

months, which unmasked the diabetes and prompted its discontinuation, could have had a pathogenetic effect, although this point is speculative. Presumably gall stones were the initial cause of the acute pancreatitis; however, we believe that they were not the cause of CCP, because a follow up CT scan failed to show them, and because gall stones are not a cause of chronic pancreatitis or CCP.¹¹

In summary, we present two cases of CCP, one in a patient with SLE and SS, and the other in a patient with scleroderma, in which the usual causes of calcification—chronic alcohol intake, nutritional deficiency, hereditary pancreatitis, and chronic hypercalcaemia—have been excluded. Because there was no definitive aetiological factor, this could be either idiopathic and unrelated to the rheumatic disease, or a real association with these collagen diseases.

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Small bowel telangiectasia in scleroderma

Scleroderma may involve the gastrointestinal (GI) tract in over 50% of patients, common problems being those of dysmotility, malabsorption and bacterial overgrowth.¹ Winterbauer documented gastric telangiectases in his case reviews in 1964,² and since then there have been sporadic reports of significant gastrointestinal bleeding from both gastric and colonic lesions.^{3–9} We report the occurrence of telangiectasia of the small bowel in a patient with scleroderma and significant GI bleeding.

This 47 year old woman was diagnosed as having scleroderma when she presented with tightness, swelling of her fingers and Raynaud's phenomena. Her autoantibody profile, specifically anticentromere and anti-Scl-70, had always been negative. An upper GI endoscopy was performed when she developed symptoms of gastro-oesophageal reflux; this showed gastric antral telangiectases in a 'water melon' pattern. At that time her haemoglobin concentration was normal. Eighteen months later she was admitted as an emergency, having had melaena. Urgent repeat endoscopy demonstrated the previous lesions, but with no evidence of bleeding; subsequent colonoscopy was unremarkable. She continued to have episodes of blood loss and needed regular admissions to hospital for blood transfusions—approximately 30 units over six months. Coeliac axis and superior mesenteric angiography was performed and revealed no arteriovenous malformation.

Laparotomy and 'on-table' enteroscopy were carried out and revealed multiple telangiectases throughout the small intestine, the distal half being most densely affected. Three separate 4–7 cm lengths of the most severely involved ileum were resected. The patient still required frequent transfusions. However, as the relative contributions of the small bowel and gastric lesions to the overall blood loss were not clear a ⁹⁹Tc labelled red cell scan was undertaken. Its appearance was consistent with a source of bleeding in the terminal ileum. Because her gastric lesions might, at other times, be contributing more than was demonstrated, it was decided to treat these with heater probe ablation. Fortunately, her transfusion requirements have now reduced and she maintains a stable haemoglobin concentration with oral iron therapy alone.

Angiodysplasia may be responsible in up to 8% of cases of upper GI bleeding and possibly around 6% of bleeding of the lower GI tract.¹⁰ Its symptomatic association with scleroderma seems rare, though when it does occur it is certainly significant. In this patient, small bowel lesions were not only shown to be present, but at one time appeared to be the main source of bleeding.

This finding is not surprising but appears not to have been described previously. The gastric telangiectasia is probably not without blame, but it is difficult to determine what

proportion of blood loss has come from any particular site. Interestingly, this lady has never displayed cutaneous telangiectasia, unlike most of the cases previously reported. Should this lady's transfusion requirements again increase, it is proposed to try further ablation therapy, and then oestrogen therapy in an attempt to reduce small bowel blood loss.¹⁰ We highlight this case in order to bring attention to the possibility of small bowel telangiectasia causing blood loss in patients with scleroderma.

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Correction

Anticardiolipin, anticentromere and antiScl antibodies in patients with systemic sclerosis and severe digital ischaemia

An author's error occurred in this paper by Dr Herrick and others (*Ann Rheum Dis* 1994; **53**: 540–2). The fifth sentence in the second paragraph on page 542 should have read: '... but a Spanish study ...' [not '... but another Japanese study ...'].

The authors wish to apologise to Dr Fonollosa and colleagues (Barcelona) for this mistake.