

Acute Hemolytic Crisis Induced by Naphthalene Inhalation from Domestic Henna Application in a 3-Year-Old Male with a Family History of G6PD Deficiency: A Case Report

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Abstract

Case Report

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is a common X-linked enzymatic disorder that predisposes erythrocytes to oxidative hemolysis. We report the case of a 3-year-old male with a maternal family history of G6PD deficiency who presented with a 1-day history of reduced appetite, pallor, and macroscopic hemoglobinuria (tea-colored urine). Initial laboratory evaluation revealed an acute normocytic anemia (hemoglobin 7.9 g/dL) with mild reticulocytosis. Conventional biochemical markers of active hemolysis including serum lactate dehydrogenase (LDH) and total bilirubin had already returned to normal reference limits at presentation, and no active bleeding was noted. A peripheral blood smear demonstrated rare bite cells (degmacocytes). Detailed history-taking revealed recent inhalation exposure to naphthalene-enriched domestic henna during Eid celebrations. This case highlights a fully resolved hemolytic event where morphological cues and detailed history-taking, proved pivotal in confirming a self-limiting hemolytic crisis induced by an unusual domestic oxidant.

Keywords: G6PD deficiency, Hemolytic crisis, Naphthalene exposure, Bite cells, Hemoglobinuria, Normocytic anemia.

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INTRODUCTION

Glucose-6-phosphate dehydrogenase (G6PD) deficiency affects millions worldwide, rendering red blood cells highly vulnerable to oxidative stress. While acute hemolytic episodes are frequently triggered by infections, fava beans, or specific medications, domestic chemical exposures remain underrecognized potent triggers. When patients present after the acute phase of hemolysis has subsided, traditional serum markers such as lactate dehydrogenase (LDH) and bilirubin can normalize rapidly, masking the underlying process. This case report illustrates the importance of detailed history taking, including social and family history in the diagnosis after biochemical markers may have normalized.

CASE PRESENTATION

A 3-year-old male presented to a primary care center with a 1-day history of reduced appetite, noticeable pallor, and the passage of dark, tea-colored urine (Figure 1). He was otherwise hemodynamically stable. Given a known maternal family history of G6PD deficiency, an immediate complete blood count (CBC)

was performed. The CBC revealed an acute anemia with a hemoglobin level of 7.9 g/dL, prompting an emergent referral to secondary care for further evaluation and monitoring.

Upon admission to the secondary care facility, clinical examination confirmed marked cutaneous and mucosal pallor. There was no evidence of active internal or external hemorrhage, organomegaly, or lymphadenopathy.



Figure 1: Tea-coloured urine. Copyright to Lecturio

INVESTIGATIONS

Initial laboratory investigations yielded unexpected results regarding conventional hemolytic markers. Both serum lactate dehydrogenase (LDH) and serum total bilirubin levels were within normal reference limits. Urinalysis confirmed macroscopic hemoglobinuria without intact red blood cells.

A peripheral blood smear was requested to evaluate the etiology of the normocytic anemia. The smear demonstrated a normocytic, normochromic erythrocyte population alongside mild reticulocytosis and bite cells (degmacytes) (Figure 12).

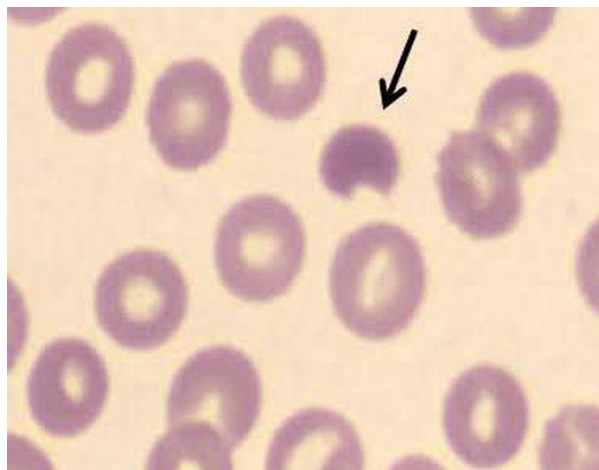


Figure 2: Bite cells or degmacytes. Copyright to MEDizzy

Laboratory Parameter	Patient Value	Reference Range
Hemoglobin	7.9 g/dL	11.5–13.5 g/dL
Mean Corpuscular Volume (MCV)	82 fL	75–87 fL
Reticulocyte Count	3.2%	0.5%–2.0%
Serum Lactate Dehydrogenase (LDH)	210 U/L	150–300 U/L
Total Bilirubin	0.7 mg/dL	0.2–1.2 mg/dL
Peripheral Blood Smear	Rare bite cells present	Normal morphology

Differential Diagnosis & History Refinement

The presence of degmacytes pointed toward oxidative hemolysis, characteristic of a G6PD-deficient crisis. However, because the biochemical markers (LDH, bilirubin) had already normalized, the hemolytic event was deduced to be fully resolved and self-limiting.

To identify the precise oxidant trigger, a targeted environmental & social history was obtained

from the parents. The review ruled out recent illness or exposure to fava beans or oxidative medications. However, the parents disclosed that during recent domestic Eid celebrations, traditional henna had been applied to family members in the household. Crucially, the henna mixture had been enriched with naphthalene (Figure 3) a traditional practice used locally to enhance the depth and longevity of the henna stain. The patient had inhaled these localized domestic fumes.



Figure 3: Naphthalene powder. Copyright to Camachem.com

Treatment and Outcome

Because the hemolytic process had already ceased organically and the patient remained hemodynamically stable without respiratory distress or further hemoglobin drop, conservative management was adopted. The child was closely monitored with serial CBCs, maintained on adequate oral hydration to protect renal function from hemoglobinuria, and the family was strictly counseled on avoiding naphthalene and other oxidative agents. The hemoglobin stabilized, the tea-colored urine cleared within 48 hours, and the patient was discharged in excellent clinical condition with a plan for outpatient G6PD enzyme activity testing once the reticulocytosis fully resolved as well as Iron and Folic Acid supplementation.

DISCUSSION

This case underlines two vital clinical insights for the acute care physician: the atypical presentations of domestic toxicities, the importance of a detailed history and the temporal limitations of biochemical hemolytic markers.

Naphthalene inhalation is a well-documented but frequently overlooked cause of severe oxidative stress in G6PD-deficient individuals. In certain cultural contexts, naphthalene is added to henna applications to darken the dye color. Because the chemical volatilizes easily, inhalation alone can rapidly precipitate an acute hemolytic crisis in G6PD deficiency.

Furthermore, this case serves as a reminder of the transient kinetics of serum markers like LDH and bilirubin. In a self-limiting or acute-onset hemolytic event, these markers peak rapidly and can return to the normal reference range within days as the liver clears bilirubin and tissues metabolize excess LDH. Conversely, structural damage to the erythrocyte membrane remains visible. Splenic macrophages pit out precipitated denatured hemoglobin (Heinz bodies), leaving behind classic "bite cells" or degmacytes that circulate for days to weeks.

When conventional biochemistry fails to align with an obvious historical drop in hemoglobin, a meticulous review of the peripheral blood smear remains the gold standard tool to identify a resolved or resolving hemolytic crisis.

Learning Points

- Morphological markers over biochemistry: In resolved or self-limiting hemolytic crises, peripheral blood smear cues like bite cells (degmacytes) can confirm a diagnosis even after serum LDH and bilirubin have completely normalized.
- Unusual domestic triggers: Naphthalene inhalation from culturally specific domestic practices, such as enriching henna dyes, is a potent trigger for acute oxidative hemolysis in G6PD-deficient children.
- Comprehensive history taking: When evaluating unexplained pediatric anemia, exploring recent festival celebrations, domestic cosmetics, and environmental chemical exposure is vital.

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