ability to ketoacidosis and their more general perceptions of control of diabetes provided useful pointers to risks of subsequent ketoacidosis during use of continuous subcutaneous infusion.

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(Accepted 13 May 1986)

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Spontaneous pneumomediastinum in two stowaways

We describe two cases of pneumomediastinum which occurred in stowaways on a banana boat arriving at Avonmouth Docks, Bristol, from Columbia, South America. The voyage took 17 days, during which time the two men ate and drank very little. The temperature in the hold of the ship was 54-59°C.

Case reports

Case 1—A 23 year old man was hypothermic (34·8°C), moderately dehydrated, and, except for resting tachycardia of 96 beats/min, showed no abnormal cardiovascular or respiratory signs. There was evidence of cold injury to both feet. Investigations showed blood urea concentrations of 76 mmol/l (458 mg/100 ml), creatinine 475 μmol/l (5·4 mg/100 ml), sodium 155 mmol(mEq)/l, chloride 105 mmol(mEq)/l, bicarbonate 21 mmol(mEq)/l, and potassium 4·4 mmol(mEq)/l. Concentration of urinary sodium was 28 mmol/l and urea 401 mmol/l (2·4 g/100 ml), indicating dehydration with prerenal uraemia and no metabolic acidosis. Biochemical values returned to normal when the patient was rehydrated. The patient complained of chest pain, worsened by breathing and recumbency. Despite the absence of physical signs in the chest a radiograph showed mediastinal and soft tissue emphysema. This resolved within five days. He was found to be infested with *Ascaris lumbricoides*.

Case 2—A 19 year old man was also hypothermic (35·2°C) and moderately dehydrated with a resting tachycardia of 100 beats/min. There were no other abnormal physical signs. The blood urea concentration was 44·5 mmol/l (268 mg/100 ml), creatinine 171 μmol/l (1·9 mg/100 ml), sodium 152 mmol/l, chloride 105 mmol/l, bicarbonate 25 mmol/l, and potassium 3·7 mmol/l. Urinary sodium concentration was 7 mmol/l and urea 696 mmol/l (4·2 g/100 ml), which again confirmed dehydration with prerenal uraemia but no metabolic acidosis. These measures became normal when the patient was rehydrated. Routine chest radiography showed emphysema of the mediastinum and chest wall. There were no symptoms or signs, however, of either of these conditions. Resolution occurred within five days. He was infested with *Trichuris trichuria*, *Necator americanus*, and *Strongyloides stercoralis*.

Comment

Air reaching the mediastinum from the interstitial tissues of the lungs results from rupture of marginal alveolar bases and may occur when there is a sudden rise of intra-alveolar pressure. The pathophysiological mechanism that results in interstitial air in cases of asthma has been elucidated by Macklin and Macklin.[1] They found that bronchospasm, mucosal oedema, and inspissation of secretions in people with asthma caused air to be trapped with resulting stretching of alveoli. Supporting structures such as the pulmonary arteries, veins, and alveolar septa have limited elasticity, and as distension increases shearing forces, developed by exaggerated respiratory effort, rupture the marginal alveolar bases. The escaping air dissects along the perivascular sheaths towards the hilum.

Similar mechanisms have been postulated in pneumomediastinum associated with artificial ventilation, parturition, strenuous exercise, and diabetic ketosis. In diabetic ketosis the rise in intra-alveolar pressure is thought to be a result of repeated vomiting. This is also thought to cause some cases of pneumomediastinum in patients with anorexia nervosa. Pneumomediastinum, however, has been reported in the absence of vomiting in a girl with anorexia nervosa and in an emaciated adolescent boy with functional anorexia of a month's duration.[2,3] Experimental work on rats' lungs has shown that an inadequate diet, by decreasing tissue elasticity and increasing surface forces, may result in air trapping owing to premature closure of the airway.[4] Microscopic examination of these lungs showed a decrease in the volume density of lamellar bodies, mitochondria, and cytoplasm. As the granular pneumocyte lamellar bodies are the site of surfactant storage, its reduction may partly account for the altered mechanics, and hence a cellular cause for the raised intra-alveolar pressure is possible.

The possibility that similar changes might occur during starvation and account for pneumomediastinum in patients with anorexia nervosa where vomiting is not a factor has previously been considered.[3,4] These stowaways had been in the ship's hold for nearly three weeks with very little food or water. Their diet before this was possibly poor, and, moreover, both were infested with intestinal worms. The mechanism of pneumomediastinum in their cases might be similar to that postulated in some patients with anorexia nervosa.

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(Accepted 13 May 1986)

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Life threatening reaction to tuberculin testing

Severe anaphylactoid reactions after tuberculin testing are rarely seen nowadays. We report on a patient who developed an acute severe systemic upset with renal failure and hepatic dysfunction after an intradermal injection of 0·1 ml tuberculin purified protein derivative at a 1:10 000 dilution (1 IU).

Case report

A 35 year old Portuguese woman, who had lived in England for 14 years, presented with a two month history of cervical lymphadenopathy. She was otherwise well. Histological examination of a lymph node biopsy specimen had shown epitheloid granulomata but no acid fast bacilli on Ziehl-Neelsen staining; the tissue was set up for culture. All initial investigations, including renal and hepatic function, were normal. A 0·1 ml sample of a 1:10 000 (1 IU) diluted tuberculin purified protein derivative was given intradermally in the right forearm. Within three hours of this injection she developed a fever with rigors, sweating, and profuse vomiting; over the next 48 hours her condition deteriorated with generalised aches and pains, dry cough, and oliguria. On admission to hospital she was feverish (39°C) with a low volume tachycardia (120 beats/minute) and a blood pressure of 70/40 mm Hg. There was no reaction at the site of the tuberculin injection.

Investigations showed a white cell count of 9×10^6/l with normal differential; concentrations of urea 24·3 mmol/k (normal range 3·0-6·5 mmol/l) (146 mg/100 ml (18-39 mg/100 ml)). sodium 130 mmol (mEq)/l (135-145 mmol (mEq)/l), potassium 3·4 mmol (mEq)/l (3·5-5·0 mmol (mEq)/l), and total bilirubin 45 μmol/l (5-17 μmol/l) (2·7 mg) 100 ml (0·3-1·0 mg/100 ml)); activities of aspartate transaminase 117 IU/l (5-40 IU/l) and alkaline phosphatase 207 IU/l (35-130 IU/l); concentration of total protein 64 g/l (60-80 g/l) with an albumin concentration of 36 g/l (30-50 g/l); prothrombin time 17 seconds (11-14 seconds) and partial thromboplastin time 43 seconds (30-40 seconds) but no fibrinogen degradation products. The patient's urinary volume was less than 2 ml/hour; a 24 hour urinary collection showed a total protein concentration of 1·20 g/l (0·05), sodium concentration 62 mmol/l (100-250 mmol/l), potassium concentration 45 mmol/l (40-120 mmol/l) and urea concentration 183 mmol/l (170-600 mmol/l (1·1 (1·0-3·6