Intestinalpseudo-obstruction as the First Manifestationof Adrenal Insufficiency: A Case Report and Review of the Literature

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ABSTRACT

Adrenal insufficiency (AI) can be presented with gastrointestinal symptoms such as nausea, vomiting, and abdominal pain. Wereport a case of acute AI presented as intestinalpseudo-obstruction. A 27-year-old man without any remarkable medical history and underlying diseases presented withnausea, vomiting, abdominal pain, anorexia, weight loss, and constipation since 2 months earlier. In studies performed on the patient, a diagnosis of small bowel obstruction with electrolyte disturbance was made. Finally, acute AI wasdiagnosed by small bowel obstruction, which was resolved by corticosteroid treatment.

Keywords: Intestinalpseudo-obstruction, Adrenal insufficiency, Case report

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INTRODUCTION

Adrenal insufficiency (AI)has non-specific symptoms, such as gastrointestinal symptoms (abdominalpain, nausea, and vomiting), and neuropsychiatric symptoms (weakness, mood depression, and disorientation). The presenting symptoms result in long delays in diagnosis and may increase the risk of complications.Gastrointestinal disturbances were seen in more than90% of the patients. These symptoms also overlap considerablywith those of bowel obstruction. Intestinal obstruction in adrenal insufficiency has been seen in reviewing literature in oncology patients and critically ill preterm infants. We report a case of acute adrenal insufficiency manifesting as intestinalpseudo-obstruction in a patient without underlying disease.

CASE REPORT:

A 27-year-old man without any remarkable past medical history and underlying diseases presented with a history of gastrointestinal (GI) complaints such as nausea, vomiting, abdominal pain, anorexia, weight loss, and constipation that had been going for two months. The abdominal pain was a colicky pain that was not alleviated by changing the patient's position and was accompanied by other GI symptoms such as vomiting, nausea, and anorexia. The pain first occurred in the epigastric region then migrated to the periumbilical area. He had no bloody vomiting containing piecesof food. The patient also complained of weight loss (12Kg in one month) and constipation, which was relieved by medications, and now itwasrelapsing again. On physical examination, the patient was fully oriented withablood pressure of 130/80mmHg, pulse rate of 76 beats

per minute, and a body temperature of 36.8°C (axillary). In the abdominal examination, there was mild abdominal tenderness and distension without organomegaly. In further evaluation, uprightabdominal radiography revealed dilated small and large bowel without any free air or air-fluid level. Colonoscopy revealed patchy erythematous mucosa and hemorrhoid in the ascending colon(Figure 1).

Furthermore, abdominal and pelvic sonography revealed non-specific pathologic changes. Small bowel transient was performed and revealed normal intestinal mucosa, and there was no sign of obstruction in the small bowel (Figure 2).

Laboratory test results performedfor the patient are shown in <u>Table 1</u>. According to the results of the tests, hyperkalemia and hyponatremia were identified (<u>Chart A</u>).Due to weight loss, electrolyte disturbances, and low blood pressure, we suspected AI and requested the necessary tests.Measurements of serum cortisol soon after wakening (e.g., 6 to 8 AM in people who sleep at night) and adrenocorticotropic hormone (ACTH)were requested with suspected AI.Due to low cortisol and ACTH levels, which were about ten times the upper limit of normal, the patient was diagnosed as having AI. The patient was treated with corticosteroids. During a follow-up period of 6 months, thepatient was in good healthcondition. DISCUSSION

AI can arise from a primary adrenal disorder, secondary to adrenocorticotropic hormone deficiency, or by suppressionof adrenocorticotropic hormone by exogenous glucocorticoid or opioid medications(1, 2).Hallmark clinical features are unintentional weight loss, anorexia, postural hypotension, profound fatigue, muscle and abdominal pain, and hyponatremia(3-7). Hyponatremia(70-80%) is the most commonbiochemical abnormality, followed by hyperkalemia (30–40%) and



Fig 1: Colonscopy, Ascending colon and terminal ileum



Fig. 2: Small bowel transient

normochromic anemia (11-15%)(8, 9).

One of the clinical manifestations of AI is GI complications that may be revealed as the primary manifestation of AI.Gastrointestinal disturbances wereseen in more than90% of the patients(10, 11). Many times abdominal pain mimic the acute surgical abdomen(12).In this case study, we reported a man with bowel pseudo-obstruction who was diagnosed withAI.

Bowel pseudo-obstruction refers to small bowel obstruction determined by the development of mechanical bowel obstruction in the absence of an obstructive ulcer. The exact pathogenesis of bowel pseudo-obstruction is unknown in AI.However,intestinal pseudo-obstruction may occur most likely due to dysfunction of the smooth muscles of the intestine, followed by impaired bowel movements due to electrolyte imbalances.

Most AI clinical features are GI symptoms such as anorexia, nausea, vomiting, and abdominal pain, which can be so severe that can be misdiagnosed with an acute abdomen.Thesesymptoms can also be seen in bowel obstruction; therefore, there is an overlap between clinical features of acute AI and bowel obstruction. Abdominal

Table 1:Laboratory tests

Hematological tests		Biochemistry	
Test	Value	Test	Value
White blood cell count	9000/mm ³	Blood sugar	80 mg/dL
• Neutrophil	• 50%	Urea	52 mg/mL
LymphocyteMonocyte	 35% 10% 	Creatinine	1.4 mg/dL
Eosinophil	• 5%	Amylase	54 U/L
Red blood cell count	5.22×10 ⁶ /mm ³	Lipase	40 U/L
Hemoglobin	13.9 gr/dL	Aspartate transaminase	27 U/L
Hematocrit	40.5%	Alanine transaminase	28 U/L
Mean corpuscular volume (MCV)	77.59 fL	Alkaline phosphatase	54 U/L
Mean corpuscular hemoglobin concentration	34.32%	Serum iron	179 µgr/dL
Mean corpuscular hemoglobin	26.63 pg	Total iron binding capacity	361 µgr/dL
Platelets	396000/mm ³	Direct bilirubin	0.2
Prothrombin Time (PT)	13 sec	Total bilirubin	0.8
nternational Normalized Ratio (INR)	1.13 sec	Uric acid	4.1 mg/dL
Partial Thromboplastin Time (PTT)	12 sec	Urine analysis	
Erythrocyte sedimentation rate	11	Test	Value
		White blood cell	10-25/hpf
Sodium (Na)	126 meq/L	Red blood cell	0-2/hpf
Potassium (K)	5.5 meq/L	Epithelial cell	5-10/hpf
Cortisol (8 AM)	1.99 µgr/dL	Bacteria	Few
Adrenocorticotropic hormone (ACTH)	578 pg/mL	Specific gravity	1.010

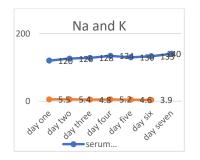


Chart 1: The process of changes in sodium and potassium

pain and vomiting often herald adrenal crisis(13).Only a handful of caseshave been reported on the association between intestinal obstruction and AI (3,4,14,15).We divided the causes of intestinal pseudo-obstruction into primary and secondary groups.Smooth muscle dysfunction was categorized as a rare and primary cause of intestinal pseudo-obstruction. Secondary causes include myxedema, opioid drugs, and Parkinson'sdisease(14,15). Rapid and dramatic correction of obstruction after glucocorticosteroid administration can also be used as a basis for the diagnosis of AI.Bowel obstruction resolved after injection of hydrocortisone in the presented case.

CONCLUSION:

Acute AI is manifested by intestinal obstruction, which is a very rare presentation of AI. This case report illustrated one of the diverse manifestations of AI. Patients presenting with unexplained pseudo-obstructions or the diagnosis of an AI should be considered. There fore, it is essential to consider AI in patients with intestinal obstruction of an undetermined cause, especially in conditions that predispose patients to steroids.

CONFLICT OF INTEREST:

The authors declare that they have no conflict of interest.

AUTHORS' CONTRIBUTIONS:

All authors have accepted responsibility for the entire content of this submitted manuscript and approved submission.

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