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Evaluation of the causes of chest pain and its relationship with the cardiovascular system in COVID-19 patients

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ABSTRACT

Aims: Chest pain is one of the most common complaints of patients in the emergency departments during the pandemic and non-pandemic period. Because the cause of chest pain can range, from an ordinary, harmless muscle pull to serious cardiac complication ultimately leading to cardiac arrest. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infects host cells through angiotensin converting enzyme 2 receptors, leading to coronavirus disease (COVID-19)-related pneumonia, and also causing acute cardiac injury and chronic damage to the cardiovascular system (CVS). COVID-19 contributes to the development of serious cardiovascular complications such as acute coronary syndrome, myocarditis, stress-cardiomyopathy, arrhythmias, cardiogenic shock, and cardiac arrest. In this study, it was aimed to determine the effects of COVID-19 on the cardiovascular system by evaluating the causes of chest pain in COVID-19 patients who applied to the emergency department with chest pain.

Methods: This retrospective study was conducted by examining the files of COVID-19 patients who applied to a district emergency department with chest pain.

Results: The files of 102 COVID-19 patients were reviewed. The most common causes of chest pain were musculoskeletal system (39.2%), respiratory system (23.5%), CVS diseases (20.6%), idiopathic causes (8.8%), gastrointestinal system diseases (7%, 9). Cardiac causes are non ST-segment elevation myocardial infarction (NSTMI), arrhythmia, ST-segment elevation myocardial infarction (STEMI) and unstable angina pectoris (USAP), respectively. Troponin value was higher in patients with cardiac chest pain (p=0.02), and ferritin value was higher in patients with pneumonia (p=0.01).

Conclusions: Chest pain or chest tightness is common in patients with active COVID-19. Although the causes of chest pain are due to musculoskeletal pathologies, both COVID-19 and cardiac origin chest pains due to direct cardiovascular system pathologies should be kept in mind.

Keywords: Chest pain, COVID-19, heart.

INTRODUCTION

In December 2019, a pneumonia outbreak caused by a new coronavirus in Wuhan, People's Republic of China, quickly spread across the world, causing the first pandemic of the 21st century. The causative virus was named "Severe Acute Respiratory Syndrome-Coronavirus-2" by the World Health Organization (WHO) (SARS-CoV-2) and the disease it causes was named COVID-19 (Coronavirus Disease 2019).[1,2] The first case was detected in Turkey on March 11, 2020, and the number of patients increased rapidly. Although the symptoms of COVID-19 disease are generally related to the respiratory system, symptoms related to the cardiovascular system (CVS) can also be observed. Patients with the highest mortality rate and most affected by the pandemic are elderly people with known cardiovascular diseases (CVD).[3] Cardiac diseases such as ischemic heart disease, HT, heart failure (HF), and atrial fibrillation are the most common conditions accompanying patients who die from COVID-19. Direct myocardial cell damage, myocardial oxygen supply/demand mismatch, acute plaque ruptures leading to acute coronary syndrome as part of systemic inflammation, catecholamine surges, and increased thrombosis have been reported as cardiac manifestations. While some of these are directly caused by the disease, others are associated with potential side effects of drugs used in the treatment of COVID-19.[1,4] In this study, we aimed to evaluate the causes of chest pain in COVID-19 patients who presented to the emergency department with chest pain and to determine the effects of COVID-19 on the CVS.

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METHODS

Approval was granted by the Ethics Committee of Kayseri City Hospital (Date: 09.2020, Decision No: 09.2020-146). This study was performed in line with the principles of the Declaration of Helsinki.

This retrospective study was conducted by examining the files of COVID-19 patients who presented to the emergency department of a district state hospital with chest pain between January 1, 2020, and December 31, 2020. Patients admitted to the emergency department with chest pain over the age of 18 and diagnosed with COVID-19 by PCR test were included in the study. Exclusion criteria were: age below 18 years; known as muscular and/or rheumatologic disease not diagnosed by PCR; mental-motor retardation; history of trauma; and incomplete file contents. General demographic characteristics (age, gender, comorbidities, smoking, etc.), vital signs (body temperature, pulse rate, arterial blood pressure (BP), oxygen saturation), type of pain, time of presentation, and time of onset of chest pain were evaluated from the patient files. Typical cardiac chest pain was considered to be a pressing, compressive chest pain behind the sternum lasting 5-15 minutes, relieved by rest and nitrate, not changing respirationally and positionally.[5] In addition, complete blood count (CBC), C-reactive protein (CRP), chest radiography, ferritin, lactate dehydrogenase (LDH), liver function tests, renal function tests, electrolytes, creatine kinase (CK), CK-MB, troponin I, electrocardiography (ECG), and echocardiography (ECHO) were evaluated. Chest pain of cardiac origin was compared with noncardiac chest pain.

Statistical Analysis

Data were evaluated using the statistical package program IBM SPSS Statistics Standard Concurrent User V 25 (IBM Corp., Armonk, New York, USA). Descriptive statistics were given as number of units (n), percentage (%), mean±standard deviation ($\bar{x}$±sh), median (M), minimum value (min), maximum value (max), first quartile (Q1), and third quartile (Q3). The log10 transformation was applied to variables with a wide distribution range. The normal distribution of the numerical variables was evaluated by the Shapiro-Wilk normality test and Q-Q graphs. The homogeneity of variances was evaluated by Levene's test. Comparisons between groups with categorical variables were evaluated with the Fisher exact test in the 2×2 and r×c tables. In cardiac and non-cardiac groups, the age variable was compared by an independent two sample t test, and the day variable was compared by a Mann-Whitney U test. Since the age variable differed between the groups, numerical variables were compared with one-way covariance analysis. A value of $P<0.05$ was considered statistically significant.

RESULTS

The study included 102 COVID-19 patients. The median age of the patients was 59.5 (24-78) years. The number of male and female patients was equal. Thirty-six of the patients (35.3%) presented with typical chest pain. The median body temperature was 36.9 (36.5-39.1) °C, the median systolic blood pressure was 120 (100-130) mmHg, the median diastolic blood pressure was 80 (70-80) mmHg, the median pulse rate was 79 (71.7-92) minutes, and the mean oxygen saturation was 93.5±5.7% (**Table 1**). The median interval between the onset of symptoms and hospital admission was 7 (1-12) days. 93.1% of the patients were admitted to the healthcare facility within the first 24 hours of experiencing chest pain. Fifty-two percent of the patients had comorbidities (Diyabetes Mellitus (DM), Hypertension (HT), Hyperlipidemia (HL), Coronary artery disease (CAD), Chronic obstructive pulmonary disease (COPD), Congestive heart failure (CHF)). 18 patients had one comorbidity, 17 patients had two, 5 patients had three, 7 patients had four, and 6 patients had five. The smoking rate was 17.6%. ECGs were normal (81.4%), T changes (7.8%), ST depression (6.9%), and ST elevation (3.9%), respectively (**Table 2**). White blood cell (WBC) median was 6.95 (5.9-9.25), hemoglobin median was 13.8 (12.3-15.2), ferritin median was 138.0 (62.7-337.2), CRP median was 8.0 (2.0-59.5), CK median was 141.5 (95.0-210.0), CKMB median was 26.0 (16.0-37.2), and 18.6% of patients had lymphopenia. Two patients died. The most common causes of GA in COVID-19 patients were musculoskeletal system (39.2%), respiratory system (23.5%), CVS diseases (20.6%), idiopathic causes (8.8%), gastrointestinal system diseases (7.9%). Cardiac causes were identified in 20.6% of chest pain and included Non ST-segment elevation myocardial infarction (NSTMI), arrhythmia, ST-segment elevation myocardial infarction (STMI), and unstable angina pectoris (USAP), respectively. 23 (28.4%) patients were diagnosed with pneumonia and 1 (1.2%) with pulmonary embolism. When patients with cardiac and noncardiac chest pain were compared, there was a significant difference between age ($p=0.01$), type of chest pain ($p<0.001$), comorbidity ($p=0.01$), and WBC count ($p=0.002$). There was a positive correlation between the number of comorbidities and the number of GA of cardiac origin ($p<0.001$). There was no significant difference between the two groups in terms of gender, day of illness, time of admission, smoking, liver and renal function tests, oxygen saturation, systolic and diastolic blood pressures, electrolytes, lymphocyte count, hemoglobin, LDH, CRP, CK, CK-MB, and troponin values (**Table 3**). There was no significant difference in age, gender, smoking, WBC, CK-MB, CK, LDH, but body temperature and

CRP values were significantly higher in patients with pneumonia (p<0.001). The troponin value was higher in patients with cardiac chest pain (p=0.02) and the ferritin value was higher in patients with pneumonia (p=0.01) (Table 4).

Tablo 1. Demographic and clinical characteristics of patients

Variables	Statistics
Age (year) M (min-max)	59.5 (24.0-78.0)
Gender, n (%)	
Female	51 (50.0)
Male	51 (50.0)
Pain type, n (%)	
Typical	36 (35.3)
Atypical	66 (64.7)
Application time, n (%)	
<12 hours	37 (36.3)
12-24 hours	58 (56.8)
>24 hours	7 (6.9)
Chest pain, n (%)	
Cardiac	21 (20.6)
Non-cardiyac	81 (79.4)
Cardiac causes, n (%)	n=21
STEMI	4 (19.0)
NSTMI	8 (38.1)
USAP	3 (14.3)
Aritmi	6 (28.6)
Non-cardiac causes, n (%)	n=81
Pneumonia	23 (28,4)
Pulmonary Embolism	1 (1.2)
Other	57 (70.4)

NSTMI: Non ST-Segment Elevation Myocardial Infarction, STEMI: ST-Segment Elevation Myocardial Infarction, USAP: Unstable Angina Pectoris.

Tablo 3. Patients' laboratory parameters according to chest pain

Variables	Chest Pain		Test statistics	
	Non-cardiac $\bar{x}$±sh	Cardiak $\bar{x}$±sh	F value	p value
WBC (10^3 /µL)	7.515±0.309	9.969±0.690	10.399	0.002
Hb (g/dL)	13.55±0.22	14.26±0.48	0.241	0.625
Lymphopenia, n (%)	16 (84.2)	18 (21.7)	0.329	0.757
logLDH(U/L)	2.408±0.027	2.513±0.061	0.847	0.360
logCRP (mg/L)	1.040±0.074	1.128±0.164	0.295	0.588
logFerritin (ml/ng)	2.104±0.073	2.198±0.163	0.151	0.04
logCK (ng/mL)	2.072±0.029	2.269±0.065	0.242	0.624
logCKMB (ng/mL)	1.407±0.037	1.591±0.081	0.283	0.596
Troponin (pg/ml)	0.012±0.118	1.260±0.262	2.403	0.124
Kreatinin (mg/dl)	0.897±0.076	0.919±0.169	0.060	0.806
AST (U/L)	34.8±1.6	39.3±3.6	0.067	0.796
ALT (U/L)	35.1±1.6	39.9±3.5	0.156	0.694

CK: creatine kinase, CRP: C-reactive protein, LDH: Lactate dehydrogenase, WBC: White blood cell, AST: Alanine aminotransferase, AST: aspartate aminotransferase. F: One-way analysis of covariance test statistics.

Tablo 4. Characteristics of patients according to inflammation parameters

Variables	Chest Pain			F value	p value
	Pneumonia $\bar{x}$±sh	Other $\bar{x}$±sh	Cardiac $\bar{x}$±sh		
CRP (mg/L)	2.041±0.077[a]	0.636±0.048[b]	1.128±0.089[c]	12.245	<0.001
Ferritin (ml/ng)	1.91±0.11[a]	2.03±0.74[b]	2.19±0.16[c]	12.160	<0.001

Superscripts a, b, and c indicate the difference between groups.

Tablo 2. Comparison of patient characteristics according to chest pain

Variables	Chest Pain		Test Statistics	
	Non-cardiac	Cardiac	Test value	p value
Age (year) $\bar{x}$±ss	54.1±15.7	63.1±13.2	t=2.665	0.011
Gender, n (%)			χ2=0.540	0.625
Female	42 (82.4)	9 (17.6)		
Male	39 (76.5)	12 (23.5)		
Type, n (%)			χ2=35.260	<0.001
Typical	17 (47.2)	19 (52.8)		
Atypical	64 (97.0)	2 (3.0)		
Starting day M (min-max)	7.0 (5.0-8.0)	8.0 (5.0-9.5)	z=1.470	0.142
Application time, n (%)			χ2=0.579	0.739
<12 hours	28 (75.7)	9 (24.3)		
12-24 hours	47 (81.0)	11 (19.0)		
>24 hours	6 (85.7)	1 (14.3)		
Additional illness, n (%)			χ2=6.220	0.015
No	44 (89.8)	5 (10.2)		
Yes	37 (69.8)	16 (30.2)		
Additional illness count, n (%)			χ2=13.939	0.016
No	44 (89.8)	5 (10.2)		
One	16 (88.9)	2 (11.1)		
Two	11 (64.7)	6 (35.3)		
Three	3 (60.0)	2 (40.0)		
Four	3 (42.9)	4 (57.1)		
Five	4 (66.7)	2 (33.3)		
Additional illness type, n (%)*				
DM	14 (53.8)	12 (46.2)	χ2=13.950	<0.001
HT	27 (71.1)	11 (28.9)	χ2=2.588	0.132
CAD	15 (65.2)	8 (34.8)	χ2=3.660	0.078
HL	10 (58.8)	7 (41.2)	χ2=5.289	0.043
CHF	6 (60.0)	4 (40.0)	χ2=2.555	0.209
COPD	7 (63.6)	4 (36.4)	χ2=1.877	0.231
Smoking, n (%)			χ2=0.206	0.759
No	66 (78.6)	18 (21.4)		
Yes	15 (83.3)	3 (16.7)		
EKG, n (%)			χ2=51.328	<0.001
Normal	76 (91.6)	7 (8.4)		
ST elevation	0 (0.0)	4 (100)		
ST depression	0 (0.0)	7 (100)		
T change	5 (62.5)	3 (37.5)		

COPD: Chronic obstructive pulmonary disease, CHF: congestive heart failure, CAD: coronary artery disease, HT: hypertension, DM:Diyabetes mellitus.

DISCUSSION

This study has shown that COVID-19 patients presenting to the emergency department with chest pain have more comorbidities, and approximately 1/5 of them have chest pain of cardiac origin. To our knowledge, our study is the first study in the literature investigating the causes of GA in COVID-19.

Chest pain is one of the most common reasons for admission to emergency departments, both before and during the pandemic. Chest pain is a common symptom in symptomatic COVID-19 patients.[6] The most common causes of chest pain in adults during non-pandemic periods are musculoskeletal system, CVS, respiratory system, psychiatric, and gastrointestinal system pathologies, respectively. In a study conducted in our country, the most common causes of chest pain were found to be the musculoskeletal system, gastrointestinal system, respiratory system, CVS, and psychological causes, respectively.[7] In our study, the most common causes of GA in COVID-19 patients were musculoskeletal system, respiratory, and CVS diseases, respectively. SARS-CoV2 virus enters the human body by attaching to angiotensin-converting enzyme-2 (ACE2) receptors in the cells of the lung and heart, esophagus, ileum, kidney, proximal tubule, and bladder.[8] ACE2 plays an important role in the neurohumoral regulation of CVS in various disease states and in normal health. The binding of SARS-CoV-2 to ACE2 leads to an alteration of ACE2 signaling pathways, which may primarily lead to acute myocardial and lung injury. As a result of the inflammatory effects of the SARS-CoV2 virus on cells, many clinical symptoms, including chest pain, can be observed.[9,10]

Cardiovascular system pathologies are the most common causes of chest pain in adults. Although the respiratory system is the system most affected by COVID-19, the highest mortality rate is due to CVS pathologies. In COVID-19, conditions such as advanced age, male gender, chronic lung disease, chronic heart disease, CRF, DM, HT, obesity, smoking, and malignancy are risk factors for mortality.[11] Especially those with advanced age and known CVD constitute the riskiest group. The mortality rate is also high in young COVID-19 patients with CVD.[3,12] The fact that advanced age and male gender are the main risk factors for CVD supports these results.[13] Similar to the literature, the patients who died in our study were male, over 65 years of age, and had comorbid diseases (DM, HT). In studies, COPD, CVD, DM, and HT were found to be comorbid risk factors.[14,15] In another study, the mean number of comorbid diseases in patients who died due to COVID-19 was found to be 2.7.[16] In our study, similar to the literature, 51.9% of our patients had comorbid diseases, and they were HT, DM, CAD, HL, COPD, and CHF, respectively.

The clinical presentation in COVID-19 patients, which is considered acute cardiac damage, has been reported as acute HF, acute myocardial infarction, myocarditis, arrhythmia and sudden cardiac arrest.[1,4] In COVID-19 infection, ECG changes and elevation in cardiac enzymes may be observed due to both direct damage of the virus to the myocardium and microvascular damage. Zhou et al.[6] found that 23% of the entire patient group and 52% of the patients who died had HF in their study. In our study, HF was detected in 9.8% of patients with chest pain and the most common ECG pathology in patients with cardiac involvement was T change, ST depression and ST elevation, respectively. Studies show that arrhythmic events are not uncommon in COVID-19 patients. Increased prevalence of arrhythmia has been found to be related to hypoxia, concomitant CVD, neurohumoral and inflammatory stress, and drugs used in treatment (18,19). One study showed that the incidence of cardiac arrhythmia was 9.3% (20). Sinus tachycardia, which occurs due to many reasons including fever, respiratory failure/hypoxemia, hemodynamic deterioration, fear/anxiety, pain, and some other physical and emotional symptoms, is the most common rhythm disorder in patients with COVID-19 infection (21). In our study, the arrhythmia rate was 28.6% of cardiac pathologies, and the most common arrhythmia was sinus tachycardia, respectively.

The most commonly used laboratory tests in COVID-19 disease include complete blood count, liver and kidney function tests, cardiac enzymes (CK, CK-MB, troponin), electrolytes, CRP, coagulation tests, procalcitonin, and LDH. Non-cardiac patients had significantly lower leukocyte counts than cardiac patients (p=0.002) and there was no difference in lymphopenia. Increased high-sensitivity cardiac troponin (hs-cTn) levels have been reported in a significant proportion of COVID-19 patients. Elevated troponin levels in these patients are considered an important indicator of poor prognosis and increased mortality (1,18). Because of the intense distribution of ACE2, the binding point of SARS-CoV-2, in cardiac myocyte cells, the high affinity of the virus to these cells and thus the destruction caused by the virus are held responsible for the elevation in troponin (1,22). In our study, the troponin values of patients with CVS involvement were higher than those of non-cardiac patients (p=0.02), but there was no significant difference in terms of CK and CK-MB. The limitation of our study is that it is retrospective and not multicenter.

CONCLUSION

Patients with a pre-diagnosis of COVID-19 presenting to the emergency department with chest pain should be considered to have more comorbid diseases. In addition, although the most common cause of chest pain is of musculoskeletal origin, it should not be forgotten that it may be caused by CAD.

ETHICAL DECLARATIONS

Ethics Committee Approval: The study was carried out with the permission of Kayseri City Hospital Ethics Committee (Date: Date: 09.2020, Decision No: 09/2020-146).

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 authors have no conflicts of interest to declare.

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REFERENCES

1. Wang D, Hu B, Hu C, et al. Clinical characteristics of 138 hospitalized patients with 2019 novel coronavirus-Infected pneumonia in Wuhan, China. *JAMA*. 2020;323(11):1061-1069. doi:10.1001/jama.2020.1585
2. Zhou P, Yang XL, Wang XG, et al. A pneumonia outbreak associated with a new coronavirus of probable bat origin. *Nature*. 2020;579(7798):270-273. doi:10.1038/s41586-020-2012-7
3. Aktoz M, Altay H, Aslanger E, et al. Türk Kardiyoloji Derneği Uzlaşı Raporu: COVID-19 Pandemisi ve Kardiyovasküler Hastalıklar Konusunda Bilinmesi Gerekenler (13 Mayıs 2020). *Turk Kardiyol Dern Ars*. 2020;48(Suppl 1):1-87. doi:10.5543/tkda.2020.36713
4. Huang C, Wang Y, Li X, et al. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. *Lancet*. 2020;395(10223):497-506. doi:10.1016/S0140-6736(20)30183-5
5. Gersh BJ, Braunwald E, Bonow RO. Chronic coronary artery desease. In: Braunwald E, Zipes DP, Libby P, editors. Heart Desease: A Textbook of Cardiovasculer Medicine. 6th. ed. Philadelphia: WB Saunders; 2001.p.1272-7
6. The European Society of Cardiology. ESC Guidance for the Diagnosis and Management of CV Disease during the COVID-19 Pandemic. Last updated on 21 April 2020.
7. Koç İ, Doğan Y, Doğan S, Köker S, Dökme A, Kaya A. Clinical follow up of patients admitting to pulmonary clinic with chest pain. *Selçuk Tıp Derg.* 2015;31(3):118-120
8. Guan WJ, Ni ZY, Hu Y, et al. Clinical Characteristics of Coronavirus Disease 2019 in China. *N Engl J Med*. 2020;382(18):1708-1720. doi:10.1056/NEJMoa2002032
9. Xiong TY, Redwood S, Prendergast B, Chen M. Coronaviruses and the cardiovascular system: acute and long-term implications. *Eur Heart J*. 2020;41(19):1798-1800. doi:10.1093/eurheartj/ehaa231
10. Li B, Yang J, Zhao F, et al. Prevalence and impact of cardiovascular metabolic diseases on COVID-19 in China. *Clin Res Cardiol*. 2020;109(5):531-538. doi:10.1007/s00392-020-01626-9
11. Wang D, Hu B, Hu C, et al. Clinical Characteristics of 138 Hospitalized Patients With 2019 Novel Coronavirus-Infected Pneumonia in Wuhan, China. *JAMA*. 2020;323(11):1061-1069. doi:10.1001/jama.2020.1585
12. Verity R, Okell LC, Dorigatti I, et al. Estimates of the severity of coronavirus disease 2019: a model-based analysis. *Lancet Infect Dis*. 2020;20(6):669-677. doi:10.1016/S1473-3099(20)30243-7
13. Palmieri L, Andrianou X, Bella A, et al. Characteristics of COVID-19 patients dying in Italy Report based on available data on March 20th, 2020. COVID-19 Surveillance Group. Available at: https://www.epicentro.iss.it/coronavirus/bollettino/Report-COVID-2019_20_marzo_eng.pdf. Accessed Mar 23, 2020.
14. Wang B, Li R, Lu Z, Huang Y. Does comorbidity increase the risk of patients with COVID-19: evidence from meta-analysis. *Aging (Albany NY)*. 2020;12(7):6049-6057. doi:10.18632/aging.103000
15. Richardson S, Hirsch JS, Narasimhan M, et al. Presenting characteristics, comorbidities, and outcomes among 5700 patients hospitalized with COVID-19 in the New York City Area. *JAMA*. 2020;323(20):2052-2059. doi:10.1001/jama.2020.6775
16. Onder G, Rezza G, Brusaferro S. Case-fatality rate and characteristics of patients dying in relation to COVID-19 in Italy. *JAMA*. 2020;323(18):1775-1776. doi:10.1001/jama.2020.4683
17. Zhou F, Yu T, Du R, et al. Clinical course and risk factors for mortality of adult inpatients with COVID-19 in Wuhan, China: a retrospective cohort study. *Lancet*. 2020;395(10229):1038.
18. Ruan Q, Yang K, Wang W, Jiang L, Song J. Clinical predictors of mortality due to COVID-19 based on an analysis of data of 150 patients from Wuhan, China. *Intensive Care Med*. 2020;46(5):846-848. doi:10.1007/s00134-020-05991-x
19. Li B, Yang J, Zhao F, et al. Prevalence and impact of cardiovascular metabolic diseases on COVID-19 in China. *Clin Res Cardiol*. 2020;109(5):531-538. doi:10.1007/s00392-020-01626-9
20. Kunutsor SK, Laukkanen JA. Cardiovascular complications in COVID-19: A systematic review and meta-analysis. *J Infect*. 2020;81(2):e139-e141. doi:10.1016/j.jinf.2020.05.068
21. Chen Q, Xu L, Dai Y, et al. Cardiovascular manifestations in severe and critical patients with COVID-19. *Clin Cardiol*. 2020;43(7):796-802. doi:10.1002/clc.23384
22. American College of Cardiology. Troponin and BNP Use in COVID-19. Available at: https://www.acc.org/latest-in cardiology/articles/2020/03/18/15/25/troponin-and-bnp use-in covid19?utm_source=accupdate&utm_medium=email_newsletter&utm_content=20200320&utm_campaign=accupdate. Accessed Mar 23, 2020.