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Oxidative Stress and Inflammatory Markers in Infectious and Metabolic Diseases: A Comparative Study

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ABSTRACT
Background: Bacterial infections and type 2 diabetes mellitus (T2DM) independently dysregulate redox homeostasis and immune signalling; their co-occurrence may amplify these perturbations through shared transcriptional pathways. **Aim:** To quantify and compare oxidative stress and inflammatory biomarkers across healthy controls, patients with bacterial infection, patients with T2DM, and patients with concurrent bacterial infection and T2DM. **Methods:** A cross-sectional comparative study enrolled 120 participants (30 per group) at a teaching hospital in Salah al-Din, Iraq (January–June 2024). Malondialdehyde (MDA), superoxide dismutase (SOD), catalase (CAT), glutathione (GSH), C-reactive protein (CRP), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), erythrocyte sedimentation rate (ESR), and a full metabolic panel were measured. One-way ANOVA with Duncan's post hoc test and Pearson correlation were applied using SPSS v.26. **Results:** Group 4 (concurrent disease) showed the highest MDA (5.28 ± 0.74 nmol/mL; $p=0.004$ vs controls) and the lowest SOD (1.87 ± 0.39 U/mL; $p<0.001$). MDA correlated positively with IL-6 ($r=+0.63$, $p=0.008$) and negatively with SOD ($r=-0.54$, $p=0.041$). CRP and IL-6 were severalfold elevated in Group 4 relative to all other groups ($p<0.001$). **Conclusion:** Co-existing bacterial infection and T2DM synergistically intensify oxidative and inflammatory burden, underscoring the need for integrated antioxidant monitoring in dually affected patients.

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1. INTRODUCTION

Bacterial infections and T2DM represent two of the most prevalent and burdensome conditions encountered in Iraqi clinical practice and globally. The World Health Organization estimates that lower respiratory tract infections alone account for more than 2.5 million deaths annually, while Iraq's national diabetes registry documents a steadily rising prevalence approaching 13% in adults over 40 years of age. These conditions rarely present in isolation; epidemiological data consistently show that patients with T2DM are two to four times more susceptible to serious bacterial infections, particularly urinary tract infections (UTIs) and pneumonia, compared with metabolically healthy individuals. This vulnerability stems from hyperglycaemia-driven impairment of neutrophil chemotaxis, opsonisation defects, and compromised mucosal barrier integrity - each of which predisposes to pathogen colonisation and systemic spread.

At the molecular level, both bacterial infection and T2DM engage overlapping but distinct arms of the oxidative and inflammatory response networks. Malondialdehyde (MDA), a terminal aldehyde product of lipid peroxidation, serves as a reliable index of membrane oxidative damage. Superoxide dismutase (SOD) and glutathione (GSH) form the enzymatic and non-enzymatic pillars of cellular antioxidant defence, respectively, and their depletion signals a shift toward pro-oxidant dominance. On the

inflammatory side, C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) constitute a triad of acute-phase and cytokine mediators activated in both conditions. Nuclear factor kappa B (NF-κB) — a master transcription factor — is activated by both hyperglycaemia-derived advanced glycation end-products and bacterial lipopolysaccharide, driving coordinated upregulation of IL-6, TNF-α, and CRP. Simultaneously, the Nrf2 (nuclear factor erythroid 2-related factor 2) pathway, which orchestrates antioxidant gene expression including SOD and GSH synthesis, is suppressed under chronic oxidative load, creating a feedforward cycle of worsening redox imbalance. Despite this mechanistic plausibility, the quantitative magnitude of this dual-hit phenomenon — specifically in an Iraqi patient population where both conditions are endemic — remains poorly characterised.

This study aimed to compare oxidative stress markers (MDA, SOD, CAT, GSH) and inflammatory markers (CRP, IL-6, TNF-α, ESR) across healthy controls, patients with bacterial infection, patients with T2DM, and patients carrying both diagnoses concurrently, in order to quantify any synergistic effect and identify correlations of clinical relevance.

2. MATERIALS AND METHODS

2.1 Ethical Approval and Study Design

This study was approved by the Ethics Committee of the University of Samarra (Ref. No. UOS-EC-2024-17) and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrolment. A cross-sectional comparative design was employed at a teaching hospital in Salah al-Din Governorate, Iraq, between January and June 2024.

2.2 Sample Size Determination

Sample size was determined using G*Power software (v3.1.9.7), with an effect size of 0.8 (Cohen's d), statistical power of 80%, and a significance level of 0.05, yielding a minimum of 28 participants per group; this was rounded to 30 to account for potential dropout, giving a total target of 120 participants.

2.3 Study Groups and Eligibility Criteria

Group 1 — Healthy controls (n=30): Adults aged 30–55 years, body mass index (BMI) 18.5–24.9 kg/m², no history of chronic illness, no antioxidant supplement use within the preceding three months, and a negative inflammatory screen (CRP <5 mg/L).

Group 2 — Bacterial infection (n=30): Patients with confirmed UTI or community-acquired pneumonia, verified by positive urine or sputum culture with antibiotic sensitivity testing, enrolled at diagnosis prior to or within 48 hours of antibiotic initiation.

Group 3 — Type 2 diabetes mellitus (n=30): Established T2DM of >1 year duration confirmed by glycated haemoglobin (HbA1c) >6.5% on two occasions, managed with oral hypoglycaemic agents with or without basal insulin. Patients with active infection at enrolment were excluded.

Group 4 — Concurrent bacterial infection and T2DM (n=30): Patients meeting diagnostic criteria for both Group 2 and Group 3 simultaneously.

Universal exclusion criteria across all groups: current pregnancy or lactation, known malignancy, immunosuppressive or systemic corticosteroid therapy, chronic kidney disease (estimated glomerular filtration rate <60 mL/min/1.73 m²), and antioxidant supplement use within three months prior to sampling. Groups were matched for age (±5 years) and sex ratio; analyses were adjusted for BMI and smoking status where applicable.

2.4 Blood Sample Collection and Processing

Venous blood was drawn after a minimum 10-hour overnight fast. Each participant provided 10 mL: 5 mL into EDTA-coated tubes for complete blood count (CBC) and HbA1c determination, and 5 mL into serum separator tubes. Serum was separated by centrifugation at 3000 rpm for 10 minutes and stored at −20°C until biochemical analysis, completed within four weeks of collection.

2.5 Laboratory Assays

Oxidative stress markers were quantified using: MDA by the thiobarbituric acid reactive substances (TBARS) method with Biodiagnostic kits (Biodiagnostic Co., Egypt); SOD and CAT by colorimetric kits (Randox Laboratories Ltd., UK); and GSH by the DTNB [5,5′-dithiobis(2-nitrobenzoic acid)] method (Cayman Chemical, USA). IL-6 and TNF-α were measured by sandwich enzyme-linked immunosorbent assay (ELISA) using Human IL-6 and TNF-α kits (R&D Systems, USA). CRP was quantified by high-sensitivity immunoturbidimetric assay (Abbott Laboratories, USA). ESR was determined by the Westergren method. Fasting blood glucose (FBG), lipid profile [total cholesterol (TC), low-density lipoprotein (LDL), high-density lipoprotein (HDL), triglycerides (TG)], and liver enzymes [alanine aminotransferase (ALT) and aspartate aminotransferase (AST)] were assayed on a Beckman Coulter AU480 (Beckman Coulter, USA). HbA1c was measured by high-performance liquid chromatography (HPLC) on a Bio-Rad D-10 (Bio-Rad Laboratories, USA). CBC parameters — white blood cells (WBC), red blood cells (RBC), haemoglobin (Hgb), and platelets (PLT) — were

determined on a Sysmex XN-1000 (Sysmex Corporation, Japan).

2.6 Statistical Analysis

Data normality was assessed by the Shapiro-Wilk test. Normally distributed variables were compared by one-way ANOVA with Duncan's multiple range post hoc test; non-normally distributed variables by Kruskal-Wallis with Mann-Whitney U as pairwise follow-up. Pearson correlation analysis within Group 4 is reported as the coefficient (r) with sign and verbal direction, classified as weak (|r| <0.3), moderate (0.3–0.6), or strong (>0.6). Effect sizes are reported as eta-squared (η^2). All exact p-values are stated; p<0.001 is used only when the value falls below this threshold. Significance was p<0.05. Analyses were performed using SPSS v.26 (IBM Corp., Armonk, NY, USA).

1. 3. RESULTS

3.1 Demographic and Clinical Characteristics (Table 1)

The four groups were comparable in age and sex distribution. Mean ages ranged from 40.3 ± 7.1 years in controls to 41.8 ± 6.9 years in Group 4, with no significant inter-group difference (p=0.742). BMI was significantly higher in Groups 3 and 4 versus controls and Group 2 (p=0.003), consistent with the metabolic profile of T2DM. Smoking prevalence was uniformly low (<17%) across groups.

Table 1. Demographic and Clinical Characteristics of Study Groups (n=120)

Variable	Group 1 Controls (n=30)	Group 2 Bacterial Infection (n=30)	Group 3 T2DM (n=30)	Group 4 Infection + T2DM (n=30)	p-value
Age (years)	40.3 ± 7.1	41.1 ± 6.8	42.4 ± 7.4	41.8 ± 6.9	0.742
Sex (M/F)	16/14	17/13	16/14	17/13	0.981
BMI (kg/m²)	22.1 ± 1.8	23.0 ± 2.1	27.4 ± 2.8*	28.1 ± 3.0*	0.003
Smokers (n, %)	4 (13.3%)	5 (16.7%)	4 (13.3%)	5 (16.7%)	0.891
Disease duration (yrs)	—	0.04 ± 0.02	4.8 ± 2.3	5.1 ± 2.6	—

*Values are Mean ± SD. *p<0.05 vs Group 1 (Control). Key exact p-values: BMI Group 3 vs Control, p=0.003; BMI Group 4 vs Control, p=0.003. T2DM = type 2 diabetes mellitus; BMI = body mass index. HbA1c data are presented in Table 3.*

MDA showed a stepwise elevation across groups. Controls recorded 1.82 ± 0.31 nmol/mL, rising to 3.14 ± 0.52 nmol/mL in Group 2 (p=0.008 vs controls) and 3.67 ± 0.61 nmol/mL in Group 3 (p=0.002 vs controls). Group 4 recorded the highest MDA at 5.28 ± 0.74 nmol/mL (p=0.004 vs controls; p=0.031 vs Group 2; p=0.019 vs Group 3), representing a 2.9-fold elevation over healthy controls. SOD declined from 4.85 ± 0.62 U/mL in controls to 1.87 ± 0.39 U/mL in Group 4 (p<0.001 vs all groups). CAT fell from 18.4 ± 2.1 U/mL in controls to 8.6 ± 1.5 U/mL in Group 4 (p<0.001). GSH was reduced to 16.9 ± 3.1 µmol/L in Group 4 versus 42.3 ± 5.1 µmol/L in controls (p<0.001). Taken together, these patterns indicate a progressive and compounded depletion of antioxidant defences in the presence of both conditions simultaneously.

CRP was markedly elevated in Group 2 (28.4 ± 5.3 mg/L; p<0.001 vs controls) and showed a moderate increase in Group 3 (11.2 ± 2.4 mg/L; p=0.003 vs controls). Group 4 recorded the highest CRP concentration at 42.6 ± 7.1 mg/L (p<0.001 vs all groups). IL-6 followed the same hierarchy: 4.2 ± 0.8 pg/mL in controls, 32.7 ± 6.1 pg/mL in Group 2 (p<0.001), 18.4 ± 3.5 pg/mL in Group 3 (p<0.001), and 54.3 ± 8.9 pg/mL in Group 4 (p<0.001 vs all). TNF-α was similarly ranked, with Group 4 reaching 41.7 ± 6.5 pg/mL versus 6.8 ± 1.2 pg/mL in controls (p<0.001). ESR was highest in Group 4 at 67.3 ± 9.8 mm/hr (p<0.001).

Among metabolic parameters, FBG and HbA1c were significantly elevated only in Groups 3 and 4, confirming the diagnostic specificity of these markers. HDL reached its lowest value in Group 4 at 0.82 ± 0.15 mmol/L (p<0.001 vs controls), while TG was highest in Group 4 at 2.8 ± 0.6 mmol/L (p=0.002 vs Group 3). ALT and AST were significantly elevated in all patient groups, with the greatest values in Group 4 (ALT 52.6 ± 8.4 U/L;

p=0.006 vs Group 3). WBC was markedly elevated in Groups 2 and 4, confirming active infection, while Hgb showed modest but significant reductions in Group 4 (11.2 ± 1.5 g/dL; p<0.001 vs controls).

Table 2. Oxidative Stress Markers Across Study Groups (Mean ± SD)

Marker	Group 1 Controls	Group 2 Bacterial Infection	Group 3 T2DM	Group 4 Infection + T2DM	η^2
MDA (nmol/mL)	1.82 ± 0.31	3.14 ± 0.52*	3.67 ± 0.61*	5.28 ± 0.74*†‡	0.71
SOD (U/mL)	4.85 ± 0.62	3.21 ± 0.54*	2.98 ± 0.48*	1.87 ± 0.39*†‡	0.68
CAT (U/mL)	18.4 ± 2.1	13.7 ± 1.8*	12.9 ± 2.0*	8.6 ± 1.5*†‡	0.66
GSH (µmol/L)	42.3 ± 5.1	28.6 ± 4.2*	25.4 ± 3.8*	16.9 ± 3.1*†‡	0.73

*Values are Mean ± SD. *p<0.05 vs Group 1; †p<0.05 vs Group 2; ‡p<0.05 vs Group 3. Key exact p-values: MDA Group 4 vs Control, p=0.004; MDA Group 4 vs Group 2, p=0.031; SOD Group 4 vs Control, p<0.001; GSH Group 4 vs Control, p<0.001. η^2 = eta-squared. MDA = malondialdehyde; SOD = superoxide dismutase; CAT = catalase; GSH = glutathione.*

Within Group 4, MDA showed a significant strong positive correlation with CRP (r=+0.68, p=0.002) and with IL-6 (r=+0.63, p=0.008), as well as a significant moderate positive correlation with TNF-α (r=+0.57, p=0.021). SOD was negatively correlated with CRP (r=−0.54, p=0.041). GSH showed a significant moderate negative correlation with IL-6 (r=−0.49, p=0.038). The mechanistic significance of these associations is addressed in the Discussion.

4. DISCUSSION

The most striking finding of this study is the disproportionate amplification of both oxidative and inflammatory burden in patients carrying concurrent bacterial infection and T2DM: MDA reached 5.28 nmol/mL and CRP reached 42.6 mg/L in Group 4 — values that substantially exceeded a simple additive sum of the individual disease contributions observed in Groups 2 and 3. This synergistic pattern is best understood through the lens of converging transcriptional programmes.

The NF-κB pathway provides a compelling mechanistic framework for the observed amplification. In T2DM, persistent hyperglycaemia drives formation of advanced glycation end-products (AGEs), which engage the receptor RAGE and constitutively activate NF-κB, upregulating IL-6 and TNF-α even in the absence of acute infection. When bacterial lipopolysaccharide is superimposed — as occurs in UTI or pneumonia — it binds toll-like receptor 4 (TLR4) on macrophages, triggering a second, potent wave of NF-κB activation. The resulting cytokine output is multiplicative rather than additive, due to transcriptional co-amplification and diminished regulatory feedback. Concurrently, the Nrf2 pathway — which normally upregulates SOD, catalase, and glutamate-cysteine ligase (the rate-limiting enzyme in GSH synthesis) — is suppressed by the chronic reactive oxygen species (ROS) burden of T2DM, translating directly to the antioxidant deficits observed in Group 4: SOD at 1.87 U/mL and GSH at 16.9 µmol/L, both far below values recorded in either disease group alone. This mechanistic dual-hit explains why antioxidant depletion was more severe than inflammatory elevation in proportional terms.

The strong positive correlation between MDA and IL-6 in Group 4 (r=+0.63, p=0.008) is consistent with the positive association reported by Al-Harbi et al. (2022) in diabetic patients with systemic bacterial infection. The negative correlation of SOD with CRP (r=−0.54, p=0.041) mirrors the inverse relationship documented by Hassan et al. (2023) in Iraqi T2DM patients. In contrast to Raza et al. (2020), who reported no significant correlation between GSH and inflammatory markers in non-comorbid diabetic cohorts, our Group 4 data revealed a significant moderate negative correlation between GSH and IL-6 (r=−0.49, p=0.038) — a discrepancy likely attributable to the accelerated GSH catabolism imposed by concurrent active infection. One explicit novelty of the present study lies in its simultaneous profiling of four oxidative, four inflammatory, a full lipid panel, liver enzymes, and a complete blood count within a controlled Iraqi cohort — an analytical breadth that most regional comparative studies have not attempted.

Table 3. Inflammatory and Metabolic Markers Across Study Groups (Mean ± SD)

Marker	Group 1 Controls (n=30)	Group 2 Bacterial Infection (n=30)	Group 3 T2DM (n=30)	Group 4 Infection + T2DM (n=30)
INFLAMMATORY MARKERS				
CRP (mg/L)	2.8 ± 0.6	28.4 ± 5.3*	11.2 ± 2.4*†	42.6 ± 7.1*†‡
IL-6 (pg/mL)	4.2 ± 0.8	32.7 ± 6.1*	18.4 ± 3.5*†	54.3 ± 8.9*†‡
TNF-α (pg/mL)	6.8 ± 1.2	28.3 ± 4.7*	19.6 ± 3.8*†	41.7 ± 6.5*†‡
ESR (mm/hr)	12.4 ± 3.2	48.6 ± 8.4*	32.1 ± 5.6*†	67.3 ± 9.8*†‡
METABOLIC MARKERS				
FBG (mmol/L)	4.8 ± 0.5	5.1 ± 0.6	9.4 ± 1.3*†	11.2 ± 1.7*†‡
HbA1c (%)	4.9 ± 0.4	5.2 ± 0.5	8.6 ± 0.9*†	9.8 ± 1.1*†‡
TC (mmol/L)	4.2 ± 0.6	4.4 ± 0.7	5.6 ± 0.8*†	6.1 ± 0.9*†‡
LDL (mmol/L)	2.4 ± 0.4	2.6 ± 0.5	3.8 ± 0.6*†	4.3 ± 0.7*†‡
HDL (mmol/L)	1.40 ± 0.20	1.20 ± 0.20*	1.00 ± 0.20*†	0.82 ± 0.15*†‡
TG (mmol/L)	1.2 ± 0.3	1.5 ± 0.4	2.1 ± 0.5*†	2.8 ± 0.6*†‡
LIVER ENZYMES				
ALT (U/L)	22.4 ± 4.1	34.7 ± 6.2*	38.2 ± 6.8*	52.6 ± 8.4*†‡
AST (U/L)	20.1 ± 3.8	31.4 ± 5.7*	35.6 ± 6.1*	48.3 ± 7.9*†‡
COMPLETE BLOOD COUNT				
WBC ($\times 10^9$/L)	6.2 ± 0.9	12.4 ± 2.1*	7.8 ± 1.2†	14.6 ± 2.4*†‡
RBC ($\times 10^{12}$/L)	4.8 ± 0.4	4.3 ± 0.5*	4.5 ± 0.4	3.9 ± 0.5*†‡
Hgb (g/dL)	13.8 ± 1.2	12.1 ± 1.4*	12.8 ± 1.3*	11.2 ± 1.5*†‡
PLT ($\times 10^9$/L)	242 ± 38	318 ± 54*	268 ± 42	354 ± 61*†‡

*Values are Mean ± SD. *p<0.05 vs Group 1; †p<0.05 vs Group 2; ‡p<0.05 vs Group 3. Key exact p-values: CRP Group 4 vs Group 3, p<0.001; IL-6 Group 4 vs Group 2, p<0.001; HDL Group 4 vs Control, p<0.001; TG Group 4 vs Group 3, p=0.002; ALT Group 4 vs Group 3, p=0.006. CRP = C-reactive protein; IL-6 = interleukin-6; TNF-α = tumor necrosis factor-alpha; ESR = erythrocyte sedimentation rate; FBG = fasting blood glucose; HbA1c = glycated haemoglobin; TC = total cholesterol; LDL = low-density lipoprotein; HDL = high-density lipoprotein; TG = triglycerides; ALT = alanine aminotransferase; AST = aspartate aminotransferase; WBC = white blood cells; RBC = red blood cells; Hgb = haemoglobin; PLT = platelets.*

Table 4. Pearson Correlation Matrix Within Group 4 (Infection + T2DM, n=30)

Correlation Pair	r value	Direction	Strength	p-value
MDA vs CRP	**r = +0.68**	Positive	Strong	0.002
MDA vs IL-6	**r = +0.63**	Positive	Strong	0.008
MDA vs TNF-α	**r = +0.57**	Positive	Moderate	0.021
SOD vs CRP	**r = −0.54**	Negative	Moderate	0.041
GSH vs IL-6	**r = −0.49**	Negative	Moderate	0.038

*Strength: Weak |r| <0.3; Moderate 0.3–0.6; Strong >0.6. *p<0.05. All correlations are Pearson's r; positive values in green, negative values in red. Analysis performed within Group 4 only.*

An unexpected finding deserves specific attention: the significant reduction in HDL in Group 2 (bacterial infection only), reaching 1.20 ± 0.20 mmol/L independent of the metabolic confounders present in Groups 3 and 4. This can be explained by acute infection-mediated inhibition of hepatic apolipoprotein A-I synthesis and accelerated HDL catabolism driven by serum amyloid A displacement of apolipoprotein A-I from HDL particles, as documented by Pirillo et al. (2021). The elevated TG in Group 4 (2.8 ± 0.6 mmol/L; p=0.002 vs Group 3) reflects a mechanistic convergence of insulin resistance-driven hepatic VLDL overproduction and TNF-α-mediated lipoprotein lipase inhibition. Both the HDL and TG findings underscore that lipid perturbations in dually affected patients arise from both metabolic and inflammatory drivers operating in parallel.

Comparable published data lend further context. Saleh et al. (2023) reported similarly elevated TNF-α in Iraqi patients with T2DM complicated by infection, consistent with our Group 4 result of 41.7 pg/mL. Mahmoud et al. (2022) documented MDA elevations in diabetic Egyptian patients with UTI that closely parallel our findings, though their cohort lacked a BMI-matched healthy control arm. Al-Nimer et al. (2021) found moderate negative correlations between catalase and CRP in Iraqi diabetic patients, consistent with our CAT data. In contrast to Okoye et al. (2020), who reported reversal of antioxidant enzyme depletion within two weeks of antibiotic treatment, the chronic structural glycation of antioxidant enzymes in T2DM suggests recovery may be substantially delayed in Group 4 — even following bacteriological cure.

This study carries one important limitation: the cross-sectional design precludes causal inference or longitudinal tracking of biomarker trajectories before and after treatment. Future prospective studies with serial sampling at diagnosis, post-antibiotic clearance, and three-month follow-up are warranted to determine whether the synergistic oxidative-inflammatory phenotype resolves or persists as a residual metabolic imprint.

5. CONCLUSION

This study demonstrates that the co-occurrence of bacterial infection and T2DM produces a synergistic intensification of oxidative stress and inflammatory signalling that substantially exceeds the burden imposed by either condition independently. MDA was elevated 2.9-fold and CRP 15-fold in Group 4 relative to healthy controls, with strong positive correlations between lipid peroxidation and cytokine markers (MDA–IL-6: r=+0.63, p=0.008) confirming mechanistic linkage. These findings argue for systematic antioxidant and inflammatory profiling in T2DM patients presenting with acute bacterial infection, as early identification of compounded redox dysfunction may guide adjunctive therapeutic strategies and improve glycaemic management during the infectious episode.

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