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Crohn's Disease Initially Treated as Avoidant/ Restrictive Food Intake Disorder in a Young Woman

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Study Design A
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Patient: Female, 19-year-old
Final Diagnosis: Crohn's disease
Symptoms: Fear of eating • nausea • postprandial abdominal pain • severe emaciation
Clinical Procedure: —
Specialty: Laboratory Diagnostics

Objective: Unusual clinical course
Background: Avoidant/restrictive food intake disorder (ARFID) can be difficult to distinguish from food avoidance that is an adaptive response to gastrointestinal disease. ARFID-like restrictive eating is relatively common in patients with inflammatory bowel disease (IBD), whereas the frequency of undiagnosed IBD among patients initially diagnosed with ARFID remains unclear. We report the case of a young woman who was admitted with presumed ARFID and was subsequently diagnosed with ileocolonic Crohn's disease.

Case Report: A 19-year-old Japanese woman was admitted to a psychiatric ward with severe emaciation (body mass index, 10.96 kg/m²), fear of eating because of postprandial abdominal pain and nausea, and no weight- or shape-related psychopathology. ARFID was initially suspected. However, the diagnosis was reassessed because of persistent diarrhea, fluctuating C-reactive protein elevation, anemia, and poor response to nutritional rehabilitation. Contrast-enhanced computed tomography and gastrointestinal endoscopy revealed ileocolonic Crohn's disease. After transfer to the gastroenterology department, nutritional therapy and risankizumab were initiated. The intestinal inflammation improved, the eating difficulty resolved, and psychiatric treatment was discontinued. She remained psychiatrically stable without recurrence of eating difficulty during 1 year of follow-up.
Conclusions: Crohn's disease can present with ARFID-like food restriction rather than persistent primary ARFID when food restriction is driven by postprandial abdominal pain, nausea, and diarrhea. Longitudinal reassessment for IBD should be considered when gastrointestinal symptoms, inflammatory markers, anemia, or poor response to refeeding persist.

Keywords: Avoidant Restrictive Food Intake Disorder • Crohn Disease • Inflammatory Bowel Diseases

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Introduction

Avoidant/restrictive food intake disorder (ARFID) is characterized by clinically significant restriction of food intake that causes weight loss, nutritional deficiency, reliance on nutritional supplementation, or psychosocial impairment, without the weight- or shape-related psychopathology typical of anorexia nervosa [1,2]. Its clinical presentation is heterogeneous and includes sensory sensitivity, lack of interest in eating, and fear of aversive consequences such as choking, vomiting, nausea, or abdominal pain [1,2]. Because these reasons for avoiding food are often related to bodily sensations, ARFID can be difficult to diagnose in patients with gastrointestinal symptoms [3,4]. According to DSM-5, ARFID can be diagnosed in patients with a medical condition only when the eating disturbance is more severe than that usually explained by the condition. However, it can be difficult to make this judgment before an underlying gastrointestinal disease has been fully evaluated [2-4].

Inflammatory bowel disease (IBD), including Crohn's disease and ulcerative colitis, commonly causes abdominal pain, diarrhea, nausea, fatigue, anemia, hypoalbuminemia, and weight loss [5,6]. Patients may restrict foods to avoid symptom exacerbation, and such restriction can become excessive, persistent, and nutritionally harmful. Studies in IBD patients have reported positive ARFID screens or ARFID risk in approximately 10% to 20% of patients [7-10], but the frequency of unrecognized IBD among patients initially diagnosed with ARFID is unknown. This issue is clinically important because psychiatric treatment and nutritional rehabilitation alone may be insufficient when food restriction is secondary to active intestinal inflammation.

We report the case of a patient who was initially diagnosed with ARFID but was later found to have Crohn's disease. The objective of this case report is to illustrate that active Crohn's disease can present with ARFID-like food restriction secondary to postprandial gastrointestinal symptoms rather than a primary eating disorder, and that longitudinal reassessment should be considered when gastrointestinal symptoms or inflammatory findings persist.

Case Report

The patient was a 19-year-old Japanese woman with no history of smoking, alcohol use, or illicit drug use. She had no remarkable medical history and no childhood history suggesting autism spectrum disorder, attention-deficit/hyperactivity disorder, or other psychiatric disorders.

At 17 years of age, after parental divorce and remarriage and interpersonal difficulties with relatives living in the same

household, she developed mild depressed mood, reduced appetite, school absenteeism, weight loss from approximately 47 kg, and diarrhea. After graduating from high school, she did not start work and remained socially withdrawn at home. At 18 years of age, she developed postprandial lower abdominal pain and nausea, and her oral intake decreased further. She visited a gynecology clinic and a neurology clinic, but no specific abnormality was identified. In January 2025, she visited a local gastroenterology clinic because of anemia, marked weight loss to 27 kg, and postprandial nausea and abdominal pain, and was referred to our hospital for inpatient treatment.

At admission, her height was 152.5 cm and body weight was 25.5 kg, corresponding to a body mass index (BMI) of 10.96 kg/m². Laboratory test results are shown in **Table 1**. Chest radiography showed a small vertical heart, and electrocardiography showed sinus tachycardia of 118 beats/min. Whole-body computed tomography (CT) showed bilateral buttock pressure ulcers but no clear intra-abdominal abnormality. She had lost 20 kg during the preceding year.

She reported fear and avoidance of eating because meals were followed by lower abdominal pain and nausea. She had mild depressed mood and insomnia, but no anhedonia, psychomotor agitation or retardation, suicidal ideation, concentration difficulties, feelings of worthlessness, hallucinations, or delusions. She denied a desire for thinness, fear of weight gain, body-image disturbance, binge eating, self-induced vomiting, and laxative misuse. Based on profound undernutrition, meal-related fear, and absence of anorexia nervosa psychopathology, ARFID with severe emaciation was suspected [2], and she was admitted to the psychiatric ward.

After admission, medical stabilization, refeeding management, supportive psychotherapy, iron supplementation, and probiotics were started. Oral intake was encouraged, but she could not increase intake because of postprandial abdominal pain; therefore, enteral nutrition through a nasogastric tube was used. Her body temperature remained in the upper 36 °C to lower 37 °C range, and C-reactive protein (CRP) fluctuated between 0.34 and 11.14 mg/dL. Despite nutritional support, her general condition did not improve sufficiently, and her body weight further decreased to 24.6 kg. Because chronic diarrhea, persistent inflammation, anemia, and poor response to nutritional rehabilitation were inconsistent with a purely psychiatric eating disorder, the diagnosis was reassessed.

Approximately 1 month after admission, contrast-enhanced abdominal and pelvic CT demonstrated inflammatory wall thickening from the ileum to the ascending colon. Upper and lower gastrointestinal endoscopy performed approximately 1.5 months after admission showed longitudinal ulcers, a cobblestone appearance, skip lesions, and luminal narrowing

Table 1. Laboratory data on admission.

Laboratory parameter	Value	Reference range
Total protein (g/dL)	5.1	6.6-8.1
Albumin (g/dL)	1.9	4.1-5.1
Total bilirubin (mg/dL)	0.5	0.4-1.5
Aspartate aminotransferase (U/L)	5	13-30
Alanine aminotransferase (U/L)	4	7-23
Alkaline phosphatase (U/L)	94	38-113
Cholinesterase (U/L)	70	201-421
Gamma-glutamyltransferase (U/L)	7	9-32
Blood urea nitrogen (mg/dL)	5	8-20
Creatinine (mg/dL)	0.38	0.46-0.79
Sodium (mmol/L)	138	138-145
Potassium (mmol/L)	4.6	3.6-4.8
Chloride (mmol/L)	100	101-108
Calcium (mg/dL)	7.8	8.8-10.1
Phosphorus (mg/dL)	3.5	2.7-4.6
Total cholesterol (mg/dL)	77	128-219
Triglycerides (mg/dL)	62	30-149
Glucose (mg/dL)	97	73-109
C-reactive protein (mg/dL)	9.77	0.00-0.14
White blood cell count (/μL)	6700	3300-8600
Red blood cell count (× 10 ⁶ /μL)	5.03	3.86-4.92
Hemoglobin (g/dL)	10.1	11.6-14.8
Hematocrit (%)	33.5	35.1-44.4
Mean corpuscular volume (fL)	66.6	83.6-98.2
Mean corpuscular hemoglobin concentration (g/dL)	30.1	31.7-35.3
Platelet count (× 10 ³ /μL)	394	158-348
Serum iron (μg/dL)	8	40-188
Unsaturated iron binding capacity (μg/dL)	80	137-325
Total iron binding capacity (μg/dL)	88	251-398
Ferritin (ng/mL)	169	10-120
Free triiodothyronine (pg/mL)	1.53	2.30-4.00
Free thyroxine (ng/dL)	1.15	0.90-1.70
Thyroid-stimulating hormone (μIU/mL)	1.360	0.500-5.000

in the intestine. She was diagnosed with ileocolonic Crohn's disease and transferred to the gastroenterology department.

After transfer, nutritional therapy through a peripherally inserted central catheter was administered. Risankizumab, an interleukin-23 inhibitor, was selected as the initial biologic therapy for this patient because of her high disease activity, the lower induction-remission rates reported for vedolizumab in this setting [11], reproductive safety concerns precluding upadacitinib in this young woman [12], and a more favorable infection-risk profile than anti-tumor necrosis factor agents given her severe malnutrition [13]. These considerations were explained to the patient and her family, who agreed to treatment with risankizumab. It was administered according to the standard regimen: induction with 600 mg intravenously at weeks 0, 4, and 8, followed by maintenance with 360 mg subcutaneously every 8 weeks. After treatment, CRP decreased to less than 0.10 mg/dL, and follow-up CT showed improvement of intestinal inflammation. Oral intake was restarted stepwise. The abdominal pain and diarrhea improved, and her fear of eating disappeared. At discharge after approximately 2.5 months of hospitalization, her body weight had increased to 29.8 kg.

One month after discharge, her body weight was 38.5 kg (BMI, 16.0 kg/m²). She had no residual depressed mood, insomnia, or other psychiatric symptoms, and psychiatric treatment was terminated. During 1 year of follow-up after discharge, she remained psychiatrically stable, with no recurrence of eating difficulty, while continuing treatment for Crohn's disease.

Discussion

The main contribution of this case report is that it illustrates a diagnostic boundary between primary ARFID, ARFID coexisting with IBD, and ARFID-like food restriction secondary to active Crohn's disease. In the present case, ARFID was clinically plausible at admission because the patient had marked weight loss, avoided eating because of anticipated abdominal pain and nausea, and did not show weight- or shape-related psychopathology. Several alternative psychiatric diagnoses were considered. Major depression was unlikely because depressive symptoms were mild and did not meet the diagnostic criteria [2]. Anorexia nervosa was also unlikely because she had no drive for thinness, fear of weight gain, or body-image disturbance. Delusional disorder or schizophrenia with somatic hallucinations was not supported because she had no psychotic symptoms, somatic hallucinations, or fixed delusional interpretation of her abdominal symptoms. Nevertheless, chronic diarrhea, fluctuating CRP elevation, anemia with low serum iron, pressure ulcers in the setting of extreme undernutrition, and poor response to refeeding were atypical for primary ARFID. The complete resolution of eating difficulty after

treatment of Crohn's disease supports the interpretation that food restriction in this patient was mainly secondary to intestinal inflammation rather than persistent primary ARFID. A previous pediatric case report described the coexistence of Crohn's disease and ARFID resulting in failure to thrive [14]. In contrast, the present case provides a clinically useful example of Crohn's disease presenting with ARFID-like restrictive eating that resolved after treatment of intestinal inflammation.

This case does not establish how often Crohn's disease mimics ARFID or how often ARFID and IBD coexist. However, it illustrates that ARFID-like food restriction in IBD may require careful interpretation. Primary ARFID can occur without organic gastrointestinal inflammation; ARFID can coexist with IBD when food avoidance persists beyond what is medically necessary; and active Crohn's disease can also produce ARFID-like symptoms when postprandial pain, nausea, and diarrhea lead to fear-based restriction. Therefore, some patients with IBD who appear to have ARFID may have food restriction secondary to intestinal inflammation rather than a true comorbid eating disorder. This possibility should be examined in future studies. Although ARFID risk or positive ARFID screening has been reported in patients with IBD [7-10], these results from screening tools do not necessarily mean that the patients meet the DSM diagnostic criteria for ARFID. A recent systematic review similarly reported wide variation in ARFID prevalence among IBD patients (10.2-51.3%) and noted that restrictive eating sometimes persists despite normalized inflammatory markers, suggesting that mechanisms beyond active inflammation alone may also contribute [15]. Future studies should clarify the diagnostic status of ARFID-like symptoms in active IBD and examine how often unrecognized IBD is found among patients diagnosed with ARFID.

A brief gut-brain interpretation may also be considered only as background speculation. Previous studies have reported default mode network alterations in Crohn's disease and possible default mode network-related findings in ARFID [16-18]. A recent narrative review similarly proposed that fear-related neural regulation and microbiota-gut-brain axis dysregulation underlie ARFID in IBD, and suggested that chronic intestinal inflammation itself can directly affect central appetite regulation and emotional processing [19]. These findings may provide a possible background for future studies on the relationship between intestinal inflammation, visceral sensation, and food restriction. However, our patient did not undergo neuroimaging or other mechanistic assessment, and these findings do not directly explain the present case.

This case has several clinical implications. Psychosocial stressors and the absence of weight- or shape-related psychopathology may support consideration of ARFID, but medical evaluation should be continued when findings suggest an organic

disease. In this patient, chronic diarrhea, fluctuating CRP elevation, anemia with low serum iron, and continued weight loss despite nutritional support led to reassessment of the diagnosis. In such cases, repeat evaluation with contrast-enhanced CT, endoscopy, and gastroenterology consultation should be considered. Fecal calprotectin was not measured in this patient; as a noninvasive marker of intestinal inflammation, it may also be a useful adjunct when persistent gastrointestinal symptoms raise suspicion of IBD. The disappearance of food-related fear after Crohn's disease treatment further supported intestinal inflammation as the main cause of the ARFID-like presentation.

Conclusions

This case illustrates that Crohn's disease can present with ARFID-like food restriction when eating avoidance is driven by postprandial abdominal pain, nausea, and diarrhea. It also suggests the importance of distinguishing primary ARFID, ARFID coexisting with IBD, and food restriction secondary to active Crohn's disease. When gastrointestinal symptoms, inflammatory markers, anemia, hypoalbuminemia, or poor response to nutritional rehabilitation persist, a primary psychiatric diagnosis should be reconsidered, and longitudinal reassessment for IBD, including Crohn's disease, should be considered.

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Department and Institution Where Work Was Done

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Patient Consent

Written informed consent was obtained from the patient for the publication of this case report.

Ethics Statement

Publication of this case was approved by the Ethical Review Committee of the Yamagata University Faculty of Medicine.

Declaration of AI and AI-assisted Technologies in The Writing Process

ChatGPT was used only to improve the English grammar and readability of this manuscript. The authors reviewed and approved all final content.