Oxidative Marker Changes in COVID-19 Patients

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Received: 03-Feb-2022, Manuscript No. JHMR- 22-51885; Editor assigned: 05-Feb-2022, PreQC No. JHMR- 22-51885(PQ); **Reviewed:** 12-Feb-2022, QC No. JHMR- 22-51885(Q); **Revised:** 16-Feb-2022, Manuscript No. JHMR-22-51885(R); **Published:** 22-Feb-2022, DOI: 10.35248/jhmr.22.04(01).102

Abstract

Covid-19 is a major human infectious disease that has afflicted a number of countries. In the pathophysiology of Covid-19, the cytokine storm plays a key role. The link between inflammation and oxidative stress has been established for a long time. The goal of this study is to evaluate oxidative stress markers in Covid-19 patients to healthy participants.The pandemic of Coronavirus Disease 2019 (Covid-19) has quickly spread over the world, posing a serious threat to global health. This disease was discovered for the first time in Wuhan, China, in December 2019 . The pathogenic agent of Covid-19 illness is the severe acute respiratory Syndrome Coronavirus 2 (SARS-CoV-2), an enveloped RNA betacoronavirus. SARS-ease CoV-2's of transmission is a crucial factor in the virus's widespread dissemination. Covid-19's clinical course might range from asymptomatic to severe symptoms (acute lung injury), 3-4 days in the hospital, transfer to the Intensive Care Unit (ICU), and even death. The Covid-19 problem has drastically increased the probability of negative consequences in recent months. To reduce the risk of hospitalisation and mortality, it is necessary to discover complex pathogenic pathways of disease. Inflammation and cytokine production are frequently associated with respiratory virus infections. Covid-19 sufferers have been shown to have high levels of cytokines and chemokines in their blood. A cytokine storm ensued in certain cases, which is thought to be a critical element in the development of acute respiratory distress syndrome and multiple organ dysfunctions. Oxidative stress plays an important role in inflammatory processes; Reactive Oxygen Species (ROS) and H2O2 can activate NF-B, triggering the generation of inflammatory cytokines. The pathophysiology of SARS-CoV-2 infection is thought to be linked to oxidative stress. In this study, we compared healthy control volunteers to covid-19 patients and assessed oxidative indicators including TOS (total oxidant status), MDA (malondialdehyde), and antioxidant markers like CAT (catalase), SOD (super oxide dismutase).

Keywords: Covid-19 • Oxidative stress • Inflammation • Pathogenesis

Introduction

Assays in biochemistry

Following the collection of blood samples, routine laboratory tests such as CBC, C- Reactive Protein (CRP), and ESR were performed. Pishgaman Sanjesh kit was also used to evaluate serum Ferritin levels. Man business kit was used to measure serum albumin. A semi-quantitative latex agglutination approach was used to measure CRP (Bionik slide agglutination test kit). A Sysmex (Kolbe, Japan) SE 9020 analyzer was used to measure ESR and a Sysmex (Kolbe, Japan) SE 9020 analyzer was used to measure CBC [1,2].

Oxidative stress markers are measured

The Yagi technique was used to determine the level of MDA in the blood. The production of Thiobarbituric Acid (TBA) reactive compounds as a reaction product was detected using spectrophotometry at a wavelength of 532 nm. The serum TOS level was determined using Erel's technique. The levels of SOD and CAT in the serum were determined using commercially available ELISA kits, following the manufacturer's instructions (KIAZIST Life Sciences, Iran [3].

Several investigations have suggested a link between oxidative stress and the aetiology of Covid-19. This study shows that Covid-19 patients had higher levels of oxidative stress and inflammatory indicators in their blood, as well as lower levels of antioxidants, when compared to the control group, especially in ICU patients. In the case groups, we found a high level of MDA in the serum. MDA is a significant oxidative stress marker. Oxidative stress indicators and respiratory viral infection, particularly RNA viruses, have a strong relationship. Some viruses have been shown to alter the redox balance of cells *in vitro* and *in vivo* studies.

Discussion and Result

The onset of oxidative stress caused by virus infection (such as respiratory syncytial virus) is required for cytokine synthesis to activate innate immunity. Furthermore, several viruses cause oxidative stress, which aids virus reproduction inside the cell. The function of macrophage respiratory burst in response to Covid-19 infection, which can result in ROS generation, is explained. ROS/RNS overproduction has a role in lung tissue injury and epithelial barrier dysfunction caused by acute respiratory viral infections. NOX2 (NADPH oxidase 2) plays a crucial role in the formation of Reactive Oxygen Species (ROS), arterial dysfunction, and thrombosis (induced by platelet activation) [4]. Over activation of NOX2 has been found in COVID-19 patients.

In human alveolar type 2-like epithelial cells and small airway epithelial cells, viruses can decrease antioxidant systems such as superoxide dismutase, glutathione S-transferase, catalase, and glutathione peroxidase. Although significant levels of antioxidant enzymes were found, this could be a protective strategy against oxidative stress. Free radicals such as ROS and RNS are neutralised in large part by CAT and SOD. Zhu Z, et al., in agreement with our findings, found elevated levels of inflammatory markers such as ESR and CRP. COVID-19 individuals have decreased oxygen delivery to the tissues, as well as disseminated intravascular coagulopathy and sepsis. Hypoxia can produce reactive species like superoxide and H2O2, which can increase inflammatory cytokine expression. In COVID-19 patients, these interactions between oxidative stress and inflammatory cytokines can lead to multiple organ failures, exacerbating the illness. The existence of a "cytokine storm" in covid-19 patients is well documented [5].

There is a definite link between oxidative stress and the severity of a variety of viral infections. Our findings revealed that Covid-19 patients experience oxidative stress, which may exacerbate their disease. One of the benefits of this study is that it eliminates any underlying conditions that could influence oxidative stress. The pathogenesis of SARS-COV is complicated by an excess of free radicals and a deficiency in the antioxidant system [6]. The interaction between oxidative stress and cytokine storm, on the other hand, may have a considerable impact on the severity of Covid-19 patient symptoms.

Patients group were divided into ICU and Non-ICU groups. ESR, CRP and serum level of ferritin were significantly higher in case group. Serum level of albumin was significantly lower in Covid-19 patients. Serum MDA and TOS was significantly increased in Covid-19 patients [7]. Also, Covid-19 patients had higher serum activity of CAT and GPX. The case group was separated into two groups based on clinical characteristics and Intensive Care Unit (ICU) admission: ICU and Non-ICU patients. The age differences between the control group (51.5 13.58 years), non-ICU patients (57.36 10.87 years), and ICU patients (55.5 15.65 years) were not statistically significant (p >0.05). All Covid-19 patients got acetaminophen codeine, enoxaparin, azitromicina, dexamethasone, dextromethorphan, kaletra, naproxen, Vitamin D, and pantoprazole, whereas the control group received no medication. Ten patients required admission to the intensive care unit (ICU) for treatment. Fever (79.6%), cough, and nausea were the most prevalent complaints on admission (62.5%). 79% of people had respiratory distress, and oxygen saturation (Spo2) is less than 88%

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Cite this article: Cullen S. Oxidative Marker Changes in COVID-19 Patients. J Health Med Res. 2022, 04 (1), 102.