RESEARCH PAPER

SEVERE PRIMARY HYPOTHYROIDISM PRESENTING AS SUBACUTE INTESTINAL OBSTRUCTION: A RARE REVERSIBLE CAUSE OF INTESTINAL PSEUDO-OBSTRUCTION : A CASE REPORT

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ABSTRACT

Severe hypothyroidism is an uncommon but important cause of gastrointestinal dysmotility and intestinal pseudo-obstruction. The condition may clinically and radiologically mimic mechanical intestinal obstruction, often leading to delayed diagnosis and unnecessary surgical intervention. We report the case of a 42-year-old male who presented with progressive abdominal distension, constipation, colicky abdominal pain, nausea, and recurrent bilious vomiting suggestive of subacute intestinal obstruction. Clinical examination revealed abdominal distension with sluggish bowel sounds. Radiological evaluation demonstrated diffuse bowel dilatation without evidence of a mechanical obstructive lesion. Further evaluation revealed profound primary hypothyroidism with a thyroid-stimulating hormone (TSH) level of 117.2 μIU/mL, markedly reduced triiodothyronine (T3) and thyroxine (T4) levels, and strongly positive anti-thyroid peroxidase antibodies. The patient was managed conservatively with nasogastric decompression, supportive care, and levothyroxine replacement therapy, resulting in complete resolution of symptoms without surgical intervention. This case highlights the importance of considering hypothyroidism in the differential diagnosis of unexplained intestinal obstruction and emphasizes the role of early thyroid function testing in avoiding unnecessary surgery.

Keywords: Hypothyroidism, Intestinal pseudo-obstruction, Myxedema ileus, Hashimoto thyroiditis, Subacute intestinal obstruction, Levothyroxine.

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INTRODUCTION

Hypothyroidism is one of the most common endocrine disorders worldwide and results from inadequate synthesis, secretion, or action of thyroid hormones. The condition affects multiple organ systems because thyroid hormones are essential regulators of cellular metabolism, growth, and physiological homeostasis. While the cardiovascular, neurological, dermatological and metabolic manifestations of hypothyroidism are well recognized, gastrointestinal involvement is often underappreciated. Gastrointestinal symptoms are generally nonspecific and commonly include constipation, abdominal bloating, anorexia, dyspepsia, and delayed gastric emptying. These manifestations are usually

mild and improve with thyroid hormone replacement. However, in severe and longstanding cases, profound gastrointestinal hypomotility may occur, leading to rare but potentially serious complications such as paralytic ileus, megacolon, and intestinal pseudo-obstruction.[1,2]

Thyroid hormones play a crucial role in maintaining normal gastrointestinal motility through their effects on smooth muscle contractility, enteric nervous system activity, autonomic regulation, and intestinal electrolyte transport. Deficiency of thyroid hormones results in reduced peristaltic activity, impaired neuromuscular transmission, and accumulation of mucopolysaccharides within the bowel wall, leading to bowel edema and diminished intestinal propulsion. These pathological changes can culminate in marked intestinal stasis and bowel dilatation, producing clinical and radiological features that closely resemble mechanical intestinal obstruction. This entity has been described in the literature as "myxedema ileus" or hypothyroidism-associated intestinal pseudo-obstruction.[3]

Intestinal pseudo-obstruction is characterized by symptoms and signs of bowel obstruction in the absence of a demonstrable mechanical cause. Patients commonly present with abdominal pain, progressive distension, nausea, vomiting, and severe constipation. Because these symptoms are indistinguishable from those of true intestinal obstruction, diagnosis can be challenging and often requires extensive radiological evaluation to exclude surgically correctable causes. Failure to recognize the underlying endocrine etiology may result in unnecessary surgical exploration, increased morbidity, prolonged hospitalization, and delayed initiation of appropriate medical therapy.[4,5]

Although several isolated case reports have documented hypothyroidism presenting as intestinal pseudo-obstruction, the condition remains exceedingly rare and is frequently overlooked in routine surgical practice. Recognition of accompanying systemic features of hypothyroidism—including fatigue, lethargy, weight gain, cold intolerance, facial puffiness, dry skin, bradycardia, and hoarseness of voice—can provide important diagnostic clues. Early thyroid function testing in patients with unexplained bowel obstruction may facilitate timely diagnosis and prevent unwarranted operative intervention.[6,7]

CASE PRESENTATION

A 42-year-old male presented to the Emergency Department with complaints of progressively increasing abdominal distension, intermittent colicky abdominal pain, constipation, nausea, and multiple episodes of bilious vomiting for the preceding ten days. The abdominal pain was diffuse, moderate in intensity, non-radiating, and occurred in intermittent spasmodic episodes. The vomiting was bilious in nature, occurred several times daily, and was associated with temporary relief of abdominal discomfort. The patient reported absolute constipation for the previous three days, with failure to pass stools and flatus. The symptoms had developed insidiously and gradually worsened, significantly affecting his oral intake and daily activities.

On detailed inquiry, the patient described a history of generalized weakness, easy fatigability, lethargy, excessive daytime sleepiness, facial puffiness, weight gain, cold intolerance, and chronic constipation for approximately six months prior to presentation. He also complained of reduced physical activity, decreased appetite, and a feeling of generalized body swelling during the same period. There was no history of fever, hematemesis, melena, hematochezia, chronic diarrhea, jaundice, urinary complaints, or significant weight loss suggestive of malignancy. There was no

previous history of abdominal surgery, tuberculosis, inflammatory bowel disease, hernia, abdominal malignancy, diabetes mellitus, connective tissue disorders, or similar episodes of intestinal obstruction. There was no significant family history of thyroid disease or gastrointestinal disorders.

On admission, the patient appeared ill-looking, lethargic, and exhibited features suggestive of hypothyroidism. He had coarse facial features, periorbital edema, facial puffiness, dry skin, and a hoarse quality of voice. Vital signs revealed a pulse rate of 58 beats per minute, regular in rhythm, blood pressure of 130/80 mmHg, respiratory rate of 18 breaths per minute, and oxygen saturation of 98% on room air. He was afebrile and hemodynamically stable. General physical examination demonstrated mild pallor and non-pitting edema of the lower limbs. There was no icterus, cyanosis, clubbing, generalized lymphadenopathy, or signs of dehydration.

Abdominal examination revealed generalized abdominal distension, more pronounced in the central abdomen. The abdomen moved normally with respiration. On palpation, there was mild diffuse tenderness without guarding, rigidity, rebound tenderness, or localized peritonism. No palpable abdominal mass or organomegaly was identified. Hernial orifices were normal, and there was no evidence of an external hernia. Percussion demonstrated a predominantly tympanitic note over the distended bowel loops. Auscultation revealed markedly sluggish and infrequent bowel sounds, suggestive of severe intestinal hypomotility. Digital rectal examination revealed an empty rectum with normal sphincter tone and no palpable intraluminal pathology.

Based on the clinical presentation, a provisional diagnosis of subacute intestinal obstruction was considered. However, the presence of systemic features including bradycardia, facial puffiness, lethargy, weight gain, chronic constipation, and cold intolerance raised suspicion of an underlying endocrine disorder, particularly severe hypothyroidism, contributing to the patient's gastrointestinal manifestations. The patient was therefore subjected to further laboratory and radiological evaluation to differentiate between mechanical intestinal obstruction and intestinal pseudo-obstruction secondary to hypothyroidism.

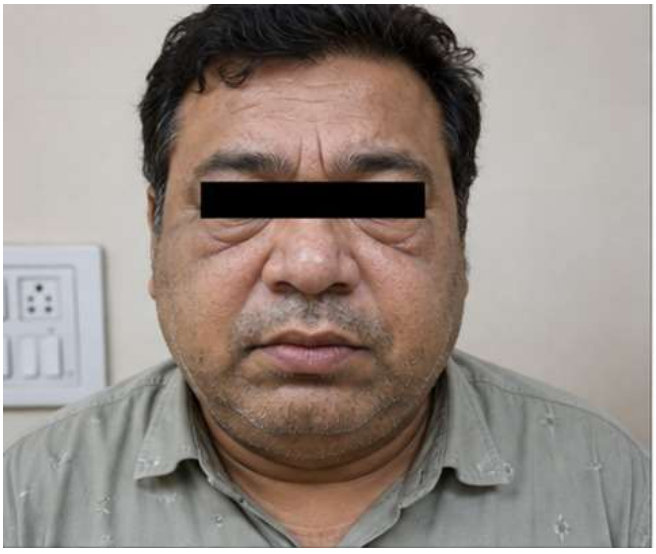

Figure 1: Clinical photograph showing facial puffiness and coarse facial features suggestive of severe hypothyroidism.

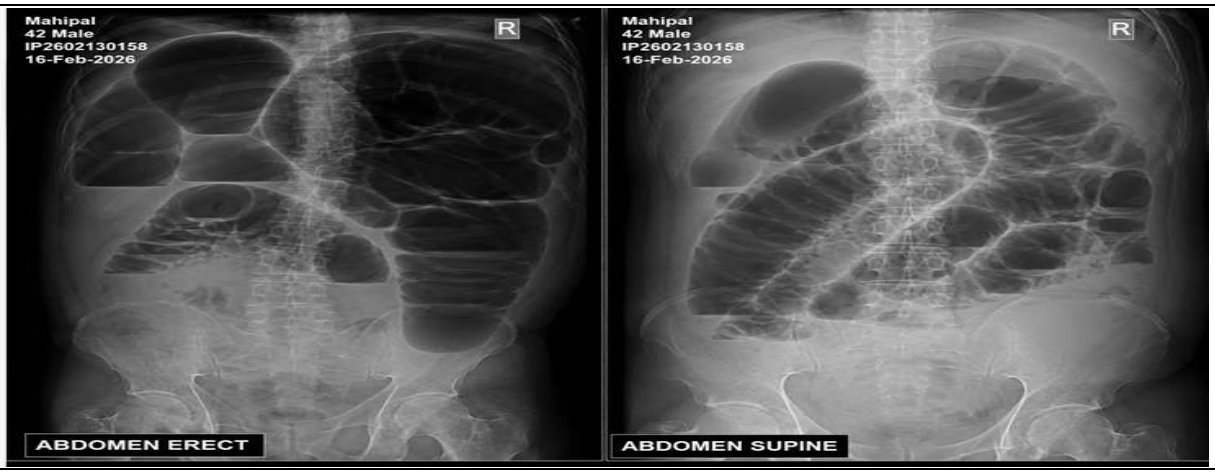

Figure 2: Plain abdominal radiograph demonstrating multiple dilated bowel loops with air-fluid levels.

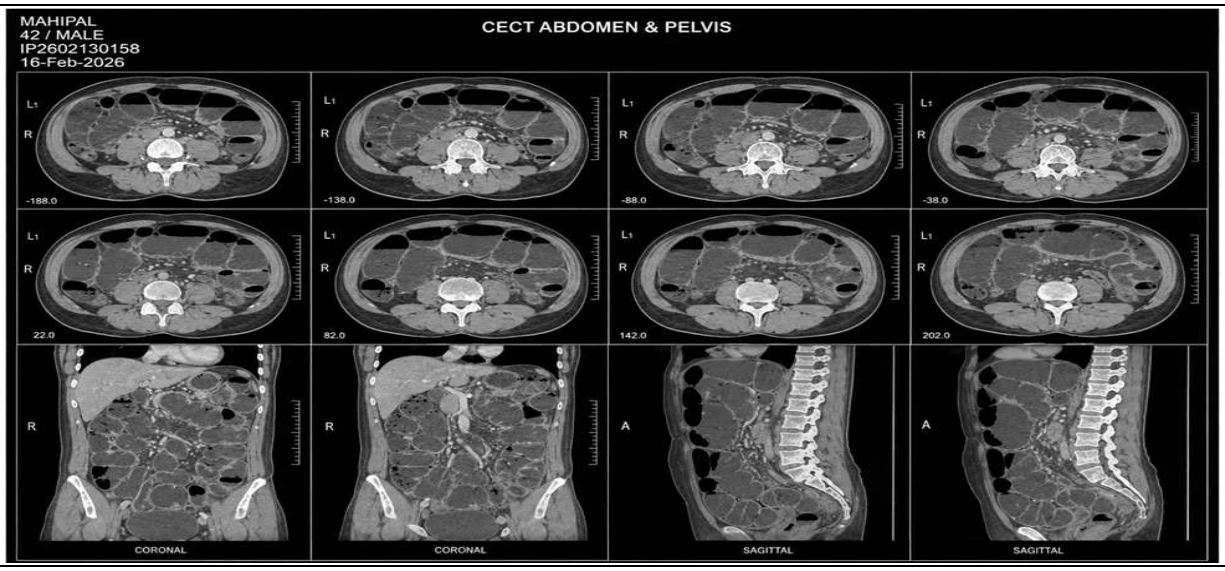

Figure 3: Axial contrast-enhanced CT image showing diffuse bowel dilatation without a transition point

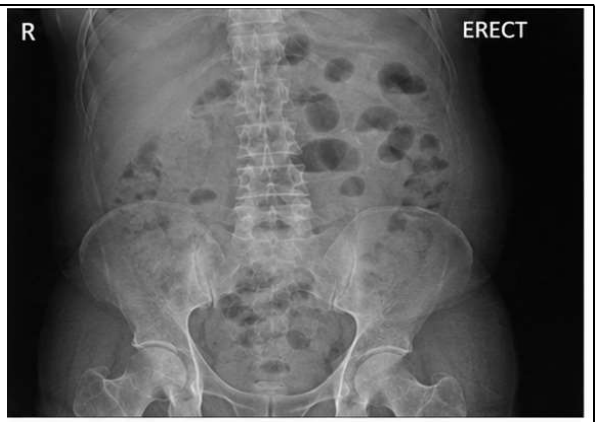

Figure 4: Follow-up abdominal radiograph demonstrating resolution of bowel dilatation after levothyroxine therapy.

INVESTIGATIONS

Laboratory investigations demonstrated mild anemia, leukocytosis, elevated erythrocyte sedimentation rate, and hypoalbuminemia. Thyroid function tests revealed severe primary hypothyroidism with a TSH level of 117.2 µIU/mL, T3 level below 0.195 ng/mL, and T4 level of 0.483 µg/dL. Anti-thyroid peroxidase antibody levels were greater than 500 IU/mL, confirming autoimmune thyroiditis.

Table 1. Laboratory Investigations

Parameter	Value
Hemoglobin	11.3 g/dL
Total Leukocyte Count	11,690/mm³
Platelet Count	5.5×10^5/mm³
ESR	50 mm/hour
Blood Urea	56.5 mg/dL
Serum Creatinine	1.17 mg/dL
Serum Sodium	139 mEq/L
Serum Potassium	3.6 mEq/L
Serum Albumin	2.72 g/dL
T3	<0.195 ng/mL
T4	0.483 µg/dL
TSH	117.2 µIU/mL
Anti-TPO Antibody	>500 IU/mL

Plain abdominal radiography revealed multiple dilated bowel loops with air-fluid levels suggestive of intestinal obstruction. Contrast-enhanced computed tomography (CECT) of the abdomen and pelvis demonstrated diffuse dilatation of both small and large bowel loops without evidence of a transition point, obstructing lesion, volvulus, stricture, internal hernia, or intra-abdominal mass. No bowel ischemia, perforation, or free intraperitoneal air was identified. The radiological findings were consistent with intestinal pseudo-obstruction rather than true mechanical obstruction.

MANAGEMENT

The patient was managed conservatively with bowel rest, nasogastric decompression, intravenous fluids, correction of electrolyte abnormalities, nutritional support, and levothyroxine replacement therapy. Continuous clinical monitoring was performed with serial abdominal examinations and assessment of nasogastric output. Surgical intervention was deferred because imaging failed to demonstrate any mechanical cause of obstruction and the clinical picture strongly suggested hypothyroidism-induced intestinal dysmotility.

Over the subsequent days, abdominal distension gradually decreased, bowel sounds improved, and nasogastric aspirates diminished significantly. The patient subsequently passed flatus and stools, indicating restoration of bowel function. Oral feeding was reintroduced gradually and was tolerated well.

FOLLOW-UP

The patient's clinical condition improved remarkably following initiation of thyroid hormone replacement. By the end of the first week, abdominal symptoms had completely resolved, and normal bowel habits were restored. He was discharged on oral levothyroxine therapy with endocrinology follow-up. At subsequent review visits, he remained asymptomatic with no recurrence of intestinal obstruction and significant improvement in constitutional symptoms including fatigue, facial puffiness, and cold intolerance.

DISCUSSION

Hypothyroidism-induced intestinal pseudo-obstruction is an uncommon manifestation of severe thyroid hormone deficiency. The pathophysiology is multifactorial and involves impaired smooth muscle contractility, autonomic neuropathy, altered intestinal electrolyte transport, and myxedematous infiltration of the bowel wall. These mechanisms result in profound gastrointestinal hypomotility, which may manifest as severe constipation, paralytic ileus, megacolon, or intestinal pseudo-obstruction.[8,9]

The diagnosis can be challenging because patients frequently present with symptoms and radiological findings suggestive of mechanical bowel obstruction. Recognition of associated systemic features such as fatigue, bradycardia, facial puffiness, weight gain, dry skin, and cold intolerance is crucial. In the present case, the absence of a transition point on CT imaging and the

markedly abnormal thyroid profile facilitated the diagnosis.

Several case reports have documented successful resolution of intestinal pseudo-obstruction following thyroid hormone replacement therapy.[10,11] Similar to previous reports, our patient showed dramatic clinical improvement after initiation of levothyroxine, thereby avoiding unnecessary surgical exploration.[12] This case underscores the importance of considering thyroid dysfunction in patients with unexplained intestinal obstruction, particularly when imaging studies fail to reveal a mechanical cause.

CONCLUSION

Severe primary hypothyroidism should be recognized as a rare but reversible cause of subacute intestinal obstruction and intestinal pseudo-obstruction. Early consideration of thyroid dysfunction in patients presenting with unexplained bowel obstruction can facilitate prompt diagnosis and appropriate medical management. Timely initiation of thyroid hormone replacement therapy can result in complete recovery and prevent unnecessary surgical intervention.

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