Case Report

Hypocalcemia in a Young Adult: A Case of Idiopathic Hypoparathyroidism with Concurrent Vitamin-D Deficiencyn

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ABSTRACT

Introduction: Hypocalcemia is a common electrolyte disturbance with diverse etiologies, including hypoparathyroidism and vitamin D deficiency. Idiopathic hypoparathyroidism, a rare nonsurgical cause, poses diagnostic and therapeutic challenges.

Case Presentation: A 22 year-old male with no known comorbidities presented with a history of diarrhoea and occasional muscle twitching for the last 3 months. He reported a weight loss of 10 kg over a two-month period. These complaints aggravated in the last few days, which brought him under emergency care. Initial investigations revealed severe hypocalcemia (serum calcium: 5.3 mg/dL), inappropriately low parathyroid hormone (PTH: 13 pg/mL), and low activated vitamin D (1, 25-dihydroxy vitamin D: 5.96 pg/mL), suggestive of hypoparathyroidism. Intravenous calcium infusion led to symptomatic improvement. Despite extensive gastrointestinal and metabolic evaluations, no underlying cause was identified, leading to a diagnosis of idiopathic hypoparathyroidism. The patient improved with oral calcium, calcitriol, and supportive management.

Conclusion: This case underscores the importance of recognizing hypocalcemia as a potential indicator of nonsurgical hypoparathyroidism and highlights the need for a systematic diagnostic and therapeutic approach for optimal long term management.

KEY WORDS: Idiopathic hypoparathyroidism, Hypocalcemia, Vitamin D deficiency, Calcium-phosphorus imbalance, Calcitriol therapy.

INTRODUCTION

Hypocalcemia is a frequently encountered electrolyte imbalance that can affect various organ systems, manifesting with cardiovascular, neuromuscular, and skeletal symptoms. Common etiologies include hypoparathyroidism, vitamin D deficiency, chronic kidney disease, and malabsorption syndromes. Hypoparathyroidism, characterised by low serum calcium with normal or inappropriately low PTH levels, is a common cause of hypocalcemia.[1] PTH plays a crucial role in calcium and phosphate homeostasis through various mechanisms.[2] While most hypoparathyroidism cases are postsurgical, approximately 25% are nonsurgical in origin.[1] Among these, autoimmune hypoparathyroidism is the most frequent and may occur in isolation or as part of autoimmune polyendocrine syndrome.[3] If exhaustive

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Access this Article Online

URL:
https://jpes.org.pk/index.php/jpes/article/view/43

Submitted: May 06, 2025 **Revision Received:** June 27, 2025

Accepted for Publication: December 31, 2025

How to cite this: *Aslam F, Fatima N, Kiran Z, Banu S. Hypocalcemia in a Young Adult: A Case of Idiopathic Hypoparathyroidism with Concurrent Vitamin-D Deficiency. JPES. 2025;2(2):80-84.*

clinical and laboratory investigations fail to identify a cause, a diagnosis of idiopathic hypoparathyroidism is made. Genetic testing should be considered in young patients or those with a family history.[4] We present a case of a young male with persistent hypocalcemia, weight loss, and gastrointestinal symptoms and discuss the diagnostic challenges and management strategies.

CASE PRESENTATION

A 22 year old man presented to the emergency department of Dow University Hospital, Karachi, with complaints of loose stools and occasional muscle twitching of the face for three months and significant weight loss of 10 kg over two months. These symptoms aggravated in the last 3 days, which brought him to medical attention and urged an emergency visit where hypocalcemia was identified as the cause of muscle twitching (5.3 mg/dl), and intravenous calcium infusion was given for symptomatic improvement. He had no history of neck surgery, familial endocrine disorders, joint pain, oral ulcers, skin rash or lower back pain. His family history was also unremarkable for such an illness. His past medication history included long-term use of proton pump inhibitors.

On examination, the patient had stable vital signs with mild dehydration, positive Chvostek's sign, muscle twitching, increased deep tendon reflexes, and mild

Table-I: hematological and biochemical profile.

CBC	*Results*	*Reference Range*
Hemoglobin	13.1 g/dl	(12-16)
White blood cells	8.2 x 109	5-10*109
Platelets	196 x 109	(150-450*109)
ESR	18	<20
Liver function tests		
SGPT	92 U/l	(<45)
Total bilirubin	0.87	(0.2-1.2)
SGOT	77 U/l	(<35)
Alkaline phosphatase	131 U/l	(53-128)
Renal Profile		
Urea	10.7 mg/dl	(12-41)
Serum Creatinine	0.81 mg/dl	(0.9-1.3)
Serum albumin	4.6 g/dl	(3.5-5)
25-hydroxy vitamin D	8.39 ng/ml	(>30)
1,25-dihydroxy vitamin D	5.96 pg/ml	(19.9-79)
Serum PTH	13.8 pg/ml	(15-65)
Electrolytes		
Serum Calcium	5.3 mg/dl	(8.6-10.2)
Serum Phosphorus	6.6 mg/dl	(2.4-4.5)
Serum Potassium	3.9 meq/L	(3.5-5)
Serum Sodium	145 meq/L	(135-145)
Serum Chloride	106 meq/L	(97-107)
Bicarbonate	23 meq/L	(22-29)
Serum Magnesium	1.72 mg/dl	(1.7-2.3)
Random blood glucose(RBS)	130mg/dl	<200mg/dl

Table-II: Other Investigations.

Thyroid stimulating hormone(TSH)	1.2 mIU/L	(0.4-4)
Serum lipase	35 U/L	(<60)
Serum amylase	101 U/L	(28-100)
Serum iron	59 ug/dl	(40-60)
Total iron binding capacity(TIBC)	228 ug/dl	(100-400)
Serum ferritin	142 ng/ml	(20-250)
Anti -TTG Ig G	1.53 AM/ml	(<20 AM/ml)
Anti-TTG Ig A	4.2 AU/ ml	<20 AU/ ml
Urinary copper	33.9 ug/day	(<60)
Serum ceruloplasmin	0.22 G/L	(0.2-0.6)
Spot urinary calcium	<0.8	
Spot urinary phosphorus	< 3.4	
urine analysis	Normal	
ANA (Anti-nuclear antibody)	Negative	
Anti-gastric parietal cell antibodies	Negative	

epigastric tenderness were noted. An ophthalmologic examination revealed a Kayser-Fleischer ring, although further investigations for Wilson's disease were negative. Laboratory investigations for weight loss and chronic diarrhoea included screening for diabetes, tuberculosis and celiac disease. The investigations did not reveal any positive findings. The clinical evaluation of the other systems did not reveal any notable findings. An electrocardiogram did not reveal any abnormalities upon admission.

Hematological investigations like complete blood picture (CP) did not show any abnormality. Biochemical investigations revealed low calcium, low PTH, and raised phosphate levels along with low 25-hydroxy vitamin D and 1, 25 dihydroxy vitamin D levels. The rest of the serum electrolytes, including sodium, potassium, chloride, and magnesium, were within normal ranges. Urinary calcium was low, which indicates no urinary loss of calcium. Further workup included Erythrocyte sedimentation rate (ESR), stool studies (stool microscopy and culture), Thyroid stimulating hormone (TSH), Monteux test, Anti-nuclear antibody profile (ANA), celiac serology (Anti TTG IgG and IgA), and workup for Wilson's disease, all of which were unremarkable. Screening chest x-ray did not show any suspicion of pulmonary pathology. Table-I and II represent the investigations profile of the patient.

The CT scan of the abdomen was unremarkable. Upper GI endoscopy revealed pan gastritis with H. pylori, confirmed by gastric biopsy. Colonoscopy showed no abnormalities.

A diagnosis of idiopathic hypoparathyroidism with concurrent vitamin D deficiency was made. Intravenous calcium infusion provided initial symptom relief, and the patient was transitioned to oral calcium (600 mg), vitamin D3 (cholecalciferol, 125 IU) and calcitriol (0.5 mcg/day). He also received H. pylori eradication therapy and supportive treatments, including probiotics and PPIs.

The patient was followed regularly. Serial laboratory monitoring (as shown in Fig.1) showed gradual biochemical normalization. After six months, serum calcium stabilized between 8.7–9.2 mg/dl and serum phosphorus decreased to 5.2 mg/dl Quality of life improved, and he regained 4 kg of the lost weight in six months.

DISCUSSION

This case highlights a rare presentation of idiopathic hypoparathyroidism with chronic hypocalcemia and vitamin D deficiency in a young adult. Hypoparathyroidism should be suspected in patients with unexplained hypocalcemia accompanied by inappropriately low PTH levels,[5] helping distinguish it from secondary causes of hypocalcemia, which

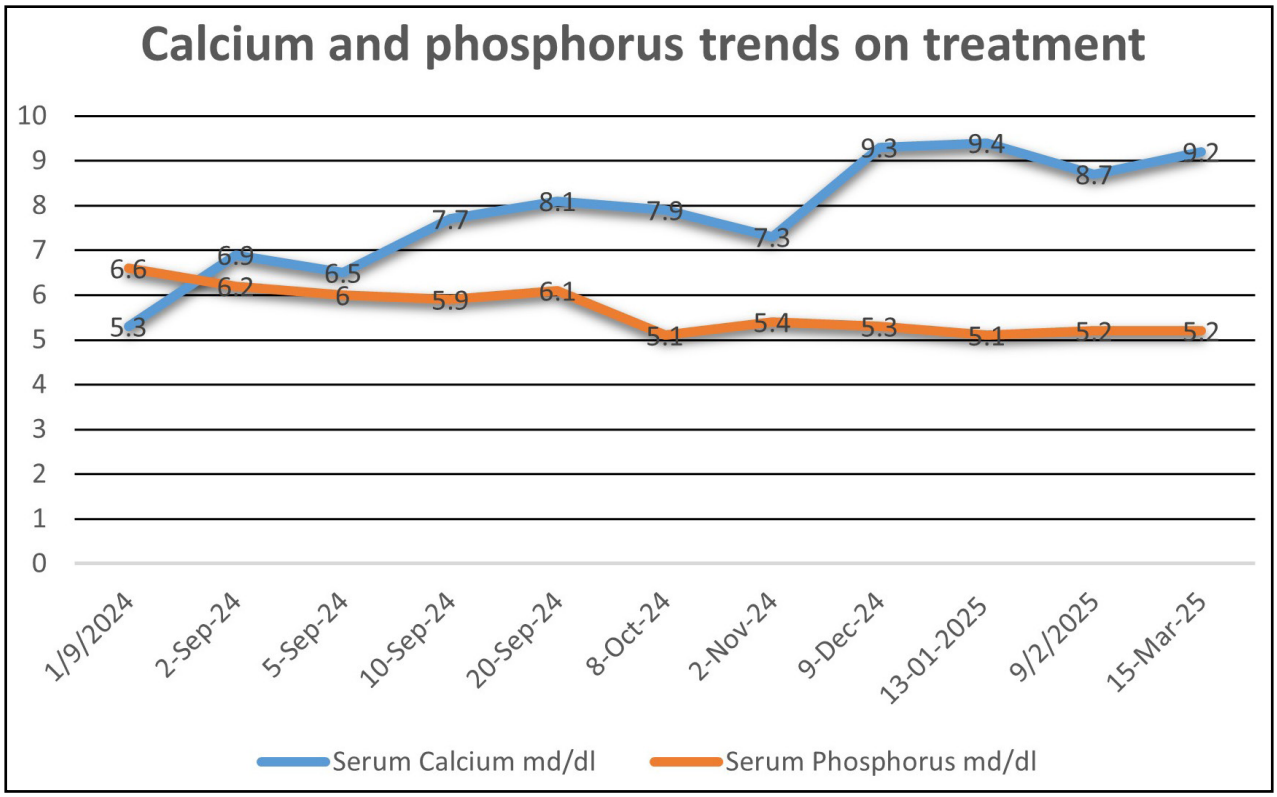

Fig.1: describes the pattern of calcium and phosphorus in the current management, and it clearly indicates the stable levels of these electrolytes over a follow-up period.

typically present with elevated PTH levels.[6] In our case, an extensive workup ruled out common etiologies.

The long-term management of hypocalcemia is crucial to prevent complications. Management goals for chronic hypoparathyroidism include:

- Preventing hypocalcaemia symptoms
- Maintaining calcium levels in the low-normal range
- Keeping the calcium-phosphorus product <55 mg^2/dL^2
- Avoiding hypercalciuria
- Preventing hypercalcemia
- Minimizing renal complications such as nephrocalcinosis or nephrolithiasis.[6]

Conventional therapy with oral calcium and active vitamin D analogues is standard. Chronic hypoparathyroidism is commonly treated with conventional therapy. Limitations include risk of hypercalciuria, fluctuating calcium levels, and long-term renal complications.[1] In refractory or poorly controlled cases, recombinant PTH analogues may be considered.[7] In a case reported in China, the patient was misdiagnosed with myasthenia secondary to muscular weakness and fatigue, which was later found to be due to idiopathic hypoparathyroidism. His symptoms improved after calcium and vitamin D replacement.[8] In another case, two patients with hypoparathyroidism presented with carpopedal spasm, brain calcification and cognitive impairment, and they improved after calcium and vitamin D treatment.[9] Similarly, our patient responded well to standard therapy and maintained stable laboratory values during follow-up. The calcium levels (8.7 mg/dL-9.2 mg/dl) are maintained within the low normal range.

CONCLUSION

Hypocalcemia necessitates a thorough, systematic diagnostic approach. Idiopathic hypoparathyroidism, although rare, should be considered when other causes are excluded. Long-term management involves careful biochemical monitoring to prevent complications associated with the disease and its treatment.

Grant support: Not applicable.

Financial disclosures: None.

IRB approval: Not applicable; informed consent was taken from the patient.

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Author Contributions:

Faiza Aslam: Conception and design of the case report and manuscript drafting.
Dr. Nazish Fatima: Patient management, literature review, and critical revision of the manuscript.
Dr. Zareen Kiran: Literature review and critical review of the manuscript.
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