A LONG WAY TO HEART FAILURE

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- Chronic low back pain
- Arterial hypertension and hyperlipidaemia
- Intolerance for ACE-inhibitors. R/ARB

- 2001: Extensive prolaps of both mitral leaflets and severe mitral regurgitation. Asymptomatic. R/conservative treatment

- 2004: first consultation at Ghent University Hospital
  - Shortness of breath, NYHA II
  - Some episodes of nocturnal dyspnea, relieved by sitting
  - Minor edema of the ankles since some weeks. This particular complaint triggered the consult.
DRUG THERAPY

• Carvedilol 25 mg b.i.d.
• Valsartan 160 mg o.a.d.
• Fluvastatine 80 mg o.a.d.
• Medroxyprogesteron 5 mg o.a.d
• Estradiol patch ~ 50 µg/day
  • (3.2 mg patch twice a week)
CLINICAL EXAMINATION

- 163 cm; 59 kg, BMI 22, BSA 1.6 m²
- BP 130/67 mm Hg, RR 73 pm

- Straight back syndrome
  - loss of normal upper thoracic spinal curvature with reduced AP diameter of the chest

- Clear lungs, normal JVP

- Limited edema under the medial malleolus
CLINICAL EXAMINATION (2)

• Displaced left ventricular impulse

• Auscultation:
  • Loud first heart sound.
  • High-pitched holosystolic murmur at the apex, radiating to the axillar area 4/6
  • Low pitch third heart sound
ECHOCARDIOGRAM

- LVED = 6.5 cm; LVES = 4.0 cm; FS = 0.38; 2D-EF = 0.78
- LVPW = 9 mm, Septum = 9 mm
- LAD = 132 ml or 82 ml/m²

- Extensive prolaps of both mitral leaflets
  - Severe mitral regurgitation grade IV
    - Systolic reversal in pulmonary veins

- PISA analysis
  - Regurgitant orifice = 1.44 cm² and regurgitant flow = 209 ml
  - LVOT flow = 53.4 ml and regurgitant fraction = 0.79
ECHOCARDIOGRAM (2)

- HR 57 pm RR
- CO = 3.04 l/min or CI = 1.88 l/min

- Mitral E = 165 cm/s & mitral A = 48 cm/s
- Septal e’ = 9.0 cm/s and a’ = 6.0 cm/s;
- Septal E/e’ = 18.3
  - Is not predictive of PCW in severe MR
  - Has an important prognostic value
- RVsyst = 21 + 5 = 26 mm Hg
WHAT DO DO?

1. Continue conservative therapy
   • Stop beta blockers
2. Perform heart catheterisation before deciding
3. Plan mitral valve repair
4. Plan mitral valve replacement
HEART CATHETERISATION (2004)

- HR 67 pm; CO = 3.2 l/min and CI 2.0 l/min
- EF 0.82
- SVR 1600 dyne*s/cm\(^5\) and PVR 139 dyne*s/cm\(^5\)
- Patent coronary arteries

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SURGERY FOR BARLOW DISEASE

- Febr 22, 2004
  - Vertical posterior incision of the LA
  - Implantation of mechanical mitral valve ATS 33 mm
  - 28 separate sutures with Ethibond 2.0
- Febr 23, 2004
  - Revision for persistent bleeding with evacuation of blood in right pleural cavity
  - Acute AF, DC shock
  - Low BP’s
- March 2, 2004
  - Discharge. **Persistent fatigue and low BP’s.**
ECG AND ECHO MARCH 18, 2004
ELECTROCARDIOGRAM

- Born: 
- Age:  
- Sex:  
- Height: 163.0 cm  
- Weight:  
- BP:  
- Med:  
- Rem:  

HR: 73 /min  
Axis:  
Intervals:  
RR: 818 ms  
P: 138 ms  
PQ: 172 ms  
QRS: 88 ms  
QT: 374 ms  
QTc: 414 ms

Interpretation:  
SINUSRITME  
NORMALE AS  
NORMAAL EGG

Validated by

25 mm/s 10.0 mm/mV  
0.05-35 Hz 50 Hz

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FOLLOW-UP 2004-2009

- Persistent low blood pressures
  - This limits 2\textsuperscript{ary} prevention of HF
- 2006 high INR and hematuria after injection of cortisone
- 2007 exercise induced dyspnea
- 2009 NYHA grade III
  - NT-pro-BNP = 1520 pg/ml
FOLLOW-UP 2009

• 2009 exercise test
  • frequent VPB with many doublets and triplets

• 2009 Holter monitoring:
  • 25% VES
  • numerous episodes of bigeminy, trigeminy, quadrigeminy
DRUG THERAPY IN 2009

• Spironolactone 12.5 mg o.a.d.
• Valsartan 80 mg o.a.d.
• Carvedilol 12.5 mg b.i.d
• Warfarin 6.25 mg o.a.d.
• Simvastatine 40 mg o.a.d.
WHAT IS GOING ON?

• Do we need additional information?
  • Is there something we miss?

• How to treat?
DO WE NEED ADDITIONAL INVESTIGATIONS?

• What we did ............

• TOE
  • No paravalvular leak

• Heart catheterisation and coronary angiography
HEART CATHETERISATION (2009)

- HR 57 pm; CO = 3.9 l/min and CI 2.4 l/min
- EF 0.28
- SVR 1872 dyne*s/cm\(^5\) and PVR 123 dyne*s/cm\(^5\)
- Normal patent coronary arteries. No significant MR.
DIAGNOSIS

• HFrEF

• Causes for HF are:
  1. Arterial hypertension
  2. Severe and longstanding volume overload referred too late for surgical correction
  3. Mitral valve replacement
  4. Peroperative apical infarction
WHAT TO DO NEXT?

1. Antiarrhythmic therapy with amiodarone
2. Antiarrhythmic therapy with other drug
3. VVI-ICD backup system one lead
4. DDD-ICD backup system two leads
5. CRT-ICD system three leads
6. Consider heart TX
WHAT WE DID.....

• Amiodarone 200 mg o.a.d.
• Backup VVI-ICD in primary prevention
EVOLUTION 2009-2011

- Increasing complaints of exercise-induced fatigue, low blood pressure and dizziness alternating with exercise-induced dyspnea

- Loss of weight till 55 kg (euthyroid function)
- BP 130/56 mmHg; HR 51 pm RR. Normal prosthetic heart sounds. No clinical signs of HF

- ECG not modified
- Exercise testing:
  - Peak load 55 Watt (0.59 of prediction)
  - Peak $O_2 = 10.8$ ml/kg/min
  - Peak HR 89 pm. No significant VPB.
DRUG THERAPY IN 2011

• Amiodarone 200 mg o.a.d.
• Spironolactone 25 mg o.a.d.
• Valsartan 80 mg o.a.d.
• Carvedilol 12.5 mg b.i.d
• Warfarin 6.25 mg o.a.d.
• Simvastatin 40 mg o.a.d.
WHAT TO DO?

1. Change drug regimen?
   1. Increase spironolactone
   2. Stop amiodarone
   3. Decrease or stop carvedilol

2. Upgrade VVI-ICD to DDD-ICD?

3. Consider redo surgery?

4. Consider screening for Heart TX?
WHAT WE DID

- Complaints are mainly due to forward failure and there is no really a problem of backward failure
- Bradycardia at rest and during exercise contribute to forward failure
  - We kept amiodarone
  - We decreased and stopped carvedilol
- Alternative would have been DDD pacing
GHENT, A COMPETITIVE UNIVERSITY IN A MEDIEVAL CITY....