are satisfied, the term NOS is used.⁴⁻⁸ Thus, FSGS of NOS type is a histologic diagnosis of exclusion.⁵⁻¹¹

Many investigations have been published in the literature on the prevalence and clinicopathological characteristics of different histologic variants of FSGS. In the case of Momeni and colleagues, it is unclear which variant of FSGS existed. Furthermore, it is better to specify the presence or absence of podocytopathy (presence of podocytic hyperplasia, podocytic hypertrophy, or podocytic capping), intracapillary hyalinosis, or glomerular enlargement, which are popular in some variants of FSGS.^{4,6,11} Furthermore, the authors mentioned the presence of mesangial proliferation addressed in the Figure 3; however, a short look to the Figure provided in the article showed absence of proliferation of mesangial cells. In fact, mesangial proliferation is generally assumed to be more than 3 cells per each mesangial area.^{2,10,12} Moreover, immunofluorescent study in this case was negative; thus, it should be better to test and mention the serum value of antineutrophil cytoplasmic antibodies or antiphospholipids antibody panel tests, too.

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Possible Nephrotoxicity After Topical Application Of A Natural Herb, Henna

Dear Editor,

Herbal products are commonly used with the conception that they are natural and safe to use; however, there are several reports on their side effects including nephrotoxicity.¹⁻³ Henna (*Lawsonia inermis Linn*) is used frequently as a hair dye in some countries including Iran. There are some reported cases of toxicity after the use of henna. Henna-induced toxicity is particularly attributed to

paraphenylenediamine (PPD), an alanine derivative, which is added as a strong oxidizer and dying accelerator to henna powder.⁴ Paraphenylenediamineinduced systemic toxicities manifest as severe edema of the face, neck, tongue, pharynx, and larynx, which can be followed by anaphylaxis, intravascular hemolysis, rhabdomyolysis, and acute kidney injury (AKI). Paraphenylenediamine toxicities are reported usually after its ingestion; however, there are some



cases of systemic toxicity after its local application as hair dye.⁵⁻⁷ We report a case of a woman who presented with nausea, vomiting, face edema, and rise in serum creatinine, possibly after application of henna to her hair.

A 62-year-old woman was referred to a nephrologist after detection of an increased serum creatinine concentration of 5.6 mg/dL and lower extremity edema. She complained of face edema, nausea, and loss of appetite, which followed by weight loss since 2 month earlier, after application of henna powder. Face edema was resolved spontaneously but nausea and dizziness continued. She mentioned decrease in her urine output during these two months. She reported the same signs about 1 year earlier after the application of henna powder that resolved after the hospital admission and treatment without receiving dialysis. She did not have any record from her previous admission. Her past medical history was positive for hypertension, which was controlled by atenolol, 100 mg, daily, without any notification that she suffered renal insufficiency.

At admission in our nephrology department, her blood pressure was 130/80 mm Hg and pulse rate was 86 beats per minute. Renal ultrasonography showed right kidney size to be 58 × 119 mm with multiple cortical cysts and no evidence of hydronephrosis, while the left kidney could not be detected. Other laboratory findings of particular importance were a serum creatinine level of 6.7 mg/dL, anemia (hemoglobin level of 7.7 g/dL), and proteinuria (545 mg in 24-hour urine collection). A dimercaptosuccinic acid renal scintygraphy reported cortical defects and developmental lobulated kidney that was suggestive for kidney failure and the left kidney was not visualized. Assuming a diagnosis of AKI, the patient was in the "loss stage" based on the RIFLE criteria.⁸ Her echocardiography showed an ejection fraction of 45%, normal left ventricle and right ventricle sizes with mild left ventricle dysfunction, mild mitral regurgitation, moderate to severe aortic insufficiency and mild tricuspid regurgitation. Normal skull radiography examination and serum and urine protein electrophoresis of the patient ruled out multiple myeloma. Secondary workup including serum levels of antineutrophil antibody, antineutrophil cytoplasmic antibody, complements C3, C4, and CH50; double-stranded DNA, anti-glomerular basement membrane antibody,

immunoglobulin A, and β -2 microglobulin were all normal. Viral markers were negative.

Due to her nausea, loss of appetite, and edema, hemodialysis was performed using a jugular doublelumen catheter. Her erythrocyte sedimentation rate was 96 mm/h, so she was instructed to follow malignancy workup as outpatient. She underwent 4 hemodialysis sessions as inpatient and was discharged after arteriovenous fistula placement.

Intoxication with PPD was first reported in 1924 as dermal allergy in a hairdresser.⁶ Although it is uncommon in the west, there are plenty of case reports from Africa and Asia. In a survey by Kaballo and colleagues, about 24% of acute tubular necrosis cases in Africa were considered as PPD intoxication.9 Basically, PPD can be absorbed from the skin, but ingestion, mostly because of suicidal attempts, could cause more serious complications. The symptoms are dose related and include angioedema, rhabdomyolysis, intravascular hemolysis, sever cardiac arrhythmias, and AKI. First symptoms could appear 4 to 6 hours after ingestion.⁷ Pathological renal changes in acute poisoning are mostly in favor of acute tubular necrosis, while the chronic intoxication can cause glomerular injuries.⁹ Mortality rate could reach up to 31% if proper supportive management is delayed.¹⁰ There is no specific antidote to PPD and treatment is primarily conservative, such as gastric decontamination, forced diuresis, and also urine alkalinisation. Patients with respiratory distress may benefit from ventilator support. Dialysis is usually required in kidney failure cases.¹¹ Our patient had contact with PPD, but it was not evident if she ingested low doses accidentally during washing her hair. Her symptoms of face edema, nausea, and loss of appetite were getting started right after henna application. She was probably a case of chronic kidney disease due to her hypertension and single kidney state, but it could be accentuated by PPD intoxication that resulted in end-stage renal disease.

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