Letters to the Editor

A Christmas thought from Professor NA Wright: HLA-DR expression in liver disease

It was with the greatest consternation that I detected, in the recent paper by Barbatis et al, a reference to a practice totally abhorrent to any right thinking pathologist, let alone a member of the Association of Clinical Pathologists: the introduction plainly refers, “in cases of liver transplantation rejection ... focal HLA-DR was identified on bile duct epithelium.” Although the reference given is to an Italian work, the implication is plain: people who eat transplanted livers express class II antigens on their bile duct epithelium. Before reinforcing my disgust at such practices, my natural curiosity prompts several questions of the authors: who is doing the eating, and whose transplanted livers are involved?

Can it be (nay!) that health professionals, jaded by paté de foie, truffled hare terrine, and salmi of partridge are rousing their gastronomical acidity by consuming resected or post mortem transplanted livers? If so, can we be enfranchised of the recipe? Personally, I prefer calf’s liver barbecued with fresh sage leaves, but perhaps the presence of a rejecting lymphoid infiltrate adds a certain piquancy to the culinary occasion? Are the authors suggesting a new cooking method, buried in the text among the intricacies of the three stage immunoperoxidase technique, which I am unable to discern? On the other hand, however great the delicacy, I would not be prepared to sample it if, as the authors suggest, my bile duct epithelium would be prepared for immunological attack. Alternatively, are the authors alluding to a revolutionary concept, that autophagy of a patient’s transplanted liver, of necessity obtained at biopsy, leads to expression of class II antigens on his bile duct epithelium?

Both these proposals of course require a mechanism; my meagre knowledge of immunology indicates that gamma interferon is responsible for the induction of HLA-DR expression on epithelial cells. Can we safely assume that absorption from the gut of proteins from transplanted liver leads to the (local) release of gamma interferon close to bile duct epithelium? My worry then becomes—is this phenomenon confined to transplanted tissue, or must we eschew our indulgence in all hepatic parenchyma?

I hope you realise, Dr Barbatis, that you and your colleagues have raised genuine shock, horror, and major doubts about the safety of such eating practices. Perhaps this letter might persuade you to look further into these startling claims, so as to set our minds at rest.

NA WRIGHT
Department of Histopathology,
Royal Postgraduate Medical School,
Hammersmith Hospital,
Du Cane Road,
London W12 0HS.

Reference
1 Barbatis C, Kelly P, Greveson J, Heryet A,

Isolated arteritis of the ovarian hilum

The histological and immunohistological features and the anatomical location of arteritis in a unique case of necrotising arteritis apparently confirmed to the ovarian hilum suggests that this is the female counterpart of isolated arteritis of the epididymis.1

A 36 year old woman had undergone hysterectomy for menorrhagia some two years previously and had since been complaining of intermittent pelvic pain. She had an unremarkable obstetric history (parity 4 + 0), had had no serious illnesses, was taking no medication, and was in good general health. At laparoscopy, a left tubal ovarian mass with adhesions was noted and a vaginal vault granulation was diathermed.

The patient was keen to have her ovarian mass removed and a bilateral salpingo-oophorectomy was performed three days after the laparoscopy. The operation was uncomplicated and the patient made an uneventful postoperative recovery.

In routine histological sections of the right ovary there was a focus of necrotising vasculitis which was apparently confined to two or three small muscular arterial branches close to the rete ovarii. The arteritic lesions were characterised by focal fibrinoid necrosis

Fig 1 · Ovarian hilar arteritis. Acute arteritis with fibrinoid necrosis and inflammation affecting part of the circumference of a small muscular arterial branch.

1484
the vessel wall with an associated infiltrate of neutrophil polymorphs, plasma cells and lymphocytes (fig 1). Eosinophil polymorphs were not seen. All the other hilar vessels showed no clinically important abnormality. Paraffin sections were stained using a standard peroxidase-antiperoxidase technique and positive staining for IgG, IgM, and Clq was seen in the arteritic lesions. The left ovary contained an endometriotic cyst. Histological material from the patient's original hysterectomy specimen was reviewed and no important abnormality found. When step sections of this material were cut, a definite arterial lesion was identified in the cervix (fig 2). To exclude systemic disease the patient underwent a full medical examination and several blood tests (including erythrocyte sedimentation rate, compliment concentrations, and antibody screen) and these all yielded normal results. The patient has been followed up for 15 months and apart from persistent pelvic pain remains in good health at the time of writing.

The aetiology of isolated arteritis is unknown. Many forms of systemic arteritis are considered to be mediated by immunological mechanisms, and isolated arteritis may represent a localised type III hypersensitivity (Arthus) reaction.² Antigens capable of initiating an Arthus type reaction in the ovarian hilum could ascend an intact and patent female genital tract. In this patient previous hysterectomy and the acute nature histological of the lesions make an ascending antigen very unlikely as a cause of ovarian hilar arteritis and it is unclear why the lesions should be apparently confined to branches of the right ovarian artery.

This patient had evidence of endometriosis and it is well recognised that foci of endometriosis which bleed usually cause a local inflammatory reaction. Necrotising arteritis is not a recognised association, and in this patient there was no endometriosis or haemosiderin deposition in the right ovarian hilum and the only inflammation was in the arteritic lesions. The possibility of a more subtle association between the arteritis and endometriosis cannot be excluded. Anti-endomterial antibodies have been described in patients with endometriosis² and whether this is an epiphenomenon or not, these antibodies and their antigens could be important in the pathogenesis of the localised acute arteritis. The association between the ovarian and cervical lesions was interesting. Although there was no acute necrotising arteritis in the cervix, the changes seen could represent resolving lesions. The hysterectomy was undertaken two years prior to the oophorectomy so that the cervical and ovarian hilar lesions were distinct anatomically and temporally. Furthermore, in other series of isolated arteritis of the cervix⁴ the disease was apparently confined to that site, although the state of the ovarian hilar vessels is not mentioned specifically.

Polyarteritis nodosa may affect the female genital tract⁶ and the diagnosis of isolated arteritis rests on the exclusion of systemic disease.

C WOMACK
Department of Pathology,
Peterborough District Hospital,
Thorpe Road,
Peterborough PE3 6DA.

References