considerably more swollen and painful after a minor twisting injury. He was otherwise well and no other joints were troubling him. He had a large effusion in the knee. The knee showed a good range of movement and was relatively pain free, though he had lateral instability of the joint. The calf was swollen and a little tender and there was also some pitting oedema around the ankle. There was a crescentic bruise clearly visible under the lateral malleolus (figure). Radiographs of the knee joint showed early osteoarthritic changes. Altogether 15 ml of altered blood were aspirated from the knee joint and he was treated with rest, reaspiration, and quadriceps exercises with full resolution and return to work.

Position of crescentic bruise.

Comment

It is important to recognise the difference between deep vein thrombosis and rupture of the knee joint. The latter is benign and settles with conservative measures, while deep vein thrombosis usually requires anticoagulants—treatment that carries an appreciable morbidity. If the fluid which has leaked from a ruptured joint contains blood then ecchymosis may be present and recognition will prevent the injudicious use of anticoagulants.

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Methaemalbuminaemia as a cause of cyanosis

In the investigation of a patient with central cyanosis, an acceptable PaO_2 , and normal haemoglobin concentration it is a logical first step to check the blood gas results on an alternative analyser. The next step is to look for an abnormal circulating haemoglobin¹; methaemoglobin may be found,² which may be due to drug or chemical toxicity, an unstable haemoglobin M, or congenitally deficient methaemoglobin reductase. A blue discoloration may persist for some time after investigations, such as lymphangiography in which methylene blue or patent blue dyes have been injected. Methaemalbuminaemia is another cause, and we describe a patient in which this was present after a severe blood loss into the tissues.

Case report

A 17-year-old girl was in the front seat of a car implicated in a road traffic accident. She sustained a severe head injury, fractures of the left arm, and severe pelvic injuries with displacement of her left femur into the abdomen. The left thigh became grossly swollen and the abdomen distended and tense, with bruising around the umbilicus and on the flank. Although the external loss was estimated at under 1500 ml, 15.5 l of blood, together with colloid and crystalloid solutions, were transfused during resuscitation. The resulting haemoglobin concentration was 13.5 g/dl, which fell over the next four weeks

Peak ODs and bilirubin concentrations after operation.

Conversion: SI to traditional units—Bilirubin: 1 μmol/l ≈ 0.058 mg/100 ml.

to 12.5 g/dl. There was no evidence of intravascular coagulopathy. She was ventilated for three days and extubated after regaining consciousness on the fifth day. After extubation she was given 28% oxygen.

After 48 hours' deepening central cyanosis and increasing tachypnoea, bronchopneumonia was diagnosed and antibiotics were started. At this time the patient was afebrile, the chest x-ray film and ECG were normal, and there were no pathognomonic physical signs of either pneumonia or fat embolism. Blood gas analyses during this period had shown a slight fall in arterial oxygen partial pressure (PaO_2) from 10.25 kPa (76.9 mm Hg) (saturation 95%) to 9.32 kPa (69.9 mm Hg) (saturation 94%) in spite of increases in inspired oxygen concentration (F_iO_2) from 28% to 40%, but despite these measures the patient remained cyanosed. The blood gas results were confirmed using a different analyser and the blood was then screened for methaemoglobin and sulphaemoglobin with negative results. It was noted that the patient's plasma was turbid and so darkly coloured that it was difficult to see the cell/plasma interface in a haematocrit tube. The pigment was shown to be methaemalbumin. The peak optical density (OD) of methaemalbumin was measured until the cyanotic discoloration had disappeared, which occurred when the OD fell to less than 1.35 (see fig). Blood taken some six weeks later showed normal concentrations of NADH-dependent and NADPH-dependent methaemoglobin reductase.

Discussion

Methaemalbumin is formed when red cells are broken down and the capacity of the normal haemoglobin-carrying protein, haptoglobin, is saturated; the excess haemoglobin then forms a loose complex with albumin. We presume that in this patient the pigment arose from the absorption into the circulation of the breakdown products of the concealed haemorrhage. Further evidence of haemoglobin metabolism may be found by monitoring the serum bilirubin concentration (figure). Methaemalbuminaemia has been reported in association with concealed haemorrhage in acute pancreatitis,³ although it is more commonly associated with intravascular haemolysis and intramedullary ineffective erythropoiesis.

With the decrease in OD of the methaemalbumin there was a concomitant decrease in both cyanosis and tachypnoea. Since the evidence for the diagnosis of bronchopneumonia was slight, it is interesting to speculate whether the methaemalbuminaemia and the increased respiratory rate were in some way related.

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