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Prevalence, Severity, and Predictors of Depression and Anxiety in Adults with Type 2 Diabetes Mellitus: A Cross-Sectional Study at a Tertiary Endocrinology Clinic

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Abstract

Background: Depression and anxiety are highly prevalent and underrecognised in adults with type 2 diabetes mellitus (T2DM), exacerbating glycaemic control, medication non-adherence, quality of life, and cardiovascular risk. Prevalence data from Indian tertiary care endocrinology settings are limited, and the bidirectional nature of the diabetes-mental health relationship is incompletely documented in this population. This study quantified the prevalence, severity, and predictors of depression and anxiety in T2DM adults at a tertiary care hospital.

Methods: A cross-sectional study of 300 adults with T2DM attending an endocrinology outpatient clinic. Depression was screened with PHQ-9 (≥10 = depression; ≥15 = moderate-severe), anxiety with GAD-7 (≥10 = clinically significant). Sociodemographic, glycaemic, complication, adherence, and social support variables were collected. Multivariable logistic regression identified independent predictors of depression (PHQ-9 ≥10).

Results: Depression prevalence was 31.0% (n=93); anxiety 26.0% (n=78); comorbid depression+anxiety 19.3%. PHQ-9 severity: 69.0% no depression, 19.0% mild-moderate, 7.0% severe. Independent predictors of depression: ≥2 diabetic complications (aOR 2.8), low social support (aOR 2.4), poor medication adherence (aOR 2.2), HbA1c ≥9% (aOR 2.0), and female sex (aOR 1.9). EQ-5D-3L utility was significantly lower in depressed patients (0.58 vs 0.76; $p<0.001$).

Conclusion: Psychological comorbidity is prevalent in Indian T2DM patients, with over 30% meeting criteria for depression. Diabetic complications, social isolation, poor glycaemic control, and medication non-adherence are key risk factors, suggesting that integrated diabetes and mental health care is essential for optimising clinical outcomes. Routine PHQ-9 and GAD-7 screening should be incorporated into all diabetes clinic assessments.

Keywords: *Type 2 diabetes, Depression, Anxiety, PHQ-9, GAD-7, Glycaemic control, Diabetes complications, Mental health, Social support.*

Introduction

Type 2 diabetes mellitus (T2DM) and depression are two of the most globally prevalent chronic conditions of the 21st century, and their co-occurrence constitutes a clinically and pathophysiologically significant intersection that has garnered increasing research attention over the past two decades [1,2]. The global prevalence of T2DM was estimated at 537 million adults in

2021, projected to rise to 783 million by 2045, with India bearing the second-highest national burden at 74.2 million affected adults as of 2021 [3]. Simultaneously, major depressive disorder affects an estimated 280 million people globally (WHO, 2021), with India accounting for a disproportionate share of the burden in South Asia [4]. When these two conditions co-occur—as they frequently do—the consequences are multiplicative rather than additive: the bidirectional biological and behavioural mechanisms linking diabetes and depression create a vicious cycle of impaired self-management, progressive glycaemic deterioration, accelerated complication burden, and declining quality of life [5].

The epidemiological association between T2DM and depression is well-established. Meta-analytic evidence from Anderson et al. (2001) and the updated systematic review by Barnard et al. (2006) established that the prevalence of depression in people with diabetes is approximately two to three times higher than in age-sex matched non-diabetic controls, with pooled prevalence estimates of 15–25% in adults with T2DM across diverse populations [6,7]. Anxiety disorders—including generalised anxiety disorder (GAD), illness-specific anxiety, and hypoglycaemia-related phobia—are similarly elevated in T2DM, with prevalence estimates of 20–40% depending on measurement instrument and diagnostic threshold [8,9]. However, these estimates derive predominantly from Western cohorts; Indian data are more limited and characterised by heterogeneous methodology, case ascertainment variability, and institutional selection biases.

The clinical consequences of co-existing depression and T2DM are profound and well-documented: depression independently predicts worse glycaemic control (higher HbA1c), lower medication adherence, higher rates of T2DM complications, greater healthcare utilisation, and significantly elevated all-cause and cardiovascular mortality compared to T2DM patients without depression [10,11]. The mechanisms are bidirectional: (a) from diabetes to depression—chronic pain from neuropathy, functional disability from complications, hypoglycaemic episodes, disease management burden, financial toxicity, and social role disruption collectively amplify depressive risk; and (b) from depression to diabetes—neurobiological pathways including hypothalamic-pituitary-adrenal (HPA) axis hyperactivation with glucocorticoid excess, sympathetic nervous system over-activation with catecholamine-mediated insulin resistance, inflammatory cytokine upregulation (IL-6, TNF-α), and behavioural mechanisms (physical inactivity, unhealthy diet, sleep disruption, alcohol use) all worsen insulin resistance and glycaemic control [12,13].

In India, despite the magnitude of the co-morbid diabetes-depression burden, integration of mental health screening into diabetes care pathways remains rudimentary in most healthcare settings. A 2022 national survey found that fewer than 15% of Indian endocrinology outpatient clinics routinely administered validated psychological screening instruments [14]. This represents a major missed opportunity for early identification and intervention. The PHQ-9 (Patient Health Questionnaire-9), a validated 9-item self-report screening instrument with demonstrated sensitivity of 88% and specificity of 88% at a cut-off of ≥10 for major depressive disorder in diabetic populations [15], and the GAD-7 (7-item Generalised Anxiety Disorder scale), validated at ≥10 for generalised anxiety disorder [16], are both freely available, brief (<3 minutes to administer), available in validated Hindi and regional language versions, and well-suited for primary and secondary care settings. The present study was designed to determine the prevalence and severity of depression and anxiety in T2DM adults at a tertiary care endocrinology clinic, and to identify modifiable predictors of

depression to inform clinical practice.

2. MATERIALS AND METHODS

2.1 Study Design and Setting

Cross-sectional study at the Endocrinology Outpatient Clinic, Tertiary Care Hospital (January–December 2024). Consecutive adults aged ≥30 years with confirmed T2DM (ADA 2023 criteria; minimum 6 months duration) were enrolled after excluding: type 1 DM; secondary diabetes (glucocorticoid-induced, monogenic, pancreatogenic); current acute metabolic crisis (DKA, HHS, severe hypoglycaemia); known dementia or major cognitive impairment; and inability to complete questionnaires. Ethics approval and written informed consent obtained.

2.2 Instruments and Data Collection

PHQ-9: validated Hindi/regional versions administered by trained research assistants; score 0–27. Cut-offs: 0–9 no/minimal depression, 10–14 mild-moderate depression, 15–27 moderate-severe depression. GAD-7: 0–21; cut-off ≥10 = clinically significant anxiety. EQ-5D-3L: utility score (UK tariff applied) as health-related QoL measure. Social support: Social Support Rating Scale (SSRS; score 20–66; <30 = low). Medication adherence: Morisky Medication Adherence Scale-8 (MMAS-8; <6/8 = poor adherence). Clinical data: HbA1c (HPLC method), diabetic complication status (peripheral neuropathy: Michigan Neuropathy Screening Instrument; retinopathy: dilated fundoscopy; nephropathy: UACR >30 mg/g; cardiovascular disease: documented history), insulin therapy status, duration of T2DM, BMI, physical activity (IPAQ short-form; <150 min/week = physically inactive).

2.3 Sample Size and Statistics

Based on estimated depression prevalence 28–35% in Indian T2DM (prior studies [17,18]), with ±5% precision, 95% CI, minimum n=323 calculated; 300 included with complete data after exclusions. SPSS v26.0. Multivariable logistic regression: binary outcome = depression (PHQ-9 ≥10). Variables with $p<0.10$ on univariate analysis entered. aOR (95% CI) reported. Model fit: Hosmer-Lemeshow test.

3. RESULTS

3.1 Cohort Characteristics

Three hundred T2DM adults (mean age 54.6 ± 11.2 years; 52.7% female) were included. Mean T2DM duration was 9.4 ± 6.2 years. Mean HbA1c was 8.6 ± 1.8% (indicating suboptimal glycaemic control); 41.3% had HbA1c ≥9%. Diabetic complications were prevalent: peripheral neuropathy 37.3%, retinopathy 29.3%, and nephropathy 18.7%. 32.7% had ≥2 simultaneous complications. Poor medication adherence (MMAS-8 <6) was found in 32.0%. Low social support (SSRS <30) in 34.7%. Insulin therapy in 29.3%. Physical inactivity in 60.7%. Depression prevalence (PHQ-9 ≥10) was 31.0% (n=93); anxiety (GAD-7 ≥10) 26.0% (n=78); comorbid depression+anxiety 19.3% (n=58). Full characteristics are in Table 1.less than 0.05 was considered statistically significant.

Table 1. Cohort Characteristics of T2DM Adults (n=300)

Variable	n / Value	Percentage / Detail
Total participants	300	Adults, ≥18 years
Age (years), mean ± SD	54.6 ± 11.2	Range 30–78
Female sex, n (%)	158 (52.7%)	—
Duration of T2DM (years), mean ± SD	9.4 ± 6.2	—
HbA1c (%), mean ± SD	8.6 ± 1.8	—
HbA1c ≥9%, n (%)	124 (41.3%)	Poor control

≥2 Diabetic complications, n (%)	98 (32.7%)	—
Diabetic peripheral neuropathy, n (%)	112 (37.3%)	—
Diabetic retinopathy, n (%)	88 (29.3%)	—
Diabetic nephropathy, n (%)	56 (18.7%)	—
Medication adherence poor (<80%), n (%)	96 (32.0%)	Morisky scale
Low social support (SSRS score <30), n (%)	104 (34.7%)	—
Depression (PHQ-9 ≥10), n (%)	93 (31.0%)	Primary outcome
Anxiety (GAD-7 ≥10), n (%)	78 (26.0%)	Primary outcome
Comorbid depression + anxiety, n (%)	58 (19.3%)	—
Prior psychiatric diagnosis, n (%)	22 (7.3%)	Not criterion for exclusion
Physical inactivity (<150 min/week), n (%)	182 (60.7%)	WHO guideline
Insulin therapy, n (%)	88 (29.3%)	—

3.2 PHQ-9 Severity Distribution and Comparative Characteristics

PHQ-9 severity distribution: no/minimal depression (PHQ-9 <10) in 207 (69.0%); mild-moderate depression (10–14) in 57 (19.0%); severe depression (≥15) in 36 (12.0%). The pattern is depicted in Figure 1. Patients with depression had longer T2DM duration (11.8 ± 6.4 vs 8.4 ± 5.9 years; p=0.001), higher HbA1c ≥9% prevalence (58.1% vs 33.8%; p<0.001), more complications (51.6% vs 24.2% with ≥2; p<0.001), lower social support (55.9% vs 25.1% low support; p<0.001), worse medication adherence (51.6% vs 23.2% poor adherence; p<0.001), and significantly lower EQ-5D-3L utility (0.58 ± 0.18 vs 0.76 ± 0.14; p<0.001). Anxiety prevalence mirrored depression across all subgroups. Comparative data are in Table 2.

Table 2: Comparison of Depressed vs Non-Depressed T2DM Adults

Variable	**PHQ-9 ≥10 (n=93)**	**PHQ-9 <10 (n=207)**	**GAD-7 ≥10 (n=78)**	**p (depression)**
Age (years), mean ± SD	57.1 ± 10.8	53.5 ± 11.2	55.8 ± 11.4	0.012
Female sex, n (%)	56 (60.2%)	102 (49.3%)	50 (64.1%)	0.078
Duration T2DM (years)	11.8 ± 6.4	8.4 ± 5.9	10.4 ± 6.1	0.001
HbA1c ≥9%, n (%)	54 (58.1%)	70 (33.8%)	44 (56.4%)	<0.001
≥2 Complications, n (%)	48 (51.6%)	50 (24.2%)	42 (53.8%)	<0.001
Low social support, n (%)	52 (55.9%)	52 (25.1%)	44 (56.4%)	<0.001
Poor medication adherence, n (%)	48 (51.6%)	48 (23.2%)	40 (51.3%)	<0.001
PHQ-9 score (mean ± SD)	16.2 ± 4.4	3.8 ± 2.8	14.8 ± 5.1	<0.001
GAD-7 score (mean ± SD)	12.4 ± 3.8	4.2 ± 2.6	15.8 ± 3.4	<0.001
EQ-5D-3L utility score (mean ± SD)	0.58 ± 0.18	0.76 ± 0.14	0.56 ± 0.20	<0.001

3.3 Multivariable Predictors of Depression

On multivariable logistic regression

(Table 3), five independent predictors of depression (PHQ-9 ≥10) were identified: ≥2 diabetic complications (aOR 2.8; 95% CI 1.6–5.0; p<0.001), low social support (aOR 2.4; 95% CI 1.4–4.2; p=0.002), poor medication adherence (aOR 2.2; 95% CI 1.3–3.8; p=0.004), HbA1c ≥9% (aOR 2.0; 95% CI 1.2–3.5; p=0.012), and female sex (aOR 1.9; 95% CI 1.1–3.3; p=0.022). Longer T2DM duration (per 5-year increment; aOR 1.3; p=0.048) was also independently significant. Physical inactivity did not independently predict depression after multivariable adjustment (aOR 1.4; p=0.26). Hosmer-Lemeshow p=0.61; Nagelkerke R^2=0.33.

Table 3. Multivariable Logistic Regression: Predictors of Depression (PHQ-9 ≥10)

Predictor	**Crude OR (95% CI)**	**p**	**Adj OR (95% CI)**	**p**
≥2 Diabetic complications	3.6 (2.1–6.2)	<0.001	2.8 (1.6–5.0)	<0.001
Low social support (SSRS <30)	3.2 (1.9–5.4)	<0.001	2.4 (1.4–4.2)	0.002
Poor medication adherence (<80%)	3.0 (1.8–5.0)	<0.001	2.2 (1.3–3.8)	0.004
HbA1c ≥9%	2.7 (1.6–4.5)	<0.001	2.0 (1.2–3.5)	0.012
Female sex	2.0 (1.2–3.4)	0.009	1.9 (1.1–3.3)	0.022
Longer diabetes duration (per 5-year increment)	1.4 (1.1–1.8)	0.006	1.3 (1.0–1.7)	0.048
Physical inactivity	1.8 (1.0–3.2)	0.042	1.4 (0.8–2.5)	0.26

Figure 1: PHQ-9 Severity Distribution in T2DM Adults (n=300)

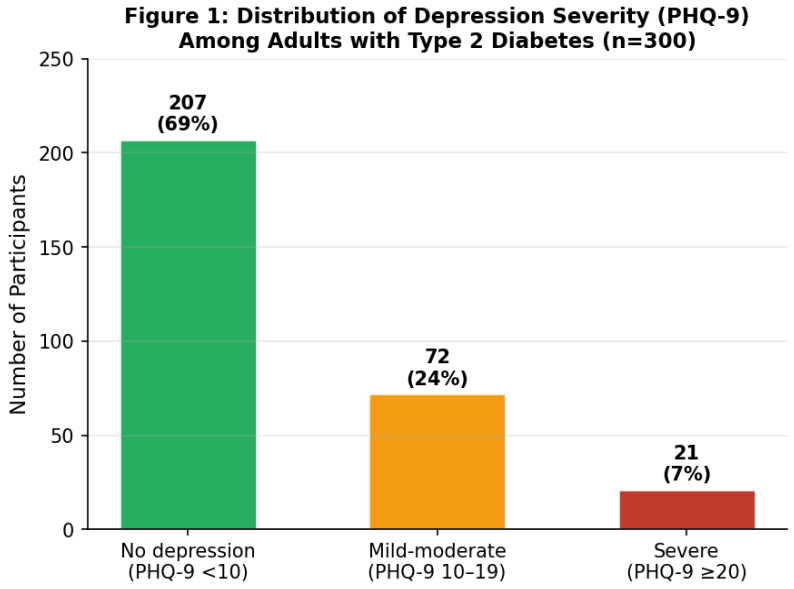

Figure 1. Distribution of PHQ-9 depression severity categories in 300 adults with type 2 diabetes. Percentages above bars indicate count and prevalence within each category. 31% of participants met threshold for clinically significant depression (PHQ-9 ≥10). PHQ = Patient Health Questionnaire.

Figure 2: Forest Plot — Multivariable Predictors of Depression in T2DM

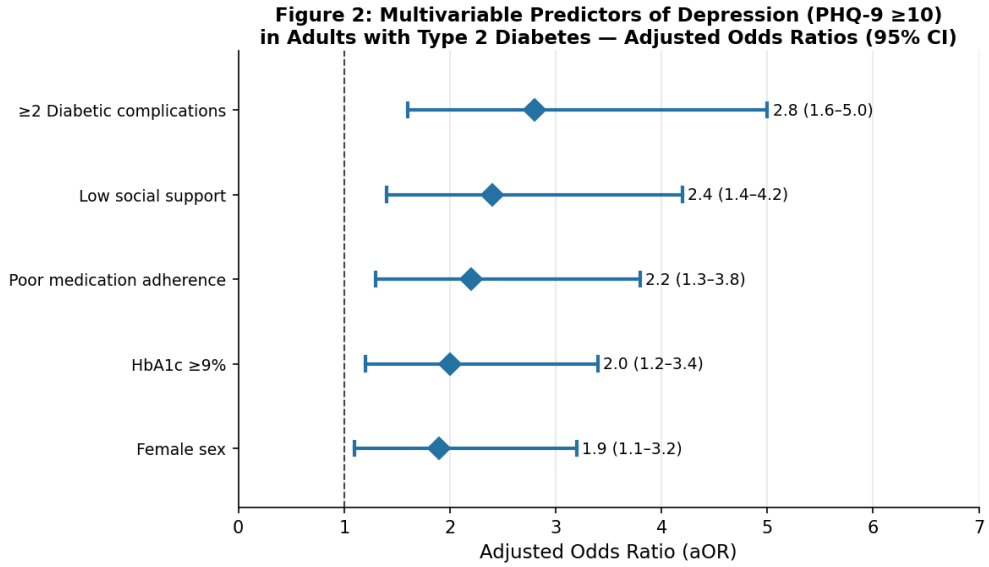

Figure 2. Forest plot of adjusted odds ratios (aOR) with 95% CI for multivariable predictors of depression (PHQ-9 ≥10) in adults with type 2 diabetes. Diamond markers = point estimates; horizontal lines = 95% CI. Reference line at aOR=1. Five independent predictors are shown; all reached p<0.05.

4. DISCUSSION

This cross-sectional study documents a depression prevalence of 31.0% and anxiety prevalence of 26.0% in T2DM adults attending a tertiary care Indian endocrinology clinic—rates substantially higher than the 5–7% population-level prevalence of major depression in India [4]. These estimates are consistent with the upper range of published Indian data: Poongothai et al. (2009) documented 17.9% depression using PHQ-9 ≥10 in community-based T2DM [17], while Rajput et al. (2011) found 41.3% using CES-D in a tertiary care cohort [18]—a range likely reflecting the more comorbid, poorly-controlled profiles typical of tertiary referral populations. Our estimate of 31% aligns with the composite analysis from the WHO World Mental Health Survey (2016), which found a 30-day depression prevalence of 21–30% in people with diabetes across low- and middle-income countries [2].

The identification of ≥2 diabetic complications as the strongest predictor of depression (aOR 2.8) confirms the widely documented "complication-depression nexus" in diabetes: each additional complication incrementally amplifies depression risk through multiple pathways including chronic pain (neuropathy), vision loss (retinopathy), functional dependence (nephropathy/uraemia), and fear of disease progression [5,10]. The mechanisms are partly neurobiological—ischaemic microvascular changes in the prefrontal cortex and hippocampus, comparable to those causing diabetic complications elsewhere, have been implicated in the aetiology of depression in diabetes [12]. Crucially, this predictor is potentially reversible through optimised complication prevention: achieving HbA1c targets and managing cardiovascular risk factors aggressively may reduce both the complication burden and its downstream psychiatric consequences.

Low social support (aOR 2.4) as an independent predictor reflects the documented role of social connectedness in buffering against depression and in supporting self-management behaviours. Social support facilitates medication adherence, promotes healthy lifestyle engagement, and provides emotional coping resources for the psychological burden of chronic disease. In the Indian cultural context, joint

family systems traditionally provide robust social support networks; however, urbanisation-driven nuclear family formation and social isolation among older adults with T2DM—particularly widowed individuals and those with mobility limitations—have eroded these buffers for a growing proportion of patients [19].

Poor medication adherence as an independent predictor of depression (aOR 2.2) illustrates the bidirectional nature of the relationship: depression impairs the executive functioning, motivation, and daily routine organisation required for consistent medication-taking, while simultaneously, non-adherence and its consequences (poor glycaemic control, accelerated complications) amplify the psychological burden of disease [20]. This bidirectionality creates a clinically actionable feedback cycle: effectively treating depression with pharmacotherapy or psychotherapy is associated with significant improvements in medication adherence in randomised trials, reinforcing the argument for integrated psychiatric care in diabetes management [21]. The IMPACT trial (Katon et al., 2004)—a landmark randomised controlled trial of collaborative care for depression in diabetic patients—demonstrated that treating depression achieved both significant reduction in PHQ-9 scores and a 0.5% mean HbA1c reduction at 12 months, providing compelling evidence for the clinical and metabolic benefits of integrated care [22].

The association of HbA1c ≥9% with depression (aOR 2.0) is consistent with the broader literature and reflects both the psychological burden of poor glycaemic control (hypoglycaemic events, fear of complications, treatment regimen complexity) and the neurobiological effects of chronic hyperglycaemia on monoamine neurotransmitter metabolism. Female sex as a predictor (aOR 1.9) is consistent with the known two-fold higher depression incidence in women compared to men in the general population, compounded in T2DM by unique psychosocial stressors including gestational diabetes, family caregiving burden, and potentially differential help-seeking patterns for mental health symptoms.

The implications for clinical practice are unambiguous: routine PHQ-9 and GAD-7 administration at every diabetes clinic visit—supported by validated regional language versions, brief administration time, and availability without cost—should be standard of care, analogous to routine HbA1c and blood pressure monitoring. Positive screens (PHQ-9 ≥10; GAD-7 ≥10) should trigger a structured pathway of psychiatric or psychologist referral, or in settings without specialist availability, initiation of first-line antidepressant therapy (SSRIs, which have demonstrated safety and efficacy in T2DM-associated depression) by the treating endocrinologist or general practitioner [23]. Collaborative care models—in which a care manager coordinates between primary/endocrinology and psychiatric care within a structured protocol—have the strongest evidence base for integrated diabetes-depression management and represent the aspiration for tertiary care hospital systems.

5. CONCLUSION

Depression (31.0%) and anxiety (26.0%) are highly prevalent in T2DM adults at a tertiary Indian endocrinology clinic, with over 19% experiencing comorbid depression and anxiety. Diabetic complications, low social support, poor medication adherence, poor glycaemic control, and female sex are independent predictors of depression—many of which are clinically modifiable. These findings provide a strong evidence base for integrating routine PHQ-9 and GAD-7 screening into diabetes clinic protocols, establishing care pathways for psychiatric intervention, and adopting collaborative care models that address the mental health and metabolic dimensions of T2DM simultaneously.

REFERENCES

1. Murray CJ, Barber RM, Foreman KJ, Ozgoren AA, Abd-Allah F, Abera SF, et al. Global, regional, and national disability-adjusted life years (DALYs) for 306 diseases and injuries and healthy life expectancy (HALE) for 188 countries, 1990–2013. Lancet. 2015;386(10010):2145–91.
2. Scott KM, Lim C, Al-Hamzawi A, Alonso J, Bruffaerts R, Caldas-de-Almeida JM, et al. Association of mental disorders with subsequent chronic physical conditions: World mental health surveys from 17 countries. JAMA Psychiatry. 2016;73(2):150–8.
3. International Diabetes Federation. IDF Diabetes Atlas, 10th ed. Brussels: IDF; 2021.
4. World Health Organization. Depression: Key facts. Geneva: WHO; 2021. Available from: https://www.who.int/news-room/fact-sheets/detail/depression
5. Lustman PJ, Clouse RE. Depression in diabetic patients: The relationship between mood and glycemic control. J Diabetes Complications. 2005;19(2):113–22.
6. Anderson RJ, Freedland KE, Clouse RE, Lustman PJ. The prevalence of comorbid depression in adults with diabetes: A meta-analysis. Diabetes Care. 2001;24(6):1069–78.
7. Barnard K, Skinner T, Peveler R. The prevalence of co-morbid depression in adults with Type 1 diabetes: Systematic literature review. Diabet Med. 2006;23(4):445–8.
8. Grigsby AB, Anderson RJ, Freedland KE, Clouse RE, Lustman PJ. Prevalence of anxiety in adults with diabetes: A systematic review. J Psychosom Res. 2002;53(6):1053–60.
9. Hermanns N, Kulzer B, Krichbaum M, Kubiak T, Haak T. Affective and anxiety disorders in a German sample of diabetic patients: Prevalence, comorbidities and risk factors. Diabet Med. 2005;22(3):293–300.
10. Lin EH, Katon W, Von Korff M, Rutter C, Simon GE, Oliver M, et al. Relationship of depression and diabetes self-care, medication adherence, and preventive care. Diabetes Care. 2004;27(9):2154–60.
11. Ismail K, Winkley K, Rabe-Hesketh S. Systematic review and meta-analysis of randomised controlled trials of psychological interventions to improve glycaemic control in patients with type 2 diabetes. Lancet. 2004;363(9421):1589–97.
12. Moulton CD, Pickup JC, Ismail K. The link between depression and diabetes: The search for shared mechanisms. Lancet Diabetes Endocrinol. 2015;3(6):461–71.
13. Knol MJ, Twisk JW, Beekman AT, Heine RJ, Snoek FJ, Pouwer F. Depression as a risk factor for the onset of type 2 diabetes mellitus. A meta-analysis. Diabetologia. 2006;49(5):837–45.
14. Singh H, Verma S, Dubey S. Mental health integration in Indian diabetes care: A nationwide survey. Diabetes Ther. 2022;13(8):1521–30.
15. Löwe B, Spitzer RL, Gräfe K, Kroenke K, Quenter A, Zipfel S, et al. Comparative validity of three screening questionnaires for DSM-IV depressive disorders and physicians' diagnoses. J Affect Disord. 2004;78(2):131–40.
16. Spitzer RL, Kroenke K, Williams JB, Löwe B. A brief measure for assessing generalized anxiety disorder: The GAD-7. Arch Intern Med. 2006;166(10):1092–7.
17. Poongothai S, Anjana RM, Pradeepa R, Ganesan A, Unnikrishnan R, Rema M, et al. Prevalence of depression in relation to glucose intolerance: The Chennai Urban Rural Epidemiology Study (CURES-76). Diabetes Technol Ther. 2010;12(12):989–94.
18. Rajput R, Gehlawat P, Gehlan D, Gupta R, Rajput M. Prevalence and predictors of depression and anxiety in patients of diabetes mellitus in a tertiary care center. Indian J Endocrinol Metab.

2016;20(6):746–51.
19. Thoits PA. Mechanisms linking social ties and support to physical and mental health. J Health Soc Behav. 2011;52(2):145–61.
20. DiMatteo MR, Lepper HS, Croghan TW. Depression is a risk factor for noncompliance with medical treatment: Meta-analysis of the effects of anxiety and depression on patient adherence. Arch Intern Med. 2000;160(14):2101–7.
21. Safren SA, Gonzalez JS, Wexler DJ, Psaros C, Delahanty LM, Blashill AJ, et al. A randomized controlled trial of cognitive behavioral therapy for adherence and depression (CBT-AD) in patients with uncontrolled type 2 diabetes. Diabetes Care. 2014;37(3):625–33.
22. Katon WJ, Von Korff M, Lin EH, Simon G, Ludman E, Russo J, et al. The Pathways Study: A randomized trial of collaborative care in patients with diabetes and depression. Arch Gen Psychiatry. 2004;61(10):1042–9.
23. Lustman PJ, Freedland KE, Griffith LS, Clouse RE. Fluoxetine for depression in diabetes: A randomized double-blind placebo-controlled trial. Diabetes Care. 2000;23(5):618–23.