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# Diagnosis and management of patients with fulminant myocarditis

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Fulminant myocarditis (FM) is a progressive and severe form of acute myocarditis, complicated by cardiogenic shock. The clinical presentation and aetiologies of FM are often heterogeneous, making it difficult to diagnose. Irrespective of how FM presents, it rapidly evolves to haemodynamic deterioration and requires prompt treatment to stabilize. As such, the use of mechanical circulatory support (MCS) devices has emerged as a critical intervention to achieve haemodynamic support, early unloading, and systemic perfusion and to prevent multiorgan dysfunction in patients with FM. Although scientific societies have proposed recommendations and management pathways, due to the heterogeneity in FM, there remains a lack of clarity in the diagnostic pathway and selection of MCS device for this young patient population. This review provides an updated and integrated overview of the diagnostic flow and important clinical considerations when managing patients with FM.

## Introduction

Myocarditis is an inflammatory condition of the heart muscle, which can vary widely in severity. Myocardial inflammation can result in impaired cardiac function, leading to heart failure, arrhythmias, and even sudden cardiac death in severe cases. Fulminant myocarditis (FM) is a rapidly progressive and severe form of acute myocarditis (AM), complicated by cardiogenic shock, requiring inotropes and/or mechanical circulatory support (MCS).[1,2] As such, it is usually a single acute event, with recurrent episodes a rare occurrence. The aetiology of FM can be secondary to infections (mainly viral infections), systemic autoimmune disorders, medications, and vaccines. Independent of the initial trigger, the subsequent inflammatory response is responsible for myocardial injury, causing myocyte necrosis, interstitial oedema, and inflammatory infiltrates.[3] The use of MCS support devices has emerged as a critical intervention in managing patients with FM.

## Diagnostic flow chart

Myocardial inflammatory diseases have heterogeneous clinical presentations and aetiologies making it sometimes challenging to diagnose. The 2013 European Society of Cardiology (ESC) expert position paper defined criteria for diagnosis of clinically suspected myocarditis based on clinical presentation and non-invasive

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diagnostic techniques.[4] Most often, FM presents as a rapidly evolving haemodynamic deterioration after the initial classic presentation of AM. Based on large registries, the most frequent symptom in patients with AM is chest pain (85–95% of cases), followed by dyspnoea (19–49% of cases), whereas syncope occurs in a minority of patients (~5%). In more than 50% of patients, cardiovascular symptoms are associated with fever or other infectious prodromal manifestations.[2] Abnormal electrocardiogram findings are present in >60% of patients with AM as ST-elevation (ST-elevation myocardial infarction-like presentation), ST-depression, or T-wave inversion, particularly involving inferior and lateral leads.[5] Myocardial inflammation may be reflected in echocardiographic changes such as wall thickening, granular echogenicity, regional hypokinesia (typically involving inferior and lateral segments), and reduced global longitudinal strain.[6] Most patients with AM (~75%) have normal global ejection fraction at presentation but may rapidly evolve towards systolic dysfunction in the first few days.[5] Furthermore, pericardial effusion may be present. High-risk features at presentation are left ventricular (LV) dysfunction (ejection fraction < 40%), ventricular arrhythmias or atrioventricular blocks, and cardiogenic shock. A recent retrospective registry showed that 26.6% of patients with AM had complicated presentation with LV systolic dysfunction, arrhythmic pattern, or cardiogenic shock accounting for 3–9% of total cases.[7]

Irrespective of FM presentation, it is fundamental to stratify the risk level of the patient to guide the diagnostic pathway and prompt appropriate treatment. Circulating levels of high-sensitivity troponin can be used as a biomarker for myocardial injury, and can be elevated to 64–100% above the levels found in a healthy individual. However, this is largely nonspecific and is indicative of different cardiac and non-cardiac conditions. A more specific marker, N-terminal pro-brain natriuretic peptide, is often elevated in AM in response to increased stretching of the heart muscle and is an independent predictor of poor outcomes.[8] Other common laboratory tests utilized for AM diagnosis include C-reactive protein.[2,5] An elevation in erythrocyte sedimentation rate suggests an associated autoimmune disorder, and the presence of eosinophils can suggest the presence of eosinophilic myocarditis.[9] Peripheral blood serological and virological tests are rarely informative and are thus not routinely indicated in AM diagnostic pathway. Exceptions are represented by settings like HIV, the presence of *Borrelia burgdorferi* antibodies, and positivity for respiratory viral genomes that can trigger AM. Additionally, influenza and coronaviruses can trigger immune-mediated lymphocytic myocarditis. In these clinical cases, a myocardial viral genome blood test and pharyngeal swabs are recommended, although its clinical and therapeutic implications remain controversial.[2,10] Autoantibodies and other immunological tests may be indicated in patients with known or possible autoimmune disorders as well.[11]

## Cardiac magnetic resonance

Cardiac magnetic resonance (CMR) is currently the gold standard non-invasive diagnostic technique for myocarditis in patients presenting with uncomplicated suspected AM. However, in critical patients, particularly patients supported by MCS, CMR should be utilized following haemodynamic stabilization to assess the presence, extent, and localization of residual inflammation and replacement fibrosis. As such, CMR allows for the recognition and quantification of inflammation and replacement fibrosis of myocardial tissue. The diagnosis of myocarditis by CMR has relied on the Lake Louis Criteria (LLC) since 2009, which refers to three hallmarks of myocardial inflammation with corresponding CMR markers and includes hyperaemia, tissue oedema, and necrosis/fibrosis.[12] The LLC have been updated recently to include MRI mapping techniques to provide quantitative characterization of myocardial tissue and increase the overall diagnostic accuracy.[13,14]

## Endomyocardial biopsy

Non-invasive imaging techniques, particularly CMR, are increasingly used in the diagnosis and monitoring of myocarditis.[7] However, endomyocardial biopsy (EMB) remains the gold standard for confirming cardiac inflammation, especially in cases of new-onset heart failure where there is suspicion of infective and autoimmune diseases. Current guidelines from the ESC Working Group on Myocardial and Pericardial disease recommend EMB in patients with suspected myocarditis,[1,15] while the AHA guidelines recommend EMB in cases of suspected FM (Class I, level of evidence B). International recommendations state EMB should be performed in patients with suspected myocarditis with new-onset heart failure or cardiogenic shock requiring inotropic or MCS device support, in cases of unexplained heart failure accompanied by ventricular arrhythmias or high-degree atrioventricular block (e.g. Mobitz 2 or higher), or if there is no response to standard medical management within 1–2 weeks.[15-17]

In suspected myocarditis with cardiogenic shock, EMB carries a complication rate of 1–5% and requires careful consideration of patient stability, operator experience, and clinical setting, with elevated risks in critically ill patients, particularly those on anticoagulation due to MCS.[7,18-21] Patients with severe presentations [Society for Cardiovascular Angiography & Interventions (SCAI) E or SCAI C-D] may require early intervention with MCS, including veno-arterial extracorporeal membrane oxygenation (VA-ECMO) or Impella, and EMB may be performed to establish aetiology, especially when coronary angiography or Impella implantation is conducted in the catheterization lab. For patients supported by VA-ECMO, a combined fluoroscopic and echocardiographic-guided approach is recommended for right ventricular EMB, with a preference for septal sampling to reduce risks of complications such as tamponade.

If haemodynamic recovery occurs, weaning from MCS and transitioning to medical therapy is prioritized, with follow-up CMR to assess disease progression and recovery. For patients without recovery after 7 days of MCS, or those not improving, EMB is advised to determine underlying pathology, as it can inform potential escalation to pulse steroid therapy or consideration for heart transplantation listing if needed. Specific histological findings, such as inflammatory infiltrates, can help classify myocarditis subtypes, which

may have prognostic and therapeutic implications.[19,22,23] For instance, acute necrotizing eosinophilic myocarditis is a rare, often fatal form of myocarditis presenting with acute heart failure or sudden cardiac arrest.[12] Patients frequently require prolonged mechanical support and have high mortality rates or need for LV assist device or heart transplantation (up to 50%).[13]

Advanced atrioventricular block can suggest specific aetiologies, such as cardiac sarcoidosis, giant cell myocarditis, Lyme carditis, or immune checkpoint inhibitor (ICI)-associated myocarditis. The presence of viral genomes, as recommended by the 2013 ESC position statement,[1] can also guide treatment strategies. For instance, the detection of cytomegalovirus in immune-suppressed patients, enterovirus in infants (where immunosuppression is not recommended), or Parvovirus B19 (although of less clear significance) can influence management.[14,24]

Safety considerations for EMB are essential given the increased risk of bleeding and cardiac tamponade, particularly in patients receiving anticoagulation with MCS.[18-21,25] The CHANGE PUMP-2 study identified predictors or poor outcomes at 90 days, including the need for VA-ECMO [hazard ratio (HR) 7.73], ventricular arrhythmias on admission (HR 3.40), and the absence of EMB (HR 1.76), indicating the potential importance of EMB in guiding therapy and improving outcomes. The optimal timing and methodology for EMB in suspected FM cases on temporary MCS remain under investigation.[26] Most data suggest that EMB be performed in patients who have stabilized,[14] to balance the risk/benefit of the procedure. When performed, CMR and/or electro-anatomically guided EBM should be considered as they yield higher sensitivity and specificity as compared with blind biopsy. Timing for EMB throughout FM management is summarized in *Figure 1*.

## Medical therapy

Fulminant myocarditis is a reversible disease, with the aim of clinicians to achieve haemodynamic support, early unloading, systemic perfusion, and prevention of multiorgan dysfunction. Patients with haemodynamically unstable heart failure should be referred to tertiary centre intensive care units, experienced with MCS, EMB, and heart transplant (HTx) when possible.[2] Patients with FM require vasoactive drug support but there is no defined recommendation on the choice of inotropic agents. International registry data suggest that dobutamine was the most frequently used (58.7%), followed by epinephrine and norepinephrine (43.0% and 41.2%, respectively).[22] A general recommendation for adult patients is to not use high doses of inotropes and to favour early MCS to prevent the risk of inotrope-induced arrhythmias in patients already at risk.[25,27]

In patients with giant cell, eosinophilic and, less frequently, sarcoidosis-associated myocarditis, FM intravenous corticosteroids are indicated. Immediate administration of high doses of steroids (1 g methylprednisolone/daily for at least 3 days), even before EMB, should be considered when immune-mediated form of FM is strongly suspected.[10] Other immunosuppressive agents are often needed in addition. Giant cell myocarditis is reported to respond to cyclosporine-based immunosuppressive therapy, suggesting an autoimmune etiopathogenesis.[28]

In instances of myocarditis due to the use of ICI drugs, the standard approach is cessation of ICI and high corticosteroid therapy (intravenous methylprednisolone 1 g/24 h) within 24 h of diagnosis. While not all ICI-related myocarditis cases are FM requiring high steroid dose, there are low grade forms that can safely continue ICI, often necessary from the oncologic perspective. Recent studies also evaluated other immunosuppressive therapies such as abatacept, a CTLA-4 immunoglobulin fusion protein, to use in addition to corticosteroids.[29] As such, maintenance of immune suppression should be based on the results of EMB and is typically useful in cases of eosinophilic myocarditis, giant cell myocarditis, and sarcoidosis, or in cases of systemic autoimmune disorders. Lastly, there are currently no antiviral therapies with approved efficacy.[1,30]

## Mechanical circulatory support

Myocarditis predominantly affects young males, which is a much different patient demographic from those who typically require MCS, as atherosclerosis is uncommon in younger patients.[31] However, the absence of atherosclerosis does not imply that vascular access is less challenging. An advantage of a younger patient population is that vessels tend to be more elastic and allow for the insertion of devices with diameters that may exceed the measured diameter of the vessel. As such, patients suffering from myocarditis should ideally have a groin-free access strategy to facilitate early mobilization, with the axillary artery being the most favourable access point.

Ventricular unloading via MCS devices can play a crucial role in mitigating the adverse effects of viral injury and autoimmune responses in myocarditis. The benefits of ventricular unloading include the following:

(1) Decreased workload of the heart: Ventricular unloading reduces myocardial stress and oxygen demand, which can limit further myocyte damage caused by the inflammatory response and preserve myocardial function.
(2) Improved coronary perfusion: Devices like microaxial flow pumps enhance coronary blood flow, ensuring better oxygen and nutrient delivery to the myocardium, which aids in the repair of damaged myocardial tissue, and reduces the extent of injury.
(3) Modulation of the immune response: Reducing myocardial stress and improving perfusion can indirectly modulate the immune response by decreasing the levels of stress-related inflammatory cytokines, mitigating the autoimmune component of myocarditis, and preventing further myocardial damage.
(4) Enhanced recovery environment: Ventricular unloading creates a more favourable environment for myocardial recovery by minimizing further injury and providing the heart with a chance to heal. This can be particularly beneficial in the context of viral myocarditis, where ongoing viral replication and immune-mediated injury are common.

The choice of which MCS strategy to pursue is not univocal. There is no consensus on device selection in FM and the decision should be patient tailored, based on the severity of cardiogenic shock, organ function, and centre

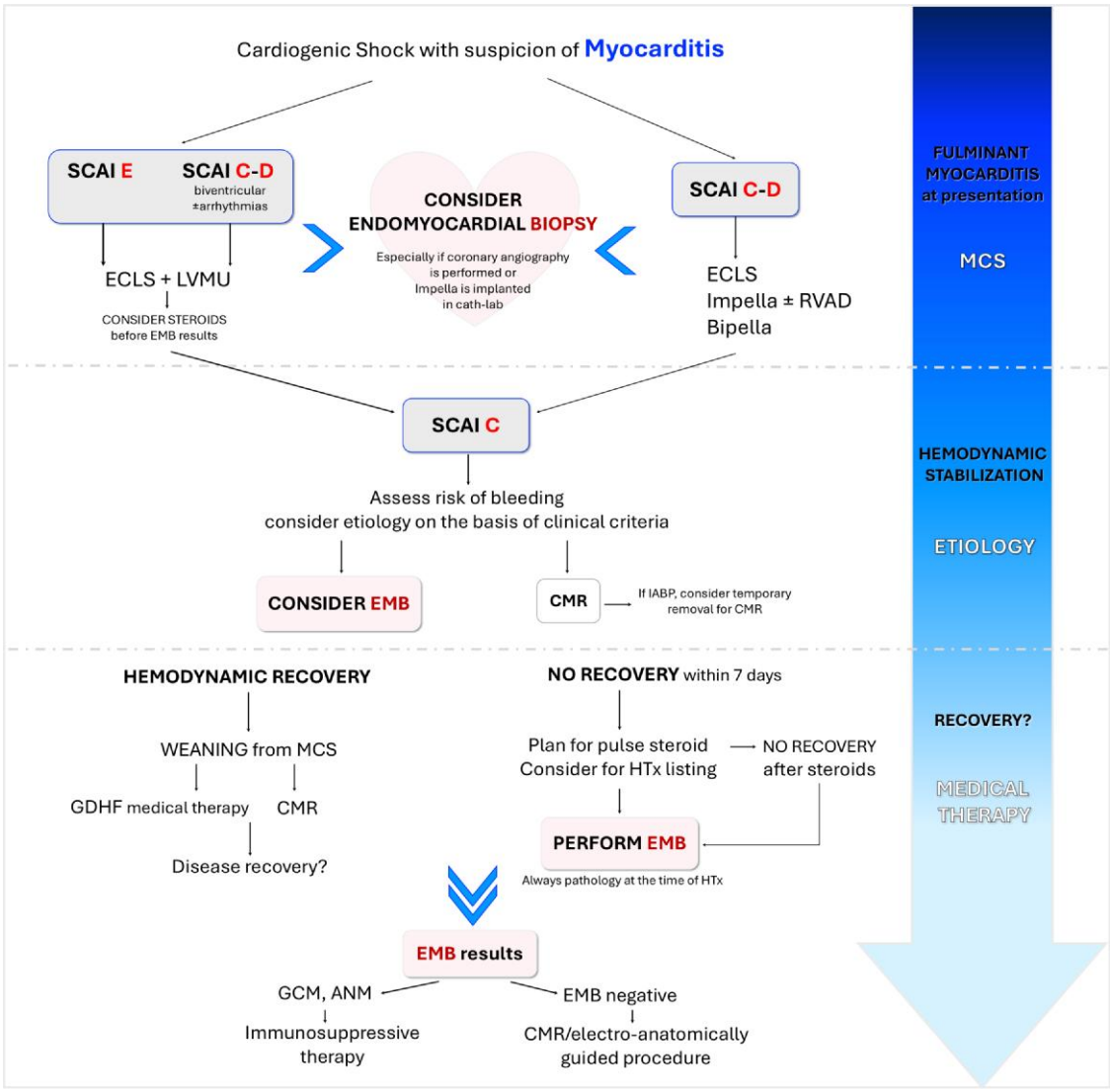


**Figure 1** Timing for endomyocardial biopsy throughout fulminant myocarditis management. ANM, acute necrotizing myocarditis; Cath-lab, catheterization laboratory; CMR, cardiac magnetic resonance; ECLS, extracorporeal life support; EMB, endomyocardial biopsy; GCM, giant cell myocarditis; GDHF, guideline-directed heart failure; HTx, heart transplant; LVMU, left ventricle mechanical unloading; MCS, mechanical circulatory support; RVAD, right ventricular assisted device; SCAI, Society for Cardiovascular Angiography & Interventions.

experience. The most frequently used short-term MCS in FM patients is VA-ECMO. However, during VA-ECMO support, LV distention and increased afterload may cause pulmonary oedema and hinder myocardial recovery. An early and effective unloading strategy should be associated. The combined ECMELLA (VA-ECMO + Impella) provides potent haemodynamic support, oxygenation, and ventricular unloading and is associated with lower mortality compared to VA-ECMO alone, despite higher complication rates. The combination in ECMELLA or BIPELLA should be considered when the right ventricular function is compromised.[31,32]

## Impella devices vs. extracorporeal membrane oxygenation in patients with fulminant myocarditis

To date, survival outcomes in patients with FM supported with short-term VA-ECMO range from ~40% to 88% at discharge.[9,11-17,22,23,32] Among the FM cases complicated by cardiogenic shock, survival rates were generally higher compared with other causes of cardiogenic shock, which may be attributable to differences in age, comorbidities, and the reversible nature of a myocarditis.[9] As such, the use of MCS devices in complicated myocarditis largely varies from site to site and is heavily influenced by local expertise and resources.[8,19] However, there is a growing number of sites that aim for LV unloading in FM cases and deploy Impella devices as a bridge-to-recovery or bridge-to-permanent ventricular-assisted devices (VADs) or HTx.[18-21,25,26,33] This is reflected by the increasing number of case series and cohort studies describing the management and outcomes of short-term use of the Impella in FM and giant cell myocarditis.[2,3,10,18,19,21,25-31,34-38] Thus, while myocarditis resolution remains incompletely understood, it has been proposed that LV unloading may suppress myocardial

infiltration by inflammatory cells and consequently enhances cardiac recovery.[11,38,39] In addition, the increased systemic and coronary blood flow achieved by Impella support could further facilitate cardiac recovery.[10,39]

Patients with FM receiving short-term support with Impella devices are usually younger and more often female compared with most patients in cardiogenic shock requiring Impella support, and the duration of support ranges from 7 to 16 days.[10,27,30,31,35,38] Moreover, the Impella CP device has been used (>80%) in the majority of cases. A previous retrospective multicentre study, in which 34 patients with FM were managed with Impella devices, had a survival rate of 62% underscoring the safety and effectiveness of this MCS device.[31] In addition, a recent analysis from the Japanese Registry for Percutaneous Ventricular Assist Devices assessed outcomes in FM cases managed with an Impella device and highlighted an overall survival rate of 74.3% at 30 days.[40] More specifically, survival was 68.5% in the ECMELLA cohort and 83.2% in the Impella-alone cohort. Nonetheless, this study also indicated a high rate of complication event rate (48.7%), specifically bleeding events (32.0%) and acute renal failure (8.6%).[40]

Due to heterogeneity of patients with myocarditis, the variation among applied Impella devices, and the lack of controlled studies, it is challenging to predict cardiac recovery with Impella support and provide evidence-based recommendations on the best timing for weaning or durable VAD or cardiac transplantation.[41] Overall, the reported rate of cardiac recovery and rate of successful weaning from the Impella devices differs largely and depends on the study cohort.[41] Early studies including mostly adult patients receiving VA-ECMO and VADs observed transplant-free survival rates to discharge of ~50%.[42,43] More recent observational data indicated transplant-free and survival rates ranging from 60% to 90%,[10,22,31,35,38] with the underlying age, comorbidities, severity of organ failure, site experience, and resources play a key role. Moreover, given that patients with FM may require prolonged Impella support to achieve adequate recovery, the risk of bleeding, haemolysis, and coagulopathy is subsequently higher.[42,43] Renal failure remains another complication, which can be further aggravated by low cardiac output and haemolysis.[42] Infectious complications (e.g. access-related) have also been a concern.[44] This risk might be additionally elevated among specific myocarditis cases requiring high doses of steroids or other immunosuppressant regimens.

To date, only observational studies have been published assessing outcomes of FM cases managed with VA-ECMO vs. ventricular unloading with Impella devices. More specifically, patients supported with Impella had less bleeding and vascular complications compared with patients supported with VA-ECMO.[45] As such, it is well established that side effects of VA-ECMO, irrespective of the presentation and implantation mode, require more resources and are more prone to complications (especially vascular and bleeding adverse events).[32] Additionally, VA-ECMO increases the afterload, which potentially aggravates LV dysfunction and increases the risk for respiratory decompensation due to pulmonary oedema, even within few hours after installation.[33,46] Collectively, and considering the recent DanGer-Shock trial,[47] we advocate for early consideration of ventricular unloading involving Impella devices in myocarditis cases with haemodynamic deterioration. This recommendation also accounts for the potentially lower bleeding and vascular complication risks with Impella devices. Similarly, in patients with myocarditis where a VA-ECMO is initially deployed or needed, the early introduction of an Impella device for LV unloading might be associated with a lower mortality risk according to recent data from a large multicentre cohort study.[33,46]

## PROPELLA

The strategy of prolonged Impella support, also termed PROPELLA, in FM is a new potential mode of action based on prolonged LV unloading via axillary implanted axial flow pump (Impella).[38] From a pathophysiological point of view, haemodynamic load is associated with the activation of cardiac mechanotransduction pathways, able to trigger inflammatory reactions.[39] As discussed above, VA-ECMO is known to increase the afterload of the LV, leading to consequent LV distension, myocardial wall stress, and activation of inflammation, thereby worsening the primitive inflammation damage and promoting unfavourable cardiac remodeling.[39] In contrast, adequate LV unloading may decrease cardiac wall stress and subsequently mitigate inflammatory response. In this context PROPELLA may provide benefits beyond its primary function of mechanical support. Moreover, it has been described that mRNA expression of innate immunity members, known to be up-regulated in myocarditis,[48] decreases during MCS and immunosuppressive therapy. This effect was abrogated after the removal of Impella support despite the continuation of immunotherapy, suggesting a primary unloading-dependent mechanism.[38]

## BIPELLA

Until recently, VA-ECMO was the only minimally invasive option to achieve biventricular support. Conversely, the Impella heart pumps unload the ventricle and require minimal anticoagulation. Few cases have been reported on the use of BIPELLA, which includes the use of Impella CP on the left and Impella RP on the right, for acute biventricular failure. In the first described case report of the BIPELLA approach,[33] the patient had suspected AM and myocardial recovery was reached. As such, this strategy mitigates shortcomings of ECMO while providing percutaneous biventricular unloading and haemodynamic support, thereby providing a potential solution in patients with acute biventricular failure.

## Device escalation in deteriorating myocarditis cases

There are several factors that must be considered when deciding if an MCS device escalation is necessary in the course of an FM case. First is the age of the patient. Certainly, physicians may be more reluctant to further escalate device therapy among patients above the transplant or ventricular assist device age criteria. On the other hand, patients with non-sinus rhythm, ventricular tachycardias, fibrillation, or established biventricular failure with compromised haemodynamics may require early consideration for advanced

haemodynamic support. In such scenarios, a surgically placed Impella 5.5 with or without VA-ECMO may represent a valuable option. In cases with respiratory failure, VA-ECMO should be evaluated. Among patients with concomitant RV failure, without respiratory failure, the Impella RP or ProtekDuo cannula could also be applied, although reports supporting those devices in myocarditis cases are lacking. When deciding upon MCS strategies, physicians should also take into account the EMB findings when available. In cases with severe histologic damage, such as damaged cardiomyocytes comprising ≥50% of the total cardiomyocytes, the prognosis appears worse and prolonged support can be expected. Of note, the requirement of MCS for more than 14 days was generally associated with worse prognosis and a higher likelihood of unsuccessful weaning.[4,16,32,49] In patients with fulminant giant cell myocarditis and an evident cardiac failure accompanied by haemodynamic deterioration, prognosis is often very poor with <20% survival at 5 years.[33,34,38,39,50] However, the early initiation of immunosuppressive therapy may reverse the course of disease.[34] Bridging the compromised ventricular and haemodynamic status using Impella devices may buy the patient the required time to halt and reverse the cardiac inflammation with immunosuppressive therapy.[10,38,39,51] Since this recovery process usually takes time, physicians may need to consider surgical access routes and Impella 5.5 devices. Nonetheless, giant cell myocarditis is often characterized by an involvement of both ventricles, and biventricular MCS seems more often required when compared to patients with myocarditis or cardiomyopathies attributable to other aetiologies.[10,43,51]

## Clinical outcomes and considerations

The prognosis of patients with AM accompanied by mild-to-moderate heart failure and LV systolic dysfunction is generally benign with optimal medical management and those patients typically recover in weeks to months.[1-3,52] Approximately 50% of the myocarditis patients with significant LV dysfunction show recovery, 25% develop chronic systolic dysfunction, and 25% evolve into end-stage dilated cardiomyopathy potentially requiring permanent VAD implant or transplantation, or even resulting in death.[3,5,6,8,9] The mortality for biopsy-verified viral myocarditis has been described as 20% and 56% at 1 and 4.3 years, respectively.[8] In general, the prognosis of myocarditis seems determined by the specific aetiology, accompanying clinical features, and comorbidities at presentation.[2,3,9] However, the clinical course and prognosis of patients afflicted by severe myocarditis, especially FM cases complicated by pump failure, arrhythmias, and haemodynamic deterioration, are often poor, irrespective of early recognition and medical management.[1,11-13,53] This is mostly attributable to the fact that the pharmaceutical options for patients with FM are often limited, and multiorgan failure is common.[17]

The definition of heart recovery is challenging in patients with FM. This is in part because the haemodynamic stabilization alone does not correspond to recovery of myocardial function[53] and the inflammation process does not end with cardiogenic shock. In chronic heart failure, LV-assisted devices that prolong LV unloading are known to be associated with reverse remodelling, with anti-fibrotic and anti-inflammatory mechanisms.[54] However, less is known about the effect of prolonged LV unloading during myocarditis. The use of PROPELLA was associated with signs of mitigation of inflammation,[38] but further investigation is needed to better understand the cellular response and clinical implication of the unloading-induced modulation of inflammatory reactions. As such, serial EMB could be considered to follow myocardial histological evolution and determine when myocardial recovery is sufficient to warrant weaning or whether unloading is acting as a treatment option for long-lasting myocarditis.[53]

## Future directions

Advancements in MCS technology and improved understanding of myocarditis pathophysiology continue to enhance patient outcomes. Innovations such as fully implantable VADs, bioengineered tissues, and regenerative therapies hold promise for future treatment paradigms. The development of next-generation microaxial flow pumps with enhanced efficiency and reduced complication rates is also a significant area of research. Surgically implanted microaxial flow pumps represent a promising avenue for extending the duration and effectiveness of mechanical support while allowing patient mobility and reducing complication risk. Additionally, ongoing studies focusing on the optimal timing, patient selection criteria, and combination therapies will further refine the use of MCS in myocarditis.

Although case series and cohort studies demonstrated encouraging short-term outcomes following the treatment of FM with Impella devices, the risk for adverse events cannot be ignored. These findings align with previous MCS studies, in various clinical scenarios, emphasizing caution regarding haemorrhagic and vascular complications. Finally, several important questions remain to be clarified in FM cases requiring MCS, including the optimal timing of initiation of an Impella device, duration of support necessary for recovery, or determination of whether permanent VADs or HTx is necessary.

## Conclusion

Mechanical circulatory support plays a pivotal role in managing severe myocarditis, offering a life-saving intervention in cases of refractory cardiogenic shock and heart failure. The choice of MCS device depends on the clinical scenario, and timely intervention is critical in maximizing patient outcomes. As technology and medical knowledge advance, MCS will continue to be a cornerstone in the treatment of this challenging condition, providing hope and improved survival for patients with severe myocarditis.

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No new data were generated or analysed in support of this research.

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