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Hypokalemic paralysis as presenting manifestation of systemic diseases: a case series

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Abstract

Hypokalemia is a common issue in clinical settings, often indicating underlying systemic conditions that require careful evaluation. This study presents three cases where hypokalemic paralysis served as the initial symptom of systemic diseases. Key evaluation methods included clinical history, physical examination focusing on volume status, and acid–base assessment. The cases highlight the diverse etiologies of hypokalemia, including primary hyperaldosteronism, Sjogren's syndrome, and Crohn's disease.

Keywords Hypokalemia, Primary hyperaldosteronism, Renal tubular acidosis, Sjogren's syndrome, Crohn's disease

Introduction

Potassium plays a crucial role in maintaining cellular function. Excitable tissues, including nerves and muscles, heavily depend on a potassium gradient across the cell membrane for their normal function [1]. Disturbance in serum potassium levels (both hypokalemia and hyperkalemia) can manifest with cardiac conduction abnormalities and muscle weakness. Disorders of potassium balance can arise from abnormal renal handling of potassium in a variety of systemic diseases. In this study, we document hypokalemic paralysis as the initial manifestation of three distinct systemic illnesses.

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Case 1

A 26-year-old female, presented with bilateral lower limb weakness of 1-day duration. The weakness was sudden in onset and was not associated with any bowel or bladder disturbance. The patient had a history of difficult-to-treat hypertension for the last 4 years and has had two pregnancy losses at 12 weeks and 28 weeks of gestation, secondary to hypertensive crisis. The patient was being managed with amlodipine 10 mg, metoprolol 100 mg, and prazosin 10 mg daily. Her systolic and diastolic BP used to be in the range of 180–200 mmHg and 100–110 mmHg respectively. The patient was compliant with medications. The patient denied any history of swelling feet, cold intolerance, episodic sweating, palpitations, recurrent oral ulcers, photosensitivity, rash, arthralgias, and arthritis. Examination revealed a heart rate of 88 beats per minute, BP of 180/120 mmHg in the right upper limb, and 170/116 mmHg in the left upper limb. All peripheral pulses were palpable and there was no radio femoral delay. Ophthalmological examination revealed bilateral grade III hypertensive retinopathy changes. The cardiac, abdominal, and respiratory examination was unremarkable. Neurological examination revealed grade 1 power in bilateral lower limbs and grade 4 power in bilateral upper limbs. Deep tendon reflexes were absent. Planters were bilateral flexors. Sensory and

cerebellar functions were intact. Initial investigations revealed normal hematological and renal parameters with severe hypokalemia (serum potassium of 2.0 meq/l). ECG showed T-wave depression with prominent U waves and features of left ventricular hypertrophy. 2D ECHO showed mild to moderate left ventricular hypertrophy. ABG revealed metabolic alkalosis. urine analysis didn't reveal any proteinuria or microhematuria. Urinary potassium to creatinine ratio was 58 mmol/g of creatinine, suggestive of urinary potassium loss. USG KUB revealed normal kidneys. Renal vascular Doppler showed bilateral normal renal flows, peak systolic velocity, pulsatility, and resistive indices. Plasma aldosterone concentration (PAC), plasma renin, and aldosterone rennin ratio were 229 ng/dl, 1.25 mIU/L, and 184.68 ng/mIU respectively (detailed labs are shown in Table 1). The patient was started on spironolactone and potassium replacement. The patient's BP improved to 110/90 mmHg on spironolactone 100 mg daily and amlodipine 5 mg daily. CECT abdomen revealed a 20.1×23×11 mm hypodense lesion in the right adrenal gland (Fig. 1) with moderate to intense enhancement of the lesion and significant washout on delayed images. The CT enhancement on the non-contrast image was less than 15 HU highly suggestive of a right adrenal adenoma (Fig. 2). Expertise in adrenal vein sampling was not available in our center. Because of suppressed plasma rennin, very high plasma aldosterone, high aldosterone rennin ratio, urinary potassium loss, and very good response to spironolactone, right adrenalectomy was done after proper informed consent. Histopathology was suggestive of an adrenal cortical adenoma (Figs. 3 and 4). Repeat plasma aldosterone post-surgery came down to 11.20 ng/dl from baseline of 229 ng/dl. The patient presently has normal serum potassium (4.2 meq/l) and is off antihypertensives with systolic and diastolic BP in the range of 110–120 mmHg and 70–80 mmHg respectively. The final diagnosis of primary hyperaldosteronism secondary to aldosterone-producing adrenal adenoma presenting with hypokalemic paralysis was made.

Case 2

A 45-year-old female, normotensive, non-diabetic, presented with a 1-day history of weakness of all 4 limbs, with an inability to walk. The patient started with bilateral lower limb weakness which progressed over the day to involve bilateral upper limbs. The patient complained of dryness of mouth with difficulty in swallowing solid foods and foreign body sensations in both eyes for the last 1 year. She denied any history of skin rashes, oral ulcers, arthralgias, arthritis, and symptoms suggestive of Raynaud's phenomenon. There was no history of recent trauma. There was no history of similar illness in the past. Examination revealed a heart rate of 92 beats per minute and BP of 110/70 mmHg in the right upper limb. Neurological examination revealed grade 2 power in all 4 limbs. Deep tendon reflexes were absent in all 4 limbs. Sensations were intact and planters were

Table 1 Laboratory parameters of patient 1

Laboratory parameters of patient 1		Reference interval
Hemoglobulin (g/dl)	11.50	13.0–14.0
Sodium (meq/l)	144	135–145
Potassium (meq/l)	2.0	3.5–5.3
Serum pH/bicarbonate (meq/l)/PCo2 (mmHg)	7.47/ 30.6/ 42	
Urinary potassium (meq/l)	29	
Urinary creatinine (mg/dl)	50	
Blood urea (mg/dl)/serum creatinine (mg/dl)	70/ 1.1	17–43/0.5–1.1
Bil(mg/dl)/AST(IU/L)/ALT(IU/L)/ALP(IU/L)	0.40 / 32/ 47/ 120	
Total protein (g/dl)/albumin (g/dl)	6.5/ 4.4	6.4–8.3/3.5–5.2
Random glucose (mg/dl)	105	< 200
Calcium (mg/dl)/phosphate (mg/dl)	9.2/ 2.8	8.8–10.8/2.5–4.5
Magnesium (mg/dl)	2.1	1.6–2.5
Urine R/M	Albumin: nil, rbcs: nil, pus cells: occasional	
Aldosterone ng/dl (baseline)	229.0	2.21–35.3
Rennin mIU/L (baseline)	1.24	4.40–46.1
Aldosterone rennin ratio ng/mlU	184.67	< 20.6
TSH (µlU/ml)	3.4	0.5–5.0
HbsAg, anti-HCV, HIV	Non-reactive	
Aldosterone ng/dl (post-surgery)	11.20	

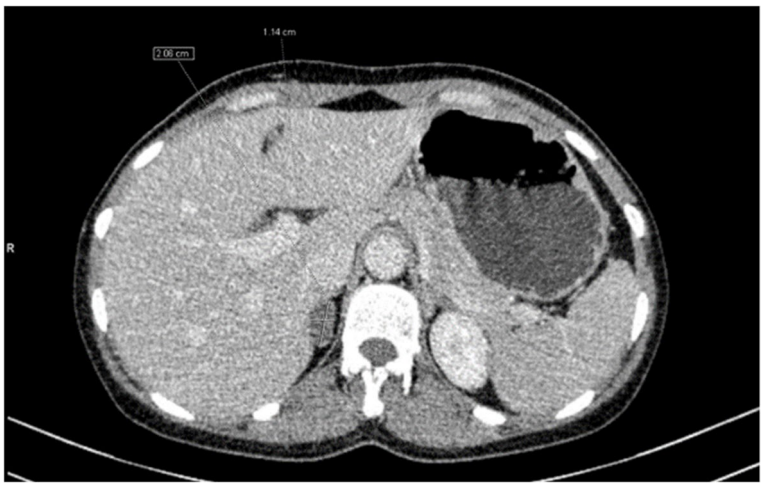

Fig. 1 CECT showing 2.06 × 1.14 cm right adrenal mass

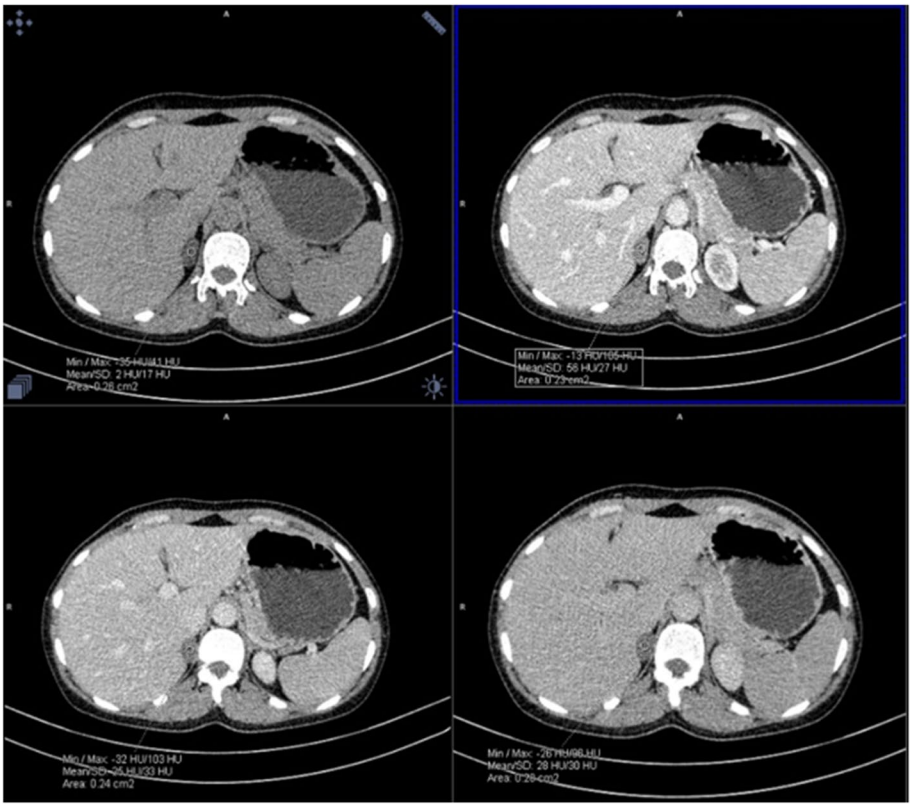

Fig. 2 CT density of the lesion in the unenhanced image is < 10HU, considered diagnostic of lipid-rich adrenal adenoma

bilateral flexor. There were no cerebellar signs. The cardiac, abdominal, and respiratory examination was unremarkable. The evaluation revealed severe hypokalemia (serum potassium of 1.9 meq/l) with severe metabolic acidosis (pH 7.23 with serum bicarbonate of 8.4 meq/l). Urine was alkaline with a pH of 6.6. urinary potassium was 44 meq/l, suggestive of renal potassium loss. There was mild renal dysfunction with a serum creatinine of 1.6 mg/dl. ANA was positive with an end-point titer of 1:320 using an immunofluorescence assay (Hep-2). ENA profile revealed 3 + positivity for anti-Ro and anti-La antibodies. Schirmer 1 on the right and left

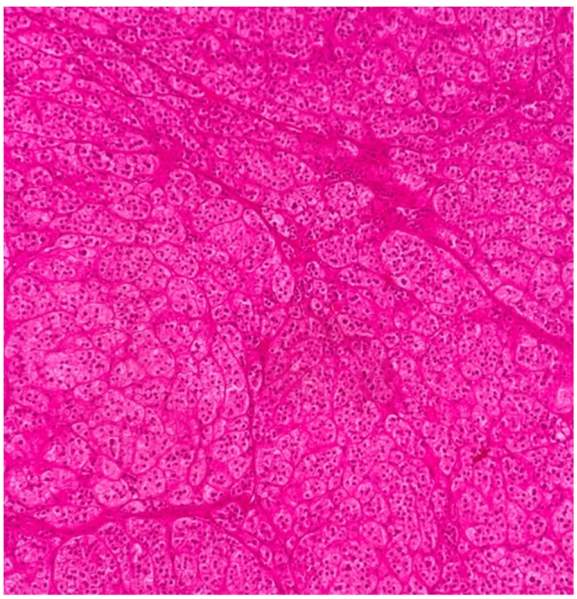

Fig. 3 H&E stained (100×) section shows circumscribed tumor composed of a solid sheet of large tumor cells forming nested pattern intersected by delicate fibrous septa

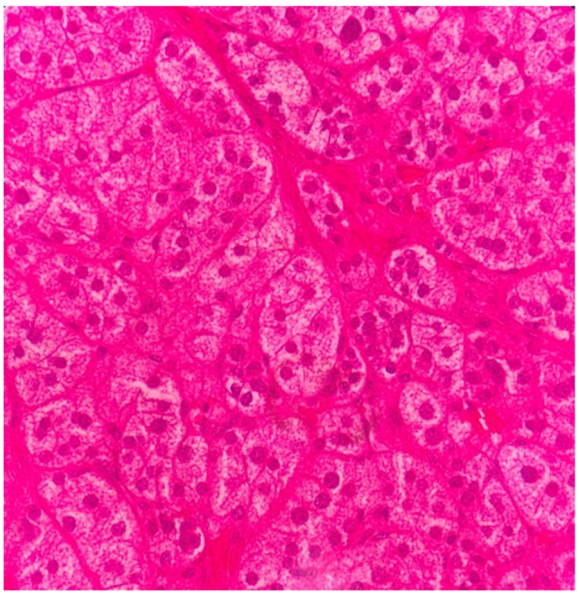

Fig. 4 H&E stained (400×) section shows mildly pleomorphic cells with round to oval central nucleus, finely clumped chromatin, conspicuous nucleoli, abundantly clear to granular eosinophilic cytoplasm, and distinct cell borders

eye showed 3 mm and 2 mm wetting respectively and Schirmer 2 showed right and left eye wetting at 2 mm and 1 mm respectively. Tear break-up time (TBUT) for the right and left eye was 4 s and 5 s respectively. USG KUB showed bilateral non-obstructing nephrolithiasis. Detailed labs are shown in Table 2. Based on the presence of symptoms suggestive of xerostomia, dry eyes, non-anion gap metabolic acidosis, severe hypokalemia, alkaline urine, positive Schirmer's test, antibodies against anti-Ro and anti-La and USG-proven bilateral nephrolithiasis, final diagnosis of Sjogren's syndrome with distal renal tubular acidosis was made. The patient was started on potassium replacement. Because of renal dysfunction, the patient was also started on prednisolone at 1 mg/kg body weight which was gradually tapered. The patient showed significant improvement in her symptoms and her renal function normalized on subsequent follow-up. The final diagnosis of Sjogren's syndrome with dRTA presenting with hypokalemic paralysis was made.

Case 3

A 17-year-old female, presented with sudden onset weakness of all 4 limbs for 3 h. She denied a history of oral ulcers, photosensitivity, rash, arthralgias, and arthritis. She denied symptoms suggestive of dryness of mouth and eyes There was no history of bowel and bladder involvement. There was no history of similar illness in the past. Examination revealed a heart rate of 102 beats per minute and BP of 100/64 mmHg in the right upper limb. Neurological examination revealed grade 2 power in all 4 limbs. Deep tendon reflexes were absent in all 4 limbs. Sensations were intact and planters were bilateral flexor. There were no cerebellar signs. The cardiac, abdominal, and respiratory examination was unremarkable. The evaluation revealed severe hypokalemia (serum potassium of 1.4 meq/l) with compensated non-anion gap metabolic acidosis (pH 7.37 with serum bicarbonate of 15.6 meq/l). Urine was alkaline with a pH of 6.5. Urinary potassium was 20 meq/l with trans tubular potassium gradient (TTKG) of 16.03, suggestive of urinary potassium loss. USG of the abdomen showed left nephrolithiasis. ANA was negative. Detailed labs are shown in Table 3. Based on non-anion gap metabolic acidosis, severe hypokalemia, renal potassium loss, alkaline urine pH, and nephrolithiasis, a diagnosis of distal RTA was made. The patient was given a potassium replacement. Her weakness improved and she was discharged home on potassium citrate. While being evaluated for the cause of dRTA, she was readmitted with 10–15 episodes of bloody diarrhea for 1 day. A colonoscopy was done which showed features of Crohn's colitis (ileocecal disease). The patient was initiated on sulfasalazine, azathioprine, and steroids. The patient entered into remission. The final diagnosis of dRTA likely secondary to Crohn's disease was made.

Table 2 Laboratory parameters of patient 2

Laboratory parameters of patient 2		Reference interval
Hemoglobulin (g/dl)	9.0	13.0–14.0
Sodium (meq/l)	138	135–145
Potassium (meq/l)	1.9	3.5–5.3
SerumpH/bicarbonate (meq/L)/PCo2 (mmHg)/ chloride (meq/L)/Anion gap	7.23/8.4/20.2/118/11.6	
Urinary potassium (meq/l)	44	
Urine pH	6.6	6.0–7.5
Blood urea (mg/dl)/serum creatinine (mg/dl)	28/1.6	17–43/0.5–1.1
Bil(mg/dl)/AST(IU/L)/ALT(IU/L)/ALP(IU/L)	0.9/28/34/140	
Total protein (g/dl)/albumin (g/dl)	6.9/4.52	6.4–8.3/3.5–5.2
Random glucose (mg/dl)	117	< 200
Calcium (mg/dl)/phosphate (mg/dl)	8.8/3.2	8.8–10.8/2.5–4.5
Magnesium (mg/dl)	1.9	1.6–2.5
Urine R/M	Albumin: nil, rbcs: nil, pus cells: 2–4/hpf	
TSH (μIU/ml)	2.4	0.5–5.0
Serum ANA by IFA (Hep 2)	Positive with endpoint titer 1: 320	
SS-A antibody	+ + +	
R0-52 antibody	+ + +	
SS-B antibody	+	
dsDNA	Negative	
HbsAg, anti-HCV, HIV	Non-reactive	

Table 3 Laboratory parameters of patient 3

Laboratory parameters of patient 3		Reference interval
Hemoglobulin (g/dl)	10.9	13.0–14.0
Sodium (meq/l)	142	135–145
Potassium (meq/l)	1.4	3.5–5.3
SerumpH/bicarbonate (meq/L)/PCo2 (mmHg)/ Chloride (meq/L)/Anion gap	7.37/15.6/27/116/10.4	
Urinary potassium (meq/l)	20	
Serum osmolality (mosm/kg)	284	
Urine osmolality (mosm/kg)	253	
TTKG	16.03	
Urine pH	6.5	6.0–7.5
Blood urea (mg/dl)/ serum creatinine (mg/dl)	77/0.58	17–43/0.5–1.1
Bil (mg/dl)/AST(IU/L)/ALT(IU/L)/ALP(IU/L)	0.58/42/44/160	
Total protein (g/dl)/albumin (g/dl)	6.2/3.9	6.4–8.3/3.5–5.2
Random glucose (mg/dl)	106	< 200
Calcium (mg/dl)/phosphate (mg/dl)	8.06/2.47	8.8–10.8/2.5–4.5
Magnesium (mg/dl)	1.7	1.6–2.5
Urine R/M	Albumin: nil, rbcs: nil, pus cells: nil	
Serum ANA by IFA (Hep 2)	Negative	
Anti endomysial antibody	Negative	
HbsAg, anti-HCV, HIV	Non-reactive	

Discussion

Hypokalemia is a common occurrence in clinical settings. It can stem from transient factors such as cellular shifts, or it can persist due to insufficient intake or excessive potassium depletion. The latter may result from either renal or extrarenal causes. By examining clinical history, conducting a thorough physical examination, and assessing the acid–base status, the underlying cause of hypokalemia can typically be identified [2]. In this report, we present three cases where hypokalemia and associated acid–base imbalances served as initial indicators of three distinct systemic diseases.

In patient 1, hypokalemic metabolic alkalosis in the presence of hypertension prompted us to the evaluation of hyperaldosteronism. Suppressed plasma rennin in the presence of high plasma aldosterone and aldosterone rennin ratio suggested primary hyperaldosteronism. However, hypokalemia is seen only in about 09 to 30% of patients with primary hyperaldosteronism [3], suggesting that hypertension in young even in the absence of hypokalemia warrants evaluation for hyperaldosteronism. The next step is to perform any one of four confirmatory tests: oral and intravenous salt loading test, the fludrocortisone suppression, and the captopril challenge test [4]. According to the Endocrine Society's clinical guidelines, confirmatory tests need not be performed if the PAC is > 30 ng/dL with undetectable plasma renin in the setting of spontaneous hypokalemia–such as in our case. Hence, we bypassed the confirmatory testing and proceeded with abdomen imaging instead. Although CT abdomen and pelvis with contrast using the adrenal protocol can be useful in identifying masses that can be potential aldosterone-producing adenomas, adrenal vein sampling (AVS) with subsequent determination of aldosterone/cortisol ratio remains the gold standard and the most accurate way to differentiate between the functional adrenal masses and non-functional incidentalomas. Expertise in AVS is not available in our center. Based on very high plasma aldosterone levels, suppressed plasma rennin, urinary potassium loss, and adequate pre-operative control of hypertension and correction of hypokalemia with spironolactone. The patient was taken for the right surgical adrenalectomy after proper informed consent. The patient is presently off anti-hypertensives with normal blood pressure and serum potassium.

In patient 2, hypokalemic non-anion gap metabolic acidosis, alkaline urine pH, and urinary potassium loss suggested renal tubular acidosis. Bilateral renal calculi suggested distal RTA as the likely subtype. Given symptoms of xerostomia and foreign body sensation in both eyes, the patient was evaluated for Sjogren's syndrome. American-European Consensus Group proposed the diagnostic criteria which include clinical information and laboratory examination values [5]. Our patient fulfilled the defined criteria for primary SS (dry eye and mouth longer than 3 months, positive Schirmer test, presence of autoantibodies SS-A and SS-B) when evaluated according to these diagnostic criteria.

Systemic manifestations occur in approximately 30 to 40% of the patients with primary Sjogren's syndrome [6]. Lymphocytic infiltration can cause interstitial nephritis, autoimmune primary biliary cholangitis, and obstructive bronchiolitis. Immune complex deposition can result in palpable purpura, cryoglobulinemia-associated glomerulonephritis, interstitial pneumonitis, and peripheral neuropathy [7]. The most commonly affected non-exocrine organ in Sjogren's syndrome is the kidney with the prevalence ranging between 2 and 67% [8, 9]. The most common form of renal involvement in Sjogren's syndrome is interstitial nephritis followed by distal renal tubular acidosis (dRTA), nephrogenic diabetes insipidus, and different forms of glomerular diseases, of which membranoproliferative glomerulonephritis (MPGN) and membranous nephropathy (MN) are the most common [10, 11]. Although how SS causes type 1 distal RTA is exactly not known, it is suggested that absent or decreased activity of H + -ATPase of intercalated cells located in collecting tubules of distal nephron may play a role in the pathogenesis. Hypokalemic paralysis seen in SS is rare and may sometimes mimic hypokalemic periodic paralysis (HPP). However, there are case reports of severe hypokalemic paralysis, which was later confirmed as Sjogren's syndrome [12] as in our case.

Both patient 1 and patient 2 denied any history of similar weakness in the past and the diagnosis of underlying systemic disorder was delayed for more than 4 years in patient 1 and more than a year in patient 2. This highlights the fact that the physician should have a very high index of suspicion for diagnosing these disorders. Hyperaldosteronism should always be suspected in any young hypertensive patient with difficult-to-control hypertension irrespective of the presence or absence of hypokalemia. A patient with renal tubular acidosis should always be evaluated for Sjogren's syndrome. Sjogren's syndrome is the second most common rheumatological disease after rheumatoid arthritis and accounts for most cases of dRTA in middle-aged females.

In patient 3, severe hypokalemia, non-anion gap metabolic acidosis, alkaline urine pH, urinary potassium loss, and nephrolithiasis suggested the diagnosis of distal renal tubular acidosis. While the patient was being evaluated for the etiology of dRTA, she presented with bloody diarrhea. A colonoscopy was done which showed features of Crohn's colitis (ileocecal disease). Diagnosis of dRTA secondary to Crohn's colitis was made.

Kidney and lower genitourinary involvement have been reported in 4–23% of patients with IBD manifesting primarily as urinary calculi, fistulas, and kidney tubular damage [13, 14]. Granulomatous interstitial nephritis, interstitial nephritis with hyperoxaluria, and renal tubular acidosis have also been reported [15]. Severe osteomalacia and renal tubular acidosis in Crohn's disease have been reported by Rui MM et al. [16]. A renal biopsy study done by Josephine M.et al. [17] of patients with IBD showed interstitial nephritis and tubular injury in 19% and 8% of cases respectively. Sulfasalazine is also known to cause renal tubulointerstitial damage, however, in the present case patient manifested with renal tubular acidosis before a diagnosis of Crohn's disease was made, indicating that dRTA in the present case is the extraintestinal manifestation of Crohn's disease. Renal tubular acidosis as a manifestation of Crohn's disease is very rare and merits evaluation for mesalamine-induced tubulointerstitial damage.

Conclusions

Hypokalemia is frequently encountered in clinical practice. Hypokalemia can be a manifestation of systemic illness and should always be evaluated. Clinical history, physical examination with particular emphasis on volume, and assessment of acid–base status allow the cause of hypokalemia to be readily determined in most cases. Hypokalemic paralysis as a manifestation of primary hyperaldosteronism, Sjogren's syndrome, and Crohn's disease are extremely rare.

Abbreviations

RTA	Renal tubular acidosis
dRTA	Distal renal tubular acidosis
PAC	Plasma aldosterone concentration
KUB	Kidney and urinary bladder

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Authors' contributions

RS, NAB, FM, and PT contributed to the data collection. RS, NAB, FM, PT, and NJ participated in the writing of the manuscript. RS and IW participated in the critical review. RS, NAB, FM, PT. NJ and IW provided approval for the final manuscript. All authors read and approved the final manuscript.

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Availability of data and materials

Data and materials will be available on request.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

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Competing interests

The authors declare that they have no competing interests.

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