Clin Res Cardiol (2012) 101:1021–1023
DOI 10.1007/s00392-012-0483-8

LETTER TO THE EDITORS

Molecular evidence of *Enterococcus faecalis* in an occluding coronary thrombus from a patient with late prosthetic valve endocarditis

Roland Klingenberg · Christian Schmied · Volkmar Falk · Thomas F. Lüscher · Andrea Zbinden · Roberto Corti

Received: 13 March 2012 / Accepted: 30 May 2012 / Published online: 12 June 2012

Sirs:

ST elevation myocardial infarction (STEMI) is commonly caused by a thrombotic occlusion of an epicardial coronary artery. The most frequent cause is plaque rupture or erosion of an atherosclerotic plaque with subsequent thrombus formation [1]. Thromboembolism constitutes an infrequent scenario attributable to dislodgement of a thrombus from a distinct site such as in venous thrombosis combined with a patent foramen ovale. Septic thromboembolism from an endocarditic valve is a very rare, but potentially fatal etiology and direct evidence of the associated microorganism in the occluding coronary thrombus has not been reported.

A 77-year-old male patient with subacute anterior ST-segment elevations on 12-lead ECG was referred to our institution for coronary angiography. His past medical history revealed a left hip joint replacement 10 years ago and aortic valve replacement with a biological prosthetic valve for symptomatic calcific aortic stenosis 5 years ago. At that time he had a severely compromised systolic LV function and non-stenotic epicardial coronary arteries (Fig. 1a). Due to persistent pain in the left hip, analysis of the joint fluid was performed 3 weeks prior to admission yielding *Enterococcus faecalis*. Surgical hip prosthesis removal was complicated by postoperative septicemia (*E. faecalis*), requiring vasopressor therapy and intravenous antibiotics (amoxicillin and tobramycin iv). During the course of this complication, the patient also experienced chest pain associated with the ECG changes mentioned above. Coronary angiography showed complete thromboembolic occlusion of the mid-portion of the left anterior descending coronary artery (LAD; Fig. 1b) and a non-stenotic lesion in the circumflex and right coronary artery. Repetitive thrombus aspiration using an aspiration catheter and stenting due to residual thrombus material was performed with subsequent restoration of normal coronary blood flow. A selective aortogram showed accumulation of contrast-dye in a perivalvular cavity protruding from the ascending aorta in proximity to the biological valve suspicious of an abscess (Fig. 1c). To assess urgency of surgery, serial transoesophagial echocardiograms over the next 2 days revealed rapidly growing vegetations on the aortic valve, relevant aortic regurgitation and an enlarged perfused perivascular cavity measuring 2.5 × 1.3 cm (Fig. 1d–f). Urgent cardiac surgery was performed (standard/logistic euroSCORE = 22/93.65 %) confirming a large abscess cavity with disentanglement of the aortic valve annulus from the mitral valve and substantial valvular vegetations. Following explantation of the prosthetic valve and debridement, the aortic root was replaced by a Freestyle-prosthesis with re-implantation of the left and right coronary ostium. The antibiotic regimen was escalated due to an intercurrent ventilator-associated pneumonia and the patient was referred 5 weeks later to a regional hospital.

Microbiological evaluation yielded *E. faecalis* for the vegetations on the prosthetic aortic valve using conventional culture techniques. The coronary thrombus aspirated from the LAD was positive for bacterial DNA of *E. faecalis* by PCR method.

R. Klingenberg (✉) · C. Schmied · T. F. Lüscher · R. Corti
Department of Cardiology/Cardiovascular Research, University Hospital Zurich, Rämistrasse 100, 8091 Zurich, Switzerland
e-mail: roland.klingenberg@usz.ch

V. Falk
Department of Cardiothoracic Surgery, University Hospital Zurich, Zurich, Switzerland

A. Zbinden
Institute of Medical Microbiology, University of Zurich, Zurich, Switzerland

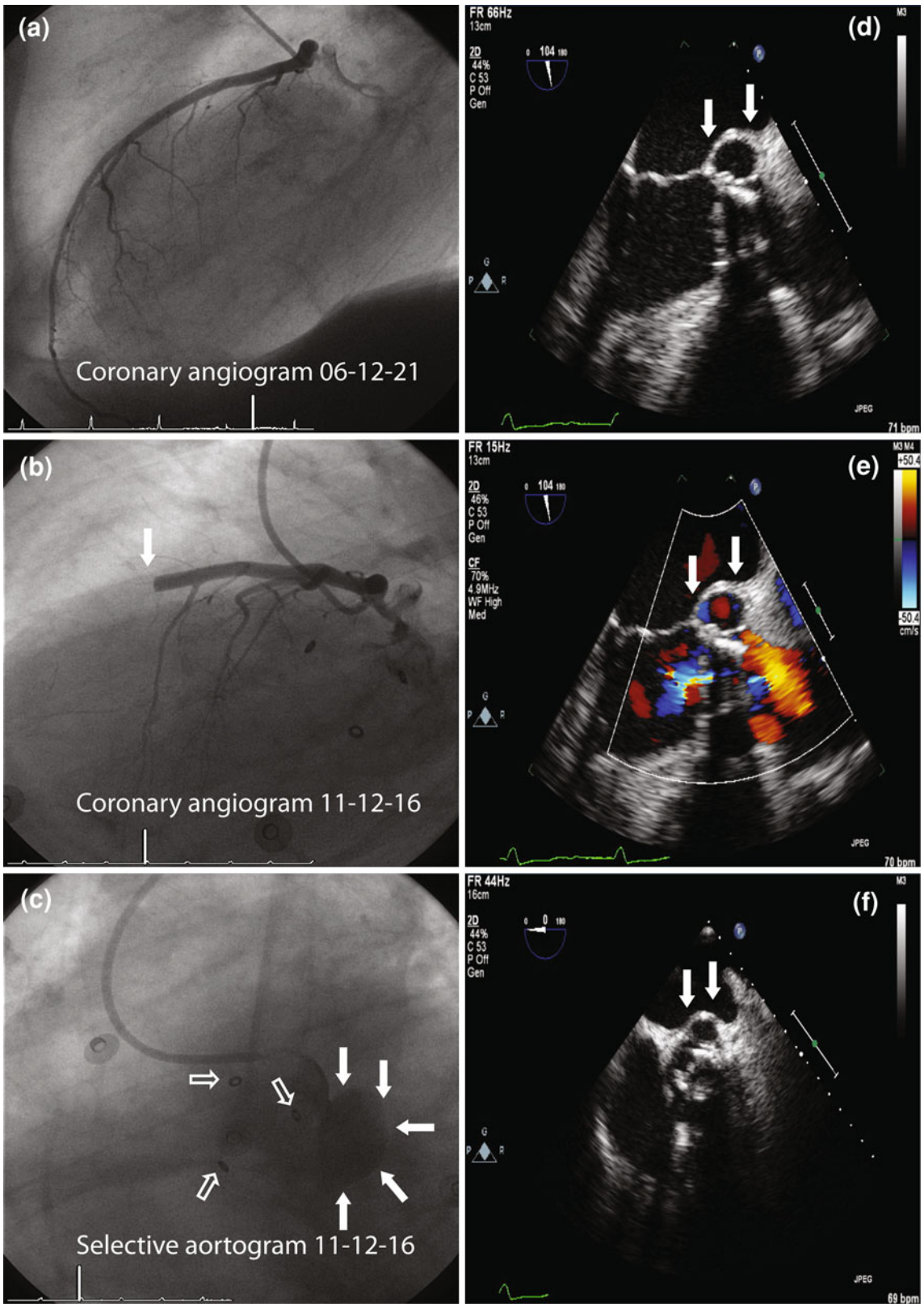

Fig. 1 Imaging of the coronary vasculature, aortic valve and aorta. **a**, **b** Coronary angiogram. *Arrow* shows thrombotic occlusion. **c** Selective aortogram showing the bioprosthetic valve (*open arrows*) and the adjacent abscess (*closed arrows*). **d–f** Transoesophageal echocardiogram ± color doppler. *Arrows* show paravalvular abscess cavity

We here report a case of STEMI due to septic embolisation as a result of infective endocarditis with *E. faecalis* leading to a rapid destruction of the prosthetic valve. We demonstrate a clear link between colonization of the prosthetic aortic valve with *E. faecalis* and PCR-based detection of the bacterium [2] in the occluding coronary thrombus. In line with our interpretation of a thromboembolic source of the thrombus, the LAD showed no detectable atherosclerotic lesion 5 years prior to the incident and also was remarkably smooth after thrombus aspiration and stenting at the time of the STEMI. The spectrum of causative organisms for prosthetic valve endocarditis ranges from frequently found bacterial species (*Staphylococcus aureus*, *Coagulase-negative Staphylococci*, *Enterococci*, *Viridans Streptococci*) [3] to rare cases caused by *Lactococcus garvieae* [4] or *Granulicatella adiacens* [5]. Despite a rising number of patients with prosthetic valves, the relative incidence of prosthetic endocarditis has declined substantially to less than 0.5 % per year for late prosthetic endocarditis [6]. This case demonstrates the dramatic course of late prosthetic valve endocarditis despite prompt initiation and maintenance of an antibiotic regimen with documented sensitivity of *E. faecalis* to the combination of a β-lactam and an aminoglycoside which had been implemented according to current ESC guidelines [7].

Acknowledgments The authors received support from the Swiss National Research Foundation Sonderprogramm Universitäre Medizin SPUM 33CM30-124112; the Swiss Heart Foundation; the Fondation Leducq, a strategic alliance with Pfizer Inc. and the Zurich Heart House—Foundation for Cardiovascular Research, Zurich.

Conflict of interest No conflict of interest to be declared.

References

1. Virmani R, Burke AP, Farb A, Kolodgie FD (2006) Pathology of the vulnerable plaque. J Am Coll Cardiol 47:C13–C18
2. Bosshard PP, Kronenberg A, Zbinden R, Ruef C, Bottger EC, Altwegg M (2003) Etiologic diagnosis of infective endocarditis by broad-range polymerase chain reaction: a 3-year experience. Clin Infect Dis 37:167–172
3. Wang A, Athan E, Pappas PA, Fowler VG Jr, Olaison L, Pare C, Almirante B, Munoz P, Rizzi M, Naber C, Logar M, Tattevin P, Iarussi DL, Selton-Suty C, Jones SB, Casabe J, Morris A, Corey GR, Cabell CH (2007) Contemporary clinical profile and outcome of prosthetic valve endocarditis. JAMA 297:1354–1361
4. Wilbring M, Alexiou K, Reichenspurner H, Matschke K, Tugtekin SM (2011) *Lactococcus garvieae* causing zoonotic prosthetic valve endocarditis. Clin Res Cardiol 100:545–546
5. Poss J, Schafers HJ, Herrmann M, von Muller L, Bohm M, Kilter H (2010) *Leukocytoclastic vasculitis* and myocardial infarction as presenting manifestations of infective endocarditis: a case report. Clin Res Cardiol 99:59–61
6. Piper C, Korfer R, Horstkotte D (2001) Prosthetic valve endocarditis. Heart 85:590–593
7. Habib G, Hoen B, Tornos P, Thuny F, Prendergast B, Vilacosta I, Moreillon P, de Jesus Antunes M, Thilen U, Lekakis J, Lengyel M, Muller L, Naber CK, Nihoyannopoulos P, Moritz A, Zamorano JL (2009) Guidelines on the prevention, diagnosis, and treatment of infective endocarditis (new version 2009): the task force on the prevention, diagnosis, and treatment of infective endocarditis of the european society of cardiology (esc). Endorsed by the european society of clinical microbiology and infectious diseases (escmid) and the international society of chemotherapy (isc) for infection and cancer. Eur Heart J 30:2369–2413