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Research Article

Covid-19's Effect on Cardiovascular and Respiratory Health in Young Adults Who Have Recovered from Covid

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Abstract

Animals and people can contact Corona viruses, one kind of virus. They have a high death rate because they might affect the pulmonary and extra-pulmonary systems. This study aims to investigate the effects of COVID-19 on respiratory and cardiovascular function in young adults who have recovered from the virus. We searched several databases, including Google Scholar, Medline, PubMed, and Research gate, using terms like "impact of COVID-19 on young adults," "SARS-CoV-2," "cardiovascular status," and "respiratory status" to create a narrative on this subject. It was discovered that while most mild and moderate cases of acute respiratory distress syndrome recover fully, a small number of severe cases continue to be hypoxemic even after receiving appropriate care, and that patients with COVID-19 symptoms have lower VO2 max values than those without symptoms. There is much debate concerning the coronavirus's long-term impact on cardiopulmonary function. It is anticipated that a subset of patients may exhibit cardiovascular problems that are both subclinical and potentially visible. Even in patients who appear to have restored cardiac function, ventricular arrhythmias, atrial fibrillation, and CAD are possible risks. Patients who have recovered but still have poor respiratory health are at a higher risk of developing heart and respiratory problems in the future.

Keywords: Young adults, Pulmonary injury, Respiratory status, Cardiovascular status, and Cardiac injury.

Introduction

The global pandemic COVID-19 is having a significant effect on the healthcare system everywhere. A cluster of pneumonia cases linked to an unknown microbial agent in Wuhan were reported by China in December 2019, and it was established that the illness had spread and was transmissible from person to person [1-3]. This new coronavirus, also known as SARS-CoV-2 (severe acute respiratory syndrome 2), is the infectious diseases etiology. The elderly were the group most impacted by the COVID-19 outbreak at first, and as it spread, so did the number of illnesses among adults and children [4,5]. It is deadly for all age groups and has resulted in major consequences [3-5]. The coronavirus is known to impact nearly every organ in the body, including the pulmonary system. However, it mostly affects the lungs, causing fever and dyspnea as early signs and eventually decreasing respiratory function [3-7]. The severity of COVID-19 infection symptoms varies, and the infection site affects both the disease's symptomatology and prognosis. Muscle soreness, muscle dysfunction, weakness, fever, chills, headache, dyspnea, diarrhea, vomiting, weight loss, and so forth are typical symptoms [1-10]. The long-term effects of COVID-19 on the respiratory and cardiovascular status of young people (18-25 years of age) have not been investigated. Extensive research has been done on the impact of COVID-19 on the pulmonary and extrapulmonary organ systems in bigger age groups and at midterm follow-up. Because of how severe COVID-19 is, people who have recovered from it are more likely to experience cardiorespiratory problems in the future.

Methodology

We searched for papers, literature, and different articles or cases for original and review articles about the effect of COVID-19 on cardiopulmonary function that had been published since the start of COVID-19 by looking through several databases, including Google Scholar, Medline, PubMed, and Research gate. The search was conducted using a combination of terms such as young adults, pulmonary injury, heart, lungs, cardiac system, respiratory system, cardiac status in young adults, myocardial injury, and pathophysiology and COVID-19, COVID, SARS-CoV-2, cardiac, cardiovascular, cardiovascular function, cardiovascular status, cardiac injury, respiratory tract, respiratory function, respiratory status, pulmonary injury, and several other terms.

Discussion

After a thorough analysis, we discovered that COVID-19 had a significant impact on a sizable population, with the young adult demographic being mainly ignored and their lack of symptoms playing a significant influence. There are several long-term consequences, but two of the most prevalent ones are respiratory and cardiovascular problems. Patients who have recovered from COVID-19 frequently experienced decreased work capacity, easy fatigue, dyspnea during exercise, and decreased respiratory endurance; these symptoms combined increase their risk of future respiratory and cardiac problems [3-5,7,8,10-13]. SARS-CoV-2 first infects the respiratory system, but it can also have a variety of effects on the cardiovascular system, including the induction of severe myocarditis and inflammation of the heart [14]. Because SARS-CoV-2 binds to the functional receptor ACE-2 (Angiotensin Converting Enzyme) on pulmonary epithelial cells, the virus has a higher chance of infiltrating and destroying lung epithelial cells [3,4,6,11-17]. Significant amounts of ACE2 are seen in the kidneys, bladder, ileum, heart, and lungs [4,5,12-17].

Recent investigations [18,19] suggested that COVID-19-recovered people also displayed varying degrees of functional, musculoskeletal (e.g., fatigue, weight loss, joint pain), radiologic, and psychosocial abnormalities. Similarly, *H Ahmed, et al.,* revealed that patients had decreased exercise ability (6-minute walk test) and impaired DLCO at 6-month follow-up. Severe illnesses were associated with higher d-dimer, fibrinogen, and delayed thrombin time, as well as a significant rate of multi-organ failure [20]. While some other studies show no change in cardiopulmonary function three or four months after infection when compared to pre-infection examinations, in their retrospective studies that COVID-19 influences cardiopulmonary function [19,21]. However, compared to non-severe patients, serious patients had a higher prevalence of pulmonary fibrosis, certain restrictive ventilatory anomalies, and impairment of the lungs' capacity to diffuse carbon monoxide (DLCO), according to a study on 110 hospitalized patients at the time of release. Depending on the kind of heart injury, there can be major repercussions if there is persistent inflammation or fibrosis [18-20,21].

According to review study, once a virus attaches itself to the ACE2, it rapidly activates pathogenic T cells and triggers the large production of neutrophils and inflammatory cytokines like IL-6, which in turn triggers the release of even more inflammatory cytokines. The inflammatory cascade is strengthened by this process. Although T lymphocytes and neutrophils can kill viruses, they can also cause cardiac damage, making them two-edged swords [16]. Opacification of ground glass was seen in CT images. Patients with symptoms had a lower VO2 max than those without symptoms. A prior investigation on 237 hospitalized patients found that 40% of the cases had cardiovascular abnormalities, and similar findings were also observed in 60% of the cases involving changes in glucose metabolism in patients with respiratory infections [22]. Myocardial infarction, arrhythmias, and disturbance of the myocardial oxygen demand-supply relationship are among the potential acute and direct cardiac injuries caused by SARS-CoV-2 [5,7,14,16,18,20,21]. Patients with COVID-19 had decreased lymphocytes and elevated troponin levels.

A notable rise in Cardiac Troponin I (cTnI) of 36% was observed in patients with severe illness compared to non-severe patients, according to a recent meta-analysis of 4 studies that examined cTnI levels in 341 COVID-19 patients. In a similar vein, a research of 1591 patients in Italy found that 49% of patients had hypertension, 21% had CVD, and 18% had hypercholesterolemia; these patients also needed greater PEEP (positive end expiratory pressure) levels while they were in the hospital [23]. Certain patients' Cardiac Magnetic Resonance Imaging (CMR) revealed early signs of scarring, myocardial inflammation, and enlarged hearts because of inadequate pumping [14,16-21]. During physical activity, there may be subtle cardiac consequences that could be fatal, such as arrhythmia, heart failure, cardiac arrest, cardiogenic shock, and pericardial effusion, as reported in several case reports involving hospitalized patients [16-23]. Comorbidities like hypertension, cardiovascular disease, pulmonary illnesses, and Acute Respiratory Distress Syndrome (ARDS) have been associated with unfavorable outcomes [4], particularly in smokers and drinkers. The detrimental effects of these behaviors on cardiopulmonary immune function make the progression of the disease unsurprising. Previous research shown that, in comparison to non-drug users, smokers, drinkers, and substance users were more likely to experience severe COVID-19 symptoms that could require ICU assistance, mechanical breathing, or even result in death [24-26].

A tiny proportion of severe cases of acute respiratory distress syndrome remain hypoxemic even after receiving the necessary care, even though the majority of mild and moderate cases of the condition recover fully. According to *Fabio Anastasio, et al.,* patients who developed ARDS and impaired DLCO after hospitalization and whose 6MWT, full spirometry, and Cardio-Pulmonary Exercise Testing (CPET) revealed residual pulmonary and functional impairment need respiratory therapy and progressive physical activity [3]. Chest physiotherapy, including coughing exercises, diaphragmatic training, stretching exercises, and at-home exercises, has been

shown to enhance FEV1, FEV1/FVC percent, and carbon monoxide Diffusing Lung Capacity (DLCO%), according to *Huang, et al.,* It also lessens the symptoms of anxiety and depression and enhances life expectancy and quality [13].

Future Research Focus

In the future, this research might be conducted experimentally, requiring participants to undergo PFT exams and using the results to support the clinical presentation. Studying's limitations Might have been conducted as an experimental study little information was known about young adults. The symptoms that had already developed from a previous exposure coincided with the COVID second wave.

Conclusion

The symptoms of corona virus disease (COVID-19) can range greatly, from acute sickness and death to asymptomatic. There is much debate over the long-term impact of coronavirus on cardiopulmonary function. Consideration should be paid to cardiopulmonary protection when treating COVID-19. It is anticipated that a subset of patients may exhibit cardiovascular problems that are both subclinical and potentially visible. Even in patients who appear to have restored cardiac function, arrhythmias (ventricular or atrial fibrillation) or CAD (Coronary Artery Disease) could still occur.

Acknowledgements

None.

Conflict of Interest

None.

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