PLACE 2026
PLATFORM OF LABORATORIES FOR ADVANCES IN CARDIAC EXPERIENCE

CONGRESSO
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When the Flu stops the heart: fatal fulminant Influenza A myocarditis

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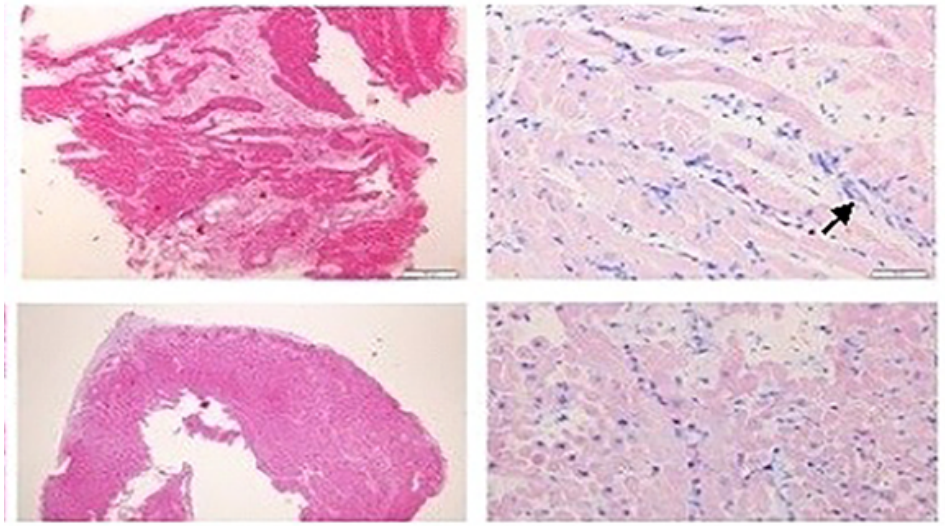
When the Flu Stops the Heart: Fatal Fulminant Influenza A Myocarditis

Myocarditis is an inflammatory disease of the cardiac muscle with various underlying causes. Its clinical course can range from paucisymptomatic forms to cases of shock requiring pharmacological and/or mechanical circulatory support.

Mortality is high in forms with a **fulminant onset**, making rapid and accurate etiological evaluation essential.



FULMINANT MYOCARDITIS - DISEASE OVERVIEW

Rare but catastrophic: approximately **10% of all acute myocarditis cases** diagnosed in hospital settings.

Clinical progression: rapidly leads to cardiogenic shock, malignant arrhytmias, and multi-organ failure.

Etiology: while ofter viral

High mortality: despite advanced mechanical support, the prognosis remains extremely guarded.

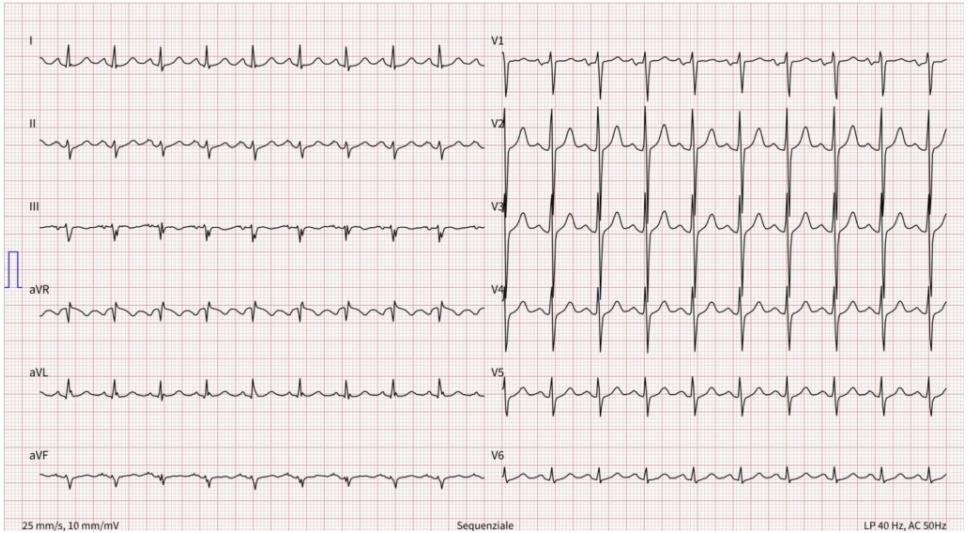
CASE REPORT

- Female, 48 years old
- No cardiovascular risk factors
- No chronic home therapy
- ER admission for astenia and flu like symptoms for a few days (h12.00)

CASE REPORT

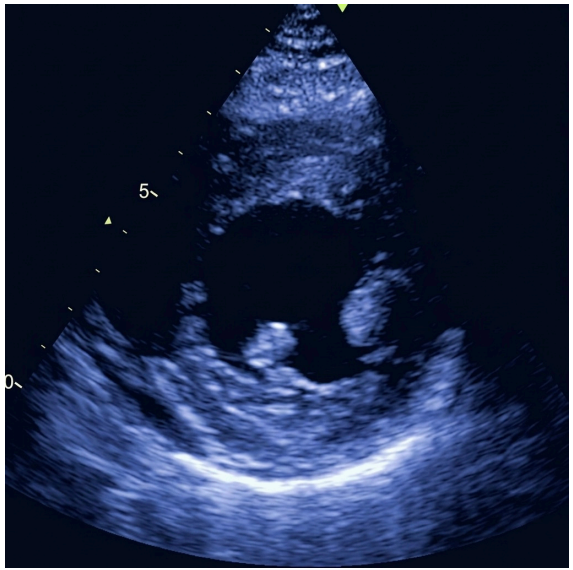
Initial investigations and diagnostic workup:

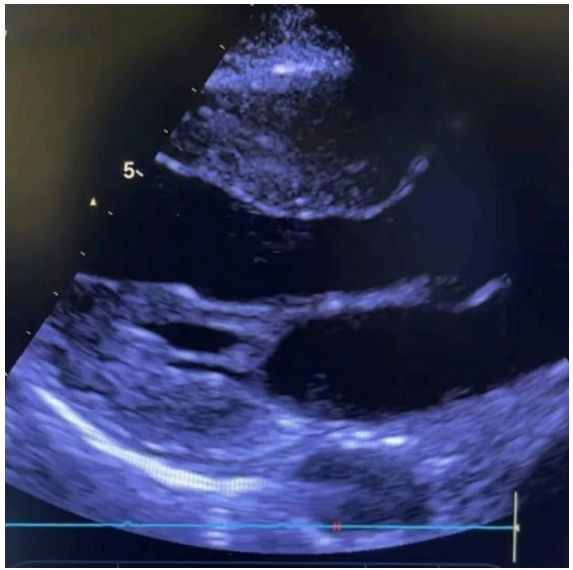
- Sinus Tachycardia, no ECG abnomalities
- Elevated Cardiac Troponin (3965 pg/dL)



CASE REPORT

Transthoracic echocardiogram: Moderate biventricular dysfunction (FE 40%), mild pericardial effusion. (h 17:00)

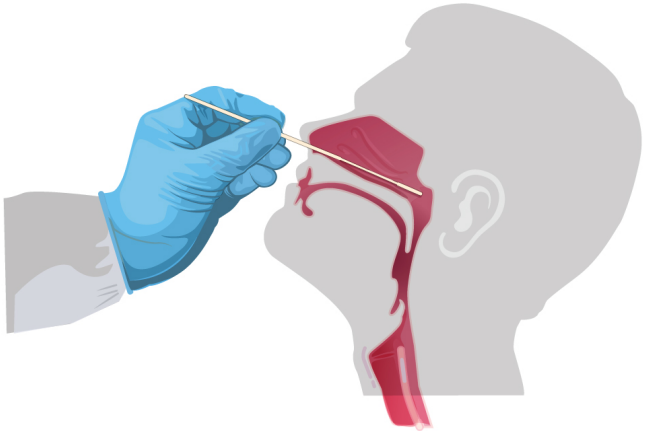




CASE REPORT

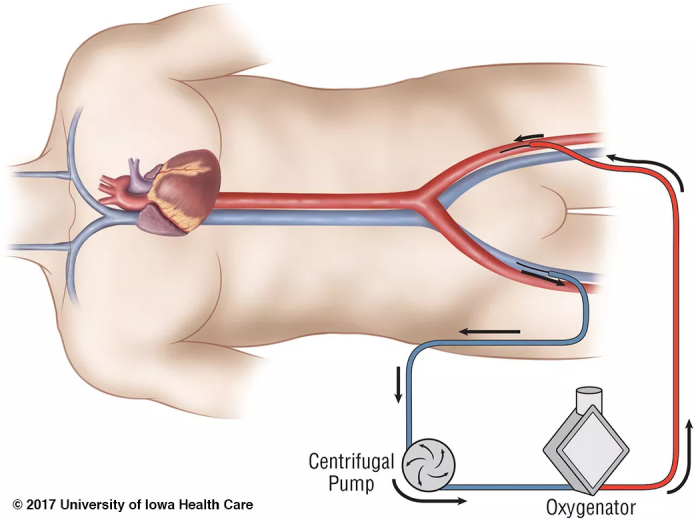
The patient was promptly **admitted to the ICCU** for monitoring and advanced supportive therapy. (h.19:00)

Infectious Etiology Workup: A nasopharyngeal swab was performed immediately at admission to run a Multiplex RT-PCR Respiratory Viral Panel (RVP), targeting common triggers of viral myocarditis.



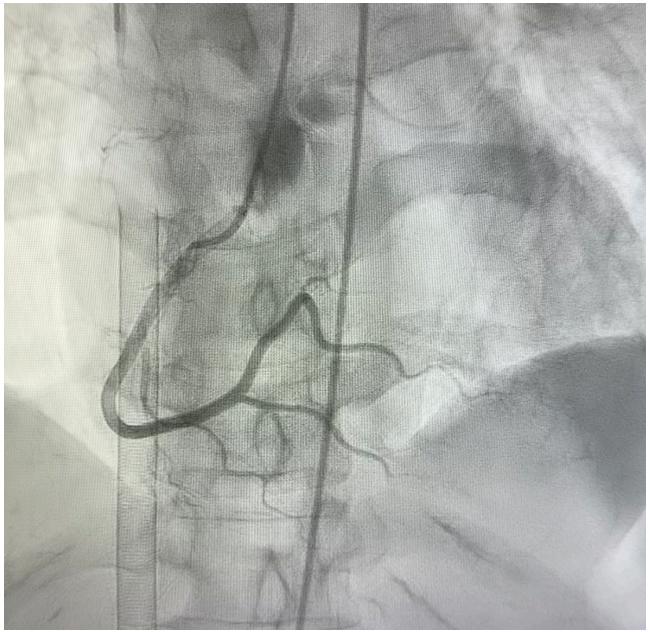
CASE REPORT

Within a few hours of admission, the patient experienced a sudden, catastrophic deterioration of her clinical status, rapidly progressing to severe **cardiogenic shock and profound systemic hypotension** refractory despite high-dose dual vasopressor and inotropic therapy. (h 6:00)



To stabilize systemic tissue perfusion and salvage cardiac work, a **VA-ECMO** system was urgently placed. (h 8:00)

CASE REPORT



Coronary angiography revealed clear vessels, ruling out CAD.

CASE REPORT

Transplant Center Transfer: the patient was urgently transferred to an advanced **Heart Transplant Center** for further management and potential organ replacement evaluation.

Endomyocardial Biopsy: To definitively establish the underlying etiology and guide targeted immunosuppressive or antiviral therapies, a right ventricular endomyocardial biopsy was performed.

Histological Findings: The biopsy analysis revealed a pronounced **lymphomonocytic and macrophagic infiltrate**, confirming the definitive diagnosis of acute active myocarditis**.**

CASE REPORT

Etiological Confirmation: respiratory viral panel performed on the admission nasopharyngeal swab returned **positive for Influenza A**.

Pathophysiological Correlation: Influenza-associated myocarditis can progress rapidly due to a combination of direct viral infiltration of cardiomyocytes and an intense, secondary host immune-mediated cytokine release.

Antiviral Initiation: Targeted antiviral therapy with **Oseltamivir** was immediately initiated to inhibit viral neuraminidase, limiting active viral replication and further myocardial cell injury.

CASE REPORT

An **Intra-Aortic Balloon Pump (IABP)** was initially positioned to decrease afterload, alleviate wall stress, and optimize coronary arterial diastolic perfusion.

Escalation to Impella 5.5: To achieve superior active venting and systemic forward support, the patient was transitioned to an Impella 5.5 transaortic microaxial flow pump, providing up to 5.5 L/min of direct forward flow to completely decompress the LV.

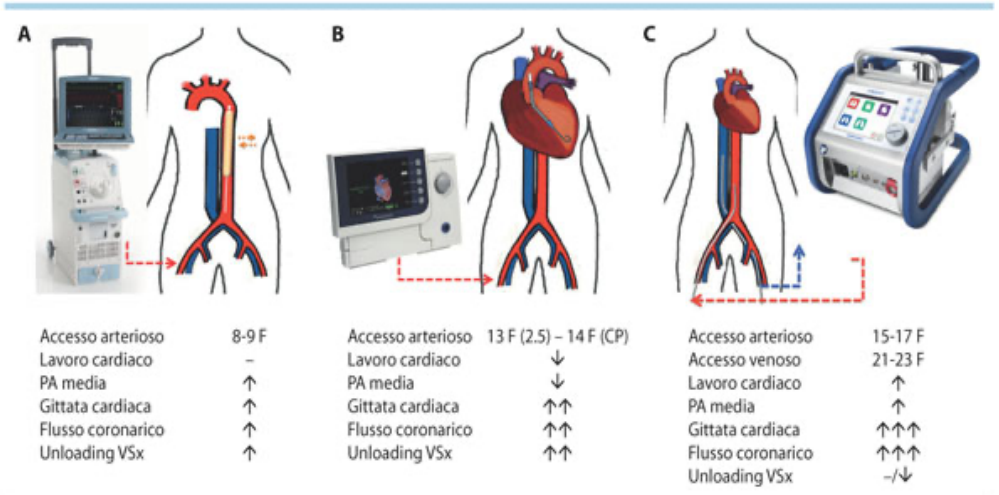
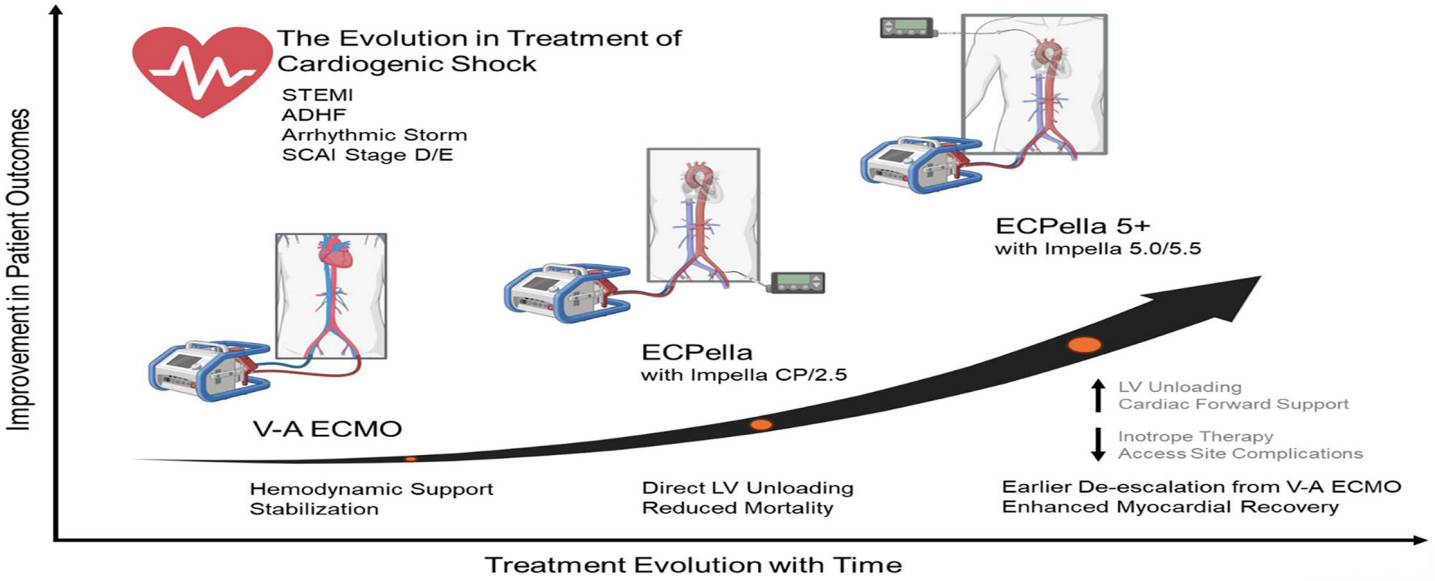


Figura 1. I principali dispositivi di supporto emodinamico: contropulsatore aortico (*A*), Impella (*B*), sistema di ossigenazione a membrana extracorporea (*C*).
PA, pressione arteriosa; VSx, ventricolo sinistro.

CASE REPORT


The Evolution in Treatment of Cardiogenic Shock
STEMI
ADHF
Arrhythmic Storm
SCAI Stage D/E
V-A ECMO
ECPella
with Impella CP/2.5
ECPella 5+
with Impella 5.0/5.5
LV Unloading
Cardiac Forward Support
Inotrope Therapy
Access Site Complications
Hemodynamic Support
Stabilization
Direct LV Unloading
Reduced Mortality
Earlier De-escalation from V-A ECMO
Enhanced Myocardial Recovery
Improvement in Patient Outcomes
Treatment Evolution with Time

CASE REPORT

REFRACTORY VASOPLEGIA AND MOF

Clinical Course Deterioration: Despite achieving optimal mechanical circulatory support (MCS) and adequate pump flows with VA-ECMO and Impella 5.5, the patient's status progressively worsened, driven by an overwhelming systemic inflammatory response.

- **Refractory Vasoplegia:** The patient developed severe, unrelenting vasoplegia, marked by persistent systemic vasodilation, low systemic vascular resistance (SVR), and escalating requirements for multiple vasopressors (Norepinephrine, Vasopressin).
- **Multiple Organ Failure (MOF):** Severe hypoperfusion triggered a rapid multi-organ failure cascade, including severe lactic acidosis, acute liver dysfunction, and oliguric acute kidney injury (AKI) requiring continuous renal replacement therapy (CRRT).

CONCLUSIONS

KEY CLINICAL MESSAGES

- **Early Detection & Monitoring** Prompt recognition of fulminant myocarditis from initial, non-specific symptoms is vital. Implementing aggressive, serial transthoracic echocardiographic (TTE) monitoring is essential to catch rapid ventricular deterioration early and preempt cardiovascular collapse.
- **Biomarkers & Cytokine Therapy** Identifying predictive biomarkers for vasoplegic response will guide timely, targeted therapies. Integrating extracorporeal cytokine adsorption techniques (e.g., cytokine filtration) or specific immunomodulatory therapies alongside mechanical circulatory support (MCS) represents a crucial frontier to mitigate multi-organ failure.

THANK YOU FOR YOUR ATTENTION