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ASSESSMENT OF LIVER FUNCTION TESTS IN CHILDREN WITH G6PD DEFICIENCY

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Abstract
Glucose-6-phosphate dehydrogenase (G6PD) deficiency is one of the most common enzymopathies worldwide, with a significant prevalence in India. This study assesses liver function tests (LFTs) in Indian children diagnosed with G6PD deficiency to determine potential hepatic involvement. The study aims to analyze liver enzyme alterations, bilirubin levels, and other biochemical markers to understand their implications in managing affected children. A total of 200 children aged 1–12 years diagnosed with G6PD deficiency were included in this observational study, along with 200 age- and gender-matched healthy controls. Biochemical markers such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), bilirubin, alkaline phosphatase (ALP), and total protein levels were assessed. Results showed that G6PD-deficient children had significantly higher levels of ALT, AST, and total bilirubin compared to controls ($p < 0.05$). ALP and albumin levels remained comparable. The findings suggest mild hepatic involvement, possibly due to oxidative stress and hemolysis. Regular monitoring of LFTs in G6PD-deficient children is recommended, especially during oxidative stress episodes. Further studies with larger cohorts are needed to establish long-term hepatic outcomes in this population.

Keywords: G6PD deficiency, liver function tests, pediatric hepatology, enzyme markers, Indian children

INTRODUCTION

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is an X-linked enzymatic disorder affecting red blood cell metabolism. It is one of the most common genetic disorders worldwide, particularly in populations with high incidences of malaria, such as India [1]. The primary consequence of G6PD deficiency is hemolysis triggered by oxidative stressors, leading to hemolytic anemia. While its hematological implications are well-established, its impact on liver function remains unclear [2].

The liver plays a crucial role in bilirubin metabolism and detoxification. Since G6PD deficiency predisposes individuals to increased hemolysis, hepatic involvement may arise due to an increased bilirubin load [3]. However, whether G6PD-deficient children experience hepatic stress beyond hemolysis remains to be explored.

Previous studies have indicated mild liver enzyme alterations in individuals with G6PD deficiency, suggesting possible transient hepatocellular stress [4]. Elevated liver enzymes, particularly alanine

aminotransferase (ALT) and aspartate aminotransferase (AST), could indicate hepatic involvement secondary to hemolysis-induced oxidative stress [5].

This study aims to assess liver function in G6PD-deficient children in India by evaluating key biochemical markers, including ALT, AST, bilirubin, alkaline phosphatase (ALP), and total protein. By comparing these markers between G6PD-deficient children and healthy controls, we seek to determine whether these children exhibit signs of hepatic dysfunction. The findings will provide insight into the need for routine liver function monitoring in G6PD-deficient pediatric populations.

MATERIALS AND METHODS

Study Design and Population This observational cross-sectional study was conducted in our tertiary care hospital. A total of 200 children aged 1–12 years, diagnosed with G6PD deficiency, were enrolled. The control group included 200 age- and gender-matched healthy children without G6PD deficiency.

Inclusion Criteria:

- Children aged 1–12 years
- Diagnosed with G6PD deficiency via quantitative enzyme assay
- No history of chronic liver disease or congenital liver disorders

Exclusion Criteria:

- Children with concurrent infections affecting liver function
- Those receiving hepatotoxic medications
- History of neonatal cholestasis or metabolic liver diseases

Biochemical Analysis Venous blood samples were collected after overnight fasting. The following LFT parameters were measured:

- Serum bilirubin (total and direct)
- ALT and AST
- ALP
- Serum albumin and total protein

Liver function parameters were compared between G6PD-deficient and control groups. Data were analyzed using SPSS, with significance set at $p < 0.05$.

RESULTS

Of the 200 children with G6PD deficiency, mild elevations in ALT and AST were observed in 32% and 28% of cases, respectively. Total bilirubin levels were significantly higher in G6PD-deficient children ($p < 0.01$). ALP and albumin levels did not show significant variation between the two groups.

Table 1: Liver Enzyme Levels

Parameter	G6PD Deficient (Mean ± SD)	Control (Mean ± SD)	p-value
ALT (U/L)	45.2 ± 10.3	32.8 ± 8.6	<0.05
AST (U/L)	42.7 ± 9.1	30.2 ± 7.8	<0.05
ALP (U/L)	210.5 ± 32.2	205.7 ± 29.5	0.08

Table 2: Bilirubin and Protein Levels

Parameter	G6PD Deficient (Mean ± SD)	Control (Mean ± SD)	p-value
Total Bilirubin (mg/dL)	1.5 ± 0.4	0.8 ± 0.3	<0.01
Direct Bilirubin (mg/dL)	0.6 ± 0.2	0.3 ± 0.1	<0.05
Albumin (g/dL)	4.2 ± 0.5	4.3 ± 0.4	0.12
Total Protein (g/dL)	6.8 ± 0.6	7.0 ± 0.5	0.07

Table 3: Haematological Parameters

Parameter	G6PD Deficient (Mean ± SD)	Control (Mean ± SD)	p-value
Hemoglobin (g/dL)	10.8 ± 1.2	12.5 ± 1.1	<0.01
Reticulocyte Count (%)	2.8 ± 0.9	1.2 ± 0.5	<0.01

Table 4: Liver Function Comparison

Parameter	G6PD Deficient (%)	Control (%)	p-value
Elevated ALT	32%	12%	<0.05
Elevated AST	28%	10%	<0.05
Elevated Bilirubin	40%	15%	<0.01

DISCUSSION

The study highlights mild hepatic involvement in G6PD-deficient children, with elevated transaminases and bilirubin levels. The increased bilirubin is likely linked to hemolysis rather than intrinsic liver dysfunction. Elevated ALT and AST suggest transient hepatocellular stress, possibly due to oxidative damage [6-10].

Previous studies have documented sporadic cases of hepatocellular injury in G6PD-deficient patients, especially during hemolytic crises [11]. The observed increase in bilirubin, particularly the direct fraction, suggests possible hepatic processing challenges rather than outright dysfunction. Although ALP and albumin levels were not significantly different, the mild increase in transaminases necessitates careful monitoring [12-14].

G6PD deficiency predisposes individuals to oxidative stress-induced hemolysis, which may contribute to secondary hepatic stress [15]. The hemolytic episodes increase heme breakdown, leading to higher bilirubin levels, as seen in our study. While liver enzyme elevations were mild, they may indicate subclinical hepatic stress requiring further evaluation.

The comparison of total protein and globulin levels between the two groups suggests that synthetic liver function remains intact, further supporting the theory that hepatic impairment is mild or transient. Additionally, the increased reticulocyte count in G6PD-deficient children aligns with expected compensatory hematopoiesis due to chronic hemolysis [16].

These findings emphasize the need for clinicians to monitor LFTs in G6PD-deficient children, particularly during oxidative stress episodes. More extensive longitudinal studies could help determine whether repeated hemolytic crises contribute to progressive liver dysfunction in adulthood.

CONCLUSION

G6PD-deficient children in India exhibit mild alterations in liver function tests, particularly in bilirubin and transaminase levels. While these changes are not indicative of severe liver disease, periodic monitoring is recommended, particularly during oxidative stress episodes. Clinicians should consider routine LFT evaluations in G6PD-deficient children to prevent complications associated with recurrent hemolysis.

REFERENCES

1. Frank JE. Diagnosis and management of G6PD deficiency. Am Fam Phys. 2005;72(7):1277–82. [PubMed] [Google Scholar]
2. Washington EC, Ector W, Abboud M, Ohning B, Holden K. Hemolytic jaundice due to G6PD deficiency causing kernicterus in a female newborn. South Med J. 1995;88(7):776–9. [DOI] [PubMed] [Google Scholar]
3. Iolascon A, Faienza MF, Perrotta S, Meloni GF, Ruggiu G, del Giudice EM. Gilbert's syndrome and jaundice in glucose-6-phosphate dehydrogenase deficient neonates. Haematologica. 1999;84(2):99–102. [PubMed] [Google Scholar]
4. Salomon F, Casimir A, Julien S, et al. Liver function assessment in glucose-6-phosphate dehydrogenase deficient neonates in Benin. Int J Biomed Res. 2018;09(06):221–5. [Google Scholar]

5. Bubp J, Jen M, Matuszewski K. Caring for glucose-6-phosphate dehydrogenase (G6PD)-Deficient patients: Implications for pharmacy. P T. 2015;40(9):572–4. [PMC free article] [PubMed] [Google Scholar]
6. Abdel Hakeem GL, Abdel Naeem EA, Swelam SH, et al. Detection of occult acute kidney injury in glucose-6-phosphate dehydrogenase deficiency anemia. Mediterr J Hematol Infect Dis. 2016;8(1):e2016038. [DOI] [PMC free article] [PubMed] [Google Scholar]
7. Nkhoma ET, Poole C, Vannappagari V, Hall SA, Beutler E. The global prevalence of glucose-6-phosphate dehydrogenase deficiency: a systematic review and meta-analysis. Blood Cells Mol Dis. 2009;42:267–78.
8. Harcke SJ, Rizzolo D, Harcke HT. G6PD deficiency: An update. J Am Acad Physician Assist. 2019;32:21–26.
9. Beutler E, Gaetani G, Der Kaloustian V, Luzzatto L, Sodeinde O. Glucose-6-phosphate dehydrogenase deficiency. Bull World Health Organ. 1989;67:601–11.
10. Olusanya BO, Emokpae AA, Zamora TG, Slusher TM. Addressing the burden of neonatal hyperbilirubinaemia in countries with significant glucose-6-phosphate dehydrogenase deficiency. Acta Paediatr. 2014;103:1102–9.
11. Isa HM, Mohamed MS, Mohamed AM, Abdulla A, Abdulla F. Neonatal indirect hyperbilirubinemia and glucose-6-phosphate dehydrogenase deficiency. Korean J Pediatr. 2017;60:106–11.
12. Nkhoma ET, Poole C, Vannappagari V, Hall SA, Beutler E. The global prevalence of glucose-6-phosphate dehydrogenase deficiency: a systematic review and meta-analysis. Blood Cells Mol Dis. 2009;42:267–78.
13. Hsia YE, Miyakawa F, Baltazar J, Ching NS, Yuen J, Westwood B, et al. Frequency of glucose-6-phosphate dehydrogenase (G6PD) mutations in Chinese, Filipinos, and Laotians from Hawaii. Hum Genet. 1993;92:470–6.
14. Nair PAK, Al Khusaiby SM. Kernicterus and G6PD deficiency-a case series from Oman. J Trop Pediatr. 2003;49:74–77.
15. Kaplan M, Hammerman C. Severe neonatal hyperbilirubinemia. A potential complication of glucose-6-phosphate dehydrogenase deficiency. Clin Perinatol. 1998;25:575–90, viii.
16. Kaplan M, Herschel M, Hammerman C, Hoyer JD, Stevenson DK. Hyperbilirubinemia among African American, glucose-6-phosphate dehydrogenase-deficient neonates. Pediatrics. 2004;114:e213–9.