Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Acute Dengue-associated Myocarditis in a Resource-limited Setting: A Case Series From Côte D'ivoire

Inès Angoran Regnier
iangoran@yahoo.fr
Université Félix Houphouët-Boigny
Adama Doumbia
Université Félix Houphouët-Boigny
Corinne Akpovo
Université Félix Houphouët-Boigny
Roland N'Guetta
Université Félix Houphouët-Boigny
Christophe Konin
Université Félix Houphouët-Boigny
Serge Paul Eholie
Université Félix Houphouët-Boigny

Case Report

Keywords: Dengue, myocarditis, heart block, expanded dengue syndrome, cardiac MRI, Côte d'Ivoire

Posted Date: September 4th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10328635/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Abstract

Dengue fever can progress to severe organ dysfunction, known as the expanded dengue syndrome. Among its atypical complications, acute myocarditis is rare, underdiagnosed, and potentially fatal. We report a series of four young male patients admitted to Treichville University Hospital (Abidjan, Côte d'Ivoire) illustrating the diverse spectrum of dengue-related myocarditis. Presentations ranged from acute heart failure to high-grade atrioventricular block requiring temporary transvenous pacing, occurring during different phases of dengue illness While elevated high-sensitivity troponin was a consistent warning sign, 3-Tesla cardiac magnetic resonance (CMR) imaging definitively confirmed myocardial inflammation via localized edema and subepicardial late gadolinium enhancement. This series highlights the need for a high index of suspicion during dengue outbreaks, where management demands a careful balance between fluid resuscitation and myocardial strain.

Introduction

Dengue fever is a rapidly spreading arboviral disease transmitted by the *Aedes aegypti* mosquito, caused by the dengue virus (DENV), a single-stranded RNA virus belonging to the Flaviviridae family [1, 2]. Four distinct serotypes have been isolated globally (DENV-1 to DENV-4), with recent escalations placing over 2.5 billion people at risk in endemic regions spanning Central America, Asia, and sub-Saharan Africa [2]. In Côte d'Ivoire, dengue maintains an endemo-epidemic status, facing seasonal outbreaks during the high rainy period; the latest significant outbreak occurred between May and July 2024, during which three co-circulating serotypes (DENV-1, DENV-2, and DENV-3) were identified across urban settings.

While the vast majority of infected individuals remain asymptomatic or present with a self-limiting, non-specific flu-like febrile illness, approximately 10% progress toward severe clinical phenotypes [1, 2]. The concept of "expanded dengue syndrome," which includes severe hepatitis, neurological dysfunction, acute kidney injury, and cardiac complications affecting patients who may not show classical signs of capillary leakage, was introduced by the WHO to include unusual or atypical visceral involvements [3, 4].

Arboviral infections are increasingly recognized as causes of cardiovascular manifestations, with an incidence ranging widely from 15% to 50% in hospitalized cohorts [5]. These problems include functional myocardial depression, transient arrhythmias, pericarditis, atrioventricular conduction blocks, and non-specific electrocardiographic (ECG) alterations [5, 6]. While mild conduction disturbances are occasionally noted during the convalescent stage, acute myocarditis complicating severe dengue remains infrequently reported and poses diagnostic challenges for clinicians [7, 8]. This article describes a case series of four male patients presenting with acute dengue-related myocarditis and varied conduction injuries at Treichville University Hospital, highlighting the chronological evolution of cardiac injury across the clinical phases of dengue and the clinical management required to balance fluid balance against an inflamed myocardium.

Case Presentations

This retrospective case series was conducted at the Infectious and Tropical Diseases Unit of Treichville University Hospital, Abidjan, Côte d'Ivoire. All consecutive patients hospitalized between January and August 2025 with laboratory-confirmed acute dengue infection were screened for cardiac involvement. During the study period, 3,189 patients were hospitalized, of whom 103 (3.2%) had confirmed dengue infection. Among these, 20 patients (19.4%) fulfilled the World Health Organization (WHO) criteria for severe dengue, and four patients (20.0%) were diagnosed with acute dengue-associated myocarditis. These four patients constitute the present case series (Figure 1).

Case 1

The first patient was a 46-year-old man with a history of smoking who presented after four days of high-grade fever, chest pain, dyspnea, and malaise. On admission, he was febrile (40.1°C), tachycardic (124 beats/min), and mildly hypoxemic (SpO_2 93%). Laboratory investigations confirmed DENV-2 infection by RT-PCR, with leukopenia, grade 3 thrombocytopenia, elevated CRP (32 mg/L), and markedly increased high-sensitivity troponin (11,800 ng/L). ECG showed left anterior hemiblock with diffuse low voltage (figure 2). Transthoracic echocardiography revealed preserved left ventricular ejection fraction (LVEF 60%) and a small pericardial effusion. Cardiac magnetic resonance (CMR) demonstrated myocardial edema with subepicardial late gadolinium enhancement, fulfilling the revised Lake Louise criteria for acute myocarditis (figure 3). The patient was managed with supportive care and guideline-directed heart failure therapy (enalapril, bisoprolol, and furosemide). His clinical condition and laboratory abnormalities progressively improved, and he was discharged after eight days.

Case 2:

A 29-year-old man living with well-controlled HIV infection presented with high fever, chest pain, dyspnea, and fatigue. Dengue virus serotype 3 infection was confirmed by laboratory testing. ECG demonstrated a new complete left bundle branch block (figure 4), while echocardiography revealed severe global left ventricular hypokinesia with an LVEF of 35%. Laboratory investigations showed leukopenia, thrombocytopenia, CRP of 84 mg/L, troponin of 14,200 ng/L, and NT-proBNP of 456 pg/mL. Continuous cardiac monitoring was instituted, and guideline-directed medical therapy for heart failure (enalapril, bisoprolol, and furosemide) was initiated. The patient's clinical status improved progressively, with normalization of platelet count and resolution of the conduction abnormality. He was discharged after eight days with complete recovery of symptoms.

Case 3:

A 28-year-old previously healthy man presented with five days of fever, myalgia, retro-orbital pain, chest pain, and recurrent syncope. Dengue virus serotype 2 infection was confirmed, while malaria was excluded. ECG monitoring revealed advanced second-degree atrioventricular block associated with severe symptomatic bradycardia (figure 5). Troponin reached 9,830 ng/L and NT-proBNP 400 pg/mL, whereas echocardiography showed preserved left ventricular systolic function (LVEF 58%). Because of persistent hemodynamic instability, a temporary transvenous pacemaker was inserted. Intravenous

methylprednisolone was administered because of suspected inflammatory involvement of the conduction system, followed by low-dose enalapril after stabilization. Sinus rhythm recovered within ten days, allowing pacemaker removal, and the patient was discharged without residual conduction abnormalities.

Case 4:

A 24-year-old previously healthy man presented after one week of fever, severe myalgia, and acute chest pain. Laboratory investigations confirmed acute DENV-2 infection with profound leukopenia, elevated CRP (125 mg/L), and markedly increased troponin (15,600 ng/L). ECG demonstrated apicolateral ST-segment elevation suggestive of acute myocardial injury (figure 6). Echocardiography showed preserved systolic function (LVEF 62%). CMR performed on day 9 demonstrated myocardial edema associated with subepicardial and intramyocardial late gadolinium enhancement, confirming acute myocarditis according to the revised Lake Louise criteria (figure 7). The patient received supportive treatment with ramipril, bisoprolol, and strict physical rest. Clinical symptoms and laboratory abnormalities resolved completely within ten days.

Table 1. Laboratory Investigations and Quantitative Clinical Characteristics

Laboratory Parameter / Constant	Case 1	Case 2	Case 3	Case 4
Demographics & Profile	46y, Male, Smoker	29y, Male, HIV+	28y, Male, Financier	24y, Male, Sales agent
Dengue Serotype / Co-infection	DENV-2 / None	DENV-3 / *P. falciparum*	DENV-2 / *P. falciparum*	DENV-2 / None
Hemoglobin (g/dL) 11.5-17.5g/dL	13.2	10.8	11.5	14.1
Total Leukocyte Count (/mm³) 4-11 ×10³/µL	3,510	1,400	2,000	700
Nadir Platelet Count 150-450 ×10³/µL	42 000	32 000	55 000	78 000
C-Reactive Protein (mg/mL) <0.5 mg/L	32	84	45	125
Alanine Transaminase 10-55 U/L	99	150	147	210
Aspartate Transaminase 8-35 U/L	48	145	136	199
Peak Cardiac Troponin (ng/L) <0.6 ng/L	11,800	14,200	9,830	15,600
NT-proBNP (pg/mL)	210	456	400	Not measured
Electrocardiogram (ECG)	LAHB, low voltage	New Complete LBBB	Second-degree AV Block	Apicolateral ST elevation
Echocardiography (TTE)	LVEF 60%, LVH, PE	LVEF 35%, hypokinesia	LVEF 58%, normal valves	LVEF 62%, normal valves

Laboratory Parameter / Constant	Case 1	Case 2	Case 3	Case 4
Cardiac MRI (CMR)	Lateral subepicardial LGE	Not performed	Not performed	Subepicardial/intramyocardial LGE
Clinical Outcome	Favorable, full recovery	Favorable, LVEF recovery	Favourable, sinus rhythm restored	Favorable, symptom resolution

Abbreviations: LAHB, left anterior hemiblock; LBBB, left bundle branch block; LVH, left ventricular hypertrophy; PE, pericardial effusion; LVEF, left ventricular ejection fraction; AV, atrioventricular; LGE, late gadolinium enhancement; LMWH, low-molecular-weight heparin; NSAIDs, non-steroidal anti-inflammatory drugs.

Discussion

Dengue fever is a viral disease with an endemic presence in more than 100 countries and a remarkable in its occurrence worldwide. These four observations illustrate the broad clinical diversity of acute dengue-related myocarditis occurring in young male patients with little to no prior cardiac history, predominantly driven by the DENV-2 serotype [5, 9]. The spectrum ranged from mild myocardial injury and isolated conduction abnormalities to severe expressions featuring profound left ventricular (LV) systolic dysfunction or high-grade atrioventricular block [5, 6, 7]. A critical finding in this series is that in this small series, cardiac manifestations appeared to occur during different phases of dengue illness, suggesting a temporal relationship between viral infection and myocardial injury., but follow a possible chronological relationship with the clinical phases of dengue virus infection, requiring highly tailored medical management [3, 8].

The pathophysiology of cardiac injury in dengue is complex and multifactorial [5]. While direct viral invasion of cardiomyocytes has been structurally documented in isolated autopsy studies, the majority of clinical data points toward an indirect immune-mediated pathway [6, 7]. The massive, systemic release of pro-inflammatory cytokines during the viremic phase alters capillary permeability and induces myocardial tissue inflammation [7, 10, 11]. In our series, the subepicardial and focal distribution of late enhancement on CMR, combined with the absence of obstructive coronary disease, strongly argues for this immune-inflammatory mechanism [5, 12]. One of the most consistent findings across our patients was the marked elevation of high-sensitivity cardiac troponin. Although troponin elevation is not specific for myocarditis, its systematic increase in all four patients, combined with compatible clinical and electrocardiographic findings, strongly supported myocardial involvement. In resource-limited settings where CMR is not routinely available, serial troponin measurements may represent an accessible tool for early detection and follow-up of cardiac injury during acute dengue infection.

Chronologically, this inflammatory cascade can manifest across different stages of the disease:

- Late Febrile / Early Critical Phase (Cases 1, 2, and 3): Myocardial injury peaks between day 4 and day 5, precisely when plasma leakage, profound thrombocytopenia, and systemic viral load induce maximum metabolic stress [3]. In Case 2, this led to severe myocardial stunning and acute heart failure with an LVEF of 35% [5, 9]. In Case 3, localized inflammation of the AV node caused high-grade conduction block [6, 7].
- Convalescence Phase (Case 4): Onset on day 7 suggests a delayed post-viral immune-mediated hypersensitivity reaction or localized immune-complex deposition within the myocardium, occurring even as the systemic hematological markers are beginning to recover [7, 8].

The Position Statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases places major emphasis on comprehensive risk stratification [13]. This includes monitoring for the development of ventricular dysfunction, high-grade conduction blocks, and utilizing specialized criteria for non-invasive tissue characterization [13, 14]. In suspected cases of myocarditis, ECG modifications, echocardiographic abnormalities, and elevated biomarkers of myonecrosis (Troponin, CK-MB) are especially helpful to formulate a timely clinical diagnosis [15, 16]. In the present series, the diagnosis of clinically suspected myocarditis was based on the European Society of Cardiology recommendations, combining compatible clinical presentation, elevated cardiac biomarkers, electrocardiographic abnormalities, and echocardiographic findings. In the two patients who underwent CMR, the diagnosis was further confirmed using the revised Lake Louise Criteria demonstrating myocardial edema and non-ischemic late gadolinium enhancement.

Our series successfully applied these standards in several ways:

- Hospitalization and Telemetry: All four patients were immediately hospitalized and placed under continuous ECG monitoring, optimizing survival odds against unpredictable tachyarrhythmias or high-grade blocks [13].
- Advanced Imaging Support: 3-Tesla CMR was utilized in Cases 1 and 4, demonstrating the typical subepicardial LGE signature of viral myocarditis and confirming diagnosis non-invasively [12, 13].
- Device and Anti-inflammatory Strategy: In Case 3, the prompt insertion of a temporary transvenous pacemaker successfully managed the hemodynamic instability caused by a high-grade AV block, while systemic corticosteroids were introduced to reduce immune-mediated conduction tissue edema [6, 7, 13].

However, translating these advanced imaging and electrophysiology standards into sub-Saharan African realities highlights substantial structural challenges [5]. Access to high-field CMR imaging and electrophysiology suites is extremely restricted in West Africa. In resource-limited settings where CMR is unavailable, clinicians must adapt by utilizing serial cardiac biomarkers (troponin and NT-proBNP) combined with structured bedside echocardiography as a reliable, accessible alternative for the early diagnosis and therapeutic follow-up of these complex cardiac presentations [12, 15, 16].

The use of corticosteroids in Case 3 should not be interpreted as routine management of dengue-associated myocarditis. Methylprednisolone was administered because of severe symptomatic high-grade atrioventricular block requiring temporary pacing and the presumed immune-mediated inflammatory involvement of the conduction system. Evidence supporting corticosteroid therapy in dengue myocarditis remains limited and controversial, and its use should be individualized until further studies become available.

Reports of dengue-associated myocarditis remain scarce in sub-Saharan Africa despite the increasing frequency of dengue outbreaks. Limited access to advanced cardiac imaging probably contributes to under-recognition of this complication. Our experience suggests that systematic cardiac evaluation should be considered in hospitalized dengue patients presenting with chest pain, dyspnea, rhythm disturbances, or unexplained elevations of cardiac biomarkers.

Limitations

This report has several limitations. First, it represents a small single-center case series, limiting the generalizability of the findings. Second, cardiac magnetic resonance imaging was available in only two patients, preventing uniform application of the revised Lake Louise Criteria [17]. Third, endomyocardial biopsy, the diagnostic gold standard for myocarditis, was not performed. Finally, long-term follow-up data were not available, precluding assessment of potential delayed cardiac sequelae. Another limitation is the absence of systematic CMR evaluation in all patients, which may have resulted in underestimation of myocardial involvement. Finally, the descriptive nature of this small case series precludes any causal inference regarding treatment efficacy.

Conclusion

Acute myocarditis is an uncommon but potentially life-threatening complication of dengue infection. Early recognition based on chest pain, electrocardiographic abnormalities, and elevated cardiac biomarkers is essential. Whenever available, cardiac magnetic resonance imaging provides valuable confirmation of the diagnosis. In resource-limited settings, combining clinical assessment, serial troponin measurements, and echocardiography may facilitate early diagnosis and improve patient management. Larger prospective studies are warranted to better define the epidemiology and optimal management of dengue-related myocarditis in sub-Saharan Africa.

Declarations

Ethics approval and consent to participate

This retrospective cohort study was conducted in accordance with the principles of the Declaration of Helsinki.

Consent for publication

Written informed consent was obtained from the patients (or their legal guardians) for the publication of their clinical details and any accompanying images.

Competing interests

The authors declare that they have no competing interests in relation to this manuscript.

Funding

This work received no specific external funding. The research was conducted using the internal resources of the home institution.

Authors' contributions

The authors contributed to the study design, as well as the collection of clinical and echocardiographic data. All authors have read and approved the final version of the manuscript.

Author List & Affiliations

Inès Angoran Regnier

Abidjan Institute of Cardiology

Adama Doumbia

Department of Infectious Diseases, Côte d’Ivoire

Corinne Akpovo

Department of Infectious Diseases, Côte d’Ivoire

Roland N’Guetta

Abidjan Institute of Cardiology

Christophe Konin

Abidjan Institute of Cardiology

Serge Paul Eholie

Department of Infectious Diseases, Côte d’Ivoire

Corresponding Author

Dr. Inès Angoran Regnier

Abidjan Institute of Cardiology

BP V206, Abidjan, Côte d'Ivoire

Tel: +225 0708651127

Email: iangoran@yahoo.fr

References

1. Guzman MG, Harris E, Dengue. Lancet. 2015;385(9966):453–65.
2. Bhatt S, Gething PW, Brady OJ, Messina JP, Farlow AW, Moyes CL, et al. The global distribution and burden of dengue. Nature. 2013;496(7446):504–7.
3. World Health Organization. Dengue guidelines for diagnosis, treatment, prevention and control: new edition. Geneva: World Health Organization. 2009. (WHO/HTM/NTD/DEN/2009.1). Accessed February 19, 2023.
4. World Health Organization, ed. Comprehensive Guidelines for Prevention and Control of Dengue and Dengue Haemorrhagic Fever. Rev. and expanded ed. New Delhi: World Health Organization Regional Office for South-East Asia; 2011.
5. Yacoub S, Wertheim H, Simmons CP, Screaton G, Wills B. Cardiovascular manifestations of the emerging dengue pandemic. Nat Rev Cardiol. 2014;11(6):335–45.
6. Virk HUH, Inayat F, Rahman ZU. Complete heart block in association with dengue hemorrhagic fever. Korean Circ J. 2016;46(6):866–9.
7. Navinan M, Yudhishdran J, Herath S, Rathnatilaka A, Rajapakse S, Sri Ranganathan S. Complete heart block in dengue complicating management of shock due to both bleeding and leakage: a case report. BMC Res Notes. 2015;8(1):68.
8. La-Orkhun V, Supachokchaiwattana P, Lertsapcharoen P, Khongphatthanayothin A. Spectrum of cardiac rhythm abnormalities and heart rate variability during the convalescent stage of dengue virus infection: a Holter study. Ann Trop Paediatr. 2011;31(2):123–8.
9. Li Y, Hu Z, Huang Y, Li J, Hong M, Wang L, et al. Characterization of the Myocarditis during the worst outbreak of dengue infection in China. Medicine. 2016;95(27):e4051.
10. Restrepo BN, Isaza DM, Salazar CL, Ramirez RE, Upegui G, Alzueta A, et al. Serum levels of interleukin-6, tumor necrosis factor-alpha and interferon-gama in infants with and without dengue. Rev Soc Bras Med Trop. 2008;41(1):6–10.
11. Hober D, Delannoy AS, Benyoucef S, De Groote D, Wattre P. High Levels of sTNFR p75 and TNFa in Dengue-Infected Patients. Microbiol Immunol. 1996;40(8):569–73.
12. Miranda CH, Borges M, de Matsuno C, Vilar AK, Gali FC, Volpe LG. Evaluation of cardiac involvement during dengue viral infection. Clin Infect Dis. 2013;57(6):812–9.

13. Schulz-Menger J, Collini V, Gröschel J, Adler Y, Brucato A, Caforio ALP, et al. 2025 ESC Guidelines for the management of myocarditis and pericarditis. Eur Heart J. 2025;46(40):3952–4041.
14. Chen J, Chen S, Li Z, Huang P, He B. Role of electrocardiograms in assessment of severity and analysis of the characteristics of ST elevation in acute myocarditis: a two-centre study. Exp Ther Med. 2020;20(5):1–8.
15. Satarasinghe RL, Arultnithy K, Amerasena NL, Seneviratne SL. Asymptomatic myocardial involvement in acute dengue virus infection in a cohort of adult Sri Lankans admitted to a tertiary referral centre. Ceylon Med J. 2007;52(3):87–90.
16. Doros G, Ardelean A, Olariu C, Popoiu MC, Doros A, Marcovici T. 173 Cardiac biomarkers in diagnosing acute myocarditis in adolescents. Arch Dis Child. 2021;106(2):A73–4.
17. Ferreira VM, Schulz-Menger J, Holmvang G, Kramer CM, Carbone I, Sechtem U, et al. Cardiovascular magnetic resonance in nonischemic myocardial inflammation: expert recommendations. J Am Coll Cardiol. 2018;72(24):3158–76.

Figures

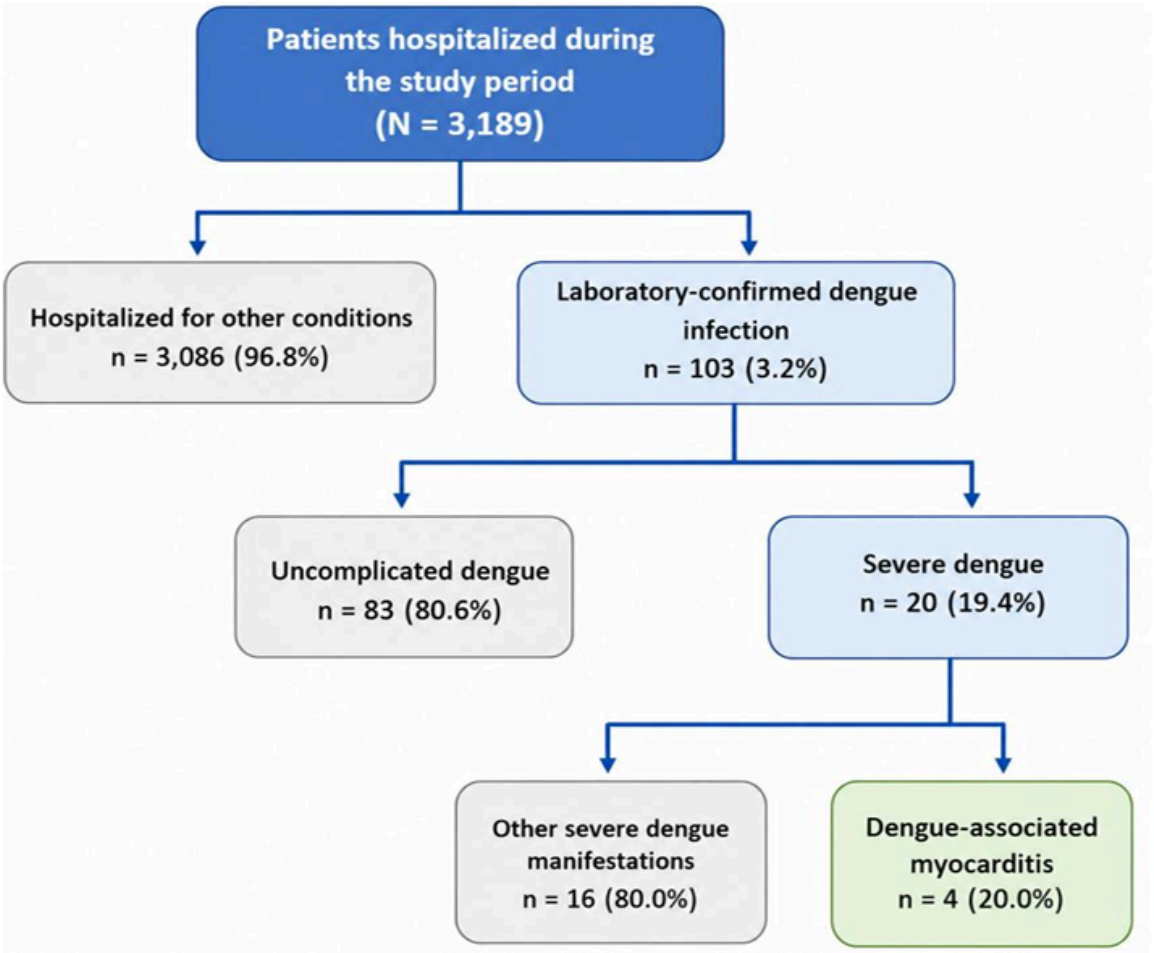


Figure 1

Flow chart of patient selection during the study period.

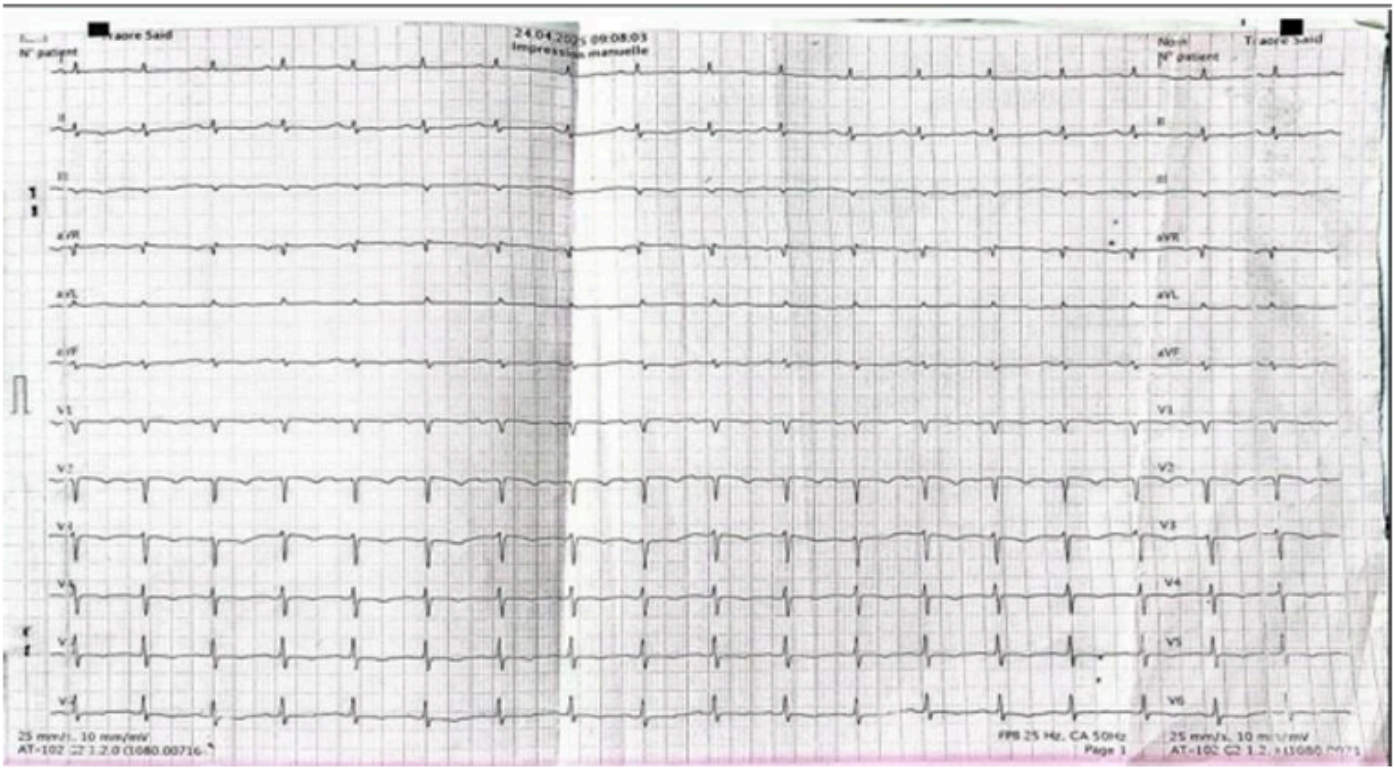


Figure 2

Electrocardiogram: left anterior hemiblock and diffuse low voltage

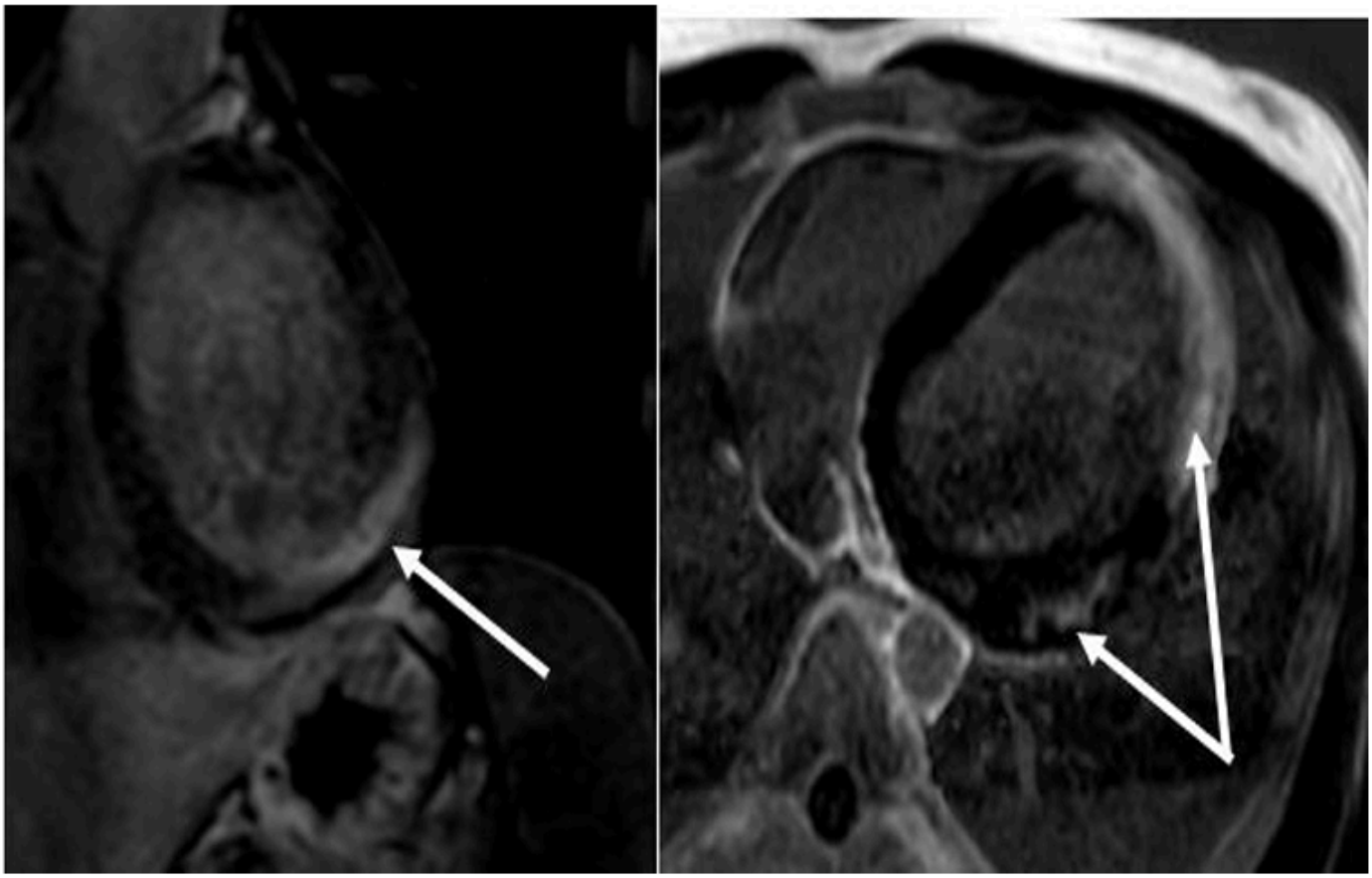


Figure 3

Cardiac magnetic resonance imaging (3 Tesla): subepicardial and intramyocardial late gadolinium enhancement (white arrows)

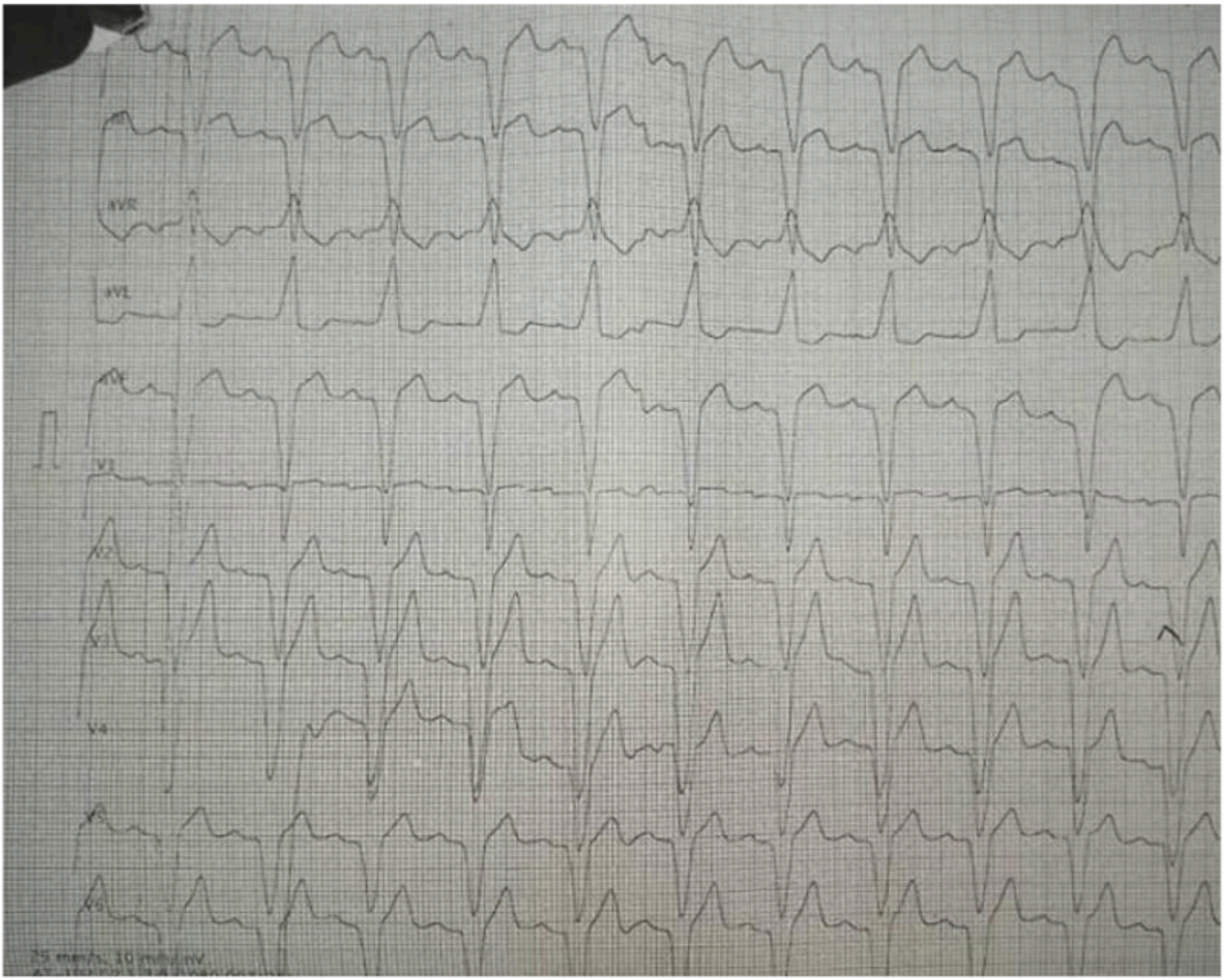


Figure 4

Electrocardiogram: complete left bundle branch block

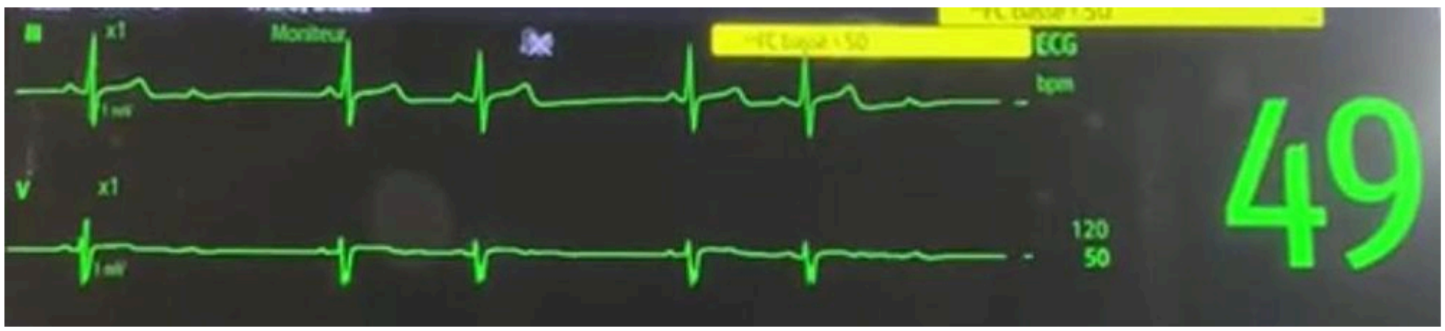


Figure 5

Electrocardiogram: second-degree atrioventricular block

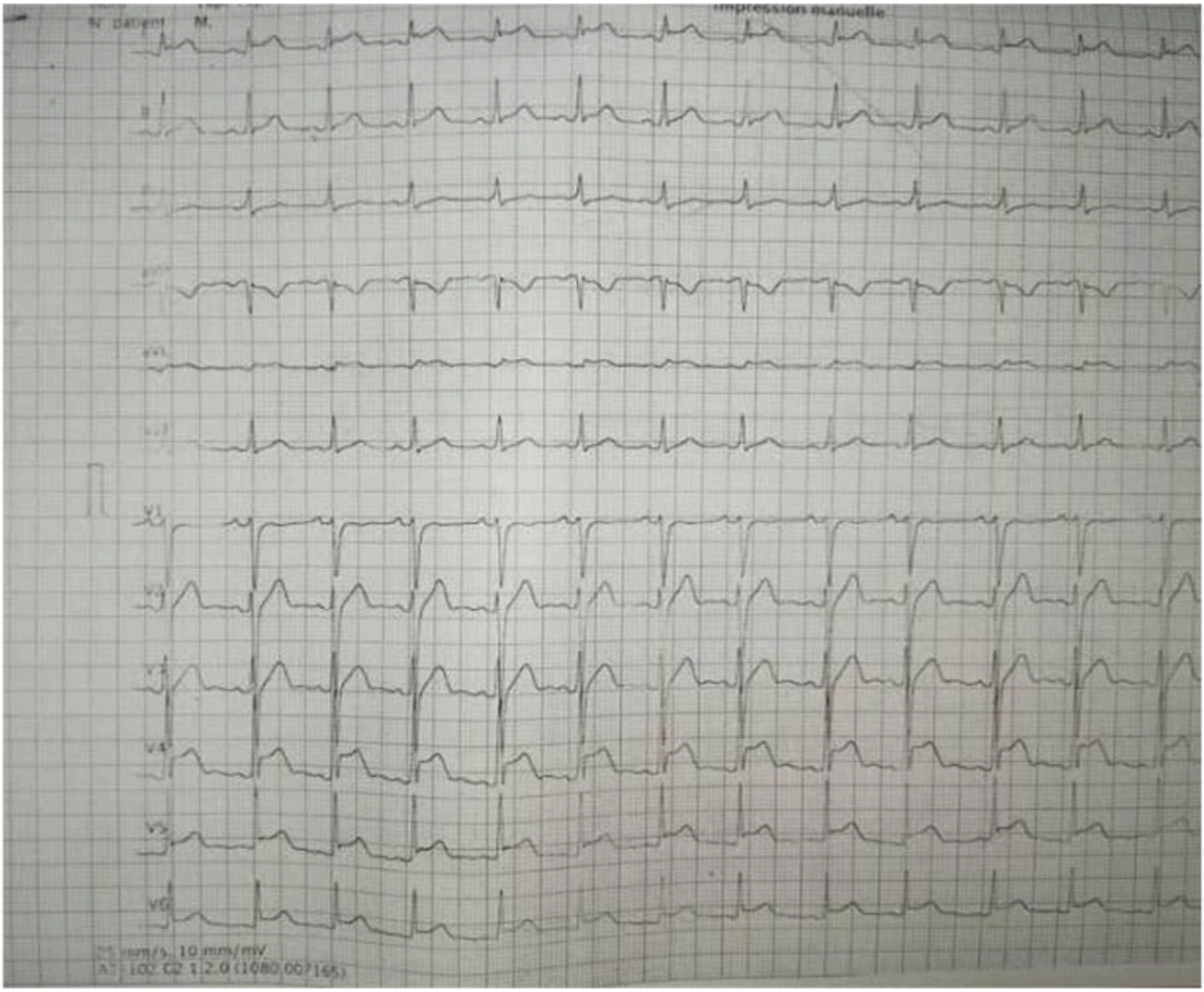


Figure 6

Electrocardiogram showing apicolateral ST-segment elevation

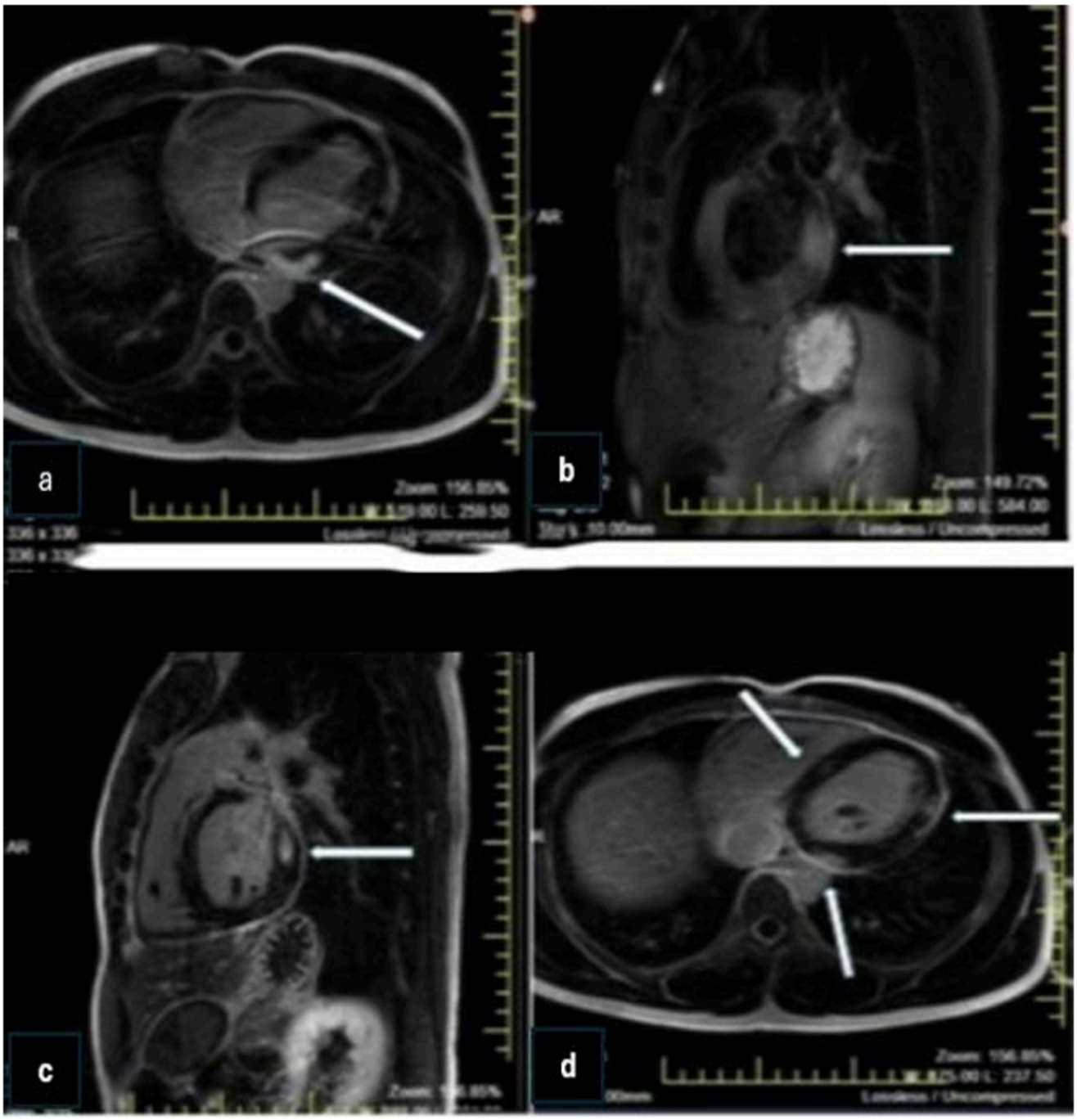


Figure 7

Cardiac magnetic resonance imaging (3 Tesla): subepicardial and intramyocardial late gadolinium enhancement on panels (a), (c) and (d); T2 hyperintensity on panel (b)