1974. Routine skeletal x ray examinations and bone biopsies yielded normal results.

He received a cadaveric renal allograft in 1976 and was in excellent general health thereafter, with satisfactory and stable renal function (mean serum creatinine concentration $156 \mu\text{mol/l}$ (1.8 mg/100 ml), serum urea concentration 9.5 mmol/l (57 mg/100 ml), creatinine clearance 51 ml/min). Other biochemical variables including ionised and total serum calcium concentrations, serum phosphate concentration, and serum alkaline phosphatase activity were consistently in the normal range. Immunosuppression after transplantation was achieved with prednisolone (initially 100 mg/day , reduced progressively to a maintenance dose of 10 mg/day) and azathioprine ($2.5 \text{ mg/kg body weight/day}$). A single, mild episode of acute rejection diagnosed on the eighth day after operation was treated with additional oral prednisolone for three days.

In September 1981 he experienced sudden pain in his right lower calf while walking. Clinical examination indicated a ruptured Achilles tendon. At surgery the diagnosis was confirmed and the tendon successfully repaired. In May 1982 he suddenly developed pain and a lump in his left upper arm while changing gear in his car. Examination showed that the long head of the biceps had ruptured near its insertion at the shoulder. No treatment was considered to be necessary.

In September 1982 he noticed that he was no longer able to extend his right little finger and that a painless swelling on the dorsum of his right hand, present for at least 18 months, had enlarged considerably. Six weeks later the right ring finger also dropped, and shortly after this he became unable to extend his right middle finger (figure). Radiographs of the wrist and

Hand showing inability to extend middle, ring, and little fingers and swelling on dorsum before operation.

hand did not show any osteoporosis, erosions, or other changes indicative of hyperparathyroidism. Serum parathyroid hormone concentration was normal ($<0.1 \mu\text{g/l}$). At operation widespread synovitis of the extensor tendons was found and rupture of the middle, ring, and little finger extensors confirmed. The extensor indicis proprius was transferred and sutured to the three ruptured tendons with a good functional result. Histological examination of the resected synovium showed granulation tissue and acellular fibrin on the surface, with a non-specific chronic inflammatory reaction in the subsynovium.

Comment

The cause of the tendon ruptures in this patient is unknown. There was no evidence of amyloid deposition, rheumatoid arthritis, or other inflammatory arthropathy. Attrition of the tendons due solely to prolonged treatment with corticosteroids is unlikely because rupture of tendons has not been observed in patients receiving long term steroids for ulcerative colitis and asthma.⁵ Steroids would not have been responsible for the synovial reaction that preceded the rupture of tendons in the right hand. Secondary hyperparathyroidism, which has been implicated in tendon ruptures in patients receiving haemodialysis, seems improbable because of the lack of any biochemical or radiological evidence of the condition. The suggestion that the chronic metabolic acidosis associated with impaired renal function causes tendon ruptures in patients receiving haemodialysis does not help to explain the present case as renal function had been almost normal for over five years.¹

In the absence of any other explanation it seems likely that the rupture of tendons in this patient was in some way a consequence of renal transplantation, perhaps related to long term immunosuppression with azathioprine and prednisolone. It is therefore surprising that spontaneous rupture of tendons has not been reported in other recipients of renal transplants. Possibly such ruptures have occurred but have not been reported because of the lack of any obvious cause.

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Fractures on chest radiographs as indicators of alcoholism in patients with liver disease

Alcoholism is increasingly being recognised as an important cause of illness in patients admitted to general hospitals, but the diagnosis is still often missed. Some patients at first deny excessive alcohol consumption, while in other instances doctors fail to take an adequate drinking history. Although mean red cell volume and serum γ -glutamyltransferase activity correlate with alcohol intake, their practical value as screening tests has been called into question.¹ Recently, reports of two studies claimed that the presence of fractures on chest x ray films is a useful indicator of alcoholism both in patients presenting via casualty services and in those with liver disease.^{2,3} Our impression that fractures were relatively rare in patients with alcoholic liver disease led us to carry out the study described here.

Patients, methods, and results

We studied 278 consecutive patients referred to the liver unit over 18 months who had undergone liver biopsy for the first time and in whom a chest x ray film was available for examination. In 103 patients alcoholic liver disease was diagnosed on the basis of a history of alcohol consumption exceeding 80 g/day for men or 40 g/day for women, as established by a standardised interview schedule,⁴ and liver biopsy appearances suggestive of alcoholic liver disease as judged by a specialist histopathologist. The remaining 175 patients had a variety of non-alcoholic liver diseases. Cirrhosis was present in 65 (63.1%) and 31 (17.7%) of the groups with alcoholic and non-alcoholic liver disease respectively. The mean ages of the two groups were similar (52.3 (SD 11.7) and 50.3 (16.4) years respectively).

Only nine patients with alcoholic liver disease had a fracture visible on the chest x ray film, which gives a sensitivity of such a finding for the diagnosis of alcoholic liver disease of only 8.7%. The specificity (the proportion of patients with non-alcoholic liver disease who did not have a fracture) was 96.6% (169/175), and the positive predictive value (the proportion of patients with a fracture who proved to have alcoholic liver disease) was 60%. Only five patients (4.9%) with alcoholic liver disease and two (1.1%) with non-alcoholic liver disease had bilateral or multiple rib fractures. Within each group there were no differences between the proportions of patients with cirrhosis who had fractures compared with those with precirrhotic liver disease and fractures.

Comment

Two previous surveys, in an alcoholism unit² and a gastroenterology unit,³ showed that 25-30% of alcoholic patients had rib or other fractures on chest x ray films. On the basis of the distribution and

radiographic appearances these were attributed to trauma. In contrast, only 8.7% of our patients with alcoholic liver disease had detectable rib fractures. This difference may reflect the different populations studied. Patients with alcoholic liver disease admitted to the liver unit usually present with symptoms of chronic liver disease; they are rarely severely dependent on alcohol,⁵ and only 5% report previous episodes of trauma. Alcoholic patients presenting to general gastroenterology wards or to alcoholism units with psychosocial problems relating to their alcoholism² are perhaps more likely to be severely dependent and to have suffered trauma during accidental falls or fights or as a result of withdrawal fits. Although cirrhosis was present in 63% of the group with alcoholic liver disease compared with 18% of the group with non-alcoholic liver disease, there was no association between the presence of fractures and the severity of liver disease.

The low sensitivity of fractures on chest x ray films for diagnosing alcoholism in patients with liver disease in the present study indicates that chest radiography has limited application in detecting patients with alcohol problems. When fractures are present they may alert the radiologist and clinician to the possibility of alcoholism, but the diagnostic yield is not sufficiently great to justify the use of chest radiography when it would not otherwise be undertaken.

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Postperinatal mortality among infants discharged from special care units

With improving neonatal care more babies of low birth weight survive into infancy. Although much is made of the reduction in perinatal mortality, little is known of the long term survival of babies discharged from intensive care units. We report on the mortality among babies discharged from one such unit.

Patients, methods, and results

We studied all postperinatal deaths (from one week to one year) that occurred among children born from 1 January 1977 to 31 December 1981 to parents resident in the Southampton health district including those who were resident outside the health district at the time of death (data from the Office of Population Censuses and Surveys). Name, age, sex, cause, and place of death were detailed in each case. For babies who had been admitted to the special care unit the following information was obtained from hospital records: date of birth, type of pregnancy (single or multiple), birth weight, duration of stay in unit, reason for admission, and treatment given.

Of 2840 babies discharged from the special care unit, 30 (10.6/1000) died postperinatally, compared with 132 (6.3/1000) out of 20 939 babies not admitted. Deaths after discharge from the unit thus accounted for 18.5% of all postperinatal deaths and consisted of 20 (67%) sudden deaths at home and 10 (33%) in hospital, the ratio of boys to girls being 6:4.

Of the 20 deaths at home, 14 were "cot deaths," three babies had congenital heart disease (one having Down's syndrome), one had suffered perinatal brain injury, and two died accidentally. Fourteen of those who died at home had been low birthweight preterm babies, six of whom were one of twins. Ten of the 14 cot deaths were in low birthweight babies, including all six twins. These 14 cot deaths together with a further 35 among babies who had not been admitted to the unit gave an overall incidence of cot death of 2:1000 live births (4.9:1000 discharged from the unit, and 1.7:1000 others).

Of the 10 babies discharged from the unit who died in hospital, six had congenital deformities, one birth asphyxia, one meningococcal septicaemia, one bronchopulmonary dysplasia, and one hydrocephalus after meningitis; six had been low birthweight infants.

When the duration of stay in the special care unit was calculated the 30 babies who died had spent a total of 951 days in hospital (mean 31.7 days/baby). The 14 presenting as cot deaths stayed 237 days (16.9 days/baby) while the others stayed 714 days (44.6 days/baby).

The figure shows the ages at death (corrected for gestation) of babies discharged from the special care unit, with a peak in postperinatal deaths at 2-3 months.

Ages (corrected for gestational age) at death of 30 babies discharged from special care unit (●—●); 14 cot deaths shown separately (○---○).

Comment

Babies discharged from special care units continue to have a higher postperinatal mortality than the rest of the population, in spite of the quality of care provided immediately after birth. None the less, the 68% increase in mortality shown in this study compares favourably with the tenfold increase found by Kulkarni *et al*¹ and Sills *et al*.²

The pattern of deaths after discharge from the unit was similar to that of postperinatal deaths generally,³ with a preponderance of boys, a high proportion of deaths at home, and a peak at 2-3 months. This pattern suggests an overlap of factors relating to the outcome of pregnancy and deaths of infants.

A striking feature was the relatively high number of cot deaths after discharge which accounted for nearly a third of all cot deaths. Of the babies presenting as cot deaths, only one had a bradycardic episode and one a hypoglycaemic attack. This strengthens the view that neonatal apnoea and cardiac arrhythmias are not major factors in cot deaths.

Babies discharged from intensive care units contribute appreciably to postperinatal mortality, and particularly cot deaths, in a way not yet completely understood. Further study of physiological mechanisms in this group of vulnerable babies could, however, provide vital clues about deaths of infants generally.