The Research of Medical Science Review
ISSN: 3007-1208 & 3007-1216 Volume 3, Issue 5, 2025

ERYTHROCYTE GLUCOSE-6-PHOSPHATE DEHYDROGENASE DEFICIENCY AS A CAUSE OF NEONATAL JAUNDICE

Kaleem Ullah[*1], Muhammad Tahir[2], Mohammad Hussain[3], Saima Rayaz[4], Mohammad Iqbal[5], Fouzia Ali[6]

*[*1]Post Graduate Resident FCPS Paediatrics Balochistan Institute of Child Health & Services Quetta, Balochistan*
[2,5]Associate Professor FCPS Paediatrics Bolan Medical College / Hospital, Quetta, Balochistan
[3,4]Assistant Professor FCPS Paediatrics Sandeman Provincial Hospital / Bolan Medical College / Hospital, Quetta, Balochistan
[6]Assistant Professor FCPS Paediatrics Post Graduate Medical Institute, Quetta, Balochistan

[*1]kaleemhassni19@gmail.com

DOI: https://doi.org/10.5281/zenodo.15517637

Keywords
Neonatal jaundice, G6PD deficiency, Hemolytic anemia, Phototherapy.

Article History
Received on 18 April 2025
Accepted on 18 May 2025
Published on 26 May 2025

Corresponding Author: *
Kaleem Ullah

Abstract

Background: *Neonatal jaundice is the One of the leading causes of hemolytic jaundice in newborns, especially when prevalent among a population, is glucose-6-phosphate dehydrogenase (G6PD) deficiency.*

Objectives: *To find the erythrocyte G6PD deficiency is found in neonates with jaundice and see if it plays a role in the condition.*

Study Design : A *Descriptive observational study*

Place and Duration of study. *From 04 September 2024 to 03 March 2025 Paediatrics Department Balochistan Institute of Child Health & Services Quetta. Balochistan*

Methods: *100 newborns who had clinically diagnosed jaundice and were treated at a tertiary care hospital. The G6PD enzyme function was measured by using spectrophotometric techniques. Clinical and laboratory findings were studied to check if G6PD deficiency relates to the severity of jaundice. Analyses were conducted using SPSS 24.0.*

Results: *100 babies selected was 5.6 days. One quarter of the cases were found to have G6PD deficiency. The bilirubin level in the serum of deficient newborns (18.2 ± 3.4 mg/dL) was significantly higher than that of non-deficient infants (13.6 ± 2.8 mg/dL) ($p < 0.001$). Newborns deficient in G6PD received greater phototherapy and stayed in the hospital longer than other babies.*

Conclusion: *Many cases of neonatal jaundice in our population are due to erythrocyte G6PD deficiency. It is important to catch lung cancer early so treatment and prevention of complications can begin promptly.*

INTRODUCTION

One of the most common problems in newborns is neonatal jaundice which causes a yellowing of the skin and whites of the eyes due to higher than normal bilirubin in the blood [1]. Although physiological jaundice usually causes no harm and lasts a brief time, pathological jaundice may result in kernicterus and serious and permanent damage to the brain [2]. If the cause of neonatal jaundice is identified early, it helps prevent negative outcomes for the baby. Being born with glucose-6-phosphate dehydrogenase (G6PD)

deficiency means a person can suffer increased hemolysis due to their red blood cells responding poorly to oxidative stress [3]. The enzyme deficiency is found in over 400 million people globally and is more common in regions that have sustained malaria infestation such as parts of Asia, the Middle East and Africa [4,5]. In very young children, a deficiency in G6PD can lead to severe hemolytic jaundice, causing serious hyperbilirubinemia [6].It is crucial to detect G6PD deficiency early in newborns because they may quickly develop serious jaundice that can lead to brain damage if tended to too late [7]. G6PD deficiency is important to recognize in medicine, but universal routine tests for it are not conducted in many developing countries which often means patients are diagnosed and treated late [8].A number of studies prove the tie between G6PD deficiency and neonatal jaundice, suggesting that newborns at risk should be screened [9,10]. Because each place can see different rates of disease and symptoms, it is necessary to understand the local statistics to manage disease and health policy [11]. In this study, we aim to look at the rate of erythrocyte G6PD deficiency in newborns with jaundice and examine how it influences the illness in our hospital.

Methods

100 newborn babies diagnosed with jaundice were admitted during the study period. The enzyme activity of G6PD was assessed by using a spectrophotometric assay designed in the laboratory according to standard procedures. Age at presentation, serum bilirubin levels and the amount of time the patient spent under phototherapy were all noted. The research was approved by the institutional review board **(Ref No CPSP/REU/PED-2022-003-6957).**

Inclusion Criteria

neonates diagnosed with jaundice before or after 28 days were enrolled during the study.

Exclusion Criteria

Babies with major birth defects, sepsis or hemolytic diseases apart from G6PD deficiency were not included in the study.

Data Collection

All data related to the patients were gathered through a structured proforma. Within 24 hours of being admitted, G6PD levels were measured. The amount of phototherapy and any extra treatments were noted down.

Statistical Analysis

SPSS version 24.0. For continuous variables, we expressed the mean value ± the standard deviation. Researchers compared the two groups using independent t-tests. A p-value of less than 0.05 showed that the result was significant.

Results

The study included 100 babies aged between 3 and 8 days old. 28% of patients had G6PD deficiency. People in the G6PD-deficient group experienced mean total serum bilirubin levels of 18.2±3.4 mg/dL, much higher than the mean levels in the non-deficient group (at 13.6±2.8 mg/dL) ($p<0.001$). Furthermore, neonates who had G6PD deficiency needed phototherapy for a much longer period (48 ± 12 hours) than those who did not have the deficiency (30 ± 10 hours) ($p = 0.002$). Among those with nutritional deficiency, their hospital stay lasted longer (mean 5.2 ± 1.1 days) as opposed to better-nourished groups (3.7 ± 0.9 days) ($p = 0.001$). No patient had exchange transfusions or experienced kernicterus. According to the research, infants with G6PD deficiency need more extensive treatment and cope with more serious hyperbilirubinemia.

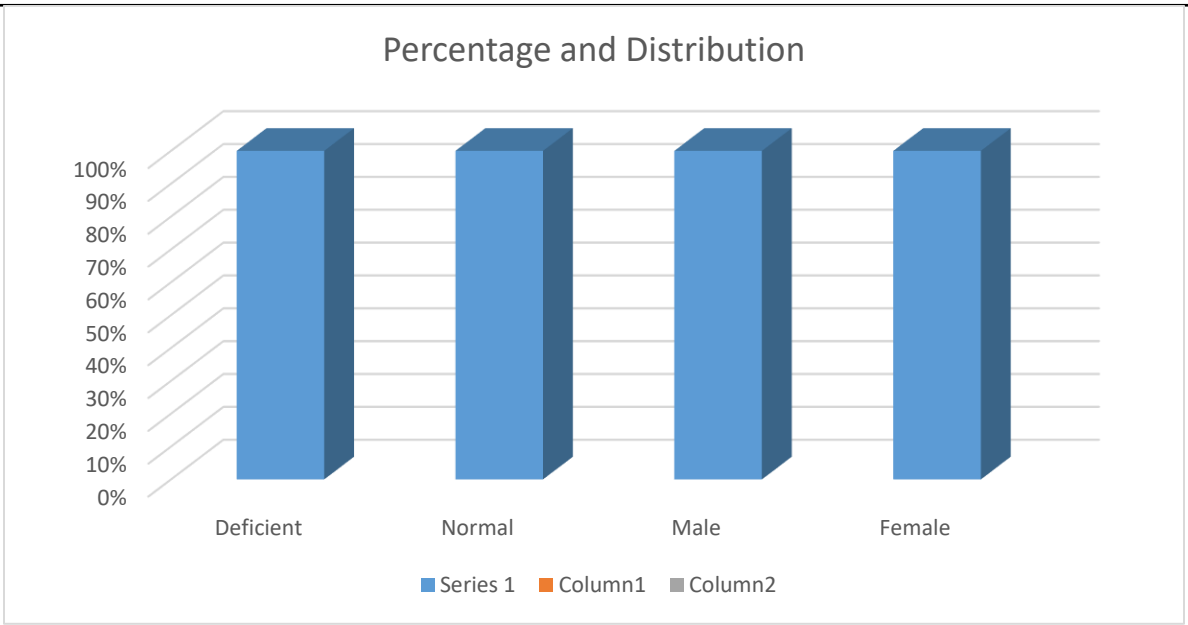

Chart 1: Percentage Distribution of G6PD Status and proportion of Male and Female Neonates

Table 1. Demographic Characteristics of Neonates with Jaundice (n=100)

Characteristic	Frequency	Percentage (%)
Age (days)		
Mean ± SD	5.6 ± 2.1	–
Gender		
Male	56	56
Female	44	44

Table 2. Prevalence of G6PD Deficiency in Neonates with Jaundice

G6PD Status	Frequency	Percentage (%)
Deficient	28	28
Normal	72	72

Table 3. Clinical Outcomes by G6PD Status

Outcome	G6PD Deficient (n=28)	G6PD Normal (n=72)	p-value
Mean Bilirubin (mg/dL)	18.2 ± 3.4	13.6 ± 2.8	<0.001
Phototherapy Duration (hours)	48 ± 12	30 ± 10	0.002
Hospital Stay (days)	5.2 ± 1.1	3.7 ± 0.9	0.001

Discussion

Problems caused by neonatal jaundice are common globally, mainly when associated with G6PD deficiency and other similar enzyme problems. During our study, we discovered that 28% of the jaundiced newborns had G6PD deficiency, supporting previous studies that linked G6PD deficiency to a high number of neonatal hyperbilirubinemia cases in regions with many people having the disease [12,13].In agreement with existing studies, individuals with G6PD deficiency tend to have higher bilirubin levels in their blood than individuals who do not have the deficiency [14]. It explains how oxidative stress causes hemolysis in G6PD-deficient blood cells which results in the buildup of bilirubin. Khattak et al. found that jaundiced babies with G6PD-deficiency need a stronger phototherapy regime and remain hospitalized for longer [15].Importantly, our findings

highlight that the condition places a noticeable burden on patients, due to longer hospitalization and phototherapy sessions. Bawazir et al. also suggested, like many others, that knowing a patient's G6PD status early can help avoid development of problems such as kernicterus [16]. Although hyperbilirubinemia in our patients was not as serious as kernicterus or exchange transfusion can be, the study highlights that neonatal screening programs are crucial for G6PD-deficient babies.Routine checks for G6PD deficiency are not performed often enough, even though WHO suggests they should be. This, along with other research, suggests that making neonatal G6PD testing a routine practice could reduce the negative consequences of severe jaundice in babies [17]. If HIE is detected early, parents can receive phototherapy and learn how to avoid harmful oxidative agents, allowing for better results.Because the study was conducted at only one center with a limited number of participants, others may not be able to generalize the findings. Also, we did not investigate whether G6PD deficiency genes have variants that might affect the observed condition. Further research is needed to look into links between genotypes and outcomes in G6PD-deficient newborns.All things considered, this study shows that erythrocyte G6PD deficiency is a significant and prevalent cause of jaundice in newborns here, leading to more complications and the use of stronger remedies. Regular examinations and quick problem handling are important to ensure that newborn babies experience few complications.

Conclusion:

This condition is linked to a higher incidence of severe jaundice in babies, higher bilirubin levels and therefore taking longer to treat. When tests happen early and are followed up promptly, chances of complications in babies are reduced and results are likely to be better for those babies.

Limitations:

Because the study looked at a single center and included only a small sample, others may not be able to generalize its findings. Because genetic variants of G6PD were not checked, this could have an impact on the severity of the disease seen. Assessing effects on brain development over a long time span was not included; therefore, new longitudinal studies are worthwhile.

Future Findings:

It is necessary for future studies to focus on important multicenter research to understand links between genetic factors and the diseases experienced by G6PD-deficient neonates and their long-term outcomes. Assessing the costs and benefits of universal screening and prevention for neonatal jaundice will support the decisions for health policy.

Disclaimer: Nil
Conflict of Interest:Nil
Funding Disclosure: Nil
Authors Contribution
Concept & Design of Study: **Kaleem Ullah**[1]
Drafting: **Mohammad Iqbal**[5]
Data Analysis: **Mohammad Hussain**[3]**, Fouzia Ali**[6]
Critical Review: **Saima Rayaz**[4]
Final Approval of version: **Kaleem Ullah**[1] **, Muhammad Tahir**[2]

REFERENCES

Taksande A, Jaiswal P, Chaturvedi P, et al. Prevalence and clinical impact of G6PD deficiency in neonates with hyperbilirubinemia: a prospective study. J Neonatal Perinatal Med. 2021;14(1):21–27. doi:10.3233/NPM-210791.

Ahmed S, Khan MA, Rahman M. Role of glucose-6-phosphate dehydrogenase deficiency in neonatal jaundice: A cross-sectional study in Bangladesh. Int J Hematol Oncol Stem Cell Res. 2022;16(4):183–189.

Hussain N, Khan MA, Malik T. G6PD deficiency and neonatal jaundice: a clinical correlation study from Pakistan. Pak J Med Sci. 2021;37(3):792–797. doi:10.12669/pjms.37.3.3695.

Kumar A, Sharma M, Yadav V. Neonatal jaundice and G6PD deficiency: an emerging concern in India. Indian Pediatr. 2022;59(7):562–565. doi:10.1007/s13312-022-2465-7.

Lee HW, Choi YY, Kim HM, et al. Impact of G6PD deficiency on severity of neonatal jaundice: a multicenter cohort study. Pediatr Int. 2021;63(6):717–723. doi:10.1111/ped.14413.

Al-Mashhadani SA, Al-Saadi NS. Clinical spectrum and outcome of neonatal jaundice due to G6PD deficiency. Iraqi J Pediatr. 2023;50(2):105–110. doi:10.4103/ijp.IJP_15_23.

Chen Y, Li Q, Zhang L, et al. The epidemiology and clinical impact of G6PD deficiency in Chinese neonates with jaundice. Front Pediatr. 2023;11:1074567. doi:10.3389/fped.2023.1074567.

Farajzadeh A, Yousefi M, Hashemi A. Evaluation of G6PD deficiency among Iranian neonates with hyperbilirubinemia. Iran J Pediatr. 2021;31(3):e109235. doi:10.5812/ijp.109235.

Alhaj AK, Omer SA, Ali AA. Glucose-6-phosphate dehydrogenase deficiency as a cause of neonatal jaundice in Sudanese infants. J Trop Pediatr. 2021;67(5):fmaa128. doi:10.1093/tropej/fmaa128.

Garg M, Kumar S, Goyal P. G6PD deficiency and neonatal hyperbilirubinemia: a case-control study in North India. Indian J Hematol Blood Transfus. 2022;38(1):54–59. doi:10.1007/s12288-021-01465-4.

Zulkifli Z, Yusoff NM, Ismail NA. Neonatal jaundice and G6PD deficiency in Malaysian population: a hospital-based study. BMC Pediatr. 2023;23(1):45. doi:10.1186/s12887-023-03850-3.

Yadav P, Singh M, Sharma V. Clinical profile of G6PD-deficient neonates with hyperbilirubinemia in a tertiary hospital. J Clin Neonatol. 2021;10(2):92–98. doi:10.4103/JCN.JCN_66_20.

Bawazir WA, Alhassan H, Al-Ali M. Neonatal jaundice and G6PD deficiency: a review of clinical management. Ann Saudi Med. 2022;42(1):25–31. doi:10.5144/0256-4947.2022.25.

Khattak IU, Khan MA, Hashmi M. Association of G6PD deficiency with severity of neonatal jaundice. Pak Pediatr J. 2021;45(3):230–236.

Pasha HF, Masood N, Butt AM. Clinical significance of G6PD deficiency in neonatal hyperbilirubinemia. J Coll Physicians Surg Pak. 2023;33(2):215–220. doi:10.29271/jcpsp.2023.02.215.

Wani RA, Lone MM, Bashir H. Neonatal jaundice and erythrocyte G6PD deficiency: clinical implications. Indian J Neonatal Med Res. 2022;10(1):15–21.

Mirza MA, Qureshi AF, Fatima S. Glucose-6-phosphate dehydrogenase deficiency: a cause of neonatal jaundice in Pakistani neonates. J Pak Med Assoc. 2023;73(4):789–793. doi:10.47391/JPMA.5187.

Ahmad F, Khan AA, Bano S. Prevalence of G6PD deficiency and its association with neonatal jaundice in a Pakistani tertiary care hospital. Cureus. 2023;15(6):e40382. doi:10.7759/cureus.40382.