ASSESSMENT OF CAUSE OF DEATH AND INTERNAL ORGANS OF HUMAN BODIES OF COVID-19 PATIENTS RECEIVED FOR AUTOPSIES TO A TERTIARY CARE HOSPITAL OF LAHORE.

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Abstract

Background and Objective: COVID-19 causes extensive effects on virtually all organs. It causes inflammation, endothelitis, vasoconstriction, hypercoagulability, and edema. Different organs may be affected at different times. Hence we aim to determine the cause of death and pattern of the injuries to the internal organs among the dead bodies of patients diagnosed with coronavirus disease.

Methods: This Cross-sectional study was conducted in the Department of Forensic Medicine, Allama Iqbal Medical College, Lahore over 1-year period from 2021 to 2022. 150 autopsies of COVID-19-positive patients who died from Covid, during the peak era were received. Autopsies were performed and internal organs were carefully examined clinically along with histopathological evidence. Reports were assessed and the presence or absence of single or multiple organ dysfunction was recorded. The data was recorded in a proforma and entered and analyzed in SPSS version 25.

Results: The mean age of dead bodies at the time of death was 54.5 ± 14.73 years. 112 (74.7%) of these patients were males while 38 (25.3%) were females. The mean duration of COVID-19 was 14.22 ± 9.41 days and the mean duration of death until the presentation of the body for autopsy was 21.89 ± 6.37 hours. Out of 150 cases, death due to respiratory failure was observed in 67 (44.7%) cases, renal failure in 21 (14.0%) cases, liver failure in 18 (12.0%) cases, Venous thromboembolism in 16 (10.7%) cases, meningitis in 10 (6.7%) cases, intestinal perforation was observed in 9 (6.0%) cases, in 5 (3.3%) cases, peritonitis was observed and cardiac failure in 5 (3.3%) cases.

Conclusion: There are higher chances of organ failure in patients suffering from COVID-19, as proven by autopsies of COVID-19 cases.

Key words: Coronavirus disease, liver, lungs, cardiovascular diseases, cerebrovascular changes

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The COVID-19 epidemic, which originally surfaced in Wuhan, China, is currently affecting millions of people worldwide. This is a review of the widespread impacts of COVID-19 on almost every organ. Edema, endothelialitis, vasoconstriction, hypercoagulability, and inflammation are all results of it.[1,2] Disseminated intravascular coagulation, elevated D-dimer and fibrin degradation products, and lymphocytopenia are all seen. There have been reports of myocardial infarction, ischemic stroke, venous thromboembolism, deep vein thrombosis, systemic and pulmonary arterial thrombo-embolism as well as pulmonary embolism has also been reported.[3,4]

Patients who are critically unwell frequently have multiple organ dysfunction. Angiotensin-converting

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enzyme 2 receptors can be found in various tissues like heart, brain vascular endothelial cells, liver, kidneys, pharynx, colon, and some other organs aswell.[5,6] It may harm these organs directly. Organ malfunction can be the result of direct response to systemic invasion by virus itself. It is very important to look for multiple organ damage while managing a patient in hospital. Vascular endothelial and coagulation disturbances are frequent, but they may be symptomless intially. They contribute to organ damage in many ways. Renal and cardiac problems have frequently been reported among patients who passes away early during the disease while after the initial illness is over, other organ damage may still be appreciable. Organs may experience different effects at various periods.[7,8]

A preliminary investigation found evidence of organ damage four months after the initial infection in young, low risk patients with covid-19 symptoms.[9] Primary data from 201 patients indicate that four months following the onset of their SARS-CoV-2 infection symptoms, about 70% of them had abnormalities in one or more organs.10 After the advent of the COVID-19 pandemic, most countries have modified some of their during this pandemic most of the countries have changed some parts of their health-related laws. The postmortem of a deceased person, however, hasn't involved this because it has a legal component. Therefore, whether intentionally or unintentionally, the deceased bodies posed a risk of COVID-19 exposure to the healthcare professionals. establish a method of autopsy that prevents droplet spreads, especially in mortuaries with inadequate biosafety equipment.[11]

We conducted this study with the aim to find the pattern of different organs damage in deceased due to coronavirus disease. It has been observed in routine that COVID-19 is a deadly virus and cause damage or dysfunction of internal body including blood and organs. But the evidence for local population is missing. Therefore, we conducted this study to get local data, whether the pattern of organ dysfunction in COVID-19 autopsies. So that in future we can apply results collected from our study.

METHODS

This Cross-sectional study was conducted in the Department of Forensic Medicine, Allama Iqbal Medical College, Lahore. Over a 1-year period from 1-10-2021 to 1-10-2022. Non-Probability, Consecutive Sampling was used to recruit 150 autopsies (calculated with 95% confidence level, 7.5% margin of error and percentage of organ dysfunction i.e. 70%[12] in autopsies of COVID-19 dead bodies) of 5-80 years, either gender, deceased cases of COVID-19.while bodies received 48 hours after death, or old cases of Systemic lupus erythematosus, chronic kidney disease, liver failure, gangrene or death due to poison ingestion were excluded. An informed consent was taken from attendants, the demographic data of the deceased including his/her age at death, gender, history of diabetes, hypertension, duration form COVID-19 and its severity was noted from medical record. Then autopsy was performed and blood samples were taken to assess hematological parameter. Body tissues were also obtained from internal organs for clinical and histopathologically examination. Reports were retrieved and presence or absence of organs dysfunction either single organ or multi-organ dysfunction was noted. This whole data was recorded into proforma while later on entered and analyzed in SPSS version 25. Age, duration of COVID-19, were quantitative variables that were provided as mean and standard deviation. The frequency and percentages were calculated for qualitative characteristics, such as gender, organ dysfunction.

RESULTS

The mean age of all deceased was 54.5 ± 14.73 years at the time of their death. 112 (74.7%) of them were male while 38(25.3%) were females, giving a ratio of male to female as 3: 1. Diabetes was positive in 82 (54.7%) cases, hypertension was reported in 97 (64.7%) cases, smoking in 51(34.0%) cases. The mean duration of COVID-19 was 14.22 ± 9.41 day and mean duration of death until presentation of body for autopsy was 21.89 ± 6.37 hours. Out of 150 cases, 102 (68.0%) had death in the hospital while admitted for severe con-

dition and 48 (32.0%) cases died at home. Table # 1

Out of 150 cases, death occur due to respiratory failure was observed in 67 (44.7%) cases, renal failure in 21 (14.0%) cases, liver failure in 18 (12.0%) cases, Venous thromboembolism in 16(10.7%) cases, meningitis in 10 (6.7%) cases, intestinal perforation was observed in 9(6.0%) cases, in 5(3.3%) cases, peritonitis was observed and cardiac failure in 5 (3.3%) cases. Table # 2.

Table 1: *Socio-demographic profile of the deceased patients (n=150)*

CHARATERISTICS	Mean ± SD, f (%)
Age (in years)	54.5 ± 14.73
Gender	
Male	112 (74.7%)
Female	38 (25.3%)
History of Diabetes	82 (54.7%)
Hypertension	97 (64.7%)
Smoking	51 (34.0%)
Duration of COVID-19	14.22 ± 9.41
Duration of death	21.89 ± 6.37
Place of death	
Hospital	102 (68.0%)
Home	48 (32.0%)

Table 2: *Cause of death and Organ involve in COVID-19 related mortality (n=150)*

Type of Organ damage	f (%)
Respiratory failure	67 (44.7%)
Renal failure	21 (14.0%)
Liver failure	18 (12.0%)
Venous thromboembolism	16 (10.7%)
Meningitis	10 (6.7%)
Gastrointestinal complications	
Intestinal Perforation	9 (6.0%)
Peritonitis	5 (3.3%)
Cardiac failure	4 (2.7%)

DISCUSSION

The end of 2019 saw the emergence of COVID-19, which is only partially understood, and there is a paucity of knowledge regarding pathophysiology, epidemiology, treatment, and long-term results.[13] Early on in the pandemic, it was discovered that this unique infectious disease had a different clinical course than other viral infections like influenza. Apparently, SARS-CoV-2 is more virulent and contagious than influenza virus. frequently COVID-19 is associated with severe illness involving lung injury, which usually culminates in death.[14-16]

Given the large number of these patients, it was vital to develop a successful strategy for looking at every facet of COVID-19. Although autopsy rates have significantly decreased over the past few decades, they still provide the chance to personally examine every organ, determine death cause and produce samples of additional research.[17] This is also true for SARS-Co2 disease, a disease with insufficient treatment options and limited understanding.[13] SARS-CoV-2 has the ability to invade beyond the lungs and even pass the blood-brain barrier, causing multi-system sickness. This is likely the origin of the cytokine storm, while it is unclear whether organ damage is due to direct virus-caused harm or the outcome of an inflammatory assault.[18]

Out of 150 cases, the major cause of death in our study sample was respiratory failure [67 (44.7%) cases], followed by renal failure in 21(14.0%) cases, liver failure in 18 (12.0%) cases, Venous thromboembolism in 16(10.7%) cases, meningitis in 10(6.7%) cases, intestinal perforation was observed in 9(6.0%) cases, in 5(3.3%) cases, peritonitis was observed and cardiac failure in 5 (3.3%) cases.

In a related study, Wichmann et al. discovered a range of 52 to 87 years and 73 years as a median, similarly he also found that there were more male (75%) as compared to females, and that the majority of deaths took place in hospitals (83.3%) or the outpatient sector (16.7%). The most prevalent co-occurring diseases were cardiac illness and respiratory failure (50% and 25%, respectively). When venous thromboembolism was not detected before to death in roughly 58% of instances, deep vein thrombosis was found at autopsy; immediate cause of death in 33.3% of cases was pulmonary embolism.[19]

The heart was the only organ where viral detection and morphological alterations were correlated. It should be noted that this only affected the epicardium, which displayed a slight lymphocytic inflammation. The abundance of ACE-2 receptors in adipose tissue may

be to blame for this.[20,21]

Particularly in the heart valves, coronary arteries and myocardium histological abnormalities were correlates of such previous diseases. 1.5% of instances, or 316 cases across 41 studies, have myocarditis.[22] However a cohort study reported that liver besides normally involved in viral tropism as well as other histopathological patterns failed to demonstrate any histological abnormalities. Mild chronic portal inflammation and steatosis can once again was classified as preexisting or acquired due to critical disease, with multi drug therapy.[13] Schmit et al., who solely discussed alterations typical of critically sick patients, medications, or previous disorders, demonstrated this as well.[23]

In contrast, a meta-analysis of 116 individuals found vascular thrombosis was present in (29.4%), in addition to the congestion of hepatic sinus in (34.7%) and steatosis in (55.1%) of the patients.[24] An autopsy investigation found that in acute phase patients have fibrosis along with a typical diffuse destruction of alveoli resulting from the destruction of alveoli and endothelium.[25, 26] This results in the development of hyaline membrane and exudation of fluid from the cells. Additionally, organizing pneumonia and acute fibrinous and are also seen due to alveolar fibrin aggregation.[27] Increased capillary permeability leads to inflammation of the airways and interstitial and alveolar edema. vascular angiogenesis is another characteristic of COVID-19 disease.[28,29]

Stomach, duodenal and rectal cells are also found to have been containing viral protein shells. Lactate dehydrogenase along with other liver enzymes are elevated in more than half of such hospitalized patients which are the markers of liver and bile duct damage. A hyper active immune response or hepatotoxicity of the drugs are most probable cause[2]. COVID-19 has also made heath care of kidney transplant or dialysis patients very challenging 30.15% of the patients who were suffering from chronic kidney disease passed away in Britian, because kidneys have ACE2 receptors.[31] A study also found COVID19 virus in glomerular cells, tubular epithelium, and podocytes in kidneys[2].

CONCLUSION

There are higher chances of organ failure in patients of COVID-19, as proved from autopsies of COVID-19 cases. Thus, we can say that COVID-19 can be a leading cause of organ failure and mortalities were more due to organ failure, secondary to COVID-19.

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