Malfunctioned cardiac resynchronization therapy attributed to acute exacerbation of cardiac sarcoidosis

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A 39-year-old man with isolated cardiac sarcoidosis who received cardiac resynchronization therapy-defibrillator implantation was undergoing gradual prednisone tapering in outpatient clinic by monitoring disease activity with fluorine-18 fluorodeoxyglucose positron emission tomography (18F-FDG PET). His myocardial 18F-FDG uptake was disappeared completely (Panel A), and his prednisone was further tapered.

He was hospitalized for acute decompensated heart failure requiring catecholamine (Panel B). Echocardiography showed left ventricular (LV) dyssynchrony and electrocardiogram exhibited a marginal change suggestive of LV alone pacing. Pacemaker interrogation revealed a marked rise in the right ventricular (RV) capture threshold leading to failure of RV pacing (arrow). Serum angiotensin-converting enzyme and soluble interleukin-2 receptor level were within normal limits. 18F-FDG PET identified increased 18F-FDG uptake including LV inferior and RV, indicating acute exacerbation of cardiac sarcoidosis (Panel C). His prednisone was increased and RV pacing output was elevated. In response to increased prednisone, catecholamine was tapered and 18F-FDG uptake was diminished (Panel D). Intriguingly, RV capture threshold was gradually decreased correlating with resolution of cardiac sarcoid activity. The patient was discharged home with greatly improved symptoms.

Despite of periodic examination in our outpatient clinic, the recrudescence of cardiac sarcoidosis associated with tapering prednisone process caused pacemaker failure to capture attributed to elevated RV myocardial pacing threshold, resulting in cardiac dyssynchrony and decompensated heart failure. The ECG change at the time of acute exacerbation was less obvious by the nature of his extended myocardial conduction disturbance, and only 18F-FDG PET told us the exact mechanisms of heart failure.

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