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Letter to the Editor
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A Rare Cause of Recurrent Cardiogenic Shock and Flash Pulmonary Edema-Concealed Atrial Lead Dysfunction

Yuan-Kun (Aden) Wu,[1] Chih-Chieh Yu[1,2] and Jiunn-Lee Lin[2,3]

Key Words: Acute heart failure • Acute mitral regurgitation • Atrial arrest • Dyssynchrony • Pacemaker dysfunction

Abbreviations	
CIED	Cardiac implantable electronic device
ECG	Electrocardiography
LA	Left atrial
LV	Left ventricular
MR	Mitral regurgitation

INTRODUCTION

For post-cardiac implantable electronic device (CIED) implantation patients, electrophysiologists rely on programmer interrogation to retrieve the stored data and manually check the pacing-lead function during interrogation. However, when all these data fail to yield any clues to the abnormality, the malfunction of the pacemaker might not be detected; therefore, the necessary measures would not be carried out. Here, we describe a case of a patient with a dual-chamber pacing for sick sinus syndrome for decades who presented with recurrent acute pulmonary edema and cardiogenic shock. The diagnosis of CIED dysfunction was delayed for approximately 1 year.

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[1]Division of Cardiology, Department of Internal Medicine, National Taiwan University Hospital; [2]Department of Internal Medicine, College of Medicine, National Taiwan University, Taipei; [3]Cardiovascular Center, Taipei Medical University Shuang-Ho Hospital, New Taipei City, Taiwan.
Corresponding author: Dr. Chih-Chieh Yu, Division of Cardiology, Department of Internal Medicine, National Taiwan University Hospital, No. 7, Chung San South Road, Taipei 100, Taiwan. Tel: 886-2-2312-3456 ext. 65257; Fax: 886-2-2395-1841; E-mail: ccyu2014@ntu.edu.tw

CASE

A 72-year-old woman with a history of sick sinus syndrome status post first dual-chamber pacemaker implantation in 1996, followed by generator exchanges in 2006 and 2015, and new ventricular lead implantation in 2020, presented to us with repeated episodes of acute pulmonary edema since the beginning of 2021. She recovered from each episode within a couple of days without an apparent identifiable cause. Thereafter, she was maintained under a heart failure post-acute care management program with a strictly regulated diet under the observation of a case manager and showed excellent adherence.

This time, she developed similar symptoms and presented to the emergency room a few hours later. The patient was hypotensive with a blood pressure of 68/53 mmHg and had a pulse rate of 86/min. Her respiratory rate was 28 breaths/min with oxygen saturation of 94% under a nasal cannula at 3 L/min. Physical examination revealed bilateral crackles with a grade IV/VI holosystolic murmur over the apex, with radiation to the axilla. No pitting edema over dependent parts was observed.

Electrocardiography (ECG) showed a predominantly dual-chamber pacing rhythm (Figure 1A). The P-wave was indiscernible, without significant evidence of underlying atrial pathology or atrial capture failure. Complete blood counts and basic metabolic panel findings were unremarkable. N-terminal pro-B-type natriuretic peptide level was 383.2 pg/mL (normal range: < 125 pg/mL). Troponin-T level was 15.68 ng/L (normal range: < 14 ng/L) and decreased on repeated testing. Chest radiography indicated bilateral pulmonary edema with ade-

quately positioned A and V leads.

Echocardiography showed severe mitral regurgitation (MR) with dilated left ventricular (LV) and left atrial (LA) sizes (LV end-diastolic diameter: 54 mm, LA dimension: 48 mm; LA volume: 101.7 mL) and normal LV ejection fraction (62.7%) (Figure 1B). The regurgitant jet was centrally directed, without leaflet motion abnormality or anatomical destruction. The severity of MR was significantly increased compared with that on echocardiographic images 2 weeks prior, without a noticeable increase in chamber dimension.

Inotrope and vasopressors were administered for cardiogenic shock at the emergency department, and the cardiology team admitted the patient to the cardiac critical care unit to investigate the etiology of acute decompensated heart failure.

Surprisingly, her inotrope demand dropped markedly without definite intervention 1 hour after admission. An ECG revealed atrial pacing with intrinsic conduction rhythm (Figure 2A). Repeated echocardiography showed significantly reduced MR severity (Figure 2B) comparable to her baseline condition. Occasional loss of atrial capture with ventricular pacing persisted, and the maximum generator output could not regain atrial capture. Atrial lead impedance was within the normal range during interrogations (Table 1). No atrial high rate episode was recorded within the past one month. There-

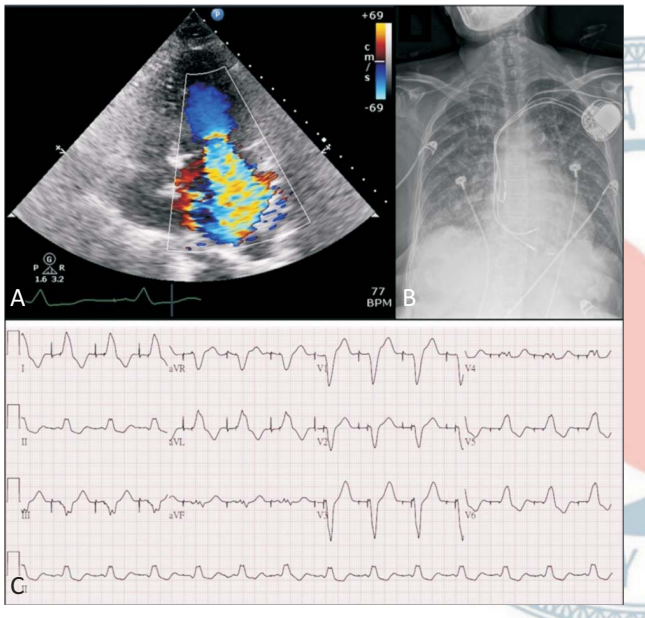

Figure 1. *Initial presentation: severe mitral regurgitation (A) with bilateral pulmonary edema (B) while under dual-chamber pacing with atrial capture failure (C).*

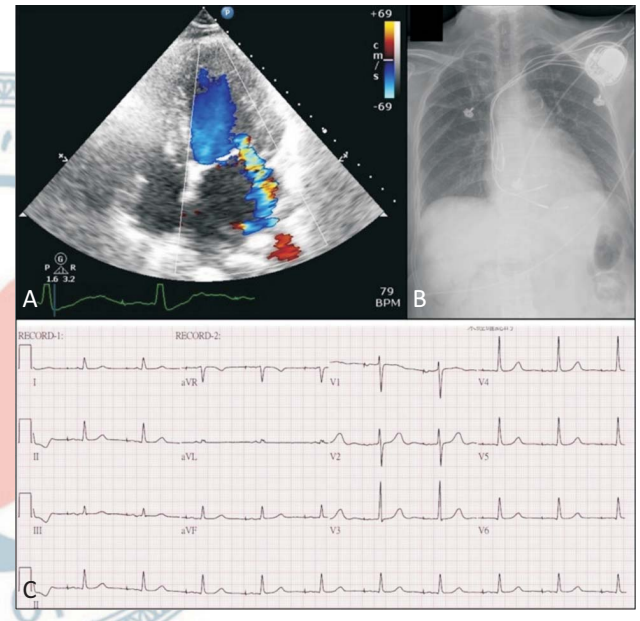

Figure 2. *After transfer to the cardiac intensive care unit, mitral regurgitation severity reduced dramatically (A) with rapid resolution of pulmonary edema on the day after admission (B) while under atrial paced rhythm (C).*

Table 1. Serial atrial lead interrogation parameters

	Baseline reading (one year ago)	Episode reading	New A-lead reading (6 months later)
Threshold (V at ms)	0.875 V at 0.40 ms	1.0 V at 0.40 ms	0.5 V at 0.40 ms
P/R (mV)	0.18	0.18	0.35
Impedance (Ohms)	588	527	519
Ap-Vp (%)	1.4	14.3	0.3
Ap-Vs (%)	98.5	83.8	99.7
As-Vp (%)	< 0.1%	1.5	< 0.1%
Total Vp (%)	< 1.5%	15.8%	< 0.4%
Atrial high rate episodes (hrs/day)	< 0.1	0.2	0

fore, intermittent loss of atrial capture and right ventricular pacing causing acute severe MR was suspected as the etiology of acute lung edema. A new atrial lead was implanted with no complication. The patient was discharged without any further episodes of discomfort for the following 9 months.

DISCUSSION

This case demonstrates a rare cause of recurrent acute pulmonary edema and cardiogenic shock. The diagnosis of atrial lead dysfunction is challenging owing to its elusive nature. However, identifying the etiology in time is pivotal since lead dysfunction is a manageable condition to prevent a devastating outcome.

A possible mechanism of intermittent atrial noncapture is focal fibrosis,[1] which may cause an unexpected exit block. Because the trends of impedance and threshold might not deviate during the routine pacemaker interrogation, lead dysfunction can remain unsuspected. Therefore, pacemaker dysfunction is often missed in an acute setting, which delays the diagnosis and treatment.

Chronic right ventricular pacing is associated with an increased incidence of dyssynchrony-induced cardiomyopathy, atrial fibrillation, and increased risk of heart failure hospitalization.[2,3] Therefore, the patient's right ventricular pacing burden was minimized by setting a long atrioventricular interval and using Managed Ventricular Pacing (MVP^{TM}). When the atrial lead is working properly, right ventricular pacing is minimized. In our case, when the the atrium lost the capture from the atrial pacing lead, there was no escape rhythm either from the atrium or His-Purkinje conduction system before pacemaker ventricular pacing. The pacemaker shifted to the dual-chamber pacing mode, and, immediately, the patient lost not only atrial kicking but also intraventricular synchrony. In this patient, the loss of intraventricular synchrony caused not only altered LV activation and depolarization sequences resulting in increased intraventricular filling pressure but also altered mitral apparatus activation; both aggravated the severity of MR, causing acute decompensated heart failure.[4-6]

The underlying mitral pathology of the patient might have contributed to this consequence. The patient had dilated atria and barely coapted mitral valve leaflet at the baseline. She had moderate functional MR when the pacemaker was in optimal status. As soon as the atrioventricular and intraventricular synchrony were lost, the severity of MR immediately worsened, making the already on-the-edge heart decompensated. Therefore, the disease courses were very sudden and devastating each time. If there had been no underlying mitral pathology or the severity of MR had been less severe at baseline, the manifestation might have been different.

The presentation of CIED dysfunction can be very diverse. The alertness of the physician is pivotal in prompt diagnosis and early treatment.

LEARNING POINTS

CIED dysfunction can be very vague and concealed sometimes. In patients presenting with heart failure with an undetermined cause, CIED dysfunction should always be considered even when the initial pacemaker interrogation reveals no abnormality. Repeated testing or longer-term monitoring might be needed to clarify the true etiology.

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DECLARATION OF CONFLICT OF INTEREST

All the authors declare no conflict of interest.

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