Acute hepatitis as an initial manifestation of cardiogenic shock with progression to heart transplantation

Samira Dias dos Passos, Felicio Chueiri Neto, Thais Piovezan Neves, Seok Woo Shin, Ilka de Fatima Ferreira Santana Boin

https://doi.org/10.1590/SciELOPreprints.14236

Submitted on: 2025-11-22
Posted on: 2025-12-11 (version 1)
(YYYY-MM-DD)

Acute hepatitis as an initial manifestation of cardiogenic shock with progression to heart transplantation.

Author Contributions (CRediT Taxonomy)

- **Samira Dias dos Passos:** Conceptualization; Writing – Original Draft; Writing – Review & Editing.
- **Thais Maria Piovezan Neves:** Writing – Original Draft Support.
- **Seok Shin Woo:** Writing – Review & Editing; Methodology/Technical Review.
- **Ilka de Fátima Ferreira Santana Boin:** Supervision; Writing – Review & Editing.
- **Felicio Chueiri Neto:** Validation; Writing – Review & Editing.

ORCID Identifier
https://orcid.org/0000-0002-8780-0451
https://orcid.org/ 0009-0007-3268-2818
https://orcid.org/0000-0002-1165-2149
https://orcid.org/0000-0003-3400-6163
https://orcid.org/0009-0006-9396-2795

Short title: Cardiogenic shock masked as acute hepatitis

Keywords: Heart Transplantation; Myocarditis; Shock, Cardiogenic

Abstract: This is a case report of a patient admitted to an academic medical center, initially presenting with acute hepatitis with a possible indication for liver transplantation. After targeted intensive evaluation, including the use of a pulmonary artery catheter and bedside ultrasonography, the patient was diagnosed with cardiogenic shock due to viral myocarditis — confirmed by endomyocardial biopsy — and was subsequently successfully treated with heart transplantation.

Case Report

A previously healthy male patient was admitted to the Intensive Care Unit after being transferred from an outside facility due to an initial suspicion of fulminant hepatitis with possible need for liver transplant evaluation, as he had already been hospitalized elsewhere for investigation of acute hepatitis. In the weeks preceding admission, family members reported that he had contact with a young child diagnosed with hand-foot-and-mouth disease, consistent with the patient's prior flu-like symptoms that initially appeared mild. He sought medical attention after developing exertional dyspnea and mild non-anginal chest pain, leading to hospitalization.

Several days later, he experienced progressive clinical deterioration, with markedly elevated transaminases, prolonged prothrombin time, hypotension, acute kidney injury, and the need for intensive care support. On the day prior to transfer, he developed a decreased level of consciousness; hepatic encephalopathy could not be excluded, and he was intubated due to concern for fulminant hepatitis. Upon arrival at the receiving transplant center, the transplant and surgical teams assumed care. Bedside assessment revealed a severely ill, anasarcatic, deeply icteric patient, cold, poorly perfused, and with clammy skin. He was intubated on moderate-to-high ventilatory settings, with low pulmonary compliance, and required moderate-dose vasopressors. Abdominal examination showed no hepatomegaly, jugular venous distention, or hepatojugular reflux.

A bedside echocardiogram was performed to clarify the etiology of shock, demonstrating—on subjective visual evaluation—features of dilated cardiomyopathy, with an estimated left ventricular ejection fraction of approximately 16% and severely reduced contractility, as demonstrated in Figure 1.

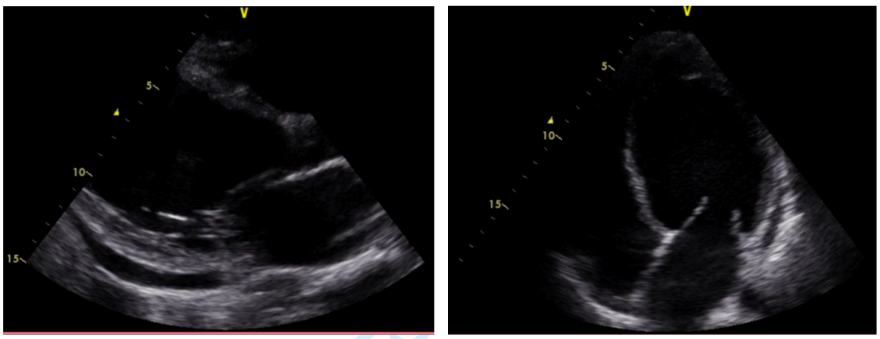

Figure 1: Bedside echocardiogram showing a parasternal long-axis view on the left and an apical four-chamber view on the right at ICU admission.

In practical terms, the clinical presentation clearly indicated cold, hypodynamic shock, although the underlying etiology remained undefined. Given the progressive development of additional organ dysfunctions, pulmonary artery catheter monitoring was pursued to clarify the hemodynamic parameters and allow for more targeted, evidence-based management decisions.

Following insertion of the pulmonary artery catheter, hemodynamic measurements demonstrated a low cardiac output (CI 1.8 L/min/m²), indexed systemic vascular resistance at the lower limit of normal, and a pulmonary capillary wedge pressure of 24 mmHg. The initial interpretation of the hemodynamic study was that of mixed shock (cardiogenic + vasoplegic), predominantly driven by cardiac failure.

In response, inotropic support was promptly initiated with low-dose dobutamine at 5 mcg/kg/min, and the cardiac surgery team was consulted for intra-aortic balloon pump placement. Both interventions were performed on the day of admission, resulting in clear improvement in the cardiac index (reaching 3.5 L/min/m²) and in systemic oxygen delivery (DO_2), with an increase of more than 12% within the first 24 hours.

I It is also important to highlight that, at the time of ICU admission, the patient simultaneously presented with acute kidney injury, previously diagnosed the day before transfer. Therefore, in conjunction with the nephrology team, continuous renal replacement therapy was initiated within the first 24 hours of ICU admission.

After five days of combined hemodynamic support with left ventricular assistance—via intra-aortic balloon pump, pharmacologic therapy with dobutamine and systemic vasodilators, and renal replacement therapy—both hepatic and renal dysfunctions were essentially resolved. This supported the hypothesis that the patient was experiencing a single syndrome—cardiogenic shock—that triggered a cascade of secondary organ dysfunctions.

Weaning from the intra-aortic balloon pump was carried out according to hemodynamic targets, seven days after its placement. The patient was able to maintain adequate cardiac output on dobutamine alone; however, due to the lack of full functional cardiac recovery, heart transplantation was indicated after 50 days of hospitalization.

A causal relationship with the underlying etiology still required clarification, given that the only positive finding was a respiratory virus detected on the viral panel performed at admission. Therefore, an endomyocardial biopsy was also indicated, which subsequently confirmed the diagnostic hypothesis of viral myocarditis.

Regarding case management, once a cardiogenic component was suspected as the main cause of decompensation—based on bedside echocardiography— invasive monitoring with a pulmonary artery catheter (PAC) was chosen. Although its use has declined, the PAC is not obsolete and remains indicated primarily in cases of cardiogenic shock, in which accurate measurements of preload and afterload are essential for clinical decision-making, or in situations where the etiology of shock is not fully established [(1).]

With a cardiac output of 1.6 L/min/m², pulmonary artery occlusion pressure of 24 mmHg, central venous pressure of 28 mmHg, and a systemic vascular resistance of 2500 dyn·s/cm^5, dobutamine—a positive inotropic agent with some vasodilatory effect—was promptly initiated [2]. After an initial improvement in contractility, but complicated by dobutamine-induced tachyarrhythmia, left ventricular mechanical support with an intra-aortic balloon pump was indicated, resulting in additional improvement in cardiac output.

After 24 hours of the initial measures for cardiogenic shock, a systemic vasodilator—sodium nitroprusside—was added to the therapeutic regimen, guided by the hemodynamic parameters provided by the PAC. Within 72 hours of cardiogenic shock management, a decline in transaminase levels was observed, along with improvement in the confounding factors that had contributed to the initial diagnostic uncertainty.

Parameter	Admission	D2	D3	D4	D5
SOFA score	**17**	**15**	**16**	**16**	**13**
AST (TGO U/L)	7412	5284	3010	1645	935
ALT (TGP U/L)	5416	4408	2730	793	401
INR	4,13	4,22	3,13	2,38	1,87
LDH (U/L)		5896		1893	1345
Sistemic vascular resistance index (SVRI dyn.s.cm^{-5}.m^2)		2500	1700	3895	1183
Norepinephrine (mcg/kg/min)	0,2	-	-	-	-
Dobutamine (mcg/kg/min)		10	15	20	20
Sodium nitroprusside (mcg/kg/min)				0,5	1
Cardiac index (L/min/m^2)	1,9	2,2	2,6	2,9	3,0
Pulmonary artery occlusion pressure (mmHg)		28	25	22	15

Table 1 *- Hemodynamic and laboratory values over five consecutive days of ICU evaluation*

It is well established in the literature that there is a correlation between acute hepatic dysfunction and circulatory shock. During cardiogenic shock, the hemodynamic state of low cardiac output leads to multiple organ dysfunctions, including hepatic impairment, which may present primarily in two forms: hypoxic hepatitis and acute liver failure. Management of both conditions consists of attempting to restore cardiac output, which in the setting of cardiogenic shock is closely related to myocardial contractility or afterload reduction [3].

In this context, the consistency between the literature and the hemodynamic findings in this clinical case becomes evident. As cardiogenic shock was managed—initially with dobutamine, through increased contractility, and subsequently with an intra-aortic balloon pump, mainly through afterload reduction—there was a clear improvement in global tissue perfusion, additionally reflected by a decline in transaminase levels, as shown in Table 1.

With the gradual increase in dobutamine dose, during the use of the intra-aortic balloon pump, **as demonstrated in Table 1**, we observed a reduction in systemic vascular resistance up to Day 3. On the fourth day, a new increase in systemic vascular resistance was noted following

evidence of asynchrony with the intra-aortic balloon pump. In addition to adjusting the inflation and deflation timing, a systemic vasodilator—sodium nitroprusside—was added, resulting in combined benefits both in increasing cardiac output and reducing systemic vascular resistance.

Left ventricular filling pressures, represented by the pulmonary artery occlusion pressure (PAOP) in Table 1, also showed a significant reduction over the course of several days. This improvement can be attributed both to hemodynamic optimization with increased cardiac output, as previously described, and to volume management, initially supported by continuous renal replacement therapy, given the presence of acute kidney injury as one of the patient's initial organ dysfunctions. Once severe myocardial dysfunction was diagnosed, it became evident that the kidney injury was, in fact, a cardiorenal syndrome; after five days of treating the underlying cause, renal replacement therapy was discontinued.

After the diagnosis of severe acute myocarditis, with hemodynamic compromise and cardiogenic shock refractory to medical therapy, the patient became dependent on dobutamine to maintain cardiac output throughout hospitalization. Additionally, criteria for advanced heart failure were met. Based on these findings and according to the latest heart transplantation guidelines, the patient received a **Class I recommendation, Level of Evidence C** for heart transplantation [4].

Five months after clinical management of heart failure, the patient ultimately underwent successful heart transplantation. Postoperatively, he continued outpatient follow-up, through which a precise etiologic investigation was completed using intraoperative myocardial biopsy. Consistent with the literature, the biopsy revealed perivascular lymphocytic myocarditis with areas of activity in both ventricles. This finding strongly suggests viral myocarditis, considering the patient's exposure to a respiratory virus, confirmed by viral panel at admission. This makes the case even more remarkable, as this virus is not among the most common etiologic agents of myocarditis. Viruses with known cardiotropic potential include adenoviruses and enteroviruses (such as Coxsackie A and B, and echoviruses). Enteroviruses like parvovirus B19, known to be vasculotropic, may also contribute to myocarditis, as can lymphotropic viruses of the Herpesviridae family—including human herpesvirus 6 (HHV-6), Epstein-Barr virus, and cytomegalovirus (CMV) [5].

Thus, it is possible to conclude that not all cases of acute liver dysfunction are necessarily attributable to primary hepatic causes. Comprehensive and individualized assessment of the critically ill patient is crucial, with persistent investigation of alternative contributors to shock. This case also illustrates that the pulmonary artery catheter still holds an important role in the intensive care setting; without it, proper management of cardiogenic shock and subsequent recovery of organ dysfunction would not have been possible. This underscores the essential role of the intensivist—together with the entire care team—in the management of critically ill patients.

Data Avaliability Statement

Data sharing is not applicable as no datasets were generated or analysed for this case report.

Ethics Committee Approval Statement

The informed consent form was signed by the patient's legal guardian, and the study was approved by the Unicamp Research Ethics Committee (CEP), CAAE number 83889524.0.0000.5404.

Conflict of Interest Statement

The authors declare that they have **no conflicts of interest**, whether financial, personal, academic, or institutional, that could have influenced the work reported in this manuscript. This statement is made in accordance with the guidelines of the Committee on Publication Ethics (COPE).

References

1. Dias FS, Rezende E, Mendes CL, Réa-Neto Á, David CM, Schettino G, et al. Parte II: monitorização hemodinâmica básica e cateter de artéria pulmonar. Rev Bras Ter Intensiva [Internet]. março de 2006 [citado 13 de dezembro de 2024];18(1). Disponível em: http://www.scielo.br/scielo.php?script=sci_arttext&pid=S0103-507X2006000100012&lng=pt&nrm=iso&tlng=pt

2. Shabana A, Dholoo F, Banerjee P. Inotropic Agents and Vasopressors in the Treatment of Cardiogenic Shock. Curr Heart Fail Rep. dezembro de 2020;17(6):438–48.

3. Kasper P, Tacke F, Steffen HM, Michels G. Hepatische Dysfunktion bei Patienten mit kardiogenem Schock. Med Klin - Intensivmed Notfallmedizin. outubro de 2019;114(7):665–76.

4. Bacal F, Marcondes-Braga FG, Rohde LEP, Xavier Júnior JL, Brito FDS, Moura LAZ, et al. 3ª Diretriz Brasileira de Transplante Cardíaco. Arq Bras Cardiol [Internet]. 2018 [citado 2 de setembro de 2024]; Disponível em: https://www.scielo.br/scielo.php?script=sci_arttext&pid=S0066-782X2018001400230

5. Tschöpe C, Ammirati E, Bozkurt B, Caforio ALP, Cooper LT, Felix SB, et al. Myocarditis and inflammatory cardiomyopathy: current evidence and future directions. Nat Rev Cardiol. março de 2021;18(3):169–93.

This preprint was submitted under the following conditions:

- The authors declare that the necessary Terms of Free and Informed Consent of participants or patients in the research were obtained and are described in the manuscript, when applicable.
- The authors declare that the preparation of the manuscript followed the ethical norms of scientific communication.
- The authors declare that they are aware that they are solely responsible for the content of the preprint and that the deposit in SciELO Preprints does not mean any commitment on the part of SciELO, except its preservation and dissemination.
- The authors declare that the data, applications, and other content underlying the manuscript are referenced.
- The deposited manuscript is in PDF format.
- The authors declare that the research that originated the manuscript followed good ethical practices and that the necessary approvals from research ethics committees, when applicable, are described in the manuscript.
- The authors declare that once a manuscript is posted on the SciELO Preprints server, it can only be taken down on request to the SciELO Preprints server Editorial Secretariat, who will post a retraction notice in its place.
- The authors agree that the approved manuscript will be made available under a Creative Commons CC-BY license.
- The submitting author declares that the contributions of all authors and conflict of interest statement are included explicitly and in specific sections of the manuscript.
- The authors declare that the manuscript was not deposited and/or previously made available on another preprint server or published by a journal.
- If the manuscript is being reviewed or being prepared for publishing but not yet published by a journal, the authors declare that they have received authorization from the journal to make this deposit.
- The submitting author declares that all authors of the manuscript agree with the submission to SciELO Preprints.