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.

Abhilasha Soni[1], Manish Chaturvedy[1], Rajesh Jhorawat[1], Nitin Bajpai[1]

[1]Department of Nephrology, AIIMS, Jodhpur, Jodhpur, India

Corresponding author: *Nitin Bajpai, Department of Nephrology, AIIMS, Jodhpur, Jodhpur, India. E-mail: drnitinbajpai@gmail.com*

How to cite this article: Soni A, Chaturvedy M, Jhorawat R, Bajpai N. Intravascular Hemolysis During Therapeutic Plasma Exchange Using 5% Human Albumin: What is Missed? Indian J Nephrol. 2025;35:312-3. doi: 10.25259/IJN_437_2024

Received: 01-08-2024; **Accepted:** 12-08-2024
Online First: 12-09-2024; **Published:** 25-02-2025

DOI: 10.25259/IJN_437_2024

"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]

Conflicts of interest: There are no conflicts of interest.

Lia Sarah Anish[1], Aishwarya P Lakshmi[2], S Mathini[1], R Ram[1]

[1]Department of Nephrology, Sri Venkateswara Institute of Medical Sciences, Tirupati, Andhra Pradesh [2]Department of Nephrology, Saveetha Medical College, Kuthambakkam, Madras, Tamil Nadu, India

Corresponding author: *R Ram, Department of Nephrology, Sri Venkateswara Institute of Medical Sciences, Tirupati, Andhra Pradesh, India. E-mail: ram_5_1999@yahoo.com*

References

1. Brieger GH. The diseases of runners: A view from the eighteenth century—A commentary. Pharos Alpha Omega Alpha Honor Med Soc 1980;43:29–32.

2. Gardner KD. Athletic pseudonephritis; alteration of urine sediment by athletic competition. J Am Med Assoc 1956;161:1613–7.
3. Jones GR, Newhouse I. Sport-related hematuria: A review. Clin J Sport Med 1997;7:119–25.
4. Miller BJ, Pate RR, Burgess W. Foot impact force and intravascular hemolysis during distance running. Int J Sports Med 1988;9:56–60.
5. Blacklock NJ. Bladder trauma in the long-distance runner: "10,000 metres haematuria". Br J Urol 1977;49:129–32.

How to cite this article: Anish LS, Lakshmi AP, Mathini S, Ram R. "Jogger's Nephritis" After Pilgrimage. Indian J Nephrol. 2025;35:313-4. doi: 10.25259/IJN_480_2024

Received: 20-08-2024; **Accepted:** 20-08-2024;
Online First: 17-10-2024; **Published:** 25-02-2025

DOI: 10.25259/IJN_480_2024