[Image focus]

Cardiac involvement in hypereosinophilic syndrome

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A 67-year-old male presented with a four-week history of aggravating exertional dyspnoea (NYHA III), orthopnoea and clinical signs of heart failure. Laboratory studies showed a white blood cell count of 9.1×10^9/L, of which 1.96×10^9/L were eosinophils (21.5%). Secondary causes of eosinophilia were excluded; cytogenetic analysis demonstrated no FIP1L1-PDGFRA gene fusion, reducing the probability of a myeloproliferative variant. The ECG revealed sinus rhythm, with abnormal R-progression in the precordial leads ($|R|\ V_4 > |R|\ V_5$) and T wave inversions in leads V_4-V_6. On transthoracic echocardiography (TTE), the left ventricle (LV) showed a slightly reduced ejection fraction (EF: 47%, modified Simpson's method), a restrictive filling pattern (E/A > 2.0 on PW-mitral inflow, e' 4 cm/s, E/e' septal 21 on Doppler tissue imaging) and apical hypokinesis. The LV apex was obliterated with an added fenestrated structure, showing no contrast captation on contrast echocardiography (figure 1). Post-gadolinium cardiac magnetic resonance imaging (MRI) showed a hypo-intense intracavitary structure on the delayed enhancement images, demarcated by shallow subendocardial hyperenhancement (figure 1) suggestive for intracavitary thrombus with endomyocardial fibrosis. The TTE and cardiac MRI findings met the diagnostic criteria for the second thrombotic stage of idiopathic hypereosinophilic syndrome (HES). The patient was treated with diuretics, corticosteroids and fenprocoumon (target INR 3); leading to significant clinical amelioration, biochemical normalization of the hypereosinophilia, and echocardiographic transition to diastolic dysfunction grade I without remaining congestion after 4 weeks, further supporting the initial diagnosis of HES.

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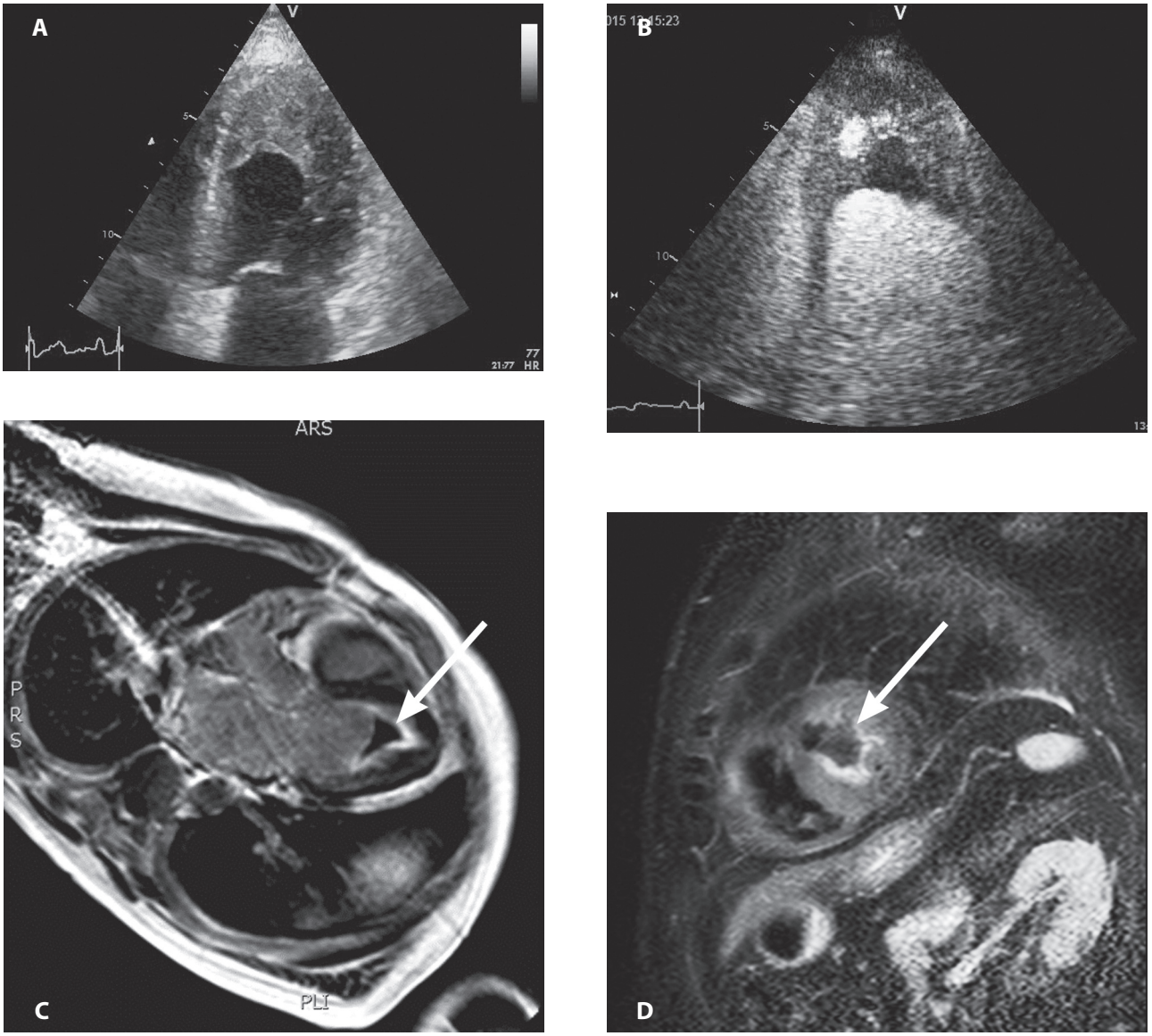

Fig. 1 (**A**) Transthoracic echocardiography (A4C view), showing LV apical obliteration by an added structure. (**B**) Contrast echocardiography illustrating presence of fenestrations in the structure with associated contrast stasis, but no evidence of contrast captation by the structure itself. (**C**) Four-chamber view cardiac MRI with delayed gadolinium enhancement showing evidence of intracavitary hypo-intense added structure (arrow), surrounded by a hyper-intense halo. (**D**) Short-axis T2-weighted images with fat suppression showing strong hypo-intense thrombus intraluminal (arrow). LV: left ventricle. MRI: magnetic resonance imaging.