

## Loeffler's Endocarditis Unveils Underlying Eosinophilic Granulomatosis With Polyangiitis

A 42-year-old female with a history of asthma presented to the emergency department with chest tightness and shortness of breath. Her electrocardiogram showed diffuse ST-segment depression with reciprocal elevation in lead aVR (Figure 1A). Physical examination revealed petechiae and purpura on the back (Figure 1B), and troponin was markedly elevated (51687 ng/L; reference <16 ng/L), prompting admission for suspected non-ST elevation myocardial infarction. Laboratory results showed leukocytosis ( $14.2 \times 10^9/L$ ) with significant eosinophilia (41.1%) and elevated C-reactive protein (82.8 mg/L). Coronary angiography revealed normal coronary arteries. Transthoracic echocardiography demonstrated a large, non-perfused mass occupying the apical and mid-cavity segments of the left ventricle (Figure 1C and Video 1), with regional wall motion abnormalities. Further history uncovered chronic sinusitis and polyneuropathy. Chest and paranasal computed tomography revealed diffuse ground-glass opacities and chronic ethmoid sinusitis (Figure 1D and 1E). Despite a normal neurological exam, brain magnetic resonance imaging identified multiple small cortical and subcortical infarcts (Figure 1F). Stool parasite examination and autoimmune serologies including cytoplasmic antineutrophil cytoplasmic antibody, and perinuclear antineutrophil cytoplasmic antibody were negative. Multidisciplinary evaluation integrating clinical, radiological, and laboratory findings supported a diagnosis of eosinophilic granulomatosis with polyangiitis (Churg–Strauss syndrome) complicated by Loeffler's endocarditis. The patient was treated with intravenous methylprednisolone (1000 mg/day for 3 days), followed by oral prednisone (1 mg/kg/day), and anticoagulated with heparin, later switched to warfarin. She showed marked improvement, with normalization of eosinophil count, inflammatory markers, and cardiac function.

Multimodality imaging played a pivotal role in unveiling Loeffler's endocarditis as the initial cardiac manifestation of eosinophilic granulomatosis with polyangiitis.

**Informed Consent:** Written informed consent was obtained from the patient.

**Declaration of Interests:** The authors have no conflicts of interest to declare.

**Funding:** The authors declare that this study received no financial support.

**Video 1:** Apical 4-chamber view on transthoracic echocardiography demonstrating a large, immobile, non-perfused mass occupying the apical and mid-cavity segments of the left ventricle, consistent with Loeffler's endocarditis.

### E-PAGE ORIGINAL IMAGE



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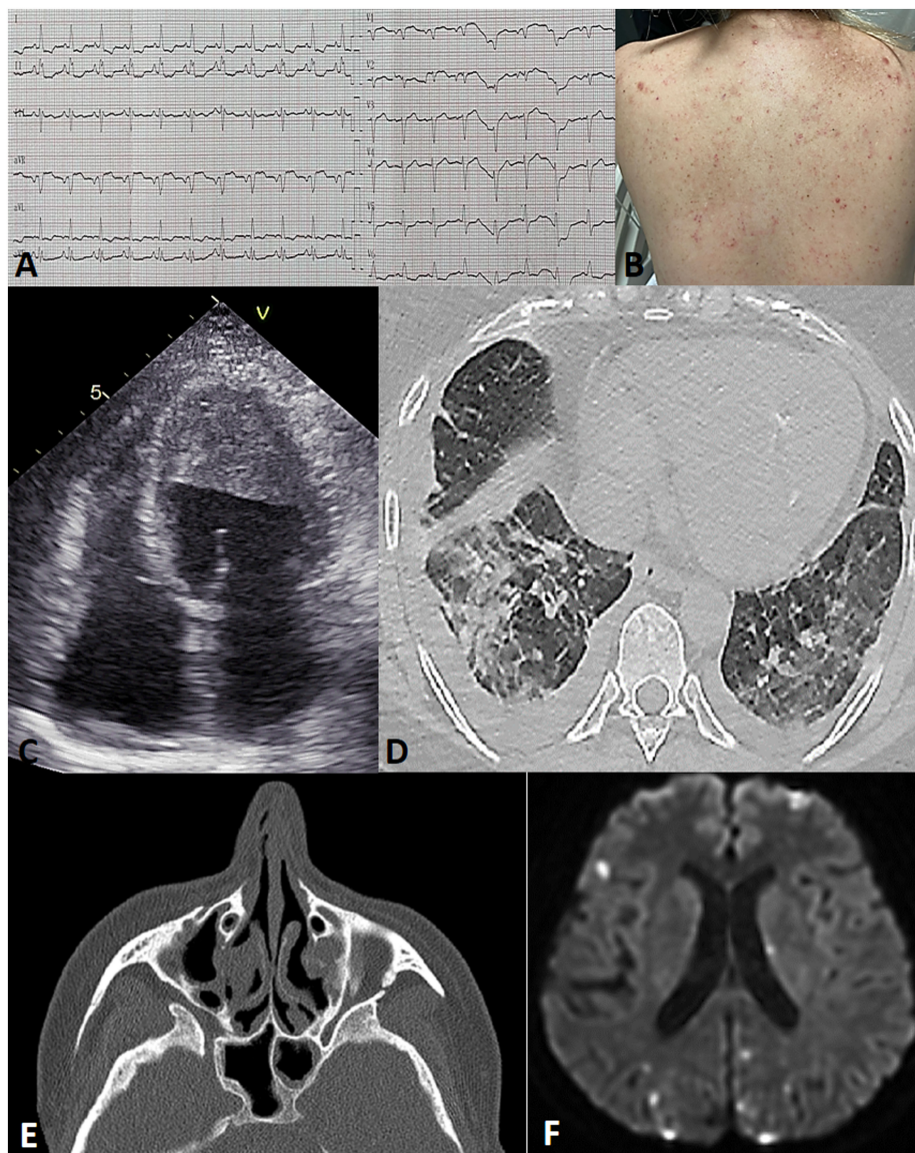
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**Cite this article as:** Kodali B, Bezgin T, Çağdaş M, İnan Çelik A. Loeffler's endocarditis unveils underlying eosinophilic granulomatosis with polyangiitis. *Anatol J Cardiol.* 2026;30(1):E-3-E-4.



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DOI:10.14744/AnatolJCardiol.2025.5708



**Figure 1.** (A) Electrocardiogram on admission showing diffuse ST-segment depression in leads I, II, III, aVL, aVF, and V5–V6, with reciprocal ST-segment elevation in lead aVR. (B) The patient's back demonstrating petechiae and purpuric lesions. (C) Transthoracic echocardiogram (apical 4-chamber view) revealing a large, non-perfused mass occupying the apical and mid-cavity segments of the left ventricle, consistent with Loeffler's endocarditis. (D) Axial view of the chest computed tomography (CT) showing bilateral diffuse ground-glass opacities suggestive of eosinophilic pulmonary involvement. (E) Axial CT image of the paranasal sinuses demonstrating chronic ethmoid sinusitis. (F) Brain diffusion-weighted magnetic resonance imaging (MRI) showing multiple acute cortical and subcortical infarcts in both cerebral hemispheres, consistent with systemic embolization.