isotopic methods may be needed to determine which is the major lesion requiring treatment.

Complete remission of gastrointestinal bleeding has followed partial gastrectomy for watermelon stomach and gastric telangiectasias but, given the propensity for multifocal lesions in CREST syndrome and the potential problems such patients can have with diarrhoea, attempts should be made to preserve as much intact bowel as possible. The authors state that endoscopic therapies are only effective in small lesions. Our experience shows that extensive vascular lesions can be safely dealt with by endoscopic means and this has been confirmed by others. Heater probe, bipolar cautery, laser and sclerant injection have been used successfully.

Gostout et al. achieved success in 87% of 45 patients with watermelon stomach with no serious complications, although 33% were left with asymptomatic antrum dysmotility.

It is surprising that gastric vascular abnormalities are a relatively uncommon reported cause of gastrointestinal bleeding in CREST patients given that a significant proportion (65%) of patients with the watermelon stomach exhibit ‘autoimmune’ or connective tissue disease type phenomena. Prednisolone may be effective in the presence of hypergastrinaemia and achlorhydria. Our patient rather unusually in this population had normal acid output and gastrin so corticosteroids were not given. Progesterone/oestrogen combinations have also been reported to be effective in bleeding gastric telangiectasias but were avoided in this case because of previous hormone-induced migraine. A high index of suspicion is often needed to make the diagnosis of watermelon stomach at endoscopy, angiography is often unhelpful and histological changes may be missed at biopsy. As the authors state the endoscopic appearance can be mistaken for gastritis.

Gastric vascular abnormalities should be considered by gastroenterologists and rheumatologists in the differential diagnosis of anaemia in CREST patients, wider recognition of these entities and associations is likely to result in more cases becoming evident. Endoscopic therapy may be all that is required.

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References

Cardioprotective therapeutics – drugs used in hypertension, hyperlipidaemia, thromboembolization, arrhythmias, postmenopausal state and as anti-oxidants

Sirs,
In addition to the cardioprotective agents cited by Kendall et al. in patients with cardiac failure of ischaemic origin, spironolactone deserves special mention because of its potential role in those patients who, as a consequence of suboptimal dosing resulting from drug-related hypotension, might not derive full benefit from angiotenin converting enzyme (ACE) inhibitors. If ongoing human studies were to validate the utility of prophylaxis against myocardial fibrosis documented in animal studies, spironolactone could be a useful adjunct to ACE inhibitors and loop diuretics, partly because the use of triple therapy for blocking tubular reabsorption of sodium at multiple sites could also reduce diuretic requirements. Diuretic-related hyper-reninaemia might thereby be ameliorated, with consequent reduction in the magnitude of hyper-reninaemia as a risk factor for myocardial infarction.

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References
A unusual case of dysphagia

Sir,

Regarding the paper of Treasure and Hall,1 we wish to report a clinical story of similar symptoms but with a different aetiology.

A 68 year old woman had progressive dysphagia that developed over a period of several months. She had the feeling of food remaining stuck in the upper third of her chest. There were no symptoms of heartburn or other chest distress and no weight loss.

Clinical examination and routine chest X-rays were normal. Barium swallow revealed an oesophageal stenosis and endoscopy demonstrated a pulsatory mass at the 25 cm level. There were no mucosal abnormalities. A computed tomographic scan with the application of an intravenous contrast medium showed an aberrant right subclavian artery (ARSA) or arteria lusoria, running vertically and behind the oesophagus and causing the stenosis. In order to demonstrate that the dysphagia in the patient was caused by the presence of the ARSA, an evaluation of deglutition with solid food was performed. The food was labelled with technetium-99 in a colloidal suspension and revealed a stagnation for solids at the level of the ARSA, confirming the diagnosis of dysphagia lusoria. The patient was treated by simple division of the ARSA with decompression of the oesophagus. A reanastomosis was not performed since at operation the blood flow to the right arm through collateral circulation was noted to be satisfactory. After the operation the woman was free of any difficulties in swallowing.

Dysphagia lusoria, the symptomatic compression of the oesophagus by the ARSA, is a rare syndrome.2,3 The anomalous vessel represents the persisting portion of the right fourth aortic arch, the cranial portion of this arch being obliterated.4 Symptoms can appear in childhood and in adults.5,6 A universally accepted explanation for the absence of clinical signs in children and young adults does not exist. Loss of mobility of the anatomical structures and a subsequent oesophageal motility disorder have been suggested. Furthermore, the dilatation, the tortuosity and rigidity of the great vessels, the increase in the dorsal kyphosis, the sclerosis and calcification of trachea, aorta and the abnormal vessels could be an explanation for the occurrence of dysphagia.2,6

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References


Polycythaemia rubra vera presenting with severe glossitis

Sir,

Polycythaemia rubra vera (PRV) can present in various ways including acute bleeding or thrombotic episodes, cerebral circulatory disturbances, gout and pruritus. We report an unusual case of PRV in whom the main presenting complaint was severe glossitis.

An 84 year old woman was admitted to hospital with a 3 month history of severe soreness of the tongue with associated loss of taste, prominent dysphagia for solids and weight loss of 12 kg. There was a past history of ischaemic heart disease and hypertension, and a mild stroke with very good recovery 6 years prior to the present admission. At the time of her stroke, routine tests including a full blood count were normal. She had no history of abdominal or respiratory disease, was a non-smoker and did not drink alcohol. Her only regular medication prior to admission was multivitamin tablets for a few weeks, but the glossitis had not improved.

On examination there was facial plethora, a smooth atrophic, 'beefy' red tongue and moderate splenomegaly. Investigations revealed: haemoglobin 17.8 g%, white cell count 42.2 x 10⁹/l, platelet count 195 x 10⁹/l, differential white cell count—neutrophils 58%, lymphocytes 17%, basophils 10%, myelocytes 10%, myeloblasts 2%; MCV 73 fl, MCH 24 pg, MCHC 33 g/dl. Blood film showed a leucoerythroblastic picture and the ESR was 1 mm in the first hour. Serum electrolytes, renal, liver, calcium and thyroid function tests, as well as serum folate and ferritin levels were normal. Chest X-ray and arterial blood gases were also normal. Serum B₁₂ was > 1,000 ng/l (normal 200–900) and leucocyte alkaline phosphatase score 182 (normal 35–100). Bone marrow trephine examination revealed panhyperplasia with the presence of moderate myelofibrosis, and no evidence of iron deficiency or metastatic disease. An abdominal ultrasound confirmed moderate splenomegaly and showed no abnormality of other abdominal viscera. A barium meal and a gastroscopy were normal. On the basis of these findings, a diagnosis of PRV with secondary myelofibrosis was made and busulphan 4 mg daily was commenced. By the end of 4 weeks' therapy the glossitis and dysphagia resolved, her weight steadily increased, and the full blood count normalized.

Glossitis could occur in PRV as a result of severe iron deficiency due to repeated bleeding or venesections with no iron replacement.1,2 However, in our patient the existence of glossitis without iron or vitamin deficiency, and its fast resolution with chemotherapy given for the