A super-responder to CRT concealed by silicosis for eight years

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Introduction: An ECG from a 79-year-old man was sent to me when he got a chest tightness during his admission in respiratory ward of our branch hospital. It was a tachycardia with wide QRS, and the sinus rhythm after cardioversion by aminodarone also showed wide QRS complex because of CLBBB(Figure 1, 2). He had been a frequent patient admitting to the department of respiratory medicine at least once a year for 8 years. Eight years ago, he began to suffer dyspnea, and was diagnosed as silicosis since he had been a drifting worker for 3 years when he was around 20 years old. His symptoms of dyspnea aggravated and exercise tolerance reduced rapidly. Now, the old man can only walk 50m (1 block) on level ground or climb 1 floor up. He didn't have any histories of hypertension or diabetes, and neither alcohol addiction nor smoking habit. Physical examination found moist rales with little dry rales over both lungs, cardiac dilated to the left, and systolic murmurs audible at apex, but we couldn't find any signs of pulmonary hypertension. So what on earth were the main causes for his symptoms of breathless. Then the patient was transferred to our department to get further management in May 9th, 2018.

Methods: ECG on admission still showed CLBBB with a prominent wide QRS duration 186ms. Echocardiogram showed obvious left ventricle dilation and severe systolic dysfunction with LVEDD 6.8cm and LVEF 24%. When we reviewed all of his hospital records during the past 8 years, we got some clues listed below: 1, the first ECG recorded in 2010 when he was admitted to pulmonary department was CLBBB and the QRS duration was 133ms, the QRS duration became wider gradually through the 8 years and now it was 186ms. 2, he had 5 echocardiograms during the 5 past years, showed his LVEDD from 5.8cm to 6.8cm and LVEF from 40% to 24% now, while the right ventricle size was normal through all these years. 3, the chest X rays and CT pulmonary scans examined almost every year showed no obvious change signs although there were some small nodules in the upper lung areas without any progressions. These findings confirmed us that he suffered a chronic heart failure even more than his silicosis since his cardiac abnormalities aggravated while the pulmonary disease was stable. There was another episode we got from his records. Two years ago when he had the chest tightness at first time, he was suspicious of AMI because of a mild elevation of TnI and received a coronary angiography. No obvious stenosis were found and no stents planted. But he was prescribed with optional medicine therapy, including ACEI-$\beta$-blocker, and aldosterone antagonist because of the reduction of LVEF but still his symptoms deteriorated. We did a CTA scan again to completely exclude possible IHD. Hence the ultimate diagnosis for this patient was revised to dilated cardiomyopathy and silicosis.

Result: According to the guidelines, he had a strong indication for CRT, and we know patients with LBBB, wider QRS, and non-ischemic can get magnitude of benefit from CRT. It's very hopeful that he could be a good responder to CRT. So a CRTD (Viva Quad XT CRT-D DTBA2QQ, Medtronic) was implanted successfully in May 16th. All of the parameters were excellent, adaptive CRT was programmed ON, and the pacing ECG showed a beautiful narrow QRS within 120ms (Figure 2), patient’ symptoms relieved significantly shortly after implantation. Seven months later, the patient complained of abdominal beating. Unfortunately phrenic nerve stimulation happened. Instant ECG showed normal
capture of ventricle, due to adaptive CRT, total VP actually LV pacing was pretty good at a rate of 98%, thresholds and other parameters were pretty well as before. Then chest x-ray revealed it. It seemed that LV lead went deeper than before. When compared to the last one at 3 days post-implanting, we found it was not the lead moving, but the cardiac size decreased sharply, much more clear compared to pre-implantation (Figure 3). And echocardiogram showed us a nearly normal heart with LVEDD 5.0cm and LVEF 60%. Since the threshold was good, we decreased the output and the stimulation alleviated. Five months later, when the patient returned for his 1 year follow-up, we are surprised to see a totally normal heart, LVEDD 4.5cm and LVEF 67%. He is indeed a super responder to CRT.

Conclusion: This story sounds really wonderful, but I share this case not because of its amazing results. Think about his suffering for 8 years before, this is key point I want to share: what is the most important aspect for this patient’s successful therapy? Although a good technology CRT is vital, as well as the new functional adaptive CRT gives him more benefits, the process the patient getting his correct diagnosis is far more important. This case illustrated that we should pay more attention to our patients, analyze all the data carefully and comprehensively, before we make a final diagnosis and decision.