CLINICAL IMAGE

# Idiopathic left ventricular subendocardial dissecting hematoma

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A 47-year-old woman, previously not treated for chronic diseases, was admitted to the hospital in a severe condition and with symptoms of New York Heart Association class IV heart failure (HF) (dyspnea at rest, peripheral edema). The results of laboratory tests showed increased levels of inflammatory parameters (white blood cell count, $17.63 \times 10^9$/l; reference range, $4.0–10.0 \times 10^9$/l; C-reactive protein, 126.36 mg/l; reference range, 0.0–5.0 mg/l; procalcitonin, 0.85 ng/ml; reference range <0.5 ng/ml), and myocardial necrosis (troponin, 111 ng/l; reference range <47 ng/l). The peripheral eosinophil count was within the reference range.

Transthoracic echocardiography showed enlargement of the heart cavities, in particular of the left ventricle (LV), akinesis of the interventricular septum, severe impairment of the ejection fraction estimated at 25%, thickening of the LV lateral wall, and LV intramyocardial dissection (in the form of a free-floating membrane) with the formation of an echolucent space between the endocardium and the myocardium. However, on color Doppler imaging, a connection between the created space and the LV cavity was not visible and the pericardium did not show pathological amount of fluid (**FIGURE 1A** and **1B**; Supplementary material, *Figure S2A–S2L*).

A computed tomography scan of the heart showed thickening of the free wall of the LV from the subendocardial side (15–37 mm). Additionally, it revealed an uneven outline to the lumen side and uneven density. A significant reduction in the subendocardial and medial density of the muscle with impaired contractility was visualized. Of note, multifocal lesions on the surface were observed, indicating thrombus formation. Nonobstructive coronary atherosclerotic lesions were visible in the coronary arteries (**FIGURE 1C** and **1D**; Supplementary material, *Figure S1A–S1C*). Coronary angiography was not performed since there were no electrical alternans on electrocardiography associated with ST-segment elevation myocardial infarction (STEMI).

Because of the critical condition of the patient, transfer to a medical center with experience in performing endomyocardial biopsy and expertise in cardiac pathology was not possible. According to current guidelines, cardiac magnetic resonance imaging (MRI) is an important diagnostic method, especially when biopsy is unavailable.[1] Therefore, once clinical stabilization of the patient was achieved during hospitalization, an MRI examination was performed, revealing LV enlargement and global LV hypokinesia with significantly impaired ejection fraction. Cardiac MRI showed diffuse myocardial edema and foci of subendocardial and mid-wall late gadolinium enhancement (LGE), which may indicate myocarditis. The presence of LGE was also due to subendocardial dissection. An extensive thrombus was visualized at the side of the LV lateral wall (**FIGURE 1E** and **1F**; Supplementary material, *Figure S1D–S1I*).

Moreover, owing to symptoms of respiratory failure, the patient required intubation and ventilator therapy. Under hemodynamic monitoring, catecholamines and levosimendan were infused. Due to accompanying generalized infection (pneumonia, sepsis) and thrombocytopenia, no attempt was made to support the circulation mechanically. Continuous renal replacement therapy was administered to reduce overhydration. Standard pharmacological treatment of HF was introduced, while methylprednisolone infusions were used as anti-inflammatory treatment.

Subsequent echocardiographic examinations did not show any intramyocardial dissection, but a thrombus in this location was visualized, most

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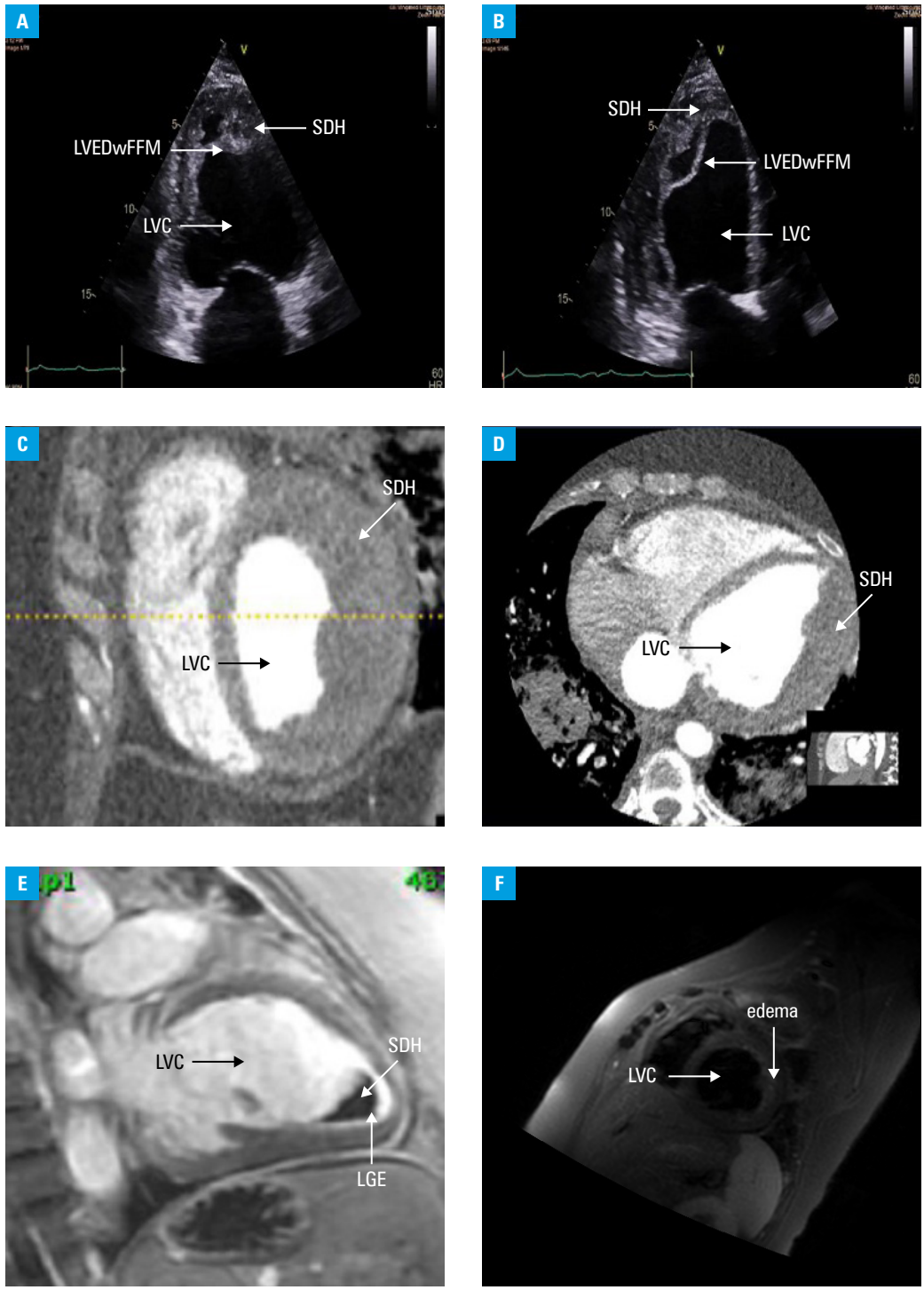


**FIGURE 1** **A**, **B** – transthoracic echocardiography visualizing an intramyocardial dissection of the anterolateral left ventricular wall with the formation of a space containing hematoma; modified 2-dimensional apical view during diastole (**A**) and systole (**B**); **C**, **D** – computed tomography angiography images showing a massive subendocardial dissecting hematoma (**C** – short axis view; **D** – 4-chamber axial view); **E** – magnetic resonance imaging showing the subendocardial dissecting hematoma and late gadolinium enhancement (2-chamber view during systole); **F** – magnetic resonance imaging showing a T2-sequence abnormality—edema (short axis view during systole)
Abbreviations: LGE, late gadolinium enhancement; LVC, left ventricular cavity; LVEDwFFM, left ventricular endocardium dissection with a free-floating membrane; SDH, subendocardial dissecting hematoma

likely stabilizing the previous dissection (Supplementary material, *Figure S2M–S2P*).

Hemorrhagic dissection of the LV wall with the formation of intramyocardial dissecting hematoma (IDH) occurs as a result of blood extravasation between myocardial spiral fibers due to the damage to capillary cells. IDH may affect the free wall of the LV, the interventricular septum, or the right ventricular wall.[2,3] This complication most often concerns patients with STEMI, with a frequency below 0.4%. Other causes mentioned in the literature are injuries

or complications of percutaneous interventions. Importantly, IDH can be mistaken for an LV lumen thrombus.[4] Consequently, introduction of anticoagulant therapy may worsen the course of IDH with a possible rupture of the free wall of the heart muscle.[5] The presented case describes an extremely rare kind of LV wall dissection. Although there was a high clinical suspicion of myocarditis, in the absence od endomyocardial biopsy, its definitive diagnosis was not possible. This case illustrates the importance of multimodality imaging in the diagnosis of IDH.

## SUPPLEMENTARY MATERIAL

Supplementary material is available at www.mp.pl/paim.

## ARTICLE INFORMATION

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