Tachycardia-Induced Cardiomyopathy

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## **Case History**

 Z.H., a 59 year old woman was admitted on January 16<sup>th</sup>, 2005 due to shortness of breath
 Her past history included: Type 2 Diabetes Mellitus Arterial Hypertension, well controlled on drugs Carcinoma of Breast with lumpectomy in 2002.



# **Cardiac History**

She was known to suffer from mitral stenosis due to rheumatic heart disease.

Over the years she developed pulmonary hypertension (PA Pressure of 60mmHg) and had chronic atrial fibrillation for 15 years prior to admission.

#### ■ The LVEF was 60%.

 On July 2004 she underwent mitral valve replacement (mechanical), tricuspid valve annuloplasty and right and left atrial MAZE procedure.



### **MAZE Procedure**

The MAZE procedure (using RF and Cryo energy) included:

 Right atrium: Intercaval line Isthmus line

Left Atrium:

Encircling PVs line LIPV to MV Annulus line Posterior line connecting Rt and Lt PV's



### Echocardiogram

An echocardiogram 1 week after surgery :
LVEF of 60% with normal regional contraction
Normally functioning mechanical mitral valve
Non significant tricuspid valve regurgitation
PA pressure of 60mmHg



### **Perioperative course**

- The spontaneous rhythm immediately following surgery was atrial standstill and a slow nodal escape (48/min).
- 2 days later she was in atrial fibrillation with complete AV block and a slow wide QRS ventricular escape.
- A DDDR permanent pacemaker (AT501, Medtronic) was implanted on the 8<sup>th</sup> postoperative day. Both atrial fibrillation prevention algorithm and atrial overdrive therapies were active.



## **Postoperative period**

- During august 2004 she began to notice palpitations.
- An echocardiogram on October 2004 showed LVEF of 50%, some tricuspid regurgitation and PA pressure of 60mmHg.
- In December 2004 she began to suffer from shortness of breath. Holter monitoring showed atrial fibrillation and atypical atrial flutter with rapid ventricular response, partially paced.
   Amiodarone was started.



An echocardiogram on December 23<sup>rd</sup>, 2004 showed a mildly dilated LV with an EF of 35%, a normally functioning mitral valve, estimated PA pressure of 80mmHg, a dilated and hypokinetic RV and mild to moderate tricuspid regurgitation. A Holter monitoring on December 29<sup>th</sup> showed atrial fibrillation and flutter with a mean rate of 100 bpm, mostly paced. Rapid atrial pacing occurred about 30% of the time, believed to represent atrial prevention and therapy.



#### **Current Hospitalization**

- In January 16<sup>th</sup> 2005 she was hospitalized with congestive heart failure.
- An echocardiogram showed further deterioration of LVEF to 20%.
- The ECG showed a paced ventricular rate of 100 bpm, with atrial fibrillation and atrial pacing spikes at 110 bpm.
- Holter monitoring, a few days later, showed mostly sinus rhythm at a mean rate of 77bpm, with intermittent atrial undersensing and non capture.
- Chest fluoroscopy did not disclose lead dislodgement.



### **Current Hospitalization**

- The pacemaker was programmed to VVIR with a lower rate of 45bpm. Drug therapy of her CHF was intensified.
- A week later she underwent atrial lead reposition. Pacemaker interrogation showed that she was in sinus rhythm around 70bpm most of the time.
  The pacemaker was programmed to DDIR 70bpm.
  She remains in normal sinus rhythm.
  The LVEF improved gradually.



### **Final Echocardiogram**

 Echocardiogram on October 7<sup>th</sup>, 2005: LV with normal dimensions and an LVEF of 50%
 RV with mild hypokinesis
 PA pressure of 60mmHg
 Mild tricuspid regurgitation



### Tachycardia-Induced Cardiomyopathy

- This patient developed symptomatic failure of both ventricles within a relatively short period of time. The most likely explanation is tachycardia induced cardiomyopathy (TIC). This is a well known, though relatively unusual cause, of CHF. The rapid recovery of ventricular function following rate control supports the diagnosis.
- β-adrenergic desensitization and dysfunction of the sarcoplasmic reticulum have been found to be the main pathophysiology of experimental TIC



# **Pathophysiology of TIC**

Several features of this case are interesting:

Ventricular dysfunction developed quite rapidly. We have the impression that there are hearts which are more prone to fail, maybe due to a smaller "contractile" reserve". In this woman could a previous rheumatic carditis, an ongoing hemodynamic burden due to pulmonary hypertension and the recent cardiac surgery be contributing factors to both left and right heart failure?



# Likely contributing factors

- The detrimental effect of the rapid and irregular rate of atrial fibrillation could be amplified by the RV apical pacing.
- Pacemaker therapy and prevention algorithms were responsible for the rapid ventricular rate about 30% of the day and may have contributed to the deterioration of the LV function.
- The loss of atrioventricular synchrony due to atrial lead dysfunction may have added to the signs of heart failure.



# Summary

This is a case of biventricular failure due to uncontrolled atrial arrhythmias following MAZE procedure . In our experience these arrhythmias are difficult to control and AVN ablation may sometimes be required. However, ventricular pacing as well as pacing algorithms may be contributory factors to the development of TIC.

