on a background of slowly progressive deterioration of mobility, personality, and intellect.

Neurological investigation included computed tomography, which showed cortical atrophy, and a non-specifically abnormal electroencephalograph, with much high voltage β activity. Random estimations of blood glucose concentrations were consistently normal except for one subnormal result in 1976 when it was thought that she had accidentally taken sulphonylurea tablets. Subsequent random blood glucose estimations were normal and this was not investigated further.

Inappropriate insulin secretion was confirmed by finding serum insulin concentrations of 9·3 and 10·2 mU/l during subsequent hypoglycaemic episodes.[3] Further episodes were effectively prevented by oral diazoxide. While undergoing investigation, however, she developed rapidly fatal septicaemia. At postmortem examination a well differentiated islet cell tumour, 15 mm in diameter, was found in the pancreas. Her brain was atrophic, and histological examination showed multiple focal infarcts of variable size in the cerebral and cerebellar hemispheres and basal ganglia, with relative sparing of the temporal lobes and hippocampus. In the cerebellum there was widespread loss of Purkinje cells.

Comment

An insidious and irreversible dementing process without acute episodes has been described with insulinoma but is rare; two major reviews of a total of 318 cases found evidence of irreversible neuropsychiatric deficit in only three cases.[2,4] The duration of symptoms before diagnosing insulinoma often exceeded 10 years,[1] and one well documented case presented with a history of over 25 years.[5] Such a delay in diagnosis is presumably more likely when the course is atypical or relatively benign.

We suggest that our patient's gradual global neurological deterioration since adolescence was the result of recurrent episodes of hypoglycaemia due to an insulinoma that had eluded diagnosis for over 30 years. This case illustrates the difficulty of detecting intermittent hypoglycaemia and the importance of excluding it with fasting blood samples in cases of progressive, unexplained neurological deficit.

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Southampton General Hospital, Southampton SO9 4XY
J A SNOOK, BM, MRCP, medical registrar
R VANDERSTAR, MB, MRCP, medical registrar
R O WELLER, PHD, FRCPATH, professor of neuropathology

Correspondence to: Dr Snook.

Predicting risk of diabetic ketoacidosis in patients using continuous subcutaneous insulin infusion

Several recent reports have discussed an increased incidence of diabetic ketoacidosis during continuous subcutaneous insulin infusion compared with conventional injection treatment.[1-3] The possibility that this increased incidence may be at least partly due to certain characteristics of the patients who use infusion treatment has not previously been explored.

Patients, methods, and results

During a feasibility study of the use of continuous subcutaneous insulin infusion in a diabetes clinic 11 patients were identified who had experienced episodes of diabetic ketoacidosis over a period of two years and six months while using the infusion treatment.[4] These patients were matched for age, sex, and duration of diabetes with 11 patients using continuous subcutaneous infusion who had not experienced ketoacidosis. All 22 patients had completed a series of newly designed psychological scales before starting the treatment. The psychometric development of these scales has been described elsewhere.[5] Ketoacidosis was defined as ketosis and hyperglycaemia with thirst, nausea, malaise and vomiting, and plasma bicarbonate concentration <15 mmol(mEq)/l.

The groups were compared for age at leaving full time education, daily insulin dosage and carbohydrate intake, duration of insulin use, initial and 12 monthly glycosylated haemoglobin concentrations and psychological measures. The psychological scales provided measures of perceived control of diabetes, which included five scales measuring internal versus external control and two scales measuring perceptions of medical factors affecting control. Scales measuring health beliefs specific to diabetes provided eight further variables. Independent group *t* tests were used for comparison between the two groups on all measures. The table shows the results.

Comparison of demographic, clinical, and psychological variables in patients who suffered episodes of ketoacidosis and those who did not. Values are means (SD)

	Developed ketoacidosis	Did not develop ketoacidosis	*t*	df	p
Demographic features					
Sex	7F:4M	7F:4M			
Age (years)	35·2 (13·8)	34·0 (13·1)	−0·21	20	NS
Age at leaving full time education	15·4 (1·3)	18·5 (3·6)	2·29	17	<0·05
Clinical and biochemical features					
Duration of diabetes (years)	13·9 (10·8)	14·2 (10·0)	0·06	20	NS
Daily insulin dosage (U/kg)	55·8 (21·5)	61·0 (19·7)	0·10	20	NS
Glycosylated haemoglobin (%):					
Initially	60·4 (9·2)	58·5 (12·2)	−0·39	20	NS
At 12 months	51·2 (6·9)	52·3 (7·8)	0·32	18	NS
Psychological features					
Perceived control scales*:					
Internality	23·9 (7·0)	25·1 (4·3)	0·47	17	NS
Personal control	22·8 (4·6)	27·7 (2·2)	2·98	16	<0·01
Foreseeability	17·9 (4·4)	24·2 (8·5)	1·92	17	0·071
Externality	12·1 (7·4)	10·2 (6·5)	−0·61	17	NS
Chance	12·7 (6·5)	11·1 (7·1)	−0·51	18	NS
Treatment	18·1 (4·0)	13·9 (4·5)	−2·09	17	0·052
Medical control	14·3 (6·3)	13·3 (7·4)	−0·30	17	NS
Health belief scales:					
Perceived severity†:					
Hypoglycaemia	3·4 (1·2)	4·4 (1·3)	1·88	20	0·075
Hyperglycaemic coma	5·4 (0·7)	5·5 (0·5)	0·71	20	NS
Perceived vulnerability†:					
Hypoglycaemia	5·0 (1·2)	4·4 (1·9)	−0·96	20	NS
Hyperglycaemic coma	4·2 (1·2)	2·9 (1·2)	−2·50	20	<0·05
Perceived severity of complications‡	20·7 (1·4)	21·1 (2·0)	0·49	20	NS
Perceived vulnerability to complications‡	15·0 (3·6)	15·4 (3·5)	0·24	20	NS
Perceived benefits of treatment§	26·1 (7·2)	28·8 (6·2)	0·95	20	NS
Perceived barriers to treatment§	16·5 (8·4)	12·0 (5·9)	−1·44	20	NS

*Scores ranged from 0 to 36: the higher the score the stronger the attribution towards the variable.
†Scores ranged from 1 to 6: the higher the score the greater the perceived severity or vulnerability.
‡Scores ranged from 4 to 24: the higher the score the greater the perceived severity or vulnerability.
§Scores ranged from 0 to 36: the higher the score the more benefits or barriers perceived.

Clinical and demographic measures did not differ between the groups, except for age at leaving full time education: patients who developed ketoacidosis had had significantly fewer years of full time education.

Psychological measures—Significant or nearly significant differences were obtained for three of the seven scales measuring perceived control—namely, personal control, foreseeability, and treatment. Compared with the patients who had not experienced episodes of ketoacidosis, those who had felt less personally in control of their diabetes, thought that outcomes were less foreseeable, and attributed more responsibility for diabetes management to the treatment. The health belief ratings indicated that patients who had had episodes of ketoacidosis tended to feel more vulnerable to problems related to their diabetes, differences between groups reaching significance for perceived vulnerability to hyperglycaemic coma. None of the differences in ratings of perceived severity of complications reached significance, although the patients who had had episodes of ketoacidosis tended to rate each problem as less severe.

Comment

The two groups of patients were distinguishable only by the psychological variables measured and age at leaving full time education, the patients who developed ketoacidosis having had fewer years of education. These patients seemed to be looking for a medical solution to their diabetes and considered that they personally had little control over the disease. Such patients may be less likely to detect metabolic problems and to initiate emergency action when necessary. Before using continuous infusion treatment the patients' who went on to develop ketoacidosis rated themselves as being more vulnerable to this complication, although prior experience of ketoacidosis was similar in both groups. These feelings of vulnerability might reflect their feelings of being less in control of their diabetes.

Over optimistic expectations of continuous subcutaneous infusion may have encouraged patients to assume that less personal responsibility for their diabetes would be required than with injection treatment. Ketoacidosis may, however, have arisen because of some characteristic of infusion treatment combined with the psychological characteristics identified here. Either way, this study indicates that patients' perceptions of their vulner-

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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Department of Psychology, University of Sheffield, Sheffield S10 2TN
C BRADLEY, BSC, PHD, director, diabetes research
D S GAMSU, BA, research associate

Royal Hallamshire Hospital, Sheffield S10 2JF
G KNIGHT, MB, MRCP, research registrar
A J M BOULTON, MD, MRCP, senior registrar in medicine
J D WARD, MD, FRCP, consultant physician

Correspondence to: Dr Bradley.

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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Departments of Medicine and Radiology, Ham Green Hospital, Bristol
SYLVIA M ALDRIDGE, MRCP, FRCR, senior registrar in radiology
S C GLOVER, MRCP, consultant physician
C JOHNSON, FRCR, consultant radiologist

Correspondence to: Dr Aldridge, Department of Radiology, Royal Infirmary, Bristol.

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