HEART FAILURE AND CARDIOMYOPATHY

Nebivolol improves coronary flow reserve in patients with idiopathic dilated cardiomyopathy

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Heart 2007;93:319–324. doi: 10.1136/hrt.2006.091751

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Accepted 3 July 2006
Published Online First
25 October 2006

Background: Impaired coronary flow reserve (CFR) is a significant predictor of poor prognosis in patients with idiopathic dilated cardiomyopathy (IDC). Nebivolol reduces mortality and morbidity in patients with heart failure and left ventricular dysfunction, including cases caused by IDC.

Objective: To assess the effects of nebivolol on CFR in patients with IDC.

Methods: CFR was measured in 21 clinically stable patients with IDC (mean (SD) ejection fraction 35.7 (6.2)) at baseline and after 1 month of treatment with nebivolol once daily. A control group of apparently healthy subjects who were matched for age and sex was used for comparison. Resting and hyperaemic coronary flows were measured using transthoracic second-harmonic Doppler echocardiography. None of the subjects had any systemic disease.

Results: After 1 month of treatment, heart rate was reduced significantly ($p < 0.001$). The blood pressure was decreased significantly ($p < 0.001$). The left ventricular end-diastolic diameter and stroke volume were not changed significantly, but end-systolic diameter was decreased significantly ($p < 0.05$). Resting rate–pressure product was lower after treatment with nebivolol, but dipyridamole-induced change was not influenced by the treatment. Nebivolol treatment reduced significantly coronary velocities at rest ($p < 0.02$) and also caused a significant increase in coronary velocities after dipyridamole ($p < 0.02$), leading to a greater CFR (2.02 (0.35) vs 2.61 (0.43), $p < 0.001$). Nebivolol induced an absolute increase of 6% in the CFR in 17 of 21 patients (80.9%).

Conclusions: In patients with IDC, 1 month of treatment with nebivolol induces a marked increase in CFR.

Previous studies have shown that despite angiographically normal epicardial coronary arteries, coronary flow reserve (CFR) is altered in patients with idiopathic dilated cardiomyopathy (IDC) due to coronary microcirculatory dysfunction.^{1–3} In addition, reduced CFR has been reported to be a poor prognostic indicator and independent predictor of subsequent cardiac events in patients with idiopathic left ventricular dysfunction.⁴ However, the mechanism of the altered CFR in IDC is not fully understood. Previous experimental and clinical studies showed that basal and stimulated releases of endothelium-derived nitric oxide (NO) were reduced in small coronary vessels excised from nonischaemic failing hearts.^{5–6} This may be attributed, at least partly, to reduced gene expression of vascular endothelial NO. More recently, it has been suggested that decreased availability of basal NO in human coronary circulation was indicated by the blunted vasoconstrictor effect of N_G -monomethyl-L-arginine on the coronary blood flow, and that blunted response to N_G -monomethyl-L-arginine was most prominent in patients with more advanced left ventricular dysfunction.⁵ Furthermore, long-term L-arginine supplementation improved small-vessel coronary endothelial function in humans.⁷

Nebivolol is a third-generation, highly cardioselective and lipophilic β_1 -adrenoceptor antagonist.⁸ It also affects the L-arginine/NO pathway, causing vasodilatation by increasing endothelial NO release.⁹ Recent clinical trials have shown that nebivolol is well tolerated and effective in reducing mortality and morbidity in patients with heart failure and left ventricular dysfunction, including cases caused by IDC.¹⁰ However, the mechanism behind the reduction of mortality in these patients by nebivolol treatment remains unclear. Therefore, we hypothesised that nebivolol treatment may contribute to reduction of

mortality in patients with IDC by improving CFR. Accordingly, this study assessed the effects of short-term nebivolol treatment on CFR by transthoracic Doppler echocardiography (TTDE) in patients with IDC.

METHODS

Patients

Thirty ambulatory patients with IDC who had not previously taken any β -blocking agents were included in this study. All patients underwent coronary angiography, which showed normal epicardial coronary arteries. Diagnosis of dilated cardiomyopathy was based on a left ventricular ejection fraction (LVEF) $\leq 45\%$ by echocardiography and coronary angiography, left ventricular end-diastolic diameter > 55 mm and/or left ventricular end-systolic diameter 45 mm by echocardiography. All patients had at least a 6-month history of IDC, had been treated with both angiotensin-converting enzyme inhibitor (ramipril) and loop diuretic (furosemide) for > 6 months at the baseline condition, and were clinically stabilised under this treatment. Other vasoactive drugs such as angiotensin II receptor blockers and digoxin were not allowed. The patients did not have any evidence of decompensated heart failure, had New York Heart Association functional class I–III symptoms and had normal sinus rhythm at the time of study.

Patients were excluded from the trial if they showed one or more of the following parameters: a history of hypertension or hypertension on clinical examination (systolic pressure

Abbreviations: CFR, coronary flow reserve; DPFV, diastolic peak flow velocity; IDC, idiopathic dilated cardiomyopathy; LAD, left anterior descending artery; LVEF, left ventricular ejection fraction; TTDE, transthoracic Doppler echocardiography

>160 mm Hg and/or diastolic pressure >100 mm Hg measured in a sitting position at three subsequent examinations); valvular or congenital heart disease; cardiac rhythm other than sinus; second-degree or third-degree atrioventricular block; diabetes mellitus; previous myocardial infarction; hypothyroidism or hyperthyroidism; hypertrophic cardiomyopathy; chronic obstructive pulmonary disease or cor pulmonale; systemic diseases such as collagenosis and pheochromocytoma; pregnancy; significant chronic renal failure (serum creatinine >159 $\mu\text{mol/l}$); and cancer that might substantially shorten survival. Patients engaged in alcohol misuse and cigarette smoking were also excluded.

Control group

The control group consisted of 15 subjects who underwent coronary angiography for clinical indications, had coronary arteries free of stenotic lesions and whose LVEF was >55%. The same exclusion criteria were also applied to this group. The control subjects had not taken any vasoactive drugs. The study was conducted according to the recommendations set by the Declaration of Helsinki on biomedical research involving humans. The institutional ethics committee approved the study protocol, and all patients and controls provided written informed consent.

Study design and drug titration

The study was performed according to an open design. After baseline assessment of CFR by TTDE, patients received nebivolol. The drug dose was initially 1.25 mg/day and increased at weekly intervals over the next 2 weeks to 2.5 mg/day and 5 mg/day (maximum 5 mg/day), if well tolerated. Each patient was controlled weekly, and nebivolol treatment was titrated according to the patient's clinical and symptomatic status. Maximum dose was achieved in all patients. At 1 month after reaching target dose or optimisation of nebivolol treatment, echocardiographic examinations including CFR measurement were repeated 6 h after giving nebivolol. During this period, two patients were admitted to the hospital because of decompensated heart failure, one patient died suddenly out of hospital, two patients developed atrial fibrillation and four patients declined to continue participating in the study. Therefore, with the exclusion of these nine patients, the study was completed with 21 patients, with results expressed accordingly. The same investigator who had been blinded to the patients' data performed all echocardiographic examinations.

Echocardiographic examination

Each subject was examined using an Acuson Sequoia C256 echocardiography system equipped with a 3V2c broadband transducer with second-harmonic capability (Acuson, Mountain View, California, USA). Two-dimensional, M-mode and subsequent Doppler echocardiographic examinations were performed on each subject in the lateral decubitus position. The echocardiographic images were recorded on video home system videotapes. Diastolic and systolic interventricular septal thickness, posterior wall thickness and left ventricular end-diastolic and left ventricular end-systolic diameters were measured on the parasternal long-axis views. All measurements were performed on M-mode images. Left ventricular end-diastolic volume, end-systolic volume and ejection fraction were calculated using the biplane Simpson formula.

The pulsed Doppler sample volume was positioned at the mitral leaflet tips. Early diastolic peak flow velocity (E), late diastolic peak flow velocity (A), E/A ratio, isovolumic relaxation time, and E wave deceleration time were measured by transmitral Doppler imaging.

CFR measurement

After at least 12 h of fasting, the subjects were examined in the left lateral position. Transthoracic second-harmonic Doppler echocardiography examination was performed on each subject using an Acuson Sequoia C256 echocardiography system equipped with a high-resolution transducer with second-harmonic capability (5V2c). Visualisation of the distal left anterior descending artery (LAD) was performed using a modified, foreshortened, two-chamber view obtained by sliding the transducer on the upper part and medially, from an apical two-chamber view, to reach the best alignment with the interventricular sulcus. Subsequently, coronary flow in the distal LAD was examined by colour Doppler flow mapping over the epicardial part of the anterior wall, with the colour Doppler velocity set in the range of 8.9–24.0 cm/s. The colour gain was adjusted to provide optimal images. The acoustic window was around the midclavicular line in the fourth and fifth intercostal spaces, with the subject in the left lateral decubitus position.¹¹ The left ventricle was visualised on the long-axis cross-section, and the ultrasound beam was then inclined laterally. Next, coronary blood flow in the LAD (middle to distal) was searched by colour Doppler flow mapping. All subjects had Doppler recordings of the LAD with dipyridamole infusion at a rate of 0.56 mg/kg over 4 min. All subjects had continuous heart rate and electrocardiographic monitoring as well as blood pressure recording at baseline, during dipyridamole infusion and at recovery. Echocardiographic images were recorded on VHS videotapes.

Two experienced echocardiographers who had been blinded to the clinical data analysed the recordings. With the sample volume placed on the colour signal, spectral Doppler of the LAD showed the characteristic biphasic flow pattern with larger diastolic and smaller systolic components (fig 1). Coronary diastolic peak velocities were measured at baseline and after dipyridamole infusion by averaging the highest three Doppler signals for each measurement. CFR was defined as the ratio of hyperaemic to baseline diastolic peak velocities. A CFR value ≥ 2.0 was considered normal.¹² CFR measurement was achieved in all subjects. To test the reproducibility of CFR measurement, it was repeated two days later in eight control subjects. The intraobserver, intraclass correlation coefficient for CFR measurement was 0.885.

Statistical analyses

The analyses were performed using SPSS V.9.0. Data were expressed as mean (SD). The IDC and the control groups were compared using the Student's *t* test. Before and after treatment, comparison analyses were made using paired samples *t* test. Values of $p < 0.05$ were considered significant.

RESULTS

Baseline characteristics of the study population

Table 1 shows the general characteristics of the patients and controls. The groups were similar regarding age, sex, body mass index, systolic and diastolic blood pressures, triglycerides and glucose values. However, the levels of total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol and high-sensitivity C reactive protein were statistically different between the groups.

Baseline haemodynamic and echocardiographic parameters of the groups

Left ventricular end-diastolic, end-systolic and accordingly stroke volumes, and left ventricular mass index were significantly higher in the IDC group (table 2). The mean (SD) LVEF was 35.5 (6.4)% in the IDC group and 67.1 (2.2)% in the control group ($p < 0.001$). Representing left ventricular diastolic

Figure 1 Mid to distal segment of the left anterior descending artery (LAD) in colour-coded transthoracic Doppler echocardiography (arrows); spectral Doppler coronary blood flow by sampling in mid to distal segment of the LAD. LV, left ventricle; S, systole; D, diastole.

dysfunction, mitral E/A ratio was 1.5 (1.4) in the IDC group and 1.1 (0.2) in the control group ($p < 0.01$), isovolumic relaxation time was 108.8 (22.4) ms in the IDC group and 86.7 (11.6) ms in the control group ($p < 0.01$), and mitral E-wave deceleration

time was 206.1 (78.9) ms in the IDC group and 193.4 (14.7) ms in the control group ($p > 0.05$). Baseline diastolic peak flow velocity (DPFV) was significantly higher (27.4 (8.5) cm/s vs 22.3 (4.3) cm/s, $p < 0.05$), but hyperaemic DPFV was

Table 1 Demographic and atherosclerotic risk characteristics of the study population

	Patients with IDC (n = 21)	Healthy controls (n = 15)	p Value
Clinical data			
Age (year)	55.9 (9.6)	55.8 (3.8)	NS
Male/female (%)	14/7 (66.3/33.3)	10/5 (66.3/33.3)	NS
Body mass index (kg/m ²)	27.5 (4.2)	27.7 (3.4)	NS
Baseline haemodynamics			
Systolic BP (mm Hg)	125.1 (13.8)	122.2 (11.2)	NS
Diastolic BP (mm Hg)	79.3 (8.9)	76.1 (6.4)	NS
Heart rate (beats/min)	74.0 (15.0)	70.1 (9.4)	NS
Haematological and biochemical data			
Total cholesterol (mmol/l)	4.64 (1.04)	5.37 (0.72)	<0.01
HDL cholesterol (mmol/l)	1.17 (0.19)	1.26 (0.26)	<0.05
LDL cholesterol (mmol/l)	2.87 (0.73)	3.30 (0.65)	<0.01
Triglyceride (mmol/l)	1.53 (0.57)	1.62 (0.69)	NS (0.09)
Haemoglobin (g/dl)	14.29 (1.01)	14.32 (3.21)	NS
hsCRP (mg/l)	4.80 (2.93)	2.30 (1.53)	<0.01
Glucose (mmol/l)	5.20 (0.47)	5.16 (0.40)	NS
Medications			
ACE inhibitor (ramipril)	100% (21/21)	—	
Diuretic (furosemide)	100% (21/21)	—	

ACE, angiotensin-converting enzyme; BP, blood pressure; HDL, high-density lipoprotein; hsCRP, high-sensitivity C reactive protein; IDC, idiopathic dilated cardiomyopathy; LDL, low-density lipoprotein.

Figure 2 Coronary flow reserve before and after nebivolol treatment. * $p < 0.05$ versus after nebivolol treatment. $\dagger p < 0.001$ versus untreated.

significantly lower in the IDC group than in the control group (53.7 (11.9) cm/s vs 63.2 (11.3) cm/s, $p < 0.05$; table 2). Therefore, CFR was significantly lower in the IDC group than in the control group (2.02 (0.35) vs 2.79 (0.53), $p < 0.001$; fig 2).

Changes in haemodynamic and echocardiographic parameters after nebivolol treatment

The study was completed on 21 of 30 patients with IDC. Table 3 summarises the changes in the IDC group after nebivolol treatment. Left ventricular end-systolic volume decreased significantly after nebivolol treatment (from 137.4 (52.7) ml to 120.8 (43.9) ml, $p = 0.03$). LVEF increased significantly (from 35.7 (6.2)% to 40.6 (7.5)%, $p = 0.005$) and left ventricular

mass index decreased slightly but not significantly (table 3). Representing left ventricular diastolic functions, left ventricular isovolumic relaxation time changed significantly; however, mitral E and A velocities, E/A ratio and mitral E wave deceleration time did not change significantly. Systolic and diastolic blood pressures and heart rate at rest decreased significantly ($p < 0.001$). Accordingly, the rate-pressure product at rest decreased significantly ($p < 0.001$) after treatment (table 3). Diastolic blood pressure after dipyridamole was decreased significantly ($p < 0.01$) and heart rate after dipyridamole was slightly reduced after nebivolol treatment. However, systolic blood pressure and rate-pressure product after dipyridamole did not change significantly after treatment.

Changes in coronary flow measurements and CFR after nebivolol treatment

The dipyridamole test during baseline testing and after nebivolol treatment did not cause major side effects. Coronary flow was measured in all patients and controls. During the 1-month treatment with nebivolol, baseline DPFV significantly decreased (27.4 (8.5) cm/s vs 23.1 (3.8) cm/s, $p = 0.02$) and hyperaemic DPFV significantly increased (53.7 (11.9) cm/s vs 60.1 (11.9) cm/s, $p = 0.02$). Therefore, nebivolol treatment significantly improved the CFR in 21 patients with IDC (2.02 (0.35), 2.61 (0.43), before and after nebivolol treatment, respectively; $p < 0.001$; fig 2).

When outside the interval of agreement for repeated measures obtained in eight volunteer patients with IDC (from -6% to +5%), the individual variation of CFR was considered biologically significant. An increase exceeding 6 percentage points in the CFR after nebivolol was detected in 17 of 21 patients (80.9%). CFR was decreased in only one patient (9.9%), and in three patients the CFR increase was <6%.

DISCUSSION

The main findings of the study are that (1) CFR was impaired in patients with IDC who were clinically stable on both angiotensin-converting enzyme inhibitor and loop diuretic treatments, had never taken any β -blocker treatment, and did not have any systemic disease; and (2) 1 month of nebivolol

Table 2 Echocardiographic findings and haemodynamic profiles of the study population

	Patients with IDC (n = 21)	Healthy controls (n = 15)	p Value
LVEDV (ml)	212.8 (72.2)	90.5 (12.6)	<0.001
LVESV (ml)	137.4 (52.7)	30.4 (5.5)	<0.001
Stroke volume (ml)	75.4 (25.7)	60.1 (8.7)	<0.01
EF (%)	35.7 (6.2)	67.1 (2.2)	<0.001
LVMI (g/m ²)	147.6 (30.2)	82.6 (14.1)	<0.001
Mitral E-wave max (cm/s)	71.1 (27.2)	71.1 (13.3)	NS
Mitral A-wave max (cm/s)	65.8 (24.4)	64.8 (12.9)	NS
E/A ratio	1.5 (1.4)	1.1 (0.2)	<0.05
Mitral E-wave deceleration time (ms)	206.1 (78.9)	193.4 (14.7)	NS
Isovolumic relaxation time (ms)	108.8 (22.4)	86.7 (11.6)	<0.01
Heart rate at rest (beats/min)	76.8 (16.1)	74.3.4 (10.0)	NS
Systolic BP at rest (mm Hg)	126.3 (13.2)	121.6 (11.3)	NS
Diastolic BP at rest (mm Hg)	77.3 (9.5)	75.7 (6.4)	NS
RPP at rest (beats/min mm Hg 10 ³)	9.23 (1.88)	9.98 (3.66)	NS
Heart rate after dipyridamole (beats/min)	91.4 (16.8)	89.4 (20.8)	NS
Systolic BP after dipyridamole (mm Hg)	121.4 (12.8)	116.5 (8.8)	NS
Diastolic BP after dipyridamole (mm Hg)	77.2 (6.4)	70.4 (7.0)	<0.05
RPP after dipyridamole (beats/min mm Hg 10 ³)	11.00 (2.12)	12.31 (4.7)	NS
Baseline DPFV (cm/s)	27.4 (8.5)	22.3 (4.3)	<0.05
Hyperaemic DPFV (cm/s)	53.7 (11.9)	63.2 (11.3)	<0.01

A, late diastolic peak flow velocity; BP, blood pressure; DPFV, diastolic peak flow velocity; E, early diastolic peak flow velocity; EF, ejection fraction; IDC, idiopathic dilated cardiomyopathy; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; LVMI, left ventricular mass index; RPP, rate-pressure product. Values are represented as mean (SD).

Table 3 Comparison of echocardiographic findings and haemodynamic profile before and after nebivolol treatment

	Before treatment	After nebivolol	p Value
LVEDV (ml)	212.8 (72.2)	199.7 (53.9)	NS
LVESV (ml)	137.4 (52.7)	120.8 (43.9)	<0.05
Stroke volume (ml)	75.4 (25.8)	78.9 (22.7)	NS
EF (%)	35.7 (6.2)	40.6 (7.5)	<0.01
LVMl (g/m ²)	147.6 (30.20)	140.8 (29.5)	NS
Mitral E-wave max (cm/s)	71.0 (27.2)	69.6 (23.6)	NS
Mitral A-wave max (cm/s)	65.8 (24.4)	65.0 (21.3)	NS
E/A ratio	1.5 (1.4)	1.3 (1.0)	NS
Mitral E-wave deceleration time (ms)	206.1 (78.9)	193.7 (67.2)	NS
Isovolumic relaxation time (ms)	108.8 (22.4)	123.5 (30.4)	<0.05
Heart rate at rest (beats/min)	76.8 (16.1)	63.0 (11.8)	<0.01
Systolic BP at rest (mm Hg)	126.3 (13.2)	118.8 (9.7)	<0.001
Diastolic BP at rest (mm Hg)	77.3 (9.5)	72.0 (7.9)	<0.001
RPP at rest (beats/min mm Hg 10 ³)	9.23.1 (1.88)	7.49 (1.47)	<0.001
Heart rate after dipyridamole (beats/min)	91.4 (16.8)	84.8 (12.4)	<0.05
Systolic BP after dipyridamole (mm Hg)	121.4 (12.8)	118.2 (11.2)	NS
Diastolic BP after dipyridamole (mm Hg)	77.2 (6.4)	71.9 (6.8)	<0.01
RPP after dipyridamole (beats/min mm Hg 10 ³)	11.00 (2.12)	10.03 (1.69)	NS
Baseline DPFV (cm/s)	27.4 (8.5)	23.1 (3.8)	<0.05
Hyperaemic DPFV (cm/s)	53.7 (11.9)	60.1 (11.9)	<0.05

A, late diastolic peak flow velocity; BP, blood pressure; EF, ejection fraction; DPFV, diastolic peak flow velocity; E, early diastolic peak flow velocity; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; LVMl, left ventricular mass index; RPP, rate-pressure product.
Values are represented as mean (SD).

treatment improves CFR reflecting coronary microvascular function in these patients.

CFR determined noninvasively by TTDE is a reliable marker of coronary microvascular function and its feasibility has been validated.¹³ Furthermore, CFR measured by TTDE has recently been shown to have excellent correlation with CFR measured by positron emission tomography.¹⁴ In previous reports, the success rate of coronary flow measured in the middle or distal part of LAD by TTDE was reported as 98–100%.^{13, 14} In our study, the success rate was 100%. Because systolic heart movement causes disturbances in Doppler signal resulting in difficulties to obtain reliable measurements during systole, we only measured the peak diastolic component of coronary flow. Impairment of CFR, which may lead to myocardial ischaemia, progression of left ventricular dysfunction, congestive heart failure¹⁵ and increased mortality,⁴ has been described in patients with dilated cardiomyopathy even when occlusive epicardial coronary arterial disease cannot be demonstrated angiographically.¹ Reduced CFR in patients with IDC is not fully attributable to haemodynamic changes such as the degree of left ventricular dysfunction and increased left ventricular diastolic pressure.^{2, 16} It also depends on functional and/or structural abnormalities of the microcirculation such as coronary perfusion pressure, lumen integrity and vasomotor control in epicardial conduit vessels.¹⁵

Coronary flow is regulated by oxygen demand in myocardial tissue much more than in other circulatory districts.¹⁷ β -blockade reduces myocardial oxygen consumption. This reduction is likely to be associated with a reduction in coronary flow.¹⁸ In addition, blunted heart rate response during β -blockade may beneficially affect the diastolic phase of myocardial perfusion during hyperaemia.¹⁹ Improvement of CFR has been recently shown in patients with IDC after short-term and long-term treatment with carvedilol.¹⁹ This improvement preceded the improvement of left ventricular dysfunction.

It has been shown that nebivolol favourably affects endothelial function, although the maximal NO release in microvascular endothelial cells is similar after nebivolol and carvedilol treatments.^{20, 21} Nebivolol has vasodilator properties with increasing endothelial NO release due to effects on the L-arginine/NO pathway that reduce peripheral vascular

resistance.⁹ Nebivolol not only increases NO release but also inhibits endothelin-1 synthesis.²² Increased circulating endothelin-1 concentration is associated with a poor prognosis in IDC with reduced cardiac function, and contributes to vascular resistance.²³ Although the favourable effects of nebivolol on cardiac performance in patients with impaired left ventricular systolic and diastolic functions, including those caused by IDC, have been known for more than a decade, only one study has shown a beneficial effect of nebivolol on CFR.¹⁷

In conclusion, a 1-month treatment with nebivolol in patients with IDC free of overt coronary artery disease improves CFR. This improvement is due to both an increase of hyperaemic blood flow velocity and a decrease of resting blood flow velocity, and is associated with the reduction of afterload and peripheral resistance.

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Competing interests: None declared.

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IMAGES IN CARDIOLOGY

doi: 10.1136/hrt.2006.090282

Blockage of bileaflet mitral valve prosthesis imaged by computed tomography virtual endoscopy

A 53-year-old man with a bileaflet mitral valve prosthesis and coronary artery bypass grafting presented with severe symptoms of heart failure. His ECG showed a common atrial flutter with 2:1 conduction and a heart rate of about 130 beats/min; there was no sign of acute myocardial ischaemia. Laboratory findings were also negative. A pharmacological conversion of the atrial flutter was unsuccessful, thus making electrical cardioversion necessary.

Both transthoracic (TTE) and transoesophageal echocardiography (TOE) were performed. TTE showed an elevated mean diastolic gradient (10 mm Hg) across the prosthesis. TOE confirmed the suspicion of prosthetic valve dysfunction showing a blockage of one leaflet. No thrombi were detected. A successful restoration of sinus rhythm by electrical cardioversion was achieved.

Because of the malfunctioning prosthesis the patient underwent valve replacement. In order to exclude progression of the coronary artery disease before reoperation, a fast computed tomographic (CT) scan (LightSpeed VCT scanner, GE Healthcare) was performed. Post-processing imaging showed in a three-dimensional format the prosthesis both from the left atrium and the left ventricle perspective, confirming the blockage of one leaflet (see panels A–D).

Virtual coronary angiography is a novel post-processing imaging modality of CT scans. This procedure makes the contrast material inside the coronary arteries appear transparent and the vessel wall grey or another colour (according to the attenuation control). At present, there is no other report of the use of this imaging modality in the diagnosis of prosthetic valve obstruction. In conclusion, post-processing imaging modality of fast CT scanning is a novel technique for imaging prosthetic obstruction.

Virtual endoscopy of a bileaflet valve. Panels A and B: view from left ventricle. Panels C and D: view from left atrium. The apparent rupture of the ring and one leaflet is due to motion-related artefact.

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