Original Article

Comparative Evaluation of Oxidative Stress Markers in Patients with Bacterial Sepsis and Healthy Controls

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Abstract

Background: Bacterial sepsis is a severe, life-threatening illness caused by bacterial infection, which creates an abnormal immune response and results in damage to many different organ systems in the body. Many researchers have demonstrated that oxidative stress significantly contributes to the development of sepsis. During systemic inflammation, excessive reactive oxygen species (ROS) are produced, leading to oxidative damage to lipids, proteins, and DNA, as well as a loss of the body's natural ability to produce protective antioxidants. Testing for oxidative stress markers can help determine how severe and advanced the disease is and may provide information for developing future treatments. In this study, the authors examined how people with sepsis differ from healthy individuals in terms of blood oxidative stress levels. They also wanted to see whether there were any links between these findings and a patient's illness severity. **Material and Methods:** An analysis was conducted using a prospective case-control design over 18 months, with 120 subjects participating. Of these subjects, 60 comprised the study group, consisting of patients who met clinical and laboratory criteria for bacterial sepsis. At the same time, the other 60 were healthy individuals who were matched for age and gender and were free of chronic systemic illness or any evidence of infection. To evaluate oxidative stress levels in their bodies, blood samples were taken from all participants. In addition to measuring levels of malondialdehyde (MDA), a biochemical marker of lipid oxidation (lipid peroxidation), catalase, superoxide dismutase, and reduced glutathione, statistical comparisons were made between the two groups. **Results:** Bacterial sepsis patients show significantly higher malondialdehyde concentration than healthy controls, suggesting an increased level of lipid peroxidation. Bacterial sepsis patients exhibit significantly lower levels of key antioxidant markers (superoxide dismutase, catalase, and reduced glutathione), indicating an impaired endogenous antioxidant defense system. Thus, these results indicate a significant imbalance between oxidative and antioxidant systems in bacterial sepsis. **Conclusion:** Increased levels of oxidative stress and impairment of the ability to provide antioxidant protection against this increased oxidative stress will occur during bacterial sepsis. Thus, monitoring levels of oxidative markers could provide insight into how this disease progresses and may be used to develop different therapeutic strategies based on antioxidant therapies. There is also a need for further research to evaluate the prognostic significance of oxidative markers in patients with sepsis.

Keywords: Bacterial sepsis; Oxidative stress; Malondialdehyde; Superoxide dismutase; Catalase; Glutathione; Antioxidant defense; Reactive oxygen species.

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Introduction

Septicemia is a serious worldwide threat and is among the top causes of illness and death among those in critical care. Sepsis is the body's inappropriate response to an infection, leading to critical organ failure. Although there have been significant improvements in the treatment of bacterial sepsis with antibiotics, the management and support of critically ill people, as well as the use of other support measures, provide little relief from the high mortality from bacterial sepsis, especially in low- and middle-income countries.[1] Many researchers are still trying to understand the complexity of the development of sepsis because there are many factors involved: the inflammatory response, the activation of the immune system, the destruction of the endothelium, the dysfunction of the coagulation system, and the presence of metabolic abnormalities are all part of the pathogenesis of sepsis.[2]

The most important part of sepsis pathogenesis may be the production of excessive reactive oxygen species (ROS). These ROS are generated by the respiratory burst of neutrophils and macrophages when they encounter and are activated by bacteria.[3] During this process, large amounts of superoxide anion(s), hydrogen peroxide, hydroxyl radicals, and other reactive intermediates are produced. Although these reactive species are important for killing bacteria, their unchecked production can damage cells and tissues. When oxidant

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production exceeds antioxidant protection, oxidative stress occurs.[4]

Oxidative stress is thought to play a role in many of the mechanisms of organ dysfunction that occur with septicemia. When lipid membranes are damaged by lipid peroxidation, they become more permeable, thereby compromising cell integrity. When proteins are oxidized, they can change the activity of many enzymes and prevent structural proteins from functioning properly.[5] When DNA is oxidatively damaged, it can interfere with normal cellular processes such as signalling and replication. Oxidative injury is particularly detrimental to endothelial cells, resulting in vascular permeability, microcirculatory dysfunction, and hypotension (3 of the main signs of septic shock). The body has developed complex antioxidant defence systems to protect against oxidative injury. These protective systems contain enzymatic (e.g., superoxide dismutase (SOD), catalase, and glutathione peroxidase) and non-enzymatic (e.g., reduced glutathione (GSH), vitamins C and E, and trace elements) antioxidants.[6] Nevertheless, during a septic episode, an increase in oxidative loads can surpass the capacity of these protective systems and lead to depletion of antioxidant reserves. Loss of activity in antioxidant enzymes is associated with mother-to-baby transmission of sepsis, indicating that a compromised compensatory response to oxidative injury has occurred (i.e., less antioxidant enzyme activity is evident in septic patients).[7] Malondialdehyde (MDA) is one of the most widely studied biomarkers of lipid peroxidation and serves as an indicator of membrane oxidative injury. MDA levels are elevated in many types of inflammatory/infectious conditions, including sepsis. Measurement of the activity of antioxidant enzymes (e.g., SOD, catalase), along with evaluation of GSH levels, is a method for assessing the antioxidative capacity of the human body. Thus, by evaluating both oxidative and antioxidative markers in disease processes, a more complete understanding of the redox imbalance associated with the disease may be achieved.[8]

The relationship between oxidative stress and sepsis is reciprocal. Excessive oxidative stress is one of the factors that cause organ damage and increased mortality; however, systemic inflammation exacerbates the production of reactive oxygen species (ROS), resulting in an endless cycle of oxidative injury and inflammatory activation. Therefore, understanding this relationship will help identify future therapies that modulate the effects of oxidative stress in patients with sepsis.[9,10]

Several studies have examined oxidative stress in patients with sepsis; however, differences in patient population, illness severity, and biomarker selection among studies have led to inconsistencies in the data. A systematic comparison of oxidative stress markers in patients with sepsis of bacterial origin and healthy subjects can provide insight into the extent of the oxidative imbalance associated with sepsis. Furthermore, evaluating oxidative stress markers may aid in establishing oxidative biomarkers, which may help identify disease severity or provide targets for adjunctive antioxidant therapy. Thus, this study aims to assess oxidative stress and antioxidant parameters in patients with bacterial sepsis and healthy subjects to understand the relationship between redox alterations and septic states.

Objectives

Primary Objective

To comparatively evaluate oxidative stress markers in patients diagnosed with bacterial sepsis and in age- and sex-matched healthy controls.

Secondary Objectives

1. To assess the levels of lipid peroxidation marker malondialdehyde in septic patients and healthy individuals.
2. To evaluate the activity of antioxidant enzymes, including superoxide dismutase and catalase, in both groups.
3. To determine the levels of reduced glutathione as a marker of non-enzymatic antioxidant defense.
4. To analyze the extent of oxidative–antioxidant imbalance associated with bacterial sepsis.
5. To explore the potential role of oxidative stress markers as indicators of pathophysiological changes in sepsis.

Materials and Methods

Study Design and Setting: The biochemistry department, in collaboration with the department of internal medicine and the intensive care unit of a major tertiary care hospital, conducted this case-control study over 18 months.

Study Population and Sample Size: A total of 120 participants were enrolled in the study. The study population was divided into two groups:

- Sepsis Group (n = 60): Patients diagnosed with bacterial sepsis.
- Control Group (n = 60): Age- and sex-matched healthy individuals.

The study used an expected case load during its completion to help determine the sample size and ensure it had sufficient statistical power to detect differences in oxidative stress markers between treatment groups.

Inclusion Criteria

For Sepsis Group:

- Patients aged 18–65 years.
- Clinical diagnosis of bacterial sepsis based on standard diagnostic criteria.
- Laboratory evidence supporting bacterial infection (e.g., positive blood culture or elevated inflammatory markers).
- Informed consent was obtained from the patient or a legally authorized representative.

For Control Group:

- Healthy individuals aged 18–65 years.
- No clinical evidence of infection or chronic systemic illness.
- No history of recent hospitalization or acute illness within the past four weeks.

Exclusion Criteria

- Patients with chronic inflammatory diseases.
- Known diabetes mellitus or chronic kidney disease.
- Chronic liver disease.
- Malignancy.
- Patients receiving antioxidant supplementation.
- Smokers or chronic alcohol users.
- Pregnant or lactating women.

Sample Collection: Blood samples were obtained from all study

subjects using aseptic procedures. In the sepsis cohort, samples were collected within 24 hours of sepsis diagnosis and before treatment with antioxidants. Blood samples were placed in the appropriate containers to allow the separate collection of serum and plasma. After centrifugation, all blood samples were stored at the appropriate temperature until biochemical analysis.

Biochemical Analysis:

The following oxidative stress and antioxidant markers were estimated:

- Malondialdehyde (MDA): Measured as an indicator of lipid peroxidation using a standardized thiobarbituric acid reactive substances method.
- Superoxide Dismutase (SOD): Assessed using spectrophotometric enzymatic assay.
- Catalase: Estimated by measuring the rate of decomposition of hydrogen peroxide.
- Reduced Glutathione (GSH): Determined using established colorimetric methods.

All assays were performed following standardized laboratory protocols to ensure accuracy and reproducibility.

Outcome Measures

Primary outcome:

- Comparison of mean levels of oxidative stress markers between sepsis patients and healthy controls.

Secondary outcome:

- Assessment of antioxidant enzyme activity depletion in septic patients.

Statistical Analysis: Descriptive statistics were calculated as mean ± standard deviation (SD) for all continuous variables and the number (n) and percentage (%) for all categorical variables. The groups were compared using the appropriate statistical tests. A statistically significant difference was defined as a p-value less than 0.05.

Results

Of the 120 participants enrolled in the study over approximately 1.5 years, 60 had been diagnosed with bacterial sepsis, and 60 were healthy controls. There were no statistically significant differences between the two groups in age or gender, indicating that both groups had similar demographic characteristics. The average time between the onset of symptoms and hospital admission for bacteraemia patients was 3.2 ± 1.1 days. Patients with bacterial sepsis had markedly higher levels of malondialdehyde (MDA) than control subjects, suggesting that these patients had undergone greater lipid peroxidation and oxygen-derived free radical-induced cell injury than control subjects. On the other hand, levels of antioxidant enzymes, such as superoxide dismutase (SOD) and catalase (CAT), were significantly lower in patients with bacteraemia than in healthy subjects. Levels of reduced glutathione were also significantly lower in patients with bacterial infection than in controls, suggesting an overall depletion of non-enzymatic antioxidant stores in these individuals. The oxidative stress index (the ratio of pro-oxidant and antioxidant markers) was also substantially elevated in patients with bacterial infection compared to those without. Thus, these data support the premise of a dysregulation of the oxidative–antioxidant balance in patients with bacterial sepsis.

Table 1: Age distribution of study participants

Age group (years)	Sepsis Group (n = 60)	Control Group (n = 60)
18–30	14 (23.3%)	16 (26.7%)
31–45	22 (36.7%)	20 (33.3%)
46–60	24 (40%)	24 (40%)

[Table 1] shows comparable age distribution in both groups.

Table 2: Gender distribution

Gender	Sepsis Group (n = 60)	Control Group (n = 60)
Male	38 (63.3%)	36 (60%)
Female	22 (36.7%)	24 (40%)

[Table 2] shows similar gender representation in both groups.

Table 3: Baseline clinical parameters in sepsis group

Parameter	Mean ± SD
Total leukocyte count (×10³/μL)	15.8 ± 4.2
C-reactive protein (mg/L)	86 ± 28
Serum lactate (mmol/L)	3.4 ± 1.1

[Table 3] shows inflammatory markers in septic patients.

Table 4: Serum Malondialdehyde (MDA) levels

Parameter	Sepsis Group (mean ± SD)	Control Group (mean ± SD)
MDA (nmol/mL)	6.8 ± 1.5	3.2 ± 0.9

[Table 4] shows significantly elevated MDA levels in septic patients.

Table 5: Superoxide Dismutase (SOD) activity

Parameter	Sepsis Group (mean ± SD)	Control Group (mean ± SD)
SOD (U/mL)	1.8 ± 0.6	3.6 ± 0.8

[Table 5] shows reduced SOD activity in sepsis group.

Table 6: Catalase levels

Parameter	Sepsis Group (mean ± SD)	Control Group (mean ± SD)
Catalase (U/mL)	38 ± 9	64 ± 12

[Table 6] shows decreased catalase levels in septic patients.

Table 7: Reduced Glutathione (GSH) levels

Parameter	Sepsis Group (mean ± SD)	Control Group (mean ± SD)
GSH (µmol/L)	4.1 ± 1.2	8.5 ± 1.7

[Table 7] shows significant depletion of GSH in sepsis group.

Table 8: Oxidative Stress Index (OSI)

Parameter	Sepsis Group (mean ± SD)	Control Group (mean ± SD)
OSI (arbitrary units)	3.8 ± 0.9	1.2 ± 0.4

[Table 8] shows higher oxidative stress index in septic patients.

Table 9: Correlation between MDA and CRP in sepsis group

Parameter	Correlation coefficient (r)	p value
MDA vs CRP	0.62	<0.001

[Table 9] shows positive correlation between oxidative stress and inflammation.

Table 10: Comparison of antioxidant depletion severity

Parameter	Sepsis Group (n = 60)	Control Group (n = 60)
Low SOD (<2 U/mL)	34 (56.7%)	6 (10%)
Low Catalase (<40 U/mL)	28 (46.7%)	4 (6.7%)
Low GSH (<5 µmol/L)	36 (60%)	5 (8.3%)

[Table 10] shows proportion of patients with markedly reduced antioxidant levels.

[Table 1] shows that participants aged 46–60 years accounted for 40% in both the sepsis and control groups, confirming a comparable age distribution across the study arms. [Table 2] demonstrates male predominance in both groups (63.3% in the sepsis group vs 60% in controls), indicating balanced gender representation. [Table 3] indicates elevated inflammatory markers in septic patients, with a mean leukocyte count of 15.8 ± 4.2 ×10³/µL and CRP of 86 ± 28 mg/L, confirming active systemic inflammation. [Table 4] shows significantly higher MDA levels in the sepsis group (6.8 ± 1.5 nmol/mL) compared to controls (3.2 ± 0.9 nmol/mL), reflecting increased lipid peroxidation. [Table 5] demonstrates markedly reduced SOD activity in septic patients (1.8 ± 0.6 U/mL) compared to controls (3.6 ± 0.8 U/mL), indicating impaired enzymatic antioxidant defense. [Table 6] shows that catalase levels were significantly lower in the sepsis group (38 ± 9 U/mL) than in controls (64 ± 12 U/mL), suggesting decreased hydrogen peroxide detoxification capacity. [Table 7] indicates substantial depletion of reduced glutathione in septic patients (4.1 ± 1.2 µmol/L) compared to controls (8.5 ± 1.7 µmol/L), reflecting compromised non-enzymatic antioxidant reserve. [Table 8] shows a significantly elevated oxidative stress index in the sepsis group (3.8 ± 0.9) compared with controls (1.2 ± 0.4), highlighting a pronounced redox imbalance. [Table 9] shows a strong positive correlation ($r = 0.62$, $p < 0.001$) between MDA and CRP levels, suggesting that oxidative stress increases with the severity of inflammation. [Table 10] shows that more than half of septic patients exhibited markedly reduced antioxidant enzyme levels (56.7% low SOD, 46.7% low catalase, 60% low GSH). In contrast, only a small percentage of controls showed similar reductions.

Discussion

The current study is a prospective case–control study conducted over 18 months and included 60 patients with bacterial sepsis and 60 matched healthy controls. As shown in the current study, the data clearly indicate a well-established and marked imbalance between the oxidative and antioxidant components in infected patients and their well-matched control groups.[11]

In essence, sepsis is an overactive inflammatory response in the body triggered by an invading microorganism, leading to systemic damage to the body's tissues and organs. The most important mechanism that causes this tissue injury is the

extensive production of reactive oxygen species (ROS).[12] Activated neutrophils and macrophages produce superoxide and other reactive metabolites during/after their activity in fighting bacterial invasion during the early onset of sepsis. When the production of reactive oxygen species exceeds the body's physiological buffering capacity, oxidative stress occurs, resulting in cellular damage with both structural and functional components.[13]

Malondialdehyde was measured to assess oxidative stress in sepsis patients in the current study, and the results revealed a significant increase in malondialdehyde levels in septic patients compared with normal controls. Malondialdehyde has been extensively referenced as a marker of lipid peroxidation and an indicator of cellular membrane oxidative injury. The fact that septic patients have nearly double(more than two times) the normal reference range of malondialdehyde highlights the amount of membrane injury and oxidative stress in that population.[7]

When lipid peroxidation occurs in cells, it disrupts cell membrane integrity, alters the ability of cells to move ions across membranes, increases vascular permeability, and can even lead to multiple-organ involvement and injury in septic patients. An antioxidant defence system that has several enzymatic components, including superoxide dismutase, catalase, and others, was significantly reduced in septic patients. Oxidative stress caused by bacteria is a leading cause of death worldwide.[9] Research has shown that individuals who survive bacterial septic shock often have reduced oxidative stress, a result of sustained exposure to bacterial toxins. A recent study published in the journal Critical Care found that septic patients have significantly decreased superoxide dismutase (SOD) and catalase (CAT) enzyme levels due to exposure to high levels of reactive oxygen species (ROS) and the inability of their bodies to compensate.[14]

This suggests that the activity of SOD and CAT enzymes is reduced due to the extreme oxidative stress induced by bacterial septic shock. A significant decrease in SOD activity also creates an opportunity for excessive accumulation of superoxide radicals. Decreased CAT activity leads to increased hydrogen peroxide accumulation and enhances the generation of reactive hydroxyl radicals (OH) throughout the body.[15]

Glutathione (GSH), another important antioxidant found in the cytoplasm, was also significantly depleted in septic patients. Glutathione neutralizes free radicals and maintains redox balance. In septic patients, GSH depletion demonstrates a continuous source of oxidative stress and an increased need for GSH for detoxification.[16]

The results of the present study further supported the idea of a "normal" profile for an oxidative stress index in septic patients. Individuals who had an increase in the oxidative stress index also had a decrease in antioxidant levels. A significant correlation between malondialdehyde (MDA) and C-reactive protein (CRP) was identified in this study. In other words, the greater the level of oxidative stress experienced, the higher the level of inflammation.[17]

Inflammatory cytokines can induce ROS production, creating a positive feedback loop in which oxidative stress feeds back into the inflammatory process, leading to even greater oxidative injury. Therefore, the interplay between oxidative stress and inflammation contributes to the progression of disease and organ failure in septic patients.[18]

There are several clinical implications of these findings. First, the monitoring of oxidative stress-related markers may aid in better understanding the pathophysiological state of septic patients. In addition, the significant depletion of antioxidants suggests that adjuvant antioxidant therapy may serve as an adjunct to treat septic patients.[19] There have been conflicting reports concerning the use of antioxidants as therapeutic agents in patients with bacterial infection; therefore, knowledge of the extent of an individual's oxidative imbalance may enable clinicians to determine which patients will benefit from antioxidant therapy and when.[20]

Strengths of this study include the clearly defined case-control design, comparable patient demographics across both groups, and the simultaneous assessment of pro-oxidant and antioxidant markers. There are, however, some limitations to this study's results. First, the study did not categorize septic patients by illness severity (i.e., septic shock vs. sepsis). Stratifying patients by illness may alter levels of oxidative stress markers. Second, serial assessments were not performed during the study, limiting the assessment of real-time changes in oxidative stress markers. Finally, long-term outcomes were not measured against oxidative stress values.

Despite these limitations, the current study provides substantial evidence that bacterial sepsis is associated with increased oxidative stress and decreased antioxidant defenses. The current findings support the assertion that redox imbalance occurs during septic shock due to bacterial presence, and this imbalance may be a target for new therapeutic approaches.

Conclusion

This study aims to assess the relationship between oxidative stress, systemic inflammation, and the development of sepsis. 120 patients with sepsis were enrolled in this prospective, 18-month case-control study. Findings show that patients with sepsis had significantly higher levels of oxidative stress than healthy patients, as evidenced by elevated serum malondialdehyde (a marker of lipid peroxidation). Malondialdehyde is a product of the oxidative breakdown of fatty acids and indicates damage to cellular membranes. Additionally, all antioxidant mechanisms were significantly depleted, with lower levels of superoxide dismutase, catalase, and reduced glutathione noted in septic patients.

Furthermore, the elevated oxidative stress index indicates the presence of a marked amount of redox balance disturbance in patients with bacterial sepsis. In addition, there was a statistically significant positive correlation between malondialdehyde and inflammatory markers, indicating that oxidative stress plays a close role in the inflammatory process and the pathophysiology of sepsis. The present study supports the premise that oxidative stress is a major contributing factor in the etiology of sepsis and may serve as a potential biomarker of disease activity. However, additional longitudinal studies are necessary to determine the prognostic value of oxidative markers in conjunction with the possible therapeutic use of antioxidants for patients suffering

from septic-related complications.

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Conflicts of interest
There are no conflicts of interest.

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