ISSN: 2573-1734

Covid-19 and Sudden Death with Pulmonary Manifestations- A Case Report

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Case Report

Volume 5 Issue 4

Received Date: November 19, 2020 **Published Date:** December 09, 2020

DOI: 10.23880/ijfsc-16000211

Abstract

Covid 19 being a pandemic has uniformly and bizarrely affected the globe in a most unpredictable manner. So it becomes very important to unravel the transmission dynamics and the effect of the virus which has made it a deadly virus so far. Covid 19 is a member of coronavirus family, which also includes the SARS virus (Severe Acute Respiratory Syndrome) and MERS (Middle East Respiratory Symptoms) virus. In September 2020, 52 years old was brought in via auto to the emergency department from his home with shortness of breath and a high-grade fever. He was a never smoker and a Teacher by profession. He was a known handicap since many years. On examination, he was declared dead at emergency department. His postmortem rapid antigen test negative. Autopsy studies of patients who died from ARDS are limited. In the only complete report available, we described lung injury characterized by exudative diffuse alveolar damage, pneumocyte hyperplasia, and septal inflammatory infiltrate.

Keywords: Covid 19; Pneumonia; Histopathology Report

Abbreviations: SARS: Severe Acute Respiratory Syndrome; MERS: Middle East Respiratory Symptoms; RBCs: Red Blood Cells.

Introduction

Covid 19 being a pandemic has uniformly and bizarrely affected the globe in a most unpredictable manner. So it becomes very important to unravel the transmission dynamics and the effect of the virus which has made it a deadly virus so far. Covid 19 is a member of coronavirus family, which also includes the SARS virus (Severe Acute Respiratory Syndrome) and MERS (Middle East Respiratory Symptoms) virus. The family of Coronavirus includes virus strains that the common cold and flu are caused [1].

Studies suggest that Covid 19 is a close relative of SARS.

SARS is a novel type of virus reported in 2007, and similar to most of SARS viruses, Covid 19 commonly affects the lungs in humans. The infection usually starts as a flu-like picture or no symptoms, and progressing to severe symptoms.

Covid 19 affects all organs of the body, primarily being the lungs. Severe cases of covid fatalities is due to ARDS and pneumonia but such a severity is not seen in all clinical spectrum of cases.

Mild symptoms will be observed in majority of cases ie.80%, while out of the remaining, only14% will exhibit pneumonia, 5% will exhibit septic shock and organ failure (mostly respiratory failure) and extreme fatalities will be seen in only 2% of cases [2].

Covid19 infected persons predominantly exhibit fever,

dizziness, breathlessness, headache, dry cough (eventually result in phlegm) and in some cases, there will be loss in smell and taste, gastrointestinal symptoms like diarrhea and fatigue are also reported in some cases.

So like any other viral infection, the disease is cured with time but it is significant to understand that the infection can take a fatal course, if the individual is suffering from any underlying illnesses like hypertension, diabetes, cardiac problems, respiratory issues or on any immune-suppressing medications. Older individuals are at higher risk, as they have deranged immunity levels coupled with vulnerability or predisposition to other illnesses. A significant and vital aspect in Covid 19 pathophysiology is its effect on various body system, especially the lungs [3].

Covid 19 predominantly affects the lungs damaging the alveoli (tiny air sacs). Alveolar units in the lungs facilitates transfer of oxygen to the blood vessels. The blood vessels or capillaries function oxygen transports to the RBCs (Red blood cells). All the internal organs in the body are perfused by oxygen rich RBC's finally.

The virus damages the wall and the lining of the alveolus and capillaries. The resultant debris consequent upon damage, which primarily are plasma proteins in nature, accumulates on the alveolus wall thereby thickening the lining. Due to wall thickening and deposition, fibrosis develops and the transfer of oxygen to the red blood cells is impaired. Transfer of oxygen to the red blood cells is mainly proportional to the amount of fibrosis or thickening of the wall developed, which predisposes to breathlessness as the body is running short of oxygen. Consequent to lack of oxygen, all the internal organs of body are deficiently and inadequately perfused. As a result, at this juncture, the body struggles to increase oxygen intake for want of adequate oxygen [4].

Case Report

In September 2020, 52 years old was brought in via auto to the emergency department from his home with shortness of breath and a high-grade fever. He was a never smoker and a Teacher by profession. He was a known handicap since many years.

On examination, he was declared dead at emergency department. His postmortem rapid antigen test negative. The body was subjected postmortem after receiving the inquest report from concerned police.

Post Mortem Examination

Deceased body was taken to Post-Mortem at Department

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On opening the chest cavity, heart and pericardium was intact, with normal weight, no signs of gross blood clot in the coronaries. Left lung weighs 600gms and right lung weighs 700gms. The heart and both lungs were subjected to histopathology examination.

On opening the abdomen cavity, all the organs were intact and weighing normally, no signs of hemo and pneumoperitoneum. No evidence of organ injuries.

On opening the skull cavity, there were no signs of intracerebral and intracranial hemorrhages. Cut section of brain was congested.

Blood and viscera were forwarded to Forensic Science Laboratory, at Bangalore, but it came negative for any kind of poison.

Histo-Pathology Report

Bilateral lung parenchymae shows diffuse interstitial fibrosis with intra alveolar edema. Few alveoli are ruptured and show hyaline membrane change with fibrin deposit. Also seen are proliferations of pneumocytes with few showing reactive changes. Also seen are few microthrombosis and macrothrombosis within blood vessels. Interstitial spaces show accumulation of mononuclear inflammatory cells infiltrate.

Right coronary artery shows grade I atheromatous change.

Left coronary artery shows grade V atheromatous change with calcification.

LADA shows grade V atheromatous change with calcification.

Cause of Death

Death is Due to Cardio-Respiratory Failure Consequent upon Bilateral Pneumonia.

Discussion

Coronaviruses are enveloped, positive sense, single-stranded RNA viruses, which can infect humans and a broad range of animals [1]. Beta-coronaviruses causes severe disease in humans SARS-CoV-2 comprises part of the B lineage of beta-coronaviruses, causing COVID-19 (coronavirus disease 2019) [2]. The present outbreak is nomenclature as SARS-COV -2 which initially originated

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in the Hubei province of People republic of China and later spread globally as a deadly pandemic. SARS-CoV-2 mainly affects the epithelial cells of the alveoli through receptormediated endocytosis using the angiotensin-converting enzyme II receptors widely present in the lungs [3]. SARS-CoV-2 presents with an array of clinical signs and symptoms in the initial stages. Patients may exhibit asymptomatically to having only isolated gastrointestinal tract symptoms [4]. Roughly 14% (13.8%, n = 44,672) of patients go on to develop severe disease, requiring hospitalization with or without oxygen support, and approximately 5% having a requirement of intensive care unit support management [5,6]. In COVID-19 symptomatic Patients, clinical picture usually is of fever, nasal stuffiness, fatigue, dry cough, and other features of respiratory infections. COVID-19 infected patients usually exhibit symptoms within one week. Assessment of asymptomatic nature in COVID-19 infected cases has not yet been accomplished in terms of percentage. The observational studies conducted to date reveals that the mean incubation period is five days, and a median incubation period to three days. Symptomatic patients usually develop pneumonia by the second to third week which may progress to a severe disease. COVID-19 classified as mild, moderate and severe illness depending on the signs, symptoms and investigations. Sometimes patients may have uncomplicated upper respiratory tract symptoms in a mild disease or some Adults may go on to develop pneumonia without any signs of progression to severity or fatalities observed.

Conclusion

Autopsy studies of patients who died from ARDS are limited. In the only complete report available, we described lung injury characterized by exudative diffuse alveolar damage, pneumocyte hyperplasia, and septal inflammatory infiltrate.

In spite of vast number of deaths from covid pneumonia and thereby the relevance of lung involvement in patients with COVID-19, data is limited regarding lung pathology .The importance of respiratory severity and progression in covid-19 cases along with possibility of other organ involvements are being studied throughout the world currently .Observations from a case report of a patient who died from COVID-19 in China revealed the histological findings in the lungs like desquamation of pneumocytes, diffuse alveolar damage, and edema typical of lung damage and compromise. In addition, Tian and colleagues described the pulmonary pathology of early-phase COVID-19 in two patients with lung carcinoma; both patients showed signs of the exudative phase of diffuse alveolar damage.

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