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 g/l 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 g/l.

Examination of the antrum showed prominent longitudinal mucosal ridges radiating from the pylorus with no 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 intra-platelet 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, sweating, 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 falci

Letters

Thrombocytopenia

Stab th...
mononuclear phagocytic system has a role in the thrombocytopenia associated with infection with *P falciparum*.

MS JAFF
D MCKENNA
SR MCCANN

**Book reviews**


This is a laboratory manual on methods for growing, purifying, and titrating viruses, written by those with a detailed knowledge of a particular virus group, and would be a useful addition to virus laboratories both with a research or clinical bias. The editor does not claim to cover all the virus groups, but important human pathogens such as polioviruses, herpesviruses (*herpes simplex* types I and II and cytomegalovirus), influenza viruses, and respiratory syncytial viruses are included.

The methodology is clearly set out with each of the steps and the buffers used, so it should be possible for someone unfamiliar with a particular virus group to produce workable quantities of pure virus.

The final chapter, Techniques in Clinical Virology, details some of the newer techniques of identification such as immunofluorescence, detection of respiratory syncytial virus, and serological techniques with special reference to rubella virus, and again all the steps are clearly set out.

This book would be useful in the laboratory, and there is sufficient clinical application to enable personnel in service departments to find it useful to have at hand, as well those carrying out virological research.


Few would contest the authors’ view that gross examination is the basis of a thorough perinatal necropsy. Most of the manual is concerned with the techniques of the gross necropsy with brief sections on definitions, instruments, examination of the brain after fixation, the placenta, and trimming tissues for histology. Comment on special techniques is limited to the perfusion and fixation of hearts and cytogenetic studies. A final section explores the role of the pathologist in discussing the necropsy findings with parents.

The manual is extensively illustrated. The detail in the text implies that it is aimed at those with little previous experience of necropsies of any kind. It provides a full but rather personal approach to technique. No alternative methods are offered, and there are no references to published findings. Its value to the trainee would be enhanced by the addition of comments on “why you do it” at the expense of some of the space devoted to “how you do it.” Good technique is important but it is not an end in itself.

**Notices**

The Benjamin Castleman Award is sponsored jointly by the Massachusetts General Hospital and the United States Canadian Division of the International Academy of Pathology. The award, financed by contributions to a fund established by former students and trainees of Dr Castleman, is presented at the annual meeting of the United States Canadian Division of the International Academy of Pathology, which in 1986, will be held in New Orleans...
Platelet phagocytosis: a probable mechanism of thrombocytopenia in Plasmodium falciparum infection.

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