normal serum IgG, IgM, and complement concentrations and normal results of serum electrophoresis. Negative or normal results were obtained for hepatitis B surface antigen, rheumatoid factor, antinuclear factor, double stranded DNA antibody, the Venereal Disease Research Laboratory test, anti-streptolysin O titre, and cryoglobulins. An intravenous pyelogram was normal.

Percutaneous renal biopsy showed 37 glomeruli, of which one was completely and some were partially sclerosed. Many showed mild mesangial hyperplasia, and some had capsular adhesions. Many arcuate and interlobular arteries were thicker than normal; there were focal areas of interstitial fibrosis. Immunofluorescence showed moderate granular mesangial deposition of IgA and C3 with moderate granular extraglomerular vessel deposition of C3. Occasional definite mesangial electron dense deposits were seen on electron microscopy.

Comment

This patient had mesangial IgA nephropathy with historical evidence of episodic macroscopic haematuria associated with mastitis. Haematuria associated with pharyngitis has been well documented, but to our knowledge this is the first reported case of haematuria associated with mastitis.

The pathogenesis of this disease is unclear. Circulating immune complexes containing IgA and lymphocytes bearing IgA were both detected in the serum, and there was evidence of decreased IgA specific suppressor T cell activity.^{2,3} IgA is synthesised by submucosal plasma cells in the respiratory and gastrointestinal tracts with increased production after local infection; the secretory component of IgA, however, is absent from glomerular lesions.⁴ The precise mechanism by which pharyngitis is linked to exacerbations of haematuria has not yet been elucidated.

Breast milk and colostrum are rich sources of IgA and the secretory component, which has been synthesised in vivo from cultured colostrum mononuclear cells.⁵ The close temporal association of repeated episodes of mastitis and macroscopic haematuria in this case is supportive evidence for an extrarenal origin of the glomerular IgA deposits.

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Minimal change nephropathy in the acquired immune deficiency syndrome

In patients with the acquired immune deficiency syndrome (AIDS) renal disease is unexpectedly common, focal and segmental glomerulosclerosis being the major cause of heavy proteinuria.¹⁻³ We report the previously undescribed association of minimal change nephropathy with heavy proteinuria in a patient with AIDS.

Case report

A 29 year old heterosexual man from the West Indies was admitted with fever, malaise, and weight loss of several weeks' duration. Two years previously he had developed opportunist infections (cryptococcal meningitis and infection due to herpes simplex virus type II) and been found to have hepatitis B virus e antigenaemia. Renal function had been normal, with no proteinuria. He denied abuse of intravenous drugs and homosexual contact and had never received blood products.

On examination he was febrile and cachectic. There was diffuse alopecia, a macular truncal and perianal rash, and generalised lymphadenopathy. The liver was palpable 5 cm below the costal margin. Funduscopy showed multiple cotton wool spots, and there was left lower quadrantanopia. Investigations showed: urea concentration 4 mmol/l (24 mg/100 ml), creatinine 78 μ mol/l (0.9 mg/100 ml), albumin 27 g/l, alkaline phosphatase 445 IU/l, aspartate transferase 168 IU/l, 24 hour urine protein 3.3-5.9 g, haemoglobin 100 g/l, white cell count 2.2×10^9 /l, lymphocytes 24% (0.5×10^9 /l), and platelet count 184×10^9 /l. A chest x ray film and intravenous urogram were normal.

Percutaneous renal biopsy showed 23 glomeruli: one was totally sclerosed, and 22 showed nil change on light microscopy. There was foot process fusion but no other appreciable abnormality on electron microscopy, and immunoperoxidase was negative for immunoglobulins G, A, and M and for C3. Liver biopsy showed only mild fatty change without evidence of chronic active hepatitis; there were nuclei of bizarre shape but no electron microscopic evidence of viral infection. No infective cause for the fever was found, but a computed tomogram showed multiple intracerebral lesions, found on biopsy to be primary cerebral lymphoma.

During the illness the ratio of T helper to suppressor cells fell from 2.0 to 0.19 (normal range 1-3). During treatment for the lymphoma with deep x ray therapy, daunorubicin, cyclophosphamide, vincristine, and dexamethasone he became severely neutropenic and died of septicaemia. His serum was subsequently found to be positive for antibody to human T cell lymphotropic virus III.

Comment

Proteinuria is common in patients with AIDS. Fourteen patients with mild proteinuria had no light microscopic changes in the glomeruli and lacked typical electron microscopic features of minimal change nephropathy as seen in our patient.⁴ Such mild proteinuria may occur as the result of infection, haemodynamic change, and nephrotoxic drugs.

The occurrence of heavy proteinuria (>3 g/24 hours) in AIDS requires further explanation. Abuse of heroin is typically associated with focal and segmental glomerulosclerosis⁵; indeed, 12 of 21 patients with heavy proteinuria and AIDS abused intravenous drugs, and nine of these had focal and segmental glomerulosclerosis on biopsy.¹⁻³ In the remaining nine patients, however, no cause was identified for their renal disease (focal and segmental glomerulosclerosis in six, mesangial proliferation in three).

Minimal change nephropathy is usually idiopathic but may be associated with an altered immune environment, as in patients with lymphoid tumours. In AIDS the pathogenesis may be lymphokine mediated injury; typical glomerular changes have been induced by supernatant from lymphocyte cultures from patients with idiopathic minimal change lesions.⁶

Our patient did not admit to any of the classical risk factors for AIDS; in view of the initial presentation with genital ulceration, however, the probable source was sexual contact. Neither proteinuria nor evidence of lymphoma was evident at the initial presentation, clinically or on computed tomography. Although minimal change nephrotic syndrome induced by tumour was a possibility, because of the patient's rapid death we could not assess whether treatment of the haematological malignancy would have led to remission of the renal lesion and hence whether the pathogenesis was related to the tumour or to the altered immune state of AIDS.

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