gene transcription (RVX-208), nuclear hormone receptor agonists, HDL delipidation, whole HDL particle infusion with CER-001, MDCO-216, or CSL-112, and gene therapy using HDL-related proteins such as Apo A-I.\textsuperscript{5,21} Further clinical evaluation of direct infusion of mutant (Apo A-1 Milano) or wild-type Apo A-1, linked with a phospholipid carrier, appears warranted since their vascular benefits have been repeatedly demonstrated in animal models and small clinical studies.\textsuperscript{5,21} Despite well-established cardiovascular benefits of statins in CAD, there remains a substantial residual CHD risk which may be mitigated by the right HDL-based intervention and, therefore, we should not give up on HDL despite its split personality.

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**References**

The list of references is available in the online version of this paper.

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**CARDIOVASCULAR FLASHLIGHT**

**Rare cause of myocardial infarction, stroke, and lung tumour**

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In a 53-year-old male admitted with acutely developing symptoms of right-sided hemiparesis and dysarthria revealed an extensive ischaemic infarct involving the left posterior cerebral artery territory. ECG showed right bundle branch block and significant anterior ST-segment elevation (Panel A). In addition, elevated cardiac enzyme levels suggested acute myocardial infarction (MI). Immediate transthoracic echocardiography showed a homogenous mass involving the apices of ventricles and the interventricular septum (Panel B and Supplementary material online, Video S1) therefore neither coronary angiography nor surgical treatment was indicated. Cine cardiovascular and T2-weighted MRI supported and better localized the tumour (Panel C and Supplementary material online, Videos S2–S4; Figure S1). Chest radiograph (Panel E) demonstrated a soft-tissue shadow in the right upper lung field (arrow), CT scan revealed a tumour mass there.

The patient died on 9th day of the hospital stay of heart failure. Autopsy revealed extensive tumour infiltration of the heart (Panel F). Major coronary arteries were intact. A single cerebral ischaemic infarct (diameter 4 cm) was found. Histology described primary malignant mesothelioma of the pericardium (Panel G).

Aetiology of the MI was the occlusion of arterioles supplying the myocardium due to interaction of hypercoagulability (arrows indicate fibrin-microthrombi in the microvessels of intact lung in Panel D) and per continuitatem tumour propagation of arterioles (arrow points to a tumour thrombus obliterating an arteriole within intact myocardium in Panel I).

Clinical appearance of the MI was fascinating, characterized by ST-segment elevation with changing maximum amplitude (Supplementary material online, Figure S2) and fluctuating the high-sensitive troponin level (0.47–0.31–0.99 μg/L). Accordingly, histology proved coexistence of acute (arrow) and subacute (arrowhead) MIs (Panel H).

This report introduces an uncommon mechanism responsible for MI.

Supplementary material is available at European Heart Journal online.