Indian Pacing and Electrophysiology Journal 20 (2020) 41–45

Contents lists available at ScienceDirect

Indian Pacing and Electrophysiology Journal

journal homepage: www.elsevier.com/locate/IPEJ

Hypokalemia unmasking underlying premature ventricular contraction induced polymorphic ventricular tachycardia: Low potassium is not always the culprit!

Manish Kumar [a, b, *], Anjali Sharma [a, b], Mark A. Marieb [a, b], David I. Silverman [a, b], Edmond M. Cronin [a, b]

[a] Hartford Healthcare Heart and Vascular Institute, Hartford Hospital, Hartford, CT, USA
[b] University of Connecticut School of Medicine, Farmington, CT, USA

ARTICLE INFO

Article history:
Received 12 June 2019
Received in revised form
26 September 2019
Accepted 12 December 2019
Available online 16 December 2019

Keywords:
PVC
PMVT
Hypokalemia
Cardiac arrest
Catheter ablation

ABSTRACT

Hypokalemia is prevalent in patients resuscitated from out-of-hospital cardiac arrest and can contribute to polymorphic ventricular tachycardia (PMVT) by prolonging the QT interval. We present an interesting scenario of malignant ventricular arrythmia initially attributed to moderate hypokalemia that persisted after correction of potassium. Subsequent electrophysiological study showed two frequent PMVT-triggering PVCs mapped to the base of the antero-lateral papillary muscle and the para-Hisian region of the right side of the interventricular septum. The patient underwent catheter ablation to prevent further recurrences and dual chamber ICD implantation for secondary prevention.

1. Introduction

Hypokalemia is prevalent in patients resuscitated from out-of-hospital cardiac arrest, and has been seen independent of prior therapy with diuretics, digoxin or propranolol, arterial pH, epinephrine or bicarbonate administration during resuscitation [1,2]. Hypokalemia may contribute to polymorphic ventricular tachycardia (PMVT) by prolonging the QT interval, especially in susceptible patients such as those with structural heart disease. We hereby describe an interesting Case of malignant ventricular arrythmia initially attributed to moderate hypokalemia that persisted after correction of potassium and subsequently found to have triggering premature ventricular complexes (PVCs) on mapping.

Case report: A 77-year-old female with hypertension, left ventricular hypertrophy, hypothyroidism, and left bundle branch block (LBBB) presented to the emergency department of a community hospital with recent onset of dizzy spells and urinary incontinence while sleeping. She had recently received a 7-day course of acyclovir for shingles and amoxicillin for a presumed urinary tract infection. Her home medications included Indapamide, Felodipine, Levothyroxine, and Rosuvastatin. On arrival, physical examination and vital signs were unremarkable except for a blood pressure of 184/47 mm of Hg. Initial laboratory work revealed serum potassium of 3.1 mmol/liter (mmol/L). EKG at presentation showed sinus rhythm with left bundle branch block (LBBB), frequent PVCs and moderately prolonged corrected QT interval. She was given potassium supplementation and was planned to be discharged home from the emergency room, however, she had two episodes of syncope from PVC-triggered polymorphic ventricular tachycardia PMVT/VF, both terminated by defibrillation. Amiodarone infusion was started and she was transferred to our hospital for electrophysiology evaluation.

EKG upon arrival to our hospital (Fig. 1A) showed sinus rhythm with left bundle branch block (LBBB) and high-grade ectopy. Her corrected QT interval was 599 milliseconds (ms). Serum potassium upon arrival to our hospital was 2.5mmol/L. The initial hypothesis was that left ventricular hypertrophy and LBBB had rendered her more vulnerable to PMVT/VF in the setting of hypokalemia due to indapamide use. Her episodes of nocturnal urinary incontinence

* Corresponding author. UCONN Health, Internal Medicine Residency Program, 263 Farmington Avenue, Farmington, CT, 06030-1235, USA.
E-mail address: mkumar@uchc.edu (M. Kumar).
Peer review under responsibility of Indian Heart Rhythm Society.

https://doi.org/10.1016/j.ipej.2019.12.006

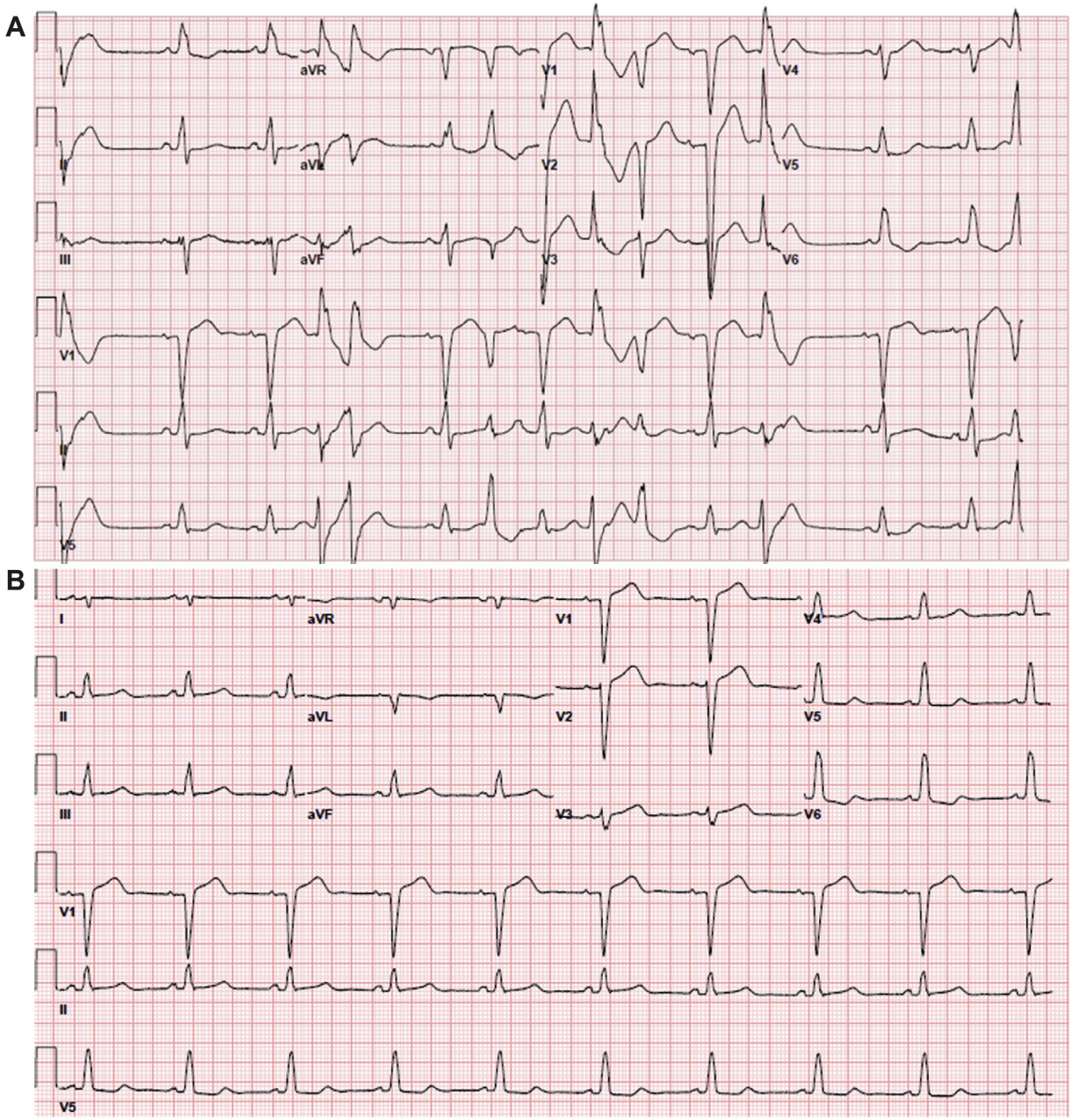

Fig. 1. EKG upon arrival to our hospital (A), showing sinus rhythm with LBBB and frequent ectopy of two different morphologies. EKG after potassium and magnesium repletion (B).

were thought to be secondary to prolonged syncope from PMVT/VF. There was no family history of sudden cardiac death, cardiomyopathy, or arrhythmia. Isoproterenol infusion was started to increase the heart rate and shorten the QTc. Amiodarone was stopped and the patient received aggressive intravenous potassium and magnesium repletion. Serum potassium levels subsequently normalized with improvement in the ectopy and normalization of QTc interval. The patient, however, continued to have frequent PVCs that seemed to originate from the antero-lateral papillary muscle and the para-Hisian region (Fig. 2A and B), and one episode of self-terminating PMVT, despite normal serum potassium (4.5 mmol/L) and magnesium (2.2 mmol/L), and a normal QTc interval (accounting for LBBB). A coronary angiogram showed very minimal coronary artery disease and excluded acute ischemia as a cause. Transthoracic echocardiogram showed mild concentric left ventricular hypertrophy with borderline normal ejection fraction (52%). Cardiac magnetic resonance imaging (CMR) showed normal chamber size with no signal abnormality, myocardial fatty infiltration or delayed enhancement. She was therefore felt to have PVC-triggered PMVT/VF, likely exacerbated by hypokalemia, which was initially thought to be the only cause.

She underwent catheter ablation to prevent further episodes. Two frequent PVCs consistent with the morphologies which triggered PMVT/VF were mapped to the base of the antero-lateral papillary muscle and the para-Hisian region of the right side of the interventricular septum, approximately 2 cm below the His bundle electrogram, respectively (Figs. 3 and 4). Both PVCs were successfully ablated with irrigated radiofrequency (35W, 30

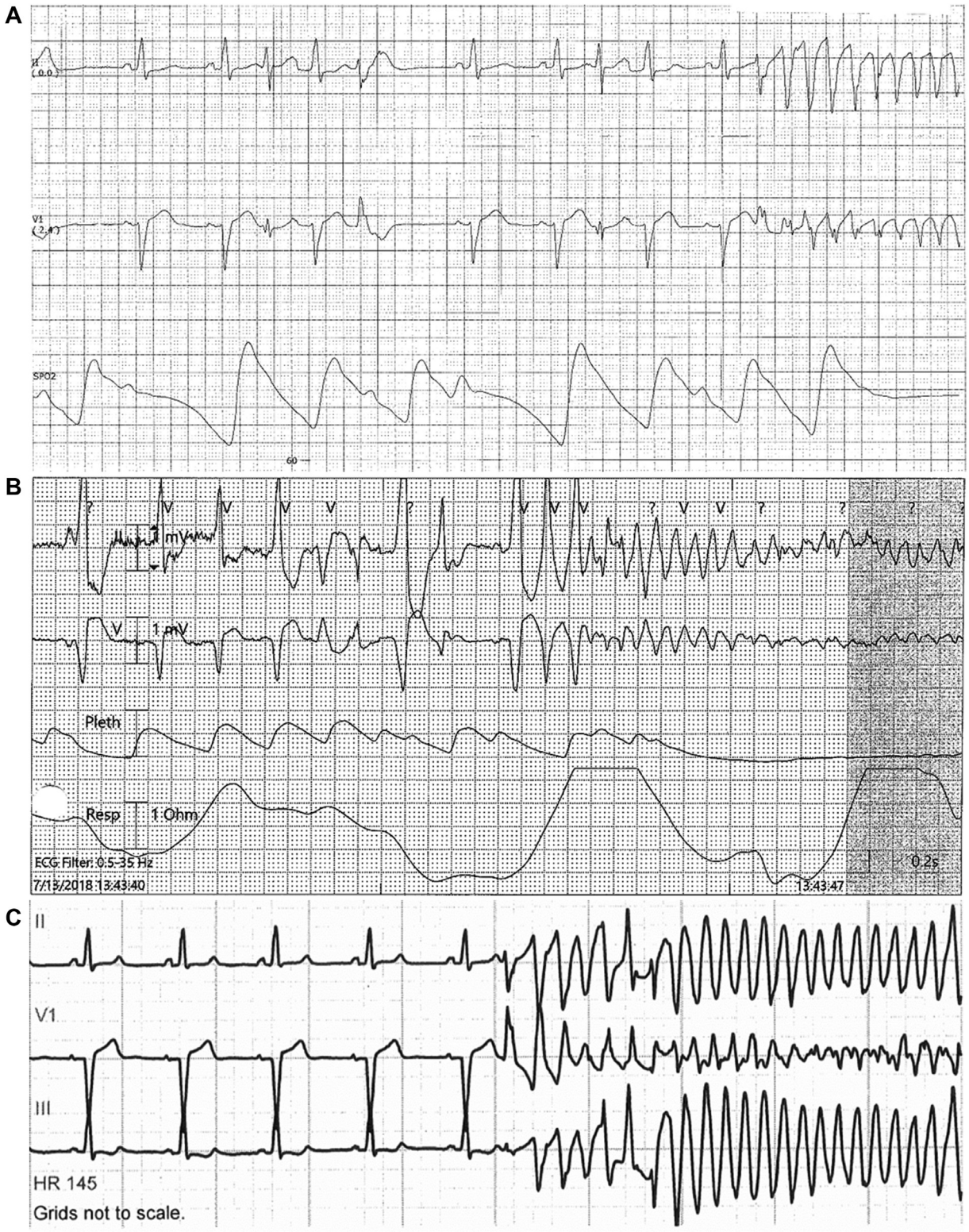

Fig. 2. PVC arising from antero-lateral papillary muscle (A) and para-Hisian (B) triggering PMVT. Recurrent episode of PMVT (C) despite normal serum potassium and QTc, initiated by a PVC from the antero-lateral papillary muscle.

seconds). Given the recurrence rate of PVC induced VF despite acutely successful ablation, a dual chamber ICD was implanted. The patient was followed up in clinic 30 days later, at which point device interrogation did not show any evidence of recurrence of ventricular arrythmia.

2. Discussion

PMVT is a type of ventricular arrhythmia characterized by continuously changing QRS configuration from beat to beat, indicating a changing ventricular activation sequence [3]. It generally

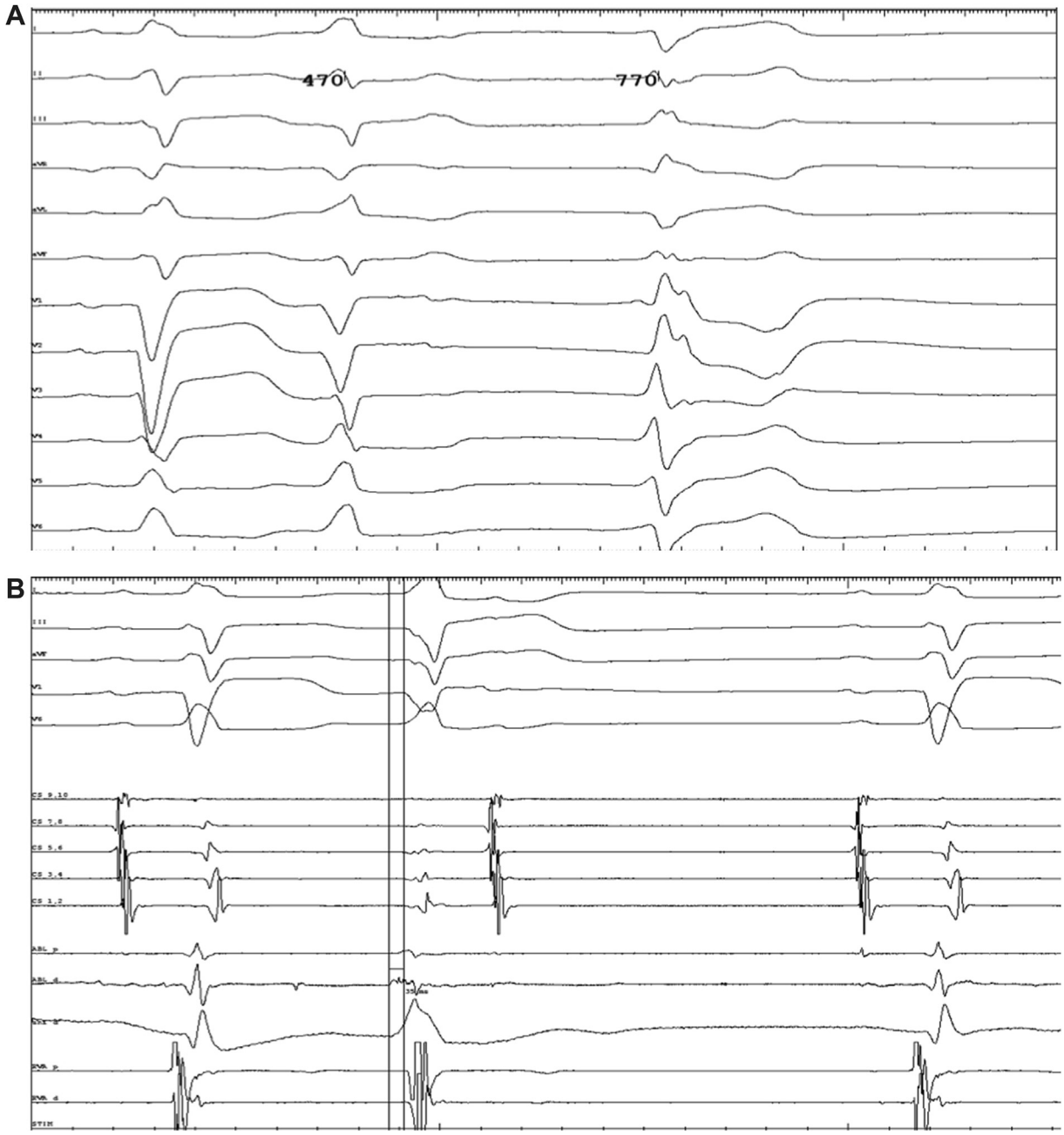

Fig. 3. A: 12-lead EKG in the electrophysiology lab showing a sinus beat with LBBB, the para-Hisian PVC and anterolateral papillary muscle PVC. B: Site of earliest activation of the para Hisian PVC, on the basal RV septum (a little below the His electrogram). Channels are, from top to bottom: surface ECG leads I, III, aVF, V1, and V5; decapolar coronary sinus catheter, from proximal to distal; ablation proximal and distal; unipolar electrogram from the distal ablation catheter (note the connection is reversed from conventional, resulting in a positive deflection); and a quadripolar catheter in the right ventricular apex, proximal to distal.

arises in the setting of structural heart diseases such as ischemic heart disease, channelopathies such as long QT syndrome, electrolyte abnormalities, and drug adverse effects or interactions and either terminates spontaneously, or degenerates to VF [3].

Hypokalemia is an important cause of PMVT/VF, and is seen in as many as 40% of patients on thiazide diuretics [4] and almost 50% of patients resuscitated from out-of-hospital ventricular fibrillation [2]. Other etiologies of hypokalemia include intracellular shifts as happens with insulin administration or activation of sympathetic system, gastrointestinal or renal loss of potassium, and drugs such as diuretics and penicillin. Given the association of hypokalemia with PMVT, it should be avoided and corrected when found. However, in this case, long-term thiazide diuretic therapy meant that the moderate hypokalemia seen was not likely new and had been well tolerated. The low potassium level in our patient, however, unmasked the underlying arrhythmogenic focus. Moreover, the hypokalemia did not explain the frequent PVCs of two consistent morphologies, and careful analysis of the telemetry strips from the referring hospital showed these two PVCs had triggered the episodes of PMVT/VF, as well as the non-sustained episode seen at our institution in the setting of normal serum potassium.

Triggering of PMVT/VF by frequent PVCs is well described [5]. At times, work up may fail to reveal any apparent cause of VF and in such conditions, is termed “idiopathic VF” [6]. PVCs which trigger

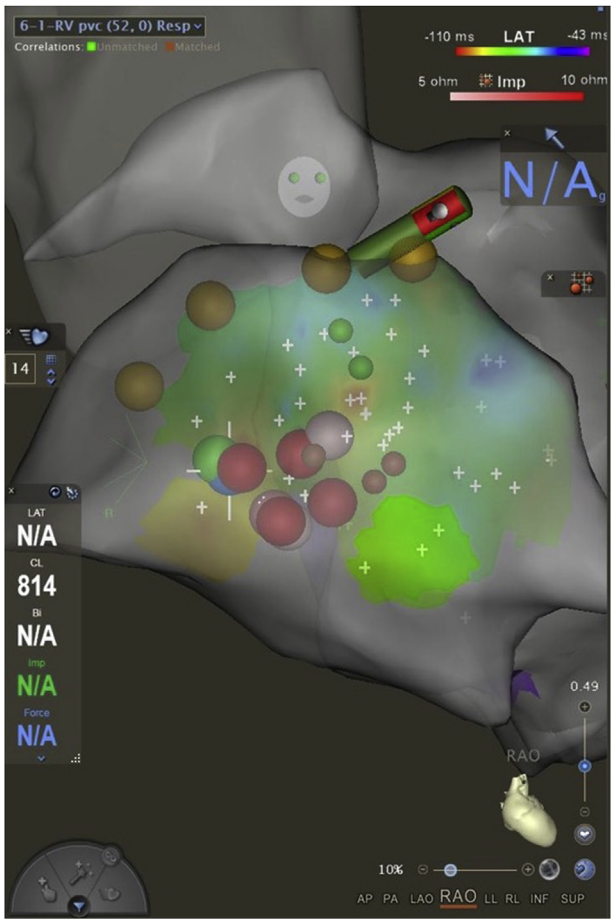

Fig. 4. Electroanatomic activation map (Carto 3, Biosense Webster, Diamond Bar, CA) in right anterior oblique projection showing the site of ablation of the paraHisian PVC. The yellow dots denote the His bundle and proximal right bundle electrograms; the pink and red dots represent ablation lesions. Radiofrequency current was initially delivered slightly further into the ventricle without effect. On mapping on the annulus, an earlier, low amplitude signal was seen. The green dot represents the site of successful ablation.

ventricular arrhythmias can also occur in the setting of structural heart disease, and after myocardial infarction. While triggering PVCs often arise from the Purkinje network, the right ventricular outflow tract and the papillary muscles are also important sites [7]. Analysis of PVC QRS morphology on telemetry strips, ECGs, and ICD electrograms can suggest the site of origin and is essential for procedural planning [3].

In patients with focally triggered VF, catheter ablation of the triggering PVC is feasible and has a high rate of success [3]. However, given the reported 11–18% recurrence rate in the published literature, an ICD is also recommended [6–8]. Patients with VT/VF thought to be due to a reversible and treatable cause such as electrolyte abnormalities or ischemia were considered to have good prognosis and often do not undergo ICD placement. These patients, however, have been shown to have a similar mortality when compared with patients who were not thought to have a reversible cause of VT/VF [9,10]. These findings suggest that the reversible cause may not be the sole causative agent and it may be unmasking the underlying arrhythmogenic predisposition as highlighted by our case. These patients, hence, require comprehensive evaluation and treatment.

Declaration of competing interest

None.

Acknowledgements

None.

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