

been available. Loss of consciousness in those carrying sugar occurred either because hypoglycaemic symptoms were not recognised or because the onset of hypoglycaemia was particularly swift.

Comment

Although the true incidence of hypoglycaemia is unknown, up to 90% of adult insulin dependent patients have experienced hypoglycaemic symptoms.³ Although great emphasis is placed on recognising symptoms and early self treatment, nearly a third of our patients were not aware of or did not follow the recommendations of the British Diabetic Association on hypoglycaemia. Serious hypoglycaemic episodes occur in up to 9% of insulin dependent diabetics each year.⁴ If the British Diabetic Association's guidelines were followed unnecessary morbidity and mortality related to hypoglycaemia could be prevented.

The failure of 23.0% of the drivers in our study to carry a separate easily accessible supply of sugar in the car puts not only themselves but other road users at increased risk. Up to 40% of insulin dependent diabetics who drive experience hypoglycaemia while at the wheel of a car,⁵ and a separate source of glucose in the vehicle is therefore essential as jackets and handbags are easily forgotten.

Those caring for diabetics need to educate their patients (particularly drivers) on the hazards of hypoglycaemia and should check that their advice is being heeded. The sound advice of the British Diabetic Association² is clearly not being taken by up to a third of our insulin dependent diabetics.

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Possible reactivation of hepatitis D with chronic δ antigenaemia by human immunodeficiency virus

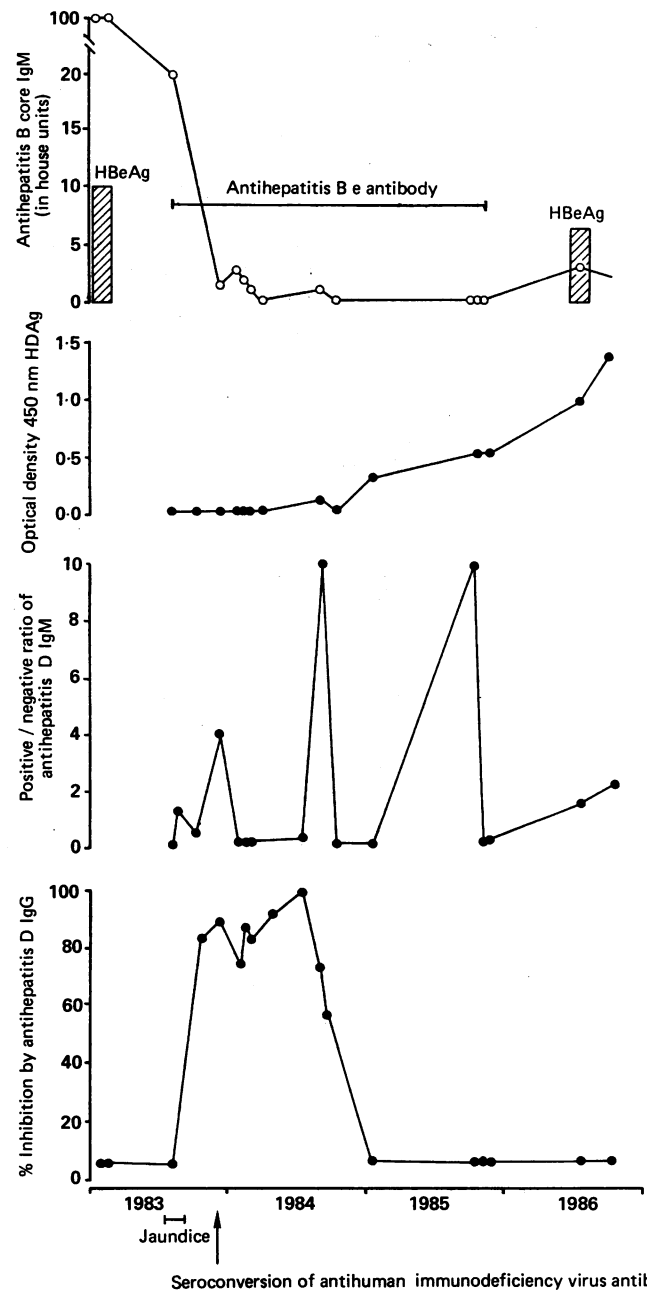
We describe a case of reactivation of infection with hepatitis D virus (δ) in an intravenous drug abuser who was a carrier of hepatitis B surface antigen (HBsAg). This condition was manifested by chronic δ antigenaemia of at least two years' standing and was probably induced by infection with human immunodeficiency virus.

Case report

Detailed serological tests for hepatitis B and D viruses were carried out on a series of specimens from a 21 year old male drug abuser in whom acute hepatitis B had been diagnosed in February 1983. At that time HBsAg was detected by commercial radioimmunoassay and enzyme immunoassay kits (Abbott); hepatitis B e antigen (HBeAg) and antihepatitis B core IgM were detected by in house enzyme immunoassays.¹ There was no jaundice and his liver enzyme activities were slightly raised. Jaundice of three weeks' duration occurred six months later, however, in August 1983.

Antihepatitis D IgG was detected in October 1983 by enzyme immunoassay (Deltassay B, Noctech). Antihepatitis D IgM was also detected by an in house enzyme linked immunosorbent assay (ELISA) using hepatitis D antigen (HDAG) derived from serum, which indicated that his hepatitis in August had been caused by a superinfection with hepatitis D virus. Antihepatitis D IgG persisted throughout 1984.

Reactivation of infection with hepatitis D virus was first detected in July 1984, when transient HDAG appeared resulting in seroconversion to antihepatitis D IgM. Reactivation was again detected in January 1985, after which he showed increasing amounts of serum HDAG (detected by enzyme immunoassay Deltassay, Noctech) with loss of antihepatitis D IgG and fluctuating amounts of antihepatitis D IgM.



Temporal changes in results of detailed serological tests for hepatitis B and D viruses in 21 year old drug abuser.

Tests for antibody to human immunodeficiency virus were carried out retrospectively. Enzyme immunoassay gave an equivocal result at first, but a Western blot analysis (using DuPont prepared nitrocellulose strips) produced a positive result in his specimen of October 1983; all subsequent tests (Wellcome enzyme immunoassay) remained positive. The activities of his liver enzymes were persistently raised and variable throughout, and a liver biopsy in October 1984 indicated the presence of chronic active hepatitis. In October 1986 he had lymphadenopathy but had not returned for follow up at the time of writing.

Comment

The serological results strongly suggest that our patient acquired superinfection with hepatitis D virus and human immunodeficiency virus about six months after acute infection with hepatitis B virus. The unusual serological pattern and the rising concentration of HDAG in serum strongly suggest reactivation of replication of hepatitis D virus. Furthermore, his most recent specimens showed reversion from antihepatitis B e antibody to HBeAg, which indicates reactivation of hepatitis B virus as well. Unfortunately the patient has been lost to follow up and the importance of this reactivation of hepatitis D virus is therefore unknown. Although this is the first reported occurrence of reactivation of infection with hepatitis D virus by human immunodeficiency virus, the large numbers of drug abusers

in Dublin who are infected with hepatitis D virus² and the increase of such infections in proportion with infections with human immunodeficiency virus (A G Shattock *et al*, international symposium on AIDS, Paris 1986) make it likely that other reactivations of infection with hepatitis D virus will occur in this susceptible population.

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Loin pain and haematuria syndrome: possible association with intrarenal arterial spasms

Loin pain and haematuria syndrome is characterised by repeated attacks of unilateral or bilateral loin pain with haematuria in patients with normal intravenous pyelograms and cystoscopy findings and sterile urine.^{1,2} We report the angiographic findings of four such patients studied in 1979-81 and followed up subsequently.

Patients, methods, and results

During 1979-81 we diagnosed loin pain and haematuria syndrome in four women (mean age 43.5 years (range 38-49)). They all had attacks of severe, incapacitating loin pain associated with microscopic haematuria. The mean duration of their symptoms at the time of diagnosis was 5.8 years (range 2-10). Bleeding times and coagulation function were normal in all patients. Urine was sterile, and no proteinuria was found. Urine cytology, intravenous urography, isotope nephrography, cystoscopy, and ultrasound examination of the kidneys showed no abnormalities. Gynaecological causes of haematuria were excluded. Phase contrast microscopy to determine the origin of the red cells in urine was not performed.

No clinical, microbiological, or serological evidence of systemic autoimmune disease or triggering or precipitating infection was found. All patients were normotensive with normal renal function. Serum concentrations of calcium, phosphate, chloride, sodium, potassium, uric acid, and parathyroid hormone were normal, as were urinary excretions of calcium and phosphate.

A needle biopsy sample taken from the side with loin pain showed only slight arteriolar hyalinosis or mild mesangial cell proliferation. Immunofluorescence studies showed minor deposits of C3 in the mesangium and in the walls of renal arterioles.

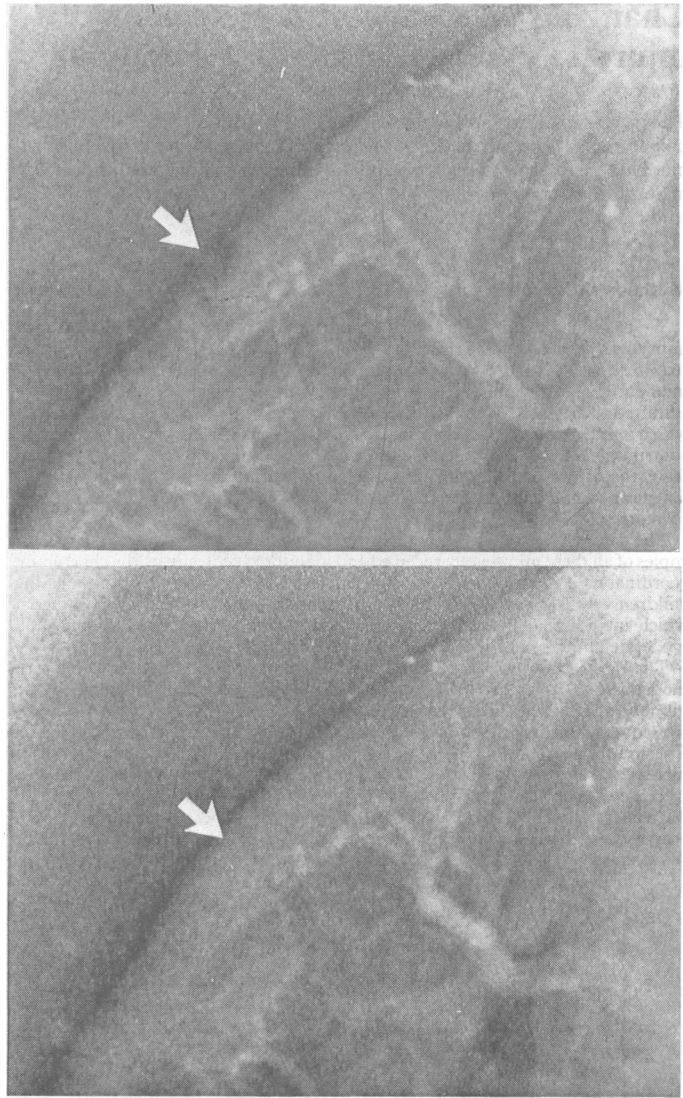
Selective renal angiography with magnification showed no irregularity of the lumen, changes in calibre, or occlusion. In two patients, however, transient vasospasms in intrarenal vessels were noted (figure); in one of these patients the arterial spasms occurred on both sides. During the five year follow up period no underlying diseases or other abnormalities developed.

Comment

Abnormalities in intrarenal peripheral arteries shown by selective renal angiography have been consistent findings in previous reports.^{1,2} In our study angiography showed no structural vascular changes in any of the four patients. Transient vasospasms in renal cortical vessels, however, were noted in two patients. According to previous reports renal artery spasms are rarely encountered during routine angiography.³ In a series of selective renal angiograms studied using the same magnification technique we found transient arterial spasm in only one of 33 kidneys studied. The present finding of vasospasm in patients with loin pain and haematuria syndrome is therefore unlikely to be merely coincidental.

The renal histological findings in loin pain and haematuria syndrome are similar to those in IgA nephropathy.⁴ Our findings on light microscopy and immunofluorescence studies are in accordance with earlier reports.

Fletcher *et al* suggested that the loin pain might be caused by renal cortical ischaemia due to vascular occlusions and thrombi.⁴ The disappearance of the pain after autotransplantation suggests, however, that it is mediated through



Above: Focused magnification of a selective right renal angiogram which, during the first injection in the arterial phase, shows a cortical perfusion defect (arrow) caused by a vasospasm in a renal cortical vessel. Below: Focused magnification of the same area. No vasospasm is seen during the second injection, given five minutes after the first.

the autonomic nervous system.⁵ Our angiographic findings support this hypothesis. Our findings also show that loin pain and haematuria syndrome may occur in patients who do not have structural changes in the intrarenal peripheral arteries.

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