CASE REPORT

Is Bacterial Translocation a Cause of Aortic Graft Sepsis?

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Introduction

Bacterial translocation (BT) is defined as the passage of gastrointestinal microflora across the lamina propria to local mesenteric lymph nodes, and from there to distant sites. BT is known to occur in humans and is associated with an increased incidence of septic morbidity.

We describe here a patient in whom the occurrence of BT was demonstrated at the time of repair of an abdominal aortic aneurysm, and who subsequently developed late graft sepsis. This finding raises the possibility that this phenomenon represents a cause of prosthetic vascular graft infection.

Case Report

A 61-year-old man underwent elective surgery for an 8-cm diameter infrarenal abdominal aortic aneurysm. At the beginning of laparotomy a lymph node was excised from the small bowel mesentery close to the ileocolic junction and sent for microbiological analysis as part of an ongoing study into bacterial translocation in surgical patients.1 Once lymph-node sampling was complete the patient received a 1.2 g intravenous dose of co-amoxiclav as prophylaxis. The operative procedure was straightforward, with a 24-mm diameter Dacron tube graft used to repair the aneurysm. The patient made an uneventful postoperative recovery and was discharged from hospital ten days later.

Culture of the lymph node sampled at laparotomy isolated the Gram-negative enteric bacillus Escherichia coli. Concomitant venous blood samples, small bowel serosal exudate collected for the duration of the procedure and mural thrombus from within the aneurysm sac were all sterile.

Two-and-a-half years later the patient was admitted as an emergency with haematemesis and melaena. A contrast-enhanced CT scan of the abdomen demonstrated a large haematoma adjacent to the proximal aortic anastomosis. The diagnosis of an aortoenteric fistula was confirmed at laparotomy. The aortic graft was excised and the lower limbs revascularised with an extra-anatomical axillofemoral graft.

Culture of the aortic graft revealed a lactose-fermenting coliform species, likely to be an E. coli, with an antibiotic-sensitivity profile identical to that of the organism isolated from the MLN at the time of the original operation.

Unfortunately, the patient developed a superficial femoral artery thrombosis the following day, necessitating femoral embolectomy, refashioning of the groin anastomosis and fasciotomies. Despite continued active management in the Intensive Care Unit, he subsequently developed adult respiratory distress syndrome (ARDS) and multiple organ failure, culminating in death a few days later.

Discussion

Prosthetic aortic graft infection is a serious problem, with an incidence of between 1 and 6% and an associated mortality rate of around 50% in most series.2 Historically, the majority of graft infections have been caused by skin commensals such as Staphylococcus aureus and Staphylococcus epidermidis.3 The majority of infections are thought to occur as a result of direct
contamination of the graft at the time of surgery, the usual source of bacteria being the patient’s skin, but others include infected lymphatics and contaminated mural thrombus or atheroma. Grafts may also become infected by direct extension of a groin wound infection or by haematogenous seeding. However, in a recent series of cases of aortic graft infection from one unit more than half of the grafts cultured grew enteric bacteria, with *E. coli* identified in 12 out of 50 cases. This case report illustrates the possibility that the process of BT across the intestinal wall represents a further cause of prosthetic aortic graft infection. BT is known to occur in significant numbers of patients undergoing laparotomy, and its presence is associated with an increased incidence of postoperative septic morbidity. 

The majority of bacteria isolated from mesenteric lymph nodes (MLNs) are indigenous enteric bacteria, predominantly *Enterobacteriaceae* such as *E. coli*. These bacteria are responsible for the majority of postoperative infections. Reduced splanchnic blood flow is inevitable perioperatively in patients undergoing AAA repair, a factor known to predispose to BT. The fact that *E. coli* was grown from the MLN obtained at the first operation, and a similar coliform species was isolated from the excised graft over 2 years later raises the interesting possibility that the translocating bacteria were able to colonise the graft at the time of insertion or soon after, with consequent infection and formation of an aortoenteric fistula. This has potential implications in terms of the possible need for further therapeutic manoeuvres such as prolonged antibiotic therapy or at least close surveillance in those patients in whom evidence of BT is identified at the time of graft insertion.

**References**


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