Review Article

Eating Disorders and Insulin-Dependent Diabetes Mellitus

SCOTT J. CROW, M.D.
PAMELA K. KEEL, A.B.
DAVID KENDALL, M.D.

The eating disorders anorexia nervosa and bulimia nervosa have been reported to occur in Type I diabetes mellitus. Although prevalence estimates vary, the most rigorous studies yield rates similar to the population at large. Intentional insulin omission is more common, especially in young diabetic women, and at times may indicate an eating disorder in Type I diabetic patients. Both diagnosable eating disorders and intentional insulin omission are associated with worse glycemic control and higher rates of secondary diabetic complications. Recognition of these conditions, followed by carefully coordinated treatment involving both diabetes care providers and mental health providers, is necessary to improve treatment outcome. (Psychosomatics 1998; 39:233–243)

Type I or insulin-dependent diabetes mellitus (IDDM) is an endocrine disorder characterized by absolute lack of insulin production due to autoimmune destruction of the pancreatic β cell. IDDM is generally diagnosed in childhood, with a prevalence rate of about 0.1% in the population. IDDM requires the use of exogenous insulin, and management of the disease is facilitated by blood-sugar monitoring, specific dietary constraints, and regular exercise. Chronic hyperglycemia in IDDM can result in a number of secondary complications, including peripheral neuropathy, autonomic neuropathy, retinopathy, and nephropathy; it is now clear that improved diabetic control can prevent the onset of, and halt the progression of, these complications.[1]

The relationship between IDDM and eating disorders has been widely studied following initial reports of an association between anorexia nervosa, bulimia nervosa, and IDDM.[2–4] Past reviews have highlighted the early studies suggesting high rates of eating disorders in young diabetic women.[5–7] Recent studies using more rigorous methods have found lower rates of traditional eating disorders. However, there has recently been mounting evidence for the adverse impact of eating-disordered behaviors such as insulin omission, as well as eating disorders, on the treatment and outcome of IDDM. We review the existing literature on eating-disordered behaviors and diagnosable eating disorders associated with IDDM. We will also examine recent studies on the impact of eating-disordered behaviors on diabetic control and the development of complications from IDDM. Finally, we will briefly examine potential treatment strategies and outline recommendations for diagnosis and management.

Received April 23, 1997; revised August 6, 1997; accepted August 19, 1997. From the Departments of Psychiatry, Psychology, and Medicine, University of Minnesota, Minneapolis. Address reprint requests to Dr. Crow, Department of Psychiatry, University of Minnesota, Box 393 UMHC, Minneapolis, MN 55455.

INTENTIONAL INSULIN OMISSION

A major symptom in persons with bulmia nervosa (and some patients with anorexia nervosa as well) is the use of behaviors such as vomiting to try to compensate for ingested calories. One form of these behaviors unique to those with IDDM is the intentional omission of insulin. If a meal is eaten and inadequate insulin is injected for the calories ingested, blood glucose may increase markedly. As a consequence, glycosuria occurs and the volume of urine produced increases. The end result is the excretion of large amounts of glucose (and thus calories) in the urine, resulting in short-term reduction in weight from fluid loss and some caloric restriction.

Several investigators have examined the frequency with which insulin-dependent diabetic persons report intentional omission of insulin to counteract food eaten or to lose weight. The results of these studies are summarized in Table 1.[3,8–16] As these data suggest, the frequency of insulin omission reported by subjects varies rather widely but tends to be higher than the rate of diagnosable eating disorders in IDDM, with up to one-third of subjects reporting this behavior at some time. When comparison data are available, rates of insulin omission are higher in female than male subjects. This picture mirrors that seen regarding bulimic behaviors in the general population, in which the rate of experimentation with bulimic behaviors far exceeds the rate of diagnosable bulimia nervosa in the young-adult female population.[17,18] Similarly, it appears that insulin omission as a weight-control strategy is far more common among insulin-dependent diabetic persons than is the development of full eating disorder syndromes. It is important to note that, due to the resulting hyperglycemia, the health risks of this behavior may be greater than those associated with bulimic experimentation seen in nondiabetic young women.

In a recent review of adherence to self-care behaviors among persons with both Type I and Type II diabetes, Robiner and Keel[19] reported significant difficulties with adherence to the diabetic regimen. However, compliance with insulin recommendations tended to be higher than any other area of self-care. Indeed, across the 13 studies the researchers reviewed, 11 reported predominantly good-to-excellent adherence with various aspects such as timing, dose, and number of insulin injections. While intentional insulin omission is a fairly frequent phenomenon in the broad population of diabetic persons, Rodin et al.[10] suggested omission was far more common in those persons with a diagnosable eating disorder (54% vs. 12%). In addition, the observed gender differences in intentional insulin omission support the notion that this behavior is often eating disorder–related rather

Table 1. Intentional insulin omission

Authors	Year	*N*	Rate of Omission, %	Response Rate, %
Hudson et al.[3]	1985	80	14.0	30
Birk and Spencer[8]	1987	385	4.9	70
Powers et al.[9]	1990	97	14.0 female 4.0 male	NR
Rodin et al.[10]	1991	103	12.0	85
Fairburn et al.[11]	1991	100	37.0 female 0 male	90
Striegel-Moore et al.[12]	1992	46	6.5	94
Marcus et al.[16]	1992	153	21.6	80.4
Peveler et al.[13]	1992	76	15.0 female 0 male	88
Biggs et al.[14]	1994	42	37.5	60
Polonsky et al.[15]	1994	341	31.0	91

Note: NR = not reported.

than a reflection of general problems with compliance.[9,10] Given these data, one might hypothesize that insulin omission, when present, may serve as a useful marker of elevated risk for an eating disorder.

The wide variability in rates of insulin omission across studies may reflect a number of methodologic issues, including the survey of different age groups; different clinical populations; and different assessment methods (i.e., interview vs. questionnaire). For example, tertiary-care referral-center populations may be particularly difficult to treat when compared with community-based diabetes-clinic populations, relating perhaps in part to higher rates of intentional insulin omission. In addition, confidentiality may be a concern for subjects, as a substantial part of diabetes treatment involves the examination of glucose monitoring and insulin-administration records; because many patients understand that this self-reporting is an important focus of the treatment, it may be difficult to obtain accurate patient reports of insulin omission. This might be particularly true if subjects are concerned about their diabetologist learning of the insulin omission. Thus, reported rates of insulin omission may be underestimates.

RATES OF EATING DISORDERS IN IDDM

At least 19 studies have examined the frequency of eating-disordered behaviors or diagnosable eating disorders in IDDM, and these data are summarized in Table 2.[3,4,11,13,14,20–30] A variety of instruments have been used to assess the presence of eating disorders, partially explaining the wide range of reported rates. Rating scales such as the Eating Disorders Inventory (EDI) or Eating Attitudes Test (EAT) tend to provide higher estimates of rates of eating pathology. These scales measure the presence or absence of pathological eating attitudes and behaviors but not their duration, and these scales do not elicit specific criteria needed to make formal eating disorder diagnoses. In addition, rating scales for eating disorders often incorporate items measuring dietary concern—which is, to some extent, prescribed as a part of diabetic management and which one might thus expect to be elevated in IDDM patients. Interview-based instruments yielding criterion-based diagnoses such as the Eating Disorders Examination (EDE) or the Structured Clinical Interview for DSM-III-R (SCID) yield lower estimates of rates of eating disorders and are generally regarded as the optimal instruments for making diagnoses. This was demonstrated by one study in which EAT scores were higher in the diabetic women than the control subjects, but rates of eating disorder diagnoses using structured interviews were equivalent.[11] However, important dimensional information about eating behaviors that are pathological but do not fit a specific diagnostic category may be missed on the SCID (in contrast to rating scales). Seven of the 19 studies reviewed here have included both men and women, usually in roughly equal proportion; the remainder have studied women only. These studies have involved adolescents or young adults almost entirely. For most of the studies, the response rates have been above 70%, with only a few exceptions.[3,14,26,30]

BULIMIA NERVOSA

Many of the studies have found rates of bulimia nervosa (BN) in IDDM patients that appear to be higher than population base rates.[3,4,8–10,16,19,20,26] In these studies, BN rates ranging from 1.4% to 35% have been reported. Notably, the response rate reported by Hudson et al.[3] was quite low (30%), which may have resulted in significantly biased sample selection, perhaps leading to higher reported rates of BN. Other studies have not confirmed these elevated rates. Fairburn and colleagues in 1991[11] found that rates of bulimic behaviors were not higher in a group of adult Type I diabetic persons than in an age-matched control group; similarly, Marcus et al. found comparable rates of eating-disordered attitudes and behaviors between diabetics and a comparison group.[16] Also, Striegel-Moore et al.,[12] Powers et al.,[9] and Peveler et al.[13] found similar rates of bulimic

Table 2. Rates of eating disorders in diabetes mellitus

Author	Year	*N*	Average Age	%M %F	Measures	Results, % AN	BN	ED NOS	Response Rate, %	Criteria
Rodin et al.[4]	1985	46	17.2	0 100	EAT-26 EDI	6.5	6.5	6.5	NR	DSM-III
Hudson et al.[3]	1985	80	18.4	0 100	BSQ	0	35	3	30	DSM-III
Rodin et al.[20]	1986	58	17.6	0 100	EAT-26 EDI	6.9	6.9	6.9	93.6	DSM-III
Rosmark et al.[21]	1986	86	28.3	52 48	EAT-36	3.5% cutoff, F>M on EAT, $P<0.05$ (9% in female sample)			84	EAT
Birk and Spencer[8]	1987	385	28.2	0 100	Mail survey Pyle	1.0	16.2	2.1	70	DSM-III
Steel et al.[23]	1987	208	16–25	0 100	Medical records	4.8	1.4	0.5	NR	Russell
Popkin et al.[24]	1988	75	31	36 64	DIS	NR	2.4	NR	100	DIS
Steel et al.[25]	1989	273	22.0	46 54	EAT-40 EDI GHQ	20% of females, EAT >18; 5% of DMM males			97 F 91 M	EAT>18
Powers et al.[9]	1990	97	15.6	53 47	GHQ	F:0 2.2 NR M:0 0 NR	NR	DSM-III DSM-III-R		
Rodin et al.[10]	1991	103	15.1	0 100	DSED	0	5	NR		
Fairburn et al.[11]	1991	100 (67F cont)	21.5	54 46	EDE EAT	DMF: 0 5.6 5.6 DMM: 0 0 0 Control F: 0 3.0 4.5			90	EDE
Peveler et al.[13]	1992	76	15.3	0 100	EDE	—	—	9 (control) 6	88	EDE
Biggs et al.[14]	1994	42	NR	0 100	EDI-2 DICA SCID	IW: 33 AN NR 27 BN current 53 BN past Non-IW: 0 AN NR 0 BN (7.4 post)			60	DSM-III-R
Pollock et al.[27]	1995	79	20.6	44 56	ISC	0	1.3	2.5	75	DSM-III

Herpertz et al.[28]	1996	246	NR	NR	EDI BSQ SIAB	0	5.9	5.0	88	DSM-IV
Vila et al.[29]	1996	69	26.7	49 51	EAT BITE	0 M 0 F	0 5	5 38	100	DSM-III-R
Cantwell and Steel[30]	1996	147	NR	0 100	EAT-40 semistructured interview	0 current 8.3 past	10.4 current	6.3 current	68.4	EAT-40 >18; DSM-III-R

Note: AN = Anorexia Nervosa; BES = Binge Eating Scale; BITE = Bulimic Investigatory Test; BN = Bulimia Nervosa; BSQ = Body Shape Questionnaire; BULIT = Bulimia Test; DICA = Diagnostic Interview for Children and Adolescents; DIS = Diagnostic Interview Schedule: DMF = Diabetes Mellitus Female; DMM = Diabetes Mellitus Male; DQOL = Diabetes Quality of Life; DSED = Diagnostic Survey for Eating Disorders; EAT = Eating Attitudes Test; EDE = Eating Disorders Examination; EDNOS = Eating Disorder Not Otherwise Specified; F = Female; GHQ = General Health Questionnaire; ISC = Interview Schedule for Children and Adolescents; IW = Insulin Withholding; M = Male; NR = Not Reported; Pyle = Pyle Eating Behavior Survey; Russell = Criteria set forth by Russell, 1979; SCID = Structured Clinical Interview for DSM-III-R; SCL-90 = Symptom Checklist 90.

behaviors in their studies of adolescent diabetic subjects vs. control subjects.

While these more recent studies do suggest strongly that BN rates are not elevated in IDDM, it may be that some cases of eventual BN were missed by some studies. Three of these studies[9,12,13] used subjects with average ages of about 15, about 3 years younger than the average age at onset for BN. It could be that some of the subjects in those studies went on to develop an eating disorder later, resulting in artifically low prevalence rates for BN. Also, while sample sizes have generally been adequate, many of the higher prevalence rates have come in studies using larger subject groups; conversely, some (but not all) of the studies finding no differences have had smaller subject groups (and thus less power to detect those differences).

ANOREXIA NERVOSA

While several studies have found rates of anorexia nervosa (AN) in patients with IDDM that are above population base rates,[4,20,22,23], [30] this relationship does not appear to be as strong as in BN. The majority of studies examining AN in insulin-dependent diabetic populations have found rates less than or equal to population norms or control groups.

BINGE EATING DISORDER

Only one study to date has assessed the frequency of Binge Eating Disorder in IDDM. Herpertz et al. found a rate of 4.2%[28]; while no comparison group was used, population-based surveys have found rates of 1%–2%.[31,32]

EATING DISORDER NOT OTHERWISE SPECIFIED (EDNOS)

EDNOS represents a major diagnostic challenge in the diabetic patient. DSM-IV[33] defines EDNOS as "disorders of eating that do not meet the criteria for any specified eating disorder." Many studies reporting diagnostic rates for AN and BN also provide rates for EDNOS.[3,4,8,11,13,20,23,27,30] Four of these studies also present rates of inten-

tional insulin omission[3,8,11,13]; in each instance, omission rates are substantially higher than EDNOS rates.

For some diabetic persons with BN, insulin omission coexists with other methods of purging or represents the sole method of purging. However, based on the DSM-IV criteria for EDNOS, it could be argued that many insulin-omitting subjects actually meet criteria for that diagnosis. Potentially, EDNOS could be the most common eating disorder found in diabetic patients.

LONGITUDINAL COURSE OF EATING DISORDER IN IDDM

Little is known about the long-term course of eating disorders and eating pathology in IDDM. One study has examined this population over a follow-up period of 4 years.[34] During the follow-up interval, a variety of pathological behaviors became more frequent, including insulin omission, dieting, and self-induced vomiting.

MORBIDITY ASSOCIATED WITH EATING-DISORDERED BEHAVIORS IN IDDM

AN and BN are associated with a wide variety of medical complications.[35] Diabetic patients with intentional insulin omission or eating disorders may be at higher risk for complications from their IDDM, due to the potential negative impact of the eating-disordered behaviors on glycemic control. Long-term diabetic control is easily assessed with the use of hemoglobin A_{1c} (glycosylated hemoglobin) measurements. Given a typical life of a red blood cell (120 days), the degree of glycosylation of the hemoglobin contained in the red cells represents an integrated assessment of glycemic control over a 4-month period. There is a strong relationship between glycemic control (as measured by HbA_{1c}) and the development of complications in diabetes mellitus. This relationship was demonstrated in a multicenter prospective study examining the effects of routine diabetic care vs. more vigorous efforts to obtain tight diabetic control.[1]

Three groups have examined the relationship between insulin omission and glycemic control, as measured by glycosylated hemoglobin (HbA_{1c}).[14,15,36] In two of the studies, glycosylated hemoglobin levels were significantly higher in the insulin-omitting subjects. Biggs et al.[14] found substantial differences in HbA_{1c} between the insulin-omitting and non–insulin-omitting subjects (15.3 vs. 10.3, $P<0.01$). La Greca and colleagues[36] found that 0% of diabetic persons with $HbA_{1c}<9.0$, 50% of those with HbA_{1c} between 9.0 and 11.9, and 70% of those with $HbA_{1c} \geq 12.0$ reported intentional insulin omission. In the study by Polonsky and coworkers,[15] insulin omission and hyperglycemia led to higher rates of diabetic complications. The insulin-omitting subjects had significantly higher rates of retinopathy, neuropathy, and hospitalization than the nonomitting subjects. Thus, persons with eating disorders who use insulin omission as a form of purging risk complications that may not be normally encountered in the general eating-disordered population.

Numerous authors have examined the relationship between eating disorder rating-scale scores and HbA_{1c}. Rodin et al.,[4] using the EAT-26, and Wing and colleague,[37] using the Binge Eating Scale, found significant correlations between pathological scores on measures of binge behavior and elevated HbA_{1c} levels, whereas Cantwell and Steel[30] did not. Similarly, several studies have compared glycosylated hemoglobin levels in eating-disordered vs. non–eating-disordered Type I diabetic persons, as summarized in Table 3.[8,10,11,20,36,38,39] In each of these studies, the eating-disordered groups have had higher glycosylated hemoglobin levels than the control groups. Lorini et al.[39] found considerably higher glycosylated hemoglobin in a binge-eating group than in the restricting anorexic group (9.33 vs. 7.02). It is of note that this study used an annualized mean of several glycosylated hemoglobin levels intended to reflect an even longer term measure of glycemic control. As previously noted, insulin omission by itself has been associated with elevated glycosylated hemoglobin levels.[9,25] Since this behavior appears more common in the eating-disordered subgroup

of IDDM patients, it may account for much of the difference between the groups. Alternatively, binge-eating behavior may itself result in higher glucose levels postbinge, leading to the increase in HbA_{1c}. The clinical significance of these differences remains the same regardless of their behavioral source.

Numerous studies have demonstrated a relationship between the co-occurrence of an eating disorder and diabetes mellitus and elevated risk for medical complications of either the eating disorder or diabetes. In their study, Carney and co-workers found greater diminishment in bone mineral density (a common complication of ED) in the eating-disordered patients with diabetes mellitus than in those without diabetes mellitus (0.76 vs. 0.96, $P = 0.025$).[40] Steel et al.[23] found painful peripheral neuropathy in 37.5% of the eating-disordered diabetic persons (vs. 0% in the non–eating-disordered subjects).

Four studies have specifically examined the rates of retinopathy in young women with Type I diabetes mellitus, with and without eating disorders. Colas et al.[38] found that the non–eating-disordered subjects were considerably more likely to be free of any retinopathy (79% vs. 38%, $P<0.001$), whereas the women with disordered eating were more likely to have retinal microaneurysms (41% vs. 7%, $P<0.001$). in reports by Steel et al.[25] and Cantwell and Steel,[30] retinopathy was more frequent in groups with high EAT-40 scores than in the low-scoring control groups (for example, 45.5% vs. 15.4%, $P<0.0530$). Rydall and colleagues showed much higher rates of retinopathy over 4.4-year follow-up in the eating-disordered vs. non–eating-disordered patients (86% vs. 24%, $P<0.004$).[34] Microalbuminuria, on the other hand, did not differ in frequency between the diabetic persons with and without an eating disorder in the same study.

One important factor in the interpretation of these data is the relative lack of information on psychiatric comorbidity in these subjects. It seems likely that eating pathology with resulting hyperglycemia is the cause of these elevated complication rates. However, psychiatric illness in general has been recently reported as a risk factor for diabetic retinopathy[41]; therefore, one cannot rule out psychiatric comorbidity as a mediator of at least some of this effect.

TREATMENT

Education is a frequent focus in treating an eating disorder in a diabetic patient. The risks of diabetic complications and the importance of dietary management are repeatedly stressed. In our experience, education about dietary management is helpful for these persons; however, the common assumption that there is a knowledge deficit about the risk of diabetic complications is often inaccurate. Patients are commonly very well informed about these risks, and further education in this regard may not be beneficial. Selected patients may benefit from knowledge about the added complication risks from eating-disordered behaviors when present concurrently with IDDM. However, overemphasis on dire outcomes can overwhelm the patient, leading to

Table 3. Glycosylated hemoglobin in eating-disordered (ED) and non–eating-disordered (non-ED) diabetic subjects

Authors	Year	*N*	ED	non-ED	*P*
Rodin et al.[20]	1986	58	12.1	11.5	NS
Birk and Spencer[8]	1987	385	8.49	6.96	= 0.01
Rodin et al.[10]	1991	103	10.0	8.9	< 0.02
Fairburn et al.[11]	1991	100	13.3	11.1	= 0.02
Colas et al.[38]	1991	58	10.8	8.1	< 0.0001
Lorini et al.[39]	1993	8	7.02 AN 9.33 BE	NR	< 0.05

Note: AN = anorexia nervosa; BE = binge eating; NR = not reported.

increased distress and increased use of disordered eating as a coping mechanism. Educational efforts may be more fruitful when directed toward themes typically addressed in eating disorder treatment. These include information on societal attitudes about shape and weight; the role of dietary restriction as a precipitant of binge eating; and the biological determinants of body weight (i.e., "Set-Point Theory").[42]

For the bulimic patient, resumption of a consistent pattern of meals and snacks is important to avoid the cycle of dietary restraint followed by binge eating. Standard approaches to treatment include structured psychotherapies such as cognitive-behavioral therapy[43] or interpersonal therapy,[44] as well as the use of antidepressants.[45] All of these treatments are effective, although the structured psychotherapies, when available, appear to yield somewhat better results. Of note, the antidepressants are effective for BN, even in the absence of depression.

The treatment of AN is less well established. Weight restoration through the resumption of adequate caloric intake is a central aspect of treatment. Psychotherapeutic strategies may be effective, although empirical data are limited at present. Family therapy may be a particularly effective strategy in the treatment of AN, especially for younger persons with AN who still live with their parents.[46] The role of pharmacologic treatments is less clear. Many agents have been tried, with limited success, but selective serotonin reuptake inhibitors have shown some promise and are under further study.[47]

The treatment of an eating disorder is usually best delivered through the coordinated efforts of the diabetes care provider and a mental health professional. This approach is essential for the eating-disordered diabetic patient. Frequent communication between professionals (and family too, for a minor) is needed; furthermore, familiarity with both eating disorders and diabetes mellitus is necessary for all professionals involved. This frequently requires some degree of education, both for mental health professionals and for primary care physicians or diabetologists. It is also helpful to provide parents with guidance about when and how to intervene in the patient's routine of eating and diabetic care.

A careful assessment for psychiatric comorbidity is an important aspect of the initial evaluation. Rates of depression and anxiety disorders are higher in eating-disordered persons than in the general population[48–50]; similar elevated rates of depression and anxiety disorders have been described in IDDM.[24,51] The impact of combining these two risk factors has yet to be determined but might contribute to still higher risk for anxiety and mood syndromes.

A final, difficult issue involves the extent to which interventions important to diabetic care potentially contradict treatment strategies effective in treating eating disorders. For example, should treatment of the diabetic, eating-disordered patient emphasize dietary restraint and/or weight loss? Dietary management is a fundamental component of diabetic management. However, it has long been recognized that dietary restraint—and subsequent dietary disinhibition when restraint fails—may be an integral part of the pathophysiology of BN.[52] Extreme dietary restraint is also a core feature of AN. Steel and colleagues[53] have shown that rating scale measures of dieting behavior, focusing on thinness and body dissatisfaction, all become more pathological over the first year following diagnosis of IDDM. It may be that adolescents with diabetes are instructed about dietary management and dietary restraint for their illness at a time when they are most susceptible to environmental messages about thinness and at highest risk for developing an eating disorder. In addition, institution of insulin therapy for newly diagnosed IDDM is associated with weight gain, as glycosuria ends and fluid volume is replenished. For adolescent girls, this may be a particularly difficult stressor.

Thus, the risks of prescribing significant dietary restriction for insulin-dependent diabetic persons, particularly in adolescent girls and young adult women, must be balanced against the potential for these dietary measures to improve diabetic control. Other forms of diabetes management such as exercise might pose much less risk, and weight-loss strategies might best

be reserved for substantially obese patients within this group. Coordinated efforts by the diabetologist and mental health provider are required to obtain optimal diabetic control while minimizing the risk of further disordered eating.

CONCLUSIONS

There are conflicting data about the frequency of eating disorders in IDDM. A few studies have found elevated rates of AN in study groups; more have found elevated rates of BN. However, this has not been an entirely consistent finding, and more recent studies that attempt to correct for the presence of the diabetic dietary regimen and avoid other confounds suggest that rates may not be elevated in comparison with nondiabetic control subjects. A careful examination of this literature reveals that those studies using appropriate control groups appear to be less likely to find elevated rates. Also, the use of more strict diagnostic criteria such as DSM-III-R (vs. DSM-III) criteria yields lower rates of diagnosis. Finally, the use of more rigorously structured interviews or criterion-based diagnostic instruments has yielded lower rates of eating disorder diagnosis than found with rating scales and cutoff scores. If there is an increased risk for formal eating disorders in IDDM, that relationship appears weaker than originally believed.

Much more convincing and quite alarming are the high rates of insulin omission in Type I diabetic persons for weight-control purposes and the finding of the poor degree of glycemic control obtained in those subjects with eating-disordered behaviors. Those studies examining intentional insulin omission have been virtually unanimous in finding it to be a common behavior in young diabetic women, although the duration of insulin omission may vary greatly. For many of these persons, insulin omission might be best classified as EDNOS. Because of the high frequency of insulin omission and its association with impaired glycemic control and diabetic complications, most of the morbidity associated with eating disorders in diabetic persons may relate to this behavior.

Integrated treatment approaches that combine traditional eating disorders treatments with diabetes care adapted to the issues unique to an eating diosrder in IDDM are recommended. Such treatments are best delivered in close collaboration between mental health and diabetes care providers familiar with both eating disorders and IDDM.

This paper was supported in part by a grant from the McKnight Foundation and by a National Institutes of Health Obesity Center pilot and feasibility grant (Grant No. DK50456).

References

1. DCCT Research Group: The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. N Engl J Med 1993; 329:977–986
2. Szmukler GI: Anorexia nervosa and bulimia in diabetics. J Psychosom Res 1984; 28:365–369
3. Hudson JI, Wentworth SM, Hudson MS, et al: Prevalence of anorexia nervosa and bulimia among young diabetic women. J Clin Psychiatry 1985; 46:88–89
4. Rodin GM, Daneman D, Johnson LE, et al: Anorexia nervosa and bulimia in female adolescents with insulin-dependent diabetes mellitus: a systematic study. J Psychiatr Res 1985; 19:381–384
5. Popkin MK: Type-1 diabetes mellitus and eating disorders: navigating nearly uncharted waters. Psychiatric Medicine 1989; 7:205–210
6. Rodin GM, Daneman D: Eating disorders and IDDM. Diabetes Care 1992; 15:1402–1412
7. Hall RCW: Bulimia nervosa and diabetes mellitus: a dangerous interplay producing accelerated complications. Seminars in Clinical Neuropsychiatry 1997; 2:24–30
8. Birk R, Spencer ML: The prevalence of anorexia nervosa, bulimia, and induced glycosuria in IDDM females. The Diabetes Educator 1987; 15:336–341
9. Powers PS, Malone JI, Coovert DL, et al: Insulin-dependent diabetes mellitus and eating disorders: a prevalence study. Compr Psychiatry 1990; 31:205–210
10. Rodin G, Craven J, Littlefield C, et al: Eating disorders and intentional insulin undertreatment in adolescent females with diabetes. Psychosomatics 1991; 32:171–176
11. Fairburn CG, Peveler RC, Davies B, et al: Eating disorders in young adults with insulin-dependent diabetes mellitus: a controlled study. BMJ 1991; 303:17–20

12. Striegel-Moore RH, Nicholson TJ, Tamborlane WV: Prevalence of eating disorder symptoms in preadolescent and adolescent girls with IDDM. Diabetes Care 1992; 15:1361–1368
13. Peveler RC, Fairburn CG, Boller I, et al: Eating disorders in adolescents with IDDM. Diabetes Care 1992; 15:1356–1360
14. Biggs MM, Ramirez Basco M, Patterson G, et al: Insulin withholding for weight control in women with diabetes. Diabetes Care 1994; 17:1186–1189
15. Polonsky WH, Anderson BJ, Lohrer PA, et al: Insulin omission in women with IDDM. Diabetes Care 1994; 17:1178–1185
16. Marcus MD, Wing RR, Jawad A, et al: Eating disorders symptomatology in a registry-based sample of women with insulin-dependent diabetes mellitus. Int J Eat Disord 1992; 12:425–430
17. Kendler KS, MacLean C, Neale M, et al: The genetic epidemiology of bulimia nervosa. Am J Psychiatry 1991; 148:1627–1637
18. King MB: Eating disorders in a general practice population: prevalence, characteristics and follow-up at 12 to 18 months. Psychol Med 1989; 14:1–34
19. Robiner WN, Keel PK: Self-care behaviors and adherence in diabetes mellitus. Seminars in Clinical Neuropsychiatry 1997; 2:40–56
20. Rodin GM, Johnson LE, Garfinkel PE, et al: Eating disorders in female adolescents with insulin-dependent diabetes mellitus. Int J Psychiatry Med 1987; 16:49–57
21. Rosmark B, Berne C, Holmgren S, et al: Eating disorders in patients with insulin-dependent diabetes mellitus. J Clin Psychiatry 1986; 47:547–550
22. Lloyd GG, Steel JM,Young RJ: Eating disorders and psychiatric morbidity in patients with diabetes mellitus. Psychother Psychosom 1987; 48:189–195
23. Steel JM, Young RJ, Lloyd GG, et al: Clinically apparent eating disorders in young diabetic women: associations with painful neuropathy and other complications. BMJ 1987; 294:859–862
24. Popkin MK, Callies AL, Lentz RD, et al: Prevalence of major depression, simple phobia, and other psychiatric disorders in patients with long-standing Type 1 diabetes mellitus. Arch Gen Psychiatry 1988; 45:64–68
25. Steel JM, Young RJ, Lloyd GG, et al: Abnormal eating attitudes in young insulin-dependent diabetics. Br J Psychiatry 1989; 155:515–521
26. Stancin T, Link DL, Reuter JM: Binge eating and purging in young women with IDDM. Diabetes Care 1989; 12:601–603
27. Pollock M, Kovacs M, Charron-Prochownik D: Eating disorders and maladaptive dietary/insulin management among youths with childhood-onset insulin-dependent diabetes mellitus. J Am Acad Child Adolesc Psychiatry 1995; 34:291–296
28. Herpertz S, Wagoner R, Albus C, et al: Diabetes mellitus and eating disorders: a multicenter study on the comorbidity of the two diseases. Paper presented at the 7th New York International Conference on Eating Disorders, New York, 1996
29. Vila G, Friedman S, Timsit J, et al: Eating disorders and insulin-dependent diabetes mellitus. Paper presented at the 7th New York International Conference on Eating Disorders, New York, 1996
30. Cantwell R, Steel J: Screening for eating disorders in diabetes mellitus. J Psychosom Res 1996; 40:15–20
31. Spitzer RL, Devlin MJ, Walsh BT, et al: Binge eating disorder: a multisite field trial for the diagnostic criteria. Int J Eat Disord 1992; 11:191–203
32. Spitzer RL, Yanovski S, Wadden T, et al: Binge eating disorder: its further validation in a multisite study. Int J Eat Disord 1993; 13:137–153
33. American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, 4th Edition. Washington, DC, American Psychiatric Association, 1994, p. 327
34. Rydall A, Rodin G, Olmsted M, et al: Disordered eating behavior and microvascular complications in young women with insulin-dependent diabetes mellitus. N Engl J Med 1997; 336:1849–1854
35. deZwaan M, Mitchell J: Medical complications of anorexia nervosa and bulimia nervosa, in Medical Issues and Eating Disorders, edited by Kaplan AS, Garfinkel PE. New York, Brunner/Mazel, 1993, pp. 60–100
36. LaGreca AM, Schwarz LT, Satin W: Eating patterns in young women with IDDM: another look. Diabetes Care 1987; 10:659–660
37. Wing RR, Nowalk MP, Marcus MD, et al: Subclinical eating disorders and glycemic control in adolescents with Type I diabetes. Diabetes Care 1986; 9:162–167
38. Colas C, Mathieu P, Tchobroutsky G: Eating disorders and retinal lesions in Type I (insulin-dependent) diabetic women. Diabetologia 1991; 34:288
39. Lorini R, d'Annunzio G, Cortona L, et al: Eating disorders in adolescents and young women with insulin-dependent diabetes mellitus (IDDM). Advances in the Biosciences 1993; 90:507–510
40. Carney C, Andersen A, Barsness J, et al: The influence of Type I diabetes mellitus on the course and outcome of eating disorders: a comparative analysis. Paper presented at the 7th New York International Conference on Eating Disorders, New York, 1996
41. Cohen ST, Welch G, Jacobson AM, et al: The association of lifetime psychiatric illness and increased retinopathy in patients with Type I diabetes mellitus. Psychosomatics 1997; 38:98–108
42. Keesey RE: A set-point theory of obesity, in Handbook of Eating Disorders, edited by Brownell KD, Foreyt JP. New York, Basic Books, 1986, pp. 63–87
43. Fairburn CG, Marcus MD, Wilson GT: Cognitive behavior therapy for binge eating and bulimia nervosa: a comprehensive treatment manual, in Binge Eating: Nature, Assessment and Treatment, edited by Fairburn CG, Wilson GT. New York, Guilford, 1993, pp. 361–404

44. Fairburn CG: Interpersonal psychotherapy for bulimia nervosa, in Handbook of Treatment for Eating Disorders, edited by Garner DM, Garfinkel PE. New York, Guilford, 1997, pp. 278–294
45. Crow SJ, Mitchell J: Pharmacologic treatments for eating disorders, in Body Image, Eating Disorders, and Obesity, edited by Thompson JK. Washington, DC, American Psychological Association, 1996, pp. 345–362
46. Russell FM, Szmukler GI, Dare C, et al: An evaluation of family therapy in anorexia nervosa and bulimia nervosa. Arch Gen Psychiatry 1987; 44:1047–1056
47. Kaye WH, McConaha C, Nagata T, et al: Fluoxetine prevents relapse in a majority of patients within anorexia nervosa: a double-blind placebo-controlled study. Paper presented at the 2nd Eating Disorders Research Conference, Pittsburgh, PA, 1996
48. Hudson JI, Pope HG, Yurgelun-Todd D, et al: A controlled study of lifetime prevalence of affective and other psychiatric disorders in bulimic outpatients. Am J Psychiatry 1987; 144:1283–1287
49. Halmi KA, Eckert E, Marchi P, et al: Comorbidity of psychiatric diagnoses in anorexia nervosa. Arch Gen Psychiatry 1991; 48:712–718
50. Brewerton TD, Lydiard RB, Herzog DB, et al: Comorbidity of Axis I psychiatric disorders in bulimia nervosa. J Clin Psychiatry 1995; 56:77–80
51. Lustman PJ, Griffith LS, Clouse RE, et al: Psychiatric illness in diabetes mellitus: relationship to symptoms and glucose control. J Nerv Ment Dis 1986; 174:736–742
52. Polivy J, Herman CP: Dieting and binging: a causal analysis. Am Psychol 1985; 40:193–201
53. Steel JM, Lloyd RJ, MacIntyre CCA: Changes in eating attitudes during the first year of treatment for diabetes. J Psychosom Res 1990; 34:313–318