Clinical Article

Henna Induced Acute Hemolysis in a G6PD-Deficient Patient: A Case Report

Murat Söker, MD; Celal Devecioglu, MD; Kenan Haspolat, MD; Bünyamin Dikicl, MD; Ömer Dogru, MD

Abstract

Lawsone is a chemical present in henna, the crushed leaves of which are used worldwide as a cosmetic agent to stain hair, skin and nails. Vitro observations showed that lawsone was capable of causing oxidative hemolysis in a dose dependent manner in rats. In the literature, acute hemolysis after henna dye application over the human body is rare except in infancy. A Glucose-6-Phosphate Dehydrogenase (G6PD) enzyme deficient patient with acute hemolysis after exposure to henna is presented. *Int Pediatr. 2000;15(2):114-116.*

Key words: Acute Hemolysis, Henna, Glucose-6-Phosphate Dehydrogenase (G6PD) deficiency.

Introduction

Glucose-6-Phosphate Dehydrogenase (G6PD) deficiency, is an important X linked enzyme deficiency of the pentose phosphate pathway. It is the most common human enzyme deficiency in the world and it affects an estimated 400 million people. Major clinical manifestations are drug-induced acute hemolysis and/or neonatal jaundice and a very small proportion of G6PD-deficient individuals have a chronic hemolytic anemia (CHA). Both of these conditions are directly related to the inability of specific cell types to regenerate reduced nicotinamide adenine dinucleotide phosphate (NADPH); the G6PD enzyme normally catalyzes this reaction. Drugs, infections, nutriments or oxidizing agents may cause non-spherocytic hemolytic anemia in G6PD enzyme deficient patients.

Henna is used for medication and cosmetics in some regions of the world especially in the middle east.[1-3] Henna is shown to cause severe hemolytic anemia in animals.[4-6] In the literature, hemolysis linked to henna application in G6PD enzyme deficient patients is rare except in infancy.[1,7]

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

From the Department of Pediatrics, Dicle University Faculty of Medicine, Diyarbakir, Turkey.

Address reprint requests to the Department of Pediatrics, Dicle University Faculty of Medicine, Diyarbakir, Turkey, sokerm@hotmail.com (Dr Söker).

Case Report

The patient (NK) was an eleven years old boy, presented to Dicle University Faculty of Medicine, Department of Pediatrics, with paleness, weakness and reddish colored urine. There was a history of psoriatic lesions throughout the whole body. These lesions were more prominent at the knees and elbows (Fig 1). He was treated with some medications repeatedly without improvement. The patient was "hennaed" throughout the whole body and the day after henna, he had reddish urine and paleness. He was hospitalized with these complaints in our pediatric hematology clinics. On physical examination he had an axillary temperature of 36.5°C, pulse of 120/min, respiratory rate of 28/min and blood pressure of

Fig 1. - General appearance of the patient.

80/60 mmHg. At inspection, reddish brown colored psoriatic lesions were spread over the skin, throughout body. The lesions were more prominent at extensor surfaces of extremities.

Other physical findings were normal. Laboratory investigations revealed hemoglobin of 4.5 gm/dL, hematocrit of 13%, white blood cell count of 6700/mm^3, red blood cell count of 1 190 000/mm^3 and platelet count 342 000/mm^3. Peripheral smear showed 70% neutrophils, 6% lymphocytes, 4% monocytes, anisocytosis(+), poikilocytosis(+), spherocytosis(-), heinz body (+) after crystal violet dye, thrombocytes enough and clustered, sickling(-) and reticulocyte count 6.2%. Biochemical analysis yielded blood urea nitrogen 90 mg/dL, aspartate aminotransferase 420 iu/L, alanine aminotransferase 104 iu/L, indirect bilirubin 5.2 mg/dL, creatinine 0.8 mg/dL, lactate dehydrogenase 721 iu/L, creatine kinase 254 iu/dL and the other biochemical parameters within normal limits. Serology for CMV, EBV, HBV and HCV was negative. Direct Coombs test was negative. Qualitative G6PD enzyme deficiency was established with Brewerin visual test. Urine analysis: red colored, specific gravity: 1015, protein (+), billirubin (+++) and sediment contained 1-2 leucocytes per high power field. Hemoglobinuria was positive with Heller test. Abdominal ultrasonography was normal.

After erythrocyte transfusion and supportive treatment, the patient recovered in short time. The family was informed about the disease and recalled for checkup after 11 days.

Discussion

Henna is a dye, the source of which is the shrub lawsonia alba. The plant is grown extensively in the Middle East and Africa.[7] 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.[7] 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.[7] Approximately 100 g of henna are needed to stain the hands and feet of an adult, and the dyeing process requires 6 hours or more.[3]

An important chemical ingredient of henna is lawsone (2-hydroxy-1,4 naphthoquinone), constituting about 1% by weight of the crushed leaves. The structure and redox potential of lawsone is similar to that of one of the naphthalene metabolites, 1,4 naphthoquinone, a potent oxidant of G6PD-deficient red cells. 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.[7] 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.[7]

In our case, a drug or infection that may cause hemolysis in G6PD deficiency was not determined. Hemolysis is thought to be caused by henna. The effect of henna on erythrocytes is known but very few cases have been reported.[1,7] Kandil et al[1] reported that henna might induce hemolysis in G6PD deficient male newborns. Zinkam et al[7] notified that in G6PD enzyme deficient patients, henna causes oxidative hemolysis and hyper-bilirubinemia. 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.[4] Although whole body is rarely stained with henna, our patient was hennaed throughout the whole body.

Henna is not only used for cosmetics, but also for medication as well. In particularly rural areas in Turkey, local henna is applied to the hands and hair for cosmetics in some traditional ceremonies.

For many years, henna has been used for the treatment of skin disorders, many of which may be fungal in origin, because of the presumed anti-fungal properties of henna, together with its structural similarity to 5-hydroxynaphthoquinone, a potent fungicide.[7]

Seborrheic dermatitis and fungal infestations are other reasons why henna is used topically on lesions in some people. In this case, henna was used for treatment of psoriatic lesions. Anti-inflammatory, antipyretic, and analgesic effects of henna in rats are reported.[8] In some trials, henna is considered to have low allergic potential and rarely causes contact dermatitis.[2,9] A case of a hairdresser who developed an immediate-type hypersensitivity with urticaria, rhinitis, and bronchial asthma on exposure to henna is reported by Majoie et al.[10]

Besides hemolysis, nephrotoxicity is another effect of henna, which is clinically presented with renal enlargement, elevated plasma levels of urea and creatinine, and tubuler necrosis.[3-5] In this patient, there was no nephrotoxic effect of henna; urea and creatinine were in normal range.

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. A local health education program should be established to prevent the use of henna dye in G6PD enzyme deficient patients.

References

1. Kandil HH, al-Ghanem MM, Sarwat MA, al-Thallab FS. Henna (Lawsonia inermis Linn.) inducing haemolysis among G6PD-deficient newborns. A new clinical observation. *Ann Trop Paediatr*. 1996; 16:287-291.
2. 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.

3. 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.
4. Munday R, Smith BL, Fowke EA. Haemolytic activity and nephrotoxicity of 2-hydroxy-1,4-naphthoquinone in rats. *J Appl Toxicol*. 1991; 11: 85-90.
5. Munday R, Smith BL, Munday CM. Comparative toxicity of 2-hydroxy-3-alkyl-1,4-naphthoquinones in rats.*Chem Biol Interact*. 1995 17; 98:185-192.
6. Babich H, Stern A. In vitro cytotoxicities of 1,4-naphthoquinone and hydroxylated 1,4- naphthoquinones to replicating cells. *J Appl Toxicol*. 1993;13:353-358.
7. Zinkham WH, Oski FA. Henna: a potential cause of oxidative hemolysis and neonatal hyperbilirubinemia. *Pediatrics*. 1996; 97:707-709.
8. Ali BH, Bashir AK, Tanira MO. Anti-inflammatory, anti-pyretic, and analgesic effects of Lawsonia inermis L. (henna) in rats. *Pharmacology*. 1995; 51:356-363.
9. Garcia ortiz JC, Terron M, Bellido J. Contact allergy to henna. *J int Arch Allergy Immunol*. 1997; 114:298-299.
10. Majoie IM, Bruynzeel DP. Occupational immediate-type hypersensitivity to henna in hairdresser. *Am J Contact Dermat*. 1996; 7:38-40.