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Case Report

Hypokalemic periodic paralysis as an atypical presentation of primary hyperparathyroidism: a case report

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ABSTRACT

Excess production of parathyroid hormone from a parathyroid adenoma or hyperplasia or carcinoma can lead to hypercalcemia. It produces a wide range of clinical manifestations ranging from abdominal pain to renal calculi. Other electrolyte abnormalities such as hypomagnesemia and hypokalemia are also seen. Complications due to hypokalemia primarily involves the cardiovascular system. Hypokalemic periodic paralysis is an unusual presentation of primary hyperparathyroidism. It presents with acute onset episodes of flaccid paralysis which usually improves following potassium supplementation. We report the case of a 37-year-old male presenting with acute weakness of the extremities with laboratory investigations suggestive of hypercalcemia, hypokalemia, normal anion gaps metabolic acidosis and alkaline urine suggestive of renal tubular acidosis. On further evaluation, the patient was identified to have a right parathyroid adenoma which was managed through surgical excision. Evaluation of secondary endocrine disorders that can lead to hypokalemia is important for early recognition and appropriate management.

Keywords: Hypokalemia, Parathyroid adenoma, AV block, Distal RTA, Hypokalemic periodic paralysis

INTRODUCTION

Primary hyperparathyroidism is one of the most common causes of hypercalcemia and results from the overproduction of parathyroid hormone. The underlying pathology in the majority of cases is due to an adenoma (80% to 90%) followed by hyperplasia of all four glands (10 to 15%).[1] Excess parathyroid hormone disrupts calcium and phosphorus metabolism producing a broad spectrum of manifestations ranging from asymptomatic hypercalcemia to osteoporosis, nephrocalcinosis and other manifestations of hypercalcemia. Hypercalcemia can disrupt alpha intercalated cells of the collecting duct leading to metabolic acidosis and hypokalemia.[2] A rare disorder resulting from severe hypokalemia is hypokalemic periodic paralysis which is characterized by recurrent episodes of acute onset flaccid paralysis. While most cases are associated with mutations in skeletal muscle sodium and calcium channels and thyrotoxicosis, hypokalemic periodic paralysis as a feature of primary hyperparathyroidism is unusual.[3] Presence of multiple clinical manifestations due to hypercalcemia and hypokalemia can make the diagnosis challenging and requires multiple laboratory investigations for prompt evaluation and treatment.

CASE REPORT

A 37-year-old male presented with complaints of recurrent episodes of palpitations for the past 3 months which were sudden in onset and had a regular rhythm. He also complained of weakness of both the lower limbs for the past 4 days. Other presenting complaints included exertional breathlessness with New York Heart

Association (NYHA) class II grading and intermittent episodes of giddiness. He had no history of syncope, orthopnea, paroxysmal nocturnal dyspnea, fever, chest pain, or recent upper respiratory tract infection.

The patient was a known case of hypokalemic periodic paralysis for the past 5 years with multiple previous hospital admissions, the latest in January 2025, improved following potassium supplementation. Muscle strength was normal between the episodes.

He has a history of bilateral nephrocalcinosis with hydroureteronephrosis, and type 1 renal tubular acidosis diagnosed during previous evaluations for recurrent hypokalemia. He had also experienced recurrent episodes of polyarthritis and cystitis.

He has no previous history of hypertension, diabetes mellitus, coronary artery disease, chronic obstructive pulmonary disease, epilepsy, or thyroid disease. There was no family history of similar paralytic episodes, endocrine disorders, or kidney disease. He was a known alcoholic but had no history of smoking or use of illicit drugs.

On examination, the patient was conscious, alert, and oriented. He was afebrile and appeared moderately built and nourished. His vitals were stable. General examination revealed mild left-sided pitting pedal edema. There was no pallor, icterus, cyanosis, clubbing, generalized lymphadenopathy, or signs of dehydration.

Cardiovascular examination revealed normal first and second heart sounds without any murmurs. Respiratory system examination demonstrated bilateral equal air entry with normal vesicular breath sounds and no added sounds. Abdominal examination revealed a soft, non-tender abdomen with normal bowel sounds and no organomegaly. Neurological examination showed no focal neurological deficits. Sensory examination was within normal limits.

For further evaluation of palpitations, electrocardiography was performed. The initial ECG demonstrated sinus rhythm with prolonged PR interval followed by a dropped QRS complex consistent with a second degree atrioventricular block and diffuse ST-segment depression involving the inferior and lateral leads, findings suggestive of hypokalemia (Figure 1).

Transthoracic echocardiography revealed structurally normal cardiac chambers with preserved left ventricular systolic function and an ejection fraction of 60%. There were no regional wall motion abnormalities, significant valvular lesions, pulmonary hypertension, or pericardial effusion. On complete blood count, anemia and leukocytosis were present.

Biochemical investigations are revealed in Table 1.

On parathyroid scintigraphy, an ovoid area of intense tracer uptake was seen in the parathyroid gland located along the medial aspect of the lower pole of the right thyroid gland indicative of a parathyroid adenoma (Figure 2).

Table 1: Biochemical investigations of serum levels.

Variables	Patient	Normal range
Urea (mg/dl)	98	15.0-40.0
Creatinine (mg/dl)	2.5	0.3-1.3
Potassium (meq/l)	2.1	3.5-5.0
Sodium (meq/l)	130.0	136.0-146.0
Chloride (meq/l)	102.4	98.0-110.0
PTH (pg/ml)	548.6	16-65
Calcium (mg/dl)	12.8	8.6-10

Following nephrology evaluation USG-KUB, urinalysis, renal function tests and arterial blood gas analysis (ABG) were done. Imaging of the urinary tract showed multiple hyperechoic calcific foci in bilateral medullary parenchyma with distension of urinary bladder. A final impression of bilateral nephrocalcinosis with grade 1 hydroureteronephrosis, consistent with chronic hypercalcemia-related renal involvement was seen. ABG demonstrated metabolic acidosis with a calculated anion gap of 9.6 mEq/l, consistent with normal anion gap metabolic acidosis (Table 2).

Table 2: Arterial blood gas analysis.

Variables	Patient	Reference range
pH	7.29	7.350-7.450
PCO_2 (mmHg)	24.8	32.0-48.0
PO_2 (mmHg)	54.3	83.0-108.0
HCO_3^- (mmol/l)	20	22.0-26.0

A urine protein to creatinine ratio (UPCR) test was done in which urine microprotein and urine protein to creatinine ratio was significantly elevated suggestive of renal disease (Table 3).

Table 3: Urine protein to creatinine ratio.

Variables	Patient	Reference range
Urine micro protein (mg/dl)	59.7	<15-20
Urine creatinine (mg/dl)	45.3	20-320
Urine protein to creatinine ratio	1.31	0.15-0.20

Urinalysis was normal except for urine pH which was 7.5. Levels of urine electrolytes was within the normal range. A persistently alkaline urine raises suspicion for distal renal tubular acidosis. Thyroid function tests were normal (Table 4).

The patient underwent ophthalmological evaluation for complaints of irritation in both eyes. On inspection the

conjunctiva was muddy in appearance. He was diagnosed with dry eye disease using Schirmer test.

Table 4: Thyroid function tests.

Variables	Patient	Reference range
Free T3 (Pmol/l)	6.8	3.1-6.8
Free T4 (ng/dl)	1.23	0.93-1.7
TSH (µIU/ml)	1.7	0.3-4.4

The recurrent episodes of hypokalemic periodic paralysis, nephrocalcinosis, renal tubular acidosis, hypercalcemia, and elevated parathyroid hormone levels led to the diagnosis of primary hyperparathyroidism presenting as recurrent hypokalemic periodic paralysis.

The patient was admitted for cardiac monitoring and correction of electrolyte abnormalities. He received intravenous potassium supplementation, intravenous fluids, and supportive care. Endocrinology and nephrology consultations were obtained for further management of primary hyperparathyroidism and its renal complications. Following correction of serum potassium, his palpitations resolved and electrocardiographic abnormalities improved. He underwent surgical management by excision of the parathyroid adenoma.

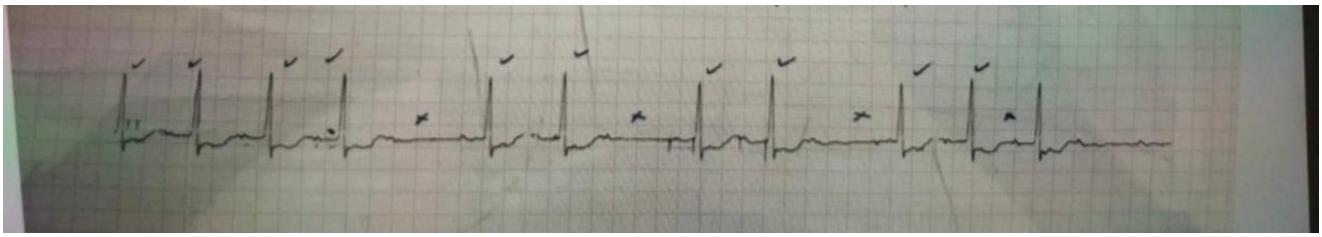

Figure 1: ECG findings of a dropped QRS complex.

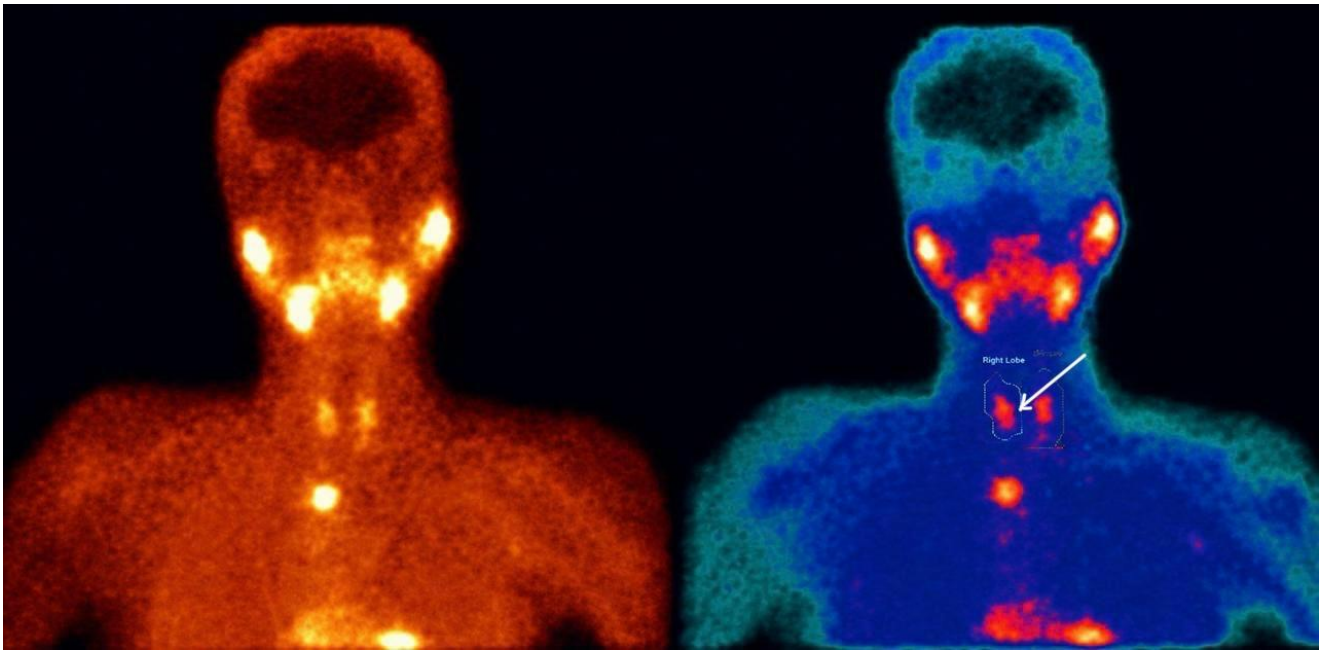

Figure 2: Parathyroid scintigraphy revealing a right parathyroid adenoma.

DISCUSSION

Primary hyperparathyroidism is a common endocrine disorder presenting in 0.1–0.4% of the population and is the most common cause of hypercalcemia. It can either be due to an adenoma, hyperplasia or carcinoma. An adenoma generally involves a single parathyroid gland as seen in the patient. It causes increased secretion of parathyroid hormone (PTH) which is normally involved in calcium and vitamin D metabolism. An increase in PTH results in increased serum calcium levels and can cause symptoms ranging from asymptomatic presentation to skeletal manifestations, fatigue, abdominal pain, renal calculi, and neuropsychiatric symptoms, the classic pentad (bones, stones, abdominal groans, psychic moans and fatigue overtones).

Although electrolyte abnormalities like hypokalemia, hypercalcemia and hypomagnesemia are common in hyperparathyroidism, hypokalemic periodic paralysis is an unusual presentation of primary hyperparathyroidism as stated by Hussain et al in their report of two patients diagnosed with hypokalemic periodic paralysis in the

setting of primary hyperparathyroidism.[3] Unlike hyperparathyroidism, the association of hypokalemic periodic paralysis with thyroid disorders is well established. Soule et al described hypokalemic periodic paralysis in a patient with Graves' disease. This form of hypokalemic periodic paralysis occurring in the setting of hyperthyroidism is called thyrotoxic periodic paralysis. It is the most common form of hypokalemic periodic paralysis and is seen primarily in Asian males. The clinical features are similar to those seen with other forms of periodic paralysis, but also include the symptoms of thyrotoxicosis such as weight loss, tachycardia, and anxiety. Any cause of hyperthyroidism can be associated with it but Grave's disease is the most common.[4]

Apart from endocrine triggers, hypokalemic periodic paralysis also exists as a channelopathy. Paydas et al in 2025 reported a case of a patient diagnosed with an inherited form of hypokalemic periodic paralysis. It is associated with mutations affecting the sodium and calcium channels present in the skeletal muscle leading to disruption of the electrical activity of the muscle fibers and absence of muscle stimulation causing flaccid paralysis. Arrhythmias, including ventricular tachycardia/fibrillation and atrioventricular block may develop due to hypokalemia and can be life-threatening.[5]

The findings of hypokalemia, normal anion gap metabolic acidosis and alkaline urine is significant for distal renal tubular acidosis where there is damage to the alpha intercalated cells of the collecting duct causing failure to secrete H^+ ions into the tubular fluid. To maintain electrical neutrality potassium gets secreted into the tubular fluid by principal cells causing hypokalemia. Excess parathyroid hormone can cause proximal renal tubular acidosis by inhibition of bicarbonate reabsorption in the proximal convoluted tubule. However, distal renal tubular acidosis due to primary hyperparathyroidism similar to our patient is rarely reported. In 2015 Lo et al described a 26-year-old man with a 3-year history of recurrent episodes of dysuria, minimal hematuria and bilateral flank pain. He had proximal muscle weakness suggestive of hypokalemic periodic paralysis and hypercalcemia attributable to primary hyperparathyroidism from a hyper functioning parathyroid adenoma. Hypokalemia, alkaline urine pH and inability to acidify urine despite acidosis led to the diagnosis of concomitant distal RTA in a patient with primary hyperparathyroidism.[6]

A common disorder which presents with acute onset flaccid paralysis similar to hypokalemic periodic paralysis is Gullain Barre syndrome (GBS). Patients with hypokalemic periodic paralysis respond quickly (hours to days) to potassium supplementation, whereas patients with GBS will take weeks to months to recover. Hence distinguishing both the presentations is important for appropriate treatment. A history of diarrhoea is suggestive of GBS. Hati et al in 2022 reported a 20-year-old woman with complaints of rapidly progressive acute onset quadriparesis and laboratory investigations revealing hypokalemia similar to hypokalemic periodic paralysis. However, a history of gastrointestinal upset and impaired joint position and vibration sense provided a diagnosis of GBS.[7] Paraesthesia and sensory loss can be present in GBS, whereas sensory symptoms are usually absent in hypokalemic periodic paralysis. Respiratory failure is seen more commonly in GBS. Pattern of muscle weakness is generalised in hypokalemic periodic paralysis in contrast to a distal predominant weakness in GBS.

Another important differential to consider in a patient with periodic paralysis is Andersen-Tawil syndrome. It is caused by a mutation in the KCNJ2 gene, which encodes the inward rectifier potassium (Kir2.1) channel. Typical presentations include periodic paralysis and cardiac manifestations, often accompanied by distinctive facial features and skeletal anomalies which includes low-set ears, micrognathia, widely spaced eyes, clinodactyly of the fifth digit, syndactyly, scoliosis, and short stature. Symptoms typically manifest early in life, usually in the first or second decade, with patients experiencing either cardiac symptoms or muscle weakness. Serum potassium levels may be elevated, normal, or low. ECG findings in Andersen-Tawil syndrome typically include a prolonged QT interval, prominent U waves, ectopic beats, and episodes of ventricular tachycardia. Exercise testing on electromyography is similar to hypokalemic periodic paralysis. Genetic testing remains crucial for confirming the diagnosis of Andersen-Tawil syndrome.[8]

There is ongoing research about the familial form of hypokalemic periodic paralysis. The two causative genes identified to date are CACNA1S, which encodes the voltage-gated Ca2+channel associated with type1 and SCN4A, which codes the voltage-gated Na+ channel associated with type 2. For years, it was unclear how mutations in these two distinct genes yielded an identical disease phenotype. Recent studies point to a mechanism in which mutations of arginine residues within the S4 voltage-sensing domain destroy the hydrophobic seal between the external fluid and the internal cytosolic crevices, thus generating an aberrant ionic leak, i.e. the gating pore current. Consistently, of twenty-two CACNA1S and SCN4A variants identified to date, fourteen have experimentally been demonstrated to exhibit gating pore currents.[9]

Samson et al conducted a randomized double blinded placebo controlled trial of dichlorphenamide in hypokalemic periodic paralysis. The majority of participants on DCP in this trial experienced less than 1 attack per week compared to more than 2 attacks per week on placebo. This trial strengthens the evidence for the beneficial effect of DCP in Hypokalemic periodic paralysis.[10] Initially acetozolamide was used in the treatment of hypokalemic periodic paralysis. But a study done in 2011 by Mathew et al showed only 54% of patients with a CACNA1S mutation reported a beneficial response compared with only 18% of those with an SCN4A mutation.[11] However correction of underlying

hypokalemia using potassium supplementation is required. Definitive treatment involves surgical excision of the parathyroid adenoma.

CONCLUSION

This case contains multiple unusual presentations like distal renal tubular acidosis in a patient with primary hyperparathyroidism and resulting hypokalemia which causes hypokalemic periodic paralysis. Presence of multiple other manifestations like nephrocalcinosis and AV block can make the diagnosis challenging. Reporting such cases is important to strengthen the evidence regarding such atypical presentations which can aid in future research.

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