Acute myocardial injury from carbon monoxide poisoning by cardiac magnetic resonance imaging

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A 40-year-old patient presented to our emergency department with altered mental status and supraventricular tachycardia. His carboxyhaemoglobin level was detected at 14%. He was found to have developed myocardial injury with a typical troponin rise and fall peaking at 22.6 ng/mL. Coronary angiography demonstrated normal coronary arteries. A battery of inflammatory and viral serologies was also negative. Cardiac magnetic resonance (CMR) was performed to evaluate the patient’s left ventricular function and myocardial damage. Steady-state free-precision cine demonstrated a mildly depressed left ventricular systolic function (ejection fraction of 54%) with hypokinesis of the anterior wall and regional akinesis of the inferior wall (Supplementary data online, Videos S1 and S2). First-pass perfusion demonstrated even perfusion throughout all wall segments (Supplementary data online, Video S3). Late gadolinium-enhancement images demonstrated multiple focal areas of high signal consistent with myocardial necrosis or fibrosis (Panels A–D).

Carbon monoxide (CO) inhibits oxygen delivery and subsequently causes ischaemic changes that can ultimately lead to multiorgan failure and death. Myocardial injury is a significant predictor of mortality in patients hospitalized with sequelae of CO poisoning. Myocardial injury from severe CO poisoning detected by CMR has been described in a previous case report (Henry TD, Lesser JR, Satran D. Myocardial fibrosis from severe carbon monoxide poisoning detected by cardiac magnetic resonance imaging. Circulation. 2008;118(7):792), in which CMR was performed months after exposure. Here, we present a case of CO poisoning that likely resulted in acute myocardial necrosis, demonstrating another type of myocardial injury that can be detected by CMR. Although T2-weighted images could not be performed due to the patient’s concurrent rhythm disorder, the typical rise and fall of troponin was highly suggestive of acute injury, and CO toxicity was the most likely cause of the observed CMR abnormalities.

Panel A. Late gadolinium-enhancement (LGE) image in the three-chamber view demonstrating a very small focal area of enhancement in the mid-anterior septum.
Panel B. LGE in the four-chamber view demonstrating a small region of subendocardial enhancement in the anterolateral wall.
Panel C. LGE image in the two-chamber view demonstrating heterogeneous patchy enhancement in the inferior wall.
Panel D. LGE image in the apical short-axis view demonstrating mid-wall enhancement in the anterior wall.

Supplementary data are available at European Heart Journal – Cardiovascular Imaging online.

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