CEREBRAL FAT EMBOLISM WITH HAEMATEMESIS

Report of a Case

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A hydrocephalic man of thirty-four was knocked down in a road accident in April 1960, and sustained a compound fracture of the shaft of the right tibia and fibula, and a closed avulsion of the lateral ligament of the left knee from the upper end of the fibula. There was no head injury or loss of consciousness and the patient was admitted to hospital in good general condition. The open fracture wound was excised and left open; a plaster back-slab was applied from toes to mid-thigh. A plaster cylinder was applied to the left lower limb from groin to malleoli after suture of a laceration on the upper part of the left leg.

On the next day the patient became drowsy. He was sweating, and petechial haemorrhages were observed in both axillae. There was an increase of muscle tone in the left upper limb, the upper limb reflexes were all exaggerated and the left plantar response was extensor. The fundi were normal. A clinical diagnosis of fat embolism was made. Four hours later further petechiae were evident and the patient appeared to be losing consciousness. Two hours later about five ounces of bright red blood were vomited. The next day the patient was still drowsy and many petechiae were present. But within two days his general condition began to improve, and a week later he was considered fit for surgical repair of the lateral ligament of the left knee. At that operation a large contusion was seen to surround the fracture of the upper end of the fibula and emulsified fat was present in the wound. The small fragment avulsed from the head of the fibula was secured to its bed by chromic catgut. The patient's further recovery was uneventful; fracture union and rehabilitation proceeded smoothly. The patient gave no history of dyspepsia before or after injury.

DISCUSSION

Haemorrhage caused by, or associated with, fat embolism has not been commented on and is said not to be a feature of the pathological picture (Sevitt 1961). Recently Sessner, Schütterle and Stummeyer (1961) described diffuse haemorrhage from the stomach and intestinal tract as a complication. There is no evidence in my case of the cause of the haematemesis; the association with signs of cerebral dysfunction is suggestive and reminiscent of peptic ulceration in disease of the hypothalamus.

It is hoped that this case report may be of some interest in its own right. It is also relevant to the use of heparin in the treatment of fat embolism. Although a trial of heparin in experimental fat embolism in the rat (Gardner and Harrison 1957) proved dangerous, interest is being shown in the use of the drug in human fat embolism; had it been given to this patient it would have been blamed unjustifiably for the haematemesis, and it may well have increased the severity of that complication.

The author would like to acknowledge information from Dr D. A. L. Bowen about four other patients with acute peptic ulceration after cerebral fat embolism.

REFERENCES

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