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Two Successive Episodes of Acute Renal Failure in the Context of Anemia Reveal a Glucose-6-Phosphate Dehydrogenase Deficiency

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Abstract **Case Report**

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is a genetic disorder that mainly affects males and manifests itself in episodes of hemolysis, often triggered by certain medications, foods, or infections. It is one of the most common genetic disorders in the world. It affects more than 400 million people, mainly in Africa, Asia, the Middle East, and the Mediterranean Basin. The frequency of G6PD deficiency is relatively high in some sub-Saharan African countries. Among the 200 variants of G6PD described worldwide, the G6PD A- variant (G202A/A376G) predominates in sub-Saharan Africa, where it affects 15 to 20% of the African population. G6PD deficiency can cause massive intravascular hemolysis triggered by oxidizing agents, and this hemolysis can complicate acute renal failure (ARF), which is usually reversible but may even require extrarenal purification. We report the case of a 16-year-old patient who had two successive episodes of ARF in the context of anemia, which investigations revealed to be due to G6PD deficiency. The objective of this study was to observe a rare case of two episodes of acute renal failure in the same patient caused by G6PD deficiency.

Keywords: Acute renal failure, anemia, G6PD deficiency.

INTRODUCTION

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is a genetic disorder that mainly affects males and manifests as episodes of hemolysis, often triggered by certain medications, foods, or infections [1]. G6PD deficiency is one of the most common human enzyme disorders [2]. It is a hereditary genetic disease that can lead to the destruction of red blood cells [3]. The relationship between G6PD deficiency and hemolytic crises was scientifically demonstrated in the 1950, particularly during the Indochina War. During this war, soldiers treated with primaquine experienced hemolytic episodes [1]. G6PD deficiency mainly affects men; hemizygous men and homozygous women have a complete deficiency, but heterozygous women are generally asymptomatic carriers [4]. It is caused by hereditary mutations in the G6PD gene, located on the X chromosome (long arm, position 28) [2]. It affects more than 400 million people, mainly from Africa, Asia, the Middle East, and the Mediterranean Basin [3]. Human migration could explain the presence of G6PD with varying frequencies in populations around the world [5]. Between 250,000 and 450,000 French people have a genetic disorder called G6PD deficiency, which is not well known among practitioners, who do not think to test for it [6]. The frequency of G6PD deficiency is relatively high in some sub-Saharan African countries [4]. Of the 200 G6PD variants described worldwide, the G6PD A- variant (G202A/A376G) predominates in sub-Saharan Africa, where it affects 15 to 20% of the African population [7]. G6PD deficiency can cause massive intravascular hemolysis triggered by oxidizing agents, and this hemolysis can complicate acute renal failure, which is usually reversible but may even require extrarenal purification [3].

The objective of this study was to observe a rare case of two episodes of acute renal failure in the same patient caused by G6PD deficiency.

OBSERVATION

This is a 16-year-old patient, not in school, Bambara, Malian, residing in SIRAKORO. He was hospitalized for the first time for impaired kidney function at 575.99 umol/l.

The patient has no known medical or surgical history other than repeated transfusions (the first transfusion since the age of 6).

This patient was hospitalized for the first time for impaired renal function at 575.99 umol/l in the context of anemia (Hb at 7 g/dl) and repeated transfusions. During this first hospitalization, he underwent several hemodialysis sessions indicated for hyperkalemia at 7.98 mmol/L, confirmed by ECG, and eventually returned to normal renal function (creatinine at 90 umol/L) and was discharged from the hospital.

Three months later, he returned to us with impaired renal function at 1213.3 umol/l, conjunctival pallor, and palmar-plantar pallor. The history of the current episode dated back three weeks and was marked by dizziness, postprandial vomiting, evening and nighttime fever, physical asthenia, and abdominal pain, for which he consulted a healthcare facility that revealed impaired renal function (creatinine level at 1213.3 umol/l) and transferred him to nephrology for better care.

Previous treatment was ignored. Physical examination: altered general condition, conjunctival pallor, subicterus, severe physical asthenia, patient lucid (Glasgow 15/15) Temperature: 37,3° C Respiratory rate 25 cycles/minute Heart rate 110 beats/minute Blood pressure 100/70 mmHg SpO2 96% Weight 66 kg Heart examination: Audible heart sounds, good intensity, regular, no additional sounds, tachycardia at 110 beats/minute. Chest: symmetrical, harmonious, vocal vibrations well transmitted, non-tympanic sound, no rales. Abdomen: soft, depressible, diffuse abdominal pain. Skin: palmar-plantar pallor, capillary refill time greater than 3 seconds.

All of this led to the following assessment (and results):

Abdominal ultrasound: Left kidney: 113X53X25 mm Right kidney: 107X50X26 mm Both kidneys are well differentiated, without lithiasis, with no dilation of the pyelocaliceal cavities. Uremia 43.59 mmol/L Creatinine 1213.3 umol/L Uric acid 637 umol/L Thick smear 25 Tropho/ul Negative dengue serology Negative HIV serology

Calcemia 2.28 mmol/L Phosphatemia 2 mmol/L Natremia 132 mmol/L Serum potassium 3.98 mmol/L Chloremia 96.6 mmol/L Magnesiumemia 1.15 mmol/L

Hemoglobin 6.7 g/dL (Normochromic macrocytic anemia) White blood cells 9970/mm3 Platelets 163000/mm3 ALAT 31.4 IU/L ASAT 28.6 IU/L Sterile urine culture 24-hour proteinuria 0.84 g/24 hours Albuminemia 37.9 g/L Proteinemia 80.9 g/L Prothrombin level 92.6% Haptoglobin 0.03 g/L LDH (lactate dehydrogenase) 2000.9 IU/L RP 4 mg/L TSHus 4.32 Hemoglobin electrophoresis: AA2 (A 96% and A2 4%)

G6PD 1.25 u/g (normal reference values: 8.0-14.9) chest X-ray, front view: Normal Electrocardiogram: Normal

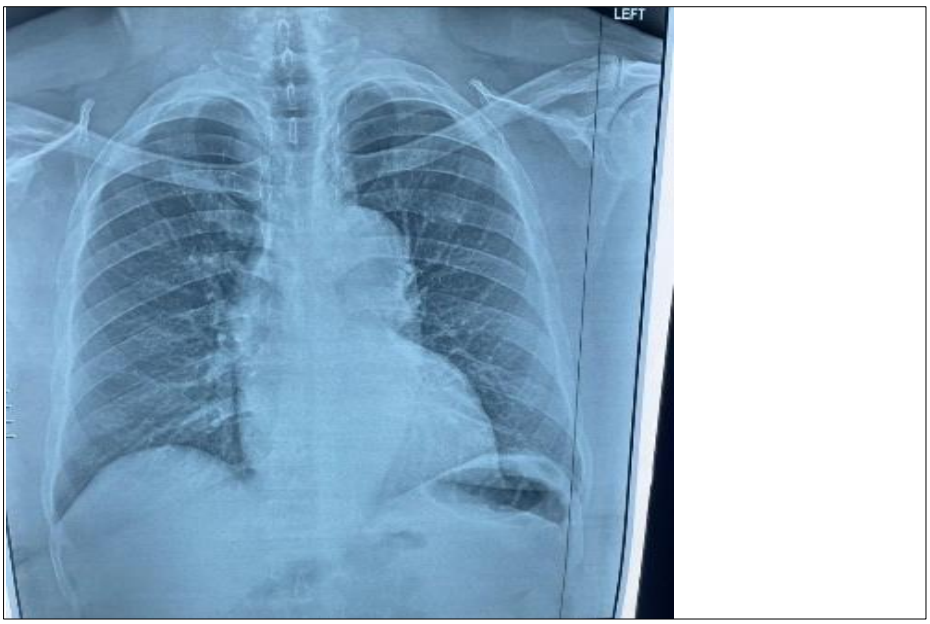

Fig. 1: Chest X-ray, front view

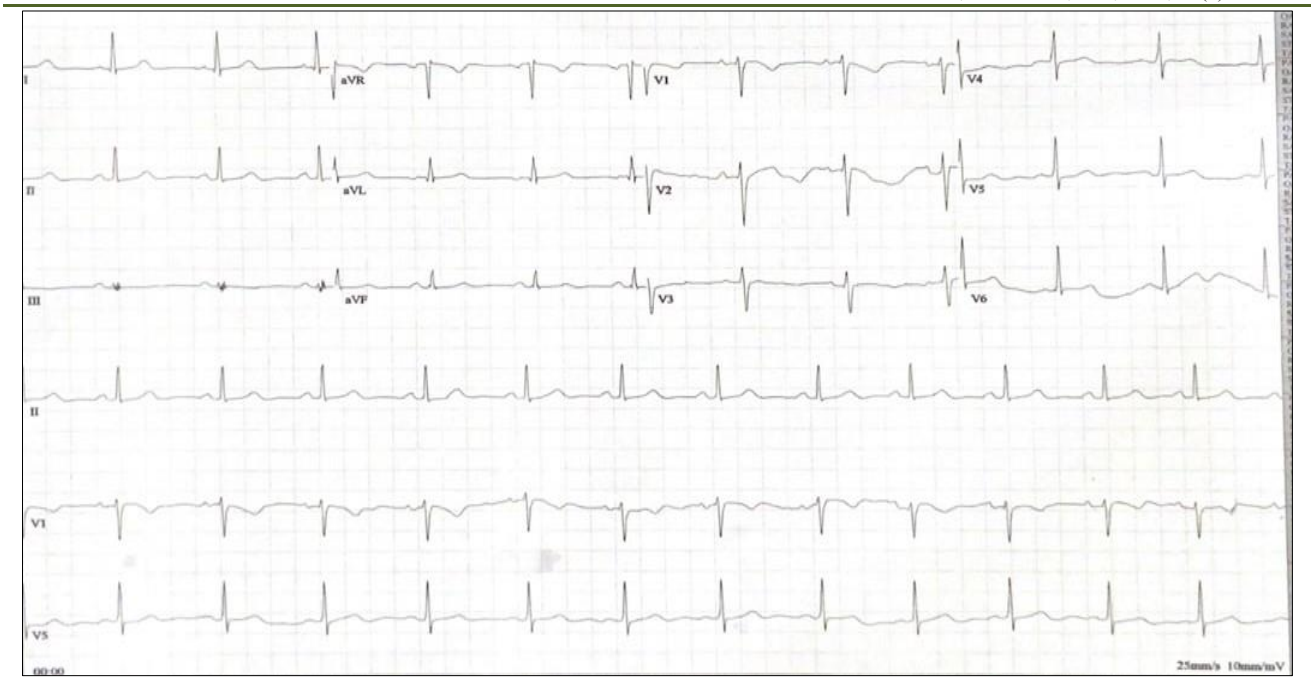

Fig. 2: ECG

Treatment:

- HEMODIALYSIS for uremic syndrome with transfusion during dialysis
- Paracetamol infusion: 1 bottle twice daily
- Lanzoprazole 30 mg capsule: 1 capsule/day
- Levosulpiride 25 mg injection: 1 bulb x 2/day
- 0.9% saline solution: 1 bottle x 2/day
- Artesunate 180mg injection: 180mgTime zero Time 12 Time 24 then Artemether+lumefantrine 80/480 mg tablet: 1 tablet twice daily

The patient's condition improved, with creatinine levels returning to normal (98.2 umol/L on discharge from hospital) after four hemodialysis sessions and an increase in hemoglobin (Hb 11.2g/L).

DISCUSSION

Glucose-6-phosphate dehydrogenase (G6PD) plays a key role in the production of ribose-5-phosphate and the generation of NADPH (reduced nicotinamide adenine dinucleotide phosphate) in the pentose phosphate pathway. This pathway is the only pathway for NADPH production in mature red blood cells, which lack the citric acid cycle [8]. NADPH, an electron donor, is essential for defense against oxidizing agents and for reductive biosynthesis reactions [9]; NADPH therefore maintains reduced glutathione, the main protector against free radicals. Without reduced glutathione, red blood cells cannot neutralize oxidizing species [10]. G6PD is remarkable for its genetic diversity. Numerous variants of G6PD, mainly resulting from missense mutations, have been described, with highly variable levels of enzyme activity and clinical symptoms [10].

G6PD deficiency can cause neonatal jaundice, acute hemolysis, or severe chronic non-spherocytic hemolytic anemia [9]. Red blood cells do not have mitochondria and depend exclusively on this NADPH pathway. In the event of G6PD deficiency, there will be less NADPH, making it impossible to regenerate reduced glutathione, resulting in the accumulation of oxidizing species in red blood cells. Oxidants cause [3,11]:

- Oxidation of hemoglobin with formation of Heinz bodies: denatured hemoglobin precipitates in the cell and these aggregates are called Heinz bodies.
- Damage to the red blood cell membrane: free radicals oxidize membrane lipids and proteins. The cell becomes rigid, deformed, fragile, and easily destroyed.
- G6PD deficiency causes two types of hemolysis: extravascular hemolysis (the main mechanism) and intravascular hemolysis (less common but possible) [12,13]:
- Extravascular hemolysis: the spleen recognizes and eliminates red blood cells containing Heinz bodies, leading to destruction in the reticuloendothelial system.
- Intravascular hemolysis: if oxidants are very high (fava beans, oxidizing drugs), red blood cells will rupture directly in the circulation.

In cases of G6PD deficiency, there are typical triggers of hemolysis [14]:

- Oxidizing drugs: Sulfonamides, dapsone, antimalarials (primaquine, tafenoquine, etc.), rasburicase, silver sulfadiazine (cream), etc.
- Infections: Cause severe oxidative stress
- Ingestion of fava beans (favism).

For our patient, the drug that could trigger hemolysis was not identified (the patient and his family were unaware of the treatment he had previously received), but the patient had malaria, which, along with fever, can also trigger hemolysis.

However, the massive hemolysis observed in G6PD deficiency can cause acute renal failure (ARF) through several mechanisms [15, 13,16]:

- massive release of free hemoglobin into the blood: during intravascular hemolysis, red blood cells burst and release free hemoglobin, heme, and free iron. When the amount of hemoglobin exceeds the capacity of transport proteins (haptoglobin), hemoglobin circulates freely in the plasma.
- Direct toxicity of hemoglobin on renal tubules: hemoglobin is nephrotoxic and causes acute tubular necrosis in the kidneys. Hemoglobin is also likely to form free radicals, increasing oxidative stress in the kidneys.

Our patient presented with hemolytic anemia, as evidenced by decreased hemoglobin, decreased haptoglobin, and elevated lactate dehydrogenase, and this hemolysis could explain the onset of acute renal failure in our patient. It should be noted that both episodes of ARF in our patient occurred in the context of anemia.

Iron and heme promote lipid peroxidation, causing cell damage and tubular cell death (therefore acute tubular necrosis) [17].

- Obstruction of the tubules by pigments: in the renal tubules, hemoglobin precipitates and forms obstructive pigments, particularly if the patient is dehydrated or if the urine is acidic.

This mechanical obstruction also contributes to acute tubular necrosis (similar to rhabdomyolysis).

- Renal vasoconstriction and decreased blood flow: free hemoglobin and heme consume NO (nitric oxide), causing:
- Renal vasoconstriction
- Reduction in renal blood flow
- Worsening of tubular damage

At the same time, hemolysis can lead to: relative hypovolemia (capillary leakage, vomiting), and hemolytic shock in severe cases [18].

The formation of microthrombi in the renal capillaries: hemolysis can activate intravascular coagulation and the formation of microthrombi, which further reduces renal perfusion.

CONCLUSION

When faced with acute renal failure in the context of anemia in a patient with a history of multiple transfusion and normal hemoglobin electrophoresis, always consider testing for glucose-6-phosphate dehydrogenase deficiency, which could explain this acute renal failure, a complication of hemolysis (G6PD deficiency).

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