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Sustained Ventricular Tachycardia Revealing Arrhythmogenic Right Ventricular Cardiomyopathy With Severe Ventricular Electrical Instability: A Case Report

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Ayoub Laaboussi [1], Nada Douari [1], Wassim Beladel [2], Amine El Houari [1], Khalil Abderrahmane Elbaz [3], Mohamed El Minaoui [3, 2]

1. Cardiology, Centre Hospitalier Universitaire Mohammed VI, Agadir, MAR 2. Cardiology, Agadir University Center, Agadir, MAR 3. Cardiology, Souss-Massa University Hospital, Faculty of Medicine and Pharmacy of Agadir, Ibn Zohr University, Agadir, MAR

Corresponding author: Ayoub Laaboussi, laaboussi1998ayoub@gmail.com

Abstract

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is an inherited cardiomyopathy characterized by fibrofatty replacement of the right ventricular myocardium, predisposing to ventricular arrhythmias and sudden cardiac death. Sustained ventricular tachycardia (VT) may be the first manifestation of the disease, and left ventricular involvement is increasingly recognized. A 53-year-old man, followed for approximately 10 years for an incompletely characterized cardiomyopathy associated with palpitations, was admitted for sustained VT with a left bundle branch block morphology and an inferior axis, suggesting a right ventricular origin. The arrhythmia was successfully terminated by intravenous amiodarone. Transthoracic echocardiography revealed a dilated and hypertrophied right ventricle with an apical aneurysm and prominent free-wall trabeculations, severe right ventricular systolic dysfunction (tricuspid annular plane systolic excursion of 11 mm, systolic tricuspid annular velocity of 7 cm/s, fractional area change of 23%, and free-wall longitudinal strain of −10.5%), as well as concomitant moderate left ventricular systolic dysfunction (ejection fraction of 43%). Cardiac MRI demonstrated mid-apical right ventricular free-wall dyskinesia, an indexed right ventricular end-diastolic volume of 145.28 mL/m², a right ventricular ejection fraction of 23%, and late gadolinium enhancement of the right ventricular free wall. Coronary angiography showed normal coronary arteries. Prolonged ambulatory ECG monitoring revealed marked ventricular instability, with 169,148 ventricular premature beats over approximately three days, including three predominant morphologies, 31,972 couplets, and 13,863 ventricular runs, and 161 episodes of ventricular tachyarrhythmia, comprising approximately 20 episodes of non-sustained VT, with the remaining episodes corresponding to accelerated idioventricular rhythm. Bradycardia developed during treatment with amiodarone and a beta-blocker, requiring dose reduction. The diagnosis of definite ARVC was established based on the diagnostic criteria. A dual-chamber implantable cardioverter-defibrillator was subsequently implanted for secondary prevention. Amiodarone was discontinued after implantation because of persistent bradycardia. The subsequent clinical course was favorable, with no recurrent symptoms or documented arrhythmic events during the available follow-up. This case illustrates the revelation of ARVC by sustained VT, the central role of multimodality imaging in establishing the diagnosis, the importance of arrhythmic risk stratification for secondary-prevention defibrillator therapy, and the need to adapt antiarrhythmic treatment to arrhythmic burden and hemodynamic tolerance.

Categories: Therapeutics, Cardiology, Emergency Medicine
Keywords: arrhythmogenic right ventricular cardiomyopathy, arrhythmogenic right ventricular dysplasia, cardiac magnetic resonance, implantable cardioverter-defibrillator, ventricular electrical instability, ventricular extrasystoles, ventricular tachycardia

Introduction

Arrhythmogenic right ventricular cardiomyopathy (ARVC), also known as arrhythmogenic right ventricular dysplasia, is an inherited cardiomyopathy characterized by progressive myocardial replacement by fibrous and fatty tissue, predominantly affecting the right ventricle (RV), and predisposing patients to ventricular arrhythmias and sudden cardiac death (SCD), particularly in young adults [1]. The disease exhibits a broad phenotypic spectrum, including biventricular and left-dominant forms, which are now encompassed within the broader concept of arrhythmogenic cardiomyopathy (ACM) [2,3]. Long-term registry data indicate that ventricular arrhythmias and heart failure are major contributors to disease-related morbidity and mortality [4].

Diagnosis is based on a multiparametric assessment integrating ECG, arrhythmic, structural, and tissue characterization findings. The 2010 revised Task Force criteria established a standardized diagnostic framework [5], subsequently refined by the Padua criteria to incorporate myocardial tissue characterization and better characterize left ventricular involvement [2].

Sustained ventricular tachycardia (VT) may be the first clinical manifestation of ARVC and should prompt comprehensive evaluation for an underlying arrhythmogenic substrate. Implantable cardioverter-defibrillator (ICD) therapy plays a central role in preventing SCD in selected high-risk patients, particularly in secondary prevention [6-8].

We report the case of a 53-year-old man presenting with sustained VT, in whom multimodality imaging and ECG assessment established a diagnosis of definite ARVC, associated with severe RV dysfunction and a high ventricular arrhythmic burden. This case report was prepared in accordance with the CARE guidelines [9].

Case Presentation

Patient information and presentation

A 53-year-old man was admitted to the cardiology department following an episode of sustained VT. He had been followed for approximately 10 years for an incompletely characterized cardiomyopathy associated with palpitations.

The patient was married, had three children, and worked as a shopkeeper. He had no known history of diabetes mellitus or arterial hypertension and was a nonsmoker. He reported no known family history of cardiomyopathy or SCD.

Approximately 20 days before admission, he began experiencing intermittent episodes of palpitations with abrupt onset, each lasting approximately two minutes and associated with generalized asthenia. Three days before admission, his condition worsened, with three episodes of syncope without postictal neurological deficits, accompanied by dyspnea corresponding to New York Heart Association (NYHA) functional class III [10] and generalized jaundice. These symptoms prompted him to seek medical attention in the emergency department.

Clinical findings

On admission, the patient was conscious and alert, with a Glasgow Coma Scale (GCS) score of 15/15 [11]. Blood pressure was 94/45 mmHg, heart rate was 181 beats/minute, and oxygen saturation was 90% on room air. Physical examination revealed generalized jaundice and jugular venous distension, without peripheral edema or pulmonary crackles. Peripheral pulses were symmetric. Despite the low blood pressure, there were no documented overt clinical signs of shock.

Electrocardiographic findings

A previous ECG performed in 2016 showed T-wave inversion extending from leads V1 to V5, in the absence of complete right bundle branch block (Figure *1*).

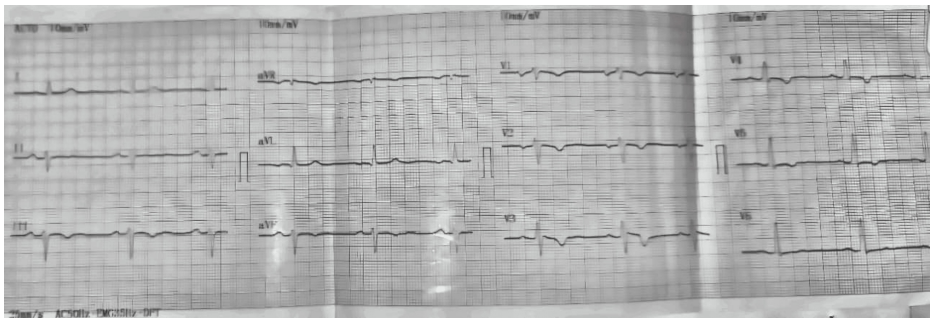


FIGURE 1: Twelve-lead ECG recorded in 2016 showing T-wave inversion in leads V1–V5, without complete right bundle branch block.

The admission ECG showed sustained VT with a left bundle branch block (LBBB) morphology and an inferior axis, suggesting a right ventricular origin (Figure *2*). Despite the absence of overt clinical signs of shock, intravenous amiodarone was administered for pharmacological cardioversion, successfully restoring sinus rhythm. Following conversion, treatment with amiodarone and a beta-blocker was initiated.

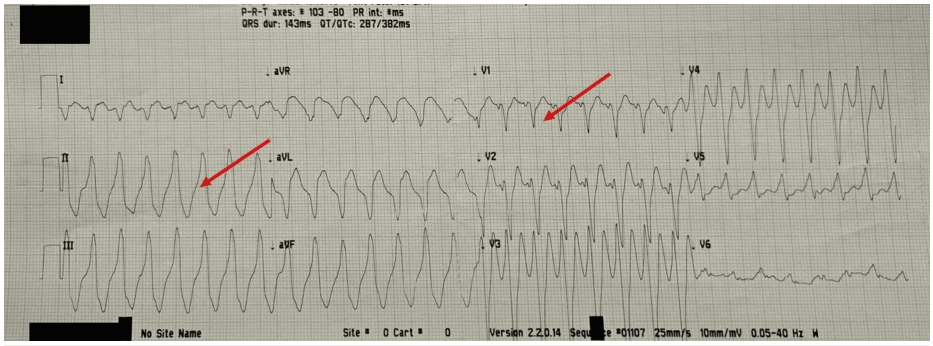


FIGURE 2: Admission 12-lead ECG showing sustained ventricular tachycardia with a left bundle branch block morphology and an inferior axis, suggesting a right ventricular origin. Arrows indicate the wide, predominantly negative QRS complexes in lead V1 and the wide, monomorphic ventricular complexes in lead II, consistent with a left bundle branch block morphology.

Laboratory findings

The initial laboratory investigations, including the complete blood count, renal and liver panels, electrolytes, cardiac troponin, and thyroid-stimulating hormone, are summarized in Table *1*. The liver panel abnormalities were consistent with hepatic congestion and a low-output state during the arrhythmic episode. Viral serologies were negative and abdominal ultrasonography was unremarkable; biological surveillance was recommended by the gastroenterology team. The absence of biological evidence of myocardial necrosis, together with the subsequent demonstration of normal coronary arteries, argued against an ischemic substrate for the VT.

Parameter	Result	Reference range
Hemoglobin (g/dL)	16.4	13.0–17.0
Leukocytes (/mm^3)	9,680	4,000–10,000
Platelets (/mm^3)	243,000	150,000–450,000
C-reactive protein (mg/L)	2.8	<5
Serum creatinine (mg/L)	11.2	7–13
Blood urea (g/L)	0.51	0.15–0.45
Sodium (mmol/L)	139	135–145
Potassium (mmol/L)	4.0	3.5–5.0
Cardiac troponin *	0.02	<0.03
Thyroid-stimulating hormone (mIU/L)	1.29	0.4–4.0
Aspartate aminotransferase (U/L)	176	<30
Alanine aminotransferase (U/L)	266	<30
Gamma-glutamyl transferase (U/L)	139	<55
Total bilirubin (µmol/L)	44	<17

TABLE 1: Initial laboratory investigations on admission.

*: Cardiac troponin is expressed relative to the local laboratory decision threshold; the measured value was below the upper reference limit and was therefore considered negative. Reference ranges are those of the local laboratory.

Transthoracic echocardiography

Transthoracic echocardiography showed a non-dilated left ventricle, with an end-diastolic diameter of 40 mm (22 mm/m²), an interventricular septal thickness of 8 mm, and a posterior wall thickness of 9 mm. The left ventricle exhibited septal dyskinesia and global hypokinesia, resulting in a moderately reduced LV ejection fraction of 43% by the biplane Simpson method.

The RV was markedly abnormal, dilated, and hypertrophied, with a basal diameter (RVD1) of 51 mm. An apical aneurysm and marked free-wall trabeculations were observed. Right ventricular systolic function was impaired, with a tricuspid annular plane systolic excursion of 11 mm, an S′ velocity of 7 cm/s, a fractional area change of 23%, and an RV free-wall longitudinal strain of −10.5%. The anteroposterior RV diameter measured 50 mm in the parasternal long-axis view (27 mm/m²) and 47 mm in the parasternal short-axis view (26 mm/m²).

The right atrium was dilated (21 cm²). Doppler assessment showed a mitral inflow pattern consistent with grade I diastolic dysfunction and a high probability of pulmonary hypertension based on the tricuspid regurgitation data. Cardiac output was 4.01 L/min, with a cardiac index of 2.3 L/min/m².

Overall, echocardiography demonstrated severe right ventricular structural and functional abnormalities strongly suggestive of ARVC, together with concomitant moderate LV systolic dysfunction.

Coronary angiography

Coronary angiography showed normal coronary arteries, excluding significant coronary artery disease as an explanation for the ventricular arrhythmia and LV dysfunction (Figure *3*).

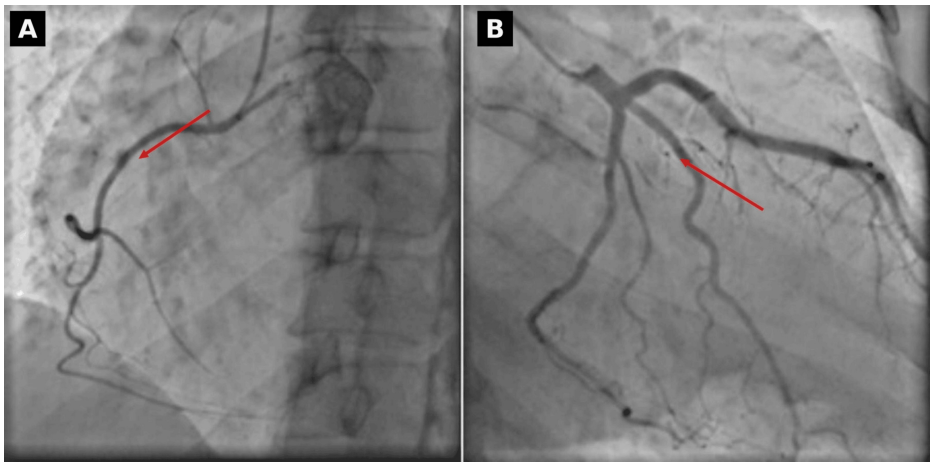


FIGURE 3: Coronary angiography showing normal coronary arteries. (A) Right coronary angiogram; the arrow indicates the smooth, aneurysm- and stenosis-free right coronary artery. (B) Left coronary angiogram; the arrow indicates the left anterior descending artery, free of significant epicardial stenosis.

Cardiac magnetic resonance imaging

Cardiac magnetic resonance (CMR) provided further evidence of an arrhythmogenic right ventricular substrate. It demonstrated regional dyskinesia of the mid-apical RV free wall, an indexed RV end-diastolic volume of 145.28 mL/m², and a severely reduced RV ejection fraction of 23% (Figure *4*). According to the imaging report, the combination of regional RV dyskinesia, marked dilatation, and impaired systolic function constituted a major criterion for the diagnosis of ARVC. Late gadolinium enhancement (LGE) of the RV free wall was also observed. However, this finding was not independently counted as a diagnostic criterion because the specific imaging pattern required by the Padua criteria was not documented in the available CMR report.

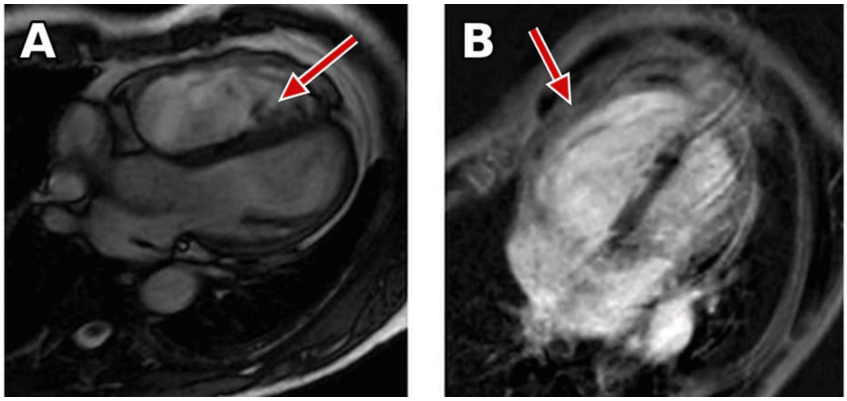


FIGURE 4: Cardiac magnetic resonance imaging. (A) Oblique axial view showing right ventricular dilatation. (B) Four-chamber long-axis view showing diffuse late gadolinium enhancement of the right ventricular free wall, with mild heterogeneous late gadolinium enhancement involving the right ventricular aspect of the interventricular septum.

Ventricular electrical instability

Following restoration of sinus rhythm, the patient continued to exhibit marked ventricular electrical instability. Prolonged ambulatory ECG monitoring was performed over three days. In total, 271,108 QRS complexes were analyzed, with an overall recording quality of 83%. Recorded heart rate ranged from 39 to 116 beats/min, with a mean of 65 beats/min.

The ventricular arrhythmic burden was substantial: 169,148 ventricular extrasystoles (maximal hourly frequency 3,593/h), 31,972 couplets, 13,863 ventricular runs, 687 episodes of bigeminy, 87 episodes of trigeminy. The recording also revealed 161 episodes of ventricular arrhythmias, including approximately 20 episodes of non-sustained VT and the remaining episodes of accelerated idioventricular rhythm (AIVR), with a maximum rate of 116 beats/min (Figure *5*). The ventricular extrasystoles were very frequent and polymorphic, occurring as trigeminy, couplets, and triplets, with three predominant morphologies. Supraventricular extrasystoles were rare, and the patient reported palpitations during the recording.

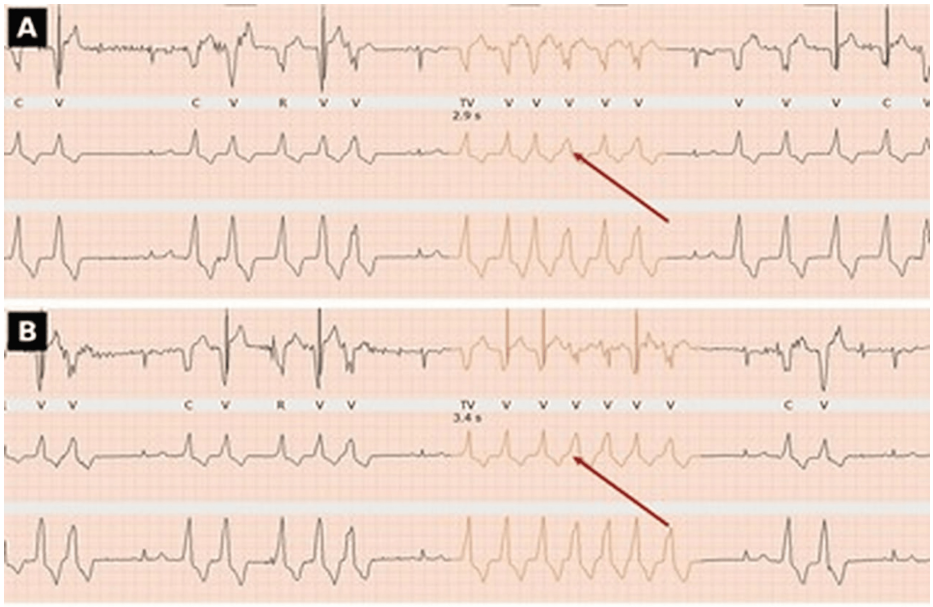


FIGURE 5: Prolonged ambulatory ECG monitoring demonstrating marked ventricular electrical instability. (A) Episode of non-sustained ventricular tachycardia; the arrow indicates the run, which lasted 2.9 seconds. (B) Episode of non-sustained ventricular tachycardia; the arrow indicates the longest documented run, lasting 3.4 seconds.

Therapeutic management and follow-up

The sustained VT was initially treated with amiodarone, with a loading dose followed by maintenance therapy, achieving conversion to sinus rhythm. The patient was then treated with bisoprolol 10 mg/day and oral amiodarone 600 mg/day. During hospitalization, he developed an accelerated idioventricular rhythm and significant sinus bradycardia, with a minimal heart rate of 38 beats/min (Figure 6), requiring down-titration of the beta-blocker and amiodarone. Bisoprolol was reduced to 2.5 mg/day and amiodarone to 200 mg every other day.

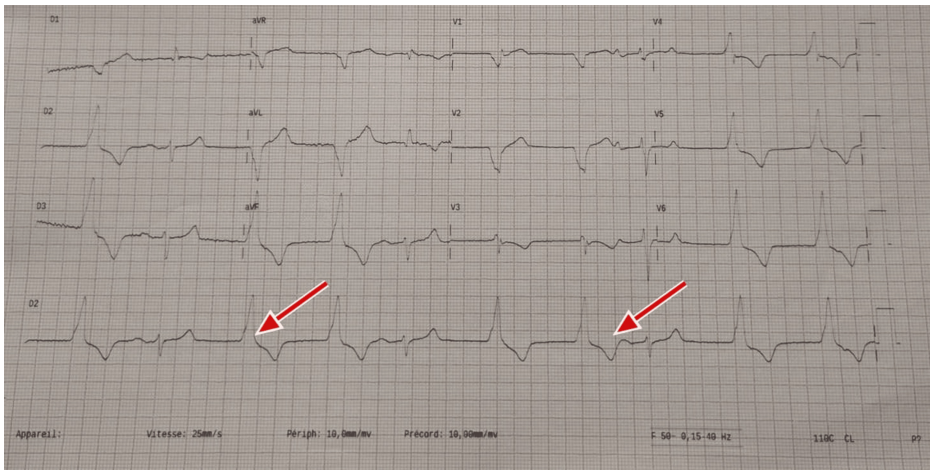


FIGURE 6: ECG recorded during hospitalization, under antiarrhythmic and beta-blocker therapy, showing the transition to bradycardia and an accelerated idioventricular rhythm. Arrows indicate the slow, wide QRS complexes of the accelerated idioventricular rhythm on the rhythm strip.

No clinical signs of congestion were observed during hospitalization, and the hemodynamic status

subsequently remained stable, with a systolic blood pressure of approximately 110 mmHg. In view of the moderately reduced LV ejection fraction, heart failure therapy was initiated and well tolerated. At discharge, treatment included low-dose bisoprolol, amiodarone, furosemide, dapagliflozin, spironolactone, and ramipril.

Given the documented sustained VT in a patient with definite ARVC, the multidisciplinary team decided to implant a dual-chamber ICD for secondary prevention of SCD. The device was successfully implanted after obtaining informed consent (Figure 7). Following implantation, amiodarone was discontinued because of persistent bradycardia, while beta-blocker therapy was maintained at the reduced dose. The subsequent clinical course was favorable, with the patient remaining asymptomatic and clinically stable, without recurrent palpitations, syncope, or dyspnea. Device interrogation showed no abnormalities or recorded arrhythmic events. Regular cardiological follow-up, including periodic device interrogation, was scheduled.



FIGURE 7: Postoperative chest radiograph after implantation of the dual-chamber implantable cardioverter-defibrillator, showing satisfactory generator and lead positioning.

Discussion

This case illustrates the diagnosis of ARVC in a patient presenting with sustained VT after approximately 10 years of follow-up for an incompletely characterized cardiomyopathy. It highlights four clinically relevant aspects: the diagnostic value of ECG and multimodality imaging findings, the assessment of ventricular electrical instability and ICD implantation, and the challenges of antiarrhythmic treatment in the presence of bradycardia.

Diagnostic approach and multimodality imaging

Sustained monomorphic VT with an LBBB morphology in an adult without coronary artery disease should raise suspicion of an RV substrate, particularly ARVC [1]. In our patient, the diagnostic workup followed the recommended stepwise approach: exclusion of an ischemic cause by biomarker assessment and coronary angiography, followed by morphofunctional characterization with echocardiography and CMR.

The diagnosis of ARVC relies on the combination of findings from different diagnostic categories, as established by the 2010 revised Task Force criteria [5]. In our patient, regional RV dyskinesia associated with marked RV dilatation and severe systolic dysfunction fulfilled a major morphofunctional criterion. Furthermore, the ECG recorded in 2016 demonstrated T-wave inversion extending from V1 to V5 in the absence of complete right bundle branch block, fulfilling a major repolarization criterion. These two major criteria from different diagnostic categories established the diagnosis of definite ARVC. The sustained VT with LBBB morphology and inferior axis constituted an additional minor arrhythmic criterion [5].

The more recent Padua criteria extend this framework by incorporating CMR tissue characterization; the late gadolinium enhancement of the RV free wall observed in our patient is consistent with myocardial fibrosis, a feature of the disease phenotype [2]. The moderately reduced LV ejection fraction with septal dyskinesia also suggests possible biventricular involvement, in keeping with the contemporary concept of ACM as a disease spectrum extending beyond the RV [1,3]. In the multicenter series of Groeneweg et al. [4], encompassing 1,001 patients and family members, LV involvement was frequent, and arrhythmic events clustered in those with overt structural disease.

Ventricular electrical instability

Ambulatory monitoring revealed an exceptionally high ventricular arrhythmic burden, with more than 169,000 ventricular premature beats over approximately three days, three predominant morphologies, and 31,972 couplets. Among the 161 recorded episodes of ventricular tachyarrhythmia, approximately 20 were identified as non-sustained VT, while the remaining episodes corresponded to AIVR. A ventricular premature beat burden exceeding 500 per 24 hours constitutes a minor arrhythmic criterion under the 2010 Task Force criteria [5]. The marked ventricular ectopy observed in this patient reflects substantial electrical instability and illustrates the complexity of pharmacological management, particularly given the development of significant bradycardia during combined amiodarone and beta-blocker therapy.

Risk stratification and implantable cardioverter-defibrillator therapy

Risk stratification in ARVC requires an individualized assessment incorporating clinical presentation, ventricular function, and arrhythmic history [3,8]. Quantitative tools have also been developed: the validated ARVC risk calculator estimates the five-year risk of sustained ventricular arrhythmia in previously event-free patients from readily available clinical variables, including age, sex, syncope, non-sustained VT, extrasystole burden, T-wave inversions, and RV ejection fraction [12].

In this case, the history of sustained VT, three syncopal episodes, severe RV systolic dysfunction, and marked ventricular ectopy supported the decision to implant an ICD for secondary prevention of SCD. Current consensus recommendations emphasize ICD implantation following cardiac arrest or sustained VT that is not hemodynamically tolerated, while ICD implantation may also be considered in selected patients with hemodynamically tolerated sustained VT after individualized risk assessment and shared decision-making [6,7]. In our patient, the absence of documented overt clinical signs of shock should be distinguished from the presence of low blood pressure at presentation.

A dual-chamber device was selected in the context of significant bradycardia observed during antiarrhythmic and beta-blocker treatment, providing an additional pacing capability should clinically significant bradycardia persist.

Pharmacological management

Antiarrhythmic drugs may reduce ventricular arrhythmia burden and symptoms in ARVC but do not replace the protective role of an ICD when indicated [3,6]. Beta-blockers are commonly used as part of pharmacological management, while amiodarone may be considered in selected patients with recurrent or symptomatic ventricular arrhythmias. In our patient, intravenous amiodarone successfully terminated the presenting VT. However, subsequent treatment with oral amiodarone and high-dose bisoprolol was complicated by significant sinus bradycardia and AIVR, requiring dose reduction. Because bradycardia persisted, amiodarone was discontinued after ICD implantation, while beta-blocker therapy was maintained at a reduced dose.

Heart failure therapy was also introduced in view of the documented LV systolic dysfunction [8]. Catheter ablation remains a therapeutic option in patients with recurrent sustained VT or repeated appropriate ICD therapies despite medical treatment [6].

Family screening and prevention of sudden cardiac death

Given the inherited nature of ARVC, the absence of a reported family history does not exclude familial disease. First-degree relatives should undergo clinical screening, including ECG, cardiac imaging, and ambulatory ECG monitoring. Genetic counselling and cascade genetic testing should also be considered when a pathogenic variant is identified [3,8]. Avoidance of high-intensity endurance exercise and competitive sports is recommended, as strenuous physical activity may accelerate disease progression and

increase ventricular arrhythmic risk. Regular clinical and device follow-up, together with appropriate family screening, remains essential for reducing the risk of adverse outcomes.

Conclusions

Sustained ventricular tachycardia with LBBB morphology may reveal long-standing, previously uncharacterized ARVC. This case emphasizes the value of a multimodal diagnostic evaluation combining ECG, echocardiography, coronary angiography, CMR, and prolonged ambulatory monitoring to establish the diagnosis, characterize the extent of RV disease and possible biventricular involvement, and quantify the arrhythmic burden. It further illustrates the importance of individualized risk stratification leading to secondary-prevention ICD implantation, as well as the ongoing adaptation of antiarrhythmic therapy according to the arrhythmic burden, clinical course, and treatment tolerance, particularly when treatment-induced bradycardia occurs.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Ayoub Laaboussi, Mohamed El Minaoui, Wassim Beladel, Amine El Houari, Khalil Abderrahmane Elbaz, Nada Douari

Acquisition, analysis, or interpretation of data: Ayoub Laaboussi, Wassim Beladel, Amine El Houari, Nada Douari

Drafting of the manuscript: Ayoub Laaboussi, Nada Douari

Critical review of the manuscript for important intellectual content: Mohamed El Minaoui, Wassim Beladel, Amine El Houari, Khalil Abderrahmane Elbaz

Supervision: Mohamed El Minaoui, Wassim Beladel

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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