Isolated non-compaction of the myocardium as a cause of coronary and cerebral embolic events in the same patient

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A 44-year-old woman with a history of smoking and previous cerebral thrombo-embolism presented to the emergency department with prolonged chest pain and ECG changes showing an acute anterior myocardial infarction. She was referred to the cath-lab for primary angioplasty. Coronary angiography showed a thrombotic occlusion at the origin of first diagonal branch without evidence of coronary stenoses in the other vessels (Panel C, arrow). The thrombus was removed using a thrombus extraction catheter (Pronto®, Vascular Solutions, Inc., Minneapolis, MN, USA) and TIMI III flow was restored without residual coronary stenosis. Two-dimensional echocardiography with intravenous echo contrast showed an enlarged left ventricle with severe left ventricular dysfunction (EF 30%). In addition, prominent trabeculations (T) and deep intratrabecular recesses at apical posterolateral wall were present (Panels E and F) suggesting the diagnosis of left ventricular non-compaction of the myocardium (LVNC). Cardiac magnetic resonance imaging (CMRI) confirmed the diagnosis of LVNC with a small apical thrombus (t) within the uncompacted layer of the myocardium. Delayed enhanced images showed an anteroapical infarct scar area (S) (Panels I and L). In addition, mild pericardial effusion was noted. Patient was discharged on ramipril, carvedilol, and oral anticoagulation with acenocumarol. As left ventricular dysfunction remains unchanged 3 months after discharge, implantation of cardioverter defibrillator is planned.

This case illustrates the importance of early detection of LVNC to eventually prevent complications as occurred in this patient and highlights the comprehensive role of contrast-enhanced CMRI in the evaluation of LVNC patients, leading in vivo detection of thrombus and extent of myocardial necrosis.

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