Persistent hypokalemia in an acute lymphoblastic leukemia patient

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Hypokalemia was evaluated (Urine k-20 mEq/L), (Urine sodium-134 mEq/L (undiluted)), (Serum osmolality-287 mosm/L), (Urine osmolality- 400 mEq/L), and TTKG-13, indicating ongoing renal loss. Presently the patient is on both magnesium and potassium replacement, but despite that, the serum potassium is low all along.

DISCUSSION

It has been clearly documented that Amphotericin-B induces renal potassium wasting and can produce substantial potassium deficit. Levels below 3 mmol/L have been reported in 12 to 40% of the patients in recent publications.[1-4] It has been proposed that both tubular injury and renal vasoconstriction play an important role in Amphotericin-B nephrotoxicity.[5,6] Our patient did not develop this complication because of the chemotherapeutic agent or antibiotic therapy, as persistent hypokalemia and mild metabolic acidosis is caused only by Amphotericin B. Our case clearly documents the fact that even after Amphotericin-B was stopped the patient could continue with hypokalemia, which might be a single indicator of tubular damage.

REFERENCES


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