Clinically apparent eating disorders in young diabetic women: associations with painful neuropathy and other complications

J M STEEL, R J YOUNG, G G LLOYD, B F CLARKE

Abstract
Of 208 young women with insulin dependent diabetes, 15 (7%) had a clinically apparent eating disorder (anorexia nervosa or bulimia), a much higher prevalence than reported in non-diabetic women. Most, but not all, of these patients had a long history of poor glycaemic control. In contrast with previous suggestions, control did not deteriorate after the onset of the eating disorder. There was a high incidence and an early onset of diabetic complications. Eleven of the 15 patients had retinopathy, six with proliferative changes; six had nephropathy; and six neuropathy. Most strikingly, four patients with anorexia nervosa developed acute painful polyneuropathy. In each case pain started when the eating disorder developed, almost coinciding with the peak of weight reduction. Remission of pain occurred as weight was regained. The symptoms were accompanied by abnormalities in peripheral nerve electrophysiology and autonomic nerve function, some improvements in which accompanied weight recovery.

It is suggested that nutritional factors may contribute to the high rate of early onset diabetic complications, particularly neuropathy.

Introduction
An association between anorexia nervosa and diabetes in young women was first suggested in 1980, when three cases were reported from our clinic. Since then several further cases of eating disorders in diabetics have been described. It has also become clear that diabetic patients may deliberately omit insulin injections or reduce the dose of insulin to control their weight instead of, or in addition to, the usual artifices used by patients with anorexia nervosa and bulimia. Few systematic studies, however, have attempted to assess the prevalence of eating disorders in diabetics.

The relation between eating disorders and diabetic control is unclear. Our original three patients were skilled in the adjustment of their insulin doses to avoid hypoglycaemia or ketonuria, but several patients have presented serious problems in diabetic management. It has been suggested that anorexia or bulimia may itself lead to poor diabetic control, but the alternative possibility, that eating disorders occur more frequently among diabetics who are already poorly controlled, has not been considered. Diabetic complications have been reported in some patients with eating disorders, but they have not been studied specifically. Nor has the possibility that the eating disorder may contribute independently to the development of complications been considered.

In an attempt to assess the prevalence of eating disorders among diabetic patients more accurately and to examine their relation with diabetic control and complications we identified and reviewed all the cases of clinically apparent eating disorders in a cohort of young women with insulin dependent diabetes.

Patients and methods
Of 208 women with insulin dependent diabetes, aged between 16 and 25, who had regularly attended the diabetic clinic at the Royal Infirmary of Edinburgh for the preceding eight years, 15 were found (by JS or RY) to have clinical evidence of an eating disorder. These were generally patients whose weight loss, as clearly documented in their records, drew attention to their problem, other diagnostic features emerging thereafter during questioning and examination. Only the patients with bulimia alone were discovered to have an eating disorder during inquiry about the cause of their persistent poor control.

Anorexia nervosa is generally thought to comprise three groups of features: (a) behaviour leading to a pronounced loss of weight (for example, 25% of previous weight or 25 lb), such as an intentional avoidance of food or self induced vomiting, or excess exercise, or all three; (b) amenorrhoea and lanugo hair growth; and (c) characteristic psychopathology, including a morbid fear of becoming fat and a distorted body image. Ten of our patients fulfilled all of these criteria, and two further patients had all the features of...
anorexia nervosa except that their weight loss did not quite amount to 25% of their previous weight or 25 lb. Two patients fulfilled the criteria for bulimia, alone and eight of those who were anorectic also admitted episodes of binge eating and vomiting. One further patient reported a morbid fear of becoming obese and binge eating with weight control achieved purely by reducing her dose of insulin; she was therefore considered to have a variant of bulimia nervosa.

In these fifteen patients glycaemic control had been assessed by measurement of glycosylated haemoglobin concentration at regular intervals (reference range 5-5.8%). All episodes of severe hyperglycaemia and ketoacidosis had been documented. Retinopathy was graded: 1=no disturbances, 2=microaneuysms and dot haemorrhages only, 3=exudates, cotton wool spots, or intraretinal microvascular abnormalities, and 4=neovascularisation. Nephropathy was classified: 1=no disturbances, 2=persistent albuminuria, 3=creatinine clearance less than 50 ml/min, and 4=requiring dialysis. Polyneuropathy was classified as acute painful neuropathy that remitted within 12 months; acute painful continuing neuropathy; chronic painful neuropathy; and painless neuropathy with reduced temperature and light touch sensation, absent tendon jerks, and neuropathic ulceration. Symptomatic autonomic neuropathy was also recorded. In the four patients with acute painful neuropathy peripheral nerve electrophysiology and cardiovascular autonomic nerve function were measured at presentation and at six monthly intervals for up to 18 months:11

All 15 patients were offered psychiatric treatment for their eating disorder. Five refused, and two patients defaulted after one interview. Five received psychiatric treatment as inpatients and four as outpatients. The others were managed as outpatients in the diabetic clinic. The outcome of treatment of the eating disorder was classified: 1=complete recovery, weight within normal range, and normal attitude to eating and body image; 2=weight normal but attitude to eating still disturbed; 3=continuing problem.

Results

Table I gives the clinical details of the 15 patients with respect to age at onset, duration of diabetes, glycaemic control, complications, treatment, and outcome.

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<tr>
<th>Case No</th>
<th>Age at onset of diabetes (yrs)</th>
<th>Age at onset of eating disorder (yrs)</th>
<th>Duration of diabetes (yrs)</th>
<th>Criteria for anorexia nervosa</th>
<th>Binging and vomiting</th>
<th>Reducing insulin to Glycosylated haemoglobin %</th>
<th>Severe hypoglycaemia</th>
<th>Retinopathy grade</th>
<th>Duration of diabetes when patient developed anorexia nervosa</th>
<th>Sympathetic peripheral neuropathy</th>
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<th>Nephropathy grade</th>
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*Refused psychiatric treatment.†Defaulted from psychiatric treatment after one visit.

TABLE I—Clinical details of 15 young diabetic women who developed eating disorders
TABLE II—Results of neurophysiological studies in four young insulin-dependent diabetic women with anorexia nervosa who developed acute painful diabetic polyneuropathy at their lowest weight

<table>
<thead>
<tr>
<th>Case No</th>
<th>Time after presentation (months)</th>
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Mean (SD) non-diabetic normal values:

- Peripheral nerve conduction velocity: 51-1 (3-1) m/s
- Sural sensory nerve conduction velocity: 7-1 (3-9) m/s
- Sural sensory nerve conduction velocity: 37-0 (3-7) m/s
- Valueto ratio: 11-0 (5-1) m/s
- Immediate heart rate response to standing: 1-43 (0-37) beats/min
- Maximum-minimum heart rate: 1-39 (0-17) beats/min
- Postural blood pressure drop: 34-2 (8-1) mm Hg

PREVALENCE OF EATING DISORDERS

Anorexia nervosa occurred in 5-8% of the patients (66% of the anorectic patients also binged and vomited), while bulimia alone occurred in 1-4% (this includes the patient who controlled her weight solely by reducing insulin). The two patients who had not lost 25% of their original weight or 5 lb were included as some definitions of anorexia nervosa do not stipulate any loss and many workers believe that the psychopathology of anorexia nervosa is more important than the amount of weight loss.10-12

CONTROL

As judged by measurement of glycosylated haemoglobin concentration, 11 of the 15 patients were classified as poorly controlled and four (cases 1, 3, 13, 14) as moderately controlled. It was striking that the poor control among the 11 patients was not confined to, or even intensified by, the duration of their eating disorders. Hyperglycaemia was equally bad before and after the eating disorder, as indicated by the range of haemoglobin concentrations (table I). Seven of the 11 poorly controlled patients had even avoided severe hypoglycaemia or ketoacidosis completely, which perhaps reflects our encouragement of self-regulated insulin dosage. Three of the four patients with a history of recurrent ketoacidosis showed no increase in frequency of episodes during their eating disorder, while the fourth (case 11) had been completely free of ketoacidosis or hospital admission only during the four years when she had anorexia nervosa.

The severity of the poor control is evident when these patients are compared with a representative group of 80 young patients with insulin dependent diabetes from our clinic, who are being followed up prospectively.11 In these patients the mean glycosylated haemoglobin concentration over three years was higher than our clinic mean for insulin dependent diabetic patients (11%) at 12% (range 7-4%—19-5%) but appreciably less than that for the 14 patients with eating disorders for whom we had complete records, whose mean concentration was 14-8% (range 11%-18%).

COMPICATIONS

Eleven patients had developed retinopathy, which was proliferative in six cases. Four of the patients with proliferative retinopathy had developed it during their eating disorder and two after they had recovered. Six had nephropathy, as judged by persistent proteinuria; two of these had renal hypertension, and one required dialysis a year after she had recovered from her eating disorder. Four patients developed acute painful neuropathy; the figure shows the relation between change in weight and the onset or remission of symptoms. Table II shows the results of the sequential somatic and autonomic nerve function tests (for one patient (case 10) nerve function had been measured previously and was included for comparison). Some recovery of nerve function, particularly of autonomic nerve function, was seen to accompany weight gain and remission of symptoms.

Discussion

Crisp et al found the prevalence of anorexia nervosa to be 0-4% in all adolescent girls but as high as 2% in girls attending independent schools. The prevalence of bulimia in young women is thought to be 1-2%. In our study the overall prevalence of eating disorders was 7-2%. As patients with eating disorders are notoriously secretive some cases of bulimia and weight control by insulin reduction were probably not identified. By contrast, diabetic patients with overt anorexia nervosa are unlikely to be missed because of routine clinic weight checks. Nevertheless, one of our patients delayed detection by filling her pockets with heavy objects and insisting that she was menstruating when she later admitted that she had had amenorrhoea. Given that we have identified only clinically apparent cases, our findings are consistent with those of Rodin et al. They assessed, by the more sensitive questionnaire technique, a much smaller group of 46 selected female diabetic patients aged 15-22 and found that 19% had anorexia nervosa or bulimia or a partial syndrome. Hudson et al reported that 35% of young female diabetics answering a questionnaire had bulimia, but unfortunately only 31% responded to their survey.12

Diabetes is a disorder that inevitably focuses attention on body weight and diet. As it may cause conflicts about autonomy and dependence, reduce self esteem, and produce stress within the family it is perhaps not surprising that diabetes is associated with an increased incidence of eating disorders. Several of our patients strongly believed that they had developed their eating disorders because they were diabetic and "had to think all the time about food." Others associated the administration of insulin with weight gain and believed that this had precipitated their eating disorder.

CONTROL

There is no clear classification of metabolic control in diabetes, but it is now obvious that patients who avoid severe hypoglycaemia and ketoacidosis are not necessarily well controlled. Indeed, they may have chronic severe hyperglycaemia. Eleven of our patients had poor control, thus defined, before, during, and after their eating disorders.

The metabolic control of those who admitted reducing their insulin dose to lose weight might have been expected to deteriorate. That it did not may be due to the fact that four had previously manipulated their insulin dose and had poor control, as reported in other groups of adolescent diabetic women,11 and all the others had also given a low dose of insulin at times past either to avoid hypoglycaemia or because they omitted their evening injection.

Our findings suggest that poorly controlled young diabetic women are at increased risk of developing an eating disorder and that the eating disorder in itself does not usually cause poor control. This contrasts with the conclusions of Hillard and Hillard, who were perhaps not judging patients in the context of lifelong diabetes when they concluded that eating disorders were the cause of poor diabetic control.15 Nevertheless, our study does not indicate to what extent clinically inapparent eating disorders might be associated...
with poor diabetic control in adolescence and early adulthood, and such information might shed a different light on the relation between eating disorders and diabetic control. We are currently engaged in a study using eating disorder scales.

**Complications**

Eleven of the patients had retinopathy, six had proliferative changes, and one was blind. Retinopathy in this age group is related to both duration of diabetes and glycaemic control, but the prevalence of visually threatening retinopathy among these patients with eating disorders was very high. Five of the six with proliferative retinopathy had been poorly controlled. We do not know if their eating disorders might be implicated in the pathogenesis, but it is striking that in four (cases 5, 7, 10, and 11) proliferative retinopathy developed when the patients were suffering from anorexia nervosa. The duration of diabetes at that stage was 12 years or less.

Acute painful polyneuropathy occurred in four of the patients with anorexia nervosa. No other young female patients in our clinic have developed painful neuropathy. In each case the pain started after the initial anorectic weight loss (15-60 months) and usually coincided with the peak of their weight reduction. All patients were treated with a stepwise programme of analgesia based on imipramine. Three (cases 7, 9, and 10) steadily regained weight during treatment, and their symptoms remitted so that drug treatment was no longer required. One patient (case 5) did not gain weight and continued to suffer severe pain 12 months after its presentation. The figure shows the relation between the onset of pain and weight improvement. In nerve function accompanying the symptomatic improvement was slightly more noticeable in cardiovascular autonomic function than in peripheral nerve electrophysiology, as described previously. None of the patients showed any improvement in glycosylated haemoglobin concentration during the acute neuropathy. Two further patients had chronic neuropathy (cases 2 and 11), which also presented while they were suffering from anorexia. Gastric autonomic neuropathy did not seem to be the cause as symptoms developed after the onset of the eating disorder, though two of the six patients with autonomic symptoms may have vomited more as a result of gastroparis. Alloway et al described two non-diabetic patients with anorexia nervosa who developed peripheral neuropathy and proximal myopathy; they postulated that the neurological disturbances were caused by protein and carbohydrate deprivation. Nutritional factors, not clearly identified, may also contribute to the development of metabolic neuronal complications in diabetic patients.

It is particularly noteworthy that only four of the 15 patients with eating disorders were free of clinical diabetic complications. Two of these four had not had diabetes for long enough (one and six years) for clinical complications to become apparent. Our observations therefore suggest an association between eating disorders and early onset severe diabetic complications in young diabetic women.

**References**


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