FAT EMBOLISM IN FOUR PATIENTS TREATED FOR FRACTURES

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FAT embolism is well recognised to be an important though relatively infrequent complication of injuries of long bones. It has been shown that this is common in surgical or accidental damage to soft and skeletal tissues (Peltier, 1956), but that only in multiple fractures of bones is it severe enough to produce recognisable clinical symptoms and signs. The time when fat embolism was a post-mortem diagnosis has passed to one in which it is diagnosed early and in some cases successfully treated. Now, though it is recognised earlier, the treatment is still largely empirical and symptomatic. Recorded below are the case histories of four patients recently seen and treated in the orthopaedic unit.

Case No. 1.

J.H., a man aged 24 years, was knocked down by a car and admitted to hospital on January 13, 1961.

His injuries were:— a comminuted fracture of the mid-shaft of the right femur, an open comminuted fracture of the shaft of the right tibia, a fractured right fibula, a rupture of the anterior cruciate ligament of the right knee, and a fracture of the base of the terminal phalanx of the right hallux. His femur was reduced and immobilised on a splint on the day of admission. The following day an open reduction and screwing of the fractured right tibia was performed under a general anaesthetic. Forty-eight hours after admission he developed sweating, abdominal pain and petechial haemorrhages in the skin of the pectoral region. His temperature rose to 101°F and remained so for two days. A provisional diagnosis of fat embolism was made. Abdominal examination revealed no evidence of injury to the viscera. In the urine fat droplets were recognised and the ether-soluble fat content was 8 mg./100 ml. No count of blood chylomicrons was made. Hb 65%. A chest X-ray showed fine mottling in the lung fields. These findings confirmed the diagnosis of pulmonary fat embolism. No specific treatment was instituted. All the symptoms cleared up within two days. He was discharged on May 12, 1961. Further out-patient treatment for his fractures was uneventful and he was finally discharged on the May 31, 1962.

Case No. 2.

W.I., a man aged 32 years, was knocked down by a car on May 18, 1962, and admitted to hospital suffering from an open fracture of the right tibia, a closed fracture of the left tibia and fibula, and

a subtrochanteric femoral fracture. There was no history of head injury and he was fully conscious.

On the day of admission under a general anaesthetic his leg fractures were manipulated and immobilised in plaster and the subtrochanteric fracture treated by skeletal traction and a Thomas's splint. He recovered full consciousness. The next morning he became drowsy and sweated profusely. He developed petechial haemorrhages in the skin at the base of the neck and upper part of the chest. Over a period of four to five hours he became deeply unconscious with decerebrate rigidity and incontinence of urine and faeces. He had a pyrexia of 102°-106°F which lasted for one week. Chest and abdomen: no abnormal physical signs. Occasional Cheyne-Stokes respiration. A diagnosis of cerebral fat embolism was made. The only confirmatory neurological finding was a local infarct in the fundus of the left eye.

He was given anticoagulants, starting with heparin and continuing with dindevan within forty-eight hours. Sedation with sodium gardenal was necessary as the patient was very restless and was pulling down the splints used to immobilise his fractures. He was given penicillin and erythromycin prophylactically because of the open fractures he sustained. He was cooled with ice packs and by tepid sponging, and began to show some improvement in one week. He was no longer deeply comatose and could respond to painful stimuli. He recovered full consciousness and mental faculties in three weeks. Control of micturition returned in seven weeks. The anticoagulant therapy was given for three weeks. He was discharged from the ward on December 7, 1962, for out-patient treatment. This out-patient supervision was uneventful and he was finally discharged on April 25, 1963, very fit and back to his former job as a machinist.

Case No. 3.

D.W., a man aged 22 years, who on September 29, 1962, while driving a motor cycle side-car combination ran into a post and was admitted to hospital with a comminuted fracture at the junction of the middle and lower thirds of the shaft of the right femur, an open dislocation of the left cuboid, an open fracture of the terminal phalanx of the left hallux, and multiple skin abrasions. He was shocked; there was no head injury.

He was given two pints of blood. Under a general anaesthetic given on the day of admission the fractures were manipulated and immobilized. He did not regain consciousness and twenty-four hours later developed decerebrate rigidity and petechial haemorrhages in the skin of the base of his neck and upper chest. There was marked respiratory

desoxycholate (10 ml. of 20% solution) which increases the emulsifying power of the blood and so reduces the size of circulating fat globules is limited because in larger doses it produces hæmolysis. Next, anticoagulant therapy has been advocated (Cobb and Hillman, 1961). Heparin is supposed to alter the lipoprotein density and would also prevent thrombosis if an occlusion did occur from the fat globules. From this small series, the impression gathered was that severe cases treated with anticoagulants recovered from the effects of fat embolism sooner than those treated without.

The treatment of these four cases has been discussed briefly; they are presented because they recovered in spite of prolonged unconsciousness, hyperpyrexia, respiratory and neurological involvement. It is also apparent that at the moment, beyond reduction of the hyperpyrexia, the quietening of the restlessness, and provision of a free airway for the patient with respiratory distress there is little to be done except to give anticoagulants, which did not interfere with fracture healing.

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INTRACTABLE PRURITUS DUE TO HEPATIC CIRRHOSIS RELIEVED BY CHOLESTYRAMINE

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PRURITUS due to liver disease is almost always due to biliary obstruction with associated retention of bile salts and other ingredients of the bile. The pruritus is always troublesome and its control is notoriously unsatisfactory. We report a case of hepatic cirrhosis whose pruritus was successfully relieved by cholestyramine.

Case Report

A married woman aged 47 attended the dermatological out-patient department on December 5th, 1962, complaining of severe generalised pruritus and tiredness for the last 5 years. At the beginning of her illness she had had bouts of violent sickness and vomiting at intervals of 6 to 7 months, but this had not troubled her much lately, although she had to avoid fried food because it made her feel sick.

She had scarlet fever in childhood. She had lived in N. Rhodesia from 1940 to 1946 where she suffered from enteritis and several mild attacks of malaria, from which she had been free since her return.

She was seen elsewhere for her skin complaint in 1960 and was put on Liq. Arsenicalis (Fowler's solution), which controlled her itching for the first time. She was kept on arsenic for about a year with reasonable control of pruritus, and thereafter this regime was stopped. Soon afterwards the pruritus returned, no other means could be found to control it, and it eventually got so bad that it interfered seriously with her sleep.

Physical examination revealed a thin, anxious looking woman with an earthy discolouration of the whole skin. There were numerous scratch marks on the body and limbs and some scattered superficial scars on the trunk and upper limbs. The provisional diagnosis was dermatitis herpetiformis and she was put on Dapsone 100 mg. b.d., which failed to control the pruritus. She was accordingly admitted on the 23rd April, 1963 for further investigation.

In hospital she was noticed to have pale coloured stools and dark urine occasionally, and her liver was palpable three finger breadths below the costal margin.

Investigations: Hb. 11 g./100 ml. PCV 35% MCHC 32%. The blood film was normal and Heinz bodies could not be found. Thrombotest 100%. ESR 37

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