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# Investigation of changes in young cardiac pathology cases before and during the pandemic process

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## ABSTRACT

**Aim**: It was aimed to evaluate the effect of pandemic in cardiac pathologies, especially acute coronary syndrome in young cases.

**Material and Method**: Between January 2019-May 2021, 510 young patients aged between 18-50 years with acute coronary syndrome, arrhythmia or pericarditis were evaluated. The patients were divided into two groups as pre-pandemic and pandemic period, and the pandemic period was divided into two groups as Coronavirus Disesase 2019 (COVID) (-)/(+). In addition, patients were divided into groups according to their diagnosis. Demographic data, diagnostic classifications, COVID-PCR results, white blood cell, mean corpuscular volume, neutrophil, lymphocyte, neutrophil lymphocyte ratio, platelet, platelet lymphocyte ratio, C reactive protein, glucose, troponin values and survival data of the patients were recorded.

**Results**: The median age of 510 patients included in the study was 44(39-48) years, 395(77.5%) were male. When the diagnoses were put into groups by time, unstable angina was the most common diagnosis in each group. In the COVID(+) group, 39(23.8%) non-ST-elevation myocardial infarction (NSTEMI), 17(10.4%) inferior MI, and 14(8.5%) anterior MI were found. Twenty three (4.5%) of all cases resulted in mortality. According to the diagnoses, the most common mortality was in the inferior MI group with 10(28.6%) cases ($p<0.001$). During the pandemic period, 13(7.9%) of the COVID(+) patients resulted in mortality ($p=0.016$).

**Conclusion**: Acute coronary syndrome cases and cardiac pathologies other than unstable angina increased in young cases during the pandemic process. Mortality rates in all groups increased significantly during the pandemic and especially in COVID(+) cases compared to pre-pandemic.

**Keywords**: Emergency department, COVID-19, acute coronary syndrome, mortality, pandemic

## INTRODUCTION

Acute coronary syndrome (ACS) is defined as all clinical symptoms of acute myocardial ischemia. Clinical diseases such as unstable angina pectoris, non-ST-segment elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI) are included in the phrase ACS (1). Although myocardial infarction (MI) is a condition that is usually seen in people over the age of 45 and frequently affects the elderly population, some younger patients may also present with MI clinic. ACS was found to affect 0.5% of men and 0.18% of women between the ages of 35 and 44. It affected 20.5% of men over 60 and 17.1% of women (2).

The 2019 Coronavirus Disease (COVID-19) is an infection requiring a comprehensive approach. In addition to kidney and liver damage, the nervous system and cardiovascular system can be affected by this infection (3). Individuals with cardiovascular disease are predisposed to COVID-19 infection. In patients with cardiovascular dysfunction who are infected with severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), there is an elevated risk of COVID-19-related adverse outcomes. There is also an increase in cardiovascular complications in these individuals (4). Most significantly, acute myocardial injury and troponin increase are severe prognosis and death indicators in COVID-19 cases. Therefore, these cases should be followed carefully and closely (5). Labile heart rate and aberrant blood pressure response to activity are common in COVID-19 patients, as are myocarditis and pericarditis, decreased myocardial flow reserve due to microvascular injury, myocardial infarction, heart failure, life-threatening arrhythmias, and sudden cardiac death. Venous and arterial thromboembolic disorders, such as coronary artery aneurysm, aortic aneurysm, accelerated atherosclerosis, and life-threatening pulmonary embolism, can also exist (6).

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We aimed to compare and evaluate acute coronary syndrome and other cardiac pathologies in terms of epidemiological, demographic, laboratory and survival before and during the pandemic period with especially young cases. In addition to the fact that there is no such data in previous studies, we were encouraged to evaluate it in a specific age range, such as young cases, which made study more meaningful.

## MATERIAL AND METHOD

The study was carried out with the permission of Medipol University Training and Research Hospital, Noninvasive Clinical Researches Ethics Committee (Date: 26.10.2021, Decision No:1064). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

### Study Design and Population

This study involves 510 patients aged 18-50 years (115 females, 395 males; median age 44(39-48) years, range 19-50 years) who presented to the emergency department between January 1, 2019 and May 11, 2021 and were diagnosed with acute coronary syndrome, pericarditis, and atrial fibrillation/flutter.

According to the hospitalization period of the patients, two groups were formed: pre-pandemic and pandemic. The pre-pandemic phase between January 1, 2019 and March 10, 2020, and the pandemic period between March 11, 2020 and May 11, 2021, each consist of 14 months. Since the 11th of March, 2020 has been designated as the beginning of the COVID-19 pandemic in Turkey, this date has been used as a point of reference. Patients who applied during the pandemic period, those who had COVID-19 disease or those with a positive Polymerase Chain Reaction (PCR) test were divided into two groups as COVID (+) and the others as COVID (-). Patients were classified into six groups based on their diagnosis: unstable angina (UA), NSTEMI, inferior MI (IMI), anterior MI (AMI), pericarditis, and arrhythmia. Patients with atrial fibrillation and atrial flutter rhythm were included in the arrhythmia group. Individuals experiencing other heart rhythms were excluded from the study. In addition, patients were analyzed based on their survival and mortality at the conclusion of the treatment process.

Cases without defined additional disease were selected in the pre-pandemic group and the COVID(-) group of the pandemic group. In the pandemic period COVID (+) group, only the cases with COVID-19 disease or positive PCR test were included. These patients were admitted to the emergency department and laboratory tests were studied. In addition, all patients had an electrocardiogram (ECG). Patients' age, gender, ECG reports, laboratory results, and clinical course were recorded in the hospital data system.

Patients with no additional disease, aged between 18-50 years, with complete demographic, laboratory and ECG reports and definitive diagnosis records were included in the study. The study excluded patients with a history of cerebrovascular disease, cardiac pathology, arrhythmia, chronic and congenital heart disease, hormone-based disease, psychiatric drug history, chronic liver disease, renal failure and dialysis, infectious disease, chronic inflammatory disease, malignancy, severe anemia and anemia treatment, hematological disease, collagen tissue disease, and pregnancy. In addition, patients who did not have hemogram and biochemistry tests, as well as ECG or missing data records, were excluded from the study. Patients under the age of 18 and over the age of 50 were also excluded from the study.

### Laboratory Analysis

Patients' levels of White Blood Cell (WBC), Mean Corpuscular Volume (MCV), neutrophil, lymphocyte, neutrophil lymphocyte ratio (NLR), platelet, platelet lymphocyte ratio (PLR), C reactive protein (CRP), glucose, and troponin were measured. A Beckman Coulter Automated CBC Analyzer was used to measure hemogram (Beckman Coulter, Inc., Fullerton, CA, USA). Cobas 6000 was used to conduct biochemistry analysis (C6000-Core, Cobas c-501 series, Hitachi, Roche, USA). Analyzers STAT Elecsys and Cobas e-411 Hitachi Roche were used to evaluate cardiac Troponin T (cTn-T). At the time of emergency room admission, a 12-lead electrocardiogram was recorded at the bedside using a Cardiofax ECG-9132K (Nihon Kohden, Tokyo, Japan). Cardiac troponin T values greater than 14 pg/mL were considered as the core (Reference range: 0-14 pg/mL).

### Statistical Analysis

SPSS 20 (SPSS Inc., Chicago, IL, USA) was used to perform statistical analyses on the data collected for this investigation. The Kolmogorov-Smirnov test was performed to check if the variables followed normal distributions. Case counts and percentages were displayed for nominal variables. Under the assumption of normality, the one-sample Kolmogorov Smirnov test concluded that the distribution was not normal in the variables at the $p<0.05$ level of significance. Therefore, the variables reported as median and interquartile range (IQR) were subjected to the non-parametric Mann-Whitney U-test and Kruskal-Wallis Test. Chi-square analysis was used to look at the connections between the categories of nominal variables. Results were considered statistically significant when the p value was less than 0.05.

## RESULTS

The median age of 510 patients included in the study was 44 (39-48) years, 395 (77.5%) were male. 227 (44.5%) of the cases formed pre-pandemic, 119 (23.3%) pandemic COVID (-), 164 (32.2%) pandemic COVID (+) group. During the pandemic period of the patients, in COVID (+) cases; WBC (p<0.001), neutrophil (p=0.039), NLR (p=0.023), CRP (p<0.001), Troponin T (p<0.001) were significantly higher. In the classification of diagnosis according to periods, UA was the most common diagnosis in each period. UA was less in 45 (27.4%) cases in the pandemic COVID (+) group than in the other groups. In the COVID (+) group, 39 (23.8%) NSTEMI, 17 (10.4%) inferior MI, and 14 (8.5%) anterior MI were observed, which was more in the evaluation made with other groups. While the frequencies of pericarditis, arrhythmia, STEMI and NSTEMI were similar in pre-pandemic and COVID (-) patients, a significant increase in all these diseases was detected in COVID (+) patients (p<0.001, **Table 1**).

There was no significant relationship between mortality and gender. Twenty three (4.5%) of all cases resulted in mortality. According to the diagnoses, the most common mortality was in the inferior MI group with 10 (28.6%) cases (p<0.001). During the pandemic period, 13 (7.9%) of the COVID (+) patients resulted in death (p=0.016). In the mortality group, CRP 24 (12-28) mg/L (p=0.018) and Troponin T 140 (114-166) pg/mL (p<0.001) values were significant, while other laboratory results were not associated with mortality (**Table 2**).

**Table 2.** Evaluation of mortality in terms of gender, laboratory results, diagnose classification and pandemic groups

| | Mortality | | P value* |
|---|---|---|---|
| | No n(%) | Yes n(%) | |
| Gender | | | 0.924 |
| Female | 110 (95.7) | 5 (4.3) | |
| Male | 377 (95.4) | 18 (4.6) | |
| Diagnose classification | | | <0.001 |
| Unstable Angina | 294(98.7) | 4(1.3) | |
| Pericarditis | 52(98.1) | 1(1.9) | |
| Inferior MI | 25(71.4) | 10(28.6) | |
| NSTEMI | 58(98.3) | 1(1.7) | |
| Arrhythmia | 34(100) | 0(0) | |
| Anterior MI | 24(77.4) | 7(22.6) | |
| Pandemic Group | | | 0.016 |
| Pre-pandemic | 221(97.4) | 6(2.6) | |
| COVID(-) | 115(96.6) | 4(3.4) | |
| COVID(+) | 151(92.1) | 13(7.9) | |
| Total | 487(95.5) | 23(4.5) | |
| | **Median (IQR)** | **Median (IQR)** | |
| Laboratuary Findings | | | |
| WBC, 103/uL | 9 (8-11) | 11(8-12) | 0.214 |
| NEU, 103/uL | 6 (5-8) | 6(5-7) | 0.601 |
| LYM, 103/uL | 3(2-4) | 4(2-4) | 0.176 |
| NLR, % | 1.75 (1.33-3) | 1.75 (1.4-2.5) | 0.525 |
| PLT, 103/uL | 257 (214-313) | 233 (208-325) | 0.660 |
| PLR, % | 87 (64.75-114.67) | 77.33 (54.25-103) | 0.096 |
| CRP, mg/L | 15 (6-24) | 24 (12-28) | 0.018 |
| Glucose, mg/dL | 113 (98-146) | 125 (104-193) | 0.087 |
| Troponin T, pg/mL | 52 (13-121) | 140 (114-166) | <0.001 |

Data are given in number (percentile) or median (IQR): (25th-75th percentile). WBC: White Blood Cell, MCV: Mean Corpuscular Volume, NEU: neutrophil LYM: lymphocyte NLR: neutrophil lymphocyte ratio PLT: platelet PLR: platelet lymphocyte ratio CRP: C reactive protein COVID: Coronavirus Disease MI: myocardial infarction NSTEMI: Non ST myocardial infarction p: Statistical Significance (<0.05) * Chi-square test was used for gender and disease classification and pandemic groups, while Man Whitney–U test was used for other variables.

**Table 1.** Evaluation of pre-pandemic&pandemic groups in terms of laboratory results and diagnose classification

| | All Patients n: 510, Median (IQR) | Pre-pandemic n:227, Median (IQR) | Pandemic | | P-value* |
|---|---|---|---|---|---|
| | | | COVID (-) n: 119, Median (IQR) | COVID (+) n: 164, Median (IQR) | |
| Baseline Chracteristics | | | | | |
| Age (year) | 44 (39-48) | 43 (39-47) | 43 (35-48) | 45 (40-48) | 0.107 |
| Gender (F(%) / M(%)) | 115 (22.5)/395 (77.5) | 56 (24.7)/171 (75.3) | 30 (25.2)/89 (74.8) | 29 (17.7)/135 (82.3) | 0.193 |
| Laboratuary Findings | | | | | |
| WBC, 103/uL | 9 (8-11) | 9 (8-11) | 9 (8-11) | 10 (8.25-12) | <0.001 |
| NEU, 103/uL | 6 (5-8) | 6 (4-8) | 5 (5-8) | 6 (5-8) | 0.039 |
| LYM, 103/uL | 3 (2-4) | 3 (2-4) | 3 (2-4) | 3 (2-4) | 0.300 |
| NLR, % | 1.75 (1.33-3) | 1.67 (1.33-3) | 1.67 (1.25-2.67) | 2 (1.4-3) | 0.023 |
| PLT, 103/uL | 256 (214-313) | 256 (210-311) | 259 (222-325) | 253 (214.25-317) | 0.149 |
| PLR, % | 86.58 (64.23-114.5) | 85.33 (64.75-108.33) | 85.33 (64-117.5) | 88.5 (63.75-117.94) | 0.705 |
| CRP, mg/L | 15 (6-25) | 12 (5-23) | 9 (5-18) | 18 (10.5-29) | <0.001 |
| Glucose, mg/dL | 114.5 (98-146.25) | 111 (98-140) | 115 (98-146) | 117.5 (100.25-160) | 0.092 |
| Troponin T, pg/mL | 60 (14-124) | 21 (6-63) | 40 (14-113) | 125 (96-158) | <0.001 |
| Diagnose Classification [n(%)] | | | | | <0.001 |
| Unstable Angina | 298 (58.4) | 182 (80.2) | 71 (59.7) | 45 (27.4) | |
| Pericarditis | 53 (10.4) | 8 (3.5) | 13 (10.9) | 32 (19.5) | |
| Inferior MI | 35 (6.9) | 10 (4.4) | 8 (6.7) | 17 (10.4) | |
| NSTEMI | 59 (11.6) | 11 (4.8) | 9 (7.6) | 39 (23.8) | |
| Arrhythmia | 34 (6.7) | 7 (3.1) | 10 (8.4) | 17 (10.4) | |
| Anterior MI | 31 (6.1) | 9 (4) | 8 (6.7) | 14 (8.5) | |

Data are given in number (percentile) or median (IQR): (25th-75th percentile). F: Female M: Male WBC: White Blood Cell, MCV: Mean Corpuscular Volume, NEU: neutrophil LYM: lymphocyte NLR: neutrophil lymphocyte ratio PLT: platelet PLR: platelet lymphocyte ratio CRP: C reactive protein COVID: Coronavirus Disease MI: myocardial infarction NSTEMI: Non ST myocardial infarction p: Statistical Significance (<0.05), * Chi-square test was used for gender and diagnose classification, while Kruskal-wallis test was used for other variables.

When the age was evaluated according to the diagnostic groups, it was 41 (29.5-47.5) years in pericarditis patients, while it was 48 (43-50) years in the inferior MI group (p=0.001). While anterior MI was present in 30 (7.6%) of male patients, it was detected in only 1 (0.9%) of female patients. The frequencies of pericarditis and NSTEMI were remarkable in both genders (p=0.016). Platelet and PLR were not associated with the diagnostic groups. Glucose 126 (112-160) mg/dL (p=0.003), WBC 12 (9-13) 103/uL (p<0.001) , neutrophil 7 (5-9) 103/uL (p=0.046) and lymphocyte 4 (2-4) 103/uL (p=0.001) values were higher in the anterior MI group. Troponin T value was highest in the NSTEMI group with 137 (110-166) pg/mL (p<0.001, **Table 3**).

## DISCUSSION

Recently, both acute cardiac pathologies and COVID-19 infection are among the most important causes of mortality and morbidity in emergency services and even in all medical units. Although the conditions of cardiac pathologies, especially acute coronary syndrome, during and before the pandemic have been partially evaluated by some studies in the general population, we aimed to evaluate the relationship between the two, especially in the young patient population, and contribute to the literature.

COVID-19 disease causes respiratory system, vascular endothelial, heart, intestine, and immune system cell infections (7). This factor enhances membrane fusion by binding to the highly expressed angiotensin-converting enzyme-2 (ACE-2) receptor via the spike protein (8). Particularly, endothelial cells and pericytes display high levels of ACE-2, which renders the cells extremely vulnerable to the COVID-19 interaction. The potential mechanisms underlying acute coronary syndrome in COVID-19 infection have not been clearly elucidated. In individuals with a confirmed diagnosis of myocardial infarction, the pathophysiological mechanisms may be explained by the angiographic appearance of unoccluded coronary arteries, numerous thrombotic lesions, and stent thrombosis (9,10). Occasionally, myocardial infarction has been examined as the initial symptom of the disease, which suggests that acute coronary syndrome is a particular thrombotic consequence of COVID-19 infection (11). The most widely acknowledged mechanisms are cytokine-mediated systemic inflammation reactions, endothelial dysfunction, prothrombotic stimulation of the coagulation cascade, and hypoxia destruction due to an imbalance in oxygen supply and demand. It may develop due to atherosclerotic plaque activation due to hyperinflammation or vasoconstriction. Another possible pathophysiological process may be related to microvascular thrombosis due to hypercoagulopathy due to COVID-19 disease (12).

In addition, despite the fact that this infection causes complications in the form of coronary plaque instability and myocardial oxygen supply, numerous researchers from all over the world have reported a significant decrease in the hospitalization rate for ACS during the peak of the pandemic. In addition to all this, increased lengths of ischemia and gate-balloon time were observed in 2020 patients (13). During this period of time, there was a correlation between a lower admission rate for STEMI and a higher incidence of cardiac arrest outside of the hospital as well as mechanical complications (14).

Showkathali et al. (15) examined the admission and clinical results of acute coronary syndrome cases in the same two-month timeframes during the pandemic process and the previous 2 years, and showed that all

**Table 3.** Evaluation of disease classification in terms of age, gender and laboratory results

| | Unstable Angina Median (IQR) | Pericarditis Median (IQR) | Inferior MI Median (IQR) | NSTEMI Median (IQR) | Arrhythmia Median (IQR) | Anterior MI Median (IQR) | P-value* |
|---|---|---|---|---|---|---|---|
| Age (year) | 43(38-47) | 41(29.5-47.5) | 48(43-50) | 44(41-48) | 46.5(41-49) | 46(41-49) | 0.001 |
| Gender | | | | | | | 0.016 |
| F (%) | 73(63.5) | 16(13.9) | 7(6.1) | 11(9.6) | 7(6.1) | 1(0.9) | |
| M (%) | 225(57) | 37(9.4) | 28(7.1) | 48(12.2) | 27(6.8) | 30(7.6) | |
| Laboratuary Findings | | | | | | | |
| WBC, 103/uL | 9(8-11) | 9(8-11) | 10(9-11) | 10.1(8-12) | 10(8.7-12.2) | 12(9-13) | <0.001 |
| NEU, 103/uL | 6(4.78-7.25) | 6(4-8) | 6(5-7) | 6(5-7) | 8(5-9) | 7(5-9) | 0.046 |
| LYM, 103/uL | 3(2-4) | 2(2-3) | 3(2-3.4) | 3(2-4) | 3(2-4) | 4(2-4) | 0.001 |
| NLR, % | 1.6(1.3-2.7) | 2.5(1.5-3.4) | 2(1.7-2.5) | 2(1.4-3) | 2.5(1.5-3.5) | 2(1.2-3.7) | 0.003 |
| PLT, 103/uL | 259(217-313) | 238(194.5-286) | 268(222-321) | 248(210-319) | 255(231-324.5) | 257(209-358) | 0.129 |
| PLR, % | 83.6(64.1-110.1) | 94(73.5-119.7) | 101.3(74-117.5) | 90(60.5-117.5) | 84(63.4-117.4) | 87.2(59.2-104.7) | 0.330 |
| CRP, mg/L | 11(5-21) | 21(13.2-39.7) | 15.5(8.5-24) | 15(9-25.2) | 15(5.5-24.5) | 15(6-34) | <0.001 |
| Glucose, mg/dL | 110.5(97-141.3) | 108(98-136) | 120(106-172) | 118(106-165) | 112(94-147.2) | 126(112-160) | 0.003 |
| Troponin T, pg/mL | 18(6-52) | 96(69.5-131.5) | 120(90-152) | 137(110-166) | 101(58-128.7) | 118(98-138) | <0.001 |

Data are given in number (percentile) or median (IQR): (25th-75th percentile). F: Female M: Male WBC: White Blood Cell, MCV: Mean Corpuscular Volume, NEU: neutrophil LYM: lymphocyte NLR: neutrophil lymphocyte ratio PLT: platelet PLR: platelet lymphocyte ratio CRP: C reactive protein MI: myocardial infarction NSTEMI: Non ST myocardial infarction p: Statistical Significance (<0.05), * Chi-square test was used for gender while Kruskal-Wallis test was used for other variables.

cases of NSTEMI, STEMI and UA decreased during the pandemic process. Again, in the study conducted for the same purpose, Braiteh et al. (16) In his study with an 8-week follow-up period, he showed a 41% reduction in applications for acute coronary syndrome. Although the decrease in isolated STEMI admissions was not significant, NSTEMI cases decreased significantly. Mimoso (17), on the other hand, stated that there was a decrease in the number of cases admitted in the pandemic and pre-pandemic evaluation, but the rate of STEMI increased during the pandemic and more clinically serious patients applied. Again, in the same study, it is not overlooked that the intervention period for these cases was prolonged. Zachariah et al. (18) evaluated the cases of acute coronary syndrome with 41,832 cases in 187 centers. They observed a 35.4% decrease in applications during the pandemic period. They found the increase in the rate of STEMI in the cases significant. Considering the mortality, the mortality rate in all myocardial infarction cases, which was 4.6% before the pandemic, was 4.9% during the pandemic period. In this study, no significant change was observed in mortality of STEMI cases, while mortality in NSTEMI cases was 2% before the pandemic, and it was found to be 2.8% in the pandemic, and it was significantly higher.

In this study, anterior and inferior myocardial infarction cases, which are STEMI groups, showed an increase in the distribution of cardiac pathologies during the pandemic process. This increase was observed more especially in COVID (+) cases. We believe that this increase is due to the fact that a fatal clinical condition such as myocardial infarction does not affect emergency hospital admissions during the pandemic process, especially in young cases, and that its numerical increase is due to the above-mentioned cardiac effects of COVID-19. In addition, there was an increase in NSTEMI cases. Contrary to similar studies, the increase seen in general myocardial infarction groups may be due to the younger age group and long period of time. Unstable angina cases were significantly decreased. We attribute these reductions to the effect of social lockdowns and curfews, to fear of coming into contact with patients infected with COVID-19. Mortality rates were found to be high during the pandemic process and especially in COVID (+) cases. The fact that the mortality rate is higher in cases with COVID (-) compared to the pre-pandemic period may be due to the late admission of the cases to the hospital or the partial deterioration of health service delivery in this period. Other potential causes include asymptomatic patients, misleading negative PCR results in patients, and negative results following COVID. We attribute the higher mortality rates in COVID (+) cases to the fact that the event has a worse prognosis with infection.

It has been previously known that myocardial damage and troponin elevation are not only indicators of myocardial infarction, but can also be seen with respiratory tract disorders. Viral diseases, of which we can give an example of the Middle East respiratory syndrome coronavirus, have been found to cause myocarditis with myocardial damage and troponin elevation (19,20). SARS-CoV-2 may also cause myocardial damage and myocarditis by directly or indirectly affecting the cardiovascular system (20). In COVID-19 individuals, the exact pathophysiology of acute pericarditis and myopericarditis is not yet completely characterized. An important step in the development of SARS-CoV-2 infection is dysregulation of the immune system, which can cause certain patients to produce an excessive amount of proinflammatory cytokines. This can lead to what is known as a "cytokine storm" (21). This elevated inflammatory response could be a contributing factor in the various cardiovascular manifestations that are linked to COVID-19, such as pericarditis and myopericarditis. Pericarditis cases, which we evaluated in cardiac pathologies in study, also increased in the pandemic process and especially in COVID (+) cases. We think that this is also related to the reaction of the infection in the cardiac layers during the inflammatory process.

Arrhythmia is among the presenting findings in patients with COVID-19 infection. It is observed that 7.3% of the patients diagnosed with COVID-19 have palpitations among their complaints (22). While arrhythmia is observed in 17% of hospitalized patients, this rate reaches 44% in patients followed in the intensive care unit (23). Patients with COVID-19 may have a higher risk of short- and long-term unfavorable clinical outcomes if they have atrial fibrillation, a common arrhythmia. Inciardi et al. (24) gathered data on 53 COVID-19 patients with a history of hospitalization for heart disease and pneumonia, including their demographics, clinical manifestations, and prognosis. Forty percent had a previous diagnosis of heart failure, and 36 percent were diagnosed with atrial fibrillation (AF). A review and meta-analysis of 187,716 people indicated that 8% of COVID-19 patients had AF, but that the true prevalence of AF could be as high as 27% due to substantial discrepancies between studies (25). In this study we conducted with young cases, we found that the frequency of arrhythmias, especially AF, increased gradually during the COVID process and in positive cases. Considering this result, we can say that the clinical status of COVID infection increases the frequency of AF in young people with the inflammatory process, but this is not associated with mortality.

The single-center and retrospective nature of the study can be considered among the major limitations. In addition, false negativity and positivity in PCR results, the possibility of partial disruption of patient registration and comorbidity information during the pandemic, and possible etiological errors in mortality data can be counted among other limitations.

## CONCLUSION

It can be said that there is an increase in cardiac pathologies other than unstable angina during the pandemic process. Mortality in young cardiovascular pathologies, especially in ACS, has increased significantly during the pandemic process and especially in COVID(+) cases. On the other hand, the cases of unstable angina have also decreased. More prospective multicenter studies are needed to reveal cardiovascular effects in young COVID-19 cases.

## ETHICAL DECLARATIONS

**Ethics Committee Approval:** The study was carried out with the permission of Medipol University Training and Research Hospital, Noninvasive Clinical Researches Ethics Committee (Date: 26.10.2021, Decision No:1064).

**Informed Consent:** Because the study was designed retrospectively, no written informed consent form was obtained from patients.

**Referee Evaluation Process:** Externally peer-reviewed.

**Conflict of Interest Statement:** The author has no conflicts of interest to declare.

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**Author Contributions:** Author declare that he participated in the design, execution, and analysis of the paper and that has approved the final version.

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