

# Intramycardial haemorrhage as a rare complication of myocardial infarction — the diagnostic value of cardiovascular magnetic resonance imaging

Krwotok w obrębie mięśnia sercowego jako rzadkie powikłanie zawału serca — diagnostyczna wartość rezonansu magnetycznego układu sercowo-naczyniowego

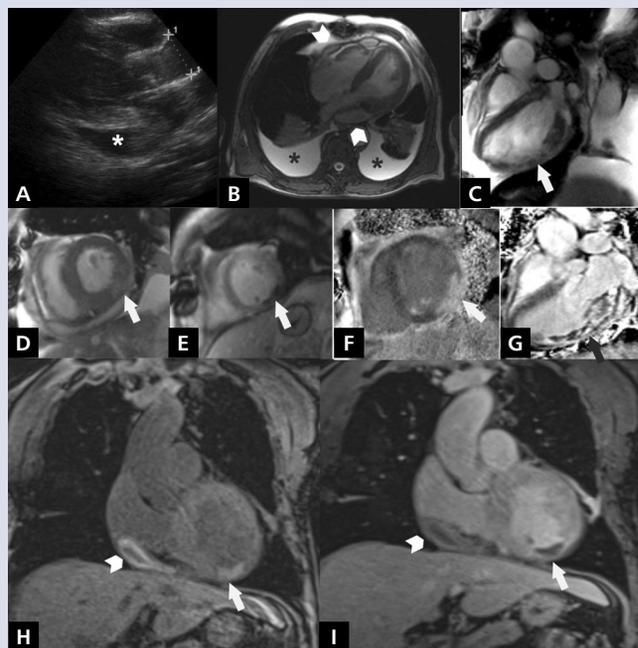
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A 77-year-old man with a one-month history of Q wave inferior-inferolateral ST segment elevation myocardial infarction (STEMI) was referred to our hospital for coronary artery bypass grafting (CABG). STEMI was treated conservatively because of delayed presentation (> 48 h after acute episode). Coronary angiography at this time revealed occlusion of obtuse marginal (OM) II (culprit artery), stenosis in the left anterior descending artery (LAD) of 80%, right coronary artery (RCA) of 90%, and OM I of 50%. On admission to our hospital, electrocardiogram (ECG) showed sinus rhythm with Q waves and ST segment elevation in leads III and AVF. On chest X-ray, cardiac enlargement was noticed. Transthoracic echocardiography revealed hypokinesis in the inferior and inferolateral left ventricular (LV) walls with the separation of pericardial layers up to 25 mm with fluid in-between (Fig. 1A, asterisk) together with bilateral pleural effusion and a markedly reduced LV ejection fraction of 35%. Pericardial and bilateral pleural effusions were considered as signs of congestion due to LV dysfunction. The patient was directed to CABG, but after sternotomy and pericardiectomy thickened pericardium and blood clots in the pericardial sac at the right ventricular (RV) free-wall and LV inferior wall without clear signs of myocardial rupture were found. The decision to perform drainage of the pericardial sac with deferment of the CABG due to the increased bleeding risk was made. 1.5 T cardiac magnetic resonance (CMR) imaging was performed for clarification of the diagnosis. CMR revealed microvascular obstruction with intramyocardial haemorrhage (IMH) in the infarcted LV inferolateral wall without rupture (Fig. 1B–I). After uneventful course, the patient was scheduled to percutaneous coronary intervention. We believe that the haemopericardium was due to myocardial necrosis that transformed into IMH with subsequent micro bleeds into the pericardial space.



**Figure 1.** **A.** Transthoracic echocardiography (TTE) (parasternal long-axis view) shows separation of pericardial layers up to 25 mm (white asterisk); **B.** Cardiac magnetic resonance (CMR) (cine four-chamber view) shows bilateral pleural effusion (black asterisks) and pericardial effusion (arrowheads); **C.** CMR (cine three-chamber heart view) shows irregular myocardium of inferolateral wall (arrow); **D.** CMR (cine short axis view) shows infarcted, irregular, and oedematous inferolateral wall (arrow); **E.** Perfusion CMR sequence shows subendocardial hypoperfusion in the inferolateral wall (arrow); **F.**  $T_1$  inversion recovery MR sequence (short axis view) showing bright signal corresponding with myocardial necrosis (arrow); **G.** The same sequence as in panel F (three-chamber heart view) shows microvascular obstruction (MVO) in the inferolateral left ventricular (LV) wall (black arrow); **H.** On native  $T_1$  vibe CMR sequence with fat suppression hyperintense area (arrow) consistent with intramyocardial haemorrhage (IMH) is seen, pericardium has hyperintense signal due to fibrin on pericardial layers with darker signal of fluid in-between (arrowhead); **I.** Post contrast  $T_1$  vibe MR sequence with fat suppression shows hypoperfusion, consistent with MVO and IMH, within the hyperintense scar (arrow), and the pericardial sac is not enhancing — there is no direct communication between LV and pericardial sac (arrowhead)

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**Conflict of interest:** none declared

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