

Portal vein thrombosis in a complicated case of Crohn's disease

A. Crowe, N. Taffinder, G.T. Layer, A. Irvine¹ and R.J. Nicholls

Departments of Gastrointestinal Surgery and ¹Radiology, St Thomas' Hospital, London SE1 7EH, UK

Summary: Portal vein thrombosis is a rare complication of ulcerative colitis and is invariably fatal. This report describes a patient with severe Crohn's disease who underwent elective surgery complicated by an anastomotic disruption with faecal peritonitis. Following emergency laparotomy he developed left hypochondrial pain which was a manifestation of splenomegaly consequent upon portal vein thrombosis. Anticoagulation was successful in preventing further spread of the thrombosis as monitored by colour Doppler ultrasound. Severe active disease, surgery and sepsis have been recognized as predisposing factors for thromboembolic complications in inflammatory bowel disease and this patient was exposed to all three. It is conceivable that portal vein thromboses occur more commonly than suspected and ultrasound scanning could ascertain the prevalence if performed prospectively.

Introduction

Thromboembolic complications of inflammatory bowel disease have been recognized and are associated with a high mortality.¹ Portal vein thrombosis is rare but has been described in 6 cases of chronic ulcerative proctocolitis, 5 of which were fatal.¹⁻³ This report purports to be the first to record extensive thrombosis of the portal venous system in a patient with Crohn's disease.

Case report

A 42 year old man suffering with Crohn's disease was investigated for iron deficiency anaemia. Twenty-seven years previously a fistula-*in-ano* had been laid open, histology revealing non-caseating granulomata. Since then, he had undergone three bowel resections which included two jejunal excisions and a colectomy resulting in a caeco-rectal anastomosis. His medication included oral corticosteroids and azathioprine.

Barium studies on this admission demonstrated two ulcerated small intestinal segments, an ileocolic stricture and a palpable rectal stricture. At laparotomy two anastomotic recurrences were excised and a limited ileo-caecal resection per-

formed with restoration of continuity by an end-to-side anastomosis, all macroscopic disease being removed. The rectal stricture was dilated.

One week postoperatively, he developed faecal peritonitis. At laparotomy, the staple line closing the blind end of the colon was found to have disrupted. The ileocolic anastomosis was resected and microscopically showed non-caseating granulomata. The rectum was oversewn and a spout ileostomy formed.

Twelve days later he developed left hypochondrial pain and a fever. A subphrenic abscess was suspected. Investigations showed a leucocytosis and thrombocytosis ($936 \times 10^9/l$), a normal coagulation profile and normal chest radiograph. Ultrasound revealed mild splenomegaly and extensive thrombosis of the superior mesenteric, splenic and portal veins. Four days later the venous phase of a selective superior mesenteric angiogram confirmed non-occlusive portal, splenic and superior mesenteric venous thrombosis with the development of a collateral circulation (Figure 1). An intravenous infusion of heparin was commenced. The patient's pain and pyrexia subsided and the ileostomy remained healthy.

The portal venous system was examined every 2 days by a real-time colour Doppler ultrasound scanner (Acuson 128) which monitored the thrombus and the portal collaterals.

The patient's recovery was further complicated by a superficial wound abscess 3 weeks later following the cessation of antibiotics. This necessitated surgical drainage but the incision healed

Correspondence: G.T. Layer, D.M., M.Ch., F.R.C.S., Department of Surgery, St George's Hospital, Blackshaw Road, London SW17 0QT, UK

Accepted: 27 August 1991

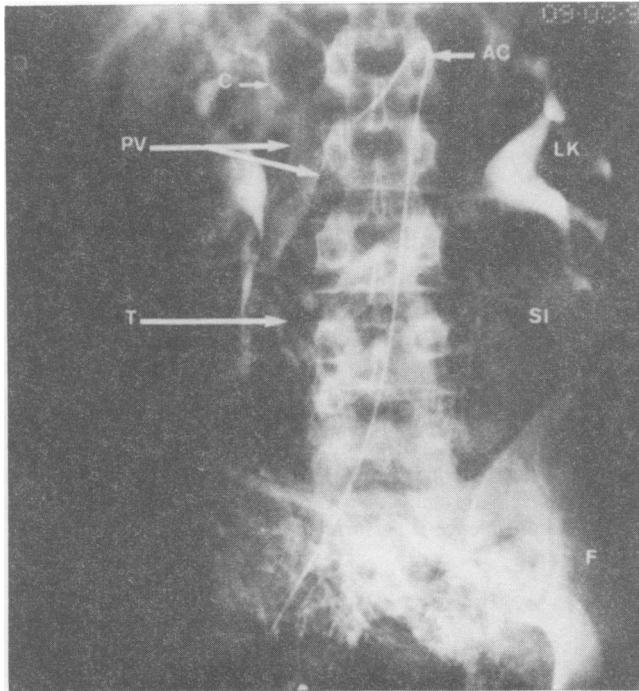


Figure 1 The venous phase of the selective superior mesenteric angiogram demonstrating thrombus in the portal venous system. AC = perforator angiogram catheter placed in the origin of the superior mesenteric artery; LK = pyelogram of left kidney; SI = small intestinal mesenteric arcades; F = flange of spout ileostomy appliance; PV = portal vein; T = thrombus outlined by contrast in the superior mesenteric vein; C = probable portal vein collateral.

soundly and he was discharged on warfarin therapy. Outpatient follow-up has shown complete resolution of left hypochondrial pain, this being his major symptom of portal vein thrombosis. He was anticoagulated for 3 months.

Discussion

The prevalence of thrombosis in association with inflammatory bowel disease varies between 1.2% and 39% depending on whether clinical or post-mortem data are studied.⁴ The thrombotic tendency has been attributed to certain risk factors. Talbot *et al.* in a retrospective review of 7,199 events identified three; active disease, surgery and sepsis.¹ Possible mechanisms are low antithrombin III and protein C levels, raised factors V and VIII levels and thrombocytosis; although these appear not to be relevant in stable disease.⁵ Our patient was exposed to all 3 risk factors and had a marked thrombocytosis but levels of antithrombin III, protein C, factors V and VIII were normal.

A recent review⁶ has noted the activation of blood coagulation in Crohn's disease, with the deposition of fibrin being a characteristic

pathological feature which correlates with the activity of the disease. It has been suggested that an activation of the immune system with induction of monocyte procoagulant activity is responsible for both the vasculitic and thrombotic lesions of Crohn's disease. Indeed, microscopic mesenteric thrombosis leading to intestinal infarction has been demonstrated to result in an appearance similar to Crohn's disease and may be an aetiological factor.⁷ Furthermore, major arterial thrombosis has also been described as a manifestation of the onset of active Crohn's disease.⁸

Hepatic and portal vein thrombosis have been recognized as heralding an occult myeloproliferative disorder.⁹ Tumour necrosis factor (TNF) has been implicated in the promotion of intravascular clot formation in both inflammatory and malignant states.¹⁰ More specifically, TNF-alpha has been demonstrated to be an important mediator of inflammation in the human gut and is increased in Crohn's disease,¹¹ this may represent another mechanism predisposing to venous thrombosis.

It is possible that many portal vein thromboses occur but remain unsuspected and undetected. A simple prospective study would be useful using Doppler ultrasound scanning to ascertain the true

prevalence.

Portal vein thrombosis has a poor prognosis.¹ Safe thrombolysis with tissue plasminogen activator has only been considered possible in highly selected patients.¹² In view of the evidence, it now seems appropriate to recommend the administration of prophylactic subcutaneous heparin to patients with inflammatory bowel disease undergoing surgery, if this policy is not already practised.

References

1. Talbot, R.W., Heppell, J., Dozois, R.R. & Beart, R.W. Vascular complications of inflammatory bowel disease. *Mayo Clin Proc* 1986, **61**: 140–145.
2. Aronson, A.R. & Steinheber, F.U. Portal vein thrombosis in ulcerative colitis. *NY State J Med* 1971, **71**: 2310–2311.
3. Capron, J.P., Remond, A., Lebrec, D., Delamarre, J., Dupas, J.L. & Lorriaux, A. Gastrointestinal bleeding due to chronic portal vein thrombosis in ulcerative colitis. *Dig Dis Sci* 1979, **24**: 232–235.
4. Lam, A., Borda, I.T., Inwood, M.J. & Thomson, S. Coagulation studies in ulcerative colitis and Crohn's disease. *Gastroenterology* 1975, **68**: 245–251.
5. Knot, E., Ten Cate, J.W., Leeksa, O.C., Tytgat, G.N. & Vreeken, J. No evidence for a prethrombotic state in stable chronic inflammatory bowel disease. *J Clin Pathol* 1983, **36**: 1387–1390.
6. Wakefield, A., Cohen, Z. & Levy, G. Procoagulant activity in gastroenterology. *Gut* 1990, **31**: 239–241.
7. Wakefield, A.J., Sawyerr, A.M., Dhillon, A.P. *et al.* Pathogenesis of Crohn's disease: multifocal gastrointestinal infarction. *Lancet* 1989, **ii**: 1057–1062.
8. Halliday, C.E.W. & Farthing, M.J.G. Arterial thrombosis in Crohn's disease. *Med J Aust* 1988, **149**: 559–560.
9. Boughton, B.J. Hepatic and portal vein thrombosis. *Br Med J* 1991, **302**: 192–193.
10. Nawroth, P.P. & Stern, D.M. Modulation of endothelial cell hemostatic properties by tumour necrosis factor. *J Exp Med* 1986, **163**: 740–745.
11. Macdonald, T.T., Hutchings, P., Choy, M.-Y., Murch, S. & Cooke, A. Tumour necrosis factor-alpha and interferon-gamma production measured at the single cell level in normal and inflamed human intestine. *Clin Exp Immunol* 1990, **81**: 301–305.
12. Fidler, H., Booth, A., Hodgson, H.J.F., Calam, J., Luzatto, L. & Hughes, J.M.B. Portal vein thrombosis in myeloproliferative disease. *Br Med J* 1990, **300**: 590–592.

Acknowledgements

We are most grateful to Professor N.L. Browse and Mr K.G. Burnand for their clinical advice, Dr B. Ayers, Dr L. Macdonald and Dr M. Creagh for their radiological expertise and Dr B. Creamer for his permission to report this case.