she had been well. Her general condition was good and her temperature was normal. She was not obese. The abdomen was uniformly distended. A non-tender irreducible right femoral hernia was present. Obstructive bowel sounds were heard. Plain abdominal x-ray films confirmed a small bowel obstruction but also showed a mediastinal fluid level. This fluid level remained even though nasogastric aspiration produced 500 ml of normal gastric fluid.

The femoral hernia was explored through a combination of a low and high approach. The sac contained frank pus and necrotic small bowel. About 15 cm of ileum was resected with end to end anastomosis. The hiatus hernia was palpated through the laparotomy wound; it admitted three fingers and the intrathoracic stomach was easily reducible. Initially she made a good recovery but about 36 hours postoperatively developed signs of septicaemic shock and died a few hours later despite intensive treatment. At necropsy half the stomach was intrathoracic through a sliding type of hiatus hernia. The intrathoracic portion was necrotic; it was extremely thin and had three large perforations in it. The pleural cavities contained two litres of gastric fluid. The small bowel was moderately distended and peritonitis was present in the right iliac fossa. The anastomosis was intact.

Comment

Hiatus hernia is liable to the same complications as hernias elsewhere.[1] Obstruction or torsion of the stomach in the hernial sac is rare but may cause gangrene and perforation with a high mortality rate.[2] Paraoesophageal hernias are more dangerous than oesophageal ones.[3] Obesity is an important cause of chronically raised intra-abdominal pressure.

Perforation usually does not follow any definite event, although Watts[4] has described ruptures resulting from the sudden increase in intra-abdominal pressure which accompanies retching and vomiting. In this patient probably postoperative distension of the bowel caused a sustained period of increased intra-abdominal pressure with compression of blood vessels at the hernial ring and ischaemic necrosis of the intrathoracic stomach. This sequence of events has not been reported.

I thank Mr J H Kilshaw for permission to publish details of his patient.

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Phaeochromocytoma with lactic acidosis

Although adrenaline administration in man has been shown to increase lactic acid concentrations,[1] the clinical coincidence of a catecholamine-secreting tumour with lactic acidosis has not yet been reported. We describe here a patient with phaeochromocytoma whose main feature on admission was severe lactic acidosis.

Case report

A 33-year-old Italian woman had been treated by her family doctor over 12 months for epigastric pain. She complained of cramps in her stomach associated with spells of paleness and sweating. Evaluation by a gastroenterologist, including gastroduodenoscopy, showed no abnormality, and symptomatic treatment was started. Repeated blood pressure recordings were normal.

On the morning of the day of admission she suffered from acute pain in the back, nausea, and palpitations. Her doctor referred her to a local hospital because of ventricular ectopic beats and epigastric pain. On admission she was pale and had dyspnoea and cold extremities. The patient was transferred to our hospital later that day because of rapidly increasing dyspnoea. She was in acute respiratory distress; she was fully conscious but her pupils were dilated and unresponsive to light. The blood pressure was 180/130 mm Hg, and an electrocardiogram (ECG) showed sinus tachycardia and suggested a subendocardial lateral wall infarction. Chest radiography showed diffuse pulmonary oedema and a normal sized heart. A naso-tracheal tube was inserted, and artificial ventilation with positive end-expiratory pressure was started. Blood gas analysis on admission showed severe metabolic acidosis. The table shows the laboratory values on admission and four hours later.

Laboratory values

	Normal range	On admission	Four hours after admission
Plasma lactate (mmol/l)	0·2-1·2	22·0	17·9
Blood glucose (mmol/l)	4·4-6·7	51·0	34·2
Plasma β-hydroxybutyrate (mmol/l)	0·02-0·10	0·67	
Plasma creatinine (μmol/l)	53-123	185	247
Plasma phosphorus (mmol/l)	0·32-0·81	6·26	
Haemoglobin (g/dl)		15·1	14·2
Arterial pH	7·35-7·45	6·67	6·9
Arterial P_{CO_2} (kPa)	4·67-6·0	6·13	4·93
Arterial P_{O_2} (kPa)	9·33-13·33	6·26	11·20
Base deficit (mmol/l)	3·3- (-1·2)	34	27
Plasma insulin (6-14 mU/l)	6-14	47	
Plasma glucagon* (pmol/l)	17·1-42·9	24·3	23·4
Plasma noradrenaline (nmol/l)	0·95-2·19	4581	3635
Plasma adrenaline (nmol/l)	0·05-0·66	3357	742
Plasma dopamine (nmol/l)	0·39-0·98	320	197

*30 K-antibody (Dr R Unger).

Conversion: SI to traditional units—Lactate: 1 mmol/l ≈ 9·0 mg/100 ml. Glucose: 1 mmol/l ≈ 18 mg/100 ml. β-Hydroxybutyrate: 1 mmol/l ≈ 10·4 mg/100 ml. Creatinine: 1 μmol/l ≈ 0·0113 mg/100 ml. Phosphorus: 1 mmol/l ≈ 3·1 mg/100 ml. Blood gas: 1 kPa ≈ 7·5 mm Hg. Glucagon: 1 pmol/l ≈ 3·48 ng/l. Noradrenaline: 1 nmol/l ≈ 16·9 ng/100 ml. Adrenaline: 1 nmol/l ≈ 18·3 ng/100 ml. Dopamine: 1 nmol/l ≈ 15·3 ng/100 ml.

A raised blood lactate concentration and severe hyperglycaemia were present. Plasma catecholamine concentrations were measured using radio-enzymatic techniques. Plasma adrenaline and noradrenaline were over 5000 times the normal range determined in normal volunteers. Drug intoxication was excluded by examining a blood specimen and by the history. Four hours after admission and after treatment with 200 mmol (mEq) bicarbonate and with 20 U of regular insulin her blood pH was still 6·9, her base deficit 27 mmol(mEq)/l, and her blood lactate 17·9 mmol(161 mg/100 ml)/l. Haemodialysis was started to try to correct the severe metabolic acidosis. Shortly afterwards the patient's condition rapidly deteriorated. Her blood fibrinogen concentrations fell to undetectable levels, which suggested the presence of disseminated intravascular coagulation. Her diastolic blood pressure dropped from 110 to 75 mm Hg; she developed pump failure and died of a cardiac arrest one hour later.

Necropsy showed a left adrenal phaeochromocytoma (52 g) with a typical histological pattern. Other pathological findings included pulmonary oedema and focal necrotic areas of the heart.

Comment

This was an unusual case of phaeochromocytoma in which the clinical course was characterised by the development of severe lactic acidosis and pulmonary oedema. The fact that the blood pressure was maintained and lactic acidosis persisted despite adequate ventilation indicates that lactic acidosis was a direct consequence of the excessive concentration of circulating catecholamines. According to Cohen's classification,[3] this was a case of type B lactic acidosis. The increase in lactate concentrations might have been caused by increased production by peripheral tissues or by a reduced rate of removal by the liver. The stimulatory effect of adrenaline on glycogenolysis and insulin resistance was probably responsible for the persistent severe hyperglycaemia. Despite the raised blood glucose value, endogenous insulin concentrations were relatively low, probably because of the inhibitory effect of the catecholamines on insulin secretion. Excessive circulating dopamine concentrations have not yet been reported in phaeochromocytoma, though raised urinary dopamine excretion has been found in rare cases of malignant phaeochromocytoma.[4]

This patient died because her condition was not diagnosed and treated before heart failure occurred. Myocardial infarction and focal myocarditis are sometimes associated with phaeochromocytoma.[5] The absence of a history of hypertension suggests that the β-adrenergic effects of the raised adrenaline concentrations dominated the early clinical course, whereas on admission vasoconstriction, decreased gut motility, and dilatation of the pupils were signs of α-adrenergic stimulation. Pupillary dilatation represented a further unique feature of the case.

The differential diagnosis of idiopathic lactic acidosis should

include phaeochromocytoma since recognition of the condition and treatment with adrenergic-blocking agents may be life saving.

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Malignant mesothelioma and gas-mask assemblers

Since the relation between malignant mesothelioma and asbestos exposure was first described in 1960[1] there have been several studies of groups exposed to asbestos. Jones[2] reported the incidence of mesothelioma at three factories which assembled service respirators containing blue asbestos and charcoal fibres during the second world war. To date, 50 workers at these factories have developed a mesothelioma. Apparently there are no reports of mesothelioma after the assembly of gas masks in the first world war. We describe one such patient, who assembled masks in the 1914-18 war, and another case in a man who was briefly employed at a mask assembly plant in Leyland, Lancashire, in the second world war.

Case reports

Case 1—An 83-year-old widow had been well until three months before presentation, when she developed dull right-sided chest pain. Three weeks before admission she had fallen and become progressively breathless, and hence came to hospital. The only finding was a large left-sided pleural effusion, which was confirmed by the chest *x*-ray film. Heavily blood stained fluid (3·5 l) was aspirated by continuous suction. Cytological appearances suggested a mesothelioma and pleural biopsy confirmed this. The effusion recurred and was tapped on two further occasions. Thoracoscopy was performed and further biopses taken (again confirming malignant mesothelioma) followed by pleurodesis with iodised talc.

She had been a housewife except during the first world war, when she had assembled gas masks at a factory in SE London. The early gas masks were filled with cloth fibres, but with the deployment of poison gas in the Battle of Ypres in 1915 there was an urgent need for adequate protection. Several types of filters were devised and for six months in late 1915 Service respirators with asbestos filters were assembled by Baldwin Bros Ltd (Munitions Department Memorandum of the War Office, 1916) where she had worked.

Case 2—A 62-year-old lorry driver who had had a transitional cell carcinoma of the bladder treated in 1971 had remained well until November 1977, when he complained of dull right-sided chest pain and a non-productive cough. The straight chest *x*-ray film was unremarkable but the lateral film showed a mass lying deep to the sternum. Tomography showed no invasion of the sternum. The mass was biopsied and the histological findings were diagnostic of malignant mesothelioma.

He had worked as a lorry driver but had never serviced his own vehicle. Nevertheless, during the second world war he had collected gas masks from a factory in Leyland, Lancashire, and for three months had assembled the filters for the masks. He had no other history of exposure to asbestos and there was therefore a 37-year latency before symptoms developed.

Comment

These cases reinforce the suggestion by Elmers and Simpson[3] that mesothelioma must be considered in anyone with a pleural lesion of insidious onset. The latent period between exposure and presentation is remarkably constant in several large series at 42 years (±SD13 years).[4] The first patient developed a mesothelioma 63 years after exposure, which equals or even exceeds the latent period described.[5] The association between gas-mask assembly in the first world war and mesothelioma has not been described, possibly because other patients with a shorter latency would have died before mesothelioma was regarded as a malignant entity. The second case typifies those described by Jones in gas-mask assemblers who have been exposed to blue asbestos, and he has reported the incidence of mesothelioma at this particular factory.[2] These cases illustrate the importance of taking a detailed history.

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Recurrent pleural effusion in Waldenström's macroglobulinaemia

Primary disease of the pleura or pulmonary parenchyma in Waldenström's macroglobulinaemia (WM) is uncommon[1] and may be the predominant manifestation.[2] We report a patient presenting with recurrent pleural effusion; this unusual mode of presentation led to delay in recognising the underlying disorder.

Case report

A 66-year-old Chinese man presented in September 1975 with a chronic cough for six months and progressive exertional dyspnoea for three months. He denied having fever, chest pain, haemoptysis, weight loss, weakness, or contact with tuberculosis. He had stopped smoking 10 years before. Chest *x*-ray film showed a right pleural effusion. Mantoux test using 1 TU PPD gave 14 mm induration. Sputum smears and cultures gave negative results for tubercle bacilli. Pleural aspiration showed straw-coloured fluid containing 70 g/l of protein, some lymphocytes, but no malignant cells. Pleural biopsy was unsuccessful. Tuberculous pleurisy was diagnosed and he was given streptomycin, isoniazid, and ethambutol. He developed bilateral effusion in December 1975. Despite the addition of prednisolone, pleural fluid reaccumulated rapidly over the months necessitating further aspirations. Fluid examination showed lymphocytes and histiocytes but no malignant cells; protein concentration varied from 50 g/l to 109 g/l. A pleural biopsy specimen in May 1976 showed focal collections of lymphocytes.

Laboratory investigations in March 1976 showed: haemoglobin 9·4 g/dl; white cell count $5·5 \times 10^9$/l (5500/mm^3); platelet count 175×10^9/l (175 000/mm^3); reticulocyte count 1·0%. Erythrocyte sedimentation rate (Westergren) 132 mm in first hour. Proteinuria 0·55 g/24 h to 1·1 g/24 h was present. Total serum protein concentration was 103 g/l; serum albumin 25 g/l; serum globulin 78 g/l; serum gammaglobulin 45 g/l. Serum electrophoresis showed a monoclonal peak in the gamma region. Serum immunoglobulin concentrations were IgM 45·5 g/l; IgG 10·0 g/l; IgA 1·92 g/l. The results of Sia test, for serum cryoglobulins, and of urine for Bence Jones protein were negative. Pleural fluid immunoglobulin concentrations were: IgM >72 g/l; IgA nil; IgG 7·2 g/l. Marrow examination showed a slight increase of plasma cells and lymphocytes. Skeletal survey was normal. Results of estimating concentrations of blood urea, serum electrolytes, and serum calcium and phosphate, and liver function tests were normal. Rheumatoid factor, LE cells, and antinuclear factor were not present.

At this stage the possibility of WM was considered. Clinically he looked wasted and weighed 50 kg. He had no finger clubbing, bleeding tendency, visual disturbance, or neurological abnormality. The liver, spleen, and lymph nodes were not enlarged. Fundi showed slight venous distension. Anti-