Advances in Bioresearch
Adv. Biores., Vol 16 (5) September 2025: 190-192

Print ISSN 0976-4585; Online ISSN 2277-1573
Journal's URL:http://www.soeagra.com/abr.html
CODEN: ABRDC3
DOI: 10.15515/abr.0976-4585.16.5.190192

Advances in Bioresearch

CASE STUDY

Complexities of PSVT in a Known Case of HOCM: Clinical Insights from a Case Report

Zainab Nalawala, Jemini Patel, Nishank Bhagat and Priyal Patel
Department of Pharmacy Practice, Parul Institute of Pharmacy & Research, Parul University, Vadodara, Gujarat
Corresponding Author: Email: zainabnala31@gmail.com

ABSTRACT
Paroxysmal supraventricular tachycardia is characterized by sudden onset of tachycardia, which usually arises above the ventricles and its rate ranges from 150 to 250 beats per minute. The case report below involves a 56-year-old female patient diagnosed with hypertrophic obstructive cardiomyopathy (HOCM) 16 to 17 years ago who was complaining of chest pain, dizziness, and palpitations. Investigations found an increased cardiac biomarker level, anemia, and echocardiographic findings confirming the progression of HOCM. The patient was treated with a multi-faceted approach using antiarrhythmic drugs, anticoagulants, and supportive care for which the improvement was remarkable with safe discharge. This is an important relationship between HOCM and PSVT which requires well-tailored care regimens to be provided for similar patients.
Keywords; *Paroxysmal Supraventricular Tachycardia, Hypertrophic Obstructive Cardiomyopathy, Atrioventricular, Creatine Kinase-Muscle/Brain, Packed Cell Volume, Left Ventricular Hypertrophy, Systolic Anterior Motion, Left Ventricular Outflow Tract, C-Reactive Protein*

Received 12.05.2025 Revised 16.07.2025 Accepted 18.09.2025

How to cite this article:
Twinkal Soni, Mrunal Bhoir, Jinal Patel"A Cross-Sectional Study of Jihwa Pariksha (Tongue Examination) In Artavadusti Vikara with Special Reference to Menstrual Disorders". Adv. Biores., Vol 16 (5) September 2025:190-192

INTRODUCTION

Paroxysmal Supraventricular Tachycardia is an arrhythmia characterized by abrupt, episodic tachycardia above the ventricles-which is often caused by reentrant processes involving the AV node or accessory pathways. [1].This results in rates of 150 to 250 beats per minute. PSVT occurs as a result of both genetic factors and anatomical changes of the structure of the heart together with reentrant circuits, which create quick, repetitive impulses. Prevalence: The whole number incidence frequency is at 2-3 cases per 1000 populations, and PSVT increases with advancing age, affecting 1-3% of adults. [2]. Symptoms might vary from moderate palpitations to severe dizziness or syncope [3]. Paroxysmal Supraventricular Tachycardia (PSVT) often results from participating premature atrial contractions that form part of reentrant pathways, especially in anatomically diseased hearts, thereby leading to intractable hemodynamic compromise. This is decidedly problematic for individuals with preexistent cardiac disease such as HOCM [4]. Hypertrophic and fibrotic cardiac tissue in HOCM forms a pro-arrhythmic substrate, increasing the incidence of PSVT episodes by raising atrial pressures and worsening arrhythmia susceptibility [5]. In this patient, HOCM-associated hypertrophy and fibrosis have produced a complex clinical situation in which PSVT pathophysiology is compounded by inadequate diastolic filling and high left atrial pressure. To prevent thromboembolic risk and successfully manage rate, a distinct rhythm control treatment approach that may include specialized anticoagulation and antiarrhythmic medications is required for this dual disease [6].

CASE REPORT

A 56-year-old female presented with a four-day history of dull, non -radiating chest pain, giddiness and palpitations. Her medical history includes a 16-17-year diagnosis of hypertrophic obstructive

cardiomyopathy (HOCM), for which she was prescribed Aspirin, Metoprolol, Atorvastatin, and Sorbitrate. Upon evaluation, she was conscious and had a high pulse rate of 117 bpm.

Laboratory findings revealed substantial abnormalities, including an increased white blood cell count of 15,400 cells/mm^3 (normal: 4,500-11,000 cells/mm^3), low haemoglobin of 10.4 g/dL (normal: 12-16 g/dL), and a decreased red blood cell count of 3.4 million/mm^3 (normal: 4.2-5.9 million/mm^3). Furthermore, the packed cell volume (PCV) was 32.8% (normal: 36–48%). Cardiac biomarkers showed significant elevations, including CK-MB at 76.80 IU/L (normal: <25 IU/L) and a positive troponin I. Total bilirubin was 0.90 mg/dL (normal: 0.1-1.2 mg/dL), albumin was 3.70 g/dL (normal: 3.5-5.0 g/dL), and urea was high at 49 mg/dL (normal: 7-20 mg/dL). The electrolytes were normal, with sodium at 139 mmol/L (normal: 135-145 mmol/L) and potassium at 4.7 mmol/L (normal: 3.5-5.0). An echocardiography in 2022 found mild concentric hypertrophy, which suggests hypertrophic cardiomyopathy (HCM) or infiltrative obstructive cardiomyopathy. In addition, imaging scans revealed free fluid in the abdomen. These observations led to the diagnosis of hypertrophic obstructive cardiomyopathy (HOCM)-associated paroxysmal supraventricular tachycardia.

Table 1; Current treatment chart of patient during hospitalization

Medication Name	Dose	Route	Frequency
Aspirin	150 mg	Oral	Once daily after lunch
Enoxaparin	0.6 cc	Subcutaneous	Twice daily
Warfarin	2.5mg	Oral	Once daily after breakfast
Atorvastatin	40 mg	Oral	Once at night
Amiodarone	200 mg	Oral	Twice daily
Metoprolol	25 mg (½ tab)	Oral	Twice daily (after meals)
Pantoprazole	40 mg	IV (inj.)	Twice daily
Ondansetron	4 mg	IV (inj.)	Thrice daily
Dobutamine	1 mcg	IV	Every 12 hours
Folic acid	5 mg	Oral	Once daily after breakfast
Laxose Syrup	30cc	Oral	Thrice daily

Following the beginning of treatment, the patient's heart rate improved significantly, reaching 96 bpm on the first day, 88 bpm on the second day, and 80 bpm on the third. Throughout the hospital stay, her blood pressure remained constant and within acceptable norms. The patient was instructed to remain in bed and have her blood pressure and heart rate properly checked on a regular basis. Furthermore, adherence to a sodium-restricted diet was encouraged. This comprehensive monitoring and management method considerably improved her overall stability and recovery. At discharge, the patient was given Aspirin 75 mg daily, Atorvastatin 20 mg at night, Pantoprazole 40 mg once day, Folic acid 5 mg every morning, Metoprolol 25 mg (half a pill in the morning and evening), and Amiodarone 100 mg BD.

DISCUSSION

Paroxysmal supraventricular tachycardia (PSVT) is an arrhythmia characterized by abrupt, fast heart rate events that originate above the ventricles and normally range between 150 and 250 beats per minute. Episodes may repeat intermittently and frequently end spontaneously, allowing for extensive periods of symptom-free time. In this case, a 56-year-old female arrived with symptoms and a clinical history that suggested progression from her long-standing hypertrophic obstructive cardiomyopathy (HOCM), which was now worsened by PSVT. Serial echocardiographic evaluations show gradual structural alterations associated with HOCM. In February 2015, a preliminary assessment revealed clinical hypertension, mild concentric left ventricular hypertrophy (LVH), diastolic dysfunction, and sclerotic aortic valve alterations. In April 2018, further imaging revealed significant concentric LVH and persistent diastolic dysfunction in the absence of systolic anterior motion (SAM) or left ventricular outflow tract (LVOT) blockage. The most recent examination, performed in June 2022, revealed modest concentric hypertrophy, a potentially developing case of HCM or infiltrative obstructive cardiomyopathy, as well as significant left atrial

dilatation, indicating increased atrial pressure and progressive diastolic dysfunction. HOCM considerably increases arrhythmia risk due to cardiac hypertrophy and fibrotic alterations that impair electrical stability, as indicated by her complaints of palpitations, chest tightness, and giddiness—symptoms common to both HOCM and arrhythmias such as PSVT (7). Elevated CK-MB, CRP, and positive troponin I levels in the blood indicate myocardial stress or damage, which is consistent with an increased arrhythmogenic risk. The impact of HOCM on diastolic function, along with left atrial dilatation, predisposes her to PSVT, since atrial strain caused by persistent left ventricular hypertrophy enhances re-entrant circuits (8). This case demonstrates the interaction between structural disease and electrical disruption in HOCM, underlining the importance of thorough cardiac monitoring and rhythm control to avoid recurring PSVT episodes and associated consequences. The patient's treatment plan incorporates treatments for both PSVT and HOCM, adhering to recognized procedures for symptom alleviation, rhythm stability, and thromboembolic prophylaxis. Antiarrhythmic medicines such as Amiodarone and Metoprolol control heart rate, but Dobutamine improves cardiac function, which is critical given the patient's admission heart rate of 117 bpm and palpitations. The patient was given Aspirin and Enoxaparin to reduce thrombotic risks associated with an increased white blood cell count (15,400 cells/mm^3) and inflammatory markers (CRP 6.10 mg/L). Intravenous fluids were administered to treat her low haemoglobin (10.4 g/dL) and RBC count (3.4 million/mm^3), which contributed to her giddiness. In addition, Atorvastatin was recommended for cholesterol control, Pantoprazole to prevent gastrointestinal problems from her heavy pharmaceutical regimen, and Folic acid to boost haemoglobin levels. After stabilization, the patient was discharged on a revised treatment regimen including Aspirin to prevent blood clotting, Atorvastatin to lower cholesterol level, Metoprolol to control heart rate, and Amiodarone to keep heart rhythm regular. Folic acid was administered to increase haemoglobin levels, while Pantoprazole was provided to protect the stomach against medication side effects, providing an all-around post-discharge care program.

CONCLUSION

In conclusion, this case exemplifies the complex relationship between HOCM and PSVT in a 56-year-old female patient. Clinical history, combined with echocardiographic findings, demonstrates the heightened risk of arrhythmias and related symptoms that characterize her heart disease. A very comprehensive treatment strategy was designed to restore her cardiac rhythm, alleviate symptoms, and prevent further thromboembolic occurrences. This case demonstrates the need for tailored therapy in the treatment of complex cardiovascular disease patients. Only through close monitoring and further review will this patient's long-term prognosis be maximized.

REFERENCES

1. Hafeez Y, Rodriguez Q, Ahmed I, Grossman SA, Haddad LM. (2025). Paroxysmal Supraventricular Tachycardia (Nursing). Treasure Island (FL): StatPearls Publishing; 2025 Jan-
2. Orejarena LA, Vidaillet H, DeStefano F, Nordstrom DL, Vierkant RA, Smith PN, Hayes JJ. Paroxysmal supraventricular tachycardia in the general population. Journal of the American College of Cardiology. 1998 Jan;31(1):150-7.
3. Yetkin E, Ozturk S, Cuglan B, Turhan H. Clinical presentation of paroxysmal supraventricular tachycardia: evaluation of usual and unusual symptoms. Cardiovascular endocrinology & metabolism. 2020 Dec 1;9(4):153-8.
4. Kautzner J, Hašková J, Cvek J, Adamíra M, Peichl P. (2024). Hypertrophic obstructive cardiomyopathy with recurrent ventricular tachycardias: from catheter ablation and stereotactic radiotherapy to heart transplant—a case report. European Heart Journal-Case Reports.8(8): ytae379.
5. Zaiser E, Sehnert AJ, Duenas A, Saberi S, Brookes E, Reaney M. (2020). Patient experiences with hypertrophic cardiomyopathy: a conceptual model of symptoms and impacts on quality of life. Journal of patient-reported outcomes. 4:1-1.
6. Al-Khatib SM, Page RL. (2016). Acute treatment of patients with supraventricular tachycardia. JAMA cardiology. 1;1(4):483-5.
7. Glavaški M, Velicki L. (2021). Shared molecular mechanisms of hypertrophic cardiomyopathy and its clinical presentations: automated molecular mechanisms extraction approach. Life. 3;11(8):785.
8. Cheng Z, Fang T, Huang J, Guo Y, Alam M, Qian H. (2021). Hypertrophic cardiomyopathy: from phenotype and pathogenesis to treatment. Frontiers in Cardiovascular Medicine. 25; 8:722340.