

IMAGE CHALLENGE

Connection between the heart and the gut

CLINICAL INTRODUCTION

A 45-year-old man with ulcerative colitis was admitted with bloody diarrhoea and chest pain. Inflammatory markers and high-sensitivity troponin were elevated (C reactive protein 57 mg/L, white cell count $10.65 \times 10^9/L$, neutrophil $6.6 \times 10^9/L$, Troponin-I 663 nmol/L). The ECG showed inferior ST-elevation. Urgent coronary angiography revealed unobstructed coronary arteries. Inpatient cardiovascular magnetic resonance (CMR) was arranged to determine the aetiology of the myocardial infarction with non-obstructive coronary arteries. The imaging protocol at 1.5 T included balanced steady-state free precession cine images, T2-weighted oedema sequences, and early and late gadolinium enhancement (LGE). Native T1 and T2 mapping images provided advanced tissue characterisation (figure 1).

QUESTION

What is the most likely diagnosis based on the MRI findings?

- Multiple embolic myocardial infarctions in the right coronary artery territory.
- Acute autoimmune myocarditis.

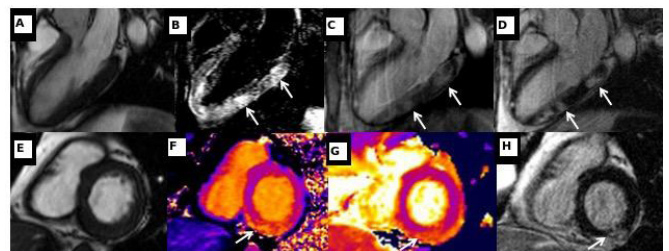


Figure 1 (A) Balanced steady-state free precession (bSSFP) left ventricular long-axis, three-chamber view. (B) T2 short-tau inversion recovery. (C) Early gadolinium enhancement demonstrating high signal intensity indicative of hyperaemia with capillary leakage (arrowed). (D) Late gadolinium enhancement with high signal intensity indicative of increased extracellular space (arrowed). (E) bSSFP left ventricular short-axis view. (F) Native myocardial T1 mapping with elevated native T1 mapping values in the inferior wall (arrowed). (G) Native myocardial T2 mapping with elevated native T2 values in the inferior wall, indicative of oedema (arrowed). (H) Late gadolinium enhancement with high signal intensity indicative of increased extracellular space (arrowed).

- Cardiac sarcoidosis.
- Stress (Takotsubo) cardiomyopathy.
- Multiple embolic myocardial infarctions in the left circumflex coronary artery territory.

For answer see page 1196

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For question see page 1148

ANSWER B

Multiple patchy, mid-myocardial, high-intensity signal areas in the inferolateral wall on T2-weighted images (B) and increased T2 mapping values on the short-axis images (G) are highly specific for oedema. These areas have high signal intensity on early (C) and late (D,H) sequences, suggesting hyperaemia with capillary leakage and increased extracellular space, respectively. Furthermore, increased native T1 mapping values in all layers but predominantly in the subepicardium of the entire lateral wall (F) are in keeping with extensive myocardial injury (figure 1). Taken in combination, these findings meet the updated Lake Louise criteria for acute myocarditis.¹ Despite extensive oedema, stress (Takotsubo) cardiomyopathy (option D) can be excluded by ventricular morphology and the presence of LGE,² and multiple embolic myocardial infarctions (options A and E) are excluded by the non-ischaemic pattern of LGE.³ Finally, based on the CMR, cardiac sarcoidosis (option C) cannot be easily distinguished from acute autoimmune myocarditis. However, the clinical presentation and absence of pulmonary changes make the latter diagnosis the most likely.⁴ Cardiac biopsy might definitively differentiate, but is not routinely performed due to associated procedural risk. The patient was treated with corticosteroids and monoclonal antibodies and responded well. The pathophysiological mechanisms underlying cardiac manifestations of inflammatory bowel disease are incompletely understood but may occur in up to one-third of patients with inflammatory bowel disease.⁵

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