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Case Report

From Covid-19 To Cardiogenic Shock: Fulminant Myocarditis Mimicking St-Elevation Myocardial Infarction

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Abstract

Fulminant myocarditis can present with electrocardiographic features that mimic ST-elevation myocardial infarction (STEMI), posing diagnostic challenges. We report a case emphasizing the importance of systematic assessment prior to invasive intervention. A 29-year-old woman with no prior medical history developed cardiogenic shock five days post SARS-CoV-2 infection. Initial ECG revealed ST-elevation with left bundle branch block morphology and wide-complex tachycardia, initially suggestive of left anterior descending artery occlusion. However, transthoracic echocardiography demonstrated diffuse hypokinesia with preserved apical function, inconsistent with coronary distribution. The levels of cardiac biomarkers serially declined, therefore supporting an inflammatory process over ischemia. The medical team deferred catheterization, prioritizing hemodynamic stabilization with inotropic therapy and Impella support. Coronary angiography on day two revealed patent vessels. The left ventricular ejection fraction rapidly recovered from 20-25% to 60-65% within five days. Impella removal was complicated by femoral artery dissection requiring vascular surgery. This case demonstrates that ECG-echo discordance, preserved apical function despite anterior ST-elevation, and falling biomarkers should raise suspicion for fulminant myocarditis over acute coronary syndrome. Deferring catheterization until hemodynamic optimization may reduce iatrogenic risk while early mechanical support facilitates rapid myocardial recovery.

Kew Words: fulminant myocarditis; SARS-CoV-2; COVID-19; cardiogenic shock, ST-elevation myocardial infarction; echocardiography; Impella; mechanical circulatory support

Introduction

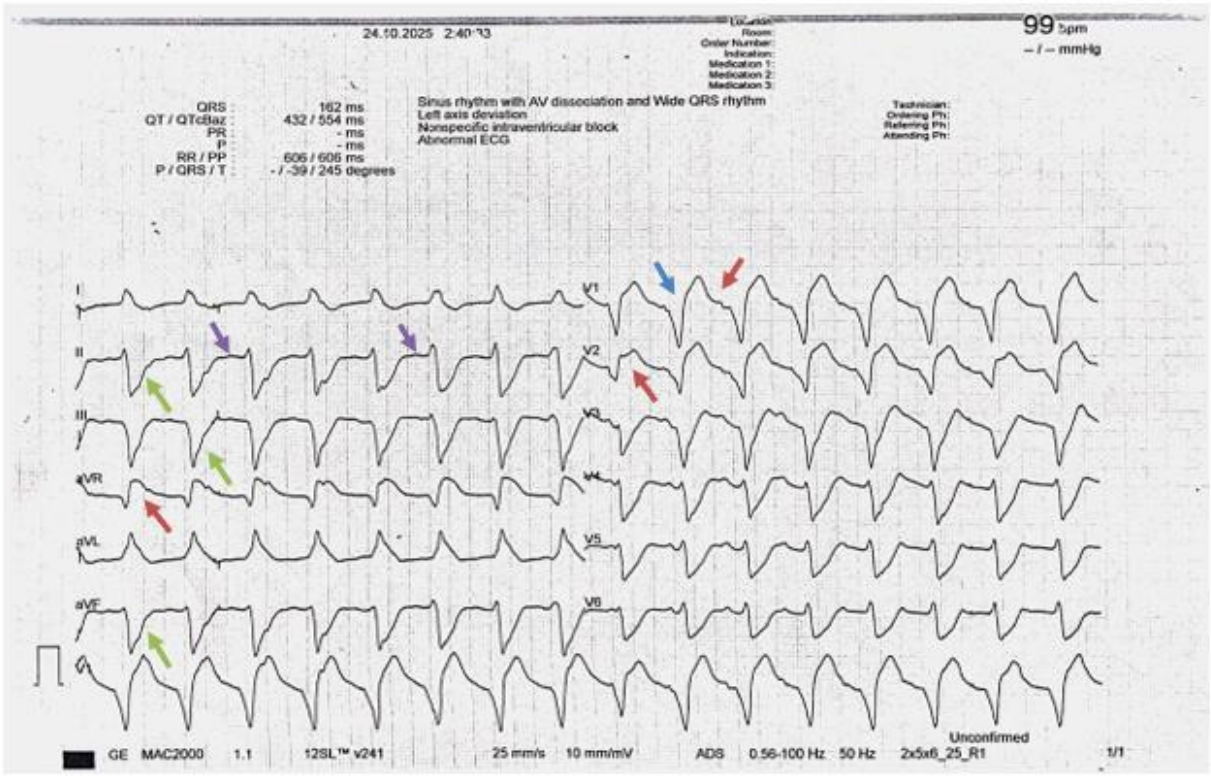
Fulminant myocarditis (FM) is a rare subgroup of acute myocarditis characterized by the sudden onset of severe, diffuse cardiac inflammation. This is a rapidly progressive syndrome that often leads to fatal outcomes due to cardiogenic shock, ventricular arrhythmias, or multi-organ system failure [1]. Clinically, it can be recognized by an acute symptom onset of less than two weeks and hemodynamic instability warranting inotropic therapy or temporary mechanical circulatory support (MCS) in the absence of other causes [2]. Although epidemiological data specific to FM is limited, it is estimated that between 5-10% of patients admitted with acute myocarditis present with a fulminant clinical picture [1]. Additionally, data suggests that the rate of FM is higher in males, and that higher mortality rates are observed in the younger population (<44 years of age) compared to those above 55 years of age [3]. The pathophysiology of acute myocarditis can be divided into three phases: 1) viral infection, 2) immune activation, and 3) myopathy. The first phase involves the reactivation of a dormant virus or new infection by a cardiotropic virus. The second phase is characterized by myocardial tissue damage resulting from viral replication and an unregulated immune response. The final phase occurs when the immune response is unable to effectively control the infection, leading to persistent inflammation and changes in the myocardium [1]. This is most frequently seen as myocardial necrosis, ventricular remodeling, and myocardial fibrosis [4]. Fulminant myocarditis can be further classified into distinct, pathological subtypes: lymphocytic myocarditis, eosinophilic myocarditis, and giant cell myocarditis. Lymphocytic myocarditis is the most frequently described subtype that is characterized by the infiltration of CD3+ T lymphocytes. It is most often precipitated by viral infection, toxin exposure, and autoimmunity. Eosinophilic myocarditis is a rarer subtype that is distinguished by the presence of eosinophils. Cases are often attributed to hypersensitivity reactions and parasitic infections. Giant cell myocarditis is characterized by the appearance of large, multinucleated cells. Although specific etiology is unknown, it is most often associated with autoimmune conditions [1]. Management of fulminant myocarditis requires a combination of medical therapy and mechanical circulatory

support. As it frequently presents as acute heart failure with progression to cardiogenic shock, initial management is aimed at achieving hemodynamic stabilization with inotropy. In the case of arrhythmias, the early use of temporary mechanical support (MCS) is necessitated [1]. Fulminant myocarditis is generally associated with worse outcomes as compared to non-fulminant myocarditis. The literature has identified higher rates of cardiac death and heart transplant at 60 days in FM cases [5]. Additionally, FM patients often present with lower left ventricular ejection fraction at follow-up [6].

Here, we report a case of a 29-year-old woman presenting with fulminant myocarditis.

Case presentation

A 29-year-old woman without prior, significant medical history presented to the Accident and Emergency Department with acute onset of hemodynamic instability preceded by five days of viral prodromal symptoms of cough, fever, sore throat, and headache. Three days before admission the patient developed gastrointestinal symptoms including nausea, diarrhea, and vomiting, followed by acute retrosternal chest pain lasting for 30 minutes on the morning of presentation. Furthermore, the patient experienced a syncopal episode at home approximately 24 hours prior to first visit. Following admission, a SARS-CoV-2 nasopharyngeal rapid test returned positive, consistent with SARS-CoV-2 infection. On presentation, the patient was hemodynamically unstable with a blood pressure of 75/45 mmHg, heart rate of 100 bpm, oxygen saturation of 95% and cool extremities. Upon initial presentation the patient was classified as Forrester Class C. The Forrester classification grades cardiogenic shock based on systemic perfusion and pulmonary congestion [7]. Class C is characterized as hypoperfusion in the absence of pulmonary congestion, often referred to as “cold and dry” shock [7]. The 12-lead electrocardiogram (ECG) initially demonstrated complex and vague findings that complicated the differential diagnosis. It revealed a wide-complex tachycardia at 100 bpm, with a broad QRS complex and undetermined basic rhythm as seen in Figure 1. Specifically, a left bundle branch block-like (LBBB) morphology was present along with atrioventricular (AV) dissociation. Changes were seen at the level of the ST-segments, particularly at the anteroventricular distribution (leads aVR, V1, and V2), along with reciprocal changes seen in the inferior leads (II, III, aVF). The P-wave was poorly visualized. The ECG pattern was highly suggestive of an acute left anterior descending (LAD) coronary artery STEMI. Following administration of intravenous amiodarone (50 mg over 5 minutes), the ventricular rate decreased from 100 to 50 bpm, with emergence of clearly visible P-waves dissociated from the QRS complexes, confirming underlying accelerated idioventricular rhythm (AIVR).



Blue arrow: LBBB morphology
Red arrow: ST-elevation
Green arrow: reciprocal inferior changes
Purple arrow: AV dissociation

Figure 1: ECG on admission

The initial bedside transthoracic echocardiogram (TTE) revealed findings highly suggestive of diffuse myocardial inflammation rather than focal ischemia. It demonstrated a mildly dilated left ventricle along with a severely reduced left ventricular ejection fraction (LVEF) of 20-25%. Moreover, the patient's TTE demonstrated wall motion abnormalities that did not follow a distinct coronary artery pattern. She had akinetic segments in the basal and mid-wall regions, with all remaining segments being hypokinetic. However, the patient had preserved apical and anterior

wall contraction. This finding is atypical for a LAD occlusion [7] and served as a diagnostic clue that helped to rule-out STEMI. The left ventricular outflow tract velocity time integral (LVOT VTI) was significantly reduced at 5.8 cm, indicating a reduced stroke volume consistent with cardiogenic shock. There was a mild degree of mitral and tricuspid regurgitation. The patient's right ventricular function was moderately reduced with a tricuspid annular plane systolic excursion (TAPSE) of 11mm, and no signs of pulmonary hypertension were observed. The pericardium was normal with no effusion noted. Serial troponin levels declined from 24 ng/L to 12 ng/L over 24 hours (reference range <14 ng/L). LDH, CK-MB, and CRP were elevated, consistent with myocardial injury and systemic inflammation. The combination of preserved apical function, diffuse wall motion abnormality, falling troponin levels, and positive rapid antigen test for COVID-19 strongly favored the diagnosis of fulminant myocarditis secondary to COVID-19 infection [8].

Initial fluid resuscitation involved administration of 500 mL of normal saline as a bolus to help restore intravascular volume. However, the patient failed to respond with only a slight improvement in her blood pressure from 75/45 to 90/60 mmHg. Given her persistent hypotension refractory to fluid resuscitation, the cardiology team initiated norepinephrine at a dose of 40 mg diluted in 250 mL of 5% dextrose at an infusion rate of 7-9 mL/hour. Despite this intervention, ongoing hypoperfusion and organ dysfunction were still present. Within 24 hours of admission, and despite receiving fluid resuscitation and pharmacological support, the patient deteriorated from Forrester Class C to Class D cardiogenic shock, which is defined as hypoperfusion with pulmonary congestion and multiorgan failure. This presented predominantly as acute kidney injury (AKI) with anuria, (urine output < 50ml/hour) along with refractory shock [7].

Despite ECG findings suggestive of LAD-territory STEMI, the cardiology team deferred immediate coronary angiography and instead pursued maximal medical therapy followed by mechanical support. They opted for this approach due to the patient's young age and absence of underlying cardiac disease [7]. Moreover, the inconsistency between the ECG rhythm, biomarker pattern, and the TTE findings (diffuse dysfunction with preserved apical and anterior function) strongly suggested infective myocarditis over the initial diagnosis of STEMI. Therefore, the deferral for urgent coronary angiography was justified. However, it was pursued at a later stage to definitively exclude any findings suggestive of ischemia at the level of the coronary arteries. By 24 hours post-admission, the patient was still in a state of refractory shock. At this point in the patient's management, mechanical support became essential. As the patient was deemed high-risk, angiography was coupled with Impella implantation (Figure 2A) to minimize complications. As anticipated, angiography revealed patent coronary arteries (Figures 2B and 2C).

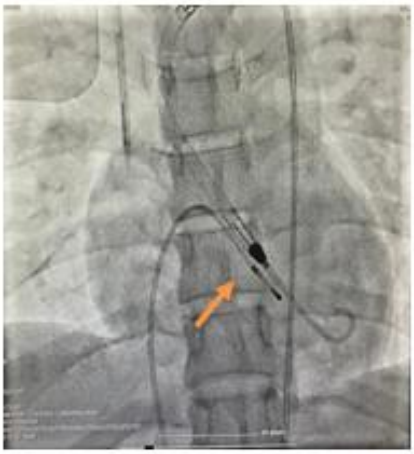


Figure 2A: Impella in position

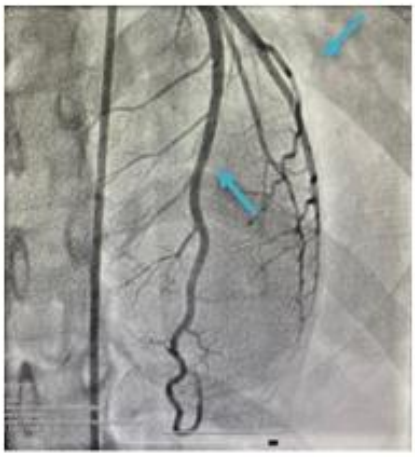


Figure 2B: LAD and LCx view

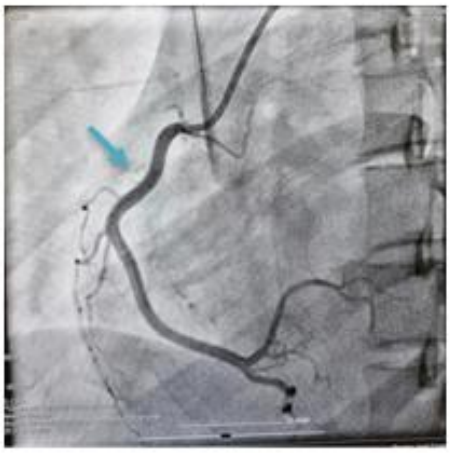


Figure 2C: RCA view

Orange arrow: Impella device inserted Blue arrow: patent coronary arteries

The Impella device was inserted via femoral artery access and provided support for five days. Repeat echocardiography demonstrated progressive recovery of ventricular function from 20–25% at presentation to 60–65% on day 5. Additionally, the ECG on day five showed resolution of ST-elevation and LBBB morphology with return to normal sinus rhythm. Hemodynamic stability was achieved with no further need for inotropic or vasopressor agents. Her stable condition justified Impella removal on day five, which was complicated by the development of right femoral artery dissection and subsequent limb ischemia. Immediate vascular surgery was scheduled to restore perfusion, with a successful final outcome.

Discussion

The underlying pathophysiological mechanism of acute myocarditis consists of infection by a virus, activation of multiple immune pathways, and subsequent myopathy. Commonly implicated viruses include parvovirus B19 and coxsackievirus, although recent research has identified a growing role for common respiratory viruses (i.e., coronaviruses) [1]. Viral-like symptoms typically appear several days prior to the onset of myocarditis. Immune activation is driven by virus-specific T lymphocytes generated by the host, which actively target and destroy myocardial tissue due to molecular mimicry [1]. This phase typically lasts from days to weeks. Dysregulated immune response and cytokine storm play key roles in the development of fulminant myocarditis. One study identified a significant upregulation in specific cytokines, such as IL-1b, IL-4, IL-17B, IL-23, and IL-10, among others [7]. This cytokine overproduction directly affects myocardial contraction and electrical transduction. Characteristic changes include ventricular wall hypokinesia, edema, and attenuated contractility [7]. In the case of myocarditis stemming from SARS-CoV-2 infection, it is proposed that direct injury to cardiomyocytes occurs due to binding of the virus to angiotensin converting enzyme 2 (ACE-2) receptors [8]. SARS-CoV-2 infection-related acute myocarditis has been characterized in the literature

as causing sudden-onset cardiac dysfunction leading to cardiogenic shock, as in our case, and fatal arrhythmias. Such cases have been reported since the viral outbreak in 2020, with some cases specifically reported post-vaccination. Fulminant myocarditis related to SARS-CoV-2 is rare, with only 108 cases reported during the peak of the pandemic between 2020 and 2022 [8]. The reported cases showed male predominance, with a male to female ratio of approximately 1.5 and a mean age of 35-years-old [8]. Majority of patients presented with a comparable clinical picture characterized by fever, dyspnea, cough, increased heart rate, low blood pressure, and reduced left ventricular ejection fraction. Moreover, it has been reported that COVID-19 infection can also precipitate STEMI. Various pathophysiological mechanisms have been implicated, including inflammatory, thrombotic, and endothelial processes that augment plaque vulnerability and interfere with coronary flow [11]. This state of increased inflammation triggers cytokine release, resulting in a hypercoagulable state through the upregulation of tissue factors and activated platelets [11]. The hypercoagulable state increases the burden of thrombus formation and subsequent thrombus mobilization during PCI intervention [11]. Viral invasion of endothelial cells through ACE-2 receptors disrupts the renin-angiotensin system, increasing the endothelial dysfunction and risk of microvascular thrombosis. This can destabilize pre-existing atherosclerotic plaques and increase the probability of developing a type I MI [12,13]. This endothelial injury lingers beyond acute infection, with data demonstrating an approximate two-fold increase in MI incidence in the months following COVID-19 infection [12]. During the initial outbreak, cases of COVID-related STEMI resulted in increased rates of cardiogenic shock and mortality compared to non-infectious counterparts [14,15]. Some works of literature attribute these poorer outcomes to the delayed interval between initial symptoms and medical attention in COVID-19 related STEMI [14]. ECG is the diagnostic tool of choice for identifying STEMI in acute emergency settings. It provides a rapid and non-invasive assessment of the heart's electrical activity. In most cases, the ECG demonstrates high specificity for STEMI when showcasing ST- segment elevation in contiguous leads. However, sensitivity is more limited which can lead to the underdiagnosis of true STEMI episodes. Currently, the standard ECG has limited sensitivity of 30–70% and specificity of 70–100% [10]. In terms of FM, abnormalities seen on ECG include nonspecific ST-segment changes, and conduction disturbances [9]. As such, ECG accuracy is highest when interpreted alongside clinical presentation and compared with previous recordings, if available. It is important to note that fulminant myocarditis can mimic STEMI electrocardiographically without underlying coronary pathology, as demonstrated in our case. This generates diagnostic uncertainty as the ECG changes could refer to a true coronary occlusion or global myocardial inflammation producing a STEMI-like image [16]. For clarity, our patient received continuous ECG monitoring to aid in revealing any new dynamic ischemic changes suggestive of a myocardial infarction or point to an alternative diagnosis. To definitively exclude STEMI, the changes observed on ECG ought to be interpreted alongside cardiac biomarkers, echocardiography, and coronary angiography. Our case therefore emphasizes the importance of evaluating electrocardiographic trace in the context of other findings to better assess the likelihood of an ACS or non-ACS presentation. The ECG presentation of this case was misleading in the first instance, which impeded the final diagnosis of fulminant myocarditis. It is generally recognized that the ST-segment elevation associated with ACS is also present in myocarditis, therefore limiting the specificity of this finding [7,17]. ECG abnormalities characterized in myocarditis broadly include ST-segment elevation, T-wave inversion, QRS prolongation, AV block, along with various forms of arrhythmias [17]. Our patient's specific ST-segment elevation distribution, along with a newly detected LBBB, would typically prompt consideration of STEMI involving the LAD. Despite her ECG trace being strongly suggestive of ACS, subsequent TTE provided diagnostic clarity. In particular, the pattern of wall motion abnormality proved critical in terms of differential diagnosis. In acute cases of LAD occlusion, regional wall motion abnormality is expected to follow a coronary distribution consistent with the LAD territory, namely the anterior wall, anteroseptal wall, and apex [19]. The discrepancy between the ECG pattern (suggesting LAD involvement) and the TTE findings seen with our patient (showing preserved apex and anterior wall with basal dysfunction) was incompatible with ACS and coronary ischemia, instead suggesting diffuse myocardial inflammation characteristic of a myocarditis picture [18]. This case exemplifies the importance of the "ECG-echo mismatch" as part of the diagnostic pathway when aiming to differentiate true episodes of STEMI from mimics. The decision to defer immediate coronary angiography and instead opt for maximal medical optimization followed by catheterization was deliberate. This approach diverges from standard STEMI/ACS protocols which require immediate revascularization. However, this was justified by clinical considerations specific to myocarditis. This was the case for our patient who was suspected of having acute myocarditis complicated by hemodynamic instability. In the setting of cardiogenic shock with reduced systemic perfusion and anuria, renal clearance of iodinated contrast is markedly impaired, increasing the risk of contrast-induced nephrotoxicity through oxidative stress, vasoconstriction, and ischemic tubular injury [20,21]. Additionally, low cardiac output promotes venous stasis, leading to contrast pooling and layering on CT imaging [22]. These findings act as a marker for cardiogenic shock, which should be promptly recognized and treated to prevent further decompensation. Therefore, the use of contrast media solution should be carefully weighed due to the high risk of worsening renal injury and contrast retention. This was taken into consideration in the context of our patient who presented with cardiogenic shock and AKI. Evidently, performing coronary angiography in a critically ill patient with refractory cardiogenic shock comes with several complications and risks. The use of radiographic contrast is known to carry the potential to exacerbate AKI and heart failure. Moreover, all clinical interventions should revolve around the Hippocratic decree of "do no harm". Deferring angiography until it was safe to do so and prioritizing the patient's hemodynamic status allowed the cardiology team to uphold this pillar. Our case represents a complex "risk-benefit" scenario, demonstrating the importance of both timing and hemodynamic optimization in an acute clinical setting such as fulminant myocarditis. Initial supportive management is strongly advised in FM, which includes mechanical ventilation, inotropic agents, and vasopressors to correct hypotension or respiratory failure, and overt cardiogenic shock. Inotropic therapy is essential to achieve hemodynamic stability in shocked patients. Dobutamine is the most frequently used agent, followed by epinephrine and norepinephrine. However, high doses of inotropic agents in adult patients are avoided to prevent precipitating tachyarrhythmias [23]. Additional pharmacotherapy may involve immunosuppressive agents, particularly in patients presenting with eosinophilic and giant cell subtypes. If appropriate, they may be initiated once active viral infection is excluded on endomyocardial biopsy by PCR [1]. Current recommendations are to administer high-dose intravenous corticosteroids (1g methylprednisolone/daily) for a minimum of three days [23]. Plasmapheresis may also be used depending on the etiology of fulminant myocarditis. In critically ill patients with low cardiac output and reduced LVEF, it is recommended to consider mechanical circulatory support (MCS) devices for cardiorespiratory support. The purpose of temporary MCS in FM is to intervene prior to the development of multi-organ failure to reduce mortality. The benefits of MCS devices include reduced cardiac

workload, improved coronary perfusion, immune response modulation, and creating an optimized environment for recovery [23]. Device selection should be tailored according to patient factors, severity of cardiogenic shock, and organ function. Extracorporeal life support, most often venoarterial extracorporeal membrane oxygenator (VA-ECMO), is considered one of the most efficient means of providing complete hemodynamic support in FM patients in the short-term. However, the consequential increase in afterload and left ventricular distention from VA-ECMO may require additional devices, such as an intra-aortic balloon pump, to support left ventricular unloading [23]. Combined ECMELLA therapy, consisting of VA-ECMO and Impella, is associated with reduced mortality as compared to VA-ECMO alone [23]. The Impella device is a percutaneous, catheter-mounted axial flow pump that directly unloads the left ventricle by aspirating blood from the left ventricular cavity and delivering it to the ascending aorta [7]. As such, it provides temporary MCS that directly assists the heart with its pumping function and maintains systemic perfusion to all organs. It is guided via fluoroscopy and inserted at the level of the aortic valve in the left ventricle through the femoral artery. The Impella device helps promote hemodynamic stability in patients experiencing cardiogenic shock, with severe coronary artery disease or during high-risk PCI [24]. Moreover, early Impella placement is associated with improved outcomes by reducing left ventricle myocardial oxygen consumption, end-diastolic compliance, and pulmonary capillary wedge pressure [25]. This rationale was applied in the case of our shocked patient who underwent Impella implantation during coronary angiography. When used as a bridge-to-recovery, duration of MCS is typically between 7-10 days due to the self-limited nature of FM. It is also important to note that Impella use has been associated with vascular complications such as bleeding. Our patient experienced access-site complication following removal of the Impella device. She developed a femoral artery dissection with thrombosis that required immediate surgical intervention. Several factors were reported to have contributed to the increased risk of femoral bleeding, such as the use of large-bore sheaths, increasing age, female sex, and antiplatelet drugs. Other complications include access site-related infection, ischemic and hemorrhagic stroke, and myocardial infarction [24]. Additionally, implantable (ICD) and wearable (WCD) cardioverter-defibrillators may also play a role in secondary prevention following the development of arrhythmias. Current recommendations support the use of ICD in patients with non-active myocarditis for sustained tachyarrhythmias to prevent sudden cardiac death (SCD) [26]. Moreover, recent data regarding the use of WCD devices suggests that myocarditis patients at risk of developing ventricular tachyarrhythmia may benefit from their use, particularly in the setting of a left ventricular ejection fraction of less than 35% or prior occurrence of tachyarrhythmia [27]. Once patients with FM recover from cardiogenic shock and are hemodynamically stable, it is advised to start treatment for heart failure [9]. Pharmacological therapy includes beta-blockers, angiotensin-receptor/neprilysin inhibitors (ARNI), mineralocorticoid receptor antagonists (MRAs), SGLT2 inhibitors and diuretics [28]. In addition, continued immunosuppressive therapy may be considered for certain subtypes of myocarditis such as eosinophilic and giant cell myocarditis, particularly in the setting of systemic autoimmune disease [9]. Despite the severe clinical course of FM, a high long-term survival rate is reported with early, aggressive, and adequate treatment. Most patients regain near-complete or complete recovery of the left ventricular function within weeks or months. Moreover, recurrence of FM is uncommon, including COVID-19-associated cases. Some literature suggests that there is an 84% lower risk of reinfection in the next seven months following the primary infection [29]. Other studies reveal that a subset of patients who developed myocarditis as a result of SARS-CoV-2 infection may be more prone to developing a second episode after receiving a dose of COVID-19 vaccination [29]. When comparing the prognosis of acute and fulminant myocarditis, one study found that over 20% of FM patients developed heart failure, ventricular dilatation or arrhythmias as compared to approximately 10% of acute myocarditis patients [7].

Limitations

Being a single case report, the observations described may not be generalizable to a wider patient population. Additionally, the diagnosis of fulminant myocarditis following COVID-19 infection was based on clinical presentation, ECG and TTE findings, and biomarker trends. It is important to note that endomyocardial biopsy, which is the gold standard for definitive diagnosis, was not performed in this case. Finally, follow-up data was limited, restricting evaluation of the patient's long-term cardiac recovery and clinical outcomes.

Conclusion

The rapid clinical course of FM requires early recognition and precise interventions to optimize patient outcomes. A high index of suspicion for FM is warranted in young patients presenting with a clinical picture of acute heart failure unresponsive to initial resuscitation, as in our case. Pursuing the incorrect treatment pathway, such as for ACS, may cause unnecessary delays and significantly worsen prognosis. In terms of our patient, discrepancies across investigations enabled the medical team to promptly revise the likely diagnosis and adjust the management approach accordingly. Moreover, the correct timing of each intervention is crucial in minimizing iatrogenic errors and safeguarding patients whilst they undergo the diagnostic pathway. Additionally, the use of MCS devices, like Impella, play a key role in supporting shocked FM patients. This rationale was applied in the management of our patient, who underwent delayed coronary angiography supported by Impella implantation. This case reflects the importance of differentiating similar medical presentations and using clinical reasoning to identify if and when to switch protocols, bearing in mind patient safety.

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