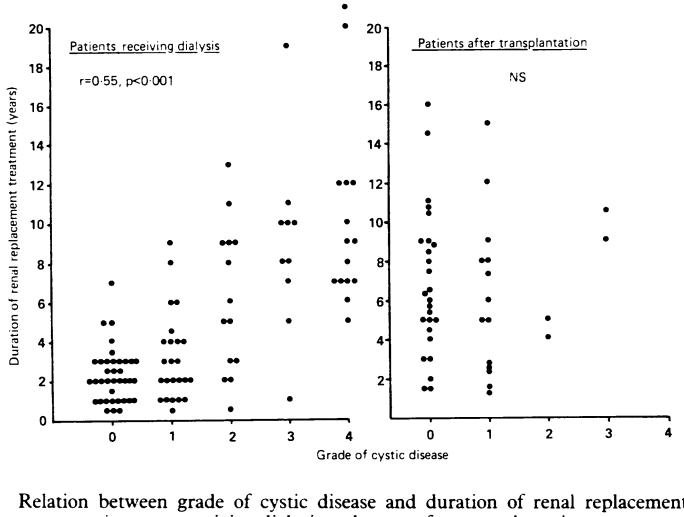
Patients, methods, and results

We studied 45 patients aged 18-72 (mean 47) who had had a functioning transplant for one to 14 (mean 4·2) years and 100 patients aged 18-78 (mean 51) who had been receiving dialysis for one to 21 (mean 4·8) years. There were no significant differences between the two groups in age, sex, or nature of underlying disease. No patient had polycystic kidney disease of adult onset. Thirty two of the patients receiving dialysis were receiving chronic ambulatory peritoneal dialysis. In the transplant group the mean total duration of renal replacement treatment—that is, the duration of dialysis plus the duration of the transplant—was 6·7 years and the mean duration of dialysis before transplantation 2·5 years.

Each patient underwent real time ultrasonography performed by one of two experienced radiologists, who used ultrasound equipment of identical resolution. Each scan was assessed by one radiologist without reference to details of the patient and graded 0-4 according to the number of cysts per kidney (0=no cysts, 1=<5, 2=5-9, 3=10-14, and 4=≥15).

Eighteen patients (40%) in the transplant group had acquired cystic disease of their native kidneys (14 grade 1, two grade 2, and two grade 3). No cysts were detected in any transplanted kidney. Sixty three patients (63%) receiving dialysis had acquired cystic disease (24 grade 1, 14 grade 2, 10 grade 3, and 15 grade 4). One patient receiving dialysis had a solid lesion in one kidney that proved to be renal adenocarcinoma. There was no correlation in the transplant group between the number of cysts and the total duration of renal replacement treatment (Kendall rank correlation) (figure). In addition, there was no correlation with



Relation between grade of cystic disease and duration of renal replacement treatment in group receiving dialysis and group after transplantation.

either the duration of the transplanted kidney or the duration of dialysis before transplantation. There was a highly significant correlation between the number of cysts and the duration of dialysis in the dialysis group (p<0·001, Kendall rank correlation) (figure). Within this group there were no significant differences between those treated by haemodialysis and those treated by chronic ambulatory peritoneal dialysis.

Comment

This study showed that acquired cystic disease of the kidneys progresses in patients treated by dialysis alone but not in patients with a functioning renal transplant. In addition, the lack of correlation between the number of cysts and the duration of dialysis before transplantation suggests that the disease may regress after successful transplantation. Cystic disease is an important complication of both haemodialysis and chronic ambulatory peritoneal dialysis and is likely to increase in prevalence as the number of patients receiving long term dialysis grows. It can cause intrarenal and extrarenal haemorrhage, which may be fatal.[2,3] The most important complication, however, is an apparently greatly increased risk of neoplasm arising in kidneys that have undergone multicystic transformation. Gardner calculated that patients with cystic disease are 14 times more likely to develop renal adenocarcinoma than patients with chronic renal failure without cystic disease.[4] We detected one neoplasm in this study, and in addition we previously reported on a patient with acquired polycystic disease who died from metastatic renal adenocarcinoma.[5] Our finding that acquired polycystic disease of the kidneys is much less severe after transplantation may therefore be an important consideration in the choice of long term renal replacement treatment.

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Severe illness associated with appearance of antibody to human immunodeficiency virus in an African

Encephalopathy and acute illnesses similar to mononucleosis associated with the appearance in serum of antibody to human immunodeficiency virus (HIV)[1-3] have not previously been reported in Africa.

Case report

A previously healthy 35 year old African man became ill in November 1984 (day 0) with vague malaise and fatigue. The next day he developed joint pains and fever (39°C), which persisted over the next 13 days. Additional problems noted on day 2 included pharyngitis, diarrhoea, and vomiting and a pruritic maculopapular rash on the face. By day 3 the rash extended to the trunk and occipital lymphadenopathy was noted. On day 4 shallow ulcers were observed on the palatal and gingival mucosa and on the penis and scrotum. He was admitted and during the next five days became progressively obtunded to the extent that he was unable to speak or recognise people; this state persisted until the 15th day, when all his symptoms dramatically improved. He was released from hospital on day 19. Antibiotics (penicillin analogues and tetracycline) were given from day 2 but had no apparent effect. During the three weeks after discharge his condition continued to improve but he developed generalised lymphadenopathy; biopsy showed only non-specific reactive hyperplasia. He resumed work at nine weeks. At his last visit (14 months) he was working full time and had regained all lost weight. The lymphadenopathy had gradually lessened from the end of the fourth month but was still present.

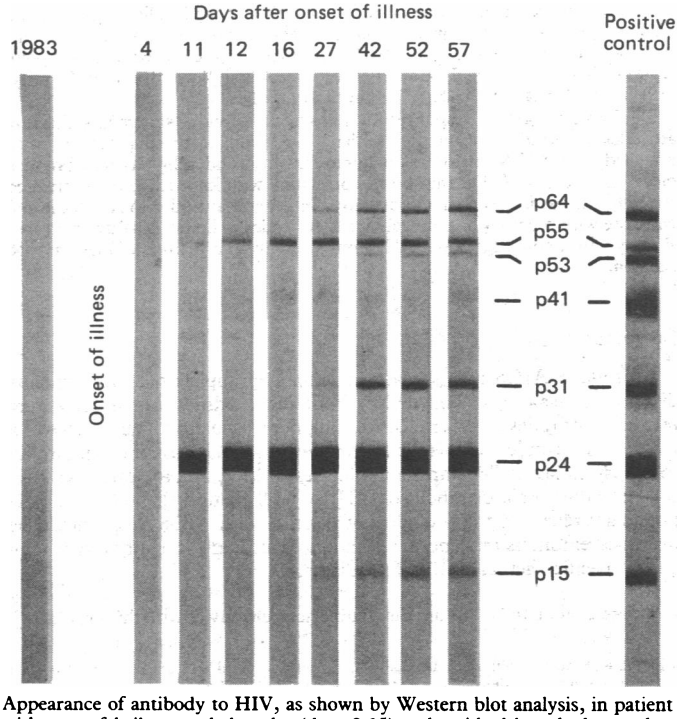
The results of Western blot analyses (figure) and enzyme linked immunosorbent assay clearly showed that antibody to HIV was present in the serum by day 11. The leucocyte count was initially $5{\cdot}0\times10^9$/l (25% lymphocytes) and gradually rose to $12{\cdot}9\times10^9$/l (29% lymphocytes) by the day of discharge. From day 11 onward, coincident with the development of antibodies, lymphocytes (up to 10%) were consistently described as reactive. Results of liver function studies were abnormal during the second week of illness. Monospot tests and tests for febrile agglutinins yielded negative results, and the titres of antibodies against Epstein-Barr virus, cytomegalovirus, and toxoplasmosis in serum obtained before and after the illness were similar. Cerebrospinal fluid, examined twice during the second week, showed only slight lymphocytosis (10 cells and 22 cells) and no evidence of cryptococcus. Bacterial cultures and blood smears for malaria yielded negative results. There were no metabolic abnormalities. To detect viruses serum, cerebrospinal fluid, and scrapings from the oral ulcers collected during the acute illness were inoculated into suckling mice and on to Vero cell cultures, all with no effect. The ratio of T lymphocyte helper to suppressor cells at nine weeks was inverted (0·5), primarily because of a high proportion of suppressor cells (22% helpers, 42% suppressors; leucocyte count $6{\cdot}9\times10^9$/l, 68% lymphocytes). At seven months the ratio remained inverted (0·85).

The patient did not know of any exposure to patients with the acquired immune deficiency syndrome or to needlesticks during the past year, denied homosexuality and drug abuse, and had never received a blood transfusion. He had not travelled outside Kenya since 1982. During the past year he had had many female sexual partners but no contact with prostitutes or venereal disease.

Comment

No other report has documented the acquisition of antibodies in relation to clinical symptoms in similar detail. Antibody against p24 and p55

appeared earliest, as previously reported,[4 5] and before day 11 of illness, coincident with the appearance of activated lymphocytes. Antienvelope protein antibodies, p41, and gp120/160 (data not shown; Gerald Robey) were first detected faintly at day 16, coincident with clinical improvement.



Appearance of antibody to HIV, as shown by Western blot analysis, in patient with acute febrile encephalopathy (days 2-15) and residual lymphadenopathy. Western blot analysis was performed with HIV grown in H9 cell lines. Serum obtained during the illness was analysed in the same tray using an avidin-biotin-peroxidase technique to show antibody attachment to HIV polypeptides.[4] Serum from 1983 was available from a previous study.

Antibodies against other antigens (p15, p31, p53, p64) appeared before day 27, coincident with the development of persistent generalised lymphadenopathy. Thus severe symptoms including an illness similar to mononucleosis, encephalopathy, and persistent generalised lymphadenopathy may occur as a consequence of primary HIV infection. Whether this profile development of antibody to HIV is typical of other seroconversions remains unknown.

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Transient ascites in progressive systemic sclerosis

The differential diagnosis for ascites of sudden onset is extensive.[1] We report transient ascites in two patients with progressive systemic sclerosis.

Case 1

A 64 year old woman had a 17 year history of progressive systemic sclerosis, manifested by Raynaud's syndrome, sclerodactyly, calcinosis, telangiectasia, and dysphagia. She developed tense ascites over three days, with oedema to mid-abdomen. No other abnormal clinical findings were present. Treatment was with penicillamine 300 mg, nifedipine retard 40 mg, and inositol nicotinate 2 g daily. Haemoglobin and electrolyte concentrations, erythrocyte sedimentation rate, and results of liver function tests (including prothrombin time and albumin concentration) were normal. α Fetoprotein and carcinoembryonic antigen were not detected. The anticentromere antibody titre was positive. Ascitic fluid contained 12 g protein/l, and the cell count was 80×10^6 cells (predominantly lymphocytes)/l. Gram staining, culture, and cytological examination yielded negative results. Urine did not contain any protein. A chest *x* ray film, echocardiogram, isotope liver scan, and hepatic phlebogram were normal. Ultrasonography and computed tomography of the abdomen confirmed ascites; findings were otherwise normal. Barium studies showed abnormalities of the oesophagus, stomach, small bowel, and proximal colon consistent with progressive systemic sclerosis. Endoscopic biopsy of the oesophagus, stomach, and jejunum did not show any evidence of malignancy.

The oedema and ascites resolved with diuretic treatment and had not recurred 18 months later (July 1986), when she was no longer taking diuretics, though the other drugs were maintained at the same dose.

Case 2

A 59 year old woman developed ascites over six weeks. She had facial telangiectasia and sclerodactyly. At laparotomy 6 litres of straw coloured fluid was removed. The liver was normal, and biopsy was performed. A "fine white mesh like appearance" of the small bowel was noted at operation. Haemoglobin and electrolyte concentrations, erythrocyte sedimentation rate, and results of liver function tests were normal. The anticentromere antibody titre was positive. Carcinoembryonic antigen was not detected, and the α fetoprotein concentration was 204 kU/l (normal <15 kU/l). Results of histological examination of the liver biopsy specimen, 24 hour urinary protein excretion, and a chest *x* ray film were normal. Radiography of the hands showed calcinosis circumscripta. Barium studies showed diminished oesophageal peristalsis, dilatation of the small bowel from mid-jejunum to ileum, and normal large bowel. Ultrasonography of the abdomen showed patent hepatic veins and no abnormality of the liver. α Fetoprotein concentration at three months and subsequently was <5 kU/l.

Nineteen months later the ascites had not recurred and she was otherwise well; she was not receiving any treatment.

Comment

These patients with progressive systemic sclerosis presented with ascites of recent onset. Extensive investigation excluded recognised causes of ascites, including liver disease, the Budd-Chiari syndrome, right heart failure, constrictive pericarditis, the nephrotic syndrome, and occult malignancy. Only one other case of otherwise unexplained ascites has been reported in a patient with progressive systemic sclerosis, who was positive for hepatitis B surface antigen and had exudative ascites with unexplained bony erosions.[2] No follow up information was given, and it is difficult to exclude recognised causes of ascites. In other series of patients with progressive systemic sclerosis ascites has been reported only in association with hepatic fibrosis[3] and cirrhosis.[4]

Pleural and pericardial fibrosis and effusions are well recognised in progressive systemic sclerosis and have been shown at necropsy to be associated with serositis.[3-5] One detailed necropsy study, however, showed that peritonitis and peritoneal adhesions were no more common in 57 patients with progressive systemic sclerosis than in controls.[5] Abnormalities of the bowel wall are well recognised in progressive systemic sclerosis and were documented in our patients; low grade peritoneal inflammation might well result in ascites in such patients. As the ascites resolved rapidly the causative factor was presumably transient. We suggest that the differential diagnosis of ascites in progressive systemic sclerosis should include this apparently benign condition, which responds to medical management.

We thank Mr D J Pinto, consultant surgeon, Tyrone County Hospital, Omagh, County Tyrone, for referring case 2 and for permission to report his findings at laparotomy.

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