LATENT SEVERE ANOXIA ASSOCIATED WITH THE FAT EMBOLISM SYNDROME

A Case Report

BY

A. P. ROSS AND J. O’HIGGINS

SUMMARY

Fat embolism syndrome was diagnosed in a 41-year-old man 42 hours after a road traffic accident. Latent severe anoxia was present and persisted despite endotracheal intubation and administration of oxygen when breathing was spontaneous. Oxygenation was restored almost immediately after instituting intermittent positive pressure ventilation. The need for frequent estimations of blood-gas tensions or oxygen saturation is emphasized.

Pulmonary fat embolism is found at necropsy in 90-100 per cent of patients who have died shortly after receiving fractures (Sevitt, 1966) but clinical manifestations of this embolism develop in less than 2 per cent (Wilson and Salisbury, 1944; Saikku, 1954). The features most commonly associated with the fat embolism syndrome are: pyrexia, tachycardia, tachypnoea, haemoptysis, variable neurological signs, coma and a petechial rash.

The following case is reported because it confirms recent evidence of latent severe anoxia associated with the fat embolism syndrome.

CASE REPORT

On May 12, 1967, a 41-year-old man, who had previously been in good health, was admitted to hospital after a road traffic accident in which he sustained a minor head injury, a compound fracture of the shaft of the left tibia, and a simple fracture of the neck of the right humerus. Plasma 1,080 ml and whole blood 1,620 ml were transfused and a course of intramuscular penicillin and streptomycin was commenced.

Thirty-six hours after injury, the patient for the first time since admission did not respond to simple commands. The body temperature was 102.2° F, pulse rate 96 beats/min, and respiratory rate 24 b.p.m. Six hours later, the respiratory rate had risen to 40 b.p.m. and a petechial rash was noted on the anterior axillary folds. A diagnosis of the fat embolism syndrome was made.

On the fourth day after injury, the abdominal reflexes were absent and early papilloedema was noted. Coarse crepitations were audible over both sides of the chest but the patient was not obviously cyanosed. However, as severe hypoxaemia without cyanosis has been reported in cases of this syndrome (Sproule, Brady and Gilbert, 1964), the Pao₂ was measured (Clark electrode) and was found to be only 23 mm Hg; the haemoglobin content of the blood was 14.2 g. Whilst the former estimation was being carried out, the patient became cyanosed and so was intubated with an endotracheal tube. He was given oxygen (12 l/min) through the tube but continued breathing spontaneously at 60 b.p.m. Half an hour later, cyanosis was no longer present but the Pao₂ had risen to only 31 mm Hg; pH was 7.56 and PaCO₂ 31.5 mm Hg. The minute volume varied between 21 and 29 l./min; the tidal volume was between 500 and 650 ml/min (Wright respirometer). The patient was sedated with pheno- peridine 1.75 mg and the lungs were ventilated with approximately 66 per cent oxygen in air using a Cape ventilator at a minute volume of 14 l./min. Subsequently, the degree of oxygenation was monitored by measuring the arterial oxygen saturation because facilities were available for this measurement to be carried out on the ward. Within half an hour of starting intermittent positive pressure respiration the arterial oxygen saturation had risen from 66 to 95 per cent (Kipp Haemoreflector) and there was a considerable reduction in the large quantities of blood-stained frothy fluid which had been aspirated previously.

On the sixth day after injury, the patient was capable of obeying simple commands. Mechanical pulmonary ventilation was being continued, using approximately 78 per cent oxygen. The arterial oxygen saturation was 98 per cent, Pvo₂ 32 mm Hg, haemoglobin content 11 g. Later in the day, mechanical ventilation was discontinued and oxygen (8 l./min) was then administered by face mask (M.C.). One hour later, the arterial oxygen saturation was 93 per cent.

Two days later, the patient was answering questions but, although aware of his surroundings, had no recollection of his accident. He was not cyanosed when breathing room air but the arterial oxygen saturation had fallen to 81 per cent. Three days later it had risen again to 93 per cent.

During the second week after injury, the patient developed a transient jaundice that was both haemolytic and hepatocellular in origin (bilirubin: total 2.8 mg/100 ml, conjugated 0.9 mg/100 ml).

DISCUSSION

Taquini, Roncoroni and Aramendia (1956) reported the case of a patient in whom the
fat embolism syndrome developed after injections of thymol in oil. They noted an arterial oxygen tension of 29 mm Hg and an oxygen saturation of 55.4 per cent. The latter value rose to 100 per cent when oxygen was administered. The cardiac output was initially very high but decreased when the patient was breathing oxygen. They believed that the cardiac output increased in an attempt to keep the mean oxygen tension of the tissues as near to normal as possible in spite of severe hypoxaemia.

Sproule, Brady and Gilbert (1964) described their experiences in the management of three young men with the fat embolism syndrome, all of whom were severely hypoxaemic but not cyanosed. In one case, the oxygen saturation was only 51 per cent but the estimated cardiac output was 30 l./min. Sproule and his colleagues believed that the high cardiac output was responsible, in part, for the absence of cyanosis and the anoxia which was initially caused by an alveolar diffusion defect. Muller and Klinger (1965) described two cases of the syndrome with arterial oxygen saturations of less than 60 per cent. Denman, Cairns and Holmes (1964) reported a severe case of fat embolism that was treated successfully with artificial ventilation and oxygen. This treatment corrected arterial oxygen desaturation and reduced the amount of pink frothy tracheal aspirate. Galloon and Chakravarty (1967) reported three cases treated successfully with artificial ventilation. All three patients remained cyanosed initially despite a high respiratory minute volume and inhalation of oxygen by face mask.

Ashbaugh and Petty (1966) believed that severe pulmonary fat embolism reduced the compliance of the lungs and that subsequently severe hypoxia might prove fatal. Denman, Cairns and Holmes (1964) noted that the compliance of the lungs was greatly reduced in their case.

However, Sevitt (1960) concluded that pulmonary fat embolism does not produce significant symptoms or cause serious lung changes in previously healthy subjects. He suggested (Sevitt, 1966) that pulmonary haemorrhage is caused by early bacterial invasion, although in our case culture of the bloody sputum produced no bacterial growth. He also believed (Sevitt, 1962) that respiratory symptoms are neurogenic in origin and caused by cerebral fat embolism. He stated that lung changes are more constant when cerebral fat emboli are present than when they are absent. However, since he also stated that the more fat there is in the lungs the more there is likely to be present in systemic vessels, it could be argued that the presence of cerebral fat emboli merely indicates a high degree of pulmonary fat embolism.

There is good evidence from animal experiments that microemboli confined to the lungs can cause dyspnoea, tachypnoea and pulmonary oedema (Dunn, 1920; Binger and Moore, 1927).

We are reluctant to accept the view that pulmonary fat emboli do not cause significant symptoms and believe that the cause of death in many cases of the fat embolism syndrome may be latent severe anoxia. Monitoring of the blood-gas tensions or arterial oxygen saturation forms an essential part of the successful management of this syndrome.

REFERENCES


LATENT SEVERE ANOXIA AND THE FAT EMBOLISM SYNDROME

ANOXIE SEVERE LATENTE ASSOCIEE AU SYNDROME DE L'EMBOLIE GRAISSEUSE

SOMMAIRE
Chez un homme de 41 ans, on a diagnostiqué un syndrome d'embolie graisseuse, quarante deux heures après un accident du traffic routier. Il y eut une anoxie latente, qui persista en dépit de l'intubation endo-trachéale et de l'administration d'oxygène lorsque la respiration était spontanée. L'oxygenation se restitua presque immédiatement après l'application de ventilation sous pression positive intermittente. On attire l'attention sur la nécessité de déterminer fréquemment la pression de gaz sanguins ou la saturation en oxygène.

LATENSTE SCHwere ANOXIE IN VERBINDUNG MIT DEM FETTEMBOLIESYNDROM: EINE FALLBESCHREIBUNG

ZUSAMMENFASSUNG

Faculty of Anaesthetists of the Royal College of Surgeons in Ireland

SCIENTIFIC MEETING: SATURDAY, MAY 25, 1968

"HAZARDS OF ANAESTHESIA"

The programme for the meeting is as follows:

9.30 a.m.  Introduction. The Dean, Dr. RAYMOND DAVYS.
9.45-10.30 a.m.  Morbidity and mortality statistics. Dr. M. D. VICKERS, Hammersmith Hospital.
10.30-11.15 a.m.  Hazards of regional techniques. Dr. ALFRED LEE, Southend-on-Sea.
11.15-11.45 a.m.  COFFEE.
11.45-12.30 p.m.  Muscle relaxants and prolonged apnoea. Dr. S. A. McDOWELL, Belfast.
12.30-1.45 p.m.  BUFFET LUNCH IN THE COLLEGE.
1.45-2.30 p.m.  Dental anaesthesia. Professor T. J. GILMARTIN, Dublin.
2.30-3.15 p.m.  Paediatrics. Dr. W. WREN, Dublin.
3.15-4.00 p.m.  Awareness in anaesthesia. Professor T. C. GRAY, Liverpool.
4.00 p.m.  TEA.

Tickets for the meeting are £1 11s. 6d. and may be obtained on application to the Secretary, Faculty of Anaesthetists, Royal College of Surgeons in Ireland, St. Stephen’s Green, Dublin 2.

PRIZE ESSAY

The Board of the Faculty of Anaesthetists of the Royal College of Surgeons in Ireland invites any Postgraduates, up to the grade of Senior Registrar, working in Anaesthesia in Ireland, to submit an essay on a subject related to Anaesthesia. A Prize of twenty guineas will be awarded to the best entry. Entries, which should preferably be based on personal investigations by the candidate, should be sent to the Secretary, Board of the Faculty of Anaesthetists, Royal College of Surgeons in Ireland, St. Stephen’s Green, Dublin 2, so as to reach him not later than Monday, July 15, 1968.