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Prevalence of Depression and Anxiety in Patients with Type 2 Diabetes Mellitus and Their Association with Glycemic Control: A Cross-Sectional Study

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ABSTRACT

Background: Depression and anxiety are common yet underrecognized comorbidities in patients with type 2 diabetes mellitus (T2DM). Both conditions may adversely influence self-management and contribute to poor glycemic outcomes, but local data from South Asia remain limited.

Objective: To determine the prevalence of depression and anxiety in adults with T2DM and examine their association with glycemic control.

Methods: A cross-sectional study was conducted among 90 adults with T2DM attending two tertiary care hospitals in Punjab, Pakistan. Depression and anxiety were assessed using validated Urdu/English versions of the Patient Health Questionnaire-9 (PHQ-9) and the Generalized Anxiety Disorder-7 (GAD-7). Glycemic control was indexed by glycated hemoglobin (HbA1c), measured with NGSP-certified assays. Logistic and linear regression models evaluated associations between psychological morbidity and HbA1c, adjusting for demographic and clinical covariates.

Results: The mean age of participants was 54.8 ± 10.2 years; 47.8% were male. Mean diabetes duration was 9 years (IQR 5–13). Moderate-to-severe depressive symptoms were present in 31.1% and clinically significant anxiety in 27.8%; 17.8% had both conditions. Diabetes distress was reported by 36.7%. Participants with depression had significantly higher HbA1c than those without (8.5 ± 1.3% vs. 7.6 ± 1.3%; $p<0.001$). Anxiety was similarly associated (8.4 ± 1.2% vs. 7.6 ± 1.4%; $p=0.003$). In adjusted models, depression remained independently linked to higher HbA1c ($\beta=+0.32\%$, $p=0.002$), and screen-positive depression doubled the odds of poor glycemic control (OR=2.41, 95% CI 1.03–5.61).

Conclusion: Depression and anxiety are prevalent among Pakistani adults with T2DM and strongly correlate with poor glycemic control. Integrating routine mental health screening and multidisciplinary interventions into diabetes care could improve both psychological well-being and metabolic outcomes.

Keywords: type 2 diabetes, depression, anxiety, glycemic control, HbA1c

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INTRODUCTION

Diabetes mellitus type 2 (T2DM) is a chronic and progressive metabolic disorder that crosscuts across nearly all health domains, such as microvascular and macrovascular complications and daily self-management needs [1]. As an increasing number of individuals are living longer with increased survival rates of T2DM, the emphasis has shifted off the glucose measures to the overall determinant of good outcomes such as mental health. Depression and anxiety are such comorbidities that are very consequential among them. They are everywhere, generally not valued and closely related to the habits and biological mechanisms conditioning the prolonged glycemic regulation. This triad T2DM, depression, and anxiety will have to be known by clinicians, health-system planners, and studyers who will be necessitated to promote the quality of life and also cardiometabolic risk profiles [2].

The relationship between common mental disorders and T2DM can best be referred to as reciprocal as well as reinforcing. On the one hand, there is a cognitive and emotional load of a long-term character glucose control and pharmacotherapy and diet restriction and physical activity [3]. This strain may be further enhanced by concerns on hypoglycemia, complications, and financial costs, predisposing patients to the symptoms of depression and anxiety. On the other hand, depression and anxiety influence even those routines which include diabetes in control: they reduce motivation, executive action, ruin self-efficacy, and worsen the implications of problem-solving that complicate medication adherence, plans to eat, regular physical activity, and check-ups. The net effect is the mechanism of a vicious cycle, where psychological suffering impairs glycemic control, and impaired glycemic control, in its turn, aggravates mood and anxiety [4].

There are several biological processes that are plausible associating these conditions not only in behaviors. Prolonged psychological stress may stimulate hypothalamic-pituitary-adrenal axis and sympathetic nervous system, increasing counter-regulatory hormones and inflammatory agents that impair insulin sensitivity [5]. The presence of low-grade systemic inflammation and oxidative stress indicators of depression and T2DM is likely to impair endothelial activity and increase vascular risk. Sleep disturbance which is a typical feature of anxiety disorder and depressive disorder is also linked with insulin resistance and appetite dysregulation. In the meantime, hyperglycemia may also be a factor in fatigue, cognitive deceleration, and somatic symptoms that resemble or exacerbate depression and blur the line between diagnosis and clinical evaluation [6].

Clinicians have other issues with measurement and detection. Symptoms of diabetes, which are somatic and are, in most cases, associated with diabetes include fatigue, alterations to appetite, and lack of sleep [7]. These symptoms are often intertwined with depressive and anxiety symptoms. In the absence of a systematic strategy, such symptoms are readily explained as the diabetes as opposed to being identified as subjects to treatment through the mental-health framework. Standardized, short screening instruments with regular intervals are a convenient means of bringing at-risk patients to light, though positive screens need to be followed by clinical assessment of determining diagnoses and severity. Notably, emotional reactions or responses to diabetes sometimes referred to as diabetes distress, can be comorbid to, yet distinct from, clinical depression or generalized anxiety. Distinct differentiation facilitates proper treatment, whether it is self-

management support (skills-based) or psychotherapy or pharmacotherapy [8].

Far reaching implications of the undetected depression or anxiety in T2DM have clinical implications. The patients with changes in their mood or anxiety symptoms whose symptoms are not managed are more likely to fail reaching their glycemic targets, to experience acute decompensations and they acquire complications more rapidly. Mental ill health also lowers the rate of seeking preventive care and increases the rate of burden on the caregivers [9]. These trends translate into increased consumption and expenditure, repeat hospitalizations and complicated pharmacologic schedules at the health-system level that might have been alleviated by integrated interventions in the past. On the other hand, as systemic cases of mental-health needs are identified and addressed using collaborative-care models that integrate behavioral health into the diabetes services, mood symptoms as well as HbA1c tend to improve, which highlights the utility of treating mood and metabolism in-vivo [10].

Risk and response are also influenced contextually. The reasons why in most low- and middle-income contexts, the psychological comorbidity thrives unnoticed include rapid urbanization, nutrition and transportation settings that do not encourage healthy dietary choices, disparities in health literacy and access to mental-health professionals [11]. Cultural beliefs can prevent talking about the mood or anxiety, and time-constrained clinics can attend to the urgent biomedical issues. Such realities render more efficient screening, stepped-care plans and team-based strategies utilizing nurses, counselors, educators, and family supports particularly critical. They also emphasize the importance of practical study that can be applied in the real-life clinics as opposed to idealized conditions [12].

Although the situation is becoming acknowledged, there are still a number of knowledge gaps. There is a wide range of prevalences estimates across studies given the disparity in the instruments, cut-offs, populations and time of evaluation hence limiting comparability [13]. Relatively few longitudinal data elucidate the temporal directionality between deteriorating mood and the increase in HbA1c has been given. There is a requirement to have evidence on what combinations of interventions psychoeducation, problem-solving therapy, cognitive-behavioral strategies, medication optimization, digital supports, social-needs navigation provide long-term benefits in psychological as well as glycemic outcomes in a variety of health-care settings. Also, regular care does not agree on the frequency of screening, the based-line threshold of clinical referral, and the most appropriate method of monitoring the mental-health outcomes and glucose measures in quality dashboards [14].

It is in this context that the current study paper fulfills two connected objectives, namely, to describe the occurrence of depression and anxiety among adults with T2DM treated in modern clinics and to investigate the linkage between these mental-health factors and the process of glycemic regulation, as indicated by hemoglobin A1c. This work aims to provide informative avenues to guide practice toward early detection and combined treatment by foregrounding validated measurement, clinically interpretable, and implementation-ready implications. Finally, to decrease the combined cost of T2DM and common mental disorders, mental health will need to be considered, not as a supplement, but as a fundamental part of high-quality diabetes management one that is necessary to attain long-term glycemic control, complications prevention, and a better quality of life of patients and their families [15].

MATERIALS AND METHODS

This was a cross-sectional, clinic-based observational study conducted over 12 months at two tertiary-care outpatient diabetes clinics in Punjab, Pakistan from January 2024 till January 2025. Consecutive adults with established type 2 diabetes mellitus (T2DM) presenting for routine follow-up were screened for eligibility during regular clinic hours by trained studyers independent of treating clinicians. The study adhered to the STROBE recommendations for observational studies and followed the ethical principles of the Declaration of Helsinki. Written informed consent was obtained prior to enrollment; ethics approval was secured from the participating institutions' review boards.

Eligible participants were men and women aged 30–75 years with a documented diagnosis of T2DM for at least 12 months and the capacity to complete interviewer-administered questionnaires. We excluded individuals with type 1 diabetes or secondary diabetes; known severe psychiatric disorders (e.g., schizophrenia, bipolar disorder); current pregnancy; advanced renal failure on dialysis; acute intercurrent illness requiring hospitalization; or use of systemic glucocorticoids within the prior three months. Patients with cognitive impairment precluding valid consent or assessment were also excluded. To minimize spectrum bias, recruitment was consecutive across clinic days and strata of age and sex were monitored weekly; when necessary, under-represented strata were preferentially approached later in the recruitment window.

The planned sample size was 90. This size was justified a priori to detect a small-to-moderate association between depressive symptom severity and glycemic control. Assuming a correlation of $r \approx 0.30$ between Patient Health Questionnaire-9 (PHQ-9) score and hemoglobin A1c (HbA1c), two-sided $\alpha = 0.05$, and power $(1-\beta) = 0.80$, a minimum of 84 participants would be required. Allowing for ~7% incomplete data or ineligible cases after screening, the final target was increased to 90. This sample also provides ≥80% power to detect a mean HbA1c difference of 0.6–0.7 percentage points between participants with and without clinically significant depressive symptoms, assuming a standard deviation of 1.2–1.4%.

Data were collected in a single study visit. Sociodemographic information (age, sex, marital status, education, occupation, monthly household income) and clinical history (diabetes duration, hypoglycemia episodes in the prior three months, comorbid hypertension or dyslipidemia, smoking status, alcohol intake, current medications including antidepressants or anxiolytics, and prior psychiatric diagnoses) were obtained using a standardized case-report form. Anthropometry included height, weight, and waist circumference measured with calibrated equipment following WHO protocols; body mass index (BMI) was computed as kg/m^2. Blood pressure was measured in the seated position after five minutes of rest using an automated sphygmomanometer; the mean of two readings taken two minutes apart was recorded.

Glycemic control was indexed primarily by HbA1c, measured on the day of assessment from venous blood analyzed in the hospital laboratory using an NGSP-certified, IFCC-aligned high-performance method. Fasting plasma glucose (FPG) and lipid profile were obtained concurrently when feasible. Inflammatory markers (e.g., high-sensitivity C-reactive protein) and thyroid-stimulating hormone were extracted from the electronic record if measured within the preceding three months; otherwise, they were recorded as missing to avoid additional venipuncture burden.

Symptoms of depression were assessed with the PHQ-9, and anxiety with the

Generalized Anxiety Disorder-7 (GAD-7). Both instruments were administered in Urdu or English according to participant preference by trained study assistants who received uniform instruction and periodic inter-rater checks. For PHQ-9, total scores range from 0 to 27; for GAD-7, from 0 to 21. Following established cut-offs, scores ≥10 on either instrument were taken to indicate at least moderate symptom severity and used to define "screen-positive" depression or anxiety for categorical analyses. Because diabetes-related emotional burden can overlap with affective symptoms, the Diabetes Distress Scale-17 (DDS-17) was also administered to enable sensitivity analyses that differentiate distress from syndromal depression/anxiety; DDS-17 scores ≥2.0 signified clinically relevant distress. Participants with high suicidality scores on PHQ-9 item 9 or severe anxiety were immediately flagged to clinic physicians for same-day evaluation according to a predefined safety protocol.

The main outcome was HbA1c which was a continuous variable. Poor glycemic control was defined as a secondary categorical outcome, where HbA1C is 7.0% (53 mmol/mol) and above. PHQ-9 and GAD-7 scores were the primary exposures that were assessed continuously (per-point change) and categorically (≥10 vs <10). Covariates were prespecified and were age, sex, diabetes duration, BMI, systolic blood pressure, whether insulin is used, the number of oral antihyperglycemics, statin use, smoking status, and socioeconomic indicators. In the case where data allowed, further modifications were done based on hypoglycemia fear (yes/no) and DDS-17 to test the confounding by diabetes distress.

Quality assurance activities included the standardization of interviewer training, a two-fold data entry process with range checks, an audit of 10 percent of records after one month and anthropometric equipment calibration logs. Assays carried out by the laboratory were done with the ISO accredited facilities where both in-house and external quality controls were recorded after every month.

R (version 4.x) and Stata (version 18) were used to conduct the statistics. Continuous variables were described as mean +SD or median (IQR) according to the distribution; categorical variables were described in counts and percentages. T-tests or Mann-Whitney U tests were utilized in instances of continuous data and 0 2 tests in instances of categorical data in group comparisons.

The prevalence of screen positive depression and anxiety were estimated using 95 confidence intervals. Multivariate linear regression was used to assess the relationship between PHQ-9/GAD-7 and HbA1c, with sequential models: Model 1 unadjusted; Model 2 age, sex, and the duration of diabetes; Model 3 further adjusted by adding parameters of BMI, systolic blood pressure, insulin use, and the use of oral agents; Model 4 further adjusted by addition of smoking and socioeconomic indicator. The odds ratios (ORs) of having poor glycemic control (HbA1c ≥7.0%) were estimated using logistic regression according to depression/anxiety category. To measure multicollinearity, it was measured using the variance-inflation factors and the model fit was measured with the help of residual diagnostics and the HosmerLemeshow test was used. Complete-case analysis was used to deal with missing data <5% in any variable; and multiple imputation by chained equations (m=10) under the assumption of missing-at-random. Sensitivity tests were used to re-fitting models having removed the participants using the antidepressants/anxiolytics and those adjusted by DDS-17 in an effort of determining the contribution of diabetes distress and

depression/anxiety. The p of less than two-sided was taken as statistically significant.

Participant safety and referral procedures were integrated into clinic workflows. Individuals screening positive for moderate-to-severe depression or anxiety received brief counseling, printed educational materials, and a same-day appointment with the in-house physician for diagnostic evaluation and management or referral to mental-health services. De-identified data were stored on encrypted institutional servers with restricted access; a separate linkage file connecting study IDs to patient identifiers was stored offline and destroyed after database lock.

RESULTS

Out of 108 adults screened, 90 participants were enrolled after exclusions and refusals. The mean age was 54.8 ± 10.2 years (range 32–74). There was a near-equal distribution of sexes (43 males, 47.8%; 47 females, 52.2%). Median duration of T2DM was 9 years (IQR 5–13). Hypertension was present in 61.1% and dyslipidemia in 58.9%. One-fifth were active smokers, predominantly men (39.5% vs 6.4% in women). The mean BMI was 28.9 ± 4.5 kg/m², with obesity more frequent among women. Clinical and demographic features are summarized in Table 1.

Table-1: Demographic and clinical characteristics of study participants (N=90)

Variable	Overall (N=90)	Male (n=43)	Female (n=47)
Age, years, mean ± SD	54.8 ± 10.2	55.7 ± 9.8	54.0 ± 10.6
30–44 years, n (%)	18 (20.0)	10 (23.3)	8 (17.0)
45–59 years, n (%)	44 (48.9)	20 (46.5)	24 (51.1)
≥60 years, n (%)	28 (31.1)	13 (30.2)	15 (31.9)
Married, n (%)	76 (84.4)	37 (86.0)	39 (83.0)
Education ≥12 years, n (%)	38 (42.2)	20 (46.5)	18 (38.3)
Median household income, PKR (IQR)	80,000 (55,000–120,000)	82,000 (60,000–115,000)	78,000 (52,000–122,000)
Diabetes duration, years, median (IQR)	9 (5–13)	9 (5–14)	8 (5–12)
Hypertension, n (%)	55 (61.1)	25 (58.1)	30 (63.8)
Dyslipidemia, n (%)	53 (58.9)	24 (55.8)	29 (61.7)
Current smoker, n (%)	20 (22.2)	17 (39.5)	3 (6.4)
BMI, kg/m², mean ± SD	28.9 ± 4.5	27.9 ± 3.8	29.8 ± 4.9
Systolic BP, mmHg, mean ± SD	134.2 ± 15.6	135.6 ± 16.1	133.0 ± 15.2
Diastolic BP, mmHg, mean ± SD	81.4 ± 9.8	82.1 ± 9.5	80.8 ± 10.1
Insulin therapy, n (%)	32 (35.6)	13 (30.2)	19 (40.4)

The mean HbA1c was 7.9 ± 1.4%, with 55.1% of participants (49/89 complete cases) classified as having poor control (HbA1c ≥7.0%). Mean fasting plasma glucose was 156.3 ± 38.9 mg/dL. Lipid profiles showed mean LDL-C of 112.4 ± 31.8 mg/dL and low HDL-C levels (41.2 ± 9.7 mg/dL). Median triglycerides were elevated at 176 mg/dL (IQR 142–224). hs-CRP values were available for 82 participants, with a median of 2.4 mg/L (IQR 1.3–4.9), indicating low-grade inflammation. Full values are presented in Table 2.

Table-2: Glycemic and biochemical measures (N=90)

Parameter	Mean ± SD or Median (IQR)	Poor control prevalence
HbA1c, %	7.9 ± 1.4	49/89 (55.1%)
Fasting plasma glucose, mg/dL	156.3 ± 38.9	
Total cholesterol, mg/dL	188.6 ± 39.7	
LDL-C, mg/dL	112.4 ± 31.8	
HDL-C, mg/dL	41.2 ± 9.7	
Triglycerides, mg/dL	176 (142–224)	
hs-CRP, mg/L (n=82)	2.4 (1.3–4.9)	

The median PHQ-9 score was 7 (IQR 4–11), with 28 participants (31.1%) classified as screen-positive for depression (≥10). The median GAD-7 score was 6 (IQR 3–10); 25 participants (27.8%) had clinically significant anxiety. Dual morbidity was frequent: 16 participants (17.8%) screened positive for both depression and anxiety. Diabetes distress was also common, affecting 33 participants (36.7%). Detailed distribution is presented in Table 3.

Table-3: Prevalence and severity of depression, anxiety, and diabetes distress (N=90)

Measure	Category	n (%)
PHQ-9 (Depression)	Minimal (0–4)	26 (28.9)
	Mild (5–9)	36 (40.0)
	Moderate (10–14)	18 (20.0)
	Moderately severe (15–19)	7 (7.8)
	Severe (20–27)	3 (3.3)
	Screen-positive (≥10)	28 (31.1)
GAD-7 (Anxiety)	Minimal (0–4)	30 (33.3)
	Mild (5–9)	35 (38.9)
	Moderate (10–14)	17 (18.9)
	Severe (15–21)	8 (8.9)
	Screen-positive (≥10)	25 (27.8)
Overlap	Depression + Anxiety	16 (17.8)
DDS-17 (Distress ≥2.0)	Clinically relevant	33 (36.7)

Patients with depression (PHQ-9 ≥10) had a mean HbA1c of 8.5 ± 1.3%, significantly higher than those without depression (7.6 ± 1.3%; p<0.001). Similarly, anxious patients had HbA1c of 8.4 ± 1.2% versus 7.6 ± 1.4% in non-anxious (p=0.003). The prevalence of poor glycemic control (HbA1c ≥7.0%) was 78.6% in depressed vs 44.4% non-depressed, and 76.0% in anxious vs 48.5% non-anxious (Table 4).

Table-4: Glycemic control by depression and anxiety status (N=89 complete cases)

Status	HbA1c %, mean ± SD	HbA1c ≥7.0%, n (%)	p-value
Depression (PHQ-9 ≥10)	8.5 ± 1.3	22/28 (78.6)	<0.001
No depression	7.6 ± 1.3	27/61 (44.4)	
Anxiety (GAD-7 ≥10)	8.4 ± 1.2	19/25 (76.0)	0.003
No anxiety	7.6 ± 1.4	30/62 (48.5)	
Depression + Anxiety	8.7 ± 1.2	14/16 (87.5)	<0.001
Neither condition	7.4 ± 1.3	19/39 (48.7)	

Multivariable linear regression showed that each 5-point increase in PHQ-9 corresponded to a 0.32% higher HbA1c (95% CI 0.12–0.51; p=0.002), even after adjusting for age, sex, BMI, diabetes duration, smoking, and treatment variables. Each 5-point increase in GAD-7 predicted a 0.24% increase (95% CI 0.05–0.43; p=0.014). In combined models, depression retained statistical significance while anxiety attenuated, suggesting stronger independent effects of depressive symptoms. Logistic regression confirmed these trends: depression (PHQ-9 ≥10) conferred an adjusted OR = 2.41 (95% CI 1.03–5.61; p=0.042) for poor glycemic control. Anxiety (GAD-7 ≥10) showed a positive but nonsignificant association (adjusted OR = 2.09; 95% CI 0.90–4.88).

DISCUSSION

This cross-sectional study involved the investigation of depression and anxiety among adults with type 2 diabetes mellitus (T2DM) and tested their relevance to the glycemic control [12]. They show that nearly one-third of the participants had moderate-severe symptoms of depression and more than one-fourth of the participants had an anxious amount of anxiety that was clinically significant. Notably, the two illnesses were both related to the elevated values of the HbA1c and the high probability of inappropriate glycemic regulation with the depression as a stronger predictor. These results suggest that the psychosocial burden of patients with T2DM is enormous and that both models of care should be integrated, whereby metabolic and mental health are taken into account separately and jointly [13].

The rates of depression (31.1) and anxiety (27.8) among this group of individuals are in accordance with the international literature. Earlier meta-analysis have documented prevalence rates of depression among individuals with diabetes to be 25-35% which is rather high compared with the general population. Similarly, there is always high anxiety levels in T2DM and may be accompanied with depressive symptoms [14]. The level of comorbidity in this study (17.8) is indicative to the earlier results, in which comorbidity is the rule rather than the exception where diabetes population is concerned. This makes it challenging to diagnose and treat, because depressive and anxious symptoms

overlap with somatic manifestations of diabetes itself, e.g. fatigue and sleep disturbance, thus under-diagnosed in clinical practice [15].

The association between depression and glycemic control observed here (mean HbA1c difference of nearly 1 percentage point) is clinically significant. A 1% increase in HbA1c is known to elevate the risk of microvascular complications by approximately 37% and macrovascular events by 14%. Thus, the impact of depressive symptoms extends beyond psychological distress to tangible biomedical outcomes [16]. Mechanistically, depression impairs motivation, problem-solving capacity, and adherence to medication, diet, and physical activity. Biological pathways may also play a role: chronic stress and depressive states activate the hypothalamic–pituitary–adrenal axis, elevate cortisol levels, and promote inflammatory cytokine release, all of which can worsen insulin resistance and glycemic dysregulation [17].

Anxiety also correlated with elevated HbA1c, though its independent effect was weaker after adjustment. This attenuation may be due to the overlap between anxiety and depressive symptoms, as well as the possibility that anxiety influences short-term behaviors (such as avoidance of glucose monitoring) rather than sustained metabolic control. Nevertheless, its clinical relevance should not be minimized, since anxious individuals often report heightened diabetes distress, poor quality of life, and increased health-care utilization [18,19].

The high burden of diabetes distress (36.7%) found in this study deserves attention. Although distinct from depression or generalized anxiety, diabetes distress refers to the emotional strain of managing a complex, lifelong illness [20]. Prior studies suggest that distress is more common than clinical depression and can equally predict non-adherence and poor glycemic outcomes. In our sensitivity analyses, depressive symptoms remained significantly associated with HbA1c even after adjusting for distress, suggesting partially overlapping but distinct constructs. Clinicians should therefore screen for both conditions to guide appropriate interventions [21].

Our findings are consistent with a growing body of evidence from low- and middle-income countries, including South Asia, where social and health-system factors compound the psychological burden of diabetes [22]. Limited access to mental health professionals, cultural stigma surrounding psychiatric illness, and the financial cost of diabetes management are key contributors. In Pakistan specifically, where diabetes prevalence is rapidly rising, routine screening for psychological comorbidity remains rare. This study adds to the evidence base by quantifying the prevalence of depression and anxiety in a clinic-based sample and linking them to objective glycemic indices [23].

Several limitations must be acknowledged. The cross-sectional design precludes establishing causality or directionality; it is possible that poor glycemic control contributes to the onset of depression, or vice versa, or that both reinforce one another in a bidirectional loop [24]. The relatively small sample size (N=90) limits generalizability and statistical power, particularly for subgroup analyses. Self-reported measures such as PHQ-9 and GAD-7, though validated, may overestimate or underestimate clinical diagnoses. Furthermore, unmeasured confounders such as physical activity, dietary intake, social support, or sleep quality may have influenced the associations. Despite these limitations, the study provides robust, clinically meaningful findings that are consistent with international literature [25].

The implications are clear: routine integration of mental health screening into

diabetes clinics is essential. Simple tools such as the PHQ-9 and GAD-7 can be administered quickly and flag high-risk individuals for further evaluation. Management should be multidisciplinary, combining psychoeducation, cognitive-behavioral interventions, and when indicated, pharmacotherapy. Collaborative care models have demonstrated improvements in both psychological well-being and HbA1c. In resource-constrained settings, task-shifting to trained nurses or community health workers could be an effective strategy [17,21].

CONCLUSION

Depression and anxiety are highly prevalent among patients with type 2 diabetes mellitus and are strongly associated with poor glycemic control. Depression, in particular, demonstrated an independent relationship with higher HbA1c levels, even after adjustment for confounding factors. Co-occurrence of depression and anxiety was linked to the worst glycemic outcomes. These findings highlight the need for early detection and integrated management of psychological comorbidities in diabetes care. Routine mental health screening and collaborative care approaches could improve both emotional well-being and metabolic control, ultimately reducing the burden of complications and improving quality of life in this high-risk population.

Conflict of Interest:

The authors report no conflicts of interest.

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Authors' contributions:

M.G.: Study conception, methodology.
S.S.: Data collection and curation.
S.H.A.: Statistical analysis and interpretation.
A.A.: Critical revision and final approval.
All authors approved the final manuscript.

Data Availability Statement:

The data used in this study are available upon reasonable request from the corresponding author, subject to ethical and institutional guidelines.

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