



A Blind Spot in Imaging: Immune Checkpoint Inhibitor-Induced Myocarditis with Negative Cardiac MRI

MULTIMODALITY
MUSEUM IMAGE

LUBNA ALNATOUR, MS

AYHAM MAHMOUD, MD

AMEER AWASHRA, MS

JONATHAN NA, MD

MOHAMMED CHAMSI-PASHA, MD

**Author affiliations can be found in the back matter of this article*

HOUSTON
Methodist
DEBAKEY HEART &
VASCULAR CENTER

ABSTRACT

Immune checkpoint inhibitor (ICI)-induced myocarditis is rare but carries a high morbidity and mortality rate. While cardiac magnetic resonance imaging (CMR) is the first-line imaging modality to support diagnosis, the need for endomyocardial biopsy is needed in negative CMR cases.

CORRESPONDING AUTHOR: Mohammed Chamsi-Pasha, MD

Houston Methodist DeBakey
Heart & Vascular Center,
Houston, Texas, US

[machamsi-pasha@
houstonmethodist.org](mailto:machamsi-pasha@houstonmethodist.org)

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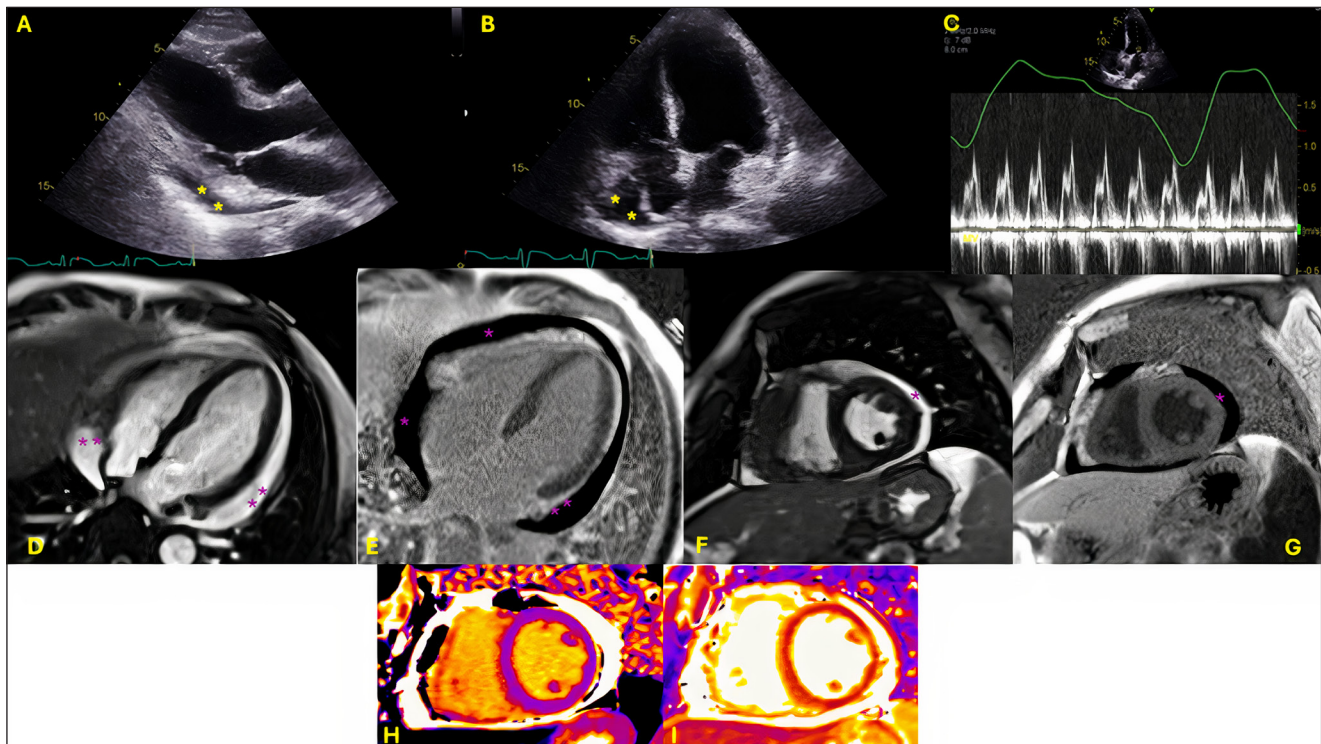


Figure 1 (A) Transthoracic echocardiogram, parasternal long-axis image with small posterior pericardial effusion (asterisk), (B) transthoracic echocardiogram, apical 4-chamber view with anterior pericardial effusion (asterisk). (C) Pulsed wave Doppler across the mitral inflow showing respiratory variation > 25% consistent with increased intrapericardial pressures. (D, F) Cardiovascular magnetic resonance imaging, balanced steady-state free precession sequence in apical 4-chamber (D) and short-axis view (F) showing circumferential pericardial effusion (moderate, asterisk). (E, G) Late gadolinium enhancement imaging suggests nulled myocardium with no evidence of ischemic damage or myocarditis. (H, I) T1 and T2 parametric mapping.

A 57-year-old male with a known case of oral cavity squamous cell carcinoma presented to the emergency department with chest pain. His current chemotherapy regimen included carboplatin, paclitaxel, and pembrolizumab (anti-programmed cell death protein 1 therapy). The patient appeared ill, with resting tachycardia (heart rate of 101 bpm) and mildly elevated jugular venous pressure at 10 cm. Labs showed elevated troponin 336 ng/L, NT-proBNP 252 pg/mL. An electrocardiogram showed sinus tachycardia and no acute ST-T-wave changes (Figure 2 A).

Transthoracic echocardiogram showed preserved ventricular function and small pericardial effusion with respiratory variation across the mitral valve consistent with increased intrapericardial pressures (Figure 1 A–C, Video 1). The hospital course was notable for ongoing myocardial injury (troponin 1185 ng/L), new right bundle branch block (Figure 2 B), and episodes of nonsustained ventricular tachycardia. To rule out other non-myocardial infarction causes of elevated troponin, cardiac magnetic resonance imaging (MRI) was performed, showing borderline reduced systolic function (left ventricle ejection fractions 51%) and moderate (10 mm) circumferential pericardial effusion but with no inflammation (Figure 1 D, F asterisks).

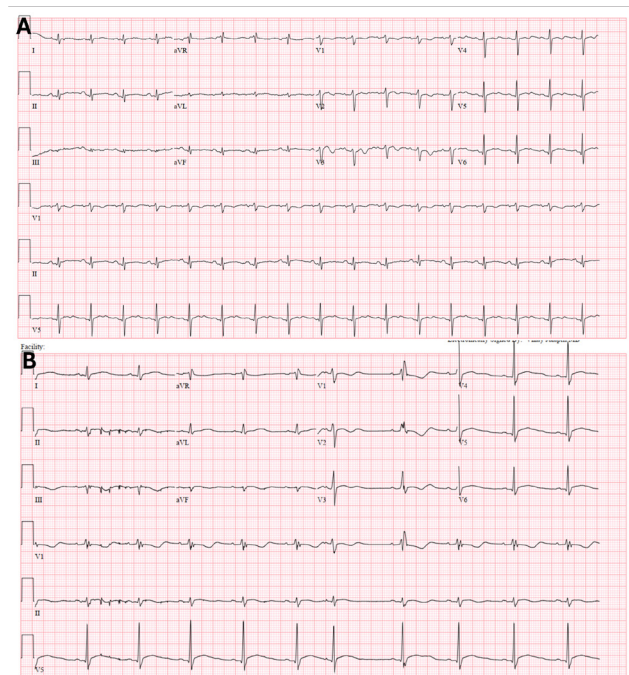
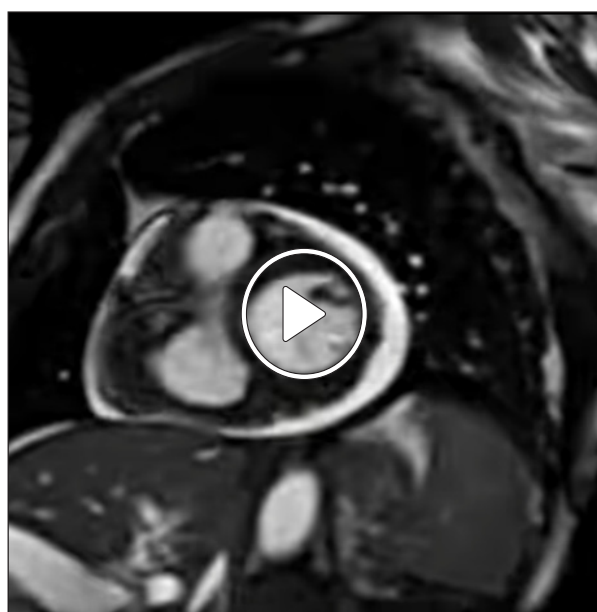


Figure 2 (A) 12-lead electrocardiography showing sinus tachycardia but no acute ST-T wave changes. (B) Subsequent electrocardiogram showing interval development of right bundle branch block.

There was no evidence of late gadolinium enhancement suggesting absence of infarction, acute injury, or myocarditis (Figure 1 E, G). In addition, the T1 and T2 relaxation times were normal on parametric mapping (Figure 1 H, I). Given the possibility of immune-checkpoint inhibitor (ICI)-myocarditis, the patient underwent an endomyocardial biopsy, which showed increased perivascular and interstitial



Video 1 Cine steady-state free precession cardiac MRI sequence (real-time imaging) in short axis stacks across the LV, 4-chamber view, 2-chamber view, and 3-chamber view. There is circumferential pericardial effusion but no evidence of chamber collapse. The left ventricular function is borderline reduced; also view at <https://youtube.com/shorts/mFHytiYCu5k>.

CD3 and CD8 positive T cells, findings consistent with ICI-myocarditis (Figure 3). The patient completed five doses of pulsed intravenous steroids and was discharged home on 60 mg prednisone with weekly taper. Immunotherapy was permanently discontinued.

First reported in 2016 and infrequently encountered,¹ this case demonstrates the need for increased awareness of ICI myocarditis, which carries a 40% risk of morbidity and mortality. Early diagnosis with interruption of ICI and initiation of immunosuppression is key to improving outcomes.²

COMPETING INTERESTS

The authors have no competing interests to declare.

AUTHOR AFFILIATIONS

Lubna Alnatour, MS  orcid.org/0009-0008-5669-5899

School of Medicine, University of Jordan, Amman, Jordan

Ayham Mahmoud, MD  orcid.org/0009-0008-5414-9018

School of Medicine, The University of Jordan, Amman, Jordan

Ameer Awashra, MS  orcid.org/0009-0002-5122-8200

An Najah National University, Nablus, Palestine

Jonathan Na, MD  orcid.org/0000-0002-5061-7493

Houston Methodist, Houston, Texas, US

Mohammed Chamsi-Pasha, MD  orcid.org/0000-0002-1445-4789

Houston Methodist DeBakey Heart & Vascular Center, Houston, Texas, US

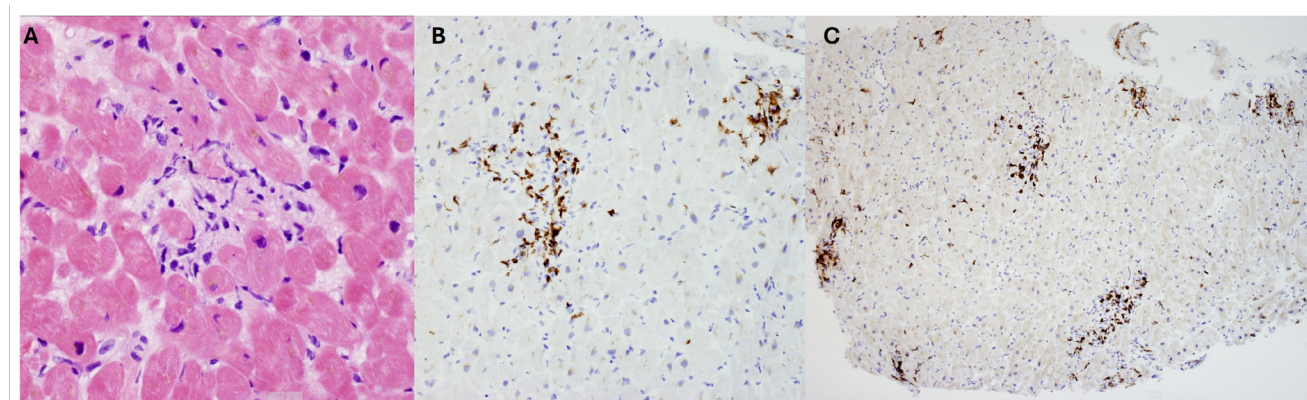


Figure 3 (A) Hematoxylin and eosin stain showing increase perivascular and interstitial CD3 positive (B) and CD8 (C) T-cells, findings compatible with immune checkpoint inhibitor-associated myocarditis.

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