Cambridge City Publishers

Case Reports in Clinical Cardiology Journal

Case Report | Vol 5 Iss 4

Right Ventricular Outflow Tract PVCs and Long QT in a 22-Year-Old Female with COVID-19 and Concomitant Nirmatrelvir-Ritonavir and Fluoxetine Use

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Received: October 19, 2025; **Accepted:** November 04, 2025; **Published:** November 15, 2025

Abstract

We present a case of a young woman who developed right ventricular outflow tract (RVOT) originating arrhythmias and QT prolongation following a COVID-19 infection and concomitant use of nirmatrelvir-ritonavir (Paxlovid) and fluoxetine. She was initially treated with mexiletine and ultimately received an implantable cardiac monitor. This case underscores the importance of evaluating medication interactions and their arrhythmogenic potential, especially in vulnerable populations, including those with a familial history of sudden cardiac death.

Keywords: RVOT PVC; Long QT; COVID-19; Paxlovid

Introduction

Premature ventricular complexes (PVCs) commonly originate from the outflow tracts, with 70-80% arising from the right ventricular outflow tract (RVOT) [1]. RVOT PVCs are typically idiopathic, occurring in the absence of structural heart abnormalities. Long QT syndrome (LQTS), a well-known cause of sudden cardiac death in the young, may be either inherited or acquired, often secondary to medications or comorbidities [2].

Here, we describe a case of a young woman, chronically on fluoxetine, who was placed on nirmatrelvir-ritonavir (Paxlovid) following a COVID-19 infection. She subsequently developed RVOT-originating arrhythmias and QT prolongation, underscoring the importance of evaluating medication interactions and their arrhythmogenic potential, especially in vulnerable populations.

Case Presentation

A 22-year-old woman with a medical history of obsessive-compulsive disorder, anxiety, depression, and ADHD presented to the emergency department with 24 hours of nausea and bilious, non-bloody vomiting. Four days earlier, she had tested positive for COVID-19 and began treatment with nirmatrelvir-ritonavir. She completed four days of therapy before discontinuing it due to worsening gastrointestinal symptoms.

At urgent care, an ECG revealed bigeminy PVCs. On admission to the community hospital, she reported chest tightness and lightheadedness but denied chest pain, palpitations, or dyspnea. She did report transient exertional palpitations in the past without syncope. Her only regular medication was fluoxetine, taken for the past eight years.

Family history was notable for sudden cardiac death in a great aunt at age 37 and a benign arrhythmia in her father. Admission ECG showed PVC couplets with left bundle branch block (LBBB) morphology in lead V1, inferior axis, and a precordial transition at V3. Lead I was low amplitude and positive. A three-beat salvo of non-sustained ventricular tachycardia (NSVT) was noted, with a corrected QT interval (QTc) of 513 ms.

Laboratory workup showed potassium of 3.5 mmol/L, magnesium of 2.0 mg/dL, and negative serum troponins. A urine drug screen was positive for cannabinoids. PCR confirmed COVID-19 infection.

Fluoxetine and nirmatrelvir-ritonavir were discontinued, and she was treated with intravenous fluids, potassium, and magnesium. Antiemetics included trimethobenzamide and lorazepam. Despite the correction of electrolytes, PVC couplets persisted.

Echocardiography revealed normal systolic and diastolic function with an ejection fraction of 60%. After transfer to a tertiary care center, PVCs resolved on telemetry. Repeat ECG showed normal sinus rhythm at 61 bpm with a QTc of 569 ms. She was started on mexiletine 200 mg every eight hours and nadolol 20 mg daily. Cardiac MRI revealed no structural abnormalities. On hospital day eight, her ECG showed sinus bradycardia at 47 bpm with a QTc of 453 ms. An implantable cardiac monitor (Medtronic Reveal LINQ) was placed, and she was discharged with plans for outpatient electrophysiology follow-up.

Discussion

The RVOT is a frequent origin of PVCs and ventricular tachyarrhythmias, often occurring in structurally normal hearts. RVOT PVCs exhibit characteristic ECG findings: LBBB morphology, inferior axis, rS pattern in V1, and R waves in V6. A positive lead suggests a posteroseptal RVOT origin [3,7]. Though traditionally considered benign, some RVOT PVCs are now understood to occur in the context of subtle structural or electrical remodeling, influenced by genetic and hormonal factors [7].

Whether inherited or acquired, LQTS is a significant risk factor for sudden cardiac death in young patients. Inherited LQTS often involves mutations in ion channel genes (e.g., KCNQ1), whereas acquired forms are typically due to medications, electrolyte abnormalities, or infections [8]. Numerous medications are implicated in QT prolongation, including antiarrhythmics (sotalol, amiodarone), antibiotics (azithromycin, levofloxacin), and antipsychotics (haloperidol).

COVID-19 itself has been associated with ventricular arrhythmias and QT prolongation [4]. Nirmatrelvir-ritonavir (Paxlovid), used for mild-to-moderate COVID-19, has been linked to bradycardia and syncope, particularly when combined with other medications. Ritonavir, a potent CYP3A4 inhibitor, can amplify the effects and toxicity of QT-prolonging drugs [9]. In this case, the concurrent use of Paxlovid and fluoxetine may have contributed synergistically to QT prolongation.

Fluoxetine is generally considered to pose a lower QT risk among SSRIs. Still, there are case reports of Torsades de Pointes and cardiac arrest in patients on chronic fluoxetine when combined with other QT-prolonging agents [10].

Though isolated PVCs are often benign, they can escalate to NSVT, sustained VT, or even ventricular fibrillation. Rarely, frequent PVCs may result in PVC-induced cardiomyopathy [11]. In symptomatic or high-risk patients, treatment may be warranted. While catheter ablation offers high success rates [1,11], pharmacologic management with beta-blockers or sodium channel blockers (such as mexiletine) remains first-line for many, especially in LQTS [5,6].

Muser et al. proposed a stepwise diagnostic algorithm for PVCs, including thorough clinical history, ECG localization, stress testing, echocardiography, and cardiac MRI [11]. Medical therapy—starting with beta-blockers or non-dihydropyridine calcium channel blockers—may be escalated to agents such as flecainide, sotalol, or amiodarone when needed. Ablation may be considered in refractory cases or in those averse to long-term medications.

Our patient was successfully managed with nadolol and mexiletine after spontaneous resolution of PVCs and normalization of QTc. This case highlights the importance of early recognition and intervention in young patients with new-onset arrhythmias, especially when multiple contributing factors—including drug interactions and viral infections—are present.

Conclusion

- A thorough clinical history is essential in young patients presenting with arrhythmias. Inherited and acquired channelopathies should remain on the differential.
- Clinicians should remain vigilant about adverse drug interactions, particularly in patients on chronic medications with potential QT-prolonging effects.
- Management of RVOT PVCs and LQTS may involve both pharmacologic and procedural strategies. Awareness of ablation as a definitive option is increasingly important.
- Cardiac MRI and continuous rhythm monitoring may be critical in risk stratification and follow-up.

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