Spontaneous Flail Chest

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SUMMARY

The flail chest syndrome usually occurs in association with severe, obvious injury. Two cases are described in which there was no trauma but in which the causative factors were pathological weakening of the thoracic cage due respectively to multiple myeloma and to chronic renal osteodystrophy associated with steroid therapy.

S. Afr. Med. J., **45**, 1262 (1971).

The flail chest syndrome has been well described in several articles.[1,2] The cause has always been related to direct obvious injury and it has been stated categorically that a 'flail chest or stove-in chest results from a severe crushing injury'.[3] This article describes 2 cases presenting with a flail chest in which no obvious trauma was involved.

CASE REPORTS

Case 1

The patient was a 54-year-old alcoholic who had maintained good health until about 8 months before admission to hospital, when he began to lose weight. This weight-loss amounted to 18·2 kg and had accelerated over the last 5 weeks. Three weeks before his admission the patient's wife had noticed 'something funny with his breathing' but was unable to define this feature more accurately. She had also noticed that he had experienced difficulty with speaking and walking and was confused. He smoked 20 cigarettes per day and produced small amounts of white sputum daily. There was no history of trauma.

On examination he was an emaciated, confused male, in whom the most obvious feature was a large anterior flail segment of the thorax involving the sternum and the ribs for about 85 mm on either side of the midline. There was no bruising or other evidence of trauma.

Respiratory rate was 36/minute, blood pressure 100/80 mmHg, pulse rate 116/minute and temperature 37·2°C. He was receiving oxygen by nasal catheters and was not cyanosed. Examination of the chest showed bilateral bronchopneumonia in addition to the flail segment. There was no lymphadenopathy or hepatosplenomegaly and clinical examination was otherwise entirely negative.

His haemoglobin concentration was 9·9 g/100 ml, white blood cell count 8 300/mm^3, and platelets 60 000/mm^3. Blood urea was 133 mg/100 ml with normal electrolytes.

*Date received: 2 June 1971.

Serum calcium was 5·3 mEq/litre. A bedside antero-posterior chest X-ray was performed and this showed multiple cystic areas in the ribs, scapulae and clavicles together with several fractures, although not all the fractures in the region of the flail segment were clearly visible. A bone-marrow examination was performed immediately and numerous primitive plasma cells, typical of multiple myeloma, were identified. The diagnosis was subsequently confirmed by protein electrophoresis which showed an abnormal fraction migrating between the beta and gamma areas. Further X-rays revealed widespread, typical lytic lesions in the skull and pelvis. There was no Bence-Jones protein in his urine.

Analysis of arterialized capillary blood showed a pH of 7·57, P_{CO_2} 65 mmHg, standard bicarbonate 27·1 mEq/litre and base excess +4 mEq/litre. These results were interpreted as being due to a combination of alveolar hypoventilation and an infusion of sodium bicarbonate which the patient had received.

The immediate problem was whether to institute intermittent positive pressure respiration for his flail chest. However, because it was doubted whether his ribs would ever heal satisfactorily, and because of the diffuse, malignant nature of his disease, it was decided not to provide artificial respiration and to treat him conservatively. Therapy for the myeloma, consisting of corticosteroid and melphalan, together with antibiotics, was instituted immediately but the patient died 12 hours later.

Case 2

The patient was a 32-year-old female who had suffered from chronic renal disease for many years. One year previously she had a bilateral nephrectomy followed by a renal transplant. Postoperatively she required very high doses of corticosteroids and azathioprine, but despite this therapy, the transplanted kidney had to be removed after 8 months because of rejection. Thereafter she was maintained on chronic haemodialysis for 4 months.

She was admitted to hospital suffering from acute upper respiratory tract obstruction of unknown aetiology. An emergency tracheostomy was performed and she improved markedly. About 60 hours later, however, it was noticed she had an area of paradoxical movement involving the right anterior chest wall. At this stage her respiratory rate was 24/minute while analysis of arterialized capillary blood showed that the pH was 7·46, P_{CO_2} 36·0 mmHg, standard bicarbonate 25·3 mEq/litre, base excess +1·5 mEq/litre and P_{O_2} 59·0 mmHg. Oxygen was administered via the tracheostomy tube, but 24 hours later her condition began to deteriorate. Respiratory rate increased to 30/minute and she was obviously distressed and in increasing respiratory difficulty. Intermittent positive pressure respiration was instituted using a Bird respirator and the respiratory distress was alleviated. Her general condition continued to deteriorate, however, and despite intensive therapy the patient died after 8 days on the respirator.

X-rays done at the time of the diagnosis of the flail segment were difficult to interpret as only portable supine pictures could be obtained. However, definite fractures of ribs 8, 9 and 10 in the right midaxillary line were visible. The cause of the fractured ribs was thought to be severe bone weakness due to a combination of renal osteodystrophy and osteoporosis resulting from prolonged high-dosage steroid treatment. Minor unnoticed trauma as a precipitating factor could not be excluded, but no history of this could be elicited.

DISCUSSION

The clinical syndrome of flail chest practically always occurs in a setting associated with obvious severe injury. This is often due to a motor vehicle accident or it occurs in the wards, following external cardiac massage. The area of paradoxical breathing is due to multiple fractures of ribs with loss of chest wall stability so that its movement responds directly to alterations of pressure in the pleural cavity. The essential lesion, therefore, is the presence of multiple fractured ribs, and in the literature this has always been associated with obvious trauma.

Severe diffuse disease, such as occurred in the 2 cases described above, would markedly weaken the bones and predispose them to multiple fractures. In neither of the cases could any history of direct trauma be obtained, and although its occurrence cannot be excluded, it could only have been so minor as to pass unnoticed. It is possible that an otherwise unimportant incident, such as cough, produced some fractures with resultant stresses at various rib sites and consequent fractures through other greatly weakened areas.

Both cases were complicated by respiratory failure. The recognized treatment for the flail chest syndrome with respiratory embarrassment is intermittent positive pressure respiration continued until the ribs heal sufficiently to enable normal thoracic movements to be carried out. This treatment was not instituted in the patient with myeloma because his disease was so diffuse that an extremely poor prognosis was indicated. In addition, it was doubted whether sufficient healing could ever be obtained to restore stability to his chest wall.

The patient who had previously had a kidney transplant was put on to controlled respiration as a second transplant was being contemplated, and, in addition, it was felt that adequate bone healing and stability might occur. However, the idea of a second transplant was abandoned and she died in renal failure.

We wish to thank Dr M. Salmon, Medical Superintendent of the Johannesburg Hospital; and Dr H. Grové, Director of Hospital Services in the Transvaal, for permission to publish.

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