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Silent Left Main Coronary Artery Obstruction in Prosthetic Aortic Valve Endocarditis: A Multimodality Imaging Case Report

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Introduction: Coronary involvement is an uncommon but potentially devastating complication of prosthetic valve endocarditis (PVE).[1] We describe an atypical presentation of extensive left main coronary artery (LMCA) obstruction associated with culture-negative PVE.

Case report: 66-year-old man with prior bioprosthetic aortic valve, aortic root and ascending aortic replacement (2024), and a recent cerebellar ischemic stroke presented with unsteadiness, left-sided thoracic discomfort and arm symptoms, without typical angina, dyspnea or fever. Troponin and inflammatory markers were elevated. Initial computed tomography suggested LMCA obstruction, as well as splenic and renal infarctions. Transthoracic and transesophageal echocardiography revealed a mobile mass on the prosthetic valve (at least 12 × 20 mm) and suspected periannular abscess extending toward the LMCA. Cardiac CT demonstrated a larger vegetation-like mass (approximately 45 × 15 × 10 mm) arising near the left and non-coronary sinuses and extending into the LMCA, causing marked luminal compromise. Cardiac magnetic resonance showed severe left ventricular dysfunction (ejection fraction 15–20%) and nonviable anterior and apical myocardium. Repeated blood cultures were negative. After multidisciplinary Heart Team assessment, surgery was deferred because of prohibitive operative risk and severe ventricular dysfunction. Antibiotic therapy was initiated with ampicillin and ceftriaxone and subsequently changed to vancomycin. The course was complicated by SCAI (Society for Cardiovascular Angiography and Interventions) stage C cardiogenic shock requiring dobutamine and acute kidney injury requiring renal replacement therapy. Following prolonged medical management, the patient stabilized, inflammatory markers normalized, renal replacement therapy was discontinued, and follow-up cardiac CT documented complete LMCA recanalization. Repeat echocardiography showed a functioning bioprosthesis with mild regurgitation. The patient was discharged on oral antibiotics.

Conclusion: PVE may cause extensive LMCA compromise and myocardial injury without classic acute coronary syndrome symptoms. Echocardiography, cardiac CT and cardiac magnetic resonance provide complementary information for diagnosis and management. Conservative treatment in this case reflected individualized high surgical risk and should not be generalized to other patients with complicated PVE.

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LITERATURE

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