CASE REPORT

A systematic approach in the management of a high output ileostomy resulting in a favorable clinical outcome

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High output ileostomy is not uncommon and when diagnosed, it should be managed expeditiously and systematically in order to prevent complications such as dehydration, electrolyte disturbance and acute kidney injury.

A multi modal regimen consisting of correction of fluid and electrolyte deficit; restriction of oral hypotonic fluids with dietary modifications and use of anti-motility drugs was sequentially and concomitantly introduced to achieve a favourable clinical outcome. Patient education regarding dietary modifications and recognition of dehydration due to high output stoma is paramount. A co-ordinated multi-disciplinary approach involving the patient, family, dietician, community nurses and hospital doctor is vital.

INTRODUCTION

High output stoma is encountered with surgically created stomas such as ileostomy, jejunostomy and colostomy. Different studies have defined a high ileostomy output as more than 1500 mL to 2000 mL per day with signs and symptoms of dehydration [1-3]. Studies have indicated that almost 16% of patients develop a high output stoma, of which 27% need to be managed conventionally [1]. This results in fluid and electrolyte imbalance culminating in a state of dehydration and acute kidney injury. Current management guidelines for high-output stomas focus on supportive measures and medications that decrease bowel motility [2]. However, response to therapy is often variable and the plan needs to be modified based on the initial fluid status, electrolyte deficit, severity of dyselectrolytemia and response to anti motility agents [3].

This case highlights the management of a high output stoma which developed following emergency laparotomy with de functioning ileostomy for a contained anastomotic leak in a patient with recent anterior resection for carcinoma sigmoid colon.

The high output ileostomy resulted in the patient developing dehydration and intractable symptomatic hyponatremia which required correction with hypertonic saline infusion and St. Mark's oral solution modified with an increased salt content.

A combination of fluid resuscitation, correction of dyselectrolytemia, anti-motility drug therapy and adherence to a dietary regimen modified for high stoma output contributed to the successful management of this case. The patient was discharged home with a patient information leaflet educating him on the maintenance of his dietary regimen and recognition of signs and symptoms of dehydration.

CASE REPORT

A 75 year old gentleman with a history of hypertension and diabetes mellitus underwent an open anterior resection for carcinoma sigmoid colon with colorectal anastomosis under general anesthesia and invasive hemodynamic monitoring. Post-operatively patient had an uneventful recovery.

On the seventh day, he developed colicky abdominal pain with distension and vomiting. A Computerised Tomography of Abdomen and Pelvis revealed a collection around the anastomotic site and intestinal obstruction.

Patient underwent an emergency laparotomy under general anesthesia and invasive hemodynamic monitoring, which revealed a contained anastomotic leak and defunctioning ileostomy was performed. Post-operatively, total parenteral Nutrition (TPN) was initiated to provide nutrition. Oral feeds were initiated once the ileostomy started functioning. TPN was tapered and stopped prior to discharge to the ward.

Patient was readmitted to the ICU after a week with drowsiness, serum sodium of 118 mM/L and an ileostomy output more than 1500 mL for two consecutive days. Arterial and Central venous access was established for volume and electrolyte status assessment and correction. Sodium deficit was calculated at 504 mM. Three-fourth of this was corrected with 3% sodium chloride and one-fourth with rehydration using normal saline (0.9%) with an aim to raise sodium by not more than 0.5 mM/L/h. Sodium level increased to 130 mM/L over a period of 24 h, following which 3% sodium chloride was discontinued and normal saline was continued as maintenance fluid. Patient was conscious and alert. On evaluating the cause of hyponatremia, the serum osmolality was 265 mOsm/kg, urine osmolality was 634 mOsm/kg and urine sodium was 21 mM/L. A diagnosis of hypovolemic hypo-osmotic hyponatremia secondary to high output ileostomy was made. Other investigations such as serum TSH, lipid profile, serum cortisol and blood glucose were within normal limits.

Since stool for clostridium difficile toxin was negative, the patient was started on loperamide. Ileostomy output marginally decreased but the patient developed mild drowsiness and serum sodium drifted to 126 mM/L over next twelve hours. Correction with hypertonic saline was restarted and the serum sodium stabilized at 134 mM/L over following 24 h. This drop in sodium was attributed to persistent high ileostomy output. The dose of loperamide was escalated, pantoprazole was increased to twice daily and oral fluids were restricted to 1.5 L/day. The patient was started on St.Mark's oral solution modified with an increased salt content to five grams/day. The salt content was titrated based on daily sodium levels and stoma output. The patient remained asymptomatic. Sodium level stabilized at 130 mM/L and ileostomy output at less than 700 mL/day. He required magnesium and potassium replacement concomitantly.

He was discharged home with modifications in his diet to regulate stomal output and restrict fluids to 1.5 L/day. He was educated to titrate the dose of loperamide according to ileostomy output and was advised to check his serum sodium every 72 h for the first fortnight. He was asked to contact the hospital if he experienced symptoms of dehydration or if serum sodium dropped to less than 130 mM/L.

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On follow-up, patient is asymptomatic to date with a serum sodium ranging from 130 to 134 mM/L and a stable ileostomy output. Patient is awaiting ileostomy reversal.

DISCUSSION

Seven to eight litres of fluid enters the upper gut every day and most of it is reabsorbed in the jejunum and ileum. About 1-1.5 L enters the colon from the ileum and 150 to 200 ml is excreted as stool [4].

When there is an ileostomy, the output depends on the length of the small bowel proximal to the stoma [3].

The proximal bowel adapts to the fluid and electrolyte losses of the stoma. After a period of adaptation, the loss of electrolytes is reduced by about 70% and the output decreases to an average of 750 ml/day (10-15 mL kg^{-1} day^{-1}) [2].

A high output stoma is defined as stomal output >l.5 L/day which subsequently causes dehydration and dyselectrolytemia [1]. In the first three weeks following surgery, almost 16% of patients with a small bowel stoma have problems with high stomal output and 27% of these require long-term treatment [4].

The effluent characteristics of an ileostomy are between normal ileal and fecal content. There is fluid and electrolyte loss as the small bowel is unable to conserve sodium, chloride and bicarbonate leading to dehydration, hyponatremia and metabolic acidosis. Long standing ileostomies also have hypomagnesemia and decreased absorption of vitamin B12 and folic acid. The psychological and practical difficulties of managing a high-output stoma should also be taken into account [5].

Common causes of a high output stoma include extensive bowel resection (secondary to Crohn's disease, bowel ischaemia or malignancies), chronic impairment of bowel function (radiation enteritis, dysmotility disorders), steroid withdrawal (following surgery for inflammatory bowel) and clostridium difficile infection [6].

This patient had a defunctioning ileostomy for a contained anastomotic leak which later on presented as a high-output stoma of more than 1.5 L/day for two consecutive days with dehydration and dyselectrolytemia.

Invasive hemodynamic monitoring was instituted by establishing an arterial and central venous access for assessment and correction of the initial volume and electrolyte status.

A step wise approach was employed to evaluate the cause of hyponatremia. The serum osmolality was measured to be 265 mOsm/kg and was less the normal range (275-295 mOsm/kg) which indicated a diagnosis of hypo-osmotic hyponatremia. Following this, the urine osmolality was sampled and measured at 634 mOsm/kg. As the urine osmolality was high (>100 mOsm/kg), the next step involved was to sample for the urinary sodium. The urinary sodium was measured to be 21 mM/L. Thus, a diagnosis of hypovolemic hypo-osmotic hyponatremia secondary to high output ileostomy was made.

The initial treatment involved replacement of the fluid and electrolyte deficit. Dehydration due to ileostomy loss was corrected by intravenous normal saline. The sodium deficit was calculated to be 504mmol by using the formula, Sodium deficit=Total Body Water X (Desired serum sodium-Measured serum sodium). This sodium deficit was corrected with with an aim to raise sodium by not more than 0.5 mM/L/h to avoid the complications associated with rapid correction of hyponatremia such as central demyelination. Symptomatic hyponatremia of 118mmol/L was corrected with 3% hypertonic saline over 24 h and serum sodium level was monitored sixth hourly. Serum sodium increased to 130 mM/L over 24 h, following which 3% sodium chloride was discontinued and normal saline was continued as maintenance fluid. Anti-motility drug loperamide was initiated once clostridium difficile toxin was negative to rule out clostridium infection which can result in a high output stoma [6].

Despite initial correction of the sodium deficit, he later on developed symptomatic hyponatremia of 126 mM/L over the next twelve hours. This was attributed to the continued high stomal output. Correction with 3% hypertonic saline was restarted and continued over the following 24 h. Loperamide dose was escalated and pantoprazole was increased to twice daily. The replacement with 3% hypertonic saline was weaned off once sodium level stabilized at 134 mM/L Further on-going sodium loss was replaced enterally with a modified St. Mark's solution.

We modified the St. Mark's solution [7] by increasing the salt content to 5 g/day with 30 g of glucose and 2 g of sodium bicarbonate in 1L solution. The patient was started on a dietary regimen to regulate stomal output and fluid intake was restricted to 1.5 L/day. The salt content was subsequently titrated based on daily serum sodium results and stomal output. Once the sodium level stabilized, standard St.Mark's solution was continued for replacement of sodium loses from the stoma.

St Mark's solution is a glucose-electrolyte mix which contains 90 mmol/L of sodium. This is formulated with 20 g of glucose, 2.5 g of sodium bicarbonate and 3.5 g of salt dissolved in one litre of water. Glucose molecules facilitates the absorption of sodium through the intestinal wall by co-transport [7].

Isotonic fluid replacement is recommended to compensate for fluid loss via the stoma to maintain fluid balance. As a rule, hypotonic fluid intake is to be avoided and nonelectrolyte fluid intake restricted to 1-1.5L [3].

The subsequent strategy employed was to limit the GI secretions. The patient was started on pantoprazole 40 mg twice daily following the diagnosis of high output ileostomy. It is recommended that proton-pump inhibitors should be initiated to reduce volume of gastric secretions and stomal volume. This can result in up to 15% reduction in stomal output [6].

As the ileostomy output remained high, an anti-diarrhoeal drug loperamide was initiated once the clostridium toxin assay was negative. Loperamide was titrated according to the ileostomy output and doses up to 16 mg/day was used.

Loperamide, an opiate analogue acts on the μ receptors in the intestinal wall, thereby reducing peristalsis and increasing transit time. It is preferred as it is neither sedating nor addictive, unlike other opioid receptor agonists such as codeine phosphate and diphenoxylate-atropine [6].

A cumulative dose of 16 mg/day of loperamide is generally accepted as the conventional maximum dose [6]. However, studies have advocated individual doses of loperamide, as high as 32 mg, to achieve an antidiarrhoeal effect [8,9].

Loperamide is well tolerated with few side effects like nausea, abdominal cramps and dry mouth [5].

If stoma output remains >2000ML daily after 2 weeks of therapy, studies have recommended that octreotide 200 mcg thrice daily for 3-5 days be initiated [10]. Octreotide and its analogues inhibits release of gastrin, cholecystokinin, secretin, motilin and other GI hormones. This decreases the secretion of bicarbonate, water and enzymes thus decreasing the volume of intestinal secretions. It also relaxes intestinal smooth muscle and increases intestinal water and electrolyte absorption [10].

Patient education plays an important role in management of a high output stoma. Patient information leaflet was provided regarding diet that might affect the stomal output. Information was provided on the recognition of dehydration and malnutrition. He was asked to add extra salt to his meals and to take frequent small quantity meals separating the solid and fluid components. He was advised to restrict fluid consumption to 1.5 L/day and to avoid food rich in sugar and fiber.

On follow-up, patient is asymptomatic to date with serum sodium ranging from 130 to 134 mM/dL and a stable ileostomy output. Patient is awaiting ileostomy reversal.

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