We describe a case of a 65-year-old patient who was hospitalized in our department due to decompensated heart failure and was diagnosed having atrial standstill.

A 65-year-old patient was referred to our department from the emergency room for cardiological consultation due to worsening dyspnea. He was diagnosed as having sick sinus syndrome a few years earlier, for which a dual-chamber pacemaker (DDDR) had been implanted.

On admission, the patient was dyspneic, having 24 breaths/min, his blood pressure was 133/96 mmHg, and pulsed oximetry showed 92% oxygen saturation. Physical examination revealed diminished breath sounds in the right lower lung region, increased jugular venous pressure with absent A waves and mild pedal edema. Chest radiography revealed right pleural effusion. The surface ECG showed pacemaker rhythm with atrio-ventricular sequential pacing, but there were no P waves visible following the atrial spike even using maximum ECG sensitivity and ventricular extrasystoles (Figure 1). The echocardiogram showed excessive biatrial enlargement, absent A waves when recording both the right and left ventricular inflow, mildly dilated right ventricle with tricuspid regurgitation and estimated systolic right ventricular pressure of 35-40 mmHg, as well as depressed left ventricular ejection fraction, visually estimated to be around 40-45%. No thrombus, spontaneous echo contrast or intracardiac shunts were detected (Figures 2, 3).

We proceeded with pacemaker interrogation and programming. The pacemaker was programmed to dual-chamber pacing function as was previously mentioned. The atrial and ventricular electrode parameters were normal. There was no electrical activity recorded via the atrial electrode even when adjusting atrial sensitivity to maximum. We then paced the atrium with maximum generator output. Under echocardiographic monitoring we paced the atrium at 7.5 V but could not obtain atrial capture. The atrial electrode recorded no atrial electrical activity; no P waves were visible on the surface ECG and we could not record A waves in either left or right ventricular inflow tract recordings. Thus, we finally programmed the pacemaker to VVIR mode.

The patient received intravenous diuretics and vasodilators to treat heart failure symptoms. Anticoagulation therapy was also initiated with oral anticoagulant bridged with unfractionated heparin to cover for possible thromboembolism due to the diag-
nosed atrial standstill. Subsequently, the patient was referred for coronary angiography, which revealed non-significant coronary artery disease managed medically.

**DISCUSSION**

Atrial standstill is a rhythm disorder in which there are no P waves in the surface ECG recordings, an atrial electrogram (EG) cannot be recorded from intra atrial electrodes and the atria cannot be paced by electrical stimuli delivered through electrodes in contact with the atria.\(^1\) It was first described in 1946.\(^1\) The diagnostic criteria involve the following: 1) Absence of P waves in the ECG and of A waves in the intracardiac recordings; 2) narrow QRS complexes; 3) evidence of atrial paralysis (absence of A waves in the jugular venous pulse, in the
atrial pressure recording and in the mitral Doppler recording); and 4) inability to stimulate the atria. Permanent atrial standstill can occur mainly in three clinical settings: 1) Long standing cardiac disease, 2) In patients with neuromuscular disease, and 3) During evaluation of vertigo, syncope or stroke.

In our case, a patient with long standing cardiac disease, permanent standstill probably developed as a result of myocardial dysfunction and fibrosis of the right atrium. The first stage is temporary atrial standstill, during which the patient responds to atrial pacing. Progression of the disease leads to a state where there is evidence of absent atrial activity on surface electrocardiograms but a very low atrial potential is recorded through intracavitary recordings and there is atrial contraction through high voltage pacing. Finally, as the disease progresses, there is no evidence of atrial activity on surface and intracavitary recordings, no evidence of A waves in pulsed wave Doppler echo interrogation and no response to high amplitude atrial pacing, similar to our case. Treatment of atrial standstill should be targeting possible complications, including syncope and embolic events. Treatment with permanent pacing and anticoagulation is deemed mandatory.

REFERENCES