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ORIGINAL RESEARCH

A Clinical-Metabolic Prediction Model for Suicidal Behaviors Risk Stratification in First-Admission Major Depressive Disorder: A Cross-Sectional Analysis

Lei Geng[1,2], Sheng Zhang[1,2], Jun Yan[1,2], Runlan Wang[1,2]

[1]Department of Psychiatry, Wuhan Mental Health Center, Wuhan, People's Republic of China; [2]Department of Psychiatry, Wuhan Hospital for Psychotherapy, Wuhan, People's Republic of China

Correspondence: Runlan Wang, Department of Psychiatry, Wuhan Mental Health Center, No. 89, Gongnongbing Road, Wuhan, People's Republic of China, Email rubywangrunlan@qq.com

Background: The clinical characteristics and biomarkers of suicidal behaviors (SB) in first-hospitalized patients with Major Depressive Disorder (MDD) remain poorly understood. This study aimed to investigate the prevalence, clinical correlates, and metabolic disturbances of SB in first-admission MDD patients in China, integrating psychosocial and biological markers to establish a predictive model.

Methods: A cross-sectional analysis was conducted on 981 first-admission MDD inpatients. Sociodemographic data, clinical symptom severity (17-item Hamilton Depression Rating Scale [HAMD-17], 14-item Hamilton Anxiety Rating Scale [HAMA-14], PANSS positive subscale [PSS], Clinical Global Impression–Severity Index [CGI-SI]), and metabolic parameters (lipid profile, fasting glucose, thyroid function) were collected. SB was assessed using the Columbia-Suicide Severity Rating Scale (C-SSRS). Binary logistic regression and ROC analysis identified correlates and model performance.

Results: The prevalence of SB was 13.46% (132/981). SB patients exhibited significantly higher psychotic symptoms, anxiety severity, and illness severity, along with elevated waist circumference (WC), diastolic blood pressure (DBP), total cholesterol (TC), and thyroid-stimulating hormone (TSH). Logistic regression identified HAMA (OR=1.72, 95% CI=1.25–2.37), PSS (OR=1.58, 95% CI=1.13–2.21), CGI-SI (OR=1.45, 95% CI=1.08–1.95), and TC (OR=1.32, 95% CI=1.04–1.68) as factors independently associated with SB (all $p<0.05$). The combined model of PSS, HAMA, and CGI-SI demonstrated strong discriminative accuracy (AUC=0.87, 95% CI: 0.83–0.91). Linear regression further linked HAMA scores to SB severity (β=0.21, $p=0.029$).

Conclusion: SB in first-hospitalized MDD patients correlates with anxiety symptoms, psychotic features, and metabolic dysregulation. A multidimensional model integrating clinical and metabolic indicators is associated with high-risk individuals, supporting targeted prevention strategies.

Keywords: major depressive disorder, suicidal behaviors, clinical prediction model, metabolic dysregulation, anxiety symptoms

Introduction

Suicide is a critical global public health issue. The World Health Organization estimates over 800,000 annual suicide deaths worldwide.[1,2] It ranks as the 18th leading cause of death across all ages, but is the second leading cause among those aged 15–29, surpassed only by unintentional injuries.[3] Alarmingly, one suicide occurs approximately every 40 seconds.[3] Suicide rates are high in many nations.[4] The United States Centers for Disease Control and Prevention reported in 2018 that the US age-adjusted suicide rate rose 33% from 1999 to 2017.[5] Critically, global rates are likely significantly underestimated. Some suicides may be misclassified (eg, as undetermined causes), potentially making actual figures 10–50% higher than reported.[6,7] Suicide deaths represent merely the tip of the iceberg: non-fatal attempts are estimated to be 10–20 times more frequent, and suicidal ideation without action is vastly more common than completed suicide.[8–10]

Suicidal behaviors (SB) arises from complex interactions between psychiatric illness, environmental stressors, and sociocultural determinants.[11] Among psychiatric disorders, Major Depressive Disorder (MDD) emerges as the most

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potent predictor, implicated in >90% of suicide fatalities.[12,13] MDD, characterized by profound disability and high recurrence rates,[14–16] diminishes quality of life,[17] disrupts occupational functioning,[18] and exacerbates socioeconomic burdens,[19] and also significantly elevates the risk of suicide.[20] Despite therapeutic advances, persistent SB vulnerability during antidepressant treatment reveals critical shortcomings in current risk stratification paradigms.[21] Specifically, the inability to identify subgroups at high risk of SB that are resistant to conventional interventions highlights the need to develop refined predictive models that integrate biomarkers.

Current literature predominantly investigates SB prevalence in mixed outpatient/inpatient cohorts or recurrent MDD populations,[22,23] with limited focus on first-hospitalized patients—a high-risk subgroup requiring urgent intervention. This gap is significant because the initial hospitalization often represents the first major clinical presentation and intervention point for severe MDD. Individuals experiencing their first severe depressive episode necessitating hospitalization may present distinct clinical profiles, biological correlates, and vulnerability to SB compared to those with chronic or recurrent illness.[24–26] Factors such as the acute onset of severe symptoms, potential treatment naivety, and the profound psychological impact of a first psychiatric hospitalization could uniquely shape SB risk trajectories.[26,27] Understanding SB determinants at this pivotal juncture is crucial for developing effective early intervention strategies. Furthermore, while metabolic dysregulation and thyroid dysfunction are increasingly recognized as SB correlates,[28] potential neurobiological mechanisms include: dyslipidemia and visceral adiposity may promote neuroinflammation,[28,29] serotonergic dysfunction,[30] and HPA axis hyperactivity;[31] elevated TSH may impair monoaminergic neurotransmission,[32] reduce GABAergic inhibition,[33] and diminish neurotrophic support.[34] These disturbances likely synergize with psychopathology to exacerbate suicide risk through convergent effects on prefrontal-limbic circuitry.[35,36] Nevertheless, clinically applicable biomarkers remain elusive despite compelling pathophysiological links.[28,37]

This study addresses these gaps through three primary objectives: 1) establishing SB prevalence in first-admission MDD patients within China's Han population; 2) identifying clinical and metabolic correlates of SB occurrence and severity; 3) constructing a multidimensional prediction model integrating psychometric and biological markers. By focusing on treatment-naïve inpatients during acute-phase MDD, our findings aim to enhance early risk detection and inform targeted prevention strategies.

Study Design and Participants

The sample size was determined using the formula:

$$n = \frac{Z^2 P(1-P)}{d^2}$$

In this equation, n stands for the sample size, Z refers to the Z-score associated with the desired confidence level (1.96 for a 95% confidence interval), P indicates the estimated prevalence or proportion (chosen as 0.2 based on the rate of dyslipidemia in the general Chinese population), and d is the acceptable margin of error (set at 0.05). Based on these values, the calculated sample size came to 246 individuals.

Participants were consecutively recruited from all first-admission MDD inpatients meeting inclusion criteria at Wuhan Mental Health Center (It is the largest public institution with psychiatric specialty in central China, visited by the patient population throughout the region) between July 2017 and August 2022. All eligible patients during this period were approached for enrollment, and those providing informed consent were included in the study. Diagnosis of MDD was confirmed through structured clinical interviews aligned with the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) criteria (Figure 1: Participant Flow Diagram).

Inclusion required: (1) no prior psychiatric hospitalization history; (2) age 18–60 years; (3) male or female; (4) Han Chinese ethnicity; (5) acute-phase depressive severity (HAMD-17 score ≥24).

Exclusion criteria encompassed: (1) pregnancy/lactation; (2) substance use disorders; (3) severe medical comorbidities or personality disorders; (4) diabetes mellitus diagnosis; (5) current use of psychotropic medications or drugs affecting metabolic/endocrine parameters; (6) cognitive/behavioral impairment precluding assessment compliance.

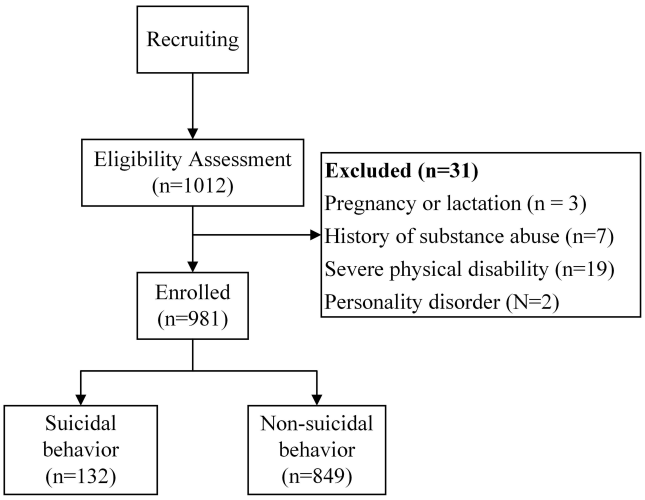

Figure 1 Study Flowchart.

The study was approved by the Ethics Committee of Wuhan Mental Health Center. All participants provided written informed consent prior to their involvement in the research, in accordance with the principles of the Declaration of Helsinki.

Clinical and Biochemical Assessments

Demographic profiles (age, sex, marital status), illness characteristics (age of onset, disease duration), and treatment history were systematically recorded. Within 24 hours post-admission, certified psychiatrists administered validated instruments:

Depressive Severity

Assessed using the 17-item Hamilton Depression Rating Scale (HAMD-17), quantifying symptoms via clinician-administered ratings (0–4/0–2 per item; total range 0–52).

Anxiety Severity

Measured with the 14-item Hamilton Anxiety Rating Scale (HAMA-14) evaluating somatic and psychic symptoms on 0–4 scales (total range 0–56).

Psychotic Features

Evaluated by the Positive subscale (P1–P7) of the PANSS (PSS) scoring seven psychotic symptoms on 1–7 severity dimensions (subscale range 7–49).

Global Illness Severity

Rated via the Clinical Global Impression–Severity Index (CGI-SI), a clinician-determined 7-point global metric (1=normal to 7=extremely ill).

Fasting venous blood samples collected on Day 2 were analyzed for:

Thyroid Function

Thyroid-stimulating hormone (TSH), free triiodothyronine (FT_3), free thyroxine (FT_4).

Metabolic Indices

Total cholesterol (TC), triglycerides (TG), high-/low-density lipoprotein cholesterol (HDL-C/LDL-C), fasting blood glucose (FBG).

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Anthropometrics

Waist circumference (WC), body mass index (BMI), systolic/diastolic blood pressure (SBP/DBP).

Suicidal Behaviors Evaluation

Current SB (past 30 days) was ascertained through semi-structured interviews with patients and corroborated by family members/guardians. The clinician-administered Columbia Suicide Severity Rating Scale (C-SSRS) quantified SB severity through six ordinal levels (passive ideation to lethal attempts) in SB-positive cases. The C-SSRS was administered by raters trained to reliability standards (kappa>0.80) through the official Chinese C-SSRS certification program. To ensure validity: All interviews followed the standardized C-SSRS structured guide.[38]

Trained psychiatrists (≥5 years experience) administered all instruments following standardized protocols, with inter-rater reliability maintained at $\kappa > 0.80$ through monthly calibration sessions.

Data Analysis

Categorical variables were expressed as frequencies (%), continuous variables as mean±SD or median (IQR) based on distribution normality (Shapiro–Wilk test). Between-group comparisons employed χ^2-tests for categorical data, independent *t*-tests for normally distributed variables, and Mann–Whitney *U*-tests for nonparametric measures. Variables with $p < 0.10$ in univariate analyses were entered into binary logistic regression (backward elimination) to identify SB correlates. Model discrimination was evaluated via receiver operating characteristic (ROC) curves, with area under the curve (AUC) >0.70 considered clinically informative.[39,40] SB severity correlates were analyzed through multiple linear regression. Significance was set at two-tailed $p<0.05$. Analyses utilized SPSS 27 and GraphPad Prism 8.4.3.

Results

Clinical and Metabolic Profile of SB Subgroups

A total of 132 cases of MDD accompanied by SB were recorded, accounting for 13.46% (132/981) of the total. Comparative analysis revealed substantial disparities between MDD patients with SB (n = 132) and non-SB counterparts (n = 849). The SB cohort demonstrated elevated psychopathological severity across multiple domains: PANSS positive symptom scores were markedly higher ($Z = -14.49$, $p < 0.001$), as were anxiety symptoms (HAMA: $Z = -12.43$, $p < 0.001$) and global illness severity (CGI-SI: $Z = -11.76$, $p<0.001$). Metabolic dysregulation was prominent in SB patients, evidenced by increased TSH ($Z = -6.59$, $p < 0.001$), WC ($Z = -2.15$, $p = 0.032$), FBG ($t = -3.98$, $p < 0.001$), and TC ($Z = -7.35$, $p < 0.001$). Notably, SB patients exhibited shorter median disease duration (9.5 vs 10.5 months, $p=0.004$) (Table 1).

Table 1 The Demographic and General Clinical Data in Different Clinical Subgroups

Index	Total Patients (n = 981)	SB (n = 132)	Non-SB (n = 849)	$t/Z/\chi^2$	*p* - value
Age - years	34 (24, 46)	38 (26, 50)	34 (23, 45)	−2.72	0.006*
Onset age - years	33 (22, 44)	37 (25, 48)	32 (22, 43)	−3.04	0.002*
Course of disease - months	10.5 (8, 12.5)	9.5 (6.25, 12)	10.5 (8.5, 13)	−2.87	0.004*
Gender				0.31	0.579
Male	333, 33.94%	42, 31.82%	291, 32.55%		
Female	648, 66.06%	90, 68.18%	558, 67.45%		
Marital status - (n, %)				1.62	0.203
Unmarried	307, 31.29%	35, 26.52%	272, 32.04%		
Married	674, 68.71%	97, 73.48%	577, 67.96%		
Treatment history				2.12	0.146
Yes	636, 64.83%	93, 70.45%	543, 63.96%		
NO	345, 35.17%	39, 29.55%	306, 36.04%		

(*Continued*)

Table 1 (Continued).

Index	Total Patients (n = 981)	SB (n = 132)	Non-SB (n = 849)	$t/Z/\chi^2$	*p* - value
Educational background				1.08	0.300
High school and below	683, 69.62%	97, 73.48%	586, 69.02%		
Bachelor and above	298, 30.38%	35, 26.51%	263, 30.98%		
PSS	7 (7, 7)	10 (7, 21)	7 (7, 7)	−14.49	<0.001*
HAMD	29 (27, 31)	32.23±3.51	29 (27, 31)	−9.75	<0.001*
HAMA	20 (18, 22)	25 (21, 28)	19 (17, 22)	−12.43	<0.001*
CGI-SI	6 (5, 6)	7 (6, 7)	6 (5, 6)	−11.76	<0.001*
TSH - uIU/mL	3.98±2.47	6.40±3.97	3.60±1.89	−7.94	<0.001*
FT_3 - pmol/L	4.9±0.69	4.93±0.65	4.90±0.70	−0.51	0.613
FT_4 - pmol/L	16.78±3.04	16.95±3.18	16.75±3.01	−0.68	0.497
WC - cm	80 (74, 86)	81.48±8.73	80 (74, 85.5)	−2.15	0.032*
FBG - mmol/L	5.26±0.63	5.46±0.8	5.23±0.59	−3.98	<0.001*
TG - mmol/L	2.11±1.00	2.11±0.99	2.11±1.00	0.05	0.962
HDL-C - mmol/L	1.32±0.23	1.28±0.23	1.32±0.23	1.84	0.066
SBP - mmHg	116.39±11.15	123.09±13.41	115.35±10.38	−6.35	<0.001*
DBP - mmHg	74.62±6.83	78.44±8.71	74.03±6.29	−5.60	<0.001*
TC - mmol/L	4.79±0.92	5.32±0.96	4.70±0.89	−7.35	<0.001*
LDL-C - mmol/L	2.67±0.74	2.84±0.83	2.64±0.72	−2.85	0.004*
BMI - kg/m^2	24.15 (23.22, 25.36)	24.21 (23.40, 25.38)	24.14 (23.2, 25.36)	−0.76	0.450

Note: *p<0.05.

Abbreviations: PSS, Positive symptom subscale; HAMD, Hamilton Depression Scale score; HAMA, Hamilton Anxiety Scale Score; CGI-SI, Clinical Global Impression Scale-Severity of Illness; TSH, Thyroid stimulating hormone; FT_3, Free triiodothyronine; FT_4, Free tetraiodothyronine; WC, waist circumference; FBG, fasting blood glucose; TG, triglycerides; HDL-C, high density lipoprotein cholesterol; SBP, systolic blood pressure; DBP, diastolic blood pressure; TC, total cholesterol; LDL-C, low density lipoprotein cholesterol; BMI, Body mass index.

Multivariate Predictors of Suicidal Behaviors

Binary logistic regression identified six factors independently associated with SB (Table 2). Anxiety severity (HAMA: OR=1.37, 95% CI=1.25–1.51) and psychotic features (PSS: OR=1.08, 95% CI=1.01–1.14) showed dose-dependent associations with SB risk. Clinical global severity (CGI-SI: OR=3.52, 95% CI=2.38–5.22) emerged as the strongest predictor, while metabolic parameters including WC (OR=1.04, 95% CI=1.01–1.07), DBP (OR=1.04, 95% CI=1.01–1.08), and TC (OR=1.51, 95% CI=1.07–2.12) contributed additively. Paradoxically, higher LDL-C levels reduced SB likelihood (OR=0.58, 95% CI=0.40–0.84).

Table 2 Binary Logistic Regression Analyses of Determinants of SB in MDD Patients

	Coefficients	Std. Error	Wald	*p*-value	95% CI for EXP (B)		
	B				Exp (B)	Lower	Upper
Constant	−20.05	2.44	67.36				
PSS	0.07	0.03	5.99	0.014*	1.08	1.01	1.14
HAMD	−0.11	0.06	3.40	0.065	0.89	0.79	1.01
HAMA	0.32	0.05	40.14	<0.001*	1.37	1.25	1.51
CGI-SI	1.26	0.20	39.40	<0.001*	3.52	2.38	5.22
WC-cm	0.03	0.02	5.32	0.021*	1.04	1.01	1.07
DBP-mmHg	0.04	0.02	5.17	0.023*	1.04	1.01	1.08
TC - mmol/L	0.41	0.17	5.56	0.018*	1.51	1.07	2.12
LDL-C - mmol/L	−0.55	0.19	8.50	0.004*	0.58	0.40	0.84

Note: *p<0.05.

Abbreviations: PSS, Positive symptom subscale; HAMD, Hamilton Depression Scale score; HAMA, Hamilton Anxiety Scale Score; CGI-SI, Clinical Global Impression Scale-Severity of Illness; WC, waist circumference; DBP, diastolic blood pressure; TC, total cholesterol; LDL-C, low density lipoprotein cholesterol.

Table 3 ROC Analysis of Factors Influencing SB

Variables	AUC	Std. Error	*p*-value	95% CI	
				Lower	Upper
PSS	0.76	0.03	<0.001*	0.71	0.82
HAMA	0.83	0.02	<0.001*	0.80	0.87
CGI-SI	0.79	0.02	<0.001*	0.75	0.84
WC-cm	0.56	0.03	0.032*	0.50	0.61
DBP-mmHg	0.64	0.03	<0.001*	0.58	0.69
TC- mmol/L	0.69	0.02	<0.001*	0.64	0.74
LDL-C - mmol/L	0.58	0.03	0.005*	0.52	0.63

Note: *$p<0.05$.
Abbreviations: PSS, Positive symptom subscale; HAMA, Hamilton Anxiety Scale Score; CGI-SI, Clinical Global Impression Scale-Severity of Illness; WC, waist circumference; DBP, diastolic blood pressure; TC, total cholesterol; LDL-C, low density lipoprotein cholesterol.

Discriminative Capacity and Severity Associations

ROC analysis demonstrated differential discriminatory capacity among SB-associated factors (Table 3). The HAMA scale achieved superior performance (AUC=0.83, 95% CI=0.80–0.87), followed by CGI-SI (AUC=0.79) and PSS (AUC=0.76). A composite model integrating these three clinical measures yielded exceptional classification accuracy (AUC=0.87, 95% CI=0.83–0.91), significantly outperforming isolated metabolic parameters (AUC range: 0.56–0.69) (Figure 2). Linear regression of SB severity (C-SSRS scores) identified HAMA as a positive contributor (β=0.21, p=0.029) and TC as a mitigating factor (β=−0.98, p=0.032), accounting for 18.7% of severity variance (adjusted R^2=0.187) (Table 4).

Discussion

This investigation provides novel insights into SB among first-hospitalized MDD patients, addressing critical gaps in characterizing this high-risk population. Four principal findings emerge: (1) SB prevalence of 13.46% in treatment-naïve inpatients; (2) distinct psychopathological and metabolic profiles in SB subgroups; (3) validated discriminative utility of a multidimensional clinical model; (4) anxiety severity as an independent correlate of SB intensity. These findings could inform risk stratification for SB and support targeted prevention strategies in high-risk clinical populations.

Current literature extensively documents the prevalence of SB in patients with MDD. A large-scale meta-analysis reveals a lifetime SB prevalence of 23.7% among MDD patients.[41] For individuals experiencing their first MDD episode and receiving outpatient care, SB detection rates range from 17.3% to 20.1%,[42,43] comparable to hospitalization-based

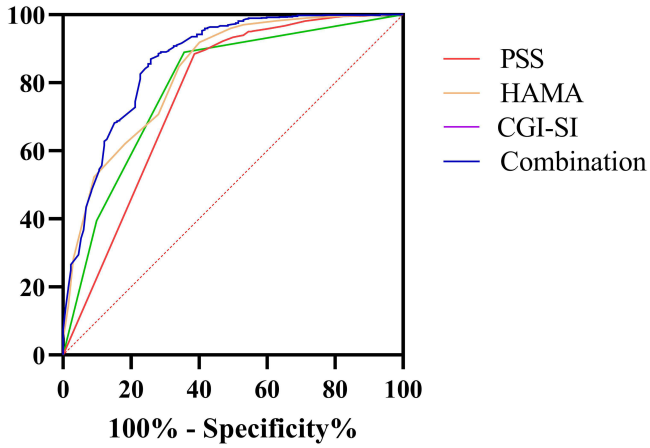

Figure 2 The discriminatory capacity of related factors for distinguishing between patients with and without SB in MDD patients. The area under the curve of PSS score, HAMA score, CGI-SI score, and the combination of these three factors were 0.76, 0.83, 0.79, and 0.87, respectively.

Table 4 Multiple Linear Regression Analysis of Correlates of SB Severity

	Coefficients	Std. Error	t	*p*-value	95% CI	
	B				Lower	Upper
Constant	16.88	3.80	4.44	<0.001*	9.34	24.43
HAMA	0.21	0.09	2.22	0.029*	0.02	0.39
DBP-mmHg	0.09	0.05	1.89	0.062	−0.01	0.19
TC- mmol/L	−0.98	0.45	−2.17	0.032*	−1.87	−0.09

Note: *p<0.05.
Abbreviations: HAMA, Hamilton Anxiety Scale Score; DBP, diastolic blood pressure; TC, total cholesterol.

SB detection rates (17.3%),[41] but notably lower than those observed in chronic/recurrent MDD cohorts (20–36%).[24,25] Intriguingly, our study identified a significantly reduced SB detection rate of 13.46%, which markedly deviates from the previously reported benchmarks. This observed pattern suggests that illness chronicity, rather than treatment setting alone, may be a more significant modulator of SB vulnerability. The coexistence of heightened psychotic symptoms (PSS), anxiety severity (HAMA), and metabolic abnormalities in SB patients aligns with integrative "brain-body" models of suicidality, wherein neuroendocrine-metabolic dysregulation synergizes with psychopathological processes was associated with increased SB risk.[28,35] Notably, thyroid dysfunction and lipid anomalies may impair prefrontal-limbic circuitry through neuroinflammatory pathways,[35] while visceral adiposity (reflected by elevated WC) co-occurred with insulin resistance, potentially reflecting a shared pathway underlying mood dysregulation.[37] The shorter disease duration in SB subgroups further suggests acute biopsychosocial decompensation—rather than illness chronicity—may drive SB emergence, urging reevaluation of duration-based risk paradigms.

Secondly, comparative analyses of sociodemographic and clinical features between MDD patients with and without SB revealed significantly heightened severity of psychopathology, psychological symptoms, and metabolic disturbances in the SB subgroup. This aligns with findings from large-scale studies in Chinese populations, which have consistently reported similar clinical and metabolic abnormalities associated with SB in MDD.[26,28,37,40,44,45] While the precise mechanisms, including lipid dysregulation, HPA axis dysfunction, neuroplasticity alterations, and inflammation, remain to be fully elucidated,[35] our findings suggest that SB in MDD co-occurs with characterized by a more adverse psychophysical state.

Thirdly, we identified several key factors associated with SB in MDD patients. These factors are multifaceted, encompassing both clinical symptoms (eg, PSS, HAMA, CGI-SI scores) and metabolic parameters. Prior research has underscored the roles of elevated anxiety symptoms, psychotic features, and specific lipid markers as factors associated with SB in MDD.[27,45,46] However, it has to be emphasized that LDL-C levels were considered by this study yet as an inverse predictor of SB in patients with MDD, contrary to the vast majority of other metabolic parameters addressed in this study. A large-scale meta-analysis yielded similar conclusions and was not confounded by ethnicity,[47,48] which emphasizes the special status of LDL-C in distinguishing it from other metabolic parameters in terms of their association with SB. While the specific clinical parameters identified in our study may differ, these findings collectively emphasize the significant association value of clinical and biological indicators in assessing SB risk in MDD.

Fourthly, we developed discriminative models for characterizing SB in MDD patients. Our analyses demonstrated robust discriminative ability of PSS, HAMA, and CGI-SI scores in distinguishing patients with and without SB. Previous studies have also explored similar approaches, achieving success using peripheral blood inflammatory cytokines and some other serum indicators.[49–51] Among these, the combination of IL-17C and TNF-β, and the combination of IL-1α, IL-5, and ICAM-1 demonstrated accuracy in distinguishing SB with AUC values of 0.848 and 0.850, respectively. However, the combination of α1-antitrypsin, transferrin, HDL-C, and apolipoprotein A1 demonstrated higher discriminatory ability (AUC = 0.928).[49–51] Our model, with a combined AUC of 0.87, also demonstrates strong capacity to differentiate SB subgroups, highlighting the efficacy of traditional clinical indicators even in the absence of advanced biomarkers.

Finally, by assessing SB severity as a continuous variable, we found HAMA scores to be predictive of more severe SB. The detrimental impact of anxiety symptoms on SB is well-established, with studies in both general university populations and

MDD patients highlighting the role of anxiety in increasing the risk of suicidal ideation and behaviors.[52–54] Consequently, MDD patients with comorbid anxiety may require augmented treatment and care to mitigate the potential for SB.

Study limitations include: (1) inherent cross-sectional causality constraints; (2) acute-phase sample homogeneity, limiting generalizability to chronic MDD; (3) undocumented antipsychotics and antidepressants exposures potentially confounding metabolic findings; and (4) potential circularity in CGI-SI assessment, as clinicians' awareness of suicide risk may inflate severity ratings. Although collinearity tests showed no critical bias, CGI-SI likely captures global severity context rather than SB-specific pathways. (5) some key psychosocial determinants of SB—including recent life stressors, substance use patterns, and socioeconomic status—were not systematically assessed. Future longitudinal designs tracking SB trajectories from first-admission through maintenance phases—while incorporating blinded CGI ratings—could elucidate dynamic risk interactions and reduce assessment bias.

Conclusion

This study establishes the clinical and metabolic signatures of SB in first-hospitalized MDD patients. The operationalized discriminative model, leveraging clinical and metabolic variables, shows utility in distinguishing high-risk subgroups.

Data Sharing Statement

All relevant data are within the manuscript.

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Disclosure

The authors report no conflicts of interest in this work.

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