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The interplay between diabetes distress, disordered eating behaviors, and antidiabetic medications in Type 2 diabetes mellitus

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ABSTRACT

Aim: Type 2 diabetes mellitus (T2DM) imposes psychological and behavioral challenges. While prior research links eating habits, distress, and glycemic control, few studies explore these factors together. This study assessed the associations between night eating, eating patterns, diabetes distress (DDS), glycemic control, BMI, gender, and medication use in adults with T2DM.

Methods: This study included 105 adults with T2DM. Night eating behavior, eating patterns, and DDS were evaluated using the Night Eating Questionnaire (NEQ), the 18-item version of the Three-Factor Eating Questionnaire-Revised (TFEQ-R18), and the Diabetes Distress Scale (DDS).

Results: NEQ score correlated with BMI (r=0.38, p=0.001) and remained significant after adjusting for age, gender, and HbA1c (p=0.002). TFEQ-R18 emotional eating inversely correlated with BMI (r=−0.36, p=0.001) and persisted after adjustment (p=0.004). Uncontrolled eating and cognitive restraint showed no links. Men scored higher on emotional eating than women (10.0 ± 2.54 vs. 8.69 ± 3.20, p = 0.02). Poor glycemic control had higher DDS emotional burden and regimen-related distress; NEQ and TFEQ-R18 scores did not differ by glycemic status or medication classes.

Conclusion: DDS, particularly emotional burden and regimen-related distress, differed across HbA1c categories, but this was not confirmed in multivariable models. BMI, not glycemic control, more consistently predicted eating behavior and distress. Given the cross-sectional design and small sample size, the no significant association between eating behavior and glycemic control does not rule out a relationship. These findings support routine distress screening in diabetes care and larger prospective studies of these associations and their relationship to body weight.

Keywords: Eating behavior, night eating, type 2 diabetes mellitus, diabetes distress.

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1. Introduction

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder that requires affected individuals to manage their condition continuously and actively. The prevalence of T2DM has increased substantially in recent decades, affecting more than 400 million

people worldwide. This number is projected to rise to over 600 million by 2045, underscoring the disease's escalating global impact [1]. Effective disease management requires sustained adherence to dietary modification, physical activity, glycemic monitoring, and pharmacological therapy. As a result, the persistent demands of self-care may place considerable psychological strain on individuals, negatively impacting mental well-being and quality of life [2].

Adherence to dietary recommendations constitutes a cornerstone of T2DM self-management, yet it remains one of the most clinically and psychologically challenging aspects of lifestyle intervention. Successful dietary adherence demands not only adequate nutritional literacy but also robust behavioral regulation to mitigate the effects of environmental cues, emotional fluctuations, and diabetes-specific distress [3]. As a result, maladaptive eating behaviors, especially uncontrolled eating, emotional eating, and cognitive dietary restraint, are more common among people with T2DM [4]. Compounding evidence indicates that these dysregulated eating behaviors severely undermine metabolic control, accelerate weight gain, and elevate long-term cardiovascular risk, thereby presenting a critical barrier to optimal clinical outcomes [5,6]. Diabetes-related stress (DDS) is associated with both disordered eating behaviors and nonadherence to treatment. This relationship creates a bidirectional burden; while the daily self-management demands of diabetes generate stress, stress can also worsen glycemic control by disrupting eating behavior and treatment adherence [7,8].

Beyond these general patterns of dysregulated eating, the temporal distribution of food intake represents an equally important yet underrecognized dimension of dietary behavior in T2DM. Night eating behavior constitutes a distinct and clinically relevant pattern of food intake characterized by evening hyperphagia, recurrent nocturnal food consumption, and morning anorexia [9]. Beyond its associations with obesity and metabolic dysregulation, night eating has been linked to disrupted circadian rhythmicity of insulin secretion and impaired postprandial glucose metabolism, thereby rendering it relevant in T2DM [10]. Notably, night eating has also been associated with elevated psychological distress, sleep disturbance, and negative affect, factors that may interact with diabetes-specific distress to further compromise glycemic stability [11].

Despite this evidence, the simultaneous assessment of diabetes distress, multidimensional eating behavior, and night eating patterns in relation to glycemic control, body composition, and antidiabetic treatment modality remains sparse in the T2DM literature. The present study, therefore, aimed to examine the relationships between diabetes-related distress, eating behavior dimensions (including night eating), and glycemic regulation status in adults with T2DM, and to explore their associations with body mass index, gender, and antidiabetic medication use.

2. Materials and methods

2.1. Study Design and Setting: This was a cross-sectional observational study conducted at a single-center endocrinology outpatient clinic. The study protocol was approved by the institutional ethics committee, and all participants provided written informed consent prior to enrollment.

2.2. Participants: A sample of 105 adults with T2DM attending routine outpatient follow-up visits was recruited. Participants needed to be between 18 and 75 years old and

have a clinician-confirmed diagnosis of T2DM, and either have available HbA1c and fasting plasma glucose measurements from the preceding three months or consent to undergo measurements as part of the study. Individuals with type 1 diabetes, gestational diabetes, or other specific diabetes subtypes were excluded, as were those with active psychotic disorder or significant cognitive impairment, a history of bariatric surgery, current pregnancy or lactation, a major change in antidiabetic medication within the three months preceding enrollment, or a concurrent condition known to substantially affect eating behavior or metabolic regulation, such as Cushing's syndrome. Patients were stratified into two groups based on glycemic control status: adequate (HbA1c <7.5%) and suboptimal (HbA1c ≥7.5%). Although most major clinical guidelines recommend an HbA1c target below 7.0% [12], this study used a 7.5% cut-off for patient categorization. This adjustment was necessary because applying the 7.0% threshold created a significant class imbalance, with only 20 patients in the ≤7.0% group, thereby reducing the statistical power of comparative analyses. The 7.5% threshold allowed for robust statistical analysis while maintaining a clinically relevant marker for treatment intensification, in accordance with national diabetes management guidelines.

During the study period, participants were systematically gathered from their routine outpatient follow-up visits. The total sample size was based on the number of individuals enrolled during the designated recruitment period, as no initial sample size calculation was conducted.

2.3. Data Collection: Participants completed a structured sociodemographic and clinical data form administered by a physician, which captured age, gender, diabetes duration, presence of complications, comorbid conditions, and current antidiabetic medications. During the visit, measurements such as body weight and height were taken to calculate BMI. Psychometric assessments were conducted using self-report measures completed by the participants.

2.4. Laboratory Parameters: Biochemical data were obtained from routine outpatient laboratory assessments. They included glycated hemoglobin (HbA1c), fasting plasma glucose, serum creatinine, low-density lipoprotein cholesterol (LDL), triglycerides (TG), total cholesterol (TC), aspartate aminotransferase (AST), and alanine aminotransferase (ALT).

2.5. Measures: Nocturnal eating patterns and circadian rhythm disruption were assessed using the Night Eating Questionnaire (NEQ), originally developed by Allison et al. and subsequently validated in Turkish by Atasoy et al. [13,14]. The scale assesses evening hyperphagia, nighttime awakenings for eating, morning anorexia, and mood and sleep issues linked to night eating. Higher scores reflect more severe night eating and a more disrupted eating and sleep pattern. Consisting of 14 items with a five-point Likert scale (0 = not at all/never, 4 = very much/always), the total score ranges from 0 to 52, with higher scores indicating more severe night eating syndrome symptoms.

Multidimensional eating behavior was assessed using the TFEQ-R18. Originally developed by Stunkard and Messick and subsequently revised by Karlsson et al, the scale was adapted for use in Turkey by Kıraç et al., with its validity and reliability duly established [15,16].

The questionnaire comprises 18 items structured around three factors, uncontrolled eating, cognitive restraint, and emotional

eating, and employs a four-point Likert-type response.

Emotional distress related to diabetes was measured with the Diabetes Distress Scale (DDS), initially created by Polonsky et al. and later validated in Turkish by Caklili et al. [17,18]. The scale includes 17 items that assess the emotional burden of living with diabetes over the past month, covering four areas: emotional burden, distress related to physicians, regimen-related distress, and interpersonal distress. Respondents rate each item on a six-point Likert scale (1 = not a problem; 6 = a serious problem). Mean scores are computed for each domain and for the overall scale. Higher scores reflect increased diabetes-related distress.

2.6. Statistical Analysis: Statistical analyses were conducted using Jamovi (version 2.6.26.0). Continuous variables are presented as mean ± SD, while categorical variables are shown as frequencies (n) and percentages (%). The Shapiro-Wilk test and histograms assessed the normality of continuous data, with all variables meeting normality assumptions. Group differences in psychometric scale scores based on gender, glycemic regulation, and antidiabetic medication were tested using independent-samples t-tests. Pearson correlation analysis examined relationships between continuous variables (NEQ, TFEQ-R18 subscales, DDS scores). For variance equality checks, Levene's test was used; if assumptions were violated, Welch's t-test replaced Student's t-test. Due to the small sample size of pioglitazone users (n=5), the Mann-Whitney U test was applied. Associations with age, gender, BMI, HbA1c, and diabetes duration were also assessed via Pearson correlations. Multivariable linear regression analyses evaluated independent effects of covariates on NEQ, TFEQ-R18, and DDS scores, including age, gender, BMI, and HbA1c. Effect sizes (Cohen's d) and 95% confidence intervals accompany p-values, with significance set at $p<0.05$.

3. Results

A total of 105 patients were included in the study. The mean age was 49.9 ± 11.2 years, and the sample comprised 58 women (55.2%) and 47 men (44.8%). Mean BMI was 31.9 ± 6.09 kg/m². Mean duration of diabetes was 11.9 ± 8.44 years. Baseline laboratory parameters and antidiabetic medication use of the patients are presented in Table 1 and 2.

Table 1. Clinical and laboratory characteristics of the study population.

Characteristic	Value
Age (years), mean ± SD	49.9 ± 11.2
Gender, n (%)	
Female	58 (55.2%)
Male	47 (44.8%)
BMI (kg/m²), mean ± SD	31.9 ± 6.09
Glucose (mg/dL)	189 ± 99.2
HbA1c (%)	8.4 ± 2.15
Creatinine (mg/dL)	0.9 ± 0.38
Total cholesterol (mg/dL)	192 ± 45.6
LDL (mg/dL)	105 ± 39.4
Triglycerides (mg/dL)	227 ± 205
AST(U/L)	20.4 ± 10.8
ALT(U/L)	22.5 ± 13.3

SD, standard deviation; BMI, body mass index. LDL, low-density lipoprotein cholesterol; AST, aspartate aminotransferase; ALT, alanine aminotransferase.

Mean NEQ total score was 22.4 ± 6.42. Regarding the TFEQ-R18 subscales, mean uncontrolled eating, cognitive restraint, and

emotional eating scores were 23.0 ± 5.12, 14.8 ± 2.78, and 9.28 ± 2.98, respectively.

Table 2. Antidiabetic medication use.

Medication	n	%
Metformin	75	71.4%
DPP-4 inhibitor	49	46.7%
GLP-1 receptor agonist	16	15.2%
SGLT2 inhibitor	68	64.8%
Sulfonylurea	17	16.2%
Pioglitazone	5	4.8%
Insulin *Mean daily dose: 55.1 ± 33.4 IU (among insulin users, n=55)*	55	52.4%

Percentages were calculated based on the total study population (N=105). Categories are not mutually exclusive, as patients may have been receiving combination therapy. DPP-4, dipeptidyl peptidase-4; GLP-1, glucagon-like peptide-1; SGLT2, sodium-glucose cotransporter-2.

With respect to diabetes-related distress, mean scores were 2.89 ± 1.15 for the emotional burden domain, 2.64 ± 1.27 for the physician-related distress domain, 3.04 ± 1.30 for the regimen-related distress domain, and 2.57 ± 1.47 for the interpersonal distress domain. The mean DDS total score was 2.81 ± 1.15. Scores for eating behavior and DDS are presented in Table 3. No statistically significant differences were observed between genders in NEQ total score (p = 0.19) or in the TFEQ-R18 uncontrolled eating (p = 0.45) and cognitive restraint (p = 0.40) subscale scores. However, women had significantly lower emotional eating scores than men (8.69 ± 3.20 vs. 10.0 ± 2.54, p = 0.02). None of the DDS subscales differed significantly by gender, including emotional burden, physician-related distress, regimen-related distress, and interpersonal distress domains (all p > 0.05). Detailed comparisons of scale scores by gender are presented in Table 4.

Table 3. Descriptive statistics of psychometric scale scores.

Scale / Subscale	Mean ± SD
NEQ Score	22.4 ± 6.42
TFEQ-R18 Subscales	
Uncontrolled Eating	23 ± 5.12
Cognitive Restraint	14.7 ± 2.
Emotional Eating	9.28 ± 2.98
DDS Subscales	
Emotional Burden	2.89 ± 1.15
Physician-Related Distress	2.64 ± 1.27
Regimen-Related Distress	3.04 ± 1.3
Interpersonal Distress	2.57 ± 1.47

SD, standard deviation; NEQ, Night Eating Questionnaire; TFEQ-R18, Three-Factor Eating Questionnaire-Revised 18-item version; DDS, Diabetes Distress Scale.

Pearson correlation analyses examined the associations between eating behavior and diabetes distress measures-including NEQ, TFEQ-R18 subscales, and DDS domains-as well as variables such as age, gender, BMI, HbA1c, and diabetes duration. The NEQ total score was significantly positively correlated with BMI (r = 0.38, p = 0.001). Among the TFEQ-R18 subscales, emotional eating showed a significant negative correlation with BMI (r = −0.36, p = 0.001).

For DDS domains, emotional burden was significantly associated with HbA1c levels (r = 0.20, p = 0.04), whereas interpersonal distress was significantly associated with BMI (r = 0.23, p = 0.03). No other significant associations were observed between the remaining eating behaviors or diabetes distress measures and age, gender, BMI, HbA1c, or diabetes duration (all p > 0.05).

Table 4. Comparison of psychometric scale scores by gender.

Scale / Subscale	Female	Male	*p*
NEQ Score	23.1 ± 6.68	21.4 ± 6.02	0.19
TFEQ-R18 Scale			
Uncontrolled Eating	22.7 ± 4.94	23.4 ± 5.35	0.45
Cognitive Restraint	14.3 ± 2.8	15.2 ± 2.31	0.11
Emotional Eating	8.69 ± 3.2	10 ± 2.54	**0.02***
DDS			
Emotional Burden	2.99 ± 1.19	2.77 ± 1.1	0.34
Physician-Related Distress	2.57 ± 1.33	2.73 ± 1.2	0.53
Regimen-Related Distress	3.12 ± 1.23	2.94 ± 1.38	0.47
Interpersonal Distress	2.66 ± 1.58	2.46 ± 1.35	0.49

*Values are presented as mean ± SD. An independent samples t-test was used for comparisons. NEQ, Night Eating Questionnaire; TFEQ-R18, Three-Factor Eating Questionnaire-Revised 18-item version; DDS, Diabetes Distress Scale; df, degrees of freedom. *p < 0.05.*

We constructed a multivariable linear regression model examining the association between NEQ total score and BMI, adjusting for age, HbA1c, and gender. In this adjusted model, BMI remained a significant independent predictor of NEQ total score (95% CI: 0.129–0.563, $p = 0.002$), whereas age, HbA1c, and gender were not significantly associated with NEQ total score (all $p > 0.05$).

In the adjusted model, the TFEQ-R18 uncontrolled eating score was not significantly associated with BMI, HbA1c, gender, or age (all $p > 0.05$). In the adjusted model, the TFEQ-R18 cognitive restraint score was not significantly associated with BMI, age, or gender (all $p > 0.05$). A borderline inverse association with HbA1c was observed (95% CI: −0.452 to 0.023, $p = 0.07$). Emotional eating score was significantly and inversely associated with BMI in the adjusted model (95% CI: −0.524 to −0.104, $p = 0.004$), independent of age, gender, and HbA1c ($p > 0.05$).

To further evaluate the association between DDS and glycemic control while accounting for potential confounders, we constructed multivariable linear regression models for each DDS domain, with age, gender, BMI, and HbA1c entered as covariates. For the DDS emotional burden domain, BMI was associated with higher scores at the 95% CI threshold (0.001–0.455, $p = 0.049$), whereas age, HbA1c, and gender were not significant predictors (all $p > 0.05$). None of the covariates, including HbA1c, BMI, age, or gender, showed significant associations with the DDS regimen-related distress scores (all $p > 0.05$). In the DDS interpersonal distress domain, BMI was notably and positively associated with scores (95% CI for β: 0.021–0.473, $p = 0.033$), regardless of age, HbA1c, and gender (all $p > 0.05$). For the DDS physician-related distress domain, none of the covariates, such as age, gender, BMI, or HbA1c, showed significant associations with scores (all $p > 0.05$).

To explore potential associations between antidiabetic medication, use and eating behavior and diabetes distress measures, exploratory independent-samples t-tests were conducted for each medication class, comparing users and non-users across NEQ, TFEQ-R18 subscales, and DDS domains. Given the markedly small number of pioglitazone users (n = 5), the non-parametric Mann-Whitney U test was used for this comparison instead. No significant differences were observed between users and non-users for metformin, sulfonylurea, DPP-4 inhibitors, SGLT2 inhibitors, pioglitazone, GLP-1 receptor agonists, or insulin across any measure (all $p > 0.05$).

Patients were stratified into adequate (HbA1c <7.5%) and suboptimal (HbA1c ≥7.5%) glycemic control groups. No significant differences were observed between the groups in NEQ total score or any TFEQ-R18 subscale score (all $p > 0.05$). With respect to diabetes-related distress, patients with suboptimal glycemic control had significantly higher DDS emotional burden (3.10 ± 1.12 vs. 2.48 ± 1.09, $p = 0.008$) and regimen-related distress scores (3.35 ± 1.29 vs. 2.49 ± 1.14, $p = 0.001$) compared to those with adequate glycemic control. No significant between-group differences were found for the DDS physician-related or interpersonal distress domains (all $p > 0.05$). Group comparisons of psychometric test scores according to glycemic regulation status are presented in Table 5.

4. Discussion

This study evaluated eating behaviors and DDSin adults with type 2 diabetes and examined their associations with clinical and metabolic characteristics. The results indicate that body mass index (BMI), rather than glycemic control alone, is a significant factor associated with eating behavior patterns. Higher BMI was independently associated with increased night eating behavior, while emotional eating was inversely associated with

Table 5. Comparison of psychometric scale scores by glycemic regulation status.

Scale / Subscale	Adequate glycemic control	Suboptimal glycemic control	*p*
NEQ Score	21.7 ± 6.11	22.8 ± 6.71	0.39
TFEQ-R18 Scale			
Uncontrolled Eating	23.5 ± 4.65	22.5 ± 5.41	0.36
Cognitive Restraint	14.7 ± 2.34	14.7 ± 2.79	0.99
Emotional Eating	9.53 ± 2.92	9.11 ± 3.07	0.49
DDS			
Emotional Burden	2.48 ± 1.09	3.1 ± 1.12	**0.008***
Physician-Related Distress	2.44 ± 1.28	2.71 ± 1.23	0.30
Regimen-Related Distress	2.49 ± 1.14	3.35 ± 1.29	**0.001***
Interpersonal Distress	2.27 ± 1.57	2.69 ± 1.37	0.16

*Values are presented as mean ± SD. Regulated, HbA1c <7.5%; Unregulated, HbA1c ≥7.5%. An independent samples t-test was used for comparisons. NEQ, Night Eating Questionnaire; TFEQ-R18, Three-Factor Eating Questionnaire-Revised 18-item version; DDS, Diabetes Distress Scale; df, degrees of freedom. *p < 0.05.*

BMI after adjustment for age, sex, and HbA1c. Eating behavior scores did not differ by glycemic control status at the HbA1c threshold of 7.5%. In relation to diabetes-related distress, suboptimal glycemic control was associated with greater emotional burden and regimen-related distress in unadjusted group comparisons. However, in multivariable analyses, BMI rather than HbA1c was associated with emotional burden and interpersonal distress. Overall, these findings indicate that eating behaviors and DDS may be influenced by distinct clinical factors, with BMI emerging as a potentially relevant correlate of both domains in patients with type 2 diabetes.

The NEQ total score was significantly and positively correlated with BMI, and this association remained significant in multivariable analyses, indicating an independent association between more pronounced night eating behavior and higher body weight in this study population. Our finding is consistent with general adult literature linking night eating to obesity and adverse metabolic outcomes [19,20]. However, the literature on T2DM shows conflicting results. While some studies report a strong link [21,22], others show no significant association [23]. The positive correlation observed in our study suggests that night eating behaviors may be associated with higher total energy intake, irregular eating patterns, and shorter fasting periods, since the cross-sectional design prevents conclusions about the causal direction of this relationship. While many individuals with T2DM eat late in the evening, this habit is not consistently associated with higher BMI across studies. Our findings may therefore reflect a broader behavioral symptom burden rather than the timing of food intake alone, a perspective consistent with evidence that dysfunctional eating in T2DM is associated with increased caloric intake and poorer dietary adherence [24]. Consequently, while our data demonstrates a clear association within this sample, further large-scale and longitudinal studies are warranted to fully clarify the complex interaction between behavioral symptom load and weight gain in T2DM.

In contrast to the positive association observed between NEQ scores and BMI, the TFEQ-R18 emotional eating subscale was significantly and inversely correlated with BMI. This inverse association contrasts with the patterns predominantly reported in the literature, where emotional eating is consistently positively associated with BMI in both general and clinical populations [6,22,23], making this finding less consistent with existing evidence. Neither cognitive restraint nor uncontrolled eating demonstrated significant associations with BMI in either univariate or adjusted analyses. Cognitive restraint, however, showed a borderline inverse association with HbA1c in the adjusted model, suggesting that patients with less stringent glycemic control may exhibit modestly lower levels of conscious dietary restraint. Given the borderline significance and the cross-sectional design, this result should be interpreted with caution and requires confirmation in larger samples. The inverse association between emotional eating and BMI may reflect diabetes-specific behavioral adaptation, structured dietary counseling as part of routine T2DM management, or sample compositional factors. However, due to the cross-sectional design and modest sample size, these interpretations remain speculative and require validation in larger prospective studies.

In the present study, men reported significantly higher emotional eating scores than women. This finding diverges from the prevailing literature, in which women

consistently report higher emotional eating than men [25,26]. No significant gender differences were observed for NEQ total score, uncontrolled eating, or cognitive restraint, which is consistent with evidence that gender effects on eating behavior tend to be dimension-specific rather than uniform across all eating constructs [26,27]. The different gender patterns of emotional eating may reflect sample-specific moderators or gender-related differences in the underlying drivers of emotional eating; in men, emotional eating has been proposed to be more closely linked to reward-seeking, alexithymia, or food insecurity rather than to the affective pathways more typically described in women [25].

Patients with suboptimal glycemic control reported significantly higher DDS emotional burden and regimen-related distress than those with adequate control. Physician-related and interpersonal distress did not differ significantly between groups. When HbA1c was analyzed as a continuous variable in multivariable models adjusting for age, gender, and BMI, it was not significantly associated with any DDS domain. Instead, BMI was a significant independent correlate of emotional burden and interpersonal distress. This discrepancy may be due to the categorical analysis capturing non-linear or threshold effects, which are reduced when HbA1c is modeled continuously, and to the categorical comparison failing to adjust for BMI or other covariates, potentially introducing confounding.

Overall, our findings suggest that BMI may be a more consistent correlate of DDS than glycemic control in this study. The observed categorical association between glycemic control and distress aligns with previous literature linking regimen-related distress, emotional burden, and poorer glycemic control [28,29]. The lack of a significant association between night eating or disordered eating patterns and glycemic control status indicates that diabetes-specific emotional burden, rather than general eating-behavior dimensions, may be more closely related to glycemic outcomes in this population. However, cross-sectional design limits the ability to determine the direction or underlying mechanisms of these relationships.

No significant associations were found between antidiabetic medication types and measures of eating behavior or DDS in the exploratory analyses, including for GLP-1 receptor agonists and pioglitazone. The small number of participants using these medications limits the statistical power of these findings, so they should be interpreted with caution. Confounding by indication is also possible, as certain drugs may be prescribed more often to patients with specific metabolic or behavioral profiles. Larger, adequately powered studies are needed to clarify whether specific antidiabetic medications are associated with eating behavior or DDS in T2DM.

Taken together, these findings indicate that BMI, rather than glycemic control, is most consistently associated with eating behavior and DDS in this cohort with T2DM. Independent associations were identified for the NEQ total score, the TFEQ-R18 emotional eating subscale, and the DDS emotional burden and interpersonal distress domains. The association between glycemic control status and DDS appears to be clinically significant, although this relationship is not evident when hemoglobin A1c (HbA1c) is analyzed as a continuous variable. This finding aligns with previous research that has linked diabetes distress to glycemic outcomes [28,29]. Integrating brief, validated distress-screening tools such as the DDS into routine diabetes

follow-up appointments is valuable for identifying patients who may benefit from targeted psychosocial support. The consistent association between BMI and measures of eating behavior and distress highlights the importance of assessing these factors, especially in patients with higher body weight. Due to the cross-sectional design, modest sample size, and limited statistical power of subgroup analyses, the lack of a significant association between night eating or disordered eating behavior and glycemic control in this sample should not be considered definitive. No significant relationships were observed between specific classes of antidiabetic medications and measures of eating behavior or diabetes-related distress. As a result, we cannot draw conclusions about the effects of specific medications on these outcomes from the current data.

4.1. Limitations: This study has several limitations to consider when interpreting the results. First, the cross-sectional design does not allow for conclusions about temporal or causal relationships among diabetes-related distress, eating behavior, and glycemic control. Longitudinal studies are needed to determine whether distress precedes or follows changes in glycemic regulation. Second, the smaller sample size limited the statistical power of certain subgroup analyses and may have impacted the accuracy of the estimates. Third, the number of patients receiving certain antidiabetic agents, especially pioglitazone (n = 5), was too small to draw definitive conclusions about medication-specific associations; these results should be viewed as exploratory. Fourth, all measures of eating behavior and distress relied on self-report instruments, which are subject to recall and social desirability biases. Fifth, as a single-center study conducted in a Turkish population, the findings may not be generalizable to other healthcare settings or cultural contexts.

4.2. Conclusion: In adults with T2DM, DDS, particularly emotional and regimen-related distress, varied significantly according to glycemic control when HbA1c was analyzed as a categorical variable. This association was not observed in multivariable models utilizing continuous HbA1c. Adjusted analyses indicated that body mass index (BMI), rather than glycemic control, was consistently associated with both eating behavior and diabetes-related distress. Given the cross-sectional design, small sample size and limited statistical power in subgroup analyses, the lack of a significant association between eating behavior and glycemic control should not be interpreted as conclusive. These results underscore the potential utility of brief distress-screening tools in routine diabetes care and highlight the necessity for prospective studies to determine the directionality of these associations and the influence of body weight on the psychological burden of diabetes.

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Ethical Statement: *The study protocol was reviewed and approved by the Ethics Committee of Basaksehir Cam and Sakura City Hospital (approval number: E-96317027-514.10-314878537; date: May 20, 2026).*

Patient Consent Statement

Written informed consent was obtained from all participants prior to enrollment in the study.

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