Causes and management of a high-output stoma

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Abstract

Aim Patients with a high-output stoma (HOS) (> 2000 ml/day) suffer from dehydration, hypomagnesaemia and under-nutrition. This study aimed to determine the incidence, aetiology and outcome of HOS.

Method The number of stomas fashioned between 2002 and 2006 was determined. An early HOS was defined as occurring in hospital within 3 weeks of stoma formation and a late HOS was defined as occurring after discharge.

Results Six-hundred and eighty seven stomas were fashioned (456 ileostomy/jejunostomy and 231 colostomy). An early HOS occurred in 75 (16%) ileostomies/jejunostomies. Formation of a jejunostomy (defined as having less than 200 cm remaining of proximal small bowel; n = 20) and intra-abdominal sepsis/obstruction (n = 14) were the commonest causes identified for early HOS. It was possible to stop parenteral infusions in 53 (71%) patients treated with oral hypotonic fluid restriction, glucose-saline solution and anti diarrhoeal medication. In 46 (61%) patients, the HOS resolved and no drug treatment was needed, 20 (27%) patients continued treatment, six (8%) of whom went home and continued to receive parenteral or subcutaneous saline, and nine died. Twenty-six patients had late HOS. Eleven were admitted with renal impairment and four had intermittent small-bowel obstruction. Eight patients were given long-term subcutaneous or parenteral saline and two also received parenteral nutrition. All had hypomagnesaemia.

Conclusion Early high output from an ileostomy is common and although 49% resolved spontaneously, 51% needed ongoing medical treatment, usually because of a short small-bowel remnant.

Keywords Ileostomy, colostomy, intestinal failure, short bowel, magnesium, nutrition support

Introduction

The formation of a stoma is common, with approximately 20 800 being formed in England each year [1]. A new ileostomy usually starts to work within 24 h of formation and initially produces about 1200 ml of watery stool, which reduces in quantity and thickens over the next 2–3 months [2]. The output may vary from 500 to 2000 ml a day, depending on the volume of food and drink consumed and the volume of gastrointestinal secretions [3,4].

There is little information on high-output stoma (HOS) [1,5–7]. A recent meta-analysis on the complications of temporary ileostomy and colostomy identified only four studies which reported data on HOS [8]. In these, the incidence of HOS was reported to be approximately 3% but the criteria used to define HOS were not described and may vary considerably between gastrointestinal units as there is no agreed definition.

The effluent from a HOS is likely to become clinically significant when the daily output exceeds 2000 ml [9], causing water, sodium and magnesium depletion, with malnutrition occurring as a late complication.

Surgery that results in less than 200 cm of residual small bowel and no colon is likely to result in a HOS. Other causes of HOS include intra-abdominal sepsis, partial or intermittent bowel obstruction, enteritis (e.g. Clostridium difficile [10] or salmonella), sudden drug withdrawal (e.g. steroids or opiates), administration of prokinetic drugs (e.g. metoclopramide), recurrent disease in the remaining bowel (e.g. Crohn’s disease or irradiation bowel disease) [9] and bacterial overgrowth from diverticula or blind loop fermentation.
Management of a HOS includes the identification and treatment of any cause for the high output, reduction and replacement of water and electrolyte losses [initially via the intravenous (iv) route], oral hypotonic fluid restriction with the use of glucose/electrolyte solution \[9,11,12\], anti diarrhoeal \[13–15\] and anti secretory medication \[9,16–23\], nutritional support, wound care and psychological support.

The aim of this study was to determine the frequency, causes, and outcome of early and late high-output ileostomies in patients treated by the Nutrition Support Team (NST) at a university teaching hospital.

**Method**

Data on all patients referred to the NST for HOS management, between January 2002 and December 2006, were collected prospectively at Leicester Royal Infirmary. Adult patients were considered to have a HOS if they were referred with a stoma output of more than 2000 ml/24 h for three or more consecutive days (and were therefore likely to develop biochemical disturbance \[9\]). They received drug or oral treatment, including loperamide, glucose-electrolyte solution and hypotonic water restriction.

The patients were placed into one of the following two groups, depending upon presentation.

1. **Group A. Early HOS.** In this group, patients had developed a HOS in the immediate postoperative phase (< 3 weeks from stoma formation) and before discharge from hospital.

2. **Group B. Late HOS.** In this group, patients had developed a HOS following discharge from hospital, during which the stoma was formed.

Patients were assigned to only one of the two groups, and there was no cross-over between the groups. The patients were classified as having a colostomy, an ileostomy or a jejunostomy. A jejunostomy was defined as having less than 200 cm of proximal remaining small bowel.

**Incidence of HOS**

The number of early HOS identified was compared with the total number of new stomas fashioned between January 2002 and December 2006. The data were obtained from the hospital clinical coding records using Office of Population, Censuses and Surveys Classification of Surgical Operations and Procedures (OPCS) Version 4.4 (April 2007). HOS patients were prospectively identified by the NST and incidence data were validated by reconciliation with stoma care nursing records.

**Cause, treatment and outcome of HOS**

The patient’s age, gender, diagnosis, type of stoma, cause and subsequent management were recorded. The standard management of HOS, in accordance with current British Society of Gastroenterology (BSG) guidelines \[9\], consisted of initial rehydration with iv saline, hypotonic oral fluid restriction (500–1000 ml/day), and treatment with glucose-electrolyte solution (3.5 g of sodium chloride, 2.5 g of sodium bicarbonate and 20 g of glucose made up to 1000 ml with tap water) and drugs (loperamide initially 2–4 mg q.i.d., half an hour before food, and esomepazole 40 mg o.d.).

**Results**

In the 5-year study period, 687 stomas were created, of which 456 (66%) were ileostomies (Fig. 1).

**Group A. Early early HOS**

**Incidence**

Seventy-five (16%) small bowel stomas were high output, and all occurred within 3 weeks of formation. Formation of a colostomy did not result in any patient having an early HOS. The median age of patients with an early HOS was 63 (range 17–88) years with a predominance of men (n = 45). The commonest indication for formation of a stoma was surgery for colorectal cancer (45%) followed by surgery for inflammatory bowel disease (Crohn’s disease, n = 6; and ulcerative colitis, n = 5) (Table 1). Fifteen (20%) of early small bowel stomas were created during emergency surgery for bowel perforation.

**Causes of early HOS**

In 37 patients, no cause for early HOS was found. Six (16%) of these patients had undergone emergency surgery for bowel perforation. There was at least one identifiable cause for early HOS in the remaining 50% of patients (Table 2); in 20 (27%) patients it was attributable to the formation of a jejunostomy.
Computed tomography (CT) identified an intestinal obstruction in eight patients, of whom, six had adhesive small bowel obstruction: that settled with conservative management. The remaining two patients required laparotomy to relieve intestinal obstruction: one for small bowel volvulus with an internal hernia and one for adhesive obstruction. In seven patients, HOS was associated with intra-abdominal or pelvic sepsis (however, there were multiple causes for a HOS in four of these patients). Management was by percutaneous drainage combined with antibiotic treatment, as indicated. One patient required surgical intervention for intra-abdominal sepsis with resection of perforated small bowel.

In five patients, early HOS was related to medication. In two patients, HOS was associated with metoclopramide treatment, which resolved following discontinuation of the drug. In one patient who had undergone a colectomy for ulcerative colitis, HOS resulted from the sudden cessation of steroids in the postoperative period. Stoma output resolved following treatment with iv hydrocortisone. Another patient had HOS related to both steroid withdrawal and a pelvic abscess, which was successfully treated with a combination of steroids and antibiotics. A further patient developed HOS during early administration of postoperative adjuvant chemotherapy; in this patient, the stoma remained high output until completion of chemotherapy.

Two patients developed postoperative enteric Clostridium difficile infection that resulted in a daily stoma output in excess of 5000 ml [10]. Both patients were treated with oral metronidazole. In one, the stoma output normalized within 4 days and in the other (who had less than 200 cm of small bowel) there was a significant decrease in stoma effluent.

Management of early HOS
In patients with an identified cause for HOS, management was directed at treating the diverse underlying causes, as detailed above. In addition, all 75 patients with HOS were given treatment conforming to the NST protocol (shown in Fig. 2). In accordance with the protocol, the use of octreotide was not widespread, only 10 patients with excessive stoma outputs (> 4000 ml/day) or less than 150 cm of residual small bowel (protocol stage 3) received octreotide, with 8 of the 10 patients also initially receiving parenteral nutrition whilst nil by mouth. A total of 28 (37%) patients required artificial nutritional support: 19 patients received parenteral nutrition, and nine patients received enteral feeding via a nasogastric tube (using polymeric feed with additional sodium).

Before referral to the NST, all patients were receiving parenteral fluids. The HOS protocol resulted in the discontinuation of parenteral fluids in 53 (71%) patients (Fig. 2). Three of six inpatients required iv fluids and remained in hospital until the stoma was reversed. Of the 10 outpatients requiring long-term iv fluid replacement, four were initially discharged without iv support but all required readmission for treatment with parenteral fluids/nutrition. Two (9%) patients required iv saline and magnesium replacement at least weekly. One patient was established on home parenteral nutrition and two patients required regular treatment with iv saline administered in hospital as a day-case procedure. A palliative oncology patient was discharged for treatment at home with subcutaneous water and electrolytes. All 10 patients who had an ongoing requirement for parenteral fluids had residual small bowel lengths of less than 200 cm (Fig. 3). Parenteral infusion was required in 62.5% of patients with less than 200 cm of small bowel.

In 34 (45%) patients, HOS was associated with hypomagnesaemia. Twenty-six (35%) patients received oral supplementation with magnesium oxide or magne-

| Table 1 Indication for stoma formation in patients with high-output stomas (HOS). |
|-----------------------------|-----------------------------|
| Diagnosis                  | Early HOS       | Late HOS       |
|                            | n (%)           | n (%)          |
| Cancer                     | 34 (45)         | 6 (23)         |
| Perforation                | 15 (20)         | 3 (12)         |
| Inflammatory bowel disease | 11 (15)         | 15 (57)        |
| Ischaemic bowel            | 8 (11)          | 0              |
| Familial polyposis coli    | 4 (6)           | 0              |
| Postoperative bleeding     | 1 (1)           | 0              |
| Defunctioned for incontinence | 1 (1)       | 0              |
| Defunctioned for pressure sore | 0            | 1 (4)         |
| Adhesive obstruction       | 1 (1)           | 1 (4)         |

| Table 2 Causes of high-output stomas (HOS). |
|-----------------------------|-----------------------------|
|                            | Early HOS       | Late HOS       |
|                            | n (%)           | n (%)          |
| Multiple causes            |                |                |
| Abdominal sepsis and short bowel | 3 (4)  | 0          |
| Enteric infection and short bowel | 1 (1)  | 0          |
| Pelvic abscess and steroid withdrawal | 1 (1)  | 0          |
| Single cause               |                |                |
| Short bowel (< 200 cm)     | 16 (22)         | 3 (12)         |
| Obstruction                | 8 (11)          | 8 (31)         |
| Medication                 | 4 (5)           | 4 (15)         |
| Intra-abdominal sepsis     | 3 (4)           | 0              |
| Enteric infection          | 1 (1)           | 0              |
| Ongoing IBD                | 1 (1)           | 1 (4)         |
| No cause identified        | 37 (50)         | 10 (38)        |

*IBD, inflammatory bowel diseases.
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STAGE 1: Exclude Potential Causes

- Rule out intra-abdominal sepsis and intermittent bowel obstruction.
- Could medication be contributing to the HOS? Consider prokinetics, withdrawal of steroids.
- Does the patient have an enteric infection? Exclude Clostridium difficile by stool toxin analysis.

STAGE 2: Initial management – Reduce fluid and electrolyte losses

- Restrict ORAL FLUIDS to 500 ml daily (Rehydrate patient with intravenous saline).
- Commence loperamide 4 mg QDS to reduce stoma losses. This should be given 30–60 min before meals and at bedtime.
- Monitor strict fluid balance, daily weights, and serum biochemistry, including magnesium levels.
- Screen for under nutrition (including BMI, % weight loss and current or expected oral intake) and refer to dietician as appropriate.
- Review stoma output after 48–72 h – if settles increase oral fluid intake.

STAGE 2: Ongoing HOS – optimise treatment with anti-secretory / diarrhoeal medication

- Continue oral fluid restriction. (If stoma output is >3000 ml/day consider placing the patient NBM for 24 h to assess gastrointestinal secretion).
- Commence St.Marks or WHO glucose-electrolyte replacement solution 1000 ml daily, orally, in addition to oral fluid restriction. Once IV fluids stopped check random urine sodium (aim >20 mmol/l).
- Increase loperamide dose to 8 mg QDS.
- Review proton-pump inhibitors. Initiate or change to omeprazole 40 mg OD-BD to reduce volume of gastric secretions.
- Continue strict monitoring (fluid balance charts, twice weekly weights, weekly magnesium levels). If serum magnesium <0.5 mmol/l give 12–16 mmol of Magnesium sulphate IV in 0.9% NaCl. Begin oral supplements (Magnesium oxide capsules 3 x 4 mmol/nocte).

STAGE 3: Evaluate efficacy of additional treatment if HOS continues

- Refer to Nutrition Support Team for further advice

Table 3 Outcome of high-output stomas (HOS).

<table>
<thead>
<tr>
<th></th>
<th>Early HOS n (%)</th>
<th>Late HOS n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>During inpatient admission</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Resolved within 2 weeks*</td>
<td>37 (49)</td>
<td>4 (15)</td>
</tr>
<tr>
<td>Ongoing until stoma reversed</td>
<td>6 (8)</td>
<td>4 (15)</td>
</tr>
<tr>
<td>Died</td>
<td>6 (8)</td>
<td>0</td>
</tr>
<tr>
<td>Ongoing treatment postdischarge</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Resolved – HOS treatment discontinued</td>
<td>9 (12)</td>
<td>0</td>
</tr>
<tr>
<td>Ongoing until stoma reversed</td>
<td>9 (12)</td>
<td>4 (15)</td>
</tr>
<tr>
<td>Refashioning of obstructed stoma</td>
<td>0</td>
<td>1 (4)</td>
</tr>
<tr>
<td>Ongoing</td>
<td>5 (7)</td>
<td>12 (47)</td>
</tr>
<tr>
<td>Died</td>
<td>3 (4)</td>
<td>0</td>
</tr>
<tr>
<td>Not known</td>
<td>0</td>
<td>1 (4)</td>
</tr>
</tbody>
</table>

*HOS treatment discontinued.

Figure 2 Management protocol for a high-output stoma (HOS).

Figure 3 Outcome of early high-output stoma (HOS): *, Resolved within 2 weeks; †, 10 patients were treated with parenteral/subcutaneous fluids and magnesium; ‡, six patients died during admission for treatment of sepsis.

**sium** glycopophosphate, and 19 (25%) required iv supplementation with magnesium sulphate. Fifteen (20%) patients required long-term supplementation with magnesium and during follow-up after discharge from hospital. Eleven (73%) patients with ongoing hypomagnesaemia had a residual small bowel length of less than
200 cm. The incidence of long-term hypomagnesaemia in patients with a short small intestine was 69%.

**Outcome of early HOS**
The outcome of patients with HOS is shown in Table 3. In 49%, the early HOS resolved during the inpatient admission, allowing treatment to be discontinued. HOS resolved in a further nine patients following discharge from hospital. Fifteen patients continued to have high effluent losses until the stoma was reversed. Overall, five patients with early HOS had late high effluent losses that required long-term nutritional support. Six patients with HOS died in hospital, all from multiorgan failure caused by severe sepsis.

**Group B. Late HOS**
A late HOS was identified in 26 patients [14 women; median age 61 (range 21–89) years] (Table 1). In all patients, the stoma had been formed more than 3 months previously (range 3 months to 26 years). Inflammatory bowel disease was the most common diagnosis [15 (6%) patients; nine with Crohn’s disease and six with ulcerative colitis] followed by colorectal cancer [six (2%) patients]. Twenty-three (88%) patients had a small bowel stoma, including ileostomy (21 patients; 12 end and nine loop ileostomies) and jejunostomy (two patients). A further three patients had a high-output colostomy, two of whom had jeuno-colic anastomoses with only 105 and 180 cm of jejunum.

Eleven (44%) patients presented with renal impairment secondary to a late HOS. Before assessment by the NST, 10 patients had been inappropriately advised to increase their oral fluid intake. None of these patients received oral fluid resuscitation with a glucose-electrolyte solution until this was recommended by the NST.

The underlying causes and outcome of late HOS are shown in Tables 2 and 3. In four (15%) patients, stoma output settled within 3 days of receiving parenteral fluids. A further four (15%) patients had symptoms of subacute bowel obstruction and a HOS that resolved following reduced intake of dietary fibre. Seventeen (66%) patients had a persistent HOS. Five were treated surgically, including closure of the ileostomy in four. The remaining 12 patients required follow up in the community for ongoing HOS and were managed in accordance with the algorithm shown in Fig. 2.

Three patients required treatment at home with parenteral nutrition or with saline and magnesium. Five patients were repeatedly re-admitted for treatment with iv water and electrolytes. In one patient, this was managed by giving twice-weekly saline and magnesium infusions as a day-case procedure. Another patient, who was stable during treatment for HOS, was re-admitted to hospital in acute renal failure after high-dose loperamide and omeprazole therapy had been stopped.

**Discussion**
This is the first study to show that 16% of small bowel stoma patients have a HOS within 3 weeks of stoma formation. The cause for this is often unclear, although intra-abdominal sepsis, medication, intermittent obstruction and reduced small bowel length (< 200 cm) account for 50% of the instances of HOS. Radiological examination is helpful as it may show the length of the remaining small bowel and possibly signs of obstruction, diverticula, active mucosal disease and entero-enteral fistula formation. If present, treatment of incomplete bowel obstruction or intra-abdominal sepsis will result in resolution of the high-output state. In this series, 20% of early HOS remained high until surgical closure of the stoma. Although the length of remaining small bowel was not documented, it is likely that a significant proportion of these HOS were jejunostomies.

Half of early HOS were transient postoperative episodes that resolved within 2 weeks of surgery. Some of these may have been caused by postoperative overload with iv saline solution, which reduces gastrointestinal motility and increases postoperative complications [24,25]. Most HOS can be managed without parenteral water and electrolytes. The most important factor in the management of HOS is oral restriction of hypotonic liquids. Unfortunately, this is likely to be associated with poor patient compliance but it is also the case that some patients are wrongly advised to increase their overall liquid intake when the stoma output is high. Before the formation of the NST, all patients in our teaching hospital with HOS were inappropriately advised to increase their intake of oral liquid. The consumption of hypotonic oral liquid low in sodium (e.g. tea, coffee and squash) results in a net efflux of sodium into the intestinal lumen, leading to sodium depletion [11,21,26]. It has been shown that most patients can manage without parenteral liquids and electrolytes if oral intake is restricted to 500–1000 mL/24 h of a glucose-electrolyte solution containing at least 90 mmol/l of sodium [12]. In the present study, 71% of patients with early postoperative HOS and 65% of patients with late HOS were managed using oral glucose-electrolyte solutions without the need for iv infusions. Education of healthcare professionals on oral water and electrolyte management is essential for the management of these patients without resorting to iv fluids.
Water and electrolyte management may be facilitated by medication to reduce motility, such as loperamide, which decreases ileostomy water and sodium output by about 20–30% [14,15]. Opiates, such as codeine phosphate, are sometimes beneficial in addition to loperamide. Both drugs should be given half an hour before meals to slow gut transit. The routine use of octreotide for HOS is not considered necessary because drugs to reduce gastric acid secretion (proton pump inhibitors such as omeprazole) reduce stoma output as effectively as octreotide [19,27].

Nearly half (45%) of patients presenting with an early HOS will have hypomagnesaemia. This may be caused by secondary hyperaldosteronism, loss of absorptive area or unabsorbed fatty acids forming soaps within the gut lumen. Oral supplementation can be given in the form of magnesium oxide [9]. Sixty-nine per cent of patients with documented evidence of less than 200 cm of residual small bowel require long-term treatment with magnesium.

Artificial nutritional support may be required in up to a third of patients with early HOS, to provide adequate and balanced nutrition to maintain nutritional status. This needs to be achieved without exacerbating stoma water and electrolyte losses. Solutions of 300 mOsm/l should be used, and additional sodium chloride can be added to increase the sodium concentration and regulate the sodium balance [28]. However, long-term parenteral nutrition and water and electrolyte support is sometimes required, and close monitoring is necessary to prevent complications.

The objective in management of late HOS is to minimize the morbidity and associated renal and electrolyte disturbances whilst maintaining quality of life. This may be achieved by dedicated healthcare professionals establishing close contact with general practitioners. Patients with late HOS can be successfully managed at home by restricting the volume of oral hypotonic liquids and by the use of glucose-electrolyte solutions. However, this requires high patient compliance, and healthcare professionals should be mindful of the need for rapid intravenous therapy when compliance is poor. A few patients in this clinical group will require regular intravenous nutrition, and water, and electrolyte supplements, but they can still be managed as an outpatient with domiciliary support. The aim should be to maintain a steady weight, a urinary output of > 800 ml/24 h, normal levels of serum creatinine and magnesium, and a random urine sodium concentration of > 20 mmol/l.

Conclusions

Early HOS is more common than previously recognized. In half of the cases, however, it is transient, and in 50% of patients an underlying cause will be apparent. HOS guidelines can direct the immediate and longer-term care of patients and ensure that a suitable management plan is implemented to reduce stoma output and its metabolic sequelae.

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References


