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Spontaneous Bacterial Peritonitis in a Patient with Anorexia Nervosa with Profound Zinc and Iron Deficiency

To the Editor:

Anorexia nervosa is a complex psychological disorder in which physical health issues can arise owing to either the direct or long-term effects of severe malnutrition. Here we describe a case of life-threatening spontaneous bacterial peritonitis in a patient with anorexia nervosa and examine the potential causative factors.

CASE REPORT

A 30-year-old woman with a history of anorexia nervosa and iron deficiency anemia attended for an intravenous ferric carboxymaltose infusion, having received ferric carboxymaltose (750 mg) 7 days before this.

On arrival she complained of severe abdominal pain and nausea. She denied any vomiting, urinary symptoms, change in bowel habit, or recent sexual activity.

On examination she was severely cachectic and febrile at 38.6°C. She had cool peripheries, pulse 88 beats per minute, blood pressure 82/54 mm Hg, and was noted to have a slightly distended abdomen, with peritonism and infrequent bowel sounds.

Initial investigations are shown in the **Table**. Initial treatment was commenced with intravenous thiamine, fluid resuscitation with 0.9% saline, and intravenous piperacillin/tazobactam. An abdominal CT scan revealed ascites and peritoneal enhancement (**Figure**).

The patient underwent diagnostic laparoscopy. This revealed purulent intra-abdominal fluid, which grew large numbers of *Escherichia coli* sensitive to co-amoxiclav. Treatment was continued with co-amoxiclav for 7 days.

Concurrent with her postoperative antibiotic therapy she underwent nutritional support with oral nutrition and iron supplementation. Despite this there was no improvement in her hemoglobin level. Further results became available, indicating that she was profoundly zinc deficient (**Table**), and after oral zinc replacement her full blood count normalized.

DISCUSSION

Spontaneous bacterial peritonitis is a life-threatening infection of ascitic fluid in the absence of any intra-abdominal, surgically treatable source of infection.[1] Spontaneous bacterial peritonitis is the most common bacterial infection seen in patients with hepatic cirrhosis and is associated with a significant mortality, both at the time of the episode and in the year following the index episode.[1] The cause of most cases of spontaneous bacterial peritonitis is bacterial translocation into mesenteric lymph nodes and subsequently into ascitic fluid;[1] in patients with cirrhosis alterations in gut microbial flora, increased gut permeability and host immune responses are probably involved.[1]

This patient did not have hepatic cirrhosis, nor was there evidence of intestinal perforation to give rise to peritoneal infection. Spontaneous bacterial peritonitis has been reported to occur in patients with nephrotic syndrome with ascites,[2] but we are unaware of any cases of spontaneous bacterial peritonitis presenting in this manner in patients with anorexia nervosa. Significant fluid retention with the transient development of ascites, pleural effusions, and pericardial effusions are recognized features of refeeding syndrome in severely malnourished individuals.

Patients with eating disorders may have altered gut motility, especially if there are associated bingeing or purging behaviors, although there is no evidence that this leads per se to altered gut micro flora or altered intestinal permeability in these patients.[3]

Approximately 50% of patients with anorexia nervosa have evidence of zinc deficiency.[4] Zinc deficiency can lead to deficient immune response owing to its importance as a secondary messenger in lymphocytes and macrophages.[5] Zinc supplementation also can be used to treat childhood diarrhea.[6]

The role of iron status and replacement and the potential for bacterial infection has been a subject of debate.[7] However, despite laboratory and animal studies suggesting potential mechanisms for iron-associated bacteremia, randomized studies in patients with renal disease or heart failure show no evidence of increased rates of bacterial infection in patients treated with intravenous iron.[7]

CONCLUSION

This case demonstrates the potential for patients with anorexia nervosa to develop profound life-threatening

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Table Selected Initial Laboratory Investigations at Presentation

Hemoglobin (g/L)	76
NR 115-160	
Mean corpuscular volume (fL)	65
NR 78-98	
White cell count ($\times 10^9$/L)	2.5
NR 4.0-11.0	
Platelets ($\times 10^9$/L)	152
NR 150-400	
Transferrin saturation (%)	3
Ferritin (µg/L)	898
NR 15-200	
Magnesium (mmol/L)	0.69
NR 0.70-1.00	
Calcium (mmol/L)	2.26
NR 2.1-2.6	
Zinc (µmol/L)	1.1
NR 10-18	
C-reactive protein (mg/L)	144
NR 0-5	
Lactate (mmol/L)	3.5
NR 0.6-2.4	
Albumin (g/L)	32
NR 30-45	
Bilirubin (µmol/L)	32
NR 3-21	
ALT (U/L)	42
NR 10-50	
AlkPhos (U/L)	31
NR 40-125	
Phosphate (mmol/L)	0.88
NR 0.8-1.4	
Amylase (U/L)	34
NR 0-100	

Other investigations: Electrocardiogram: Sinus rhythm. Chest x-ray: Normal paucity of bowel gas in pelvis and upper abdomen. Appearances suggestive of free fluid in abdomen.

AlkPhosph = alkaline phosphatase; ALT = alanine aminotransferase; NR = normal range.

illnesses, not commonly observed in healthy young adults. The development of profound asymptomatic zinc deficiency and the potential to develop life-threatening spontaneous bacterial peritonitis is a clear indication that these patients require close medical monitoring by experienced physicians with training in acute and chronic medical illness. In particular, there is a need to remember that fever and persistent abdominal pain are not features of refeeding and should prompt rapid evaluation by specialist medical practitioners.

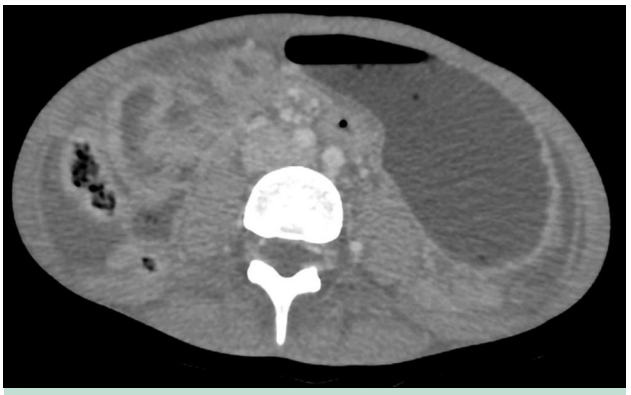

Figure Computed tomographic image demonstrating ascites with abnormal peritoneal enhancement without evidence of intestinal perforation.

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