stress, although there is no bacterial infection. In these situations, after the elevation of procalcitonin values, a rapid decline is observed in the follow-up measurements. [3] On the other hand, procalcitonin can have false negative or lower values in early course and localized site of an infection. [3] In this context, repeated measurements of procalcitonin could have been performed. Therefore, it would have been better if the authors had mentioned these conditions as limitations.

Lastly, procalcitonin is affected by a variety of infectious agents. Microbiological assessment is crucial while evaluating the procalcitonin levels. In comparisons of Gram negative agents with Gram positives, procalcitonin levels have been found to be higher in Gram negatives. [4] Therefore, it would have been more accurate, if the authors had evaluated procalcitonin levels according to infectious agents in greater detail in this study.

In conclusion, further studies are needed to determine the association between procalcitonin and VUR. We are of the opinion that procalcitonin should be evaluated with other independent variables and markers (eg, bacterial agents, C-reactive protein, and erythrocyte sedimentation rate) to provide the required information about the inflammatory status of the patient.

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Re: Kartagener Syndrome With Focal Segmental Glomerulosclerosis

Dear Editor,

The article published by Momeni and colleagues, entitled "Kartagener syndrome with focal segmental glomerulosclerosis" in the esteemed *Iranian Journal of Kidney Diseases* had some interesting points.[1] In this article, they explained a 27-year-old female patient as a case of Kartagener syndrome. Renal biopsy in this case showed segmental scar with adhesion to the Bowman capsule, which was indicative of focal segmental glomerulosclerosis (FSGS).[1] We would like to remind a few points. Indeed, it is indispensable to classify the variant of FSGS, too. In 2004, a group of renal pathologists suggested a consensus-based morphological classification system for FSGS based exclusively on light microscopic examination, commonly known as the Columbia classification.[2-5] According to this classification, 5 morphologic variants of FSGS are described: FSGS, not otherwise specified (NOS) or the classic type; cellular variant; collapsing variant; perihilar variant; and tip variant.[5-7] In brief, tip variant of FSGS necessitates the exclusion of collapsing variant and presence of at least one glomerulus with segmental lesion involving the tip domain of the glomerular capillary tuft. In the perihilar variant, the segmental sclerotic lesion is situated at the vascular pole and requires the exclusion of collapsing, tip, or cellular lesion. The cellular variant needs exclusion of collapsing and tip lesions, and is defined by segmental endocapillary hypercellularity occluding lumina in at least 1 glomerulus.[5-8] Collapsing variant was defined by collapse of at least 1 capillary loop with hyperplasia and hypertrophy of overlying visceral epithelial cells, irrespective of the presence of other variants of FSGS. In cases where none of these definitions

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] Paraphenylenediamine-induced 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