Case Report

Spectrum of supraventricular and ventricular arrhythmia: A case series highlighting AVNRT and arrhythmogenic right ventricular dysplasia

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Abstract

Background: Supraventricular tachycardia (SVT) and ventricular arrhythmia's are common causes of palpitations and syncope, with variable clinical presentations and outcomes. Radiofrequency (RF) ablation and cardioversion remain mainstays of definitive therapy in selected cases.

Case Series: We report three cases highlighting the clinical spectrum of arrhythmias. Case 1 involved a 44-year-old female with AVNRT and comorbid hypertension and hypothyroidism, successfully treated with RF ablation using 3-D electro anatomical mapping (EAM). Case 2 described a 38-year-old male with drug-refractory AVNRT and mild mitral valve prolapse, also successfully managed with RF ablation. Case 3 involved a 15-year-old boy with arrhythmogenic right ventricular dysplasia (ARVD) presenting with ventricular tachycardia; arrhythmia was refractory to antiarrhythmic therapy but reverted to normal rhythm after synchronized DC cardioversion.

Conclusion: This series illustrates the diverse presentation of supraventricular and ventricular arrhythmias, the role of advanced electrophysiologic interventions, and the importance of individualized management strategies to achieve optimal outcomes.

Key words: Supraventricular tachycardia (SVT); Radiofrequency (RF); Arrhythmogenic right ventricular dysplasia (ARVD)

Citation: Shirlin M.S, Vignesh R. Spectrum of supraventricular and ventricular arrhythmia: A case series highlighting AVNRT and arrhythmogenic right ventricular dysplasia. Kauverian Med J. 2026;3(6):93-99.

Academic Editor: Dr. Venkita S. Suresh

ISSN: 2584-1572 (Online)

1. Introduction

Supraventricular tachycardia (SVT) and ventricular arrhythmias are significant contributors to cardiovascular morbidity across all age groups. AV nodal re-entrant tachycardia (AVNRT) is the most common form of SVT in adults, often presenting with palpitations, dizziness, or syncope. Although generally benign in structurally normal hearts, arrhythmia can cause hemodynamic instability and life-threatening complications, particularly in pediatric patients or those with underlying cardiomyopathies.

Arrhythmogenic right ventricular dysplasia (ARVD) is an inherited cardiomyopathy characterized by ventricular arrhythmias and structural abnormalities of the right ventricle (RV). ARVD results from progressive replacement of right ventricular myocardium with fatty and fibrous tissue. ARVD is the second most common cause of sudden cardiac death in young people (after HOCM), accounting for up to 10% of sudden cardiac deaths in patients < 65 yrs of age. Prevalence ~ 1 in 5000.

2. Clinical Features

- ARVD causes symptoms due to ventricular ectopic beats or sustained VT (with LBBB morphology), and typically presents with palpitations, syncope or cardiac arrest precipitated by exercise.
- The first presenting symptom may be sudden cardiac death.
- Over time, surviving patients develop features of right ventricular failure, which may progress to severe biventricular failure and dilated cardiomyopathy.
- There is usually a family history of sudden cardiac death.

The electrocardiogram (ECG) expression of this depolarization delay was first described in 1978, when Fontaine5 named these tiny signals found during endocardial mapping (and also in the surface ECG) located right after the QRS complexes in leads V1-V3, as ‘epsilon waves.

The goal of ARVC management is to minimize the risk of sudden cardiac death, slow the progression of the disease, and enhance the quality of life by reducing the burden of arrhythmia and symptoms of heart failure. The management of ARVC involves a range of approaches, such as clinical management, pharmacological treatment, catheter ablation, implantation of a cardiac defibrillator, and, if required, cardiac transplant.

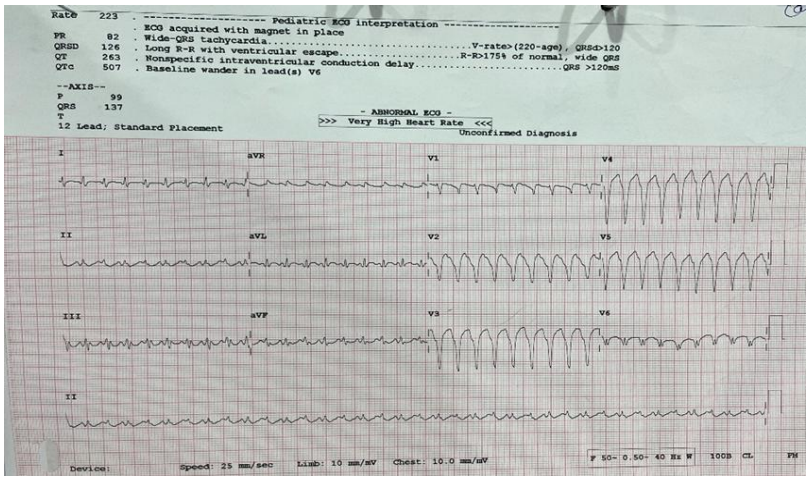
Advances in electrophysiology, including three-dimensional electro anatomical mapping (3-D EAM) and radiofrequency (RF) ablation, have revolutionized the management of SVT, offering curative treatment in drug-refractory cases.

It requires rapid recognition and intervention to prevent sudden cardiac death. This case series highlights the spectrum of arrhythmia presentations, management approaches, and outcomes in diverse patient populations.

3. Case 1

A 44-year-old female with a history of hypertension and well-controlled hypothyroidism presented with intermittent palpitations for the past two years. Baseline laboratory investigations revealed normal renal function, with blood urea of 29 mg/dL and serum creatinine of 0.7 mg/dL, along with normal liver function tests.

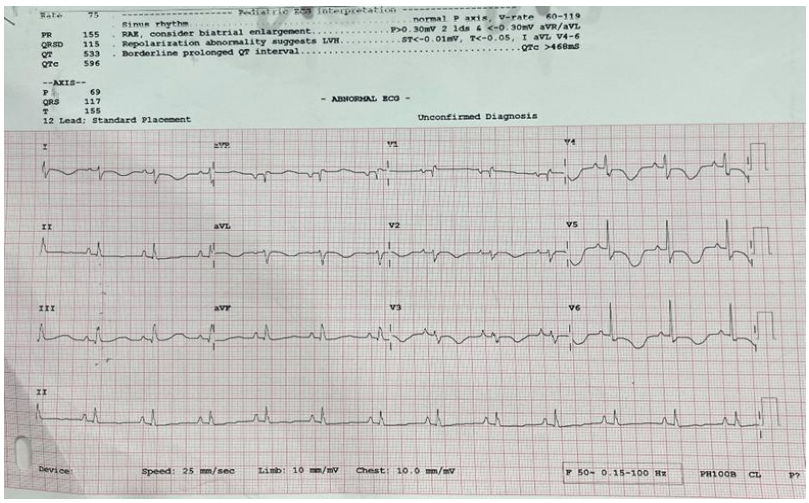
Electrocardiography revealed ventricular tachycardia with no ST changes, absent P wave, upright T wave and normal QRS complex.



She was evaluated and diagnosed with supraventricular tachycardia, specifically atrioventricular nodal re-entrant tachycardia.

She underwent an electrophysiology (EP) study, during which inducible AVNRT was identified. Subsequently, radiofrequency (RF) ablation was performed using three-dimensional electro anatomical mapping (3-D EAM), achieving successful ablation of the arrhythmogenic substrate.

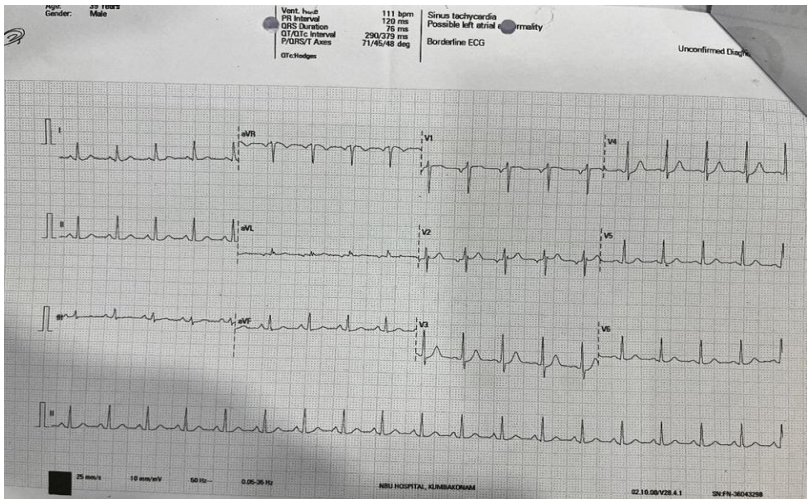
3.1. Post ablation ECG



The patient remained hemodynamically stable throughout the procedure. Post-procedure, she was discharged on her regular antihypertensive and thyroid medications, along with aspirin for one month. Symptom relief is anticipated, and she was advised regular follow-up for arrhythmia monitoring.

4. Case 2:

A 38-year-old male with no known comorbidities presented with complaints of intermittent palpitations for the past four years. Echocardiography revealed LA dilation, mild MR, mild TR, mild AR, mild PAH and mild MV prolapse. Electrocardiography revealed inverted P wave, ST depression in L2, ST elevation in AVR. Severe ST depression in V2-V4.



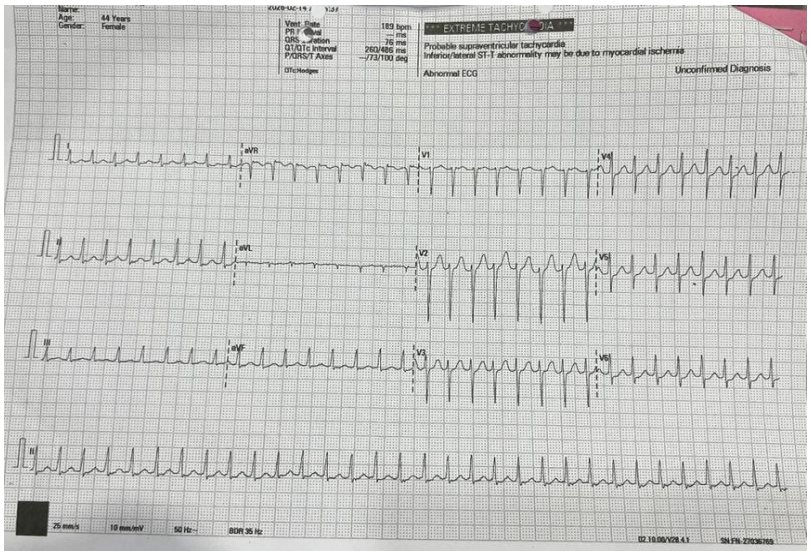
He was diagnosed with supraventricular tachycardia (SVT), specifically atrioventricular nodal re-entrant tachycardia (AVNRT), and was refractory to medical therapy. Echocardiography revealed normal left ventricular function, anterior mitral leaflet (AML) prolapses, and mild mitral regurgitation (MR). The patient underwent an electrophysiology (EP) study with radiofrequency (RF) ablation using three-dimensional electro anatomical mapping (3-D EAM), which demonstrated inducible AVNRT. Slow pathway modification was successfully performed. The patient remained stable post-procedure and was discharged on aspirin and pantoprazole with advice for regular follow-up.

5. Case 3

A 15-year-old boy presented with sudden onset palpitations, giddiness, and syncope while performing routine activities. He spontaneously regained consciousness and was initially managed at a local hospital, where his heart rate was approximately 240 beats per minute and later reduced with medical therapy. However, he subsequently developed recurrent symptoms, including abdominal pain, giddiness, and an episode of cardiac arrest, from which he was successfully resuscitated. Despite initial stabilization and discharge, he experienced recurrence of palpitations the same day and was referred for further evaluation.

On admission, his heart rate was 217 beats per minute, and blood pressure was 100/70 mmHg. Baseline laboratory investigations revealed leukopenia (WBC: 1,500 cells/mm^3), anemia (hemoglobin: 11.5 g/dL; RBC count: 3.95 million/mm^3), and thrombocytopenia (platelet count: 50,000/mm^3). Serum electrolytes showed hyponatremia (sodium: 123 mEq/L), mild hyperkalemia (potassium: 5.1 mEq/L), and hypochloremia (chloride: 94 mEq/L). Bicarbonate level was reduced (15 mEq/L), suggestive of metabolic acidosis. Serum calcium was 6.5 mg/dL.

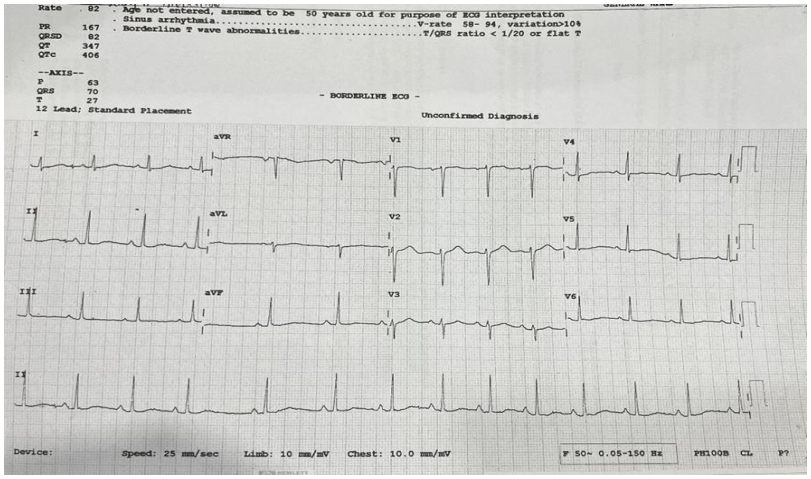
ECG revealed tachycardia, long QT intervals and a wide QRS complex.



He was treated with intravenous Amiodarone (150 mg loading dose), followed by continuous infusion (60 mg/hour for 6 hours, then 30 mg/hour for 18 hours), but there was no response.

The patient was diagnosed with arrhythmogenic right ventricular dysplasia (ARVD), presenting with ventricular tachycardia. Due to persistent arrhythmia, synchronized DC cardioversion (150 Joules) was performed, successfully restoring normal sinus rhythm.

5.1. Post ablation ECG



Cardiac MRI was planned for further structural and functional evaluation.

6. Discussion

The cases demonstrate the heterogeneity of arrhythmia presentation and management:

Case 1 involved a middle-aged female with comorbid hypertension and hypothyroidism who presented with intermittent palpitations due to AVNRT. She was successfully treated with RF ablation using 3-D EAM, underscoring the procedure's safety and efficacy even in patients with stable comorbidities.

Case 2 highlighted a younger adult male with drug-refractory AVNRT and mild mitral valve prolapse. This case emphasizes the importance of structural heart evaluation, even when left ventricular function is preserved. Successful slow pathway modification via RF ablation demonstrates its role as definitive therapy in pharmacologically resistant cases.

Case 3 represented a paediatric patient with ARVD presenting with ventricular tachycardia, syncope, and metabolic derangements. The arrhythmia was unresponsive to intravenous antiarrhythmic therapy but reverted successfully after synchronized DC cardioversion. This case highlights the critical need for rapid intervention in life-threatening arrhythmia, the role of imaging (cardiac MRI) in diagnosing structural cardiomyopathies, and the complexity of paediatric arrhythmia management.

Collectively, these cases illustrate the spectrum from typical AVNRT to malignant ventricular arrhythmias. They highlight the importance of individualized management strategies, prompt intervention, and long-term follow-up to prevent recurrence and complications.

7. Conclusion

This case series demonstrates the diverse spectrum of supraventricular and ventricular arrhythmias. AVNRT in adults is effectively managed with RF ablation, while pediatric patients with structural heart disease, such as ARVD, may present with life-threatening ventricular arrhythmias requiring urgent cardioversion. Arrhythmogenic right ventricular dysplasia/cardiomyopathy is a genetic cardiomyopathy that leads to right ventricular failure, arrhythmias, and sudden death. Early recognition, tailored intervention, and comprehensive follow-up are essential to optimize outcomes and reduce morbidity and mortality associated with arrhythmias.

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