BAPEN
Putting patients at the centre of good nutritional care

Managing a High Output Stoma

Jeremy Nightingale and the BIFA Committee

A high output stoma (HOS) is when the stoma output causes the patient to become water, sodium and magnesium depleted. This tends to occur when the output is more than 2 L / 24 hours though this varies according to the amount of food/drink taken orally (if 4 L is consumed a 2 L output may not be a problem but if only 0.5 L is consumed dehydration will follow). This occurs at some time in up to 30% of small bowel stomas. It rarely occurs if there is more than half of the colon in continuity with the small intestine.

Key points

1. Ensure good skin protection/bag fitting.
2. Exclude causes other than a short bowel (e.g. obstruction).
3. Rehydrate and stop thirst.
4. Restrict oral hypotonic fluid.
5. Sip a glucose/salt solution +/- magnesium supplements.
6. Commence loperamide (high dose) before food.
7. Commence a proton pump inhibitor (especially if a net 'secretory output').
8. Consider reducing oral insoluble fibre intake.
9. Consider subcutaneous fluid before resorting to parenteral fluids.
10. Monitor random urinary sodium concentration and serum magnesium.
11. If an oral/enteral supplement is needed due to malnutrition, the osmolarity aims to be near to 300 mOsm/L and the sodium concentration 100-140 mmol/L.
12. Patients who need admission for rehydration should not just be rehydrated and discharged.
13. Patients with a short bowel (less than 2 m remaining) needing parenteral support may be considered for a glucagon-like peptide-2 analogue.

Explanations

1. A high stoma output can cause a stoma bag to become detached causing sore peri-stomal skin.

2. While a short bowel (less than 200 cm jejunum remaining) is the most common reason for a HOS (occurring more than 3 weeks after formation), other easily treatable causes should be sought. Intermittent or partial obstruction is common. A history of sudden onset colicky abdominal pain, borborygmi, swelling/visible peristalsis and temporary stopping of the stoma are suggestive. The HOS tends to occur as the obstruction resolves. A stenosis at or near to the stoma is common and may be detected by visual inspection or inserting a finger into the stoma. A low fibre diet may help, but often a surgical resection is needed. Other causes include opioid or steroid withdrawal or the giving of prokinetic drugs (e.g. metoclopramide). In the perioperative period (less than 3 weeks after formation) and especially if the output has changed to becoming high, abdominal infection (with low albumin, raised CRP and neutrophils, and an ileus) may be associated and infection should be actively sought (CT abdomen). Other causes include small bowel diverticula (bacterial overgrowth), an ischaemic segment of small bowel (often associated with hypoalbuminaemia), recurrent bowel disease (e.g. Crohn's disease), coeliac disease, hyperthyroidism or clostridium difficile infection.

3. Rehydrating and stopping thirst may be done acutely by giving intravenous saline (e.g. 0.9% saline), while taking little or no oral intake (be conscious to monitor and time limit so patients do not experience prolonged restrictions on oral intake). The sodium concentration of small bowel stomal fluid is always (whatever the oral intake) about 100 mmol/l (range 80-140). Daily weight and fluid balance charts will help with monitoring; bag leaks may cause the losses to be underestimated.

4. Hypotonic fluid (e.g. water) causes a net efflux of sodium into the bowel lumen and this is lost through the stoma. Patients must restrict oral hypotonic fluid (e.g. to 0.5 – 1.0 L / 24 hrs). They must not be told to "drink as much as possible". In addition, they should not take hypertonic fluid (e.g. elemental diet) as this will cause a net flow of fluid into the bowel lumen (together with sodium) exacerbating water and sodium depletion.

5. An oral rehydration solution is sipped (e.g. St Mark's modified WHO cholera solution, double strength Dioralyte®) (1 L / 24 hrs). The glucose is absorbed with sodium. Rarely oral sodium chloride tablets may be taken. Oral magnesium supplementation may be needed but correcting sodium depletion alone may be adequate.

6. Loperamide up to 16 mg qds 30 - 60 minutes before food may reduce transit and increase absorption. Care is needed with higher doses (more than 4 mg four times a day) due to possible cardiac arrhythmias. The addition of codeine phosphate 30-60 mg four times a day may further help and reduce output (but does cause sedation and is addictive). If the stomal output increases with anti-diarrhoeal drugs, consider a partial obstruction.
7. A proton pump inhibitor (e.g. omeprazole 40 mg daily), will reduce output in 'net secretors' (those whose output is greater than their oral intake). As proton pump inhibitors may cause hypomagnesaemia, osteoporosis and cardiac arrhythmias they should not be given routinely and certainly stopped if no effect. The dose of omeprazole can be titrated upwards so that the stomal pH exceeds 5. Octreotide is rarely recommended due to a painful injection, gallstones and worry about its effect in inhibiting bowel growth hormones. It is equivalent to omeprazole in the amount to which it reduces the volume of stomal output in net 'secretors'.
8. While insoluble fibre increases the rate of colonic transit, its effect on small bowel transit time is unclear. It has been used to thicken ileostomy output. However, as is common, a small bowel stricture (often due to adhesions) may be present, and by causing partial intestinal obstruction (which can be painless) it can increase the stomal output; hence a low fibre intake is commonly recommended. If patients are malnourished or there is concern about the nutritional balance of their diet, they may benefit from referral to a dietitian with expertise in gastroenterology and surgical nutrition for a more thorough dietetic assessment and individualised advice.
9. If a patient still becomes dehydrated on maximal therapy, subcutaneous saline – 0.9% NaCl with up to 8 mmol/L magnesium, usually 1-4 times a week – may be considered before intravenous saline (0.9% NaCl) supplementation.
10. A patient who is sodium depleted will have high aldosterone concentrations which causes sodium reabsorption in the kidney at the expense of magnesium and potassium which are excreted. Thus, a random urine sodium concentration (of less than 20 mmol/L) indicates sodium depletion due to secondary hyperaldosteronism; treatment aims for a urine sodium concentration of greater than 20 mmol/L. If the kidney has been damaged or the patient takes a diuretic then the urine sodium concentration may not be helpful. Magnesium depletion, although often asymptomatic, is common and treated with correcting hyperaldosteronism, oral magnesium supplements, reducing fat in the diet (unless the patient is malnourished) and 1-alpha-hydroxy-cholecalciferol (Alfacalcidol). A third of serum magnesium is bound to albumin so concentrations will appear low in the presence of hypoalbuminaemia.
11. Unless the bowel is very short or there is concurrent disease, malnutrition is not usually a major problem. However, if an oral nutritional supplement is needed then a polymeric one is preferred ideally with added salt. An elemental diet is hyperosmolar so can so can exacerbate salt and water losses so should be avoided.
12. Patients admitted with a HOS should not only be rehydrated but also fully assessed for the aetiology of the HOS and preventative measures/treatments given. Generally, a patient admitted with a HOS will need follow up appointments.
13. A glucagon-like peptide-2 analogue (e.g. Teduglutide) to mainly stimulate upper gut mucosal growth may be considered in those dependent upon parenteral support, but needs to be given by a recognised home parenteral nutrition unit or integrated intestinal failure centre, in accordance with UK NICE guidance.

Suggested reading:

- Nightingale JMD (2021). How to manage a high-output stoma. Frontline Gastroenterol.; 13(2): 140-151.
- British Intestinal Failure Alliance (BIFA) (2018). Position Statement: The use of high dose loperamide in patients with intestinal failure. Available online: www.bapen.org.uk/pdfs/bifa/position-statements/use-of-loperamide-in-patients-with-intestinal-failure.pdf (Mar 2023)
- British Intestinal Failure Alliance (BIFA) (2018). Position Statement: Use of peptide growth factors for adult patients with intestinal failure. Available online: www.bapen.org.uk/pdfs/bifa/position-statements/use-of-peptide-growth-factors-for-adult-patients-with-intestinal-failure.pdf (Mar 2023)
- British Intestinal Failure Alliance (BIFA) (2022). Managing Crohn's Disease in Intestinal Failure. BIFA Top Tips Article Series. Available online: www.bapen.org.uk/pdfs/bifa/bifa-top-tips-series-20.pdf (Mar 2023)
- British Intestinal Failure Alliance (BIFA) (2021). Nutritional and fluid assessment of patients with intestinal failure. BIFA Top Tips Article Series. Available online: www.bapen.org.uk/pdfs/bifa/bifa-top-tips-series-16.pdf (Mar 2023)
- Mitchell A, England C, Perry R, *et al.* (2021). Dietary management for people with an ileostomy: a scoping review. JBI evid synth.; 19(9): 2188-2306.
- NICE technology appraisal guidance [TA804]. Teduglutide for treating short bowel syndrome. 30 June 2022.