Clinical Reports

Anorexia nervosa and necrotizing colitis: case report and review of the literature

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Summary: A 20 year old woman with anorexia nervosa and severe weight loss developed extensive necrotizing colitis after being admitted to hospital for stabilization of her weight. Necrotizing colitis with anorexia nervosa has been reported previously. We report a second case. We believe necrotizing colitis is a true complication of anorexia nervosa and should be considered in the differential diagnosis in patients with primary anorexia who develop an acute abdomen.

Introduction

Necrotizing colitis is a severe patchy gangrenous disease of the bowel manifested histologically as mucosal infarction with vascular thrombosis of small vessels and gas cysts in the bowel wall. It is caused by clostridial pathogens which release toxins locally. Anorexia nervosa has been previously reported in association with necrotizing colitis. We report another case of anorexia nervosa complicated by necrotizing colitis which we believe is a true though rare complication of anorexia nervosa.

Case history

A 20 year old women with anorexia nervosa, and a history of depression and obsessional problems was transferred to the casualty department from a psychiatric hospital with a short history of watery diarrhoea and mild abdominal pain followed by sudden severe abdominal pain mainly in the right iliac fossa with circulatory collapse.

She had been admitted to the psychiatric ward 3 days earlier after the loss of approximately 50% of her body weight which was 30 kg on admission. Overeating was suspected 2 days earlier but not proven.

On admission she was shocked with a rigid, slightly distended abdomen. She was hypothermic with a temperature of 34.5°C. Her serum urea, electrolytes, amylase and glucose were normal.

The haemoglobin was normal, 13.6 g/dl with white cell count of 11.4 × 10⁹/l and platelet count of 54 × 10⁹/l. The prothrombin time was 21 seconds with control of 14 seconds. The activated partial thromboplastin time was 77 seconds (control 36 seconds). An abdominal X-ray showed no fluid levels and thumb prints appearance or free gas in the hepatobiliary system. After resuscitation with fluid, fresh frozen plasma and platelets, emergency laparotomy was performed. The colon was gangrenous from the ileo-caecal junction to the splenic flexure with no evidence of arterial occlusion. Extended right hemicolectomy and a double-barrelled colostomy were performed. Postoperatively she remained in a critical condition. A second laparotomy was performed with further necrotic bowel resected. After 11 days she deteriorated and repeated laparotomy showed no viable gastrointestinal tract from the stomach to rectum. She died the next day. The pathological specimens confirmed the operative diagnosis of necrotizing colitis with positive tissue Gram’s stain.

Discussion

Severe anorexia nervosa affects about one in 200 teenage females. The associated mortality is between 15% and 20% at 20 years and is related to the percentage of body weight loss. Fluid imbalance, suicide and inanition are the common causes of death. Necrotizing colitis may be another lethal complication.

Clostridium perfringens is the causative pathogen of necrotizing colitis but its relation to anorexia is not clear. A similarity between bulimia and acute clostridial colitis (pig bel) has been suggested. In both conditions acute gastric dilatation occurs after alteration in eating habits. B toxins are
particularly susceptible to breakdown by pancreatic proteases, the outlet of which may be markedly reduced by involuntary starvation due to gastric dilatation and stasis. In anorexia nervosa voluntary starvation followed by refeeding even at a normal rate may trigger stasis and gastric dilatation resulting in necrotizing colitis. This has a similarity to reported cases of enteritis necroticans in prisoners of war after the intake of comparatively small amounts of food.

In anorexia nervosa the gastric dilatation appears to follow 2 weeks or so of refeeding. Several studies on patients with primary anorexia nervosa and bulimia have shown a significant reduction of the emptying rate of both the liquid and solid phases of the meal. Gastric secretion has also been shown to be reduced in anorexic patients. It has been suggested that constipation and faecal impaction, which are common in anorexic patients, may lead to large bowel obstruction and subsequent necrotizing colitis. The role of the major tranquillizers and antidepressants as a cause for gastrointestinal stasis in anorexic patients has not been investigated.

In our case we believe that the prolonged starvation followed by the refeeding of a normal diet was the possible triggering factor. Whether a secretive bingeing habit or the major tranquillizers were additional contributory factors is unclear.

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References

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