dL, creatinine 4.16 mg/dL, alkaline phosphatase 1220 U/L, serum sodium 142 mEq/L, potassium 3.6 mEq/L, calcium 8.5 mg/dL, albumin 3.85 g/dL, phosphorus 2.2 mg/dL, vitamin D 122.8 ng/mL, serum bicarbonate 11.0 mEq/L, thyroid-stimulating hormone 1.9 µIU/mL, and urine albumin-to-creatinine ratio 283.6 mg/g. Her urine examination showed glycosuria and proteinuria. The tubular maximum phosphate reabsorption was 2.0 mg/dL, suggesting phosphaturia [Supplementary Table 1]. Ultrasonography was suggestive of bilateral small kidneys with medical renal disease. Dual-energy X-ray absorptiometry scan showed a Z-score of –3 at the left hip joint, indicating osteoporosis. The patient was diagnosed with Fanconi syndrome with right femoral neck fracture with osteoporosis. Tenofovir was discontinued, and she was started on sodium bicarbonate, telmisartan, calcium, iron, potassium citrate syrup, phosphorus, and injection denosumab 60 mg subcutaneously. Over the six-month follow-up period, the patient had marked symptomatic improvement and her renal function improved.

Tenofovir can lead to acute kidney injury, chronic kidney disease, proximal tubular cell damage, and Fanconi syndrome as well as bone problems like osteopenia and osteoporosis. Therefore, patients on long-term tenofovir should undergo close monitoring of renal parameters and bone mineral density markers to prevent nephrotoxicity and adverse bone effects.[2]

Conflicts of interest: There are no conflicts of interest.

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Intravascular Hemolysis During Therapeutic Plasma Exchange Using 5% Human Albumin: What is Missed?

Dear Editor,

Therapeutic plasma exchange (TPE) is the backbone of ABO-incompatible transplant. An untoward event like hemolysis during pretransplant procedures can lead to unnecessary blood transfusions, impacting graft survival. We highlight a rare case of hemolysis during a TPE session using centrifugation method with 5% human albumin.

A 38-year-old woman with end-stage renal disease was scheduled for ABO-incompatible renal transplantation from her 44-year-old husband with 6/6 human leukocyte antigen (HLA) mismatch. To lower pretransplant antibody titer, the patient underwent plasmapheresis, and 5% human albumin of the brand Albufirst (Halstead Pharma Pvt. Ltd., Hyderabad) was used as the replacement fluid.

During the first session, after processing half of the calculated plasma volume with 5% albumin, the patient experienced severe abdominal pain with cramps and the TPE machine triggered a red cell detector alarm. Plasma waste bag observation revealed reddish discoloration [Figure 1] suggesting hemolysis and was confirmed by the

Figure 1: Reddish discoloration of plasma waste bag due to hemolysis during TPE procedure. TPE: Therapeutic plasma exchange.

pinkish discoloration of the centrifuged post-procedure blood sample.

The procedure was paused to check for any reversible cause of hemolysis like equipment-related or technical factors (faulty clamping, high blood flow rate, or access issue). Subsequently, hemolysis was attributed to 5% albumin solution, which failed quality control standards, including low sodium concentration (24 mmol/L), albumin content of 4.98 g/dL, and a notably low osmolality (70 mmol/kg). Subsequent TPE sessions using fresh frozen plasma instead of 5% albumin proceeded without complications. Posttransplant, the patient had a successful outcome with normal graft function.

As an immediate corrective measure, the remaining hospital stock was returned to the supplier. The hospital administration was promptly informed, and the analysis report was also forwarded to the hospital's pharmacovigilance cell for further review. Going forward, a mandatory hemolysis test at our blood center will be required for each new albumin lot. This incident highlights the critical need for stringent quality control in replacement fluids for TPE, as lapses can jeopardize patient safety.

Conflicts of interest: There are no conflicts of interest.

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"Jogger's Nephritis" After Pilgrimage

Dear Editor,

A 42-year-old lady, nondiabetic and normotensive, presented with complaints of painless, red-colored urine of 3 days duration. She had been on a pilgrimage to Tirumala. She had walked from her village, about 270 km away, with her family. After that, she took steps to Tirumala, another 16.9 km. There was no history of decreased urine output, dysuria, frothy urine, or pyuria. She had no history of non-steroidal anti-inflammatory drugs intake.

Clinical examination revealed blood pressure of 110/70 mm Hg, and systemic examination was normal except for tenderness of the lower limbs. A diagnostic algorithm was followed to exclude all causes of hematuria; urine examination showed the presence of trace albumin and 8–10/hpf red blood cells (RBCs). The RBCs were isomorphic, which was confirmed with phase contrast microscopy. Other investigations revealed the following: serum creatinine: 1.0 mg/dL; serum potassium: 4.8 meq/L; serum bilirubin: 0.4 mg/dL; lactate dehydrogenase: 105 IU/L; C3: 209.15 mg/dL; C4: 33.75 mg/dL; ASO titer: 83.57 IU/mL; serum myoglobin: 36 ng/mL; urine myoglobin: negative; and 24-hour urine protein: 328 mg. Ultrasound of the abdomen revealed right kidney: 9.3 × 4.1 cm and left kidney: 9.4 × 4.2 cm; no calculi, Doppler of renal arteries and veins did not reveal nutcracker syndrome. Her renal biopsy, including electron microscopy, was normal. After 96 hours of admission, the urine color changed from reddish brown to light red and pale yellow. The patient was discharged, and in the subsequent follow-up, there were no RBCs in the urine. It was hypothesized that the hematuria could be due to her long walk.

Hematuria in runners was first reported in the 18th century by Italian physician Bernardini Ramazzini,[1] who was later named "athletic pseudo-nephritis."[2] The pathogenesis of hematuria is complex and multifactorial. The mechanisms proposed are as follows: (a) Renal vasoconstriction and ischemia due to preferential shunting of blood to the exercising muscles;[3] (b) "Foot strike hemolysis," or trauma to the RBCs circulating through the sole;[4] (c) Long-distance running is known to cause trauma to the bladder, possibly due to repeated impacts of the flaccid wall of the bladder against the bladder base.[5]

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