54. Optimized the CRT Improved the Heart Failure For Hypertrophy Myocardiopathy and Left Bundle Branch Block

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Body

Introduction: Hypertrophy myocardiopathy (HCM) is one of the cause of heart failure and difficult treatment. Beside the optimal medical treatment, the cardiac resynchronize therapy (CRT) can improve the heart failure for HCM with left bundle branch block (LBBB)

Case: The reporting case 87-year-old male patient had history hypertension, normal coronary artery and heart failure. He had some episodes of dyspnea and hospitalization for 3 years and improved after optimal medical treatment. This admission, he has had episode of dyspnea and wheezing, mild edema low extremities, heart irregular 87 bpm, lung crackle both sides, mild liver enlargement. Blood test showed normal inflammation and high cardiac marker NTproBNP.

The sinus rhythm with LBBB 152ms and ventricle premature complex bigeminy on ECG and the echocardiography showed left ventricular hypertrophy, EF 47% Simpson, severe mitral regurgitation.

The decompensated heart failure control with optimal medical treatment was performed (Valsartan/Sacubitril 200mg bid, Nifedipine long acting (LA) 60mg, Isosorbide mononitrate 120mg, empaglifozin 10mg, Furosemide 40mg tid) however he has had episode of wheezing and acute pulmonary edema, so the pneumonia was treated as risk factor.

After that, the cardiac MRI was performed and showed more likely the Amyloidosis myocardiopathy and less likely Hypertrophy myocardiopathy. The blood test also showed increasing light chain. So, the fatty biopsy and gene screening hypertrophy myocardiopathy were performed to rule out which disease

For 1 week, the kidney function had been better, the optimal medical treatment with Furosemide 40mg IV tid, Valsartan/Sacubitril 200mg bid, Verospiron 25mg, Tolvaptan 15mg, Isosorbide mononitrate 120mg qd, Nifedipine LA 60mg, and antibiotic was done. The patient still had some episode of dyspnea at night and high NTproBNP dramatically.

The fatty biopsy result said negative congo red amyloid stain. The screening gene of HCM result showed one heterozygous variants of uncertain significance in TNNT2 gene (NM_000364.4) with NextSeq-Illumina system.

Beside the optimal medical treatment for heart failure, the strategy of CRT was performed for HCM with LBBB as Class IIa. The right ventricular (RV) lead was put in the mid septal, the left ventricular (LV) lead was put in the posterior lateral and right atrial lead in right atrial appendage.

After implantation, the optimized of CRT was performed with vector LV2-4, RV to LV 20ms and sensitivity atrial ventricle (SAV) 120ms.

The patient felt better, decreasing the furosemide dose, controlled blood pressure, improved from NYHA IV to NYHA II, decreasing cardiac marker NTproBNP from 56000 to 11000.

Discussion: The 1st changeling here was hypertension was controlled blood pressure, however the patient still has had dyspnea, heart failure NYHA IV and episode of acute pulmonary edema and hospitalization. The left ventricular hypertrophy was rather difficult to rule out the hypertension.

After heart failure was diagnosed with dyspnea and high NTproBNP, low LVEF 47%, the Valsartan/Sacubitril was combined other medications. However there was hard to control the heart failure. Some episodes of dyspnea, acute pulmonary edema still happened and hospitalization.

Then the heart failure due to hypertrophy myocardiopathy has indicated beta blocker which was tried. However the 1st AV block appeared after using medication and make severe conduction disorder. And the episode if acute pulmonary edema was still happened a lot of times that stopped beta blocker.

The heart failure NYHA IV, LVEF 47% was treated with Valsartan/Sacubitril Class IIB haven't showed the good outcome. The finally test was gene screening which showed abnormal for HCM. And the heart failure NYHA IV patient had the conduction disorder LBBB following the indication of CRT-D. Although the heart failure of HCM with LBBB was class IIa, however this is also the last chance to the patient.

During the procedure, the strategy was narrow QRS complex less than natural QRS complex. The RV lead must be put in the mid septal and showed QRS complex less than natural ones. After that the LV lead was put in the posterior lateral to get the QRS complex less than natural one. There 4 electrodes in the LV lead, so the best vector was screening during the lead test.

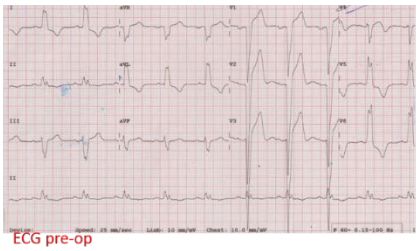
After CRT implantation, programming CRT showed vector LV 2-3, RV and LV same time and SAV 80ms that was good QRS complex 116ms. However there were some episodes of acute pulmonary edema.

Finally, the vector LV2-4, SAV 120ms and RV to LV 20ms showed the good QRS complex 126ms.

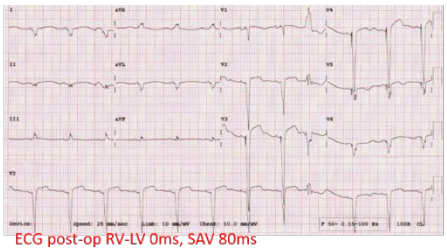
With optimized CRT in HCM was rather difficult due to the thick of the left ventricle, the action of LV lead was sooner or later that also make loss of synchronization between the LV and RV pacing.

After 1 weeks the heart failure improved from NYHA IV to NYHA II, decreasing NTproBNP.

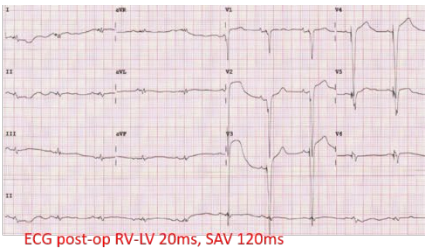
Conclusion: This is difficult to treat the heart failure HCM. The CRT-D implantation is the last chance for the heart failure of HCM with LBBB. And the optimized of CRT was also very important treatment.



ECG pre-op



ECG post-op RV-LV 0ms, SAV 80ms



ECG post-op RV-LV 20ms, SAV 120ms