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Case report

Acute adrenal insufficiency presenting as high output ileostomy

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We report a patient with ulcerative colitis who, in the period following surgery, developed recurrent episodes of acute adrenal insufficiency presenting clinically as a high output ileostomy.

Key words: High output – Ileostomy – Acute adrenal insufficiency

Complications associated with long-term steroid use in ulcerative colitis are well known. High output ileostomy of unknown aetiology has been reported in all published series of pouch surgery for ulcerative colitis.[1,2] We report a patient with ulcerative colitis who, in the period following surgery, developed recurrent episodes of acute adrenal insufficiency presenting clinically as a high output ileostomy.

Case report

A 31-year-old man with histologically proven ulcerative colitis since 1992 underwent a restorative proctocolectomy with pouch-anal anastomosis and a defunctioning ileostomy in April 1997. He was given the standard peri-operative cover with intravenous hydrocortisone having stopped his regular steroids (prednisolone) 1 month prior to surgery.

He recovered well from surgery, but from the 10th day developed a high-output stoma producing more than 5 l/day along with general lethargy and fever with serum biochemistry showing sodium levels of 117 mmol/l, potassium 5.5 mmol/l, urea 16.2 mmol/l and creatinine 129 mmol/l. On examination, he was hypotensive, the abdomen was soft with normal bowel sounds, a rectal examination revealed an intact anastomosis, a CT scan of the abdomen showed no intra-abdominal collections and stool cultures proved negative. In view of the clinical findings of lethargy, diarrhoea presenting as high-output stoma and abnormal biochemistry, a possibility of acute adrenal insufficiency was considered in spite of high blood cortisol levels (night-time level of 534 nmol/l and day-time level of more than 1380 nmol/l). The patient showed immediate clinical response to intravenous hydrocortisone (100 mg) and stomal output reduced to less than 1 l/day within 24 h. He was subsequently discharged on a reducing dose of oral steroids.

The patient was re-admitted within a few days with generalized lethargy, poor appetite, sore throat and a high-output stoma. He was found to be hypotensive. Investigations revealed hyponatraemia, hyperkalaemia and a raised white cell count. Again, there was no evidence of intra-abdominal sepsis and stool cultures proved negative. A diagnosis of acute adrenal insufficiency precipitated by infection was made. An endocrinologist's opinion concurred with the diagnosis of secondary adrenal suppression. No other clinical features of Addison's disease or hypopituitarism were identified. The 'normal' serum cortisol levels in the initial postoperative period were thought to be less than required/expected for the degree of the patient's illness. The standard replacement doses of hydrocortisone were probably inadequate in his case. The high salt and fluid loss from the ileostomy may have caused the need for

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increasing doses of steroids. An addition of fludrocortisone to the steroid replacement therapy was recommended which led to a rapid improvement in the patient's condition.

Discussion

Steroids induce and maintain remission in ulcerative colitis more effectively than is possible without their use. Long-term steroid use, however, induces suppression of the patient's own adrenal cortex. This may precipitate acute adrenal insufficiency by either a sudden withdrawal of steroid therapy or when the patient experiences stress, such as from infection or surgery.[3] The mineralocorticoid deficiency results in increased renal sodium loss and potassium re-absorption causing decreased intravascular volume, vascular tone, cardiac output and renal perfusion. The glucocorticoid deficiency causes fasting hypoglycaemia, muscle weakness and gastrointestinal disturbances (*e.g.* nausea, vomiting, diarrhoea and abdominal pain).

The patient in our case report developed Addisonian crisis as a response to stress of surgery. The diarrhoea as part of the crisis manifested itself as a high output ileostomy. Output from a newly constructed ileostomy is usually high (1–1.5 l/day) in the first 2 weeks, settling subsequently to 500–800 ml/day. A high output ileostomy has an effluent of more than 1 l/day.[4] Various causes have been described for a high output ileostomy, for example, resolution of postoperative ileus, intra-abdominal sepsis, gastro-enteritis, recurrent Crohn's disease, partial small bowel obstruction and short bowel syndrome. Our case report identifies Addisonian crisis as another important and readily treatable cause of high output ileostomy.

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