liver function values in patients with SLE, and rarely it may also be hepatotoxic. These patients may develop hypersensitivity to ibuprofen more often than patients with other diseases. More experience with this drug is essential to assess whether it may safely be given to patients with SLE without the addition of steroids.

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SHORT REPORTS

Alkaline phosphatase: changes in serum levels after a fracture

Large increases in serum alkaline phosphatase concentrations after fractures in elderly patients have been noted in specialist journals,¹ but standard textbooks either make scant reference² or comment that there is little or no rise.³ We studied alkaline phosphatase concentrations in elderly patients who had been admitted to an orthopaedic ward because of a fracture.

Patients, methods, and results

So far as possible we chose patients admitted consecutively who were expected to remain in hospital for at least four weeks. Serum alkaline phosphatase was measured in 45 patients (42 women, 3 men) with an average age of 77 (range 62-93) years. Thirty-nine had fractures of the femoral neck and the other six had fractures of other bones.

The mean serum alkaline phosphatase concentration within one week of the fracture was 59 IU/l, rising to a peak of 111 IU/l in the fourth week (see table). Thereafter concentrations of serum alkaline phosphatase declined slowly but had still not returned to the baseline by eight weeks. Increases in serum alkaline phosphatase concentrations varied considerably between individuals—from 0 to 515 % above baseline. There were no differences in the percentage rise above the baseline with age or types of fracture or according to whether the patient had undergone surgery. Likewise there was no difference in the behaviour of the serum concentrations between those who remobilised rapidly and those who had a slow course because of general illness or periods of traction.

Changes in serum alkaline phosphatase isoenzymes—The isoenzyme pattern was studied in 11 patients by electrophoresis on polyacrylamide gel.⁴ Four patients who had only a small rise in total alkaline phosphatase concentration showed no change in the proportion of bone and liver isoenzymes. In three ill patients the rise was mainly or entirely due to a rise in the concentration of liver isoenzymes. Four patients who had been generally fitter and more active had a rise in both isoenzymes, with that in the bone dominating.

Comment

This study has further confirmed that a significant change in total serum alkaline phosphatase concentrations occurs after a fracture, so that on average there is a doubling of baseline values, but with considerable individual variation.

These changes might be thought to represent osteoblastic activity after bone damage and repair, but the limited isoenzyme data available showed that the increase was not always due to the bone isoenzyme

and in some cases was entirely due to the liver isoenzyme. These changes may represent postoperative liver damage due to anaesthetic agents or other factors. Detailed studies of postoperative liver function values have suggested that a rise in total alkaline phosphatase concentrations may occur in about a third of patients in the first few days after operation.⁵

From a practical point of view, the diagnostic value of changes in alkaline phosphatase concentrations in elucidating the cause of jaundice or indicating the presence of osteomalacia or other bone disease is greatly reduced.

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Splints in severe osteogenesis imperfecta

Osteogenesis imperfecta often presents at birth with multiple fractures and may be rapidly fatal. Nevertheless, many children survive and subsequently demand great care and attention from their family and medical attendants. The stress put on the family by the possibility of further fracture is considerable and often a cause of parental anxiety and morbidity. Recurring fractures may result in gross bony deformity, especially in the long bones, and further fractures in these deformed

Serum alkaline phosphatase concentrations after a fracture

Week No:	1	2	3	4	5	6	7	8
Alkaline phosphatase (IU/l):								
Mean ± SE of mean	59 ± 2	80 ± 7	104 ± 7	111 ± 8	100 ± 7	103 ± 8	97 ± 7	87 ± 7
Range	35-59	32-270	39-227	43-284	35-5-256	53-199	43-213	60-135

limbs cause great discomfort for the child, further difficulties in management (especially lifting), and time lost from school. Conventional conservative measures to splint the limb are usually inappropriate, as reduction in joint mobility prevents manipulation of the limb without excessive pain. Here we describe a method for overcoming some of the difficulties in splinting limbs.

Method

By using the "vacuum consolidation" technique^{1 2} an exact replica of the limb can be made, around which an effective splint can be moulded without causing unnecessary pain. This uses an airtight plastic bag containing Fillite—a very fine particle, the byproduct of ash produced in electricity generating stations. It is fitted with a filtered outlet tube and attached to a vacuum source.³

The child is positioned so that the affected limb lies on a bag of Fillite and this is manoeuvred to produce a mould for the lower half of the limb (see figure). Evacuation causes the Fillite bag to assume a rigid shape, exactly reproducing the contours of the limb. A second, smaller bag is then placed over the limb completely covering it; evacuation will then complete the mould and the child may be removed by separating the two bags. The remaining negative is an exact replica of the limb, and an accurate reproduction may be produced by using plaster of paris poured into the mould between the two bags. After five to ten minutes of drying only, the vacuum may be released on both bags, allowing the positive cast to be removed and made smooth before it hardens completely.

A splint may now be made by moulding Plastazote around the cast, reinforcing this with longitudinal strips of polypropylene, and this is finished with strips of Velcro. A rough fitting of the splint can be made within two hours of the first impression's being taken.

The lower Fillite-filled bag moulded around the contours of the limb. The type of bony deformity apparent in this condition is easily seen.

Antithrombin III deficiency, hypertriglyceridaemia, and venous thromboses

A few families have been described whose members suffer from repeated thrombotic episodes and have decreased concentrations of antithrombin III in their blood. This report of a family based in the north of England suggests the need for greater awareness and screening for this condition, which has hitherto been thought to be rare in Britain.

Case report

A 48-year-old woman due for an elective cholecystectomy was referred for investigation of recurrent deep vein thromboses occurring over three years. No relatives, apart from her dead mother, had any history of thrombotic disease. The mother had suffered repeated attacks from the age of 48 until her death at 82. The patient was four years postmenopausal and smoked up to 10 cigarettes a day. She was moderately obese, and xanthelasmata were noted.

Laboratory investigations, including routine haematological, coagulation, and liver function tests, gave normal results apart from a fasting plasma triglyceride level of 3.1 mmol/l (274 mg/100 ml) (normal <2.0 mmol/l (177 mg/100 ml)). The overnight stored plasma showed diffuse lactescence. In view of the history antithrombin III levels were measured by immunodiffusion (Hoechst Partigen) and found to be low (0.7 mg/l compared with the normal range in serum of 2.0-3.5 mg/l). Her blood relations were subsequently tested (see diagram), and seven were found to have low antithrombin III concentrations (0.7-1.0 mg/l). Only the patient's elder daughter had a raised fasting triglyceride level of 3.0 mmol/l (266 mg/100 ml) as well as a low antithrombin concentration.

The patient was advised to give up smoking and take a low calorie diet. She was anticoagulated with warfarin uneventfully for six months. The dose was reduced preoperatively and a prophylactic dextran infusion was given during the cholecystectomy. The dose of warfarin was increased to a therapeutic level immediately after operation and her subsequent course was uneventful. Her daughter was advised not to take oral contraceptives and to consider a low calorie diet.

Results of investigation of four generations of a family affected by antithrombin III deficiency.

Comment

The antithrombin levels and autosomal dominant pattern of inheritance in this family conform with those in other cases.¹ The clinical effects vary among cases, however; they occur in about half those at risk² and are often serious. Apart from her mother, who none the less survived to become an octogenarian, the propositus was the only clinically affected member of the family. Therefore hypertriglyceridaemia should be considered as an additional factor since it has been found in two previous thrombotic patients with antithrombin III deficiency.³

Whether the patient's mother also had a raised lipid level can only be guessed at but autosomal dominant inheritance has been described in some cases.⁴ If this were so the patient's daughter, who has inherited both abnormalities, may well be at risk as she grows older, for most reports show an increasing number of thromboses with age.

Our findings underline the need for considering hereditary causes

Conclusion

This method of splinting limbs grossly deformed by osteogenesis imperfecta using the vacuum consolidation technique is quick, effective, and exposes the patient to little manipulation or pain.

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