

carbohydrate metabolism.¹ The hypotonia persisted until lithium was undetectable in the serum. The apparently poor development at 1 year is disappointing, but it is not clear whether this can be attributed to the very high lithium values at birth or cerebral hypoxia associated with a poor cardiac output in the immediate postnatal period.

The management of neonatal lithium toxicity of this degree has not been described before. Exchange transfusion did little to reduce serum lithium values. Haemodialysis or peritoneal dialysis are the recommended treatments in adult lithium poisoning⁴ but, with all their attendant risks it is doubtful whether these are the best treatments in neonates. Our case shows that recovery in the acute phase of lithium toxicity can be expected provided adequate supportive treatment is given.

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'Windswept deformity'

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SUMMARY We describe 8 children with 'windswept deformity'—a valgus deformity of 1 knee in association with a varus deformity of the other. The disease is a physeal osteochondrosis and conservative treatment with serial corrective plaster casts is as effective as corrective osteotomies.

'Windswept deformity' is the abrupt development of angulation of 1 knee into varus deformity and the other knee into valgus (Fig. 1). Although the disease has received little attention in published reports, it is common in Nigeria and other parts of Africa.¹ We describe 8 children with this condition whom we have treated and discuss the aetiology, natural history, and management.

Clinical features

Relevant clinical, radiological, and laboratory data were obtained while treating 8 children who presented with 'windswept deformity' at this hospital in the first 3 months of 1982. In all cases early developmental milestones were reached and the deformity arose spontaneously shortly after the previously healthy child had begun to walk. Most patients were girls. The deformity was not preceded

by any local or systemic illness and there was no clinical or radiological evidence of rickets or of other generalised bone disorder. Serum calcium, phosphorus, and phosphatases were within normal limits. General metabolic disease, malnutrition, and haematological disorders were not found.

Radiographic appearances

The radiological features were characteristically osteochondritic and the lesions occurred in the distal end of the femur or the proximal end of the tibia, or both. The medial portion of the epiphysis in genu varum or the lateral in genu valgum became wedge shaped and more dense, while the adjoining metaphysis developed a beak like projection. Radiological examination showed 3 types: Blount's tibia vara² associated with physiological valgus deformity³ in the other knee; tibia valga with a physiological varus⁴ component (Fig. 2); and Blount's disease coexisting with tibia valga (Fig. 3).

Treatment

Our first 2 patients were treated by corrective osteotomies of the proximal tibia and the rest by serial corrective plaster of Paris knee cylinder casts.

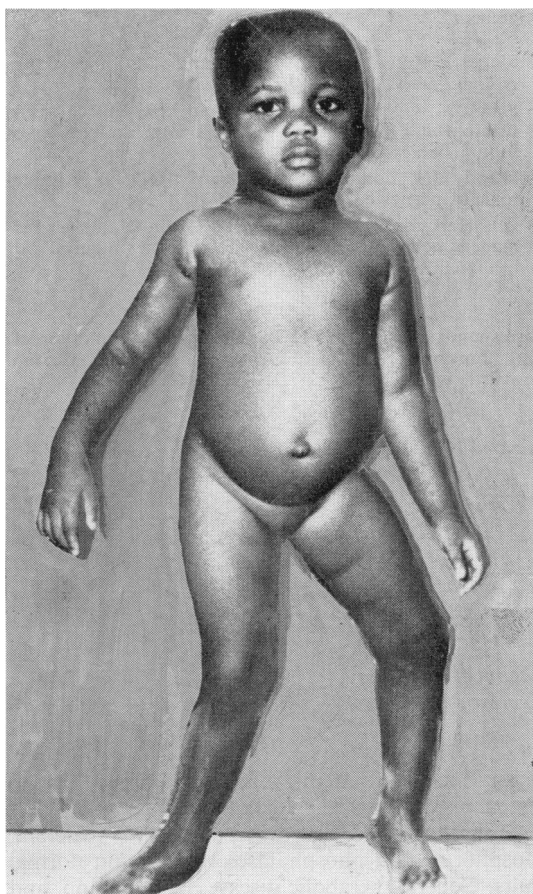


Fig. 1 The 'windswept deformity'.

All patients were cured, but the 2 who had corrective osteotomies were cured more quickly (6–8 weeks) than the others (8–12 weeks).

Discussion

The clinical and radiological features suggest that this condition is an osteochondrosis. The onset is abrupt, the children are healthy, and the disease arises from a formerly normal epiphysis. The pathological lesions are identical to those of osteochondroses and the disease can be classified as a physeal osteochondrosis.

It seems that the radiological changes in each bone may pass through 6 stages, each stage, representing the severity but not necessarily the progression of the pathological changes. In stages I to V the disease is confined either to the lower end of the femur or to the upper end of the tibia, while in stage VI both



Fig. 2 'Windswept deformity'—tibia valga associated with non-pathological varus deformity (simple bowleg) in other knee.



Fig. 3 'Windswept deformity'—tibia vara-valga.

bones are affected. Longstanding 'windswept deformity' may be associated with other postural or compensatory skeletal abnormalities such as a mild dorsolumbar scoliosis, metatarsus adductovarus or pes planovalgus, or both.

There seems to be no correlation between clinical or radiological severity, or both, and prognosis.

'Windswept deformity' is commonly seen in children in Africa but residual deformities are not as often found in adults, suggesting that a considerable proportion must correct spontaneously. Because of this caution should be exercised when recommending the more drastic method of treatment for this condition.

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Pancreatitis during sodium valproate treatment

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SUMMARY A girl aged 1 year died of acute haemorrhagic pancreatitis while taking sodium valproate. Necropsy showed widespread vascular disease that may have contributed to the onset of pancreatitis. Previous reports of pancreatitis in children receiving valproic acid are reviewed and although the association is rare, a causal relation between pancreatitis and valproic acid seems to have been established.

Although some 1 000 000 patients have been treated with sodium valproate,¹ serious or fatal reactions are rare and most of the published reports describe liver damage.¹ Acute pancreatitis has, however, been reported in 7 children receiving valproic acid, 1 of whom died.²⁻⁶ We report on a child who died of pancreatitis while taking sodium valproate.

Case report

A normal baby girl aged 3 months suffered brief right sided clonic fits during a short febrile illness. The cause of the fits was not known but they stopped after treatment with phenobarbitone. Her subsequent development was normal and phenobarbitone was stopped at age 9 months. After another febrile illness at 1 year, the right sided fits recurred, together with a right hemiparesis and frequent vacant episodes. These seizures were completely controlled with 25 mg/kg per day of sodium valproate.

Ten days after starting treatment with sodium valproate the baby was readmitted to hospital with a 4 day history of drowsiness, anorexia, and vomiting. Abdominal distension and peripheral circulatory failure were found. Resuscitation failed

and she died shortly after admission. Investigations before death showed a serum valproate concentration of 5.9 mg/100 ml (therapeutic range 5-10 mg/100 ml) and a serum amylase of 3520 IU/l (normal range 100-400 IU/l).

Necropsy showed extensive haemorrhagic pancreatitis that had been present for several days. More recent areas of necrosis were found throughout the intestinal mucosa. In addition there was a widespread long standing non-inflammatory endothelial proliferation of many medium sized arteries. Obliteration of some of these arteries had resulted in areas of ischaemic necrosis of varying ages, particularly in the kidney and left cerebral hemisphere. The aetiology of the vascular lesions was unknown.

Discussion

The clinical details of the children in whom valproic acid has been associated with pancreatitis are shown in the Table. A relation between pancreatitis and valproic acid is established in the 4 patients who again developed pancreatitis on re-exposure to valproate. In 1 patient 4 separate courses of treatment resulted in 4 episodes of pancreatitis.⁶ Four patients were receiving other anticonvulsant agents. In 2 patients who were taking phenytoin the pancreatitis resolved when valproate was stopped, despite the continuation of phenytoin treatment. Re-exposure to valproate again resulted in pancreatitis in 1 of these patients.⁵ There does not seem to be any relation between pancreatitis and the patient's sex, type of fits, past or present medical history, or the dose of valproic acid. Pancreatitis occurred within the first 6 months of treatment in all the children. One patient died after laparotomy