CASE REPORTS

Right sided infective endocarditis: Tempus fugit!

Nico R. Van de Veire*, An-Kristin Ascoop, Michel De Pauw, Johan De Sutter, Thierry C. Gillebert

Department of Cardiovascular Diseases, Ghent University, Ghent, Belgium

Received 6 January 2005; received in revised form 5 April 2005; accepted 20 April 2005
Available online 25 May 2005

KEYWORDS
Infective endocarditis; Right sided endocarditis; Intravenous drug use

Abstract We report a case of an intravenous drug user who already had a tricuspid bioprosthesis implanted after an infective endocarditis with massive tricuspid regurgitation one year ago. Now he presents with a large mass on the atrial side of the bioprosthesis that led to obstruction; hemocultures contained Enterococcus faecalis. After one-week therapy with antibiotics, aspirin and enoxaparin the mass untangled to a swinging structure and moderate to severe tricuspid regurgitation developed; surgery appeared inevitable. After two weeks however the mass was gone, tricuspid insufficiency subsided and the patient became asymptomatic. This case illustrates the potential but controversial role of anticoagulation in the treatment of patients with infective endocarditis.

© 2005 The European Society of Cardiology. Published by Elsevier Ltd. All rights reserved.

Introduction

Cardiologists working in hospitals situated in large urban communities are nowadays frequently treating a new kind of patients. These patients are commonly young, male IV drug users seeking medical advice because of fever.1,2

Case description

A 45-year-old male patient came to the emergency department of our institution because of fever up to 40 °C accompanied by shivering. He also complained of general malaise and anorexia.

One year ago the same patient had been admitted with an infective endocarditis of the tricuspid valve caused by auto-injection of illegal substances with contaminated needles. Despite adequate antibiotic treatment extensive valvular destruction had occurred and he developed intractable right heart failure. The valve was replaced by a biological 33 mm Mosaic (Medtronic) — prosthetic valve.

Since the operation last year, the patient was on methadone 50 mg once daily to control withdrawal symptoms and received psychosocial treatment on an outpatient basis. Despite these measures the patient admitted still having multiple addiction problems. He smoked two packs of cigarettes daily, drank large amount of alcoholic beverages
and was occasionally using cocaine and heroin intravenously. He mentioned that injections with dirty needles were often followed by shivering and fever. This provided him with a kick rather than deterring him.

Clinical examination revealed a cachectic patient with puncture holes on both arms. He was hemodynamically stable with a blood pressure of 113/70 mmHg and a heart rate of 90 beats per minute. There were no clinical signs of right-sided heart failure. Inflammatory markers in his blood were markedly increased: 22,000 white blood cells/μl and a CRP of 7 mg/dl. The patient tested positive for hepatitis B and C but had a negative HIV status.

Transthoracic and transesophageal echocardiography were performed on the day of admission (Fig. 1). A 2.5 × 2.5 cm mass, suggestive for a vegetation was attached to the atrial side of the tricuspid bioprosthesis. This mass was partly obstructing blood flow and caused a functional stenosis with a maximal gradient of 13 mmHg and a mean gradient of 7 mmHg. There was a tricuspid regurgitation grade 1 (systolic transvalvular gradient 16 mmHg) without arguments for pulmonary artery hypertension.

As pulmonary embolism is a typical complication of right sided endocarditis, a CAT scan of the thorax was performed. The scan was negative for pulmonary embolism, infarction, abscesses, pleural effusions and empyema. Before initiating empirical antibiotic therapy several blood cultures were drawn. Four days after admission the blood cultures became positive for Enterococcus faecalis. On basis of the antibiogram treatment with ampicillin 6 × 2 g and gentamycin 300 mg intravenously was initiated. Given the size of the thrombus and the thrombotic nature of endocarditis lesions, additional antithrombotic therapy was initiated with acetylsalicylic acid 160 mg/day and with enoxaparin two times 60 mg/day.

A transthoracic echocardiogram performed 5 days after presentation (Fig. 2) showed a dramatic progression of disease compared to the first echo. The vegetation attached to the atrial side of the tricuspid bioprosthesis was now untangled and presented as a large mobile oscillating structure swinging into the right ventricle during diastole. Functionally the inflow obstruction had disappeared but the tricuspid bioprosthesis had developed a moderate to severe insufficiency.

Treatment was continued with antibiotics, diuretics, methadone and fluid restriction. Surgical reintervention appeared inevitable. On a new transthoracic echocardiogram (Fig 3, panel A) performed two weeks after admission however, the large mobile oscillating structure had disappeared. A small filiform mobile structure at the atrial side of the bioprosthesis (1.2 cm) was the last reminder of the original vegetation. CAT-scans of the brain, thorax and abdomen were all normal.

Echocardiography three weeks after initial presentation showed tricuspid valve leaflets without vegetations (Fig. 3, panel B) and a tricuspid regurgitation grade 1. The patient was further treated conservatively and antibiotics could be stopped after 6 weeks. The clinical and biochemical evolution was favourable.

The patient was granted permission to leave the ward during one day to sort out his personal affairs but did not return to the hospital.

One week later he came to the emergency department with complaints of shivering. Inflammatory markers were normal, hemocultures remained negative and the transthoracic echocardiogram showed a tricuspid bioprosthesis without vegetations. Despite a complicated infective endocarditis

Figure 1 Transthoracic echocardiography at the time of admission. Panel A shows a detail of the apical four chamber view with a 2.5 × 2.5 cm mass (arrow), suggestive for a vegetation, attached to the atrial side of the tricuspid bioprosthesis. Panel B shows tricuspid regurgitation grade 1. Abbreviations: RV, right ventricle; RA, right atrium.
resulting in cardiac surgery, a novel episode of endocarditis, methadone substitution and intensive psychosocial counselling our patient started using IV drugs again: the urine sample tested positive for heroin.

**Discussion**

In this case report we describe a drug addict who had an enterococcus endocarditis of his tricuspid bioprosthesis.\textsuperscript{1–3} The diagnosis in our patient was

---

**Figure 2** Transthoracic echocardiography five days after admission. Panel A–C depict in apical four chamber view an untangled large mobile oscillating structure (arrow) swinging from the right atrium (panel A) into the right ventricle (panel B and C) during diastole. Panel D illustrates tricuspid regurgitation grade 3 on colour flow mode. Abbreviations: RV, right ventricle; RA, right atrium.

**Figure 3** Panel A: Transthoracic echocardiography two weeks after admission. In this apical four chamber view the large oscillating structure has disappeared. A 1.2 cm filiform structure (arrow) is now attached to the atrial side of the bioprosthesis. Panel B: Transthoracic echocardiography three weeks after admission. The tricuspid bioprosthesis leaflets are free from vegetations. Abbreviations: RV, right ventricle; RA, right atrium.
not difficult to make since he had two major Duke criteria (an oscillating intracardiac mass on the tricuspid bioprosthesis and Enterococci in his blood cultures) and two minor criteria (fever and intravenous drug use).

He presented with a large mass on the atrial side of the bioprosthesis that led to obstruction. After one-week therapy consisting of antibiotics, aspirin and enoxaparin the mass untangled to a swinging mass that obviously interfered with valve closure; obstruction disappeared and was replaced with regurgitation. After two weeks the mass was gone and tricuspid regurgitation was diminished. A lung scan showed no signs of embolisation.

Analyzing these images retrospectively we could speculate on the true nature of the mass. The mass was certainly infected as suggested by fever, shivering, increased CRP and positive blood cultures. The mass probably consisted mainly out of infected thrombotic material, slowly unfolding at first (mimicking valve destruction) and dissolving eventually. It is hypothesized that platelet-fibrin deposition occurs spontaneously in persons vulnerable to endocarditis and that these deposits, called nonbacterial thrombotic endocarditis, are the sites at which microorganisms adhere during bacteremia to initiate infective endocarditis.4 It is because of the known thrombotic nature of bigger endocarditis lesions that we decided to treat this particular patient with acetylsalicylic acid and low molecular weight heparin. Data on use of antithrombotic drugs in endocarditis are controversial and the disease is commonly considered as a relative contraindication for anticoagulation.

From this case report, but also from animal experimental studies it seems that it could be valuable to test in a randomised way if aspirin or heparin or the combination of both would be beneficial in patients with infective endocarditis especially with big vegetations. It could also be wise to continue aspirin as a secondary prevention given the presence of nonbacterial thrombi on damaged endothelium as pre-endocarditis lesions.

Acknowledgements

Nico Van de Veire is a research assistant and Johan De Sutter a senior clinical investigator of the Fund for Scientific Research — Flanders (Belgium) (F.W.O — Vlaanderen).

References