Case Report

Complications of non-occlusive mesenteric ischaemia

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Case: A 65-year-old arteriopath with a history of myocardial infarction 5 months previously presented with classical signs of mesenteric infarction that led to a right hemicolectomy with an end ileostomy.

Outcome: Postoperative complications occurred due to unusually large volume ileostomy output in the subsequent 4 weeks, resulting in severe volume depletion and the sequelae that required intensive care support. These were triggered and prolonged by two episodes of intra-abdominal sepsis.

Conclusions: Sepsis-induced high ileostomy output following intestinal resection for non-occlusive mesenteric ischaemia is a serious complication. Early restoration of intestinal continuity following bowel resection for established infarction may prevent this complication.

Key words: Ileostomy, intensive care, non-occlusive mesenteric ischaemia, pharmacotherapy, sepsis

INTRODUCTION

ACUTE INTESTINAL ISCHAEMIA is defined as a threatened or established ischaemic necrosis of the major part of the bowel. Two-thirds of cases are occlusive in origin, with approximately 70% affecting the superior mesenteric artery (SMA), and a third of cases show no gross arterial or venous occlusion, i.e., non-occlusive.¹ Non-occlusive mesenteric infarction occurs in low cardiac output states due to sepsis, congestive cardiac failure, hypovolaemia, aortic insufficiency, renal or hepatic disease, or following cardiac surgery.^{1,2} The mortality from a non-occlusive mesenteric infarction is greater than 80% due to the premorbid state of these patients and at least partly due to the vague symptomatology of the disease often delaying diagnosis.^{1,3} The extensive collateral circulation (Fig. 1) and increased oxygen extraction would compensate for a 75% reduction of mesenteric blood flow for up to 12 h.³ The majority of cases involve spasm of branches of the SMA supplying the small intestine and proximal colon with a higher mortality from colonic infarct.^{1,4} There is role for early

pharmacotherapy in correcting the underlying cause of the hypoperfusion and/or relieving arterial spasms or vasoconstriction.⁵

CASE

A 65-YEAR-OLD CAUCASIAN, RETIRED office and factory maintenance worker, was admitted in casualty for sudden onset OF severe abdominal pain. This was initially colicky in nature, in the periumbilical area, but later became constant in the right iliac fossa. It was associated with nausea, vomiting, and sweating. In casualty he suddenly became hypotensive for which he received i.v. fluids, but then went into congestive heart failure, for which he was treated with i.v. frusemide. Five months previously he had a large anterior myocardial infarction and a multi-gated acquisition scan had indicated a poor left ventricular ejection fraction of 10% with a grossly dilated left ventricle. He also had a history of peripheral vascular disease manifesting as intermittent claudication and a retrograde aortogram had demonstrated a left external iliac stenosis with reconstitution via collaterals. He smoked 20 cigarettes a day until the myocardial infarction and was a mild alcohol drinker. He was on Frusemide, aspirin, and digoxin. On examination, that patient looked ill, pale, and clammy. He was pyrexial, his heart rate was 91 b.p.m. and regular, and he was hypotensive with a blood pressure of 90/60 mmHg. He had normal jugular venous pressure. Chest examination revealed mild rhonchi and bilateral fine basal crepitations. Heart examination revealed multifocal ventricular ectopics. Abdominal

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Fig. 1. Schematic diagram of anatomy of the superior mesenteric artery. Reproduced from Boley et al. (1969), with permission from the American Physiological Society.

examination revealed voluntary guarding and rebound tenderness in the right iliac fossa. Bowel sounds were very diminished. The hernia orifices were normal and rectal examination was unremarkable. An electrocardiogram showed sinus tachycardia with atrial ectopics. Full blood count and serum biochemistry were unremarkable except for low potassium of 3.2. Serial electrocardiograms and cardiac enzymes were normal. An erect chest X-ray showed pulmonary interstitial oedema and cardiomegaly. A supine abdominal X-ray and an ultrasound scan of the abdomen were unremarkable.

An emergency laparotomy for peritonitis revealed free fluid in the peritoneal cavity, gangrenous patches on the caecum, and an ischaemic area of the terminal ileum and caecum with bleeding at the edges. A right hemicolectomy was carried out and a right iliac fossa ileostomy and a mucous fistula fashioned. Immediate anastomosis was avoided because of the progressive nature of gangrene and the local oedema and friability at the ends.

The pathologist reported the receipt of 30 cm of terminal ileum and 19 cm of caecum and colon. No definite thrombi were identified and the site of the injury was limited to the caecum with a clear cut-off point. Sections of the terminal ileum did not show ischaemic changes. The first 6 days postoperatively were uneventful and the patient was commenced on total parenteral nutrition. On the seventh day he showed clinical signs of volume depletion and circulatory failure, i.e., dehydration, pale and cold peripheries, water-

depleted hypernatraemia, dyspnoea, and tachycardia. These were associated with a deteriorating renal function, indicated by decreased urine output, high creatinine and urea, and a rapid rise in serum potassium levels. The patient also showed signs of intra-abdominal sepsis, with a high white blood cell count (WBC, 24.7) with metabolic acidosis and hypoxia. He had large ileostomy fluid loss of 50–70 mL/h, or 1.2–1.7 L/day (normal, 700 mL/day) that continued for the next 5 days. There was protein-losing enteropathy with a constant hypoalbuminaemia of 25 despite parenteral nutrition. Blood cultures were negative. He was managed in the intensive care unit by slow rehydration with fluids under central venous pressure monitoring, a dopamine infusion, oxygen support, and i.v. broad spectrum antibiotics.

The second episode of major complications commenced on the 17th day following surgery when the patient showed marked ileostomy losses of 200 mL/h (4.8 L/day). This was manifested as salt-depleted hypovolaemia with a progressively impaired renal function and hyperkalaemia. There was leukocytosis of 42 with mild jaundice and pyrexia, an abnormal clotting screen, metabolic acidosis, and hypoalbuminaemia (serum albumin 23). Blood cultures were still negative. He was managed in the intensive care unit with a combination of i.v. gentamicin, metronidazole, flucloxacillin, and appropriate fluids. His ileostomy loss steeply increased for the next 7 days with a peak ileostomy loss of 6.4 L/day on day 25. His WBC count remained high within this period. The ileostomy loss gradually decreased to the normal range 14 days later paralleling a declining WBC count. As the ileostomy effluent became more semi-solid the serum biochemistry returned to normal. He was discharged on the 39th day after surgical resection of the ischaemic bowel and had lost 12 kg in weight.

DISCUSSION

THIS CASE ILLUSTRATES the risk of a high ileostomy output and its sequelae following intestinal resection and defunctioning for non-occlusive mesenteric ischaemia (NOMI). The high ileostomy output (1–6 L/day), occurring when only 30 cm of terminal ileum was resected, may have been due to partial ischaemia affecting the mucosal surfaces of the rest of the small intestine as it is being supported by the extensive arterial anastomosis and series of arcades of the left branches of the SMA (Fig. 1). The pathological effect of NOMI is greater on the right colon with the anatomically lower blood supply from the right of the SMA.^{3,4} The production of endotoxin and translocation of bacteria across the ischaemic gut mucosa induces third space loss and thus the sepsis-induced high ileostomy output. Culture of the ileostomy effluent may have excluded *Clostridium difficile*

and methicillin-resistant *Staphylococcus aureus*, which are common nosocomial causes of severe sepsis-induced high ileostomy output following prolonged antibiotic treatment.⁶ In a case report, high output ileostomy due to acute adrenal insufficiency precipitated by infection following bowel surgery responded rapidly to i.v. hydrocortisone.⁷

The specific problems encountered in this patient included the amount of parenteral fluid required to accommodate the volume and electrolyte loss without having an overload effect on the failing ischaemic heart. Neither oral glucose/saline solution nor the antimotility drug loperamide will be effective in the absence of functioning absorptive intestinal mucosa.^{3,8} Inhibitors of gastric acid secretion (i.v. histamine-2 antagonists or proton pump inhibitors) would not reduce stoma output enough to reduce the severity of intestinal failure and thus prevent the need for parenteral fluid and electrolyte replacement.^{3,8} However, in adults, the antisecretory somatostatin analogue octreotide (50 µg s.c. b.i.d.), although expensive and not easily available, reduces stomal output with a reduction of volume of parenteral supplements.⁸ A complication of the short bowel in this case, due to NOMI-associated loss of absorption, is disruption of the ileocolonic brake mechanism mediated by hormones such as glucagon-like peptide 1 and 2 and peptide YY, which leads to gastric hypersecretion, rapid gastric and intestinal transit, and poor intestinal adaptation. Clinical studies of their replacement with the peptide analogue teduglutide reduced parenteral fluid support.⁹ The necessary provision of parenteral nutrition in the hypercatabolic patient with temporary intestinal failure should be judicious to avoid pulmonary oedema and respiratory acidosis.¹⁰

In this patient, early closure of the stoma would have obviated the problems of high ileostomy loss with derivation of the benefits of a functional colon including energy from anaerobic bacterial fermentation of carbohydrate to short chain fatty acids.⁸ The prognosis would, however, still depend on how much small bowel is left and any other coexistent systemic disease.

CONCLUSIONS

SEPSIS-INDUCED LARGE VOLUME stoma output following intestinal resection with an end ileostomy for NOMI is a serious complication that may require intensive care support. Early restoration of intestinal continuity is important in obviating this complication.

Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the editor of this journal.

CONFLICT OF INTEREST

NONE.

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