Infective Endocarditis and COVID 19:  
A Systematic Review

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Abstract  Coronavirus Disease-19 (COVID-19) is a pandemic caused by severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2). COVID-19 is known to cause a wide variety of cardiovascular manifestations, including myocarditis, pericarditis, myocardial infarction, stroke, thrombus, pulmonary embolism and acute ventricular failure. In this paper, we explore cases of infective endocarditis (IE) that occurred in patients who were concurrently infected with COVID-19 and discuss its association in contributing factors that can ultimately lead to the development of infective endocarditis. Some of these factors that contribute to IE in COVID-19 include severe inflammatory response, endothelial damage and dysfunction and immunosuppression caused by medications that are used to treat COVID-19. In this systematic review, 12 papers detailing 15 pertinent cases of IE following an infection with COVID-19 were identified. Data from these cases were tabulated and analyzed. The ages of the patients ranged from 20-70 years, with 73% of the patients developing IE of native heart valves and the remaining 27% developing IE of mechanical and bioprosthetic valves. The three most common organism implicated were Enterococcus faecalis (28.57%), Methicillin Resistant Staphylococcus aureus (MRSA) (14.28%), and Methicillin Sensitive Staphylococcus aureus (MSSA) (14.28%). Medical management of these cases involved antibiotic therapy and was reported in 80% of the cases, while only one patient (6.67%) underwent surgical valve replacement. The mortality rate of the patients in this review was quite high at 38%, and other major complications included cardioembolic stroke (20%) and septic embolization to the extremities (6.67%). COVID-19 infection results in a severe inflammatory response caused by a variety of mechanisms. This severe degree of widespread inflammation may result in damage to the endocardium, thus creating an environment to which microorganisms can adhere to and colonize. Additionally, the immunosuppressive medications used in a COVID-19 infection can result in an increased risk of developing infections that have the potential to spread to the endocardium via a hematogenous route. Physicians should be aware of occurrences of IE in COVID-19, as delay in diagnosis and management may cause significant morbidity and mortality.

Keywords: Coronavirus Disease-19 (COVID-19), severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), infective endocarditis

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1. Introduction

Coronavirus disease 2019 (COVID-19), due to severe Acute Respiratory Syndrome Coronavirus 2 (SARS-COV2), remains a pandemic. Though initially considered a disease primarily of the respiratory system, subsequent data and emerging experiences are highlighting the wide variety of manifestations this disease can have. The cardiovascular system, in particular, is of special interest because cardiovascular risk factors and preexisting chronic cardiovascular conditions are prevalent among COVID-19 patients and are known to be associated with adverse outcomes [1]. Some of the cardiovascular complications of COVID-19 include ST-elevation myocardial infarction [2], myocarditis [3], apical takotsubo cardiomyopathy [4], cardiogenic shock [5], arrhythmias [5], pericardial effusions [5], and cardiac tamponade [5]. Thromboembolic complications are seen as well, including stroke [5], deep vein thrombosis [5], and pulmonary embolism via a thrombus in transit [6]. Infective endocarditis (IE) is a life-threatening disease with serious sequelae even in those that have been treated [7]. While there have been advances in medical and surgical management, IE still has substantial mortality and morbidity [8]. In addition, many of the symptoms of IE such as fever, chills, dyspnea, fatigue, cough, and myalgia may overlap with those of COVID-19, making it difficult to suspect the presence of IE [9]. Studies have shown increased incidence of IE during the COVID pandemic [10]. Here we present a systematic review of the cases of IE in COVID-19 patients in order to understand the clinical profile of these patients as well as the organisms implicated, the valves affected, management strategies employed and the outcomes.

2. Methods

A systematic search was conducted on January 15, 2021 using PubMed, Google Scholar, CINAHL, Cochrane CENTRAL and Web of Science databases (Figure 1). Studies listing the keywords “Coronavirus disease-19”, “COVID-19”, “SARS-COV2”, and “infective endocarditis” were used to identify cases of IE in patients with COVID-19. The reference list of each report was also checked for additional cases. All cases were reviewed in detail. Data reviewed included demographic details, symptoms, cardiovascular risk factors, echocardiographic findings, valves affected, blood cultures and organisms, treatment and outcomes. A total of 12 papers were included, from which 15 pertinent cases were identified.

3. Results

A total of 15 cases were identified from the systematic review (Table 1). The mean age was 54.13 ± 16.7 years and the median age of 60± 16.7 years. 85% of the cases affected males. All of the patients in the study (93%) had active COVID-19 infections on admission and 73% reported confirmed serologic testing with RT-PCR. Only 6% of the patients were reported to have had a previous COVID-19 infection. Of the patients admitted, 20% had presented initially with an additional infection source (detailed in Table 1). The presenting complaints have been tabulated (Table 2), with the majority presenting with fever (80%), dyspnea (66.66%) and cough (60%). The associated cardiovascular risk factors and co-morbidities in the population were as follows: Hypertension (20%), diabetes mellitus type II (13.33%), hyperlipidemia (13.33%), history of rheumatic heart disease (13.33%), obesity (6.67%), mitral valve replacement (6.67%), Mitral and Aortic prosthetic valve (6.67%), history of closure of Patent Foramen Ovale (6.67%), Bicuspid Aortic valve and Aortic Coarctation repair in childhood (6.67%).
A majority of the patients developed IE of a native valve (73%) and the remaining patients developed it in artificial valves (27%). The most common valves affected were the native mitral (33.33%) and aortic valve (33.33%), followed by prosthetic aortic valve (20%), prosthetic mitral valve (6.67%) and native tricuspid valve (6.67%) (Table 3). The four most common organism implicated were Enterococcus faecalis (28.57%), followed by S. aureus (20%), and Staphylococcus aureus (14.28%). Additional organisms that were identified also included Methicillin-Resistant Staphylococcus aureus (7.14%), Pseudomonas aeruginosa (7.14%) and Candida albicans (7.14%). Only one case (7.14%) reported culture negative findings (Table 4). Medical management of the cases consisted mainly of antibiotic therapy and was reported in 80% of the cases, with only 1 patient (6.67%) undergoing surgical valve replacement. Major complications included cardioembolic stroke (20 %) and septic embolization of extremities (6.67%). Fatality occurred in 38% of the cases (Table 5). The Echocardiographic findings for each case have been tabulated and displayed (Table 6).

Table 1. Cases included in the systematic review detailing COVID-19 status on admission and disease manifestations

| Case number | Year, Author | Age/SEX | COVID-19 Status | Diagnostic test | COVID-19 manifestations | Additional Infections on admission |
|-------------|--------------|---------|----------------|-----------------|------------------------|----------------------------------|
| 1           | Ramos-Martinez [10] | 70/Female | Active infection on admission. No prior infection noted | Positive RT–PCR for SARS COV-2 | - | Forearm phlebitis |
| 2           | Ramos-Martinez [10] | 70/Female | Active infection on admission. No prior infection noted | Positive RT–PCR for SARS COV-2 | - | Sternal wound infection |
| 3           | Ramos-Martinez [10] | 60/- | Active infection on admission. No prior infection noted | Positive RT–PCR for SARS COV-2 | - | - |
| 4           | Ramos-Martinez [10] | 60/- | Active infection on admission. No prior infection noted | Positive RT–PCR for SARS COV-2 | - | Central venous catheter infection |
| 5           | Alizadehshali [11] | 24/Male | Covid-19 infection 3 weeks prior. No prior infection noted | Positive RT–PCR for SARS COV-2 | - | Viral pneumonia |
| 6           | Amir [12] | 61/Male | Active infection on admission. No prior infection noted | Positive RT–PCR for SARS COV-2 | - | Viral pneumonia |
| 7           | Dias [13] | 36/Male | Active infection on admission. No prior infection noted | Positive RT–PCR for SARS COV-2 | - | Severe respiratory distress |
| 8           | Hussain [14] | 69/Male | Active infection noted. No prior infection noted | Positive RT–PCR for SARS COV-2 | - | Viral Pneumonia |
| 9           | Kwon [15] | 65/Male | Active infection on admission. No prior infection noted | - | - | - |
| 10          | Sanders [16] | 38/Male | Active infection on admission. No prior infection noted | Positive RT–PCR for SARS COV-2 | - | - |
| 11          | Spinoni [17] | 57/Male | Active infection on admission. No prior infection noted | Positive RT–PCR for SARS COV-2 | - | Acute respiratory distress syndrome (ARDS) |
| 12          | Velez-Paez [18] | 53/Male | Active infection on admission. No prior infection noted | Positive RT–PCR for SARS COV-2 | - | Acute respiratory distress syndrome (ARDS) |
| 13          | Regazzoni [19] | 70/Male | Active infection on admission. No prior infection noted | Positive RT–PCR for SARS COV-2 | - | Bilateral pneumonia |
| 14          | Mantero [20] | 59/Male | Active infection on admission. No prior infection noted | - | - | - |
| 15          | Toth [21] | 20/Male | Active infection on admission. No prior infection noted | - | - | Pneumonia |

*Age in years.
RT-PCR: Reverse transcription polymerase chain reaction.
SARS COV-2: Severe Acute Respiratory Syndrome Coronavirus 2

Table 2. Chief complaint at COVID-19 presentation

| Manifestation | Frequency |
|---------------|-----------|
| Fever         | 80%       |
| Dyspnea       | 66.66%    |
| Cough         | 60%       |
| Lower limb edema | 6.67% |
| Chest pain    | 6.67%     |
| Diarrhea      | 6.67%     |
| Aphasia and right facial deficit | 6.67% |
| Arthritis     | 6.67%     |
| Meningitis    | 6.67%     |

Table 3. Affected heart valves in COVID-19 associated endocarditis

| Valve Type     | Frequency |
|----------------|-----------|
| Native aortic valve | 33.33% |
| Native mitral valve | 33.33% |
| Prosthetic aortic valve | 20% |
| Prosthetic mitral valve | 6.67% |
| Native tricuspid valve | 6.67% |

Table 4. Organisms implicated in COVID-19 Infective endocarditis

| Organism Type          | Frequency |
|------------------------|-----------|
| Enterococcus faecalis  | 28.57%    |
| MRSA                   | 14.28%    |
| MSSA                   | 14.28%    |
| Staphylococcus aureus  | 14.28%    |
| Methicillin resistant Staphylococcus hominis | 7.14% |
| Pseudomonas aeruginosa | 7.14%    |
| Candida albicans       | 7.14%    |
| Culture-negative       | 7.14%    |

MRSA: Methicillin-resistant Staphylococcus aureus MSSA: Methicillin-sensitive Staphylococcus aureus.
Table 5. Table showing the implicated organisms and affected valves for the listed cases, as well as the therapeutic management and outcomes

| Case number | Year, Author          | Age/Sex | Organism implicated       | Valve affected     | Valve type | Medical management                      | Valve replacement | Complications                                      | Death |
|-------------|-----------------------|---------|---------------------------|--------------------|------------|-----------------------------------------|------------------|---------------------------------------------------|-------|
| 1           | Ramos-Martinez [10]    | 70/Female | Enterococcus faecalis     | Mitral valve       | Native     | Ampicillin, ceftriaxone                | -                | None                                              |       |
| 2           | Ramos-Martinez [10]    | 70/Female | Candida albicans          | Aortic valve       | Prosthetic | Anidulafungin, fluconazole             | -                | Yes                                               |       |
| 3           | Ramos-Martinez [10]    | 60/-    | Enterococcus faecalis     | Mitral valve       | Native     | Ampicillin, ceftriaxone                | -                | None                                              |       |
| 4           | Ramos-Martinez [10]    | 60/-    | Methicillin-sensitive     | Mitral valve       | Native     | Cefazolin                              | -                | None                                              |       |
| 5           | Alizadehasl [11]       | 24/Male | Staphylococcus aureus     | Mitral valve       | Mechanical | Azithromycin, hydroxychloroquine, corticosteroids | -                | None                                              |       |
| 6           | Amir [12]              | 61/Male | Culture negative          | Mitral valve       | Native     | -                                      | -                | Septic embolization to the right leg and right hand | Yes   |
| 7           | Dias [13]              | 36/Male | Methicillin-resistant     | Mitral valve       | Native     | Vancomycin, meropenem, gentamicin      | -                | Yes                                               |       |
| 8           | Hussain [14]           | 69/Male | Staphylococcus aureus     | Aortic valve       | Prosthetic | Gentamicin, flucloxacillin             | -                | Cardioembolic stroke causing right sided hemiparesis | None  |
| 9           | Kwon [15]              | 65/Male | Pseudomonas aeruginosa    | Mitral valve       | Native     | Piperacillin/tazobactam, levofloxacin  | -                | None                                              |       |
| 10          | Sanders [16]           | 38/Male | Enterococcus faecalis     | Aortic valve       | Native     | Ampicillin, ceftriaxone                | Yes              | None                                              |       |
| 11          | Spinoni [17]           | 57/Male | Methicillin-resistant     | Aortic valve       | Native     | Linezolid, cefazidime/avibactam, Fosfomycin | -                | None                                              |       |
| 12          | Velez-Paez [18]        | 53/Male | Methicillin-resistant     | Aortic valve       | Native     | Vancomycin, gentamicin                 | -                | Yes                                               |       |
| 13          | Regazzioni [19]        | 70/Male | Methicillin-sensitive     | Aortic valve       | Native     | -                                      | -                | Embolic stroke                                     | -     |
| 14          | Mantero [20]           | 59/Male | Enterococcus faecalis     | Aortic valve       | Prosthetic | Ampicillin, ceftriaxone                | -                | Embolic stroke                                     | -     |
| 15          | Toth [21]              | 20/Male | -                         | Aortic valve       | Native     | -                                      | -                | Yes                                               |       |

Table 6. Cases included in the systematic review and corresponding echocardiography findings

| Case number | Year, Author          | TTE          | ETE                                             |
|-------------|-----------------------|--------------|-------------------------------------------------|
| 1           | Ramos-Martinez [10]    | -            | Vegetation 7 mm on mitral valve                 |
| 2           | Ramos-Martinez [10]    | -            | Hypoechoic aortic perivalvular thickening (1.3 cm). Extension to mitral-aortic junction and aortic root (abscess) |
| 3           | Ramos-Martinez [10]    | -            | A 22mm Vegetation on the mitral valve. Moderate mitral insufficiency |
| 4           | Ramos-Martinez [10]    | -            | Mild mitral insufficiency without vegetations   |
| 5           | Alizadehasl [11]       | -            | Several typical vegetations on the posterior prosthetic mitral valve leaflet |
| 6           | Amir [12]              | Flail mitral leaflet with a vegetation measuring 10 mm × 3 mm in size, producing severe mitral regurgitation | - |
| 7           | Dias [13]              | 5 cm mobile vegetation on the tricuspid valve with severe tricuspid regurgitation | - |
| 8           | Hussain [14]           | -            | Vegetations on all three cusps of the aortic valve, with no significant aortic regurgitation, |
Table 1: CASE REPORTS

| Case number | Year, Author | TTE | TEE |
|-------------|--------------|-----|-----|
| 9           | Kwon [15]    | Vegetation of approximately 5 mm on the mitral valve | - |
| 10          | Sanders [16] | 10 x 11 mm mobile echo-density on the right coronary cusp as well as a possible smaller vegetation on the left coronary cusp of the aortic valve. | Two aortic valve vegetations: 8 x 14 mm on the right coronary cusp and 3 x 4 mm on the left coronary cusp. |
| 11          | Spinoni [17] | A poor ultrasound window, that was not conclusive for IE and showed mild mitral regurgitation | Presence of endocarditis vegetation (6 x 7 mm in diameter) on the non-coronary cusp of the aortic valve |
| 12          | Velez-Paez [18] | Vegetation was observed in the aortic valve without signs of valve insufficiency | - |
| 13          | Regazzoni [19] | - | Large vegetations on the aortic valve with severe regurgitation |
| 14          | Mantero [20] | IE at the level of the biological prosthetic valve and a suspected periprosthetic abscess. | - |
| 15          | Toth [21] | Severe aortic regurgitation, a large aortic valve vegetation, and aortic root abscess | - |

TTE: Transthoracic Echocardiography  TEE: Transesophageal Echocardiography  IE: Infective endocarditis

4. Discussion

Despite many advancements in care, IE is a disease that still carries a high rate of morbidity and mortality. Mortality rates can be approximately 25% even with appropriate management [7], and it carries a 1-year mortality of about 40% [9]. The occurrence of IE in the setting of COVID-19 infection could possibly be explained by the degree of inflammation caused by the COVID-19 infection. Damaged endocardial surfaces are a prime location for pathogenic organisms to adhere to and subsequently proliferate. Inflammation induced endocardial damage can be an appropriate site that a pathogen can adhere to and colonize [7]. The excessive inflammatory response of COVID-19 is already well defined through numerous mechanisms including a cytokine storm [21], so we can therefore hypothesize that the excessive inflammatory response may lead to damage of the endocardium of native heart valves. This can create a suitable surface to which pathogens can adhere to and precipitate the development of IE. (Figure 2) illustrates a summary of the events that a COVID-19 infection may lead to IE.

Additional factors that can increase the risk of developing IE include sepsis, immunosuppression and possessing prosthetic heart valves. Sepsis creates one of the core requirements for IE to take place, that is, the presence of an organism that is able to adhere to and colonize the endocardial surface [7]. COVID-19 patients that develop critical illness are at a higher risk of developing bacteremia or even fungemia [23]. This risk of sepsis is also increased due to widespread use of immunosuppressive agents involved in the treatment of COVID-19 patients. Lastly, prosthetic heart valves are a commonly known risk factor for IE [7] and it was seen in 27% of the patients in this study. All of these risk factors can compound with one another and increase the risk of IE. Unfortunately, many of the symptoms can overlap with other conditions and make diagnosis of IE difficult. Due to the high rate of mortality, knowledge of this potential complication is important so that timely treatment can be given and improve survival chances.

Since this systematic review focused on the published literature that reported an instance of stent thrombosis occurring in the setting of COVID-19 infection, it might be subject to selection bias. In addition, due to the limited number of published literature regarding this incidence, the low sample size is a limiting factor of this study. Lastly, all of the referenced studies were either case reports or case series without any control group to enhance the accuracy of the comparison.
5. Conclusions

The majority of patients with IE and COVID 19 were elderly males who presented with chief complaints of fever, dyspnea and cough. The most common valves that were affected, were the native mitral and aortic valves, followed by prosthetic aortic valves. The most common organism implicated was Enterococcus faecalis. One patient needed valvular replacement and a high mortality of 38% was noted. One of the main goals of this study is to alert physicians of coexisting IE in COVID-19 patients.

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