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PATCHY SMALL BOWEL ISCHAEMIA SECONDARY TO SEPSIS

Editor,

Insufficient blood perfusion to the small bowel may result from arterial occlusion by embolus or thrombosis, thrombosis of the venous system, or non-occlusive processes such as vasospasm or low cardiac output. Patterns of segmental, skipped, or patchy small bowel ischaemia have been reported post abdominal aortic aneurysm repair and is thought to imply that microembolisation has played an important role[1]. Microvascular thrombi in various organs can result from disseminated intravascular coagulation[2]. We present a case of patchy small bowel ischaemia in a septic patient who had developed evidence of disseminated intravascular coagulation.

Case presentation: A 50-year-old man prescribed four weeks of diclofenac for a muscular strain presented with frank haematemesis secondary to an eroding gastric ulcer measuring 7 cm in diameter. A partial gastrectomy and Roux-en-Y reconstruction was performed and a massive blood transfusion of 25 units was required peri-operatively. The patient was referred to a tertiary care centre for intensive care where on day six post operatively, he developed peritonitis as a result of a biliary leak from his duodenal stump. His blood tests at that time were suggestive of non-overt disseminated intravascular coagulation secondary to sepsis with an elevated D-dimer of 7.36 µg/ml (0.01-0.5 µg/ml). An emergency exploratory laparotomy was performed. This revealed a duodenal stump blowout and a 2 inch segment of proximal jejunum containing multiple less than 1 cm blisters in a triangular arrangement (figure 1). These were found to be necrotic areas and were resected. Histology of the specimen confirmed areas of transmural haemorrhagic ischaemic necrosis associated with a marked serosal exudate and incipient perforation. Thrombi were histologically identified within the omentum. Later in the post-operative period he developed bleeding from his duodenal stump for which he had successful radio-embolisation of the gastroduodenal artery. After a complicated postoperative period, he was discharged home. Although, patchy bowel ischaemia has been reported in past, this pattern in a mid jejunal small bowel segment containing multiple less than 1 cm areas of necrosis in a septic patient has not been reported to our knowledge.

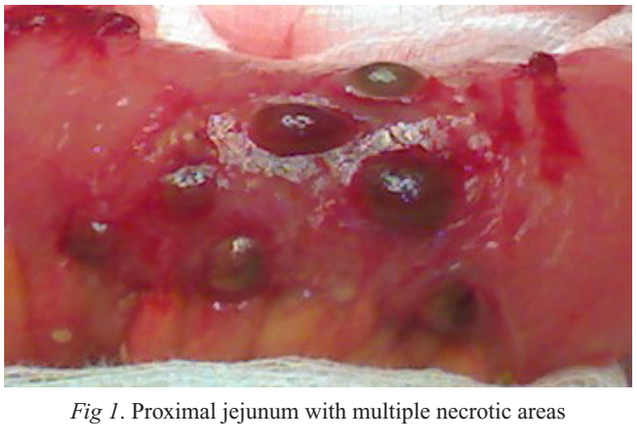

Fig 1. Proximal jejunum with multiple necrotic areas

Discussion: Patchy small bowel ischaemia has been noted in patients following abdominal aortic aneurysm repair[1]. Some of these patients have been found to have widespread microembolisation or to have pathological evidence of microemboli. As a result it has been postulated that this pattern of ischaemia is a direct consequence of microembolisation[3]. Sepsis almost invariably leads to haemostatic abnormalities. These range from the insignificant to severe disseminated intravascular coagulation. Compelling evidence from clinical and experimental studies suggests that disseminated intravascular coagulation is involved in the pathogenesis of microvascular dysfunction and that deposition of microvascular thrombi can occur[3,4]. Given the above patient had an elevated D-dimer and evidence of thrombi on histology it is our feeling that the likely mechanism behind this unusual intra-operative finding was microembolisation. With sepsis related disseminated intravascular coagulation being the most likely cause.

Conclusions: We conclude that in septic patients, the bowel should be carefully examined as missing such pathology will have serious implications for critically ill surgical patients.

The authors have no conflict of interest

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CLINICAL EXPERIENCE WITH INTRAVENOUS IMMUNOGLOBULIN AND TNF-A INHIBITOR THERAPIES FOR RECURRENT PREGNANCY LOSS

Editor,

We report on a 22 year-old non-smoking nulligravida who presented with her husband for in vitro fertilisation (IVF). She was in good general health and had five prior unsuccessful IVF treatments, all with implantation failure. While her TSH and T4 were normal, a strongly positive (1:25,600) thyroid peroxidase antibody (ATA) titre was noted. Their sixth IVF cycle included IVIG infusion x3 as had been used in the immediately preceding cycle. However, etanercept (Enbrel®;

Immunex Corp., Thousand Oaks, California USA) was added for the first time as a series of 25mg subcutaneous injections commencing four weeks before ovulation induction and continued on four-day intervals thereafter. Eight etanercept injections were given until commencement of gonadotropins, and then discontinued. Two blastocysts were transferred fresh and two were frozen at day five. Following an unremarkable obstetrical course, the patient delivered male/male twins by Caesarean at 34½ weeks' gestation. While the strongly positive ATA titre finding in our patient was concerning, we admitted that the mechanism of how ATA impacts reproductive outcome is presently unknown. ATA have been documented more often in women with recurrent pregnancy failure than controls, and a prospective clinical trial of women with "immunologic abortion" evaluating multiple autoimmune variables found ATA to be the most frequently encountered immunopathology—present in 53% of patients[1]. Our case, believed to be the first published report of its kind in Ireland, is parallel with those who have described a highly-circumscribed use of immunomodulators for refractory cases where an immune diathesis exists[2,3] and given only under closely monitored conditions. While immunomodulators are inappropriate in IVF for unselected populations and should not be regarded as first-line therapy, dampening of immune responses antagonistic to implantation and embryo development may be a derivative of IVIG + etanercept therapy. Should our patient decide to enlarge her family and return for transfer of cryopreserved embryos in future, the role of further immunomodulator treatment will require consideration.

The authors have no conflict of interest

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