Multivessel myocardial infarction

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Figure 1. Panel A. Electrocardiogram showing an acute apical and lateral myocardial infarction. Panel B. Coronary angiogram: occlusion of circumflex (Cx), obtuse margin (OM) artery (white arrows), left anterior descending (LAD) and first diagonal (D1) artery (black arrows). Panel C. Follow-up ECG: Q waves and R-wave loss in multiple leads and in addition showing incomplete ST-segment recovery (persistent ST-segment elevation). Panel D. T1-weighted inversion recovery short-axis image, 10 minutes after injection of 0.2 mmol/kg gadolinium showing transmurular hyperenhancement of the mid anterolateral and inferolateral segments (white arrows), with a subendocardial hypoenhanced core consistent with microvascular obstruction (black arrow).

A 56-year-old female patient with hypertension, obesity and chronic intermittent cauda equina compression suffered an acute myocardial infarction five

days after a lumbar hernia operation. The electrocardiogram (ECG) showed ST-segment elevation in multiple leads, consistent with an extensive acute apical and lateral myocardial infarction (figure 1, panel A). Acute coronary angiography revealed occlusion of the end-arteries of the left coronary artery in the absence of significant atherosclerotic disease (figure 1, panel B). Neither thrombosuction nor balloon dilatation were successful. Due to the recent operation, only acetylsalicylic acid, low-molecular-weight heparin and clopidogrel were given. The electrocardiogram 24 hours later showed signs of extensive myocardial damage, with loss of R waves, the occurrence of Q waves in multiple leads and incomplete ST-segment recovery, consistent with low or absent myocardial perfusion (figure 1, panel C). Cardiac magnetic resonance imaging five days later revealed pericardial effusion and akinesia of the mid anterolateral and inferolateral segments (movie).

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A large region without contrast enhancement within transmurally infarcted segments can be seen on late enhancement imaging, indicative of microvascular obstruction (figure 1, panel D).

The overall prevalence of myocardial infarction in patients with otherwise angiographically normal coronary arteries is approximately 3%. The aetiology,

predominantly vasospasm and acquired or inherited coagulation disorders, can be found in only one third of patients despite a systematic search.¹ Other proposed mechanisms are cocaine abuse, increased inflammatory state, embolism, endothelial dysfunction or extraluminal coronary artery disease.² Despite a thorough search, the exact cause is suggestive of thromboembolism but remains a mystery in our case.

Moving images illustrating akinesia of the anterolateral and inferolateral segments are shown on the journal's website at www.cardiologie.nl. ■

In this section a remarkable 'image' is presented and a short comment is given.

We invite you to send in images (in triplicate) with a short comment (one page at the most) to Bohn Stafleu van Loghum, PO Box 246, 3990 GA Houten, e-mail: l.jagers@bsl.nl.

'Moving images' are also welcomed and (after acceptance) will be published as a Web Site Feature and shown on our website: www.cardiologie.nl

This section is edited by M.J.M. Cramer and J.J. Bax.

References

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