Acute coronary syndrome in aortic infective endocarditis

David Attias1, David Messika-Zeitoun1*, Michel Wolf2, Laurent Lepage1, and Alec Vahanian1

1Department of Cardiology, AP-HP, Bichat Hospital, 46 rue Henri Huchard, 75018 Paris, France and 2Intensive Care Unit, AP-HP, Bichat Hospital, Paris, France

Received 24 May 2008; accepted after revision 31 May 2008; online publish-ahead-of-print 16 June 2008

KEYWORDS
Infective endocarditis; Acute coronary syndrome; Periannular complications

Case report

A 34-year-old man, with a history of active intravenous drug abuse, was admitted to the intensive care unit for a non-ST-elevation myocardial infarction. Blood pressure was 120/60 mmHg. He had no fever, and a systolic and diastolic murmur was audible. ECG showed an atrial fibrillation with fast ventricular cadence and a profound and diffuse ST-segment depression (Figure 1A). Inflammatory markers were markedly elevated (leucocytes count: 22 500/ml, C-reactive protein: 154 mg/L). Troponin was mildly elevated (1.62 μg/L). Transthoracic and transoesophageal echocardiography showed severe mitral and aortic damages with large vegetations on both valves and severe aortic regurgitation (Figure 1B). There was a 3 mm fistula between the ascending aorta and the left atrium (Figure 1C). Periannular complications were responsible for an extrinsic compression of the left main coronary artery (Figure 1D). Left ventricular ejection was preserved. One hour after admission, the patient died before he could

Figure 1  ECG showing an atrial fibrillation with fast ventricular cadence and a profound and diffuse ST-segment depression.

* Corresponding author. Tel: +33 1 40 25 66 01.
E-mail address: david.messika-zeitoun@bch.ap-hop-paris.fr

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have been operated on. Post-mortem blood cultures isolated *Streptococcus sanguis*.

Acute coronary syndrome occurs in only 1–3% in infective endocarditis. It is classically associated with virulent microorganisms, aortic valve infection, severe valvular regurgitation, and extensive periannular complications. It dramatically increased mortality. The two main mechanisms responsible for myocardial ischaemia are coronary embolism and exceptionally extrinsic coronary compression as in our patient.