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CASE REPORT

Hepatic Dysfunction Secondary to Severe Cardiogenic Shock Evolving to Heart Transplantation

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ABSTRACT

Introduction: Viral myocarditis is a rare but potentially life-threatening cause of cardiogenic shock in young adults. Secondary hepatic dysfunction due to systemic hypoperfusion may mimic primary acute hepatitis, delaying the correct diagnosis. We report the case of a young adult initially admitted with suspected fulminant hepatitis, whose investigation revealed severe cardiogenic shock secondary to viral myocarditis. The patient presented with acute hepatic and renal dysfunction, characterizing multiple organ failure. Invasive hemodynamic monitoring with a pulmonary artery catheter showed a low cardiac index (1.8 L/min/m^2) and elevated pulmonary capillary wedge pressure (24 mmHg), confirming cardiogenic shock. Treatment with dobutamine, intra-aortic balloon pump, and systemic vasodilator therapy led to progressive reversal of hepatic dysfunction and hemodynamic stabilization. Due to persistent inotropic dependence, the patient underwent successful heart transplantation. This case highlights the importance of recognizing hepatic dysfunction as a secondary manifestation of cardiogenic shock and of invasive hemodynamic monitoring in guiding therapy and achieving organ recovery.

Descriptors: Heart Transplantation; Myocarditis; Shock, Cardiogenic; Multiple Organ Failure; Liver Failure, Acute.

Disfunção Hepática Secundária a Choque Cardiogênico Grave Evoluindo Para Transplante Cardíaco

RESUMO

A miocardite viral é uma causa rara, porém potencialmente grave, de choque cardiogênico em adultos jovens. A disfunção hepática secundária à hipoperfusão sistêmica pode mimetizar hepatite aguda primária, retardando o diagnóstico correto. Relata-se o caso de um adulto jovem inicialmente admitido com suspeita de hepatite fulminante, cuja investigação revelou choque cardiogênico grave secundário a miocardite viral. O paciente apresentava disfunção hepática e renal agudas, configurando falência multiorgânica. A monitorização invasiva com cateter de artéria pulmonar evidenciou baixo índice cardíaco (1,8 L/min/m^2) e pressão capilar pulmonar elevada (24 mmHg), confirmando o diagnóstico de choque cardiogênico. O tratamento com dobutamina, balão intra-aórtico e vasodilatador sistêmico resultou em reversão progressiva da disfunção hepática e estabilização hemodinâmica. Persistindo dependência inotrópica, foi indicado e realizado transplante cardíaco com sucesso. O caso destaca a importância do reconhecimento da disfunção hepática como manifestação secundária ao choque cardiogênico e da monitorização hemodinâmica invasiva na reversibilidade orgânica e condução terapêutica.

Descritores: Transplante de Coração; Miocardite; Choque Cardiogênico; Insuficiência de múltiplos órgãos; Falência Hepática Aguda.

CASE REPORT

This case report was conducted in accordance with the CARE (CAse REport) guidelines for clinical case reporting[1].

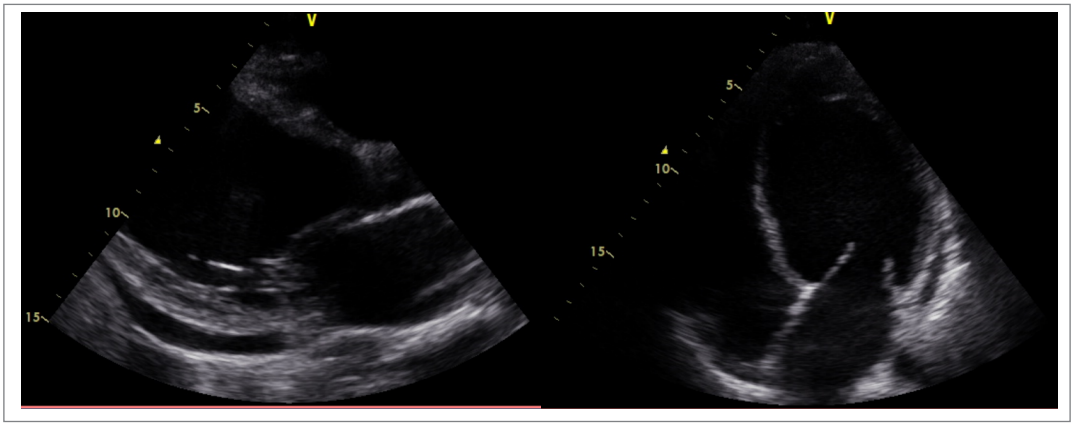
A 23-year-old previously healthy male patient was admitted to the Intensive Care Unit (ICU) of the University of Campinas Hospital de Clínicas (*Hospital de Clínicas da Universidade de Campinas - Unicamp*) following transfer from an external facility, presenting with suspected fulminant hepatitis and potential evaluation for liver transplantation. This hypothesis was raised due to a significant elevation in transaminases, prolongation of prothrombin time, and clinical deterioration during a prior admission at another facility.

Regarding recent medical history, approximately 15 days before admission, family members reported that the patient had been in contact with his 1-year-old niece, who was diagnosed with hand, foot, and mouth disease. Subsequently, the patient presented with symptoms compatible with an influenza-like illness, with no apparent signs of severity. He sought medical attention after the onset of dyspnoea on moderate exertion associated with mild, atypical, non-anginal chest pain, and was then admitted for investigation.

Approximately five days after initial hospital admission, the patient experienced progressive clinical deterioration, characterized by an elevation in transaminases exceeding ten times the upper limit of normal, prolongation of prothrombin time, hypotension, and the development of acute kidney injury prompting transfer to the ICU. On the day before transfer to our facility, he presented with a reduced level of consciousness. Given the inability to exclude hepatic encephalopathy in the context of suspected fulminant hepatitis, he underwent orotracheal intubation.

In the Campinas region, cases with suspected fulminant hepatitis and a potential need for liver transplantation are frequently referred to the University of Campinas Hospital de Clínicas (*Hospital de Clínicas da Unicamp*). Consequently, the patient was initially admitted by the liver transplant team. However, the initial bedside assessment revealed clinical findings suggestive of low cardiac output: the patient was anasarcous, jaundiced, cold, poorly perfused, and clammy, with a concurrent requirement for moderate doses of vasoactive drugs. He was intubated and on mechanical ventilation with moderate-to-high settings and low lung compliance. The abdominal examination showed no evidence of hepatomegaly, jugular venous distension, or hepatojugular reflux.

Given the presentation of shock with an etiology that was not yet fully established, a bedside echocardiographic assessment was performed, demonstrating ventricular dilation with severe systolic dysfunction, with an estimated ejection fraction of approximately 16% and a significant reduction in global contractility (Fig. 1). These findings redirected clinical reasoning toward severe cardiac dysfunction as the primary cause of the shock and associated organ dysfunctions.



Source: Authors.

Figure 1. Echocardiogram upon ICU admission: parasternal long-axis view (left) and apical four-chamber view (right).

Given the presence of hypodynamic shock and the need for more precise hemodynamic characterization, invasive monitoring with a pulmonary artery catheter was chosen. Following insertion, a reduced cardiac index (1.8 L/min/m²), elevated pulmonary capillary wedge pressure (24 mmHg), and a systemic vascular resistance index at the lower limit of normal were observed, consistent with mixed shock (cardiogenic associated with a vasoplegic component) with a predominance of cardiac failure.

Given these findings, inotropic support with dobutamine was promptly initiated at 5 μg/kg.min, and an evaluation by the cardiac surgery team was requested for Intra-Aortic Balloon Pump (IABP) implantation. Both interventions were performed on the day of admission, resulting in a significant improvement in the cardiac index to 3.5 L/min/m² and an increase of more than 12% in oxygen delivery (DO_2) within the first 24 hours.

At the time of ICU admission, the patient also presented with acute kidney injury, which had been diagnosed on the day before transfer. In conjunction with the nephrology team, continuous renal replacement therapy was indicated within the first 24 hours of admission.

After five days of combined hemodynamic support (Table 1)—including left ventricular assistance with an intra-aortic balloon pump (IABP), inotropic therapy, systemic vasodilation guided by hemodynamic parameters, and renal replacement therapy—there was progressive resolution of hepatic and renal dysfunction, reinforcing the hypothesis that these abnormalities were secondary manifestations of cardiogenic shock.

Table 1. Evolution of hemodynamic and laboratory parameters during the first five days of ICU admission.

	Admission	D2	D3	D4	D5
SOFA Score	**17**	**15**	**16**	**16**	**13**
AST (U/L)	7412	5284	3010	1645	935
ALT (U/L)	5416	4408	2730	793	401
INR	4.13	4.22	3.13	2.38	1.87
LDH (U/L)		5896		1893	1345
SVRI (dyn·s·cm^{-5})		2500	1700	3895	1183
Norepinephrine					
(mcg/kg/min)	0.2	-	-	-	-
Dobutamine					
(mcg/kg/min)	5	10	15	20	20
Sodium nitroprusside					
(mcg/kg/min)				0.5	1
CI (L/min/m^2)	1.8	2.2	2.6	2.9	3.0
PCWP (mmHg)	24	28	25	22	15

Source: Elaborated by the authors.

Weaning from the IABP was performed gradually, in accordance with hemodynamic goals, after seven days of support. Despite clinical stabilization, the patient remained dependent on dobutamine without full functional recovery of ventricular function, leading to the indication for heart transplantation after 50 days of hospitalization.

The etiological investigation suggested a possible viral origin, given that the respiratory viral panel performed upon admission was positive. To further clarify the diagnosis, an endomyocardial biopsy was indicated; the result was still pending at the time this report was prepared.

DISCUSSION

Pathophysiology of Hepatic Dysfunction in Cardiogenic Shock

The correlation between acute hepatic dysfunction and circulatory shock states is well established in the literature. In the context of cardiogenic shock, low cardiac output results in systemic hypoperfusion and reduced tissue oxygen delivery, which can culminate in multiple organ dysfunction syndrome. Among these manifestations, hepatic dysfunction stands out and can present primarily in two forms: hypoxic hepatitis and acute liver failure[2-4].

Hypoxic hepatitis, also termed shock liver, is characterized by an abrupt and marked elevation of transaminases resulting from reduced hepatic perfusion associated with venous congestion[5]. Consequently, the management of hepatic dysfunction in this context is not primarily directed at the liver itself, but rather at correcting the underlying hemodynamic instability. In cardiogenic shock, this strategy essentially involves restoring cardiac output, achieved either by increasing myocardial contractility or by reducing systemic afterload[4].

In the present case, the progressive improvement in hepatic laboratory markers accompanied hemodynamic optimization, suggesting a strong causal relationship between hepatic dysfunction and low cardiac output.

Hemodynamic monitoring and support strategy

The hemodynamic management of this case was based on invasive monitoring with a pulmonary artery catheter—a resource that, despite a decline in its use in recent decades, remains indicated in selected situations, particularly in cardiogenic shock or when the etiology of the shock is not clearly established. Initial assessment evidenced a low cardiac index associated with elevated filling pressures, consistent with severe left ventricular failure. Given these findings, the introduction of inotropic therapy with

dobutamine was initiated, aiming to increase myocardial contractility and improve cardiac output[3]. Subsequently, mechanical circulatory support with an intra-aortic balloon pump was instituted, a strategy that helps reduce afterload and improve coronary perfusion[7].

The hemodynamic evolution shown in Table 1 illustrates the correlation between therapeutic interventions and progressive improvements in circulatory parameters. A gradual increase in the cardiac index and a reduction in left ventricular filling pressures were observed over the first days of treatment. Furthermore, the introduction of sodium nitroprusside enabled a further reduction in systemic vascular resistance, thereby improving cardiac output efficiency.

These interventions resulted in a global improvement in tissue perfusion, as reflected in the recovery of hepatic function and the reversal of previously observed acute kidney injury.

Multiorgan Dysfunction in Cardiogenic Shock

In addition to hepatic dysfunction, the patient presented with early acute kidney injury, initially managed with continuous renal replacement therapy. Following the recognition of severe myocardial dysfunction, it became evident that the renal impairment was embedded within the context of cardiorenal syndrome, characterized by the bidirectional interaction between cardiac and renal dysfunction.

With hemodynamic optimization and improved cardiac output, a progressive recovery of renal function was observed, allowing discontinuation of renal replacement therapy after 5 days of treatment. This finding underscores the importance of recognizing the underlying hemodynamic etiology of organ dysfunctions in critically ill patients[8].

Indication for heart transplantation

Following the diagnosis of severe acute myocarditis with significant ventricular dysfunction and dependence on inotropic support, the patient progressed to meet the criteria for advanced heart failure. In this condition, when there is refractoriness to optimized medical therapy, heart transplantation constitutes an established therapeutic strategy. According to the guidelines of the Brazilian Society of Cardiology, patients with advanced heart failure refractory to medical treatment have a formal indication for transplantation, particularly in the presence of persistent dependence on inotropic support or progressive clinical deterioration (Class of recommendation I, level of evidence C)[9].

In the present case, the sustained dependence on dobutamine throughout the hospitalization characterized a profile of persistent hemodynamic instability, fulfilling the criteria for evaluation and listing for heart transplantation. The therapeutic decision also took into account the overall clinical context, including the evolution of organ dysfunction associated with cardiogenic shock and the absence of significant ventricular recovery despite the support measures instituted.

In similar scenarios, mechanical circulatory support strategies can be considered as a bridge to recovery or transplantation, including ventricular assist devices or extracorporeal life support. However, the indication for these modalities depends on institutional availability, the patient's clinical characteristics, and the assessment of myocardial recovery potential.

In the present case, the persistent dependence on inotropes and the severity of ventricular dysfunction substantiated the indication for heart transplantation as a definitive therapeutic strategy, following multidisciplinary evaluation and the exclusion of contraindications, in compliance with the criteria established in current guidelines.

Viral Etiology and Contextualization within the Literature

Subsequent etiological investigation revealed, by means of intraoperative myocardial biopsy, findings consistent with perivascular lymphocytic myocarditis with areas of inflammatory activity in both ventricles. This histological pattern strongly suggests a viral etiology, a hypothesis further supported by a history of viral syndrome and a positive respiratory viral panel upon admission.

Classically, viruses with the greatest myocardial tropism described in the literature include adenoviruses and enteroviruses, especially coxsackieviruses A and B. Other viral agents have also been associated with the development of myocarditis, such as parvovirus B19 and viruses from the Herpesviridae family, including human herpesvirus[6], Epstein-Barr virus, and cytomegalovirus[10].

Although respiratory viruses are not among the agents most frequently associated with myocarditis, isolated cases have been described, suggesting a possible triggering role in certain clinical contexts.

CONCLUSION

This case illustrates that acute hepatic failure can represent a secondary manifestation of severe cardiogenic shock, initially mimicking fulminant hepatitis. Early recognition of myocardial dysfunction and invasive hemodynamic monitoring allowed for appropriate therapeutic guidance and the reversal of organ dysfunctions. Furthermore, it highlights the importance of early

evaluation for heart transplantation in patients with severe ventricular dysfunction and persistent dependence on inotropic support.

ETHICS

The patient provided informed consent for the publication of this case report and associated images, with anonymity guaranteed. According to institutional guidelines, isolated case reports are exempt from formal approval by a Research Ethics Committee.

CONFLICT OF INTEREST

Nothing to declare.

AUTHOR'S CONTRIBUTION

Substantive scientific and intellectual contributions to the study: Chueiri F Neto, Passos SD; **Conception and design:** Passos SD; **Data analysis and interpretation:** Chueiri F Neto, Neves TMP, Passos SD, Shin SW; **Article writing:** Passos SD; **Critical revision:** Chueiri F Neto. **Final approval:** Chueiri F Neto, Passos SD.

DATA AVAILABILITY STATEMENT

All datasets were generated or analyzed in the current study.

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Not applicable.

DECLARATION OF USE OF ARTIFICIAL INTELLIGENCE TOOLS

The authors declare that no artificial intelligence tools were used in the preparation, writing, data analysis, or review of this manuscript.

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