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**CLINICAL VIGNETTE**

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**Splenatic infarction due to multiple left ventricular mobile thrombi in hypereosinophilic endomyocarditis**

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A 30-year-old man was admitted for rapidly developing dyspnoea. He was a basketball player and had not felt chest discomfort until a few days earlier. A chest radiograph showed severe pulmonary congestion and the electrocardiogram revealed sinus tachycardia with ST-segment depression and inverted T waves in inferior leads. Echocardiography demonstrated increased left ventricular (LV) wall thickness and restrictive transmitral flow pattern. We could also observe severe spontaneous echo contrast and multiple mobile thrombi in the LV (Panels A and B). Despite the immediate anticoagulation therapy, these thrombi did not disappear. On day 7, he revealed left upper abdominal pain and computerized tomography scan showed a thrombo-embolic splenic infarction (Panel C). Total blood eosinophil counts incrementally increased and endomyocardial biopsy taken from the right ventricle showed eosinophilic infiltration predominantly in endocardium but also in myocardium, resulting in geographic loss or sporadic damage of myocardial fibres in association with granulation tissue proliferation and fibrosis (Panel D).

The clinical and pathological findings suggested the acute necrotic stage of hypereosinophilic endomyocarditis. Steroids therapy provided a dramatic improvement of clinical and echocardiographic findings. However, recurrent inflammation occurred when we reduced the dose of oral prednisolone.

Panels A and B. Echocardiographic images showing spontaneous echo contrast in LV lumen and multiple mobile mural thrombus (arrows).

Panel C. Computerized tomography scan showing a splenic infarction (arrow).

Panel D. High power view of myometrium showing sporadic degeneration of myocytes in association with severe eosinophilic infiltration, fibrosis, and small number of Langhans giant cells (arrow) (haematoxylin and eosin stain, × 400).

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