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Original research article

Inflammatory markers between cases of severe and non-severe COVID-19 infection at admission

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Abstract
The phenomenon known as a "cytokine storm" is characterised by a surge in the production of a large number of cytokines, which are known to induce lung tissue fibrosis as well as damage that persists over time. This condition may be recognised by the fact that there is an increase in the overall quantity of cytokines that are released into the circulation. It is not quite clear whether these shifts came about as a result of the immunomodulation that the medicine delivered or whether they were the result of the illness process itself. In addition, more research is required to shed light on the probable relationship that exists between these inflammatory markers and the progression and severity of COVID-19. This connection has not yet been established. The purpose of this study was to describe any changes that may have occurred in the inflammatory markers of symptomatic COVID-19 patients and to establish a connection between these changes and the severity of the disease and its prognosis.
Keywords: Inflammatory markers, severe, non - severe, COVID-19

Introduction
The condition that was eventually identified as COVID-19 that was discovered in December 2019 in Wuhan, China, was linked to a roughly 2% increase in the likelihood of passing away. This information was found in association with the disease. [1]. The Severe Acute Respiratory Syndrome Coronavirus 2, often known as SARS-CoV-2, is the infectious agent that is responsible for producing this sickness [2]. This virus was just recently found. The coronavirus disease known as SARS-CoV was found for the first time in China in 2002, and the coronavirus infection known as Middle East Respiratory Syndrome (MERS-CoV) was found for the first time in Saudi Arabia in 2012 [3, 4]. Both of these illnesses were shown to be caused by coronaviruses. These coronaviruses are communicable between humans, animals, and other individuals [5]. Each one of these coronaviruses is encapsulated and has a positive strand of RNA. They were discovered in bats first, and it was determined that bats were responsible for their proliferation. Despite the fact that they exhibit similar clinical signs, recent research has shown that these two conditions are distinct in a number of important respects [5]. The first stage of the illness is brought on by viral replication, and it is possible that this stage will be followed by a second stage that is brought on by an inflammatory reaction from the host [6]. Both phases are defining characteristics of the condition. The radiological indications that are typical of a SARS-CoV-2 infection can be used to derive the hyperimmune response that is associated with acute respiratory distress syndrome [7]. A situation known as a "cytokine storm," which is characterised by an increase in the release of numerous cytokines that cause lung tissue fibrosis and long-term damage, may occur in the most critically sick persons [8]. A "cytokine storm" is characterised by an increase in the release of many cytokines that produce lung tissue fibrosis and long-term damage. The phenomenon known as a "cytokine storm" is characterised by a surge in the production of a large number of cytokines, which are known to induce lung tissue fibrosis as well as damage that persists over time. This condition may be recognised by the fact that there is an increase in the overall quantity of cytokines that are released into the circulation. It is not quite clear whether these shifts came about as a result of the immunomodulation that the medicine delivered or whether they were the result of the illness process itself. In addition, more research is required to shed light on the probable relationship that exists between these inflammatory markers and the progression and severity of COVID-19. This connection has not yet been established. The purpose of this study was to describe any changes that may have occurred in the inflammatory markers of symptomatic COVID-19 patients and to establish a connection between these changes and the severity of the disease and its prognosis. The variance in the levels of inflammatory markers was another objective of the experiment that was being carried out.

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Aims and Objectives

To do a comparison and draw some conclusions about the levels of inflammatory markers that were present in patients with severe and non-severe Covid-19 infections when they were admitted.

Materials and Methods

Our institution's Institutional Ethics Committee was the first to approve the study before it was carried out. In addition to this, authorization was obtained not only from the District Medical Officer but also from the medical superintendents of the respective hospitals. Patients who were seen at our hospital's flu clinic and who fulfilled both the inclusion and the exclusion criteria were approached with an invitation to take part in the research. The individuals who took part in the research were given the opportunity to provide their thoughts on the objectives of the study. Written informed permission was obtained from all of the participants in the study who agreed to take part in the research. After receiving the participant's consent, relevant information was collected. Patients were classified as having either category A or category B infection according to the criteria set forth by the Indian government. These criteria were based on the degree to which the patient was affected by the Covid-19 virus. Group A consists of those who have no symptoms at all or very few symptoms and includes asymptomatic people as well.

Patients in the category B exhibit signs and symptoms of mild to moderate pneumonia, but no indications of severe illness; their respiratory rate is between 25 and 30 cycles per minute, and their SPO2 is between 90 and 94% when tested in room air. Patients in this category are not at risk for developing severe pneumonia.

Symptomatic patients with severe pneumonia who have a respiratory rate of more than 30 cycles per minute or a pulmonary oxygen saturation of less than 94% when using oxygen; patients with ARDS and septic shock also fall into this category (confusion, drowsiness, decrease in urine output, lower blood pressure, and tachycardia). Patients with severe pneumonia who have a respiratory rate of more than 30 cycles per minute or who have a pulmonary oxygen saturation of less than 94% when using oxygen.

On the day that each patient was admitted, standard blood tests were performed on them. These tests included a complete blood count (CBC), liver function tests (LFT), renal function tests (RFT), and senior electrolytes.

Results

Table 1: Mean comparison of inflammatory markers between cases of severe and non-severe Covid-19 infection at admission

Variables (admission)	Severe Covid	N	Mean	SD	p- value
Eosinophils (%)	No	30	1.34	1.64	<0.01
	Yes	30	3.4	0.2	
CRP (mg/L)	No	30	51.4	66.8	<0.01
	Yes	30	124.0	105.1	
LDH (U/L)	No	30	386.5	221.8	0.042
	Yes	30	586	169.3	
Ferritin (ng/ml)	No	30	764	1385.3	0.530
	Yes	30	910.02	605.8	
D-Dimer (mg/L)	No	30	2.97	23.6	0.710
	Yes	30	4.15	2.6	

Mean eosinophil count at admission was significantly lower in cases with severe Covid-19 infections (0.2% vs 1%; $p<0.01$) while CRP levels (125 vs 52.4 mg/L) and LDH levels (465.5 vs 388.5 U/L) were higher significantly ($p<0.05$).

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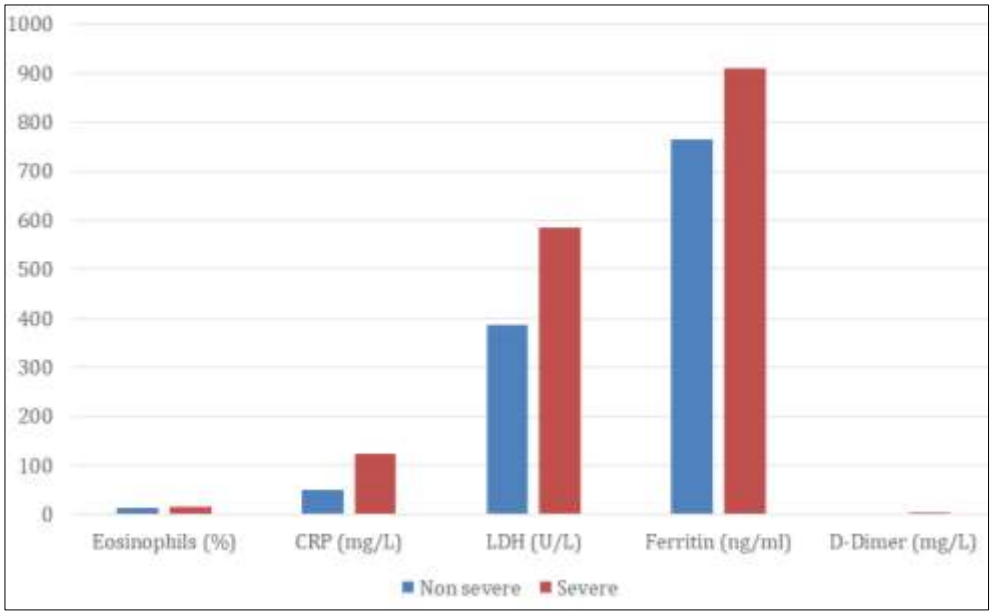

Graph 1: Mean comparison of inflammatory markers between cases of severe and non-severe Covid-19 infection at admission

Discussion

We are all currently suffering through a pandemic that is being produced by the coronavirus. The Severe Acute Respiratory Syndrome Coronavirus 2, often known as SARS-CoV-2, is the infectious agent that is responsible for producing this sickness [2]. This virus was just recently found. The first stage of the illness is brought on by viral replication, and it is possible that this stage will be followed by a second stage that is brought on by an inflammatory reaction from the host [6]. Both phases are defining characteristics of the condition. A situation known as a "cytokine storm," which is characterised by an increase in the release of numerous cytokines that cause lung tissue fibrosis and long-term damage, may occur in the most critically sick persons [8]. A "cytokine storm" is characterised by an increase in the release of many cytokines that produce lung tissue fibrosis and long-term damage. This disorder is distinguished by a rise in the total quantity of cytokines that are discharged into the bloodstream. Instances that entail serious infections are related with a significantly increased risk of morbidity as well as fatality. Therefore, in order to accurately estimate the prognosis of a patient's condition, one needs a diagnostic marker that is not only simple to compute but also easily accessible and contains a sufficient level of diagnostic accuracy. Recent studies have focused their attention on severe indicators such as neutrophils, lymphocytes, ESR, CRP, ferritin, LDH levels, D-dimer, and IL-6. In this context, these indicators have been the primary focus of study. These are only some of the many indications that have been taken into consideration. It is not quite clear whether these shifts came about as a result of the immunomodulation that the medicine delivered or whether they were the result of the illness process itself. In addition, further research has to be done in order to shed light on the probable relationship that exists between these inflammatory markers and the development of COVID-19 as well as its severity. As a consequence of this, the goal of this study was to investigate the accuracy of the inflammatory markers as a predictive indication for severity in the COVID-19 patients and make comparisons. Patients diagnosed with COVID-19 were recruited for the research and asked to provide their CRP, LDH, ferritin, and D-dimer levels.

Conclusion

More research is needed to shed light on the possible link that exists between these inflammatory markers and the development and severity of COVID-19 from different geographical locations in order to shed light on the possible relationship that exists between these inflammatory markers and the development and severity of COVID-19.

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