teaching on the endometrial cycle is ever modified.

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References


Watermelon stomach, or antral gastritis

We were interested to read the case report entitled Antral hypertrophic gastritis: a rare cause of iron deficiency by GW Stamp et al. This is similar to a recent case that we have seen which we regarded as gastric antral vascular ectasia (watermelon stomach), an entity recently reported by Jabbari et al. It seems that the two reports have discussed the same entity. We now summarise our case and comment on the two papers.

A 52 year old woman was referred for investigation of chronic iron deficiency anaemia (haemoglobin 8.2 g/l) that had not responded to treatment with iron and necessitated repeated blood transfusions. Extensive investigations at a peripheral hospital, including upper gastrointestinal series, barium enema, and coeliac angiography were unremarkable, but faecal occult blood tests yielded persistently positive results. Repeat air contrast upper gastrointestinal series showed thickened antral folds but no definite ulcer craters. Endoscopy showed prominent, friable, antral mucosal folds with red linear stripes radiating from the pylorus. Mucosal biopsies from the antrum showed fibromuscular replacement of the lamina propria with scattered organising superficial microthrombi. There was no appreciable inflammatory infiltrate. Surface foveolar epithelium appeared villous with regenera-

Fig. 1 Gastric antral mucosa with antral glands separated by vertically orientated fibromuscular bundles radiating from muscularis mucosae (Haematoxylin and eosin.) × 175.

Fig. 2 Superficial hyperplastic gastric antral mucosa with organising capillary thrombus. (Haematoxylin and eosin.) × 410.
tive changes. Mucosal biopsies of the body of the stomach showed chronic atrophic gastritis with intestinal metaplasia. Subsequent investigations showed gastric achlorhydria with raised serum gastrin concentrations. A peripheral blood film was consistent with iron deficiency anaemia as were results of serum iron studies. Serum proteins, protein electrophoresis, and serum IgE concentrations were normal. Blood transfusions were required to correct the anaemia and were complicated by a pyrogenic reaction. In spite of treatment with iron her haemoglobin remained only at 8-8 gl after three months. Because of refractory iron deficiency anaemia, a partial gastrectomy and Roux-en-Y anastomosis were performed. At operation the wall of the gastric antrum appeared thickened, particularly at the pylorus. No bleeding site was visible. Three months postoperatively the haemoglobin concentration was 12-2 gl.

Examination of the antrum showed prominent longitudinal mucosal ridges radiating from the pylorus with an ulceration. The pyloric muscle appeared slightly thickened. Histological examination elicited features similar to those of earlier mucosal biopsies. There was advanced chronic atrophic gastritis with intestinal metaplasia in the proximal stomach. The antrum, particularly distally, showed a paucity of inflammatory cells but striking fibromuscular obliteration of the lamina propria with vertical orientation of smooth muscle cells (Fig. 1). Numerous superficial organising microthrombi were seen (Fig. 2). Submucosal vessels appeared dilated and congested, but no vascular malformation was evident.

The clinical history and morphology of this case is identical with that of Stamp et al. Although they consider the histology to be that of a diffuse form of hyperplastic (regenerative) polyp that has not been previously described, we suggest that this case could be better categorised as gastric antral vascular ectasia as described by Jabbari et al. The endoscopic appearance of the longitudinal hypertrophic folds and the histological features of hyperplastic surface mucosa, with dilated and thrombosed capillaries and fibromuscular hyperplasia of the lamina propria with ectatic submucosal venous channels, seem to be characteristic.

The Montreal group emphasised the endoscopic appearance, in particular the red column of blood vessel in the hypertrophic rugal folds described as being of "watermelon striped appearance," as an aid to the clinical endoscopic recognition of this unusual form of antral gastritis. The endoscopic findings were not emphasised by Stamp et al., but friable and thickened antral mucosal folds were found. The histology of both cases was the same. We would agree with the suggestion of Jabbari et al. that the typical morphology may be a result of distal antral mucosal prolapse towards the pylorus, and the morphology does appear similar to that of the rectal mucosal prolapse syndrome. We must emphasise the rarity of this condition (nine reported cases, including this one) and the importance of recognising characteristic morphological features. The association with chronic atrophic gastritis and intestinal metaplasia is interesting but as yet unexplained.

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References

Platelet phagocytosis: a probable mechanism of thrombocytopenia in Plasmodium falciparum infection

Thrombocytopenia has often been encountered in infection with Plasmodium falciparum, especially when the organisms are resistant to chloroquine. It has been suggested that platelet consumption, as part of a disseminated intravascular coagulation, is a possible mechanism, although this is unlikely to be the basis of thrombocytopenia in all cases. It has been shown, using platelets labelled with chromium-51, that production of platelets is normal or increased during the development of thrombocytopenia associated with malaria. Bone marrow failure is therefore unlikely to be responsible for the development of thrombocytopenia.

Sequestration and destruction of platelets in the spleen is another possible mechanism of thrombocytopenia, in conjunction with splenomegaly. Excessive removal of platelets, whether normal or immunologically changed by a hypertrophied reticuloendothelial system has been postulated. Electron microscopic confirmation of intraplatelet parasitism suggests that this is a possible cause of the decreased lifespan of platelets.

We report a case of malarial infection resistant to chloroquine in which thrombocytopenia seemed to be due to the phagocytosis of platelets by monocytes.

Case report

A 35 year old man who had recently returned to Ireland from a three week holiday in Kenya presented to his general practitioner complaining of fever, sweats, anorexia, nausea, vomiting, and weight loss with generalised arthralgia. Physical examination did not show any abnormality, and his haematological indices were normal. Blood smear, which was initially negative for malaria parasites, was positive for P falciparum seven days later. Treatment with chloroquine was started, but no clinical or haematological improvement occurred. During this period his platelet count dropped to 110 × 10^9/l, haemoglobin concentration was 10/6 g/l, and leucocyte count was 4.6 × 10^9/l, showing a monocyte count of 1.38 × 10^9/l (normal monocyte count 0.2-0.8 × 10^9/l); 80% of these monocytes showed platelet phagocytosis, and some featured rosetting (Figs. 1a and b). Treatment with quinidine sulphate and Fansidar (pyrimethamine and sulfadoxine) was given, bringing about considerable clinical improvement. Blood smears at this stage showed gametocytes, with no further evidence of platelet phagocytosis (platelet count 180 × 10^9/l). Two weeks after treatment the patient was well, and his blood film was negative for malaria parasites. There was no clinical or laboratory evidence of disseminated intravascular coagulation, and platelet associated IgG values were normal (staphyloccocal protein A). Bone marrow aspiration and electron microscopy to show platelet parasitism were not carried out.

Comment

Failure to show increased platelet associated IgG makes it unlikely that an immune mediated mechanism was responsible for the thrombocytopenia. Non-immune platelet damage with subsequent phagocytosis by monocytes most probably contributed to the thrombocytopenia in our patient. This agrees with the previous suggestion that the