**Case Report**

**Ogilvie’s syndrome following renal transplantation**

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**Introduction**

Gastrointestinal complications following renal transplantation are not unusual; they include gastritis and ulcer of the stomach or duodenum in about 20% of the cases, disordered motility with paralytic ileus in 5%, and colonic perforation in 2%. Most of these complications can be treated successfully by conservative means, but colonic perforation is associated with a high mortality rate of 30–60% [1–9]. It has been suggested that high-dose corticosteroid rejection therapy, CMV infection, ischaemic or pseudomembranous colitis, and graft dysfunction play a role in severe gastrointestinal complications post-transplant, but there is little evidence to support a direct causative effect with any of these various factors.

‘Ogilvie’s syndrome’ or acute colonic pseudo-obstruction was first described by H. Ogilvie in 1948 [10]. The archetypal patient reported by Ogilvie suffered months of abdominal distension and colicky pain. At surgery malignant invasion through the coeliac axis was found, but the colon, while dilated, appeared otherwise healthy and not obstructed. Ogilvie’s syndrome is characterized by dilatation of the distal colon or of the caecum and right colon without any mechanical obstruction, and is known to occur in association with many medical and surgical conditions. When it occurs following abdominal surgery, certain factors are common, namely prior chronic constipation, diabetic autonomic neuropathy, retroperitoneal haematoma, electrolyte disturbance, treatment with opioid analgesics, kayexalate, and other drugs. It has been suggested that the pathogenesis of Ogilvie’s syndrome is a persisting neurapraxia of the sacral parasympathetic nerves S₂–₄ associated with a severe underlying illness or injury [11–15]. In spite of the increasing number of renal transplants, a typical case of Ogilvie’s syndrome after renal transplant has not been reported to date. We report a case of a 65-year-old man who developed Ogilvie’s syndrome 7 days after renal transplantation.

**Case report**

A 65-year-old man (A−, HbsAg−, CMV+, HCV−) with end-stage renal disease (minimal-change glomerulonephritis) was admitted for cadaveric renal transplantation, after receiving conventional haemodialysis for the previous 3 years. There was no history of gastrointestinal complaints. The patient received a cadaveric kidney on a double immunosuppressive protocol,

![Fig. 1. Plain film demonstrating gross colonic distension with caecal diameter 12 cm of the right colon 14 days after renal transplantation. There is no sign of perforation.](image)
and the transplantation procedure was without complications. He remained oliguric in the immediate postoperative period, and histological examination showed a primary-non-function of the transplant. On day 6 the patient developed a distended, tympanitic non-tender abdomen without high-pitched or reduced bowel sounds. An abdominal film showed dilatation of the entire colon. Rectoscopy showed no evidence of pseudo-membranes or ischaemia. Conservative treatment with nasogastric decompression aspiration was commenced but the patients abdominal distension persisted for 5 days. Repeat abdominal X-ray and CT-scan did not show perforation, colonic diverticulum, pelviperitonitis or retroperitoneal bleeding (Figure 1, 2).

The patient’s abdominal distension worsened without any pneumoperitoneum in the X-ray. Without fever or peritoneal signs the white blood cell count was elevated, and persisting abdominal distension led to surgery being carried out on day 16. The dose of immunosuppression was also reduced (cyclosporin 25 mg/day, methylprednisolone 8 mg/day). On day 18 after the renal transplantation the laparotomy revealed a fresh caecal perforation with local peritonitis. The histological examination indicated no primary disease of the colon. The intensive care treatment was uneventful. Two weeks later the patient suffered acute bleeding from the stomach due to a peptic ulcer. A Billroth-I resection was performed on day 22. Another bleeding complication—retroperitoneal haematoma formation with known lack of the factor XI—led to a third laparotomy on day 47. Again there was no primary disease of the colon or local peritonitis. Postoperative respiratory failure required a prolonged artificial ventilation and the patient finally died of cardiovascular insufficiency on day 89.

Discussion

The reason for gastrointestinal complications following renal transplantation is not always known, especially for intestinal perforation in renal transplantation. The clinical symptoms are often atypical resulting in delayed diagnosis and treatment [16]. The mortality of colonic perforation is declining as improvements are being made in the recognition, prevention, and surgical management of the disease [16–20]. However, acute colonic pseudo-obstruction with potentially fatal complications after kidney transplantation has only been rarely associated with non-obstructing colonic dilatation.

Frequent serial abdominal radiographs and colonscopic decompression have a key role in the management of the condition [21,22]. Early surgery in the case of increasing abdominal pain with or without tenderness and leukocytosis may be needed to protect the patient from uncontrolled sepsis. The pathogenesis might be explained by Laplace’s law relating wall tension to the radius of a hollow viscus. The caecum, which has by nature a larger diameter than the remainder of the large intestine, has the highest wall tension. Wangensteen estimated that an intracaecal pressure of 25 cm H₂O was necessary for perforation [23].

In general initial treatment of caecal dilatation of less than 10 cm diameter consists of a conservative approach, including nasogastric decompression, withholding of narcotics, and correction of any electrolyte abnormalities. Recent reports document tocolysis with a use of therapeutic levels of magnesium and nifedipine [24,25]. If the colon shows a progressive enlargement on X-ray, perforation may develop. In the last 10 years, since the introduction of the flexible fibreoptic colonoscope for colonscopic decompression, the perforation and mortality rate have decreased.

Conclusion

Mild cases of intestinal pseudo-obstruction which resolve spontaneously are not uncommon after renal transplantation. It is important to recognize the occasional patient with a more severe and persistent form of the disorder who is at risk of developing potentially fatal colonic perforation. The most important feature remains early recognition of Ogilvie’s syndrome followed by colonscopic decompression, repeated if necessary to avoid the subsequent vascular insufficiency, necrosis, and ultimate colonic perforation.

References

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