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Case Report

Henna: A cause of life threatening hemolysis in G6PD-deficient patient

Homa Ilkhanipur[1], Neda Hakimian[2]

ABSTRACT

Glucose-6-phosphate dehydrogenase deficiency is the most important disease of the hexosemonophosphate pathway and is responsible for neonatal jaundice that can be very severe in G6PD deficiency and induces permanent damage to the brain and causes kernicterus and death. Henna is a traditional cosmetic agent to stain the hair, skin and nails. It can cause hemolysis in G6PD deficient patients because of lawsone (2-hydroxy-1, 4naphthoquinone) that has oxidative properties similar to naphtalin. We report on a 35 days boy with jaundice (bil: 50.2), hemogluinuria and kernicterus symptoms after application of henna on his skin. In laboratory test his G6PD enzyme activity was deficient.

KEY WORDS: Acute Hemolysis, Henna, Glucose-6-Phosphate Dehydrogenase (G6PD) deficiency.

doi: http://dx.doi.org/10.12669/pjms.291(Suppl).3549

How to cite this:

Ilkhanipur H, Hakimian N. Henna: A cause of life threatening hemolysis in G6PD-deficient patient. Pak J Med Sci 2013;29(1) Suppl:429-431. doi: http://dx.doi.org/10.12669/pjms.291(Suppl).3549

INTRODUCTION

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most important disease of the hexose monophosphate pathway[1,2] and is responsible for 3 clinical syndromes, episodic hemolytic anemia induced by infections, certain drugs or, rarely, fava beans, spontaneous chronic nonspherocytic hemolytic anemia and neonatal jaundice.[3] This X-linked enzyme deficiency affects more than 200 to 400 million people worldwide.[2,4] Mediterranean mutation is the most common variant of enzyme deficiency and often associated with favism.[4,5] The prevalence of G6PD deficiency in Iranian population is 10-14.9% reported by WHO.[3] G6PD deficiency was discovered for the first time when hemolytic anemia occurred in some persons who consumed antimalarial drug named primaquine.[1,5]

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Henna is a cosmetic dye that is used for dying hair, nails[6,7] and also for treatment dermatitis[8] especially in Middle East,[9-11] like southern of Iran. Some studies identified that henna can induce hemolytic anemia.[12-14] In the literature, hemolysis linked to henna application in G6PD enzyme deficient patients is rare except in infancy.[1,15]

We report a case of a G6PD enzyme deficient patient with acute hemolysis and kernicterus symptoms after exposure to henna is presented.

CASE REPORT

The patient was a 35 days old boy, who presented to us with yellowish skin, poor sucking, poor feeding and opistotonus position, since three days ago. There was a history of total bilirubin 12 in 27th day of birth and history of using henna throughout the whole body in 30th day of birth.

On physical examination he had an axillary temperature of 36.5°C, pulse of 160/min, respiratory rate of 28/min and blood pressure of 80/60 mmHg. Other physical findings were normal. Laboratory investigations revealed hemoglobin of 4.7 gm/dL, hematocrit of 14.3%, white blood cell count of 6600/mm3, red blood cell count of 1 037 000/mm3 and platelet count 469 000/mm3.

Peripheral smear showed 77% neutrophils, 5% lymphocytes, anisocytosis(+), poikilocytosis(+), spherocytosis(-), heinz body (+) and reticulocyte count 5.5%. Biochemical analysis yielded blood urea nitrogen16 mg/dL, aspartate minotransferase 54 iu/L, alanine aminotransferase 44 iu/L, indirect bilirubin 50.2 mg/dL,creatinine 0.5 mg/dL, and the other biochemical parameters within normal limits. Serology for HAV¸ HBV and HCV was negative. Direct Coombs test was negative. Qualitative G6PD enzyme deficiency was established with Brewerin visual test. Urine analysis: yellow colored, specific gravity: 1015, billirubin (++), Hemoglobinuria was positive with Heller test. Abdominal ultrasonography reported sludge ball (20mm*9mm) in gullbladder.

After intensive photo therapy and the two times exchange the patient symptoms and lab data were recovered. After seven days the patient was discharged with follow up.

DISCUSSION

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most important disease of the hexose monophosphate pathway[1,2] and is responsible for neonatal jaundice[3] that can be very severe in G6PD deficiency and induces permanent damage to the brain and causes kernicterus and death.[16]

In the usual pattern of G6PD deficiency, symptoms develop 24–48 hours after a patient has ingested a substance that has oxidant properties. Drugs that have these properties include aspirin, sulfonamides, and antimalarials, such as primaquine.[2]

The most important way for prevention and reduction in the incidence of clinical symptoms of G6PD deficiency is to avoid oxidative agents like infection, fava beans and oxidative drugs that induce hemolysis, also screening of newborns for early diagnosis of G6PD deficiency and proper education is recommended.[17]

The plant of henna is grown extensively in the Middle East and Africa.[6] When the dried leaves are soaked in water and applied to the hair, skin, or nails, an auburn to red color develops, hence, its worldwide use as a cosmetic agent.[6] In some countries, ceremonial and social events, including weddings and circumcisions, are celebrated by the application of henna to the skin to create a variety of designs and patterns.[6] Of course henna is used in some traditional and non-scientific treatment for decreasing hyperbilirubinemia andanti-inflammatory, antipyretic, and analgesic effects of henna in rats are reported.[18]

An important chemical ingredient of henna is lawsone (2-hydroxy-1, 4naphthoquinone). Its structure and redox potential is similar to 1,4naphthoquinone, a metabolite of naphthalene and potent oxidant of G6PD deficient cells.[6] Because of these similarities, together with the knowledge that percutaneous absorption of naphthalane may hemolyze G6PD-deficient red cells, studies were designed to determine whether lawsone also may cause oxidant injury to red cells.[6] These in vitro observations indicate that lawsone, a chemical constituent of henna, is capable of inducing oxidative injury to G6PD-normal red cells, and even more so to G6PD (A-)red cells.[6]

The effect of henna on erythrocytes is known but very few cases have been reported.[6] G6PD deficient newborns admitted for hyperbilirubinaemia in Kuwait had significantly higher Serum billirubin and reticulocyte counts after exposure to henna.[9] However, their haemoglobin concentrations were neither critical nor different from G6PD deficient babies without henna exposure. Life threatening consequences of henna application have only been described in Sudan, namely angioneurotic edema associated with admixture of para-phenylenediamine to henna— not with haemolysis, and with a clearly different pathophysiological basis[9,11] but Raupp et all collected 4 cases over one year, suggested a life threatening potential of henna causing acute haemolysis in G6PD deficient children.[12]

Kandil et al reported that henna might induce hemolysis in G6PD deficient male newborns.[9] Zinkam et al notified that in G6PD enzyme deficient patients, henna causes oxidative hemolysis and hyper-bilirubinemia.[6] Lawsone was found to cause hemolysis, in a dose-dependent manner, as reflected by decreased blood packed cell volumes and hemoglobin levels and by histopathological changes in spleen, liver and kidney.[13]

In our case, his bilirubin was 12 and after using of henna (for treatment of hyperbillirubinemia!), raised to 50 and kernicterus symptoms was appeared. A drug or infection that may cause hemolysis in G6PD deficiency was not determined, fever or other evidence of acute infection was not observed either, so hemolysis was thought to be caused by henna. Our case suggests a life threatening potential of henna causing acute hemolysis in G6PD deficient infants that can lead to kernicterus.

We have reported this case because when the literature is searched thoroughly, acute hemolysis after applying henna to the whole body is a very rare condition. We suggest that a contiuous health

education program and universal G6PD screening should be initiated to prevent the use of henna dye in G6PD enzyme deficient patients.

REFERENCES

1. Mehta A, Mason PJ, Vulliamy TJ. Glucose-6-phosphate dehydrogenase deficiency. Best Prac Res Clinaematol. 2000;13:21-38.
2. Nelson Segel GB. Hemolytic anemia. In: Behrman, K Liegman, Jenson. Nelson Textbook of Pediatric. 18th ed. Philadelphia, Saunders. 2007:1636-1638.
3. WHO Working Group. Glucose-6-phosphate dehydrogenase deficiency. Bull WHO 1989;67:601-611.
4. Noori-Daloii MR, Najafi L, Ganji SM, Hajebrahimi Z, Sanati MH. Molecular identification of mutations in G6PD gene in patients with favism in Iran. J Physiol Biochem. 2004;60:273-277.
5. Beutler E. G6PD deficiency. Blood. 1994;84(11):3613-3636.
6. Zinkham WH, Oski FA. Henna: A Potential Cause of Oxidative Hemolysis and Neonatal Hyperbilirubinemia. Pediatr. 1996;97:707-709.
7. McMillan DC, Sarvate SD, Oatis JE, Jollow DJ. Role of Oxidant Stress in Lawsone-Induced Hemolytic Anemia. Toxicol Sci. 2004;82:647-655.
8. Seyedzadeh A, Hemmati M, Gheiny S. Henna- Induced Severe Hemolysis:In Glucose 6- Phosphate Dehydrogenase Deficiency. Pak J Med Sci. 2007;23:119-121.
9. Kandil HH, Al-Ghanem MM, Sarwat MA, Al-Thallab FS. Henna (Lawsoniainermis Linn.) inducing haemolysis among G6PD deficient newborns. A new clinical observation. Ann Trop Paediatr. 1996;16:287-291.
10. Al-Sheikh OA, Gad el-Rab MO. Allergic contact dermatitis: clinical features and profile of sensitizing allergens in Riyadh, Saudi Arabia. Int J Dermatol. 1996;35:493-497.
11. Sir Hashim M, Hamza YO, Yahia B, Khogali FM, Sulieman GI. Poisoning from henna dye and para-phenylenediamine mixtures in children in Khartoum. Ann Trop Paediatr. 1992;12:3-6.
12. Raupp P, Hassan JA, Varughese M, Kristiansson B. Henna causes life threatening haemolysis in glucose-6-phosphate dehydrogenase deficiency. Arch Dis Child. 2001;85:411-412.
13. Munday R, Smith BL, Munday CM. Comparative toxicity of 2-hydroxy-3-alkyl-1,4-naphthoquinones in rats. Chem Biol Interact. 1995;98:185-192.
14. Babich H, Stern A. In vitro cytotoxicities of 1,4-naphthoquinone and hydroxylated 1,4-naphthoquinones to replicating cells. J Appl Toxicol. 1993;13:353-335.
15. Shihabi A, Li WG, Miller FJ, Weintraub NL. Antioxidant therapy for atherosclerotic vascular disease: the promise and the pitfalls. Am J Physiol Heart Circ Physiol. 2002;282:H797-H802.
16. Kaplan M, Hammerman C. Glucose-6-Phosphate Dehydrogenase Deficiency: A Worldwide Potential Cause of Severe Neonatal Hyperbilirubinemia. Pediatr Rev Neo Rev. 2000;1:e32-e39.1.
17. Farhud DD, Yazdanpanah L. Glucose-6-phosphate dehydrogenase (G6PD) Deficiency. Iranian J Publ Health. 2008;37(4):1-18.
18. Ali BH, Bashir AK, Tanira MO. Anti-inflammatory, antipyretic and analgesic effects of Lawsoniainermis L. (henna) in rats. Pharmacology. 1995;51:356-363.