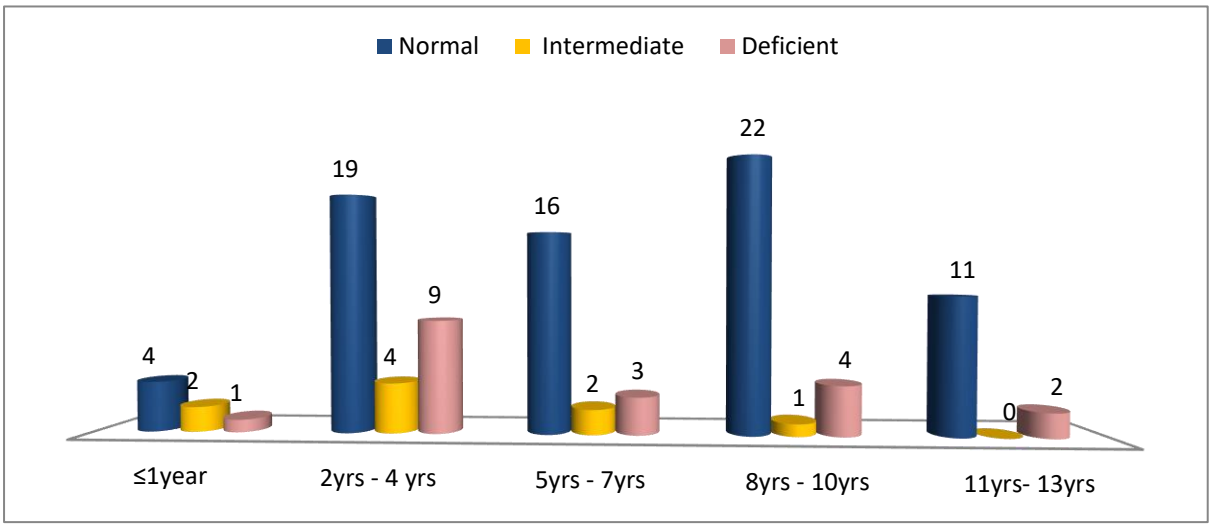

Figure 5: Bar Chart Showing G6PD activity across various ages.

Table 5 describes the statistical analyses for two variables Sex and Age along with their corresponding p-values and decisions: Mann-Whitney U test was used to analyze difference between male and female, Kruskal Wallis test was used to evaluate differences across age groups. P-value of 0.00 for sex indicates there's a significant difference (meaningful) difference, while for "Age," the observed differences are not statistically significant. The p-value of 0.29 suggests that there is no statistically significant difference in the variable "Age" across the groups. Therefore, the Kruskal Wallis test concludes that age does not significantly differ among the age groups under consideration.

Variables	Test	p-value	Decision
Sex	Mann-Whitney U	0.00	Significant
Age	Kruskal Wallis	0.29	Not significant

P<0.05(Significant), p>0.05(Not Significant)

Table 5: Mann-Whitney u and Kruskal Wallis tabl

Discussion

G6PD deficiency is the most common human enzyme deficiencies in the world, the evaluation of G6PD activity levels across different categories sheds light on the prevalence rates within the study population. In the overall analysis of the study, 19% of the participants in Abakaliki were identified as G6PD deficient, while 9% exhibited intermediate activity and 72% showed normal enzyme activity. The prevalence of G6PD deficiency, though not alarmingly high, is significant, considering the potential health implications, especially in a paediatric cohort. The prevalence of G6PD deficiency gotten in the study is similar to the research done at Usmanu Danfodiyo University Teaching Hospital in Sokoto, where 17% of G6PD-deficient children were recorded (10). It also correlates with the study of Akanni et al in Oshogbo, Nigeria among some children which indicated G6PD deficiencies of 19.5 % (4)

In research carried out by Brearley et al 2013 among Nigerians of different ethnic groups. 1,122 children (561 males and 561 females) were screened for G6PD deficiency, and it was discovered that the overall prevalence of G6PD deficiency was 15.3% (24.1% in males, 6.6% in females). Yoruba children had a higher prevalence (16.9%) than Igede (10.5%), Igbo (10.1%), and Tiv (5.0%) children (11). The regional prevalence of G6PD deficiency in Africa ranges from 15 to 26% (12). The prevalence of G6PD is generally lower among Asian

populations than sub-Saharan Africans. In the United States, African Americans are primarily affected, with a prevalence of about 10%; however, it is also seen among Italians (especially those of Sardinian ancestry), Greeks, Turkish people, Southeast Asians, people of Asian ancestry, and Sephardic Jews (13). G6PD deficiency prevalence in India is 10.5%, and in the Middle East, the prevalence varies from 3% to 29%. In Brazil, a few studies have found prevalence between 1.7% and 6.0% (13). Internationally, the geographic prevalence of the disorder correlates with the distribution of malaria. The highest prevalence rates (with gene frequencies ranging from 5-25%) are found in the following regions: tropical Africa, the Middle East, tropical and subtropical Asia, and some areas of the Mediterranean.

Furthermore, the sex distribution in this study showed that 12(24%) of the deficient children were males and 7(14%) were females. This gender-based variation is in line with existing literature, which often reports a higher prevalence of G6PD deficiency in males due to the X-linked inheritance pattern. However, the difference observed in this study deserves careful consideration, as it may have implications for gender-specific healthcare interventions. Additionally, this study is similar to a study carried out in the Eastern Province, which has the highest prevalence of G6PD deficiency in Saudi Arabia in both males and females, Males had a higher G6PD deficiency rate (26%) than females (9.9%) (18). In another study carried out by Adias et al in March 2012 at Usmanu Danfodiyo University Teaching Hospital, Sokoto, Nigeria, for G6PD deficiency among 118 children. It was found that 17 (14.4%) were G6PD-deficient, and the prevalence of G6PD deficiency was concentrated predominantly among male children (22.1%) (10). Furthermore, research on glucose-6-phosphate dehydrogenase deficiency in children from Brazzaville, Republic of Congo, two hundred and twelve children were successfully genotyped for G6PD variants. Overall, 13% (27/212) of the children were G6PD deficient, and 25% (25/100) females were heterozygous (11 BA− and 14 A+A−). The remaining 160 children had a normal G6PD genotype. Additionally, in this study, there was a significant difference between genders, which is similar to various studies. In a study by Jidda and colleague they had significant difference between gender out of the 164 subjects tested, 144 (87.8%) were normal while 20(12.2%) were G6PD deficient out of the 82 male subjects studied, 64 (78%) were normal while 18 (22%) were G6PD deficient compared to 80 (97.6%) and 2 (2.4%) females subjects who were normal and deficient for G6PD respectively (16). This is because G6PD deficiency is an X-linked recessive disorder, and the fact that only male hemizygotes and female homozygotes are most often affected. Female heterozygote's who are G6PD deficient, acquired this through the phenomenon of normal X-chromosome inactivation of the Lyon-Hypothesis(15).

In this study, analysing G6PD deficiency prevalence across different age groups illuminates interesting tendencies. Among children aged 2 to 4 years, there is a notable spike in G6PD deficiency 9 (28%), highlighting a potential vulnerability during this developmental stage. The prevalence gradually decreases in older age groups, with the lowest prevalence observed in the 11- to 13-year-old category. This age-specific variation underlines the importance of considering developmental factors in understanding the prevalence of G6PD deficiency. This is similar to the study carried out by Adias et al in March 2012 at Usmanu Danfodiyo University Teaching Hospital, Sokoto, Nigeria, for G6PD deficiency among 118 children. The highest prevalence occurred among children between the ages 2- to 5-years of the 17 G6PD-deficient children (10). Additionally, in this study, blood film from G6PD-deficient children indicated the following morphological changes; Heinz bodies, schistocytes, target cells, nucleated red cells, spherocytes, and polychromasia. suggesting haemolytic anaemia this is because G6PD deficiency can lead to the destruction of red blood cells (hemolysis), and the presence of these morphological changes in the blood film is indicative of various abnormalities in the red blood cell structure and shape. These changes are consistent with the consequences of oxidative stress on erythrocytes in individuals with G6PD deficiency, leading to haemolysis and the observed morphological alterations (14). G6PD deficiency is an X-linked condition associated with damage to red blood cells, Individuals with this disorder can generally enjoy normal health (asymptomatic) except if they are exposed to oxidative stress, such as from certain drugs, chemicals, or infections like Hepatitis Virus A and B (15). Pneumonia and typhoid fever are all notable causes of infection-induced haemolysis. Ingestion of certain llegumes(fava beans), drug-induced acute hemolytic antimalarial drugs (primaquine, chloroquine, and primaquine), sulfonamides (sulfanilamide, sulfamethoxazole, and mafenide), analgesics (aspirin, phenazopyridine, and aacetanilide), and nonsulfa antibiotics (nalidixic acid,

nitrofurantoin, isoniazid, and furazolidone) that are commonly used in the management of illnesses in our environment is potentially harmful to people with G6PD deficiency(16). Acute haemolysis results in jaundice, and if the paediatric does not receive timely treatment, excessive bilirubin may accumulate in the brain, causing irreversible damage to the brain(3). Symptoms of G6PD deficiency can include: rapid heart rate, shortness of breath, dark or orange-yellow urine, fever, dizziness, paleness, and jaundice(17).G6PD deficiency can be managed by avoiding agents that trigger anaemia, such as drugs or infections, and fava beans should be avoided. However, in patients who have class 3 variants such as G6PD A-, it may be possible to continue essential drug therapy with careful monitoring of the blood count; blood transfusion is only occasionally required when severe anaemia results(7).

Conclusion

This study showed that the prevalence of G6PD deficiency among children attending pediatric clinic among hospitals in Abakaliki is 19%. Male had the highest prevalence of G6PD deficiency among the children studied, this is due to X-linked disorder in which more males are affected than females. G6PD deficiency is highest between 2-4 years, and haemolysis is due to exposure to oxidants, infection, and ingestion of fava bean. There is a need for the routine screening of children for G6PD deficiency in our environment to allow for evidence-based management. Family health education regarding drugs, chemicals, food items that could adversely affect the patients leading to red blood cell hemolysis, jaundice, and anemia due to G6PD deficiency should be implemented. Quantitative measurement of the enzyme is also important to determine the World Health Organization (19) class of the disease to predict the natural course of G6PD deficiency. Furthermore, there is also a need to offer protective vaccination against hepatitis A and B to affected children against infection-induced attacks. There is also a need to build capacity among pediatricians in our setting to ensure the effective management of children with G6PD deficiency.

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