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DIAGNOSTIC COMPLEXITY OF CROHN'S DISEASE IN A PATIENT WITH SEVERE MALNUTRITION AND ANOREXIA NERVOSA: A CASE REPORT

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ABSTRACT

Crohn's disease (CD) is a chronic inflammatory bowel disease characterized by transmural inflammation and a relapsing course. Accurate assessment of disease activity requires integration of clinical findings, laboratory markers, endoscopic evaluation and

imaging studies. In patients with severe malnutrition and coexisting eating disorders, this assessment may be particularly challenging due to overlapping symptoms and potential inconsistency between clinical presentation and inflammatory biomarkers.

We report the case of a 46-year-old woman with CD admitted for exacerbation of gastrointestinal symptoms in the setting of profound malnutrition and anorexia nervosa. Despite moderate-to-severe clinical disease activity, inflammatory markers remained low, complicating evaluation of true disease severity. Management included intravenous corticosteroid therapy and intensive nutritional support with total parenteral nutrition. The patient developed hypophosphatemia consistent with refeeding syndrome, requiring careful metabolic correction. Clinical improvement was achieved through coordinated gastroenterological, nutritional, and psychiatric management. She is currently awaiting initiation of biological therapy.

This case illustrates the diagnostic difficulties of CD when coexisting with an eating disorder, underscoring the importance of early and accurate diagnosis and the prompt introduction of appropriate, multidisciplinary treatment to optimize patient outcomes.

Keywords: Crohn's disease, anorexia nervosa, malnutrition, inflammatory bowel disease, refeeding syndrome, eating disorders, CDAI.

INTRODUCTION

Diagnosing Crohn's disease (CD) is rarely straightforward, but in the presence of a coexisting eating disorder, it can become a clinical puzzle with serious consequences for the patient. CD is a chronic inflammatory bowel disease (IBD) characterized by transmural inflammation and a relapsing course. Diagnosis relies on clinical findings supported by laboratory tests, endoscopy with histopathology, and imaging [1]. Assessment of disease activity may be particularly challenging in patients with severe malnutrition or coexisting eating disorders, as symptoms such as weight loss and gastrointestinal complaints may overlap [2].

CD and other IBDs are frequently associated with mental health disorders, such as depression and anxiety. These often emerge alongside the earliest gastrointestinal symptoms, even before the disease is formally diagnosed [3].

Several cases have been reported in which CD was misdiagnosed as anorexia nervosa (AN). After complete treatment, which often required surgery, the patients achieved

about 80% of target body weight and symptom severity decreased considerably [4].

We present the case of a 46-year-old woman with CD whose clinical course was markedly affected by severe malnutrition and coexisting eating disorders. She had been diagnosed with CD two years prior to admission and had already been treated with oral and intravenous corticosteroids, azathioprine, and methotrexate. Mental health problems were identified many years earlier. This case also aims to highlight the diagnostic inaccuracies that can arise at the intersection of CD and AN, and to underscore the critical importance of early, accurate diagnosis and the timely introduction of appropriate, multidisciplinary treatment.

CASE DESCRIPTION

A 46-year-old woman was admitted to the hospital due to exacerbation of CD. For five days prior to admission, she had experienced worsening gastrointestinal symptoms, including approximately 10 watery stools per day with mucus. She had been diagnosed with CD two years earlier and had previously been treated with oral and intravenous corticosteroids, azathioprine, and methotrexate.

Her medical history was significant for AN, bulimia nervosa, and mixed anxiety-depressive disorder. She reported recurrent constipation and regular use of macrogols (polyethylene glycol). The history also revealed long-term nonsteroidal anti-inflammatory drug (NSAID) use and opioid use disorder secondary to chronic non-inflammatory pain.

The patient had previously undergone rectal suspension with sigmoid resection for rectal prolapse. Due to postoperative complications, further bowel resection was required with temporary stoma formation. Intestinal continuity was restored two years later. She also underwent two adhesiolysis procedures for adhesive small bowel obstruction.

At admission, the Crohn's Disease Activity Index (CDAI) was 448, indicating moderate-to-severe disease activity (Tables 1 and 2).

Table 1. Crohn's Disease Activity Index (CDAI) — Patient Scores.

CDAI COMPONENT	PATIENT VALUE	SCORE
Liquid/soft stools — avg. 8/day over 7 days (×14)	8	112
Antidiarrheal use (diphenoxylate/loperamide)	No	0
Abdominal pain — 7-day average	Mild	35
General well-being — 7-day average	Very poor	147
Arthritis/arthralgia	Yes	20
Other fistula	Yes	20
Fever >37.8°C in the past week	Yes	20
Abdominal mass	None	0
Hematocrit deviation (42 − 32%) × 6	10%	60
Body weight deviation (standard: 57.57 kg)	34%	34
Total CDAI		**448**
Interpretation: Moderate-to-severe disease activity (221–450)		

CDAI calculated according to: Best WR, Becktel JM, Singleton JW, Kern F Jr. Development of a Crohn's disease activity index. National Cooperative Crohn's Disease Study. Gastroenterology. 1976 Mar;70(3):439–444. PMID: 1248701.

Table 2. Crohn's Disease Activity Index (CDAI) — Score Interpretation.

CDAI SCORE	INTERPRETATION	PATIENT
0 – 149	Asymptomatic remission	
150 – 220	Mildly to moderately active Crohn's disease	
221 – 450	Moderately to severely active Crohn's disease	×
451 – 1100	Severely active to fulminant disease	

× Indicates patient's score (CDAI = 448). CDAI calculated according to: Best WR, Becktel JM, Singleton JW, Kern F Jr. Development of a Crohn's disease activity index. National Cooperative Crohn's Disease Study. Gastroenterology. 1976 Mar;70(3):439–444. PMID: 1248701.

On physical examination, the patient appeared cachectic, with lanugo hair and pale, dry oral mucosa. Body weight was 38 kg (approximately 66% of ideal body weight according to the Potton formula), height was 164 cm, and BMI was 14.1 kg/m^2. Vital signs were stable. The abdomen was soft and non-tender with normal bowel sounds. No peripheral edema was present at admission.

Laboratory tests revealed microcytic anemia (hemoglobin 10.3 g/dl, reference range: 12–15.1 g/dl), mild hyponatremia, and hypokalemia. Renal function was impaired (creatinine 1.4 mg/dl, reference range: 0.5–0.9 mg/dl; eGFR 47 mL/min/1.73 m^2, reference range: >90 mL/min/1.73 m^2). Despite severe malnutrition, albumin remained within the normal range. Hypokalemia was attributed to diarrhea and laxative use; it was corrected during hospitalization.

Endoscopic examinations performed one year prior to admission showed inflammatory changes consistent with CD. Upper gastrointestinal endoscopy revealed metaplasia and atrophy in the gastric antrum, a fundic gland polyp in the gastric corpus, and esophageal mucosal desquamation likely secondary to recurrent vomiting. Colonoscopy demonstrated segmental inflammatory lesions, mucosal atrophy in the terminal ileum, and postsurgical alteration of the ascending colon due to prior anastomosis, with three large pseudodiverticula. Ulcerations covered with fibrin, aphthous lesions, and fistulas were observed in the ascending and transverse colon. Histopathological examination revealed chronic inflammatory changes and raised the

possibility of NSAID-induced mucosal injury in addition to Crohn's-related lesions. The patient had discontinued NSAIDs three months prior to these examinations.

During hospitalization, abdominal ultrasound showed bowel wall thickening up to 4.5 mm without other significant abnormalities. Follow-up colonoscopy performed during the same admission revealed patchy hyperemic mucosa with contact bleeding, multiple longitudinal scars, and inflammatory changes most pronounced in the ascending colon, involving the entire circumference. A single concentric stricture and an inflammatory polyp were identified.

The main clinical challenge was determining whether the symptoms were due to active CD or related to severe malnutrition, laxative use, and chronic medication exposure.

Intravenous corticosteroid therapy was started and later transitioned to oral treatment, resulting in partial clinical improvement. She is currently awaiting initiation of biological therapy for CD. Due to severe malnutrition and insufficient oral intake, total parenteral nutrition (TPN) was started once a central venous catheter had been placed.

After initiation of TPN, serum phosphate levels decreased from 4.1 mg/dl to 1.5–1.8 mg/dl (reference range: 2.6–4.5 mg/dl), suggesting refeeding syndrome. Phosphate supplementation was introduced, and caloric intake was adjusted. Phosphate levels gradually improved to 2.6 mg/dl over the following days.

During hospitalization, recurrent peripheral edema and pleural effusion developed. These responded well to diuretic therapy and did not compromise respiratory function.

The patient's clinical condition improved. The number of stools decreased to two per day, and laboratory parameters progressively normalized. The patient was discharged in good general condition with recommendations for continued gastroenterological, nutritional, and psychiatric follow-up.

DISCUSSION

CD is defined as a chronic IBD characterized by transmural inflammation that can involve any segment of the gastrointestinal tract. It typically follows a relapsing and remitting course, with risks of stricturing, fistulising and extra-intestinal complications [1]. The pathogenesis of CD is multifactorial, involving genetic susceptibility, dysregulated immune response, and interaction with intestinal microbiota. Dysregulated T-

helper cell pathways, particularly Th1 and Th17, contribute to excessive cytokine production (e.g., TNF-α, IL-12, IL-17), leading to chronic inflammation and mucosal injury [1].

Since there is no single definitive diagnostic test for CD, diagnosis is established through an integrated approach that includes clinical evaluation, laboratory testing, endoscopy with histopathology, and cross-sectional imaging. This comprehensive strategy is essential given the heterogeneous presentation of the disease and the need to differentiate CD from other gastrointestinal disorders [2]. Routine laboratory tests, including complete blood count and C-reactive protein, serve as non-specific markers of systemic inflammation, while fecal calprotectin more specifically reflects intestinal inflammation. Ileocolonoscopy with biopsies remains the gold standard for diagnosing CD, as it allows direct visualization of mucosal lesions; typical findings include segmental skip lesions, deep ulcerations, and transmural inflammation. Histopathological analysis of biopsies may additionally reveal chronic inflammatory changes and granulomas [5]. Cross-sectional imaging, specifically magnetic resonance and computed tomography enterography, is increasingly used in the diagnostic work-up to detect strictures, fistulae, and extra-intestinal complications that may not be apparent on endoscopy [6]. Integrated diagnostic strategies that combine these modalities are essential for improving diagnostic accuracy, reducing treatment delays, and optimizing long-term outcomes [7].

Diagnostic delay in CD has been investigated, with the mean delay estimated at 3–4 years. It is particularly significant in children and adolescents, in whom the disease is most commonly mistaken for colonic lesions, appendicitis, and functional bowel disturbance [8].

Given the overlap in presenting symptoms such as weight loss, food avoidance, and gastrointestinal discomfort, differentiation between CD and AN can be diagnostically challenging, often requiring comprehensive somatic and psychiatric assessment [2]. CD is characterized by objective gastrointestinal inflammation, endoscopic findings and biomarker abnormalities, whereas AN primarily involves intentional restriction of intake and distorted body image without organic gut pathology [9]. The core symptoms of AN are intentional weight reduction, disturbed body image and amenorrhoea. Patients lose weight not only by restricting food intake but also, in some cases, by self-induced vomiting, the use of laxatives or diet pills, and excessive exercise [10].

However, P. Mallett and S. Murch noted that weight loss and anorexia can arise as manifestations of inflammatory bowel disease itself, without the presence of body image

distortion or other characteristics of a primary eating disorder [11].

Moreover, it has been observed that IBDs are associated with several mental health disorders. Symptoms of depression and anxiety may arise as a consequence of early manifestations of the gastrointestinal condition, even before it has been formally diagnosed. Notably, the majority of cases of elevated anxiety or depression identified following the diagnosis of IBD occur within the first year after diagnosis. This pattern indicates that these mental health issues most likely arise because of the IBD itself, rather than being present beforehand [3].

A pediatric case report highlights the association between AN and IBD. It describes a female patient who presented with CD. She initially responded well to induction therapy with exclusive enteral nutrition and maintenance therapy with thiopurine. Later, infliximab, an anti-TNF alpha monoclonal antibody, was used to reinduce and maintain remission. A few years later, she experienced a disease relapse and was switched from infliximab to adalimumab, another anti-TNF alpha monoclonal antibody, combined with exclusive enteral nutrition at 2,400 kilocalories per day. Despite these interventions, she continued to lose weight rapidly, developed electrolyte disturbances, and exhibited severe malnutrition. Multidisciplinary assessment, including continuous supervised feeding and psychiatric evaluation, led to the diagnosis of anorexia nervosa, which caused persistent restriction of food intake despite active inflammatory bowel disease. Intensive child psychiatry and psychology interventions, along with parenteral nutrition when necessary, stabilized her condition, allowing for safe discharge and structured outpatient follow-up [12].

Several cases have been described in which CD was misdiagnosed as AN. One case involved a female patient with a three-year history of weight loss, intermittent abdominal pain, constipation, and episodes of diarrhea. A tender mass was found in the right iliac fossa. Enterography revealed a fistula between the small intestine and sigmoid colon. Subsequent laparotomy confirmed characteristic Crohn's lesions affecting the mucosa of the terminal ileum, caecum, and descending and sigmoid colon. After a resection of the descending and sigmoid colon, and modified right hemicolectomy, she achieved 80% of target body weight, and her symptoms improved noticeably. The other three patients were also young women who had been misdiagnosed with AN. After treatment, which often required surgery, they were able to gain weight and experienced a reduction in their symptoms [4].

The causal connection between CD and AN remains unclear; however, genetically predicted AN was significantly associated with an increased risk of acute gastritis and CD.

The reverse Mendelian randomization analysis did not reveal a causal relationship [13].

Current ECCO guidelines recommend a personalized approach to therapy, including corticosteroids, immunomodulators and biologics, coupled with surgical intervention for complications, with nutritional optimization playing a supportive role [14]. Treatment of anorexia nervosa typically involves a team approach, with family physicians collaborating with psychotherapists, psychiatrists, and eating disorder specialists, who also support nutritional planning. For children and adolescents, coordination with schools is essential. Successful therapy depends on a strong patient-therapist relationship, family involvement, and close teamwork among all care providers [15].

CONCLUSION

This case demonstrates the significant impact of severe malnutrition and eating disorders on the course of CD. Coexisting anorexia, laxative use, chronic NSAID exposure, and opioid use disorder substantially complicated the clinical picture and obscured assessment of disease activity.

Effective management required anti-inflammatory therapy, intensive nutritional support, monitoring for refeeding syndrome, and interdisciplinary care. Early and accurate diagnosis, recognition of factors that may complicate the disease course, and the timely introduction of appropriate treatment are all essential in patients with IBD and coexisting eating disorders.

DISCLOSURE

Author Contributions

Conceptualization, writing — original draft, and final approval of the manuscript: Zofia Wcisło.

Clinical care of the patient, literature review: Grzegorz Szmit, Weronika Basak, Zofia Wcisło.

Data curation and data presentation: Anna Kułach, Adrianna Purwin, Karolina Lach.

Writing — review and editing: Gabriela Krok, Łukasz Lamparski, Maria Chmielewska, Wiktoria Modrzejewska.

All authors have read and agreed to the published version of the manuscript.

Ethics

The patient consent has been obtained for this clinical case.

Use of Artificial Intelligence

AI-assisted technologies were used in the preparation of this manuscript solely to improve the readability and language quality. The authors reviewed, verified, and edited all modifications, and take full responsibility for the final content and accuracy of the reported data.

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