Tamoxifen and liver damage

Tamoxifen in the treatment of metastatic carcinoma of the breast is usually well tolerated with few side effects. We report on a patient in whom tamoxifen was associated with deterioration of liver function.

Case report

A 75 year old woman, who had undergone radical mastectomy in 1978 for breast carcinoma, developed bony metastases by April 1982 and was treated with tamoxifen 10 mg twice daily. Ten weeks later she developed nausea, vomiting, and hip pain due to progressive skeletal disease. Serum liver enzyme activities, serum calcium concentrations, and a liver scan were all normal. Prednisolone 5 mg twice daily was added to her treatment, but, despite regular antiemetics, nausea and vomiting persisted. Serum bilirubin concentration and aspartate aminotransferase activity continued to rise (figure). All drugs except diazepam and prednisolone were stopped on 29 July, and aspartate aminotransferase activity and bilirubin concentration fell for two weeks (figure). Serological tests for hepatitis A and B proved negative, and an ultrasound scan of the liver was normal. A liver biopsy carried out 11 days after tamoxifen was stopped showed slightly swollen hepatocytes, cholestasis, prominent Kupffer's cells filled with pigment, and an inflammatory portal lesion with minimal duct changes.


Severe peripheral ischaemia during concomitant use of beta blockers and ergot alkaloids

Certain drugs may adversely affect perfusion through peripheral tissue. Ergot alkaloids, for instance, cause arterial vasoconstriction, which may give rise to ischaemia leading to gangrene of the toes and fingers. Blockers may diminish blood flow to striated muscle by inhibiting beta mediated vasodilatation, by causing beta mediated depression of cardiac output, and possibly through cerebral mechanisms. Clinical signs of this condition may be cold fingers and toes and even peripheral gangrene. Blockers used in conjunction with ergot derivatives may have a synergistic effect on perfusion through peripheral tissue, which could be hazardous. We report two cases in which this interaction may have occurred.

Case reports

CASE 1
A 21 year old policeman was admitted to hospital with progressive severe pain in his legs and feet of one week's duration. He had suffered from migraine for 10 years and had been taking methysergide (3 mg) and propanolol (120 mg) daily for two years. On admission his feet were pale, cold, and poorly perfused and only the femoral pulses were palpable in his legs. Aortography disclosed extreme bilateral narrowing of the superficial femoral arteries consistent with severe spasm. Intravenous infusions of 10% dextrose 40 in saline (Rhopharmcicides) and heparin (500 units/hour) and a lumbar epidural block with 1% lignocaine failed to afford any improvement.