A patient with persistent sinus arrest was evaluated on electrophysiology and right atrial voltage mapping using a 3-D electroanatomical mapping (CARTO) system. The system was useful for visually identifying the injured area after suspected acute myocarditis.

A 55-year-old man was referred to hospital after experiencing a prolonged fever that persisted for 3 days even though he was prescribed medicine at another clinic. He complained of nausea and chest pain on admission and had no past history of this. He was diagnosed as having acute myocarditis based on

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blood examination, 12-lead electrocardiogram (ECG), transthoracic echocardiogram, and computed tomography. A 12-lead ECG on day 1 showed junctional rhythm without P waves and ST segment elevation in leads I, aVL, and V2–6, and ST segment depression in leads II, III, and aVF (Figure 1A). We performed emergency cardiac catheterization and pericardiocentesis because of hemodynamic instability due to cardiac tamponade. Left ventriculography showed regional hypokinesis in septal and apical walls. Coronary angiography showed no significant stenosis. Unfortunately, endomyocardial biopsy was not performed. Treatment included aspirin, gammaglobulin, and antibiotics. Paired serology for viral antibodies detected no specific pathogens. He suffered from acute respiratory distress syndrome possibly due to acute pneumonia on day 5. The patient was given steroids (methylprednisolone 62.5 mg/day) as well as sivelestat sodium hydrate 250 mg/day because of critical respiratory failure. The same night the bradycardia gradually worsened, leading to Torsades de pointes. A temporary pacemaker was inserted and artificial ventilation was continued. His general condition improved, except for sustained absence of P waves. The wall motion of both atria was akinetic (Movie S1) and transmitral and trans-tricuspid inflow velocities were monophasic without A on echocardiography even 14 months after the acute myocarditis (Figure 1B). On day 27, his heart rate remained at 40–50 beats/min. He underwent electrophysiology and CARTO system. Intracardiac electrograms (Figure 2A) showed isolated His bundle and ventricular electrograms a normal HV interval, and no atrial depolarization was recorded from the electrodes in the right atrial appendage and coronary sinus. Stimulation at 10 V in the coronary sinus at CS 5–6 did not capture the atrium. Consecutive bipolar voltage mapping and pace mapping at 10 V with the CARTO system did not show either any atrial depolarization or any atrial capture at each site in the entire right atrial chamber, which suggested that the right atrium had lapsed into electrical scar (Figure 2B). Atropine sulphate and isoproterenol increased the heart rate from 40 to 50 beats/min, but did not enable pacing. In order to detect possible atrial depolarization, we recorded local high-gain bipolar electrograms at 49 sites in the right atrium including the septum, septum near the His bundle, free wall, and high free wall, but only noise electrograms (0.03 mV at 22 sites, 0.02 mV at 26 sites, and 0.01 mV at 1 site) could be detected. These data indicate that there was at least no atrial electrograms larger than 0.03 mV.
Thus, we considered these sites as electrical scar and colored these sites as gray in the CARTO mapping. Given that the patient recovered well, with the exception of electrical scar in the atrium, we decided to implant a dual-chamber pacemaker in DDD mode. The thresholds of sensing and pacing were within normal range. An atrial lead was also implanted for potential recovery in the future, and the pacemaker mode was set to VVI-R mode. The patient was discharged on hospital day 38. Atrial depolarization was not detected in a pacemaker check-up 9 months after implantation and the atrium could not be paced even at 7.5-V stimuli. A 12-lead ECG at a check-up 5 months before admission showed normal sinus rhythm with P waves, and saddleback-type ST segment elevation in right precordial leads (V1 and V2), which suggested Brugada type ECG findings. The patient, however, had not experienced palpitations or syncope episodes before the admission. There was no family history of sudden cardiac death. According to the patient, he had no family members who had abnormal ECG findings.

We encountered a rare case of suspected acute myocarditis complicated with persistent sinoatrial arrest. The atrium seemed to be predominantly impaired, although the left ventricle was also transiently impaired. CARTO system was useful for visually identifying the injured area.

Sinus arrest due to acute myocarditis has been reported in animals. In humans, acute atrial myocarditis has been proposed as a mechanism of atrial arrhythmia, but not of sinus arrest. In these cases complete resolution of atrial myocarditis occurred spontaneously after 6 months. Thus, to the best of our knowledge, this is the first human case of sustained sinoatrial arrest. The atrium seemed also transiently impaired. CARTO system was useful for visually identifying the injured area.

The present diagnosis of acute myocarditis was not definite because the patient did not undergo endomyocardial biopsy, cardiovascular magnetic resonance imaging, or voltage mapping of the ventricle. Despite this, the likelihood of acute myocarditis was high based on the other clinical data.

References

Supplementary Files
Supplementary File 1
Movie S1. Atrial wall motion of both atria on apical 4-chamber view. There was no atrial contraction.
Please find supplementary file(s); http://dx.doi.org/10.1253/circj.CJ-12-0475