AFTERMATH OF PANDEMIC COVID-19 ON CARDIAC PATIENTS

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Abstract:

According to preliminary research, COVID-19 is linked to high-rate cardiac arrhythmias. COVID-19 patients who have underlying cardiovascular problems have a higher death rate. Acute coronary syndrome, venous thromboembolism, myocardial injury, and arrhythmias can all be brought on by COVID-19. Among other things. Cardiac biomarkers are essential for the quick and accurate diagnosis, treatment, and prognosis of acute coronary syndrome. It is essential to receive a diagnosis during the "golden period" inorder to start therapy right away and maybe reverse cardiac damage. In this study, the risk of cardiac arrest, and arrhythmias such Atrial-Fibrillation (AF) and Brady arrhythmias was evaluated, as well as to see if covid-19 might affect cardiac patients' blood parameters. It will also assist us in determining the degree of cardiac arrest and arrhythmias and how cardiac markers will react following a CVS. We look at people who have been diagnosed with CVS and been a COVID-19 hospital admission during the months of November 2021 and March 2022, data was gathered at the Allama Social Security Hospital in Gujranwala, Pakistan. The research took around 5 months to complete. In this study, 110 patients between the ages of 30 and 60 were recruited after giving their informed permission. The study participants proceeded to the hospital, where trained staff members who were in charge of gathering specimens and making sure that all vials were appropriately labelled collected Covid-19 samples. Covid-19 samples are collected using a nasopharyngeal swab or an or pharyngeal swab. As indicated in table, our study included 110 Cardiac and COVID-19 patients varying in age from thirty to sixty, with 51 patients being male (46.4%) and 59 being female (53.6%). The age groups of patients show us the statistics of Cardiac markers (CK-MB, SGOT, CPK, CRP) and CBC (HB, WBC, Platelets and RBCS). The (Graph 3,4,5 and 6) shows us the relationship of Cardiac and Covid-19 patients with Cardiac Markers. The (Graph 7, 8, 9 and 10) shows us the relationship of Cardiac and Covid-19 patients with CBC Blood parameters. The cardiac markers are disrupted as a result of cardiac arrest, and the patients are diagnosed with cardiac arrhythmia. If a patient is already Covid-19 positive, changes in blood parameters will occur. As a consequence, cardiac indicators will be affected more, making the situation worse. As a result, heart rhythms will be disrupted, and blood circulation throughout the body will be disrupted. If the problem is not handled quickly, the patient will automatically get seriously ill. Dysrhythmias and cardiovascular arrests are going to be coming from a systemic illness, and COVID-19 will alter blood parameters, potentially affecting heart circulation in patients with severe cardiac disease.

Keywords: CKMB, CPK, SGOT, CRP, Arrhythmias, Cardiac Arrest and COVID-19.

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1. Introduction

By the beginning of May 2020, more than 1 million people in the United States had been infected by the severe acute respiratory syndromes' corona virus 2 (SARS-CoV-2)-caused Corona virus disease 2019 (COVID-19), which has spread globally. According to preliminary data from China, 17% of COVID-19 patients hospitalized developed cardiac arrhythmias overall. The probability of arrhythmia was higher (44%) for COVID-19 patients who were admitted to the critical care unit (ICU). The virus that causes Covid-19, known as the severe acute respiratory syndromes' corona virus 2 (sars-cov-2), is exceedingly contagious and dangerous. It originally surfaced in Wuhan, China, and since then, has spread globally. A single-strand positive strand RNA virus called the sars-cov-2 virus causes severe respiratory illness in humans. Over 0.8 million individuals have died from the corona virus epidemic in 2019 (covid-19) globally, has become a major pandemic between December 2019 and August 2020. Due of COVID-19's rapid spread, the WHO has designated it as a public health emergency of global concern.[2] Countries like Italy, the United States, and China have very high rates of sickness. Limited access to testing in many clinical settings contributes to the rapid spread of infection transmission in asymptomatic and early symptomatic kinds. In December 2019, Wuhan, Hubei Province, China, announced the first case of COVID-19 transmission, which was connected to the Huanan Seafood Market.[3]

The infection has since spread to 216 other locations and areas. The World Health Organization (WHO) reports that COVID-19 first showed signs of a pandemic on January 30, 2020, and that it was subsequently deemed a worldwide pandemic in March 2020. 4 Corona viruses disturb the respiratory, digestive, and central nervous systems in both humans and animals, which can lead to health problems and financial loss. Other corona viruses, such sars-cov-2, cross the barrier of species into humans and spread lethal and severe epidemics of respiratory diseases.[5] The SARS-COVID was 1st discovered in, bats and then spread to various species around the globe. The sars-cov pandemic first hit people in 2003, thanks to animal infections in China's open-air marketplaces.6 The Centers for Disease Control and Prevention (CDC) currently advises that those who have had direct touch with the subject who has SARS-CoV-2 infection quarantine (avoid contact with other people) for 14 days after the last known contact in order to prevent further transmission of SARS-CoV-2, the virus that causes corona virus disease 2019 (COVID-19).Quarantine, on the other hand, may be difficult to maintain for an extended length of time. Shorter quarantines may enhance compliance, and the CDC offers two alternatives for reducing quarantinelengthdepending upon local conditions and the availability of testing, for close relationships without symptoms: 1) If no test is performed, quarantine may end on day 10; 2) if a negative test result is received, quarantine may end on day[7,8]. The term "corona" refers to the spikes that resemble crowns that are seen on the virus's outer surface.9 Corona viruses are smaller (65–125 nm in diameter) and have single-stranded RNA with a size range of 26–32 kbs as their nucleic materialCorona viruses are grouped into the corona virus family and are referred to as alpha (-cov), beta (-cov), gamma (-cov), and delta (-cov).[10] The -cov genus of viruses has genetic ancestry with the alpha and beta corona viruses, which have the potential to infect mammals.[11]

The Centers for Disease Control and Prevention (CDC) currently recommends that individuals who have come into close contact with a person who has the infection quarantine (avoid interactions with individuals) for 14 days after the last known contact) in order to prevent the spread of SARS-CoV-2, the virus that causes corona virus disease (COVID-19)." There has been more research in this area our understanding of the underlying pathologic processes that occur in patients[12]. Cardiovascular (CV) disease has been diagnosed and treated using a number of cardiac indicators. Due to a lack of sensitivity and specificity, however, the need for unique, specialized molecules to treat cardiac muscle necrosis persists. In order to boost specificity to CV illnesses such acute coronary syndrome (ACS) or heart failure, several new drugs have been produced and studied in the previous ten years (HF).[13] The covs are further divided into families (a, b, c, and d): mers-cov belongs to lineage c, which has more than 500 viral sequences, whereas sars-cov and sars-cov-2 belong to lineage b, which has over 200 known virus sequences.[14] Myocardial necrosis (CK-MB fraction, myoglobin, and cardiac troponins), ischemia (ischemia modified albumin), myocardial stress (natriuretic peptides), myocardial stress under stress, and inflammation and prognosis (inflammatory markers) are the four categories of cardiac markers (C-reactive protein [CRP], soluble CD40 ligand [sCD40L], and homocysteine).[15] As the preferred cardiac markers for patients with ACS, cardiac troponins have exceeded CK-MB and myoglobin in terms of clinical value . Indeed, cardiac troponin is crucial for identifying acute myocardial infarction, according to the consensus criteria of the American College of Cardiology (ACC) and the European Society of Cardiology (ESC) (MI).[16]

Due to its greater sensitivity and precision in patients who have MI suspicion at the time of presentation, cardiac troponin is the sole biomarker that is advised for the diagnosis of acute MI at this time, following

these recommendations.[17] Increases in biomarkers such as cytokine production, cellular adhesion molecules, acute-phase reactants, plaque regime change and rupture biomarkers, biomarkers of ischemia, and biomarkers of myocardial stretch, which are upstream from markers of necrosis, have been found to be useful in identifying patients who are more likely to experience an adverse event.[18] Unknown is the exact method by which this virus spreads.[19] However, like with other viral pathogens, the most typical mode of transmission is likely to be colloidal suspension transmission via germs, either directly or indirectly. There is currently no evidence that the virus can spread through the air.19 Although viral fragments have been discovered in faces samples from symptomatic and recovering individuals, the risk of feco-oral transmission is uncertain.[20]

The reason will be to explore how covid-19 affects cardiac patients. By conducting this type of research, we will be able to learn how cardiac patients interact with covid-19 and how covid-19 causes more issues in the body of cardiac patients.

2. Material and Method

During the months of November 2021 and March 2022, data was gathered at the Allama Social Security Hospital in Gujranwala, Pakistan. This research is cross-sectional and used a practical sampling method. Research took around 5 months to complete. In this study, 110 patients between the ages of 30 and 60 were recruited after giving their informed permission. The study participants proceeded to the hospital, where trained staff members who were in charge of gathering specimens and making sure that all vials were appropriately labelled collected Covid-19 samples. A nasopharyngeal swab or an oropharyngeal swab is used to collect Covid-19 samples.

Nasopharyngeal swab:
We place the swab horizontally (not upwards) in one nostril and move it down the bottom of the nasal tube for a few centimeters until we reach the nasopharynx (resistance will be met). The distance between the nose and the ear provides an idea of how far the swab should be placed, after which we gently twist the swab 180 degrees to ensure an appropriate specimen is acquired. To ensure secretary absorption, leave the 1-2 seconds with the swab in place. Take away the swab then place it into the vial containing vtm by inserting it at least 12 inches below the surface of the media. Remove the extra swab handle and connect the cap firmly to the transport medium vial.

Oropharyngeal swab:
We tell the patient to open his or her mouth for oropharyngeal. With a tongue depressor, push the outer two-thirds of the tongue down, revealing the tonsils and the posterior wall of the throat. Avoid contacting the teeth, tongue, or depressor with the swab. Swab the tonsillar pillars and the posterior oropharynx with the swab. This will induce the patient to vomit for a short time. Insert the swab into the vial of vtm (same vial as the first np swab). Remove the extra swab handle and connect the cap firmly to the transport medium vial. Include the patient's ID number on the label, along with the date and time the sample was collected. Complete sample tracing record with patient ID, collection time, and collection date.

COVID-19 test results:
After sample collection, nucleic acid as from viral sample will be extracted, and parts of its genome will be amplified using reverse transcription PCR (rt-PCR). They now have a larger sample size to evaluate the viral strains thanks to this. There are two genes in the sars-cov-2 genome. Positive results occur when both genes are found; unclear results occur when only 1 gene is found; and negative results occur when neither gene is discovered. A thorough blood count will be performed as well (CBC).

Cardiac Evaluation:
In addition to testing the patient's blood for other compounds like blood fats (including blood lipids), vitamins, and minerals, we take blood from them to look for any cardiac problems. Blood testing can identify the toxins and reveal the extent of your heart muscle damage as well as whether it has occurred. Blood tests such as CKMB, CPK, SGOT, and CRP will also be performed to check the severity and extant of CVS.

Explanatory variable:
To acquire demographic information and medical co-morbidities, we meticulously reviewed all clinical records. We also kept track a profile for admission, which includes vital signs, lab testing, and antiviral medication administered during the stay. Age, sex, and marital status were among the demographic factors. History of AF, insulin, obstructive sleep apnea, chronic renal failure, hepatitis, respiratory illness, etc. any history of high blood pressure, cardiac disease, or heart failure. were considered to be co-occurring illnesses (CKD). We also looked at any previous implanted cardioverter-defibrillator or permanent pacemaker installation procedures.

Inclusion Criteria:
Cardiac patients who have been involved in the research have been diagnosed with covid-19.

Exclusion Criteria:
Patients who have had renal illness or any other condition in the past will not be allowed to participate in the trial.

Statistical Analysis:

To assess the relationship between certain clinical parameters, such as age and sex, we employed logistic regression. CK-MB, CPK, SGOT, CRP. Age-adjusted multivariable models, sex, CK-MB, CPK, SGOT, CRP. The study was used to analyze statistical version 22.

Tables and Graphs:

Table 1: Statistics

		Age in Group	Gender
N	Valid	110	110
	Missing	0	0

Table 2: Age in Group

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	30-35	4	3.6	3.6	3.6
	36-40	13	11.8	11.8	15.5
	41-45	48	43.6	43.6	59.1
	46-50	30	27.3	27.3	86.4
	51-55	12	10.9	10.9	97.3
	56-60	3	2.7	2.7	100.0
	Total	110	100.0	100.0	

Table 3: Gender

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Male	51	46.4	46.4	46.4
	Female	59	53.6	53.6	100.0
	Total	110	100.0	100.0	

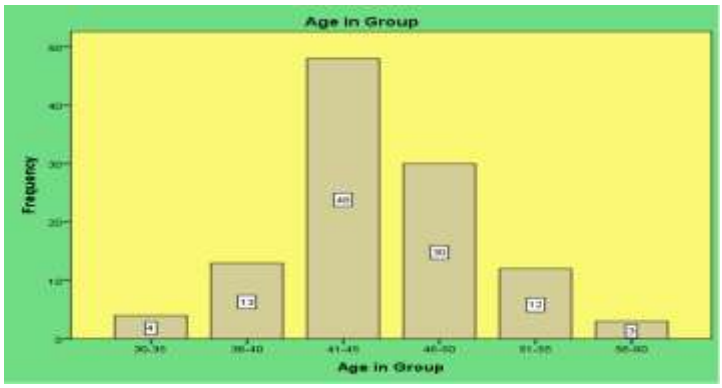
Table 4

Statistics

Covid19	Cardiac			CKMB	CPK	SGOT	CRP	RBC	HB	WBC	Platlets
Positive	Positive	N	Valid	83	83	83	83	83	83	83	83
			Missing	0	0	0	0	0	0	0	0
		Mean		232.08	391.23	46.95	39.04	4.96	14.36	7.67	244.80
		Std. Deviation		38.681	64.843	7.638	6.722	.847	1.805	2.683	75.109
	Negative	N	Valid	12	12	12	12	12	12	12	12
			Missing	0	0	0	0	0	0	0	0
		Mean		112.08	133.17	50.08	38.83	5.00	14.67	7.67	256.42
		Std. Deviation		24.224	47.916	11.851	5.167	.739	2.270	2.425	74.667
Negative	Positive	N	Valid	14	14	14	14	14	14	14	14
			Missing	0	0	0	0	0	0	0	0
		Mean		241.36	395.00	50.21	38.64	5.14	14.21	7.93	271.50

Graphs:

Graph 1

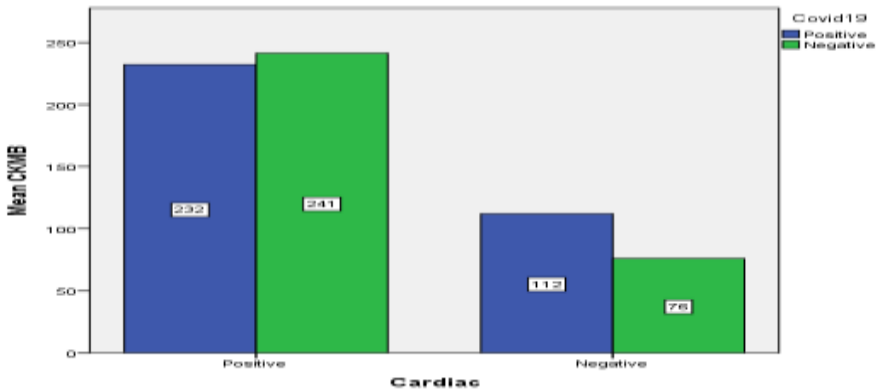


The Graph 1 shows us the Frequency of Patients with different age Group

Graph 2

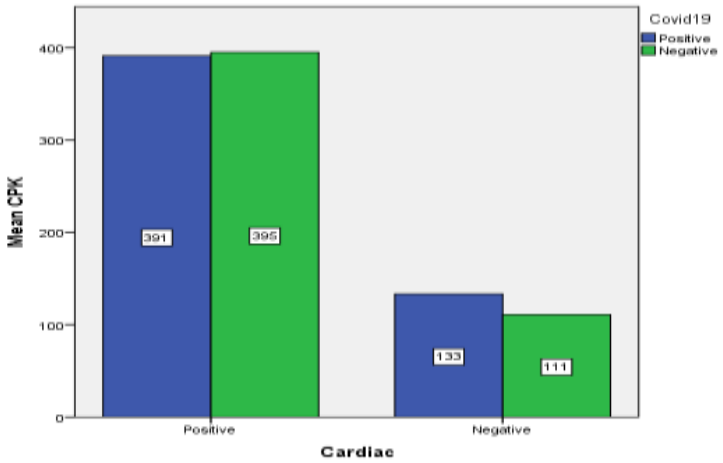
The Graph 2 shows us the frequency of Gender Distribution.

Graph 3



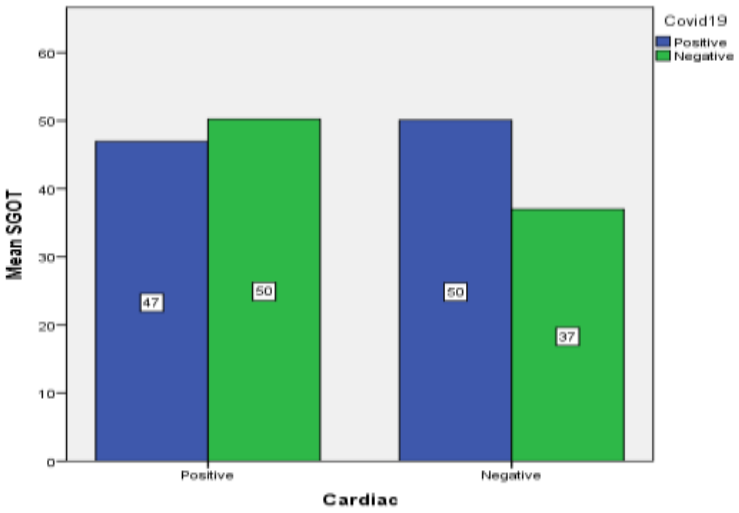
The association between Cardiac and Covid 19 patients and the CK-MB marker is seen in Graph 3

Graph 4



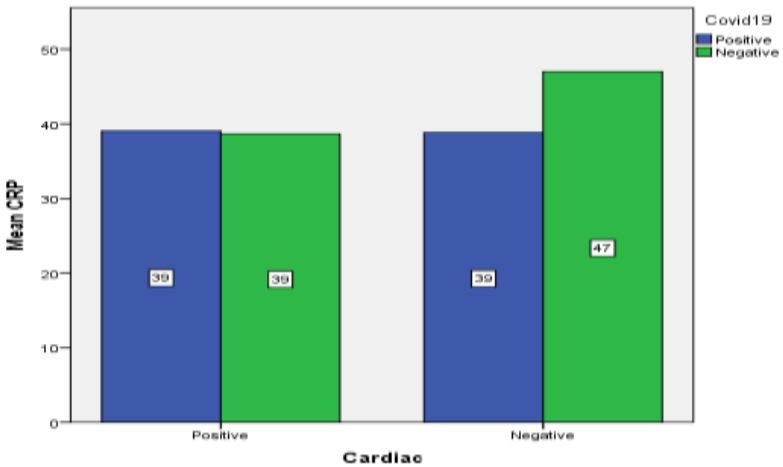
The association between Cardiac and Covid 19 patients and the CPK marker is seen in Graph 4.

Graph 5



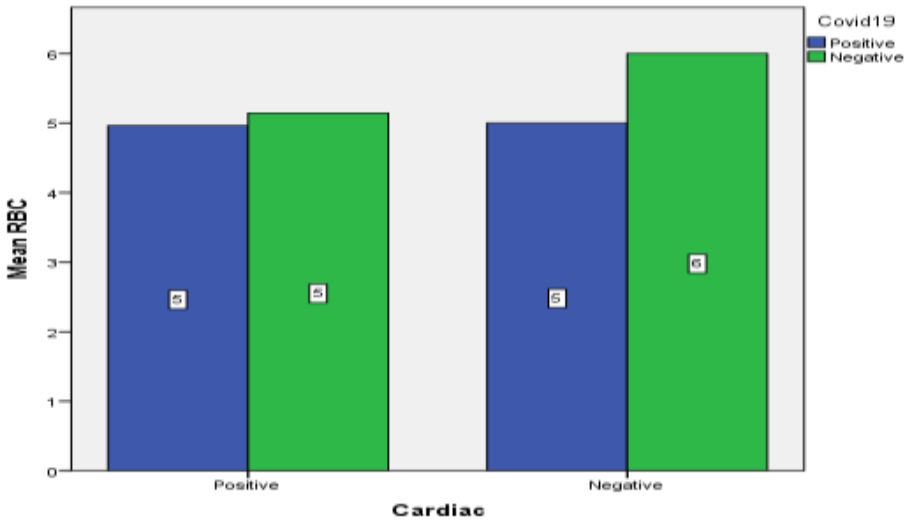
The association between Cardiac and Covid 19 patients and the SGOT marker is seen in Graph 5.

Graph 6

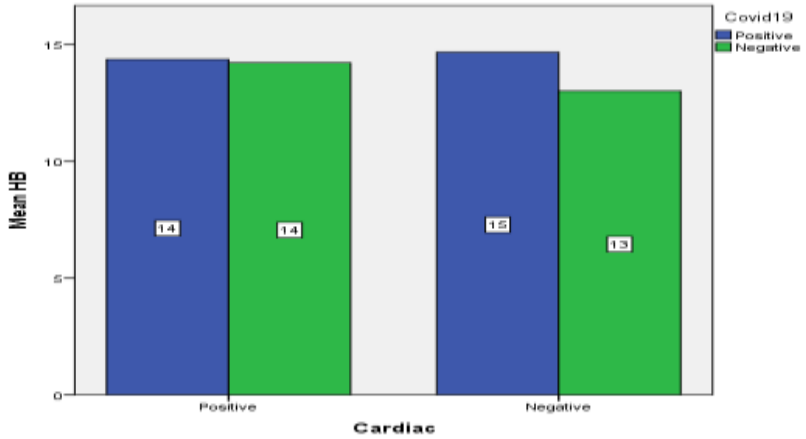


The association between Cardiac and Covid 19 patients and the CRP marker is seen in Graph 6.

Graph 7

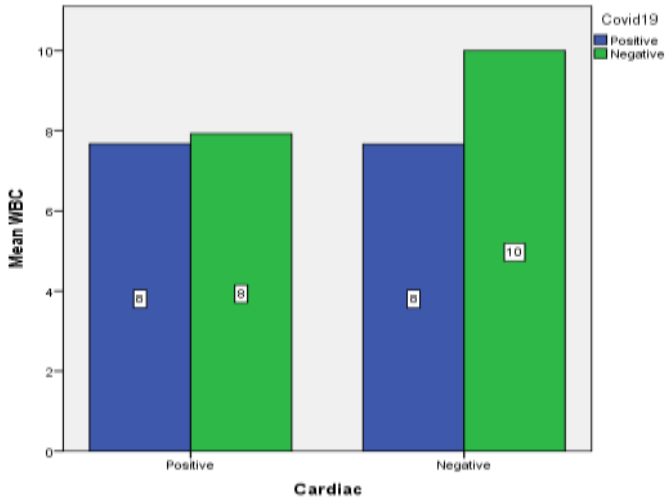


The association between Cardiac and Covid 19 patients and the RBC parameter is seen in Graph 7.
Graph 8



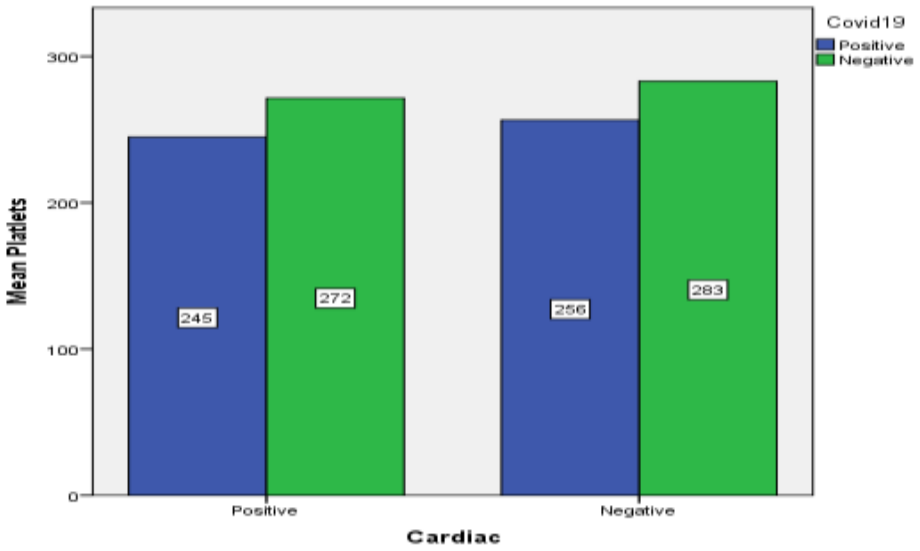
The association between Cardiac and Covid 19 patients and the HB parameter is seen in Graph 8.

Graph 9



The association between Cardiac and Covid 19 patients and the WBC parameter is seen in Graph 9.

Graph 10



The association between Cardiac and Covid 19 patients and the Platelets parameter is seen in Graph 10.

3. Results

As indicated in table, our study included 110 Cardiac and COVID-19 patients between the ages of 30 and 60, with 51 patients being male (46.4%) and 59 being female (53.6%). **(Table 2 and 3 and Graph 2)**. The age groups of patients are shown in **(Table 2 and Graph 1)**, with four individuals between the ages of 30 and 35 having a proportion of (3.6 percent). There were 13 individuals aged 36 to 40 years old with a proportion of (11.8 percent). Another age group, 41 to 45, included 48 patients, with a proportion of (43.6 percent.) Thirty patients, ranging in age from 46 to 50, have a proportion of (27.3 percent). There were 12 individuals in the 51 to 55 age group who had a percentage of (10.9 percent). Three individuals in the 56-to-60-year-old age group have a proportion of (2.7 percent). The **(Table 4)** shows us the statistics of Cardiac markers (CK-MB, SGOT, CPK, CRP) and CBC (HB, WBC, Platelets and RBCS).

The association between Cardiac and Covid 19 patients and the CK-MB marker is seen in **(Graph 3)**. This indicates that if a patient is cardiac positive as well as covid 19 positive, the CKMB level will rise over the usual threshold. If the patient is cardiac positive but not covid-19 negative, the CK-MB level will rise even higher, indicating that the heart muscles have been damaged as a result of cardiac arrest. On the other hand, if the patient is cardiac negative and Covid 19 negative, the CK-MB be level will be lower or normal, however if the patient is cardiac negative and Covid 19 positive, the CK-MB be level will be somewhat higher than normal. The **(Graph 4)** shows the correlation with covid-19 and cardiac patients with the CPK marker. It demonstrates that if a patient is cardiac positive and also covid-19 positive, the level of CPK marker will be higher than normal, however if the patient is both cardiac positive and covid-19 negative, the level of CPK marker will be somewhat higher. If the patient is cardiac negative and covid 19 positive, the CPK marker level will be close to normal; however, if the patient is cardiac negative and covid 19 negative, the CPK marker level will be normal.

The relationship between covid-19 patients, cardiac patients, and SGOT cardiac marker is seen in **(Graph 5)**. It shows that if a patient is cardiac positive and Covid 19 positive, the level of SGOT will increase more than normal, but if the patient is cardiac positive and Covid 19 negative, the level of SGT will increase more. If, on the other hand, the patient is cardiac negative but covid-19 positive, the SGOT level will rise, indicating that covid 19 has an effect on the SGOT level. If the patient is cardiac negative and covid-19 negative, on the other hand, the SGOT level will stay normal. The association between cardiac patients and covid-19 patients and the cardiac marker CRP is seen in **(Graph 6)**. It reveals that if a patient is cardiac positive and covid-19 positive, the CPK level will be somewhat higher than usual, but the level will remain the same when compared to a cardiac positive and covid-19 negative patient. If the patient is cardiac negative and covid-19 positive, the level will be somewhat higher than normal, but if the patient is cardiac negative and covid 19 negative, the level will be greater than all prior findings.

The new findings relate to various detailed blood profile measurements. The connection between RBC and cardiac and covid 19 individuals is depicted in **(Graph 7)**. It shows that if the patient is cardiac positive and covid-19 positive, the RBC level will be normal, and if the patient is cardiac positive and covid-19 negative, the RBC level will also be normal. On the other hand, if the patient is cardiac negative and covid 19 positive, the RBC level will be normal, and if the patient is cardiac negative and over 19 negative, the RBC level will be completely normal. The association between HB and cardiac and covid-19 patients is seen in **(Graph 8)**. It shows that if a patient is cardiac positive and covid-19 positive, the level of HB will rise; similarly, if a patient is cardiac positive but covid-19 negative, the level of HB will rise. This is because cardiac arrest affects the level of HB in the blood. If the patient is cardiac negative and covid-19 positive, however, the level of HB will raise more since covid-19 increases the HB level in the body. If the patient is cardiac negative and covid 19 negative, however, the level will remain normal. The connection between WBC and cardiac and covid 19 individuals is seen in **(Graph 9)**. It reveals that if a patient is cardiac positive and covid-19 positive, the amount of WBC in the blood will fall, however if the patient is cardiac positive but covid 19 negative, the level of WBC in the blood will remain low. If the patient is cardiac negative and covid-19 positive, the WBC level will likewise be elevated; however, if the patient is cardiac negative and covid-19 negative, the WBC level will stay normal. The **(Graph 10)** depicts the link between platelet levels in the blood and cardiac and covid-19 patients. It shows that if a patient is cardiac positive and covid-19 positive, the platelet level will alter somewhat in comparison to a normal person. In comparison to earlier results, if the patient is cardiac positive and covid-19 negative, there will be an increase. If the patient is caring negative and covid-19 positive, the platelet level will be disturbed; nevertheless, if the patient is cardiac negative and covid 19 negative, the platelet level will be normal.

Each patient is placed in the arrhythmia category based on the assessment of chosen characteristics such as age, sex, CVS, diabetes, hypertension, Cardiac Markers status, and CBC on admission. At the time of their first presentation and prior to the onset of cardiac arrest, all of the patients who experienced a heart attack were admitted to the ICU. The research also reveals the link between CVS and Covid 19. The cardiac markers are disrupted as a result of cardiac arrest, and the patients are diagnosed with cardiac arrhythmia. If a patient is already covid positive, changes in blood parameters will occur. As a consequence, cardiac indicators will be affected more, making the situation worse. As a result, heart rhythms will be disrupted, and blood circulation throughout the body will be disrupted. If the problem is not handled quickly, the patient will automatically get seriously ill.

4. Discussion:

Out of 110 individuals admitted to the hospital, 95 had COVID-19, according to our data. Patients admitted to the ICU had a 10-fold greater total acute death rate than individuals who were hospitalized to a non-ICU setting. 97 cardiac patients were found. 25 episodes of AF that occurred by chance, and 9 clinically important brad arrhythmias. According to the research, there are several criteria that may be used to determine the severity of cardiac arrest in individuals. Our findings indicate that the prevalence of cardiac arrests among COVID-19 patients is correlated with the risk of their illness and not only the virus itself. There are differences in cardiac marker levels when compared to normal values. The degree of COVID-19 +ve in cardiac patients is also determined using a complete blood profile. Azithromycin was not given to any of the patients at our facility. Our findings go on to say that virtually all of the heart attacks among our COVID-19 cohort were non-shock able rhythms such pulse less electrical activity or systole, which are not shock able. According to our findings, general infections, inflammation, and illness are more likely to play a role in the etiology of heart failure than direct viral infection or necrosis of the myocardium. From March 30, 2020 to May 3, 2020, 163 individuals (including those with pulmonary, autoimmune, cardiovascular, endocrine, and other conditions) participated in a research that included socio-demographic assessments and the 4- Dimensional Symptom Questionnaire.42 This study looked at how a severe isolation at COVID-19 in Greece affected the anguish, anxiety, despair, and somatization of people with chronic illnesses. In chronic illness patients, distress and somatization were higher, and anxiety and sadness raised heart activity pressure, which might contribute to cardiovascular disease. Particularly affected were patients with cardiovascular and endocrine disorders. Corona virus is potential to influence blood parameters in the body, as well as the respiratory system, resulting in serious consequences if not addressed appropriately. According to new research at the University of Alabama in Birmingham, compared to the non-ICU group, the ICU population had a greater likelihood of experiencing atrial tachycardia. Long-termanticoagulant medication should be considered in view of these consistent findings. Thrombotic problems, such as arterial and venous thrombosis, can occur as a result of COVID-19. The inflammatory mediators and

cytokine response induced by SARS-Cov-2 infection of the vascular endothelium are thought to be the source of endothelial and hemostatic activity. If AF is present, this inflammatory state may increase the risk of venous thromboembolism effects, which might result in the development of new cardiovascular disease.3

A meta-analysis and comprehensive study by Emir Yonas on the impact of cardiac arrest on COVID-19 results, was published in 2021. He used the search phrases "heart failure" and "COVID-19" to carry out a thorough literature search in the databases Cochrane Central Database, Medrxiv, SCOPUS, PubMed, and Europe PMC. The result of interest was mortality and a poor prognosis in individuals with underlying heart failure and corona virus disease About 65 publications did not report on the outcome of interest, whereas four publications were review, two experiments were meta-analyses, three pieces were literature reviews, hence he removed 74 research. COVID-19 increases the likelihood of hospitalization, poor prognosis, and according to his findings, death among people with heart failure. Patients with and without heart failure died at significantly different rates, with heart failure patients dying more often.22 There are some limitations in our research. This research was conducted at a single location that served a big metropolitan population. Therefore, our findings might not be relevant to all COVID-19 patients worldwide. As an outcome, we wouldn't be able to find these individuals' asymptomatic tachycardia as easily. Finally, we only included inpatient follow-up in our research. We are therefore unable to establish if the existence of arrhythmic events affects the long- term health of our COVID-19-treated patients

5. Conclusion

The population in the study had a higher prevalence of cardiac arrests and arrhythmias. It shows how covid -19 affects cardiac function in patients who have already been diagnosed with CVS. Cardiac indicators indicate how serious an ailment is. COVID-19 will immediately affect the respiratory system as well as blood function, resulting in an increase in pressure throughout the human circulatory system. This has a direct influence on the heart's circulation and is more likely to happen as a result of systemic sickness or cardiac arrhythmias.

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