plexes suggests their responsibility in the recurrence of pulmonary haemorrhage.

The origin and nature of these immune complexes remain unknown. They may be composed of basal membrane antigens and the corresponding antibodies or they may result from an intercurrent immunological attack of some sort. Our findings emphasise the importance of assaying for immune complexes in patients with Goodpasture's syndrome who suffer recurrences of pulmonary haemorrhages.

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Haemoperfusion for theophylline overdose

Self-poisoning with theophylline is becoming increasingly common, and probably reflects the introduction of sustained-release formulations and their extensive use. Helliwell and Berry¹ have drawn attention to the serious nature of such overdoses, noting that hypotension, cardiac arrhythmias, and hypokalaemia are associated with a high mortality while tachycardia, nausea, and vomiting, the early features of theophylline intoxication, are not universally present. Indeed, the severity of the overdose may be shown only when convulsions occur, and Zwillich² has noted that such a presentation carries a 50% mortality. Relatively small overdoses (1.5-4.0 g) from sustained-release formulations have caused severe symptoms, whose onset may be delayed up to 10 hours after ingestion. We report here such a patient who was successfully treated by charcoal haemoperfusion.

Case report

A 15-year-old girl (43 kg) who had been prescribed sustained-release theophylline (Phyllocontin) 225 mg twice a day ingested 1.575 g of theophylline (seven tablets) as a result of personal stress. The ingested dose was confirmed by a count of the remaining tablets. Gastric lavage two hours after ingestion produced only a small amount of tablet debris. She remained deceptively well for 10 hours before the sudden onset of hypotension and tachycardia, followed by a cardiorespiratory arrest. After resuscitation she had a profound acidosis, hypokalaemia, and cardiac arrhythmias, notably ventricular tachycardia and supraventricular tachycardia.

The theophylline concentration measured 16 hours after ingestion was 146 mg/l (therapeutic range 5-15 mg/l). In view of this high level, her clinical deterioration, and the onset of frequent convulsions, she was

transferred to Guy's Hospital for further management. Haemoperfusion was initiated with a charcoal column (DHP-1 Hydron Haemoperfusion Cartridge, Kuraray Co Ltd, Japan) and continued for three hours, reducing the theophylline level to 31 mg/l. Analysis performed eight hours after the end of haemoperfusion showed that there had been no rebound in the drug level. Her recovery was delayed because of chest complications, including an aspiration pneumonia which had followed the cardiorespiratory arrest. Peritoneal dialysis and then haemodialysis were required for acute renal failure, presumed to be secondary to the profound hypotension.

Comment

There are two American single case reports^{3,4} of charcoal haemoperfusion being used successfully to manage theophylline overdose. In our patient the mean drug clearance was 97 ml/min and the average flow rate through the column 140 ml/min. During 170 minutes of haemoperfusion the venous theophylline concentration was reduced from 120 mg/l to 31 mg/l, and we calculate that 920 mg of theophylline (58% of the stated ingested dose) was cleared from the blood.

This case illustrates the serious nature of theophylline overdose and the value of measuring the plasma concentration early. Relatively small doses, particularly of the sustained-release preparations, seem to produce serious complications. An additional factor may be concurrent therapeutic administration of theophylline, as in our patient. Our results suggest that haemoperfusion may be of value for severely intoxicated patients and should be considered early as a form of treatment in those who show signs of clinical deterioration in association with high plasma concentrations.

We thank the many doctors and nurses who were involved in the care of this patient.

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Anorexia nervosa in diabetes mellitus

Heightened awareness of carbohydrate consumption is a feature of both diabetes mellitus and anorexia nervosa. One authority has noted that the association of these relatively common conditions is surprisingly rare¹ and we have found only one case reported.² We describe the cases of three insulin-dependent diabetic women who presented to one diabetic clinic with anorexia nervosa. Two patients with a milder condition who refused psychiatric referral were also treated during the same six-month period.

Case reports

(1) The patient developed diabetes when aged 11 years. Difficulties in establishing diabetic control were in part attributed to the fact that the diabetes became a focus of family conflict. The patient rarely tested her urine or co-operated with dietary restrictions. When aged 17 she started to diet. Over six months her weight fell from 55.5 kg to 40.6 kg (standard weight 54.9 kg) and she ceased menstruating. No physical abnormalities were found and anorexia nervosa was diagnosed. Rapid weight loss did not lead to deterioration in diabetic control. In the psychiatric ward, however, manipulation of the diabetes complicated the management programme. Secret vomiting, refusal to provide urine specimens, and rejection of food after taking insulin all contributed to unexpected severe hypoglycaemic attacks.

Diabetic control was very difficult. Avoiding confrontation by giving the patient responsibility for both her eating and managing her diabetes merely resulted in further weight loss. After seven months she discharged herself weighing 46 kg. Four months later her pattern of eating remained disturbed. She was not testing her urine but her weight had increased to 49 kg.

(2) This patient developed diabetes when aged 13 years. Her management was complicated by a fear of injections and occasional dietary indiscretions. At 16 she started to diet and over six months her weight fell by 14 kg to 38.2 kg (standard weight 50.3 kg). She reduced her insulin from 92 to 16 units/day in order to accommodate to the low carbohydrate intake. Menstruation ceased. She talked of feelings of intense guilt whenever she had glycosuria. On admission to a psychiatric hospital her weight was 39.4 kg. This increased steadily with a behavioural management programme. After 12 weeks her weight was 48.5 kg. Now, one year after discharge, her weight remains satisfactory, but she is still preoccupied with food and eating and remains amenorrhoeic.

(3) This patient developed diabetes when aged 17 years. She found the dietary restrictions difficult to accept but her management was straightforward. At 23 she narrowly escaped serious injury in a road traffic accident. After this and a broken engagement she began to have difficulty swallowing. Soon she took only fluids, her weight fell from 62 kg to 46 kg (standard weight 64 kg), and she ceased menstruating. Physical investigations were unremarkable and diabetic control remained satisfactory. Reluctantly she accepted admission to a psychiatric hospital. Her difficulty in swallowing rapidly resolved and her weight increased over two months to 49.9 kg. Since discharge her weight has increased and diabetic control remains satisfactory.

Comment

Interestingly, problems with diabetic control secondary to the low carbohydrate intake were infrequent in these patients. None developed hypoglycaemia or ketonuria. Apparently girls with anorexia nervosa can skilfully adjust their insulin dosage to match their greatly reduced carbohydrate consumption. But problems in psychiatric management arise when they use their diabetes as a powerful trump card in any confrontation with the psychiatric team. There are several possible explanations for the apparent rarity of the association. Conceivably the presence of a life-threatening condition may in itself reduce the chance of developing anorexia nervosa or externally imposed carbohydrate restriction lessens the likelihood of self-imposed restriction. Alternatively, the association may be much commoner than has been recognised, with many cases successfully managed by physicians.

We thank Professor R E Kendell and Dr B F Clarke for permission to report cases of patients under their care.

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Axillary hyperhidrosis, 20% aluminium chloride hexahydrate, and surgery

A highly selected group of 38 patients with axillary hyperhidrosis waiting for plastic surgery were treated with 20% aluminium chloride hexahydrate. Twenty-six of them requested surgery by the end of the sixth month.

Patients, methods, and results

Thirty women and eight men whose ages ranged from 18 to 41 years were treated with 20% aluminium chloride hexahydrate and a placebo on a double-blind basis. The solutions were applied on alternate nights. After 14 days whichever solution relieved symptoms was applied to the opposite axilla on alternate nights for 14 days, and when necessary to the axilla where relief had already been obtained. In all other respects the treatment was as outlined by Scholes *et al.*¹ who applied the solution initially for seven successive nights. Two patients defaulted during treatment and were excluded from the study.

Two groups were identified—a stress-positive group of 23 patients with symptoms exacerbated by emotion, and a stress-negative group of nine patients with severe symptoms that stress did not increase. The remainder could not be categorised. After two weeks 24 patients had obtained considerable relief with the active compound. The compound, however, was acceptable to only 19, and the remaining five opted for surgery. Six patients showed no improvement, two patients obtained relief with the placebo, and four obtained equal relief with both solutions. After six months only six out of the 19 patients with an effective initial response had sustained relief, 26 out of the original 38 had opted for surgery, nine required twice-weekly applications to maintain control, and one required an application every fortnight. One of the nine stress-negative patients was controlled with the active solution compared with nine out of 26 for the remainder. Pain and itch (which could not be correlated with the effectiveness of response) were unacceptable in 10 patients.

Comment

Relief of symptoms was excellent in 64 out of 65 of Scholes's patients treated with 20% aluminium chloride hexahydrate.¹ Our different results may be due to the following factors. Our trial was undertaken on all patients simultaneously, which eliminated seasonal variation in severity of symptoms. Our patients showed no significant thermal response (two claimed to be worse in the cold). Our severely affected patients may not have had the usual nocturnal inhibition of secretions, and thus their skin was never dry enough to prevent the formation of an acidic solution causing pain etc. Six patients (17%) showed a significant placebo effect (and this among a severely affected group). Scholes's patients were treated over a 15-month period, possibly for a wider range of severity, and some of them may have been near the time of spontaneous involution. Twenty (55%) of our patients had significant local reactions, which were unrelieved in 15 by hydrocortisone cream. Scholes *et al.* recorded a reaction in 1.5% of cases. We modified the initial "loading" from Scholes's daily application to alternate days because of unfavourable reactions in five patients (not included in the trial). Shelley and Hurley² and Stillians³ obtained effective relief with application on two successive nights and three applications on alternate nights, respectively. This and the initial good response in 19 of our own patients would seem to argue against the differing results being attributable to the different initial loading regimens.

Our results show that there is a group of patients with axillary hyperhidrosis who either cannot tolerate treatment with 20% aluminium chloride hexahydrate or will not respond to it. The results of surgery can be improved by excising the axillary skin with direct closure incorporating a Z-plasty in such a way that the transverse limb of the Z comes to lie in the apex of the axilla. This avoids a linear scar contracture. Relief of the condition more than consoles most patients for the appearance of stretched scars in the axilla.

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Corrections

Vitamin D supplements in pregnant Asian women: effects on calcium status and fetal growth

An error occurred in this paper by Dr O G Brooke and others (15 March, p 751). Table II, row 6 should have read "Heat-labile (non-placental) alkaline phosphatase activity (IU/l)."

Fluid deprivation due to Althesin solution affecting drop size

An error occurred in this article by Dr W J Wraight and Mr D Cox (29 March, p 904). The formula given in the article should have been described as the formula for determining drop volume, not drip rate.