Discussion

Between 1968 and 1978 the probability of an Asian schoolchild being admitted to Glasgow hospitals with rickets was one in 29. A survey of one general practice in 1979 indicated that one Asian child in 12 (8%) had been treated for rickets at city hospitals. One in seven (14%) schoolchildren in the survey unprotected by vitamin D supplementation showed radiological evidence of active rickets and two out of five (40%) biochemical evidence, while 44% had serum 25-OHD concentrations of less than 12.5 nmol/l (5 ng/ml). These results are similar to those found in our previous surveys in Glasgow in 1961¹ and 1971² and Bradford in 1973.³

The above facts provided clear evidence of the failure of previous prophylactic measures and the need for a more effective campaign. The basis for such a campaign was provided by our three dose-response studies relating vitamin D intake to serum 25-OHD concentrations, which showed that supplements equivalent to 10 µg vitamin D daily would provide effective prophylaxis. Successful prevention depends on making the supplements widely available and easily accessible. The pattern of serum 25-OHD concentrations in the children who were the subject of the small-scale campaign that preceded the Board's official campaign indicates that supplement taking in many children is at best intermittent. This study showed, however, that even intermittent consumption of supplements will reduce the prevalence and severity of Asian rickets, although being inferior to adequate food fortification as a prophylactic measure.

The key administrative decision in devising more effective preventive measures was the formation of a small multidisciplinary working group able to make clear recommendations to the Health Board. Health education officers and community dietitians who are often charged with responsibility for preventing problems such as Asian rickets may not be powerful enough or near enough to the decision-making process to implement effective policies by themselves. Asian community leaders, although willing to help, lack the expertise to implement preventive policies, which are the province of the community health services.

The ultimate success of the Glasgow rickets campaign remains to be evaluated, and the working group remains in being to monitor progress. In the interim, for an annual expenditure of

about £12 000 at 1979 prices, the campaign seems likely to reduce the prevalence of rickets in Asian children in Glasgow. Other health authorities faced with similar problems may find the Glasgow experience useful in planning their own preventive measures.

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SHORT REPORTS

Renal failure in tetanus

Renal failure is not a recognised complication of tetanus. After the introduction of intermittent positive-pressure respirators, which prevent early death in many of the most severe cases of tetanus, the shock syndrome emerged as an important threat to survival. The syndrome appears to be due to tetanus intoxication of the sympathetic nervous system with attendant tachycardia and wide fluctuations in blood pressure compromising tissue perfusion.¹ In some patients blood pressure varies widely, and terminally patients may develop hypotension and a cold periphery.^{2,3}

Patients, methods, and results

One hundred and six black patients with tetanus admitted to the respiratory unit over three years were studied to determine possible factors causing renal failure. All were initially given chlorpromazine. Fifty received intermittent positive-pressure respiration and were given diazepam 100 mg four-hourly and alcuronium 10-20 mg intravenously when necessary. The incubation period was 9.8 ± SD 4.8 days and the period of onset 38 ± 22.5 hours. None of the patients had received tetanus toxoid or antitetanus serum.

Patients were divided into three groups. Those in group 1 had normal blood urea concentrations on admission and during the course of the illness; in group 2 urea concentrations were normal on admission but rose during the course of illness; and in group 3 urea concentrations were raised on admission. Blood volume, blood chemistry, central venous pressure, pulse rates, blood pressures, and blood and urine cultures were monitored in all patients. Sympathetic overactivity was diagnosed when labile blood pressures, pronounced tachycardia, and increased sweating were all noted.

Mortality in each group of patients

Group	No of patients	No of deaths	Significance
1	83	15	Group 1 v 2, $p < 0.001$
2	15	10	Group 2 v 3, $p > 0.05$
3	8*	5	Group 1 v 3, $p < 0.02$

*Three patients were dehydrated.

The table shows the mortality in each group. Necropsy showed histological evidence of acute tubular necrosis in five patients. Sympathetic overactivity occurred in 28 of the 106 patients, of whom 19 (68%) had renal failure. Hypotension was treated with 200 ml of 4% salt-free human albumin, followed if necessary by dopamine or isoprenaline.

Twenty-three patients, aged 11-56 years, were uraemic, of whom 21 (91%) received intermittent positive-pressure respiration. Twenty-one of these 23 patients had non-oliguric renal failure, the mean urine output being 1.83 ± 0.83 l daily; the two remaining patients were anuric. Blood urea concentration varied from 9 to 60 mmol/l (54 to 360 mg/100 ml) with a mean of 20 ± 13 mmol/l (120 ± 78 mg/100 ml). In the 15 patients in group 2 the mean time to the onset of a raised blood urea concentration was 7.7 ± 1.6 days. The rate of rise of the concentration was 5 ± 3.7 mmol/l/day (30 ± 22.2 mg/100 ml/day). Serum potassium concentration was above 7 mmol (mEq)/l in two patients. The mean serum osmolality was 318 ± 42 mmol (mosmol)/kg and mean urine osmolality 532 ± 143.9 mmol/kg in the patients in group 3. In group 2 the mean serum osmolality was 288 ± 9.1 mmol/kg and the mean urine osmolality 503.1 ± 102.9 mmol/kg.

Blood cultures were positive in four patients. Four patients were given an aminoglycoside antibiotic before the onset of uraemia.

Comment

Renal failure was associated with sympathetic overactivity in 19 of the 23 patients. Sympathetic overactivity in severe tetanus is common in our respiratory unit, a possible explanation for its high incidence being that many patients have not been immunised before the onset of the disease.⁴ Hypotension owing to dehydration was not the cause of the renal failure, since adequate hydration was maintained in all patients. Septicaemia and nephrotoxic antibiotics appeared not to be major aetiological factors. Myocarditis was absent in all patients.

Non-oliguric renal failure may be due to early treatment of shock, which may decrease the number of cases of oliguria without always preventing renal failure.⁵ The non-oliguric renal failure in most of our patients may possibly have been due to early treatment of the hypotension with sympathomimetic agents. Renal failure associated with tetanus has a high mortality, and 15 of our 23 patients (65%) died.

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³ Corbett L, Spalding JMK, Harris PJ. Hypotension in tetanus. *Br Med J* 1973;ii:423-8.

⁴ Omar MAK, Wesley AG, Pather M. Labetalol in severe tetanus. *Br Med J* 1979;iii:274.

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Monoarthritis from blackthorn injury: a novel means of diagnosis

Establishing the correct diagnosis in a patient with persistent monoarthritis is difficult.¹ Reaction to a foreign body will rarely appear to be the cause as this is often hard to prove. We describe two cases of synovitis induced by blackthorn injury and a novel and simple means of diagnosis in a patient presenting with an associated synovial effusion.

Case reports

Case 1—A schoolboy aged 15 fell into a blackthorn bush while riding. One thorn pierced the proximal interphalangeal joint of the right middle finger. The thorn was removed in two pieces. Within an hour the finger

had become red, hot, swollen, and painful. During the next week local applications produced slight improvement, although the inflammation fluctuated in intensity. Nine days later the joint again became acutely inflamed and he was treated by his general practitioner with flucloxacillin. Within 24 hours the inflammation had settled but the joint remained swollen; he was therefore referred to this hospital. We suspected that fragments of the thorn remained, and at arthrotomy multiple blackthorn fragments were removed with the aid of a dissecting microscope (figure). His condition subsequently settled completely.

Case 1—(a) Blackthorn embedded in synovium with associated foreign-body reaction; (b) Same section viewed with polarised light microscopy. Case 2—(c) Blackthorn fragments within centrifuged synovial deposit; (d) cell wall of vegetable tissue highlighted with polarised light (note multiple small fragments).

Case 2—A diabetic schoolboy aged 12 was admitted to hospital for stabilisation of his diabetes. One week before he had injured his left knee on a blackthorn bush, and within hours the joint had become painful, hot, red, and swollen. Two thorns had pierced the knee and were said to have been removed intact. He was treated with ampicillin and flucloxacillin but improved slowly. His diabetes subsequently became poorly controlled and he was therefore admitted. He was treated with intravenous antibiotics (cephradine) and the knee rested in a plaster back-slab. The swelling settled but did not resolve completely. Synovial fluid aspirate was sterile, though many polymorphs showing toxic granulations were observed in the film. A subsequent synovial aspiration was performed one week later, this time with a white cell count of 4.5×10^9 /l. The remaining synovial fluid was centrifuged (3500 rpm for 15 min), the supernatant decanted, and the deposit fixed in 10% neutral formalin. The proteinaceous plug was easily handled by conventional means, obviating the use of gelatin as a support medium. Paraffin block section showed an inflammatory exudate composed of lymphocytes and polymorphs: present within the exudate were small fragments of birefringent vegetable material, suggesting blackthorn fragments within the knee (figure).

Comment

There are many causes of chronic monoarticular arthritis, but despite thorough investigation 30% of cases usually remain undiagnosed. The diagnosis of a foreign-body reaction will either be easy when the penetrating injury is recalled or be most difficult when it is not. Plant thorns, unlike many other foreign bodies, are not detected radiographically. The puncture site is rarely found clinically. The synovial fluid white cell count is raised, most cells being polymorphs, but this is a non-specific and relatively unhelpful finding. Only careful microscopical examination of the synovium will show the foreign body, giant cells, and vegetable tissue; blind or arthroscopic synovial biopsy may therefore be unrewarding. Finding birefringent vegetable material within the spun synovial precipitate may be the easiest way of diagnosing this condition, though the incidence of positive results in patients with known plant-thorn synovitis must be established. Birefringent material may be derived from other sources—for example, crystals of urate, pyrophosphate, or intra-articular steroid preparations—and the vegetative nature of this birefringent material must be established.

The blackthorn is a perennial shrub common in Britain; it has many thorns that can easily penetrate the synovium, stimulating an inflammatory reaction.² Interestingly, though the thorn remains