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COVID-19-induced Acute Coronary Syndrome: A Review of 77 Cases

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Abstract

BACKGROUND: Existing research related to the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which caused the COVID-19 pandemic, has found that this is not only a respiratory disease. Instead, it can affect other organs such as the heart, increasing disease severity.

AIM: This study aims to review acute coronary syndrome (ACS) cases that result from COVID-19 to improve comprehension of the presentation, clinical features, diagnostic process, and outcomes of this rare but potentially severe complication.

METHODS: In this review, case report studies confirmed ACS and a reported diagnosis of COVID-19 was included in the study. A review of literature was performed on PubMed, Web of Science, and Scopus. However, only 77 cases met the inclusion criteria.

RESULTS: Regarding the clinical features of COVID-19 infection, the symptoms patients most frequently presented with were shortness of breath (SOB), fatigue, fever, and cough. In addition, it was found that the right coronary artery (RCA) was the most involved vessel.

CONCLUSION: The cases with ACS frequently displayed typical symptoms of fever, chest pain, SOB, and a cough. However, this review shows that no association was found between ACS and the COVID-19 pandemic.

Introduction

COVID-19 is a disease caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Clinical manifestations of COVID-19 show significant variation from asymptomatic to critical. Patients most commonly present with fever, fatigue, shortness of breath (SOB), a cough, expectoration, anorexia, and sputum production. The less frequent clinical symptoms include headache, sore throat, confusion, hemoptysis, and chest tightness [1]. Advanced age, obesity, and existing comorbidities such as diabetes and hypertension have been established as risk factors that could lead to more severe cases, along with organ damage, which most frequently affects the heart, liver and/or kidneys, and even death [2].

The primary symptom of COVID-19 is acute hypoxemic respiratory failure (AHRF). However, a range of associated cardiovascular (CV) issues has been identified. These are more likely to be linked with more severe disease, worse outcomes, and above all higher mortality [3]. Reports suggest that acute CV manifestations have an incidence rate of 12–28% on average, which can increase to 31% among patients in the intensive care unit (ICU) [3]. The leading COVID-19 CV complications are acute cardiac injury, acute myocardial infarction (AMI), myocarditis, arrhythmia, heart failure, and shock [4]. Moreover, acute coronary syndrome (ACS) was also recorded in a significant percentage of COVID-19 patients [5].

The underlying pathogenesis is still uncertain. However, several possible mechanisms have been suggested, such as direct viral cellular damage and systemic inflammatory response with cytokine-mediated injury, microvascular thrombosis, endothelial dysfunction, and an oxygen supply/demand imbalance due to a severe hypoxic state [6]. As with other infective diseases, COVID-19 may also promote atherosclerotic plaque instability and thrombus formation, which, in turn, precipitates acute coronary syndrome events [7]. This article thus seeks to review ACS cases that result from COVID-19 to improve comprehension of the presentation, clinical features, diagnostic process, and outcomes of this rare but potentially severe complication.

Materials and Methods

The research strategy involves two stages, as described below.

Review articles on COVID-19-induced acute coronary syndrome

Between May 2021 and June 2022, a systematic search was performed on PubMed, Web of Science, Embase, Scopus, and Google Scholar. The terms employed included coronavirus OR COVID-19 OR MERS OR SARS AND acute coronary syndrome OR ACS OR coronary heart disease OR coronary artery disease.

Data extraction

Two authors independently screened the research title and abstracts. They then extracted data related to patient characteristics, investigation, management, and outcomes onto a standardized form. Inclusion criteria included only case reports confirmed with ischemic heart disease and reported diagnosis of COVID-19 and articles in English-language peer-reviewed studies. Articles other than case reports or not written in the English language and/or studies that did not report ACS (e.g., myocarditis, animal studies, or cardiac injury) were excluded from the study. All duplicate results were removed. From 1332 initial results, the search was reduced to 533 articles following the removal of duplicates. After the application of the inclusion criteria, 77 case reports from different countries remained (Figure 1). The included studies were critically appraised, with the modified Pierson and Newcastle-Ottawa scale used to assess the quality of the case reports [8].

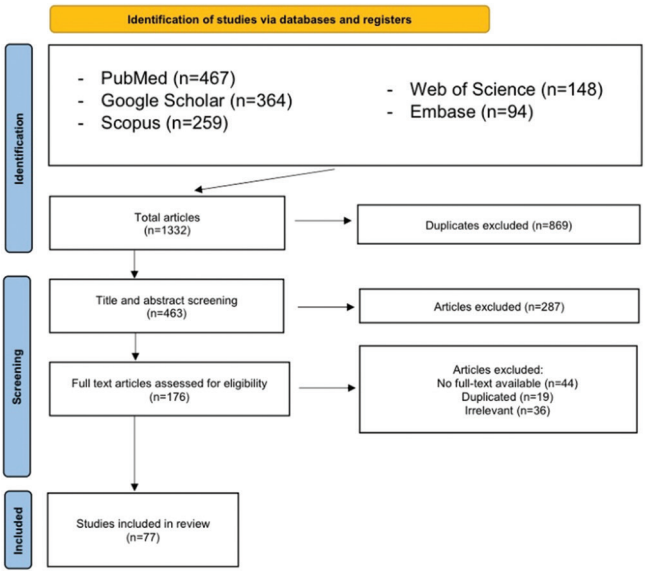

Figure 1: Preferred reporting item for systemic review and meta-analysis flow diagram of the process of literature search, screening, and inclusion of the studies

Statistical analysis

Following a review of all articles that included study flow charts and a summary of pooled ACS cases in COVID-19 patients with different variables, the data were entered and coded with IBM SPSS version 22 (SPSS, Inc. Chicago, IL). Statistical analysis was performed using two-tailed tests. $p < 0.05$ was statistically significant. All variables including personal data, medical history, management, and outcome presented as frequency and percentage. Furthermore, Pearson Chi-square test was used to test the relation between variables and patients with confirmed or probable cases.

Results

Of an initial 1332 articles, 533 remained after duplicate results were removed. From these, 77 case reports accorded with the inclusion criteria. Regarding demographics, 67.5% (i.e., 52 cases) were male. The most common comorbidities were hypertension (46.8%), followed by obesity (29.9%) and hyperlipidemia (9.1%) (Table 1).

Regarding the clinical features of COVID-19 infection among the reviewed patients, the most frequent symptoms they presented with were SOB (23.2%), fatigue (22.1%), fever (16.8%), cough (15.5%), chest pain (13.5%), nausea and/or vomiting (N/V) (2.6%), and hypoxia (1.3%). About 5% of reviewed cases were found to be asymptomatic. Regarding the clinical features of ACS, meanwhile, patients most commonly presented with chest pain (74%), SOB (44.2%), and orthopnea/paroxysmal nocturnal dyspnea (2.6%). Moreover, 6.5% were asymptomatic.

Concerning the treatment protocols adopted for COVID-19 infection among the reviewed cases, anti-viral was the most frequently reported (19.5%). Steroids were also used in 15.6% of reviewed cases. On the subject of the treatment protocol for ACS among reviewed cases of COVID-19 infection, meanwhile, thrombolytic thereby was the most reported (49.4%). Percutaneous coronary intervention (PCI) was reported in 32.5% of cases, whereas only 2.6% reported diagnostic coronary angiography.

Regarding the laboratory findings of the reviewed cases, troponin level was found to be elevated in 45.5% of patients. Pro B was elevated at 11.7%. White blood count (WBC) ranged from 6.5 to 23.4 × 109/L, with an average value of 11. In 18.2% of cases 7 × 109 was elevated. C-reactive protein (CRP) was high at 35.1% and lactate was high at 14.3%. Ferritin levels were high at 19.5%, ranging from 243 to 145,000 g/dl, with an average level of 1030 g/dl. D-Dimer was high in 35.1% of patients, with a level that ranged from 1.8 to 88,868 and an average of 1200. Lactate dehydrogenase (LDH) was high in 13% of cases.

Concerning electrocardiogram (ECG) change, ST-segment elevation in the inferior lead was most frequently reported in the reviewed cases (28.5%). This

Table 1: Characteristic of published cases of coronavirus-induced ischemic heart disease

Variable	Categories	Total cases (n = 77) (%)	Confirmed COVID-19 (n = 69) (%)	Possible COVID-19 (n = 8) (%)	p-value
Gender	Male	52 (67.5)	45 (65.2)	7 (87.5)	0.203
	Female	25 (32.5)	24 (34.7)	1 (12.5)	
Presenting symptoms	Chest pain	21 (27.3)	18 (26)	3 (37.5)	0.493
	Fever	26 (33.8)	24 (34.7)	2 (25)	0.580
	SOB	36 (46.6)	32 (46.3)	4 (50)	0.846
	Cough	24 (31.2)	23 (33.3)	1 (12.5)	0.228
	Fatigue	17 (22.1)	17 (24.6)	0	0.112
	Hypoxia	2 (2.6)	0	2 (25)	0.000
	N/V	4 (5.2)	4 (5.7)	0	0.484
ACS presentation	Chest pain	57 (74)	50 (72.4)	7 (87.5)	0.359
	SOB	34 (44.2)	30 (43.4)	4 (50)	0.725
	Orthopnea/PND	2 (2.6)	2 (2.8)	0	0.626
	Asymptomatic	5 (6.5)	4 (5.7)	1 (12.5)	0.466
Co-morbidities	HTN	36 (46.8)	35 (50.7)	1 (12.5)	0.400
	DM2	19 (24.7)	1 (1.4)	0	0.647
	Hyperlipidemia	7 (9.1)	6 (8.6)	1 (12.5)	0.723
	Obesity	23 (29.9)	21 (30.4)	2 (25)	0.751
ECG	ST-segment elevation in inferior lead	22 (28.5)	16 (23.1)	6 (75)	0.000*
	ST-segment elevation in anterior-lateral lead	10 (12.9)	10 (14.4)	0	
	ST-segment elevation in lateral lead	7 (9)	5 (7.2)	2 (25)	
	ST-segment elevation in anterior lead	6 (7.7)	4 (5.7)	2 (25)	
	ST-segment change	33 (42.3)	30 (43.4)	3 (37.5)	
COVID-19 management	Steroid	12 (15.6)	11 (15.9)	1 (12.5)	0.799
	Anti-viral	15 (19.5)	15 (19.5)	0	0.142
ACS management	Thrombolytic thereby	38 (49.4)	36 (52.1)	2 (25)	0.146
	coronary Angio	2 (2.6)	2 (2.8)	0	0.626
	Diagnostic PCI	25 (32.5)	23 (33.3)	2 (25)	0.634
Vessels involved	RCA	19 (24.7)	17 (24.6)	2 (25)	0.885
	Proximal LAD	1 (1.3)	1 (1.4)	0	
	Posterior descending	1 (1.3)	1 (1.4)	0	
	artery	3 (3.9)	2 (2.8)	1 (12.5)	
	LCx	15 (19.5)	13 (18.8)	2 (25)	
	LAD	4 (5.2)	31 (44.9)	3 (37.5)	
	Multi	4 (5.2)	4 (5.7)	0	
Serology	Troponin I	35 (45.5)	32 (46.3)	3 (37.5)	0.892
	Pro BNP	9 (11.7)	9 (13)	0	0.256
	WBC	14 (18.2)	12 (17.3)	2 (25)	0.422
	CRP	27 (35.1)	24 (34.7)	3 (37.5)	0.981
	Lactate	11 (14.3)	11 (15.9)	0	0.223
	Ferritin	15 (19.5)	15 (21.7)	0	0.400
	D-dimer	27 (35.1)	25 (36.2)	2 (25)	0.560
	LDH	10 (13.0)	10 (14.4)	0	0.248
ICU admitted		32 (41.6)	29 (42)	3 (37.5)	0.907
Outcomes	Death	18 (23.4)	15 (21.7)	3 (37.5)	0.426
	Recover/discharge	51 (66.2)	46 (66.6)	5 (62.5)	
	Undetermined	8 (10.4)	8 (11.5)	0	

was followed by ST-segment elevation in the anterior-lateral lead (12.9%) and ST-segment elevation in the lateral lead (9%). T-wave change, arrhythmia, and ST-segment elevation in the anterior lead were reported in 7.7% of cases, with ST-segment change, therefore, reported in 42.8% of the reviewed cases.

Respecting vessels involvement, among the reviewed cases of COVID-19 infection, the right coronary artery (RCA) was the most involved (24.7%). This was followed by the left anterior descending artery (LAD; 19.5%), left circumflex artery (LCX; 3.9%), proximal LAD, and posterior descending artery (both 1.3%). Multiple vessel involvement was found in 5.2% of reviewed cases.

A total of 52 of the 77 patients were discharged alive, and 18 died, whereas eight had undetermined outcomes in the case reports. Of 69 patients with ischemic heart disease (IHD) and COVID-19 infection, 29 were admitted to an ICU.

Discussion

From the start of the COVID-19 pandemic, there have been major concerns regarding the impact the disease may have on the lungs, heart, and other organ systems. This study was therefore undertaken to review cases of ACS that may have been caused by COVID-19. The term ACS is used in cases where myocardial infarction (MI) is suspected or confirmed [9]. MI has been reported in up to 12% of patients hospitalized with COVID-19 [9], [10]. Recent guidelines identify ACS from increased or decreased values of cardiac troponins in addition to symptoms suggestive of myocardial ischemia, evidence of ECG abnormalities, and indicative recent myocardial functional impairment or intracoronary thrombosis [11]. The plausible mechanisms of COVID-19 myocardial injury are a direct effect mediated by the SARS-COV-2 virus harnessing the angiotensin-converting enzyme-2 receptor to access the host cell [12]. This may also result in endothelial cell and microvascular dysfunction/occlusion [13]. Additional mechanisms have been suggested, including increased thrombosis and pro-coagulative changes, plaque rupture, demand ischemia, or vasospasm [14].

Patients with acute MI commonly present with chest pain, dyspnea, and fatigue, symptoms much like those from viral infections such as COVID-19 with or without IHD [15]. Most cases included in the review had at least one of these symptoms, along with other symptoms such as a cough, diarrhea, N/V, and sweating.

The true prevalence of chest pain in COVID-19 patients remains unknown, however [16], [17]. In part, this is because few ACS patients elected to visit a hospital during the pandemic because they feared contracting COVID-19 [18]. Our data indicate that no relation exists between COVID-19 and increased ICU admission or mortality rates. In contrast, other research found that cardiac injury, described by increased cardiac troponins, correlated with an increased need for ICU treatment, increased severity of COVID-19, and higher risk of mortality. The last mentioned is likely linked with the mortality directly associated with COVID-19, coupled with an inability to provide sufficient ACS management to all patients with COVID-19 [19], [20]. Furthermore, patients hospitalized with ACS had at least one comorbidity, most frequently diabetes and hypertension, which is similar to our data [21].

Regarding ACS patient management, for those known to be noninfectious, the usual approach of primary PCI was employed [22]. In confirmed COVID-19 cases, fibrinolytic therapy was more liberally used as the choice for early perfusion due to viral exposure risks for health-care personnel [23]. Elevated cardiac troponin was also recorded in between 10% and 30% of hospitalized COVID-19 patients [6], [19], [20], [21], [22], [23]. Most patients with troponin elevation and COVID-19 did not clinically present with ACS [24]. This review, therefore, found no relation between troponin elevation and induced ACS. All patients suspected of having COVID-19 should have a baseline ECG performed on admission to the hospital [25]. Given this, the review found that most patients with suspected or confirmed COVID-19 had ST elevation. Moreover, there are several reports of ST elevation known to be associated with COVID-19 [26]. In a pre-pandemic and cohort study of 489 patients presenting to emergency department (ED) with an ECG indicating ST elevation, 11% of cases were found to have no causative lesion on coronary angiography [27]. The most common causes were pericarditis/myocarditis, early repolarization, and Takotsubo syndrome [27]. The left coronary artery was most frequently affected in ACS, followed by the right coronary artery, then the circumflex artery [28]. The pathophysiological mechanism implicated in this discrepancy is still unclear; however, the hemodynamic or anatomical difference between the right and left coronary artery may have a significant impact here [29]. Interestingly, the review found the most common affected artery is the RCA.

Research limitations

This review's main limitation was that it included only case reports that had no evidence of relationship and are not therefore generalizable. The risk of information bias is also high because the data are not complete in all cases. It is also possible that a patient's condition has been affected by comorbidities related to the renal, gastrointestinal, and/or respiratory systems. ACS could develop due to COVID-19 infection or pre-exist in patients, which means that the causal relationship was not possible to determine. Last, the review may be limited by the heterogeneous nature of the research it drew on; some studies were of moderate quality only. Moreover, prospective and observational studies may be better analyzed to detect significant association between COVID-19 and acute coronary syndrome.

Conclusion

This review identified 69 patients with COVID-19 and eight with possible COVID-19. The cases with ACS frequently displayed typical symptoms of fever, chest pain, SOB, and a cough. No association between IHD and the COVID-19 pandemic was found. However, the future research should focus on the reliable diagnosis of IHD in an outbreak scenario, thereby characterizing these patients in larger and prospective studies.

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