tive effect of oral oestrogens and show an apparent increase in bone mass after treatment with subcutaneous oestradiol and testosterone.

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Reversal of renal failure in nephritis associated with antibody to glomerular basement membrane

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Nephritis associated with antibody to glomerular basement membrane generally presents as rapidly progressive renal failure. Histological examination shows crescentic nephritis with linear deposits of IgG on the glomerular basement membrane. Pulmonary haemorrhage may occur. Effective treatment comprises immunosuppression and plasma exchange.¹ Recovery of useful renal function is considered to be unlikely but has been reported in patients dependent on dialysis.² We describe such a case in a patient with acute oliguric renal failure.

Case report

A 35 year old woman was referred in March 1987 with a one month history of tiredness and nausea. She complained of haematuria and oliguria but did not have any respiratory symptoms. She had a history of essential hypertension, which had been treated with a thiazide diuretic. Her renal function had been normal eight months earlier. On examination her blood pressure was normal and she did not have a rash. Radiographic examination of her chest showed no abnormalities. Her haemoglobin concentration was 101 g/l, urine volume 0.4 l, serum creatinine concentration 435 μ mol/l, and creatinine clearance 12 ml/minute. Her urine showed haematuria and red cell casts on microscopy.

Two days later her renal function had deteriorated further (serum creatinine concentration 715 μ mol/l and creatinine clearance 2 ml/minute). She underwent haemodialysis, after which percutaneous renal biopsy

was performed. On histological examination two of the four glomeruli obtained showed florid necrotising crescentic glomerulonephritis; there was an inflammatory reaction around the glomeruli and associated tubulointerstitial tissue. The other two glomeruli showed mild mesangial hypercellularity. No arteriolar lesions were observed. Immunofluorescence showed strongly positive linear deposits of IgG along the surviving capillary walls of the glomeruli. Her serum contained a high titre (80%) of antibody to glomerular basement membrane (normal value <12%) and a transient low titre of antibody to the cytoplasm of neutrophils on radioimmunoassay.

Treatment with 3 mg cyclophosphamide/kg, 60 mg prednisolone, and daily plasma exchange (4 litres) was started. The titre of antibody to glomerular basement membrane decreased to normal values (figure). She

Changes in titre of antibody to glomerular basement membrane and creatinine clearance during treatment with plasma exchange and cyclophosphamide and prednisolone

remained dependent on dialysis for four weeks; subsequently her renal function gradually recovered almost to normal (serum creatinine concentration 140 μ mol/l and creatinine clearance 60 ml/minute). Plasma exchange was stopped after six weeks and cyclophosphamide after eight weeks. Ten months after

presentation she remained well and did not require further immunosuppression.

Comment

We believe that this case shows that nephritis associated with antibody to glomerular basement membrane, which rapidly leads to the patient becoming dependent on dialysis, may be reversible if early diagnosis and treatment are possible. Because of the few glomeruli in the biopsy specimen assessment of the extent and severity of the disease was difficult. The presence of some fairly normal glomeruli and the short history of rapidly progressive renal failure suggested that treatment might be effective. The assay for determining circulating antibodies to glomerular basement membranes was invaluable in monitoring the activity of the disease and encouraged us to persist with treatment as the antibody titres declined. The presence of antibody to the cytoplasm of neutrophils initially is interesting, but its importance in this patient is unclear. It has been reported as being of diagnostic value in Wegener's granulomatosis and microscopic polyarteritis.³ Our patient did not have features of vasculitis. The basic histological diagnosis was crescentic nephritis due to antibody to glomerular basement membranes.

The poor prognosis of patients with this condition, which is associated with renal failure necessitating dialysis, has led to doctors being reluctant to undertake or persist with prolonged immunosuppression and plasma exchange.^{1,5} We suggest that patients with renal impairment of recent onset and with some viable glomeruli on histological examination may regain renal function despite having needed dialysis for several weeks.

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Highly sensitive screening tests for hepatitis B surface antigen in transfusion centres of developing countries

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Although routine screening of donor blood for hepatitis B surface antigen (HBsAg) is desirable in our region, which has a moderately high prevalence of carriers,¹ screening is reported not to be cost effective in west Africa.² Therefore we assessed the risks, costs, and benefits when blood from donors was not screened or was screened with tests of low, medium, and high sensitivity. We avoided giving transfusions of infectious blood but calculated the risk of infection from the prevalence of carriers of HBsAg in the donor population and of susceptibility to infection among potential recipients.

Methods and results

Serum from 500 consecutive volunteer blood donors was screened for HBsAg by counterimmunoelectrophoresis,³ reverse passive haemagglutination with commercial kits, and enzyme linked immunosorbent assay (ELISA) (Auszyme monoclonal, Abbott, Chicago). Serum from 500 consecutive patients who were cross matched for transfusion was screened sequentially for

HBsAg, antibody to hepatitis B core antigen (Corzyme, Abbott), and antibody to HBsAg (Ausab, Abbott). All serum samples positive on ELISA were retested to confirm the result.

Four (0.8%) of the donors were positive for HBsAg on counterimmunoelectrophoresis; 12 (2.4%) yielded positive results on reverse passive haemagglutination screening, 11 (2.2%) of which were confirmed by reverse passive haemagglutination blocking tests; and 35 (7%) yielded positive results on ELISA (table). All samples of serum positive on counterimmunoelectrophoresis were positive on reverse passive haemagglutination, and all samples positive on reverse passive haemagglutination were positive on ELISA. The sensitivities of counterimmunoelectrophoresis and reverse passive haemagglutination were only 11% and 31%, but their specificities were 100% and 99%, respectively.

Among the 500 potential recipients of transfusions 99, 108, and 39 were positive for HBsAg, antibody to hepatitis B core antigen, and antibody to HBsAg, respectively; 254 (50.8%) did not have any markers of the virus and were thus susceptible to infection. Without screening, the probability of one unit of infectious blood being given to a susceptible individual was 0.036 (50.8% × 7%). With routine screening by counterimmunoelectrophoresis the risk of infection from a three unit transfusion reduced from 0.108 (0.036 × 3) to 0.0945 (50.8% × 6.2% × 3). Routine screening with reverse passive haemagglutination decreased the risk to 0.073 (50.8% × 4.8% × 3).

Counterimmunoelectrophoresis, reverse passive haemagglutination, and ELISA cost US \$0.50, \$1.00,

Risks of transfusing blood positive for hepatitis B surface antigen (HBsAg) with different screening tests

Screening test	% Of units positive for HBsAg detected	% Of units positive for HBsAg not detected*	Risk of an uninfected recipient receiving HBsAg if he or she receives:			% Of transfusions (of 3 units) in which infection averted by screening†
			1 Unit	2 Units	3 Units	
None	0	7.0	0.036	0.072	0.108	0
Counterimmunoelectrophoresis	0.8	6.2	0.031	0.063	0.094	1.4
Reverse passive haemagglutination	2.2	4.8	0.024	0.049	0.073	3.5
Enzyme linked immunosorbent assay (ELISA)	7.0	0	0	0	0	10.8

*Percentage detected by ELISA minus percentage detected by other method.

†(Risk associated with unscreened blood minus risk associated with given method) × 100.