Original Article

Biochemical Markers of Neuroinflammation in Depression and Anxiety Disorders

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Abstract

Background: Neuroinflammation has emerged as a critical biochemical mechanism underlying depression and anxiety. Cytokine dysregulation and oxidative stress are believed to disturb neuronal homeostasis and neurotransmitter balance, contributing to emotional and behavioural disturbances. The objective is to assess key inflammatory and oxidative stress biomarkers in patients with depressive and anxiety disorders and to determine their relationship with clinical symptom severity. **Material and Methods:** A total of 120 participants were enrolled and divided into three groups: major depressive disorder (n = 45), generalised anxiety disorder (n = 40), and healthy controls (n = 35). Serum concentrations of C-reactive protein (CRP), interleukin-6 (IL-6), tumour necrosis factor-alpha (TNF-α), and malondialdehyde (MDA) were measured by ELISA and spectrophotometric assays. At the same time, total antioxidant capacity (TAC) was determined using the ferric reducing ability of plasma (FRAP) method. Clinical severity was assessed using the Hamilton Depression (HAM-D) and Hamilton Anxiety (HAM-A) scales—statistical analysis employed one-way ANOVA, Pearson correlation, and chi-square (χ^2) tests. **Results:** Significantly elevated levels of CRP, IL-6, TNF-α, and MDA were observed in depression and anxiety groups compared with controls (CRP: 5.62 ± 1.48 vs. 2.13 ± 0.84 mg/L; IL-6: 8.42 ± 2.17 vs. 3.12 ± 0.98 pg/mL; $p < 0.001$). TAC was significantly reduced (0.92 ± 0.21 vs. 1.36 ± 0.27 mmol/L; $p < 0.001$). IL-6 levels correlated positively with HAM-D scores ($r = 0.64$, $p < 0.001$). Elevated CRP (> 3 mg/L) showed a strong association with depressive/anxiety symptoms ($\chi^2 = 18.72$, $p < 0.001$). **Conclusion:** Patients with depression and anxiety exhibit a consistent biochemical pattern of increased inflammatory cytokines and oxidative stress with reduced antioxidant defence. These findings reinforce the role of neuroinflammation in affective disorders and highlight the potential use of these biomarkers for early identification and therapeutic monitoring.

Keywords: Neuroinflammation; Depression; Anxiety; Cytokines; Oxidative stress; C-reactive protein; Interleukin-6; TNF-α; Antioxidant capacity.

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Introduction

Depression and anxiety are among the most prevalent psychiatric disorders worldwide, contributing substantially to the global burden of disability and poor quality of life.[1] Traditionally, these disorders have been explained by alterations in monoamine neurotransmitters such as serotonin, norepinephrine, and dopamine. However, increasing evidence indicates that their pathophysiology extends beyond neurotransmitter imbalance and involves chronic, low-grade inflammation within the central nervous system.[2] The concept of neuroinflammation has reshaped the understanding of mood disorders by linking peripheral immune activation, oxidative stress, and altered neuronal signalling pathways.[3]

Neuroinflammation refers to the activation of microglia and astrocytes, leading to the release of pro-inflammatory cytokines such as interleukin-6 (IL-6), tumour necrosis factor-alpha (TNF-α), and C-reactive protein (CRP). These molecules can influence neurotransmitter metabolism, synaptic plasticity, and neurogenesis, thereby promoting the biological changes associated with depression and anxiety.[4]

Elevated levels of circulating cytokines have been demonstrated in patients with major depressive disorder (MDD) and generalised anxiety disorder (GAD), supporting the hypothesis that systemic inflammation communicates with the brain through humoral and neural routes.[5]

Pro-inflammatory cytokines can disrupt the hypothalamic–pituitary–adrenal (HPA) axis, leading to sustained cortisol secretion and altered sensitivity of the glucocorticoid receptor. This results in enhanced oxidative stress and mitochondrial dysfunction, both of which contribute to neuronal injury and

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dysregulation of mood circuits.[6] The oxidative damage marker malondialdehyde (MDA) and reduced total antioxidant capacity (TAC) are frequently reported in depressive and anxiety states, indicating an imbalance between free radical generation and antioxidant defence mechanisms.[7,8] Furthermore, chronic inflammation induces indoleamine 2,3-dioxygenase (IDO) activation, diverting tryptophan metabolism toward kynurenine rather than serotonin synthesis, which exacerbates depressive symptoms.[9]

The interaction between inflammation and oxidative stress establishes a self-perpetuating cycle that amplifies neurodegeneration and impairs neurotransmission. This biochemical crosstalk explains, at least in part, why conventional antidepressant and anxiolytic therapies fail in a subset of patients who present with high inflammatory profiles.[10] Identifying and quantifying these biochemical markers could therefore improve diagnostic precision and help tailor anti-inflammatory or antioxidant-based therapeutic strategies.[11]

Although several studies have reported elevated cytokines in psychiatric illnesses, variations in findings across populations and the limited integration of oxidative stress parameters warrant further investigation. Hence, this study aims to evaluate serum levels of CRP, IL-6, TNF-α, MDA, and TAC in patients with depression and anxiety compared with healthy individuals, and to determine their relationship with clinical symptom severity.[12] Establishing such correlations may enhance understanding of the molecular underpinnings of mood disorders and open new avenues for biomarker-guided diagnosis and therapy.

Materials and Methods

Study Design and Setting: A cross-sectional, hospital-based observational study was conducted in the Department of Biochemistry at Kakatiya Medical College, Hanumakonda, Telangana, in collaboration with the Department of Psychiatry. The study period extended from June 2023 to May 2024. Ethical approval was obtained from the Institutional Ethics Committee (IEC No. KMC/IEC/BIO/Faculty/2023/07), and written informed consent was obtained from all participants before their inclusion.

Study Population: The study included a total of 120 participants, both males and females, within the age range of 20 to 55 years. Participants were divided into three distinct groups based on clinical diagnosis and mental health status. Group I comprised 45 individuals clinically diagnosed with Major Depressive Disorder (MDD), while Group II included 40 patients suffering from Generalised Anxiety Disorder (GAD). The third group, Group III, consisted of 35 apparently healthy individuals who served as controls and had no history of psychiatric illness, chronic systemic disease, or current medication use that could influence biochemical parameters.

All patients in the depressive and anxiety groups were evaluated and diagnosed by a qualified consultant psychiatrist from the Department of Psychiatry, following the diagnostic criteria outlined in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5). The diagnosis was confirmed through structured clinical interviews and psychiatric assessment to ensure diagnostic accuracy and uniformity across the study population.

The control group was carefully selected to match the patient groups in terms of age and sex distribution, thereby minimising confounding variables. Healthy participants were drawn primarily from hospital staff, postgraduate students, and attendants accompanying patients to the outpatient department, ensuring they were free from any ongoing psychological or physiological disorders. Before inclusion, all participants underwent a brief health screening and provided written informed consent after receiving a detailed explanation of the study objectives and procedures. This approach ensured the comparability and representativeness of the study population for evaluating biochemical markers associated with neuroinflammation in depressive and anxiety disorders.

Inclusion Criteria

Participants were enrolled in the study based on well-defined inclusion and exclusion criteria to ensure the accuracy and reliability of biochemical and clinical comparisons. Individuals between the ages of 20 and 55 years who were either newly diagnosed or drug-free cases of depression or anxiety disorder were considered eligible for inclusion. Only those who were willing to participate voluntarily and provided written informed consent after being informed about the study's purpose and procedures were recruited. These criteria ensured that the participants represented genuine cases of depressive and anxiety disorders without interference from pharmacological or age-related confounders.

Exclusion Criteria

Exclusion criteria were applied stringently to eliminate factors that could influence inflammatory or oxidative stress markers. Patients suffering from chronic infections, autoimmune conditions, or metabolic and endocrine abnormalities such as diabetes or thyroid dysfunction were excluded. Those with hepatic or renal impairment, malignancy, or a history of substance abuse were also not included in the study. Participants who were on long-term anti-inflammatory or antioxidant therapy were omitted to avoid biochemical variability caused by drug effects. In addition, pregnant and lactating women were excluded due to physiological and hormonal fluctuations that could alter oxidative and inflammatory biomarker levels. These selection parameters were carefully designed to ensure homogeneity of the study groups and to allow a more accurate interpretation of the association between neuroinflammatory biomarkers and psychiatric disorders.

Sample Collection: Venous blood samples were collected from all participants following an overnight fasting period of at least eight to ten hours to avoid postprandial variations in biochemical parameters. Approximately 5 mL of blood was drawn from the antecubital vein using a sterile disposable syringe under strict aseptic precautions. The collected samples were transferred into plain vacutainer tubes and allowed to clot at room temperature for about 20 to 30 minutes. Subsequently, the samples were centrifuged at 3000 revolutions per minute (rpm) for 10 minutes to obtain clear serum. The separated serum was carefully aspirated and transferred into pre-labeled microcentrifuge tubes

using sterile pipettes to prevent cross-contamination. All serum samples were immediately aliquoted to avoid repeated freeze–thaw cycles, which could degrade sensitive biochemical constituents. The aliquoted sera were stored at –80°C in a deep freezer until further biochemical analysis. Throughout the collection and storage process, standard laboratory safety protocols and biospecimen handling guidelines were strictly adhered to, ensuring the integrity and stability of the analytes being studied.

Biochemical Analysis: All biochemical estimations were conducted in the Department of Biochemistry using standardised laboratory protocols to ensure consistency and accuracy of results. Each assay was performed in triplicate, and mean values were taken for analysis to minimise intra-assay variation. Instruments were routinely calibrated, and reagents were prepared freshly according to the manufacturer's instructions. Internal quality control samples were analysed alongside test specimens to ensure analytical reliability.

Serum CRP concentration was determined using a quantitative enzyme-linked immunosorbent assay (ELISA) kit supplied by Calbiotech, USA. This immuno-turbidimetric technique relies on the interaction between CRP in the sample and specific anti-CRP antibodies, leading to the formation of antigen–antibody complexes that increase turbidity. The optical density of the resulting suspension was measured using a microplate reader, and the concentration of CRP was calculated from a standard calibration curve.

Serum interleukin-6 (IL-6) levels were estimated using a sandwich ELISA kit. In this method, IL-6 from the serum binds to monoclonal antibodies coated on the microtiter wells. After washing away unbound proteins, an enzyme-labelled secondary antibody specific to IL-6 was added. The substrate solution subsequently produced a colour reaction proportional to the cytokine concentration, which was measured spectrophotometrically at the recommended wavelength.

Tumour necrosis factor-alpha (TNF-α) concentrations were determined using a sandwich ELISA kit from RayBiotech, USA. This assay involves binding of TNF-α to a capture antibody immobilised on the plate, followed by reaction with an enzyme-conjugated detection antibody. The addition of a chromogenic substrate generated a colour intensity corresponding to the amount of TNF-α present in the sample. Absorbance was measured at 450 nm, and results were derived from a standard curve prepared using kit calibrators.

The extent of lipid peroxidation was assessed by estimating serum MDA levels using the thiobarbituric acid reactive substances (TBARS) assay. Under acidic and high-temperature conditions, MDA reacts with thiobarbituric acid (TBA) to form a pink-colored MDA–TBA complex. The intensity of the colour was measured spectrophotometrically at 532 nm using a spectrophotometer. The MDA values, expressed in micromoles per litre, indicated the degree of oxidative stress in the study subjects.

Total antioxidant capacity (TAC) was evaluated using the ferric reducing ability of plasma (FRAP) method, which assesses the reducing potential of antioxidants in the serum. The principle of this method is based on the reduction of ferric (Fe^{3+}) to ferrous (Fe^{2+}) ions in the presence of tripyridyltriazine (TPTZ), resulting in a blue-coloured Fe^{2+}–TPTZ complex. The colour intensity was measured spectrophotometrically at 593 nm, and the results were compared with a ferrous sulfate standard curve to express TAC in millimoles per litre.

All spectrophotometric readings were taken using the same instrument to maintain analytical consistency. To prevent degradation of sensitive analytes, samples were handled with care, and repeated freeze–thaw cycles were avoided. These biochemical estimations provided precise and reproducible data for evaluating inflammatory and oxidative stress markers associated with neuroinflammation in depression and anxiety disorders.

Statistical Analysis: The collected data were analysed using IBM SPSS Statistics software, version 26.0 (IBM Corp., Armonk, NY, USA). All continuous variables were summarised and expressed as mean values accompanied by their respective standard deviations (SD) to describe the central tendency and variability within each group. Before statistical testing, the normality of the data distribution was verified using the Shapiro–Wilk test to ensure that the assumptions for parametric analyses were met.

Comparisons among the three study groups—depression, anxiety, and control—were performed using one-way analysis of variance (ANOVA). This test was applied to determine whether significant differences existed in biochemical marker levels across groups. When the ANOVA revealed statistical significance, Tukey's post-hoc test was applied to identify which specific group pairs showed meaningful differences. For categorical variables, such as elevated biomarker prevalence, the chi-square (χ^2) test was used to assess associations and the strength of relationships between variables.

The degree of linear association between continuous biochemical parameters (CRP, IL-6, TNF-α, MDA, and TAC) and clinical scores, including Hamilton Depression Rating Scale (HAM-D) and Hamilton Anxiety Rating Scale (HAM-A), was determined using Pearson's correlation coefficient (r). This correlation analysis provided insight into how variations in inflammatory and oxidative markers corresponded with symptom severity. A p-value of less than 0.05 was considered statistically significant for all analyses, showing that the observed differences were unlikely to have occurred by chance.

Ethical Considerations: The study was carried out in accordance with the ethical principles outlined in the Declaration of Helsinki (2013 revision). Prior approval was obtained from the Institutional Ethics Committee of Kakatiya Medical College, Hanumakonda. Participants were fully informed about the nature and purpose of the study, and written consent was obtained before their inclusion. All data were treated with strict confidentiality, and the identity of participants was protected at every stage of the research process. Results were used solely for academic and scientific purposes, ensuring that participants' privacy and rights were maintained throughout the investigation.

Results

Demographic and Clinical Characteristics: A total of 120 participants were included in the analysis: 45 patients with MDD,

40 with GAD, and 35 healthy controls. The mean age of participants was 38.4 ± 8.2 years in the depression group, 36.7 ± 7.9 years in the anxiety group, and 37.1 ± 7.5 years among controls, with no significant inter-group difference ($p = 0.62$). The male-to-female ratio was comparable across the three groups ($\chi^2 = 1.84$, $p = 0.39$), suggesting successful matching by demographic variables.

Patients with depression and anxiety exhibited significantly higher mean scores on the HAM-D and HAM-A compared with controls (both $p < 0.001$), confirming the diagnostic validity of the clinical classification.

Table 1: Demographic and clinical profile of study participants

Parameter	Depression (n = 45)	Anxiety (n = 40)	Control (n = 35)	p-value	χ^2 value
Age (years, mean ± SD)	38.4 ± 8.2	36.7 ± 7.9	37.1 ± 7.5	0.62	—
Sex (M/F)	21 / 24	19 / 21	17 / 18	0.39	1.84
HAM-D score	23.6 ± 5.1	11.8 ± 3.6	4.2 ± 2.3	< 0.001	—
HAM-A score	12.1 ± 3.8	22.9 ± 4.7	5.1 ± 2.0	< 0.001	—

Serum Inflammatory and Oxidative Stress Markers: Substantial alterations in inflammatory and oxidative stress parameters were observed among patients compared with healthy controls. Mean serum CRP levels were markedly elevated in both depression (5.62 ± 1.48 mg/L) and anxiety (4.91 ± 1.26 mg/L) groups relative to controls (2.13 ± 0.84 mg/L). IL-6 and TNF-α also showed significant increases in the patient groups, whereas total antioxidant capacity (TAC) was reduced (Table 2; Figure 1). The differences were statistically significant by one-way ANOVA ($p < 0.001$ for all parameters).

Table 2: Comparison of biochemical markers among study groups

Biomarker	Depression (mean ± SD)	Anxiety (mean ± SD)	Control (mean ± SD)	F value	p-value
CRP (mg/L)	5.62 ± 1.48	4.91 ± 1.26	2.13 ± 0.84	58.37	< 0.001
IL-6 (pg/mL)	8.42 ± 2.17	7.86 ± 2.04	3.12 ± 0.98	76.21	< 0.001
TNF-α (pg/mL)	11.26 ± 3.04	10.78 ± 2.85	4.37 ± 1.22	92.54	< 0.001
MDA (µmol/L)	6.18 ± 1.45	5.79 ± 1.28	3.24 ± 0.91	81.09	< 0.001
TAC (mmol/L)	0.92 ± 0.21	0.98 ± 0.24	1.36 ± 0.27	49.46	< 0.001

Post-hoc analysis using Tukey's test indicated that both depression and anxiety groups differed significantly from controls ($p < 0.001$), but not significantly from each other ($p > 0.05$), suggesting a common biochemical pattern of inflammatory activation in both conditions.

Association of Elevated CRP with Psychiatric Status

To explore categorical relationships, participants were stratified based on CRP concentration (> 3 mg/L considered elevated). A significantly higher proportion of subjects in the depression (80 %) and anxiety (72.5 %) groups had elevated CRP compared with controls (25.7 %), yielding a chi-square value of $\chi^2 = 18.72$, $p < 0.001$ [Table 3].

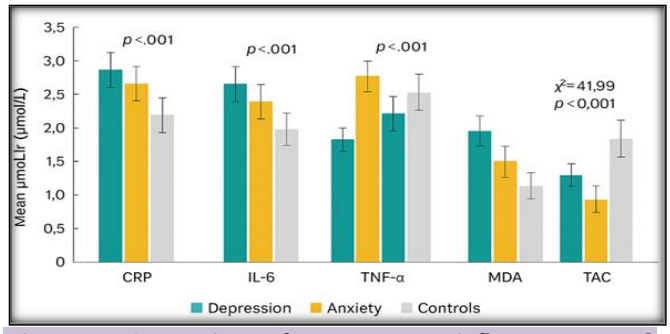

Figure 1: Comparison of mean serum inflammatory and oxidative stress markers among study groups

Table 3: Association between elevated CRP levels and psychiatric diagnosis

CRP level	Depression (n = 45)	Anxiety (n = 40)	Control (n = 35)	Total	χ^2	p
> 3 mg/L	36 (80.0 %)	29 (72.5 %)	9 (25.7 %)	74 (61.7 %)	18.72	< 0.001
≤ 3 mg/L	9 (20.0 %)	11 (27.5 %)	26 (74.3 %)	46 (38.3 %)	—	—

This observation reinforces the presence of systemic inflammation in both depressive and anxiety disorders relative to healthy individuals.

Depression severity, as measured by the HAM-D. The scatter plot reveals that participants with higher HAM-D scores tended to exhibit elevated IL-6 levels, indicating an active pro-inflammatory state in individuals with greater depressive symptomatology ($r = 0.64$, $p < 0.001$). This linear relationship suggests that IL-6 may serve as a sensitive biochemical marker reflecting the degree of neuroinflammatory involvement in depressive disorders. The finding supports the growing evidence that systemic inflammation contributes to the pathophysiology of depression, possibly through cytokine-mediated alterations in neurotransmission, neuroplasticity, and hypothalamic–pituitary–adrenal axis regulation.

Correlation between Biochemical Parameters and Symptom Severity: Pearson's correlation analysis demonstrated significant positive associations between inflammatory cytokines and symptom severity scores. IL-6 levels showed a strong correlation with HAM-D scores ($r = 0.64$, $p < 0.001$) and a moderate correlation with HAM-A scores ($r = 0.58$, $p < 0.001$). TNF-α also correlated positively with both HAM-D ($r = 0.61$*) and HAM-A ($r = 0.54$*) scales. Conversely, TAC exhibited a negative correlation with symptom severity ($r = –0.46$*, $p < 0.01$), indicating that a

greater oxidative imbalance is associated with higher levels of psychological distress [Figure 3].

Table 4: Correlation coefficients between biochemical markers and clinical scores

Parameter	HAM-D	HAM-A
CRP	0.59**	0.52**
IL-6	0.64**	0.58**
TNF-α	0.61**	0.54**
MDA	0.49**	0.45**
TAC	–0.46*	–0.43*

*Significant at $p < 0.05$; *Significant at $p < 0.001$

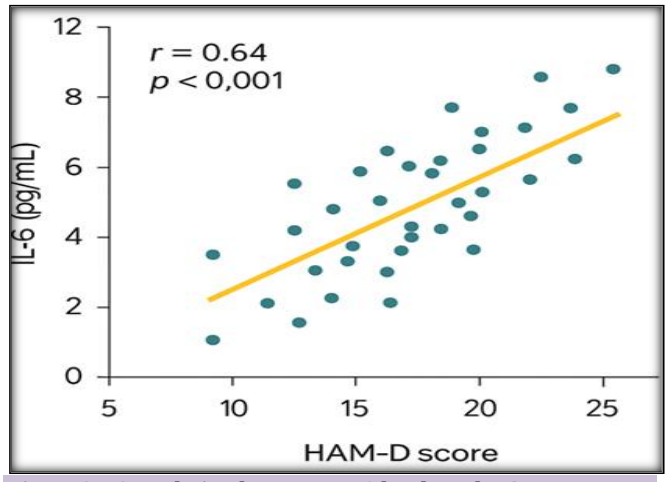

Figure 2: Correlation between IL-6 levels and HAM-D scores

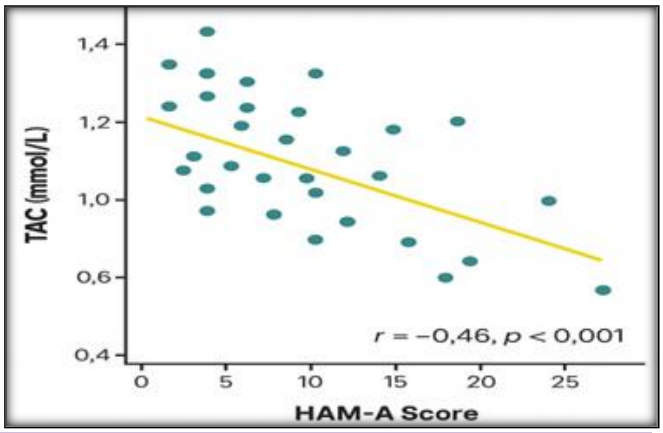

Figure 3: Inverse correlation between TAC and HAM-A scores

These results confirm that higher inflammatory and oxidative stress marker levels correspond to greater symptom intensity in both disorders.

Discussion

The present study demonstrates significant elevations in serum inflammatory cytokines (CRP, IL-6, and TNF-α) and a lipid peroxidation marker (MDA), along with a reduced total antioxidant capacity (TAC), among patients with major depressive disorder and generalised anxiety disorder compared to healthy controls. These findings reinforce the hypothesis that depression and anxiety are accompanied by systemic inflammation and oxidative stress, indicating a shared biochemical pathophysiology that links immune activation and neurochemical dysregulation.

The elevated CRP levels observed in both patient groups are consistent with previous studies that identified CRP as a peripheral marker of neuroinflammation and disease severity in depressive disorders. A meta-analysis by Haapakoski et al. (2015) reported that individuals with depression consistently exhibit higher circulating CRP concentrations compared with controls, suggesting that persistent inflammatory activity contributes to depressive symptomatology.[13] Similarly, the significant rise in IL-6 and TNF-α levels in the present study aligns with findings by Dowlati et al., who demonstrated that these cytokines play crucial roles in mediating sickness behaviour, fatigue, and anhedonia.[5] These inflammatory molecules may influence central neurotransmission by modulating serotonin, dopamine, and glutamate pathways, thereby linking immune activation with altered mood and cognition.[14]

The observed positive correlation between IL-6 concentrations and HAM-D scores in this study further supports the role of cytokines in determining symptom severity. Previous research has shown that IL-6 crosses the blood–brain barrier and activates microglial cells, triggering a cascade of neuroimmune responses that impair synaptic plasticity and neuronal survival.[15] Moreover, TNF-α has been implicated in synaptic pruning and glutamatergic dysfunction, both of which are associated with cognitive and affective disturbances in depression and anxiety.[16] These pro-inflammatory cytokines are known to activate the indoleamine 2,3-dioxygenase (IDO) pathway, diverting tryptophan metabolism from serotonin synthesis toward kynurenine production—an established mechanism contributing to mood dysregulation.[9,17]

In the current study, oxidative stress was evidenced by elevated MDA levels and a concomitant decline in TAC, indicating an imbalance between pro-oxidant and antioxidant systems. This observation is in agreement with Maes et al. (2011), who proposed that chronic inflammation leads to increased production of reactive oxygen and nitrogen species (ROS/RNS), resulting in lipid peroxidation and neuronal damage.[7] Oxidative stress and inflammation often act synergistically: pro-inflammatory cytokines promote the generation of free radicals, while oxidative byproducts further amplify cytokine release, creating a vicious cycle that sustains neuroinflammation.[18] Reduced TAC in both depression and anxiety groups reflects a depletion of antioxidant reserves, likely due to excessive utilisation in counteracting ongoing oxidative damage.[19]

The negative correlation between TAC and HAM-A scores in this study suggests that lower antioxidant defence is associated with heightened anxiety severity. This relationship mirrors the findings of Hovatta et al. (2010), who reported that increased

oxidative burden correlates with anxiety-related behaviours in both clinical and experimental models.[20] Antioxidant depletion may impair mitochondrial energy metabolism and neuronal signalling, thereby contributing to autonomic hyperactivity and the somatic manifestations of anxiety disorders.

Interestingly, despite the overlapping biochemical changes, no significant difference was noted between depression and anxiety groups in cytokine or oxidative marker levels. This finding underscores the growing recognition that these disorders share a common neuroinflammatory substrate rather than being entirely distinct entities. The concept of a "shared inflammatory phenotype" in affective disorders has been increasingly supported by recent meta-analyses.[21,22] The lack of difference between groups could also be attributed to overlapping symptom domains, stress exposure, or genetic susceptibility influencing cytokine expression patterns.

From a clinical perspective, these findings have several important implications. The consistent elevation of CRP, IL-6, and TNF-α suggests that inflammatory biomarkers could serve as potential diagnostic adjuncts or therapeutic targets. Anti-inflammatory agents, such as cytokine inhibitors and omega-3 fatty acids, have shown promising adjunctive effects in improving depressive symptoms in patients with elevated baseline inflammation.[23] Similarly, antioxidant supplementation—through compounds such as N-acetylcysteine, vitamin E, or polyphenols—may help restore redox balance and mitigate neuroinflammatory cascades, thereby complementing conventional antidepressant therapy.[24]

However, it is essential to acknowledge the limitations of this study. The cross-sectional design limits causal inference, and although the sample size is adequate for statistical power, it may not fully capture population-level heterogeneity. Moreover, the study focused on peripheral biomarkers, which may not directly reflect central nervous system inflammatory status. Longitudinal studies incorporating cerebrospinal fluid cytokine profiling and neuroimaging correlates are warranted to elucidate temporal and mechanistic relationships.

In summary, the present findings support the growing body of evidence that both depression and anxiety are associated with chronic low-grade inflammation and oxidative stress. Elevated CRP, IL-6, TNF-α, and MDA, together with reduced TAC, form a distinct biochemical signature indicative of neuroimmune activation. These alterations correlate strongly with clinical severity, suggesting that inflammatory and oxidative markers could serve as useful adjunctive indicators for disease monitoring and targeted therapeutic intervention.

Conclusion

The present study establishes that patients with major depressive disorder and generalised anxiety disorder exhibit a consistent biochemical pattern characterised by elevated inflammatory cytokines (CRP, IL-6, TNF-α), increased oxidative damage marker (MDA), and reduced total antioxidant capacity (TAC). These findings strongly support the concept that both conditions share a common pathophysiological mechanism involving chronic low-grade inflammation and oxidative stress. The observed correlations between cytokine levels and clinical severity scales further highlight the dynamic interaction between immune activation and affective symptomatology.

The evidence suggests that inflammatory and oxidative stress biomarkers may serve as valuable adjuncts in early detection, prognostic assessment, and therapeutic monitoring of mood and anxiety disorders. Integrating anti-inflammatory and antioxidant strategies with conventional psychotropic therapy could potentially improve clinical outcomes and promote neuroprotection. However, larger longitudinal studies incorporating neuroimaging and central biomarker analyses are warranted to validate these associations and clarify causal mechanisms underlying neuroinflammatory processes in psychiatric illnesses.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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