Clinical picture

Severe intestinal oedema due to nephrotic syndrome

Nephrotic syndrome is defined by oedema, proteinuria (>3 g/24 h) and hypalbuminaemia (<30 g/l). Nephrotic syndrome may occur as a result of primary renal or systemic diseases, commonly diabetes mellitus in the adult population. The cause of the oedema is not completely understood, but may result from loss of plasma oncotic pressure due to the hypalbuminaemia or primary sodium retention in the collecting tubules due to abnormal tubular function. There is usually no evidence of arterial under-filling and most patients respond to a combination of fluid and salt restriction in combination with diuretics, and treatment of the underlying glomerular pathology.

The oedema is easily demonstrated in the peripheries but can also be found in the pleural and peritoneal cavity, giving rise to respiratory and abdominal symptoms and signs. A further recognized consequence of nephrotic syndrome is intestinal oedema.

A 47-year-old offshore oil rig worker developed nephrotic syndrome. At presentation, his serum albumin was 24 g/l, serum creatinine was 167 μmol/l and urinary protein leak was 79 g/24 h. His serum cholesterol was 9.7 mmol/l. Oedema was present to his sacrum. Renal biopsy demonstrated minimal change disease. Initial treatment with oral loop diuretics was commenced. His serum albumin fell below 15 g/l and serum creatinine peaked at 307 μmol/l. Intravenous diuretic therapy and high-dose oral prednisolone (60 mg daily) was commenced following the exclusion of associated infection and malignancy.

The patient developed abdominal pain, with signs of an acute abdomen. Plain abdominal X-rays showed no evidence of obstruction. A CT abdomen showed marked bowel wall thickening with almost complete occlusion of the lumen consistent with severe oedema. The oedema was segmental, only involving part of the small bowel (Figure 1). The symptoms were slow to settle and surgical exploration was considered. A repeat CT scan was performed, by which time the abdominal symptoms were improving. No intra-abdominal pathology was identified other than intra-peritoneal and pelvic free fluid. The initial bowel oedema had resolved (Figure 2). His oedema continued to improve with a rise in serum albumin. Complete resolution of nephrotic syndrome occurred after 3 months of oral steroids.

Severe intestinal oedema may occur as a result of fluid overload or any cause of hypoalbuminaemia, and as in this case, nephrotic syndrome. Abdominal pain may also be present and mimic an acute surgical abdomen, but should resolve with resolution of the oedema. Intussusception has been described in relation to severe intestinal oedema1 as has mesenteric vein thrombosis,2 neither of which was observed in this case. Appropriate investigation of patients with abdominal pain and nephrotic syndrome is essential.
Conflicts of interest: None declared.

References

Figure 2. Despite the development of ascites the small bowel oedema has improved.

Clinical picture