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Case Report

Premature Ventricular Contractions as an Underrecognized Cause of Chronic Cough: A Case of Misdiagnosis in a Patient with Bronchiectasis

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INTRODUCTION

Cough in adults typically is classified as acute, subacute, or chronic, with chronic cough defined as lasting longer than eight weeks.[1] Common causes of chronic cough include asthma and gastroesophageal reflux disease (GERD), while less common causes include premature ventricular contractions (PVCs) and anatomic airway abnormalities.[1,2] Although PVCs frequently are encountered, their role as a cause of chronic cough is documented but often overlooked.[2]

The most widely accepted mechanism by which PVCs cause cough involves irritation of the phrenic nerve and/or diaphragm due to the strong ventricular contraction following a PVC.[3] Another theory suggests that stimulation of cardiac vagal nerve endings directly may provoke cough or heighten cough reflex sensitivity, given the shared vagal innervation of the heart and airway.[3]

PVCs as the sole cause of chronic cough are rare.[2] In one cross-sectional study, fewer than 1% of individuals with PVCs had chronic cough solely due to PVCs, while approximately 4% had cough attributed to PVCs in combination with another cause.[4]

This case report presents a patient with a chronic dry cough initially attributed to bronchiectasis but ultimately found to be caused by PVCs.

CASE REPORT

The patient was a 71-year-old woman with a history of coronary artery disease status post coronary artery bypass grafting (CABG), hyperlipidemia, hypertension, hypothyroidism, GERD, chronic kidney disease (CKD) stage 3a, and a remote smoking history (<20 pack-years). She had been diagnosed with bronchiectasis of unclear etiology in 2003 following evaluation for chronic dry cough, hemoptysis, and dyspnea.

Initial high-resolution chest computed tomography (CT) showed bronchiectasis, most prominent in the left lower and right middle lobes. A comprehensive workup, including bronchoscopy, immune deficiency screening, autoimmune and genetic panels, was unremarkable. Pulmonary function testing (PFT) revealed mild obstruction. Management included azithromycin, inhaled bronchodilators, and airway clearance therapy. PVCs were noted on electrocardiogram (EKG), but her symptoms were attributed to bronchiectasis.

Despite ongoing therapy, her cough and dyspnea worsened over the next 15 years. A 2017 CT confirmed multifocal cylindrical bronchiectasis (Figure 1), yet PFTs remained stable. Her pulmonologist questioned whether bronchiectasis fully explained her symptoms. Ongoing PVCs, exertional dyspnea, and palpitations prompted a referral to cardiology.

Exercise testing revealed frequent uniform PVCs, including bigeminy at peak exercise (Figure 2). Echocardiography showed moderate mitral regurgitation, and 24-hour ambulatory electrocardiographic monitoring (Holter monitor) recorded an 18.8% PVC burden with frequent couplets and trigeminy (Figures 3-5). She was started on acebutolol, which improved both cough and dyspnea.

Coronary angiography confirmed significant proximal anterior descending artery stenosis, and she underwent uncomplicated CABG. Post-operatively, exertional dyspnea improved significantly, and a repeat Holter showed a reduced PVC burden (7.3%). However, a month later, she reported increased nighttime cough and palpitations, despite the lower PVC burden. Empiric treatment with intranasal fluticasone and omeprazole for postnasal drip and reflux improved her nighttime cough.

Subsequent stress testing and a 30-day event monitor showed further PVC reduction to 4%. Over the following year, her cough resolved, PVCs decreased, and PFTs remained stable. At her 2019 follow-up, she had no bronchiectasis exacerbations, jogged 2.5 miles daily, and her EKGs were normal.

By 2023, spirometry showed improved forced expiratory volume in 1 second (FEV1) without airflow obstruction. However, in 2024, her cough recurred alongside increased PVCs, as noted on a wrist-worn wearable device (Apple Watch, Apple Inc., Cupertino, CA). EKG confirmed bigeminal PVCs. Since acebutolol 400 mg was no longer effective, her dose was increased to 600 mg daily in divided doses, resulting in improved symptom control.

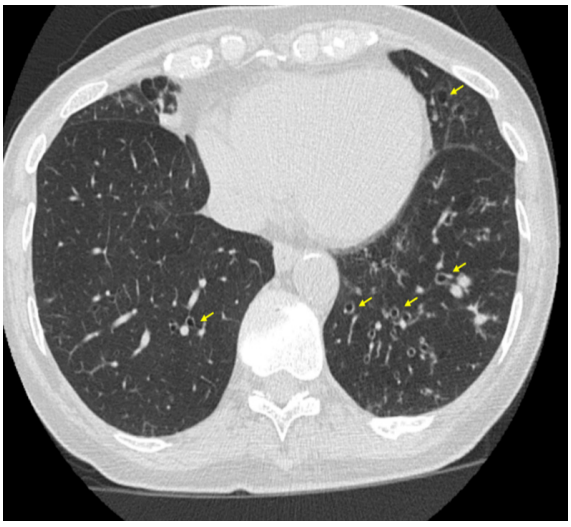

Figure 1. Computed tomography (CT) of the chest showing multifocal cylindrical bronchiectasis (yellow arrows), predominantly in the left lower lobe.

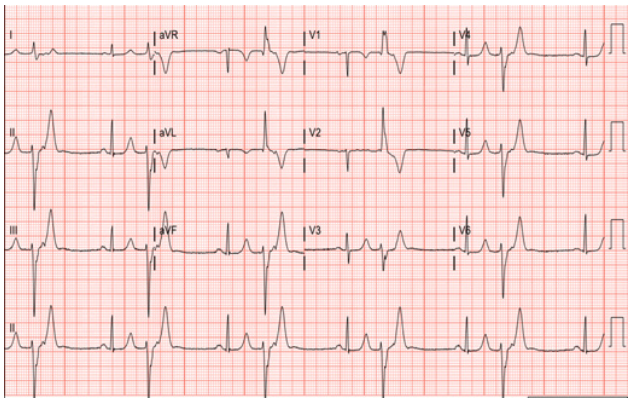

Figure 2. Electrocardiogram in 2017 demonstrating frequent premature ventricular complexes (PVCs) in a bigeminal pattern.

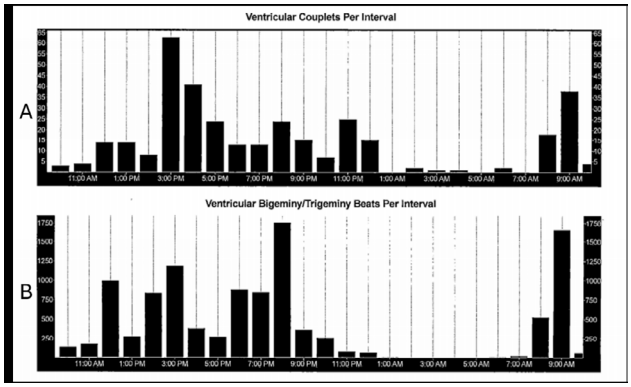

Figure 3. Ventricular events on 24-hour Holter monitor. A - Frequency of premature ventricular complex (PVC) couplets, B - Frequency of ventricular bigeminy/trigeminy.

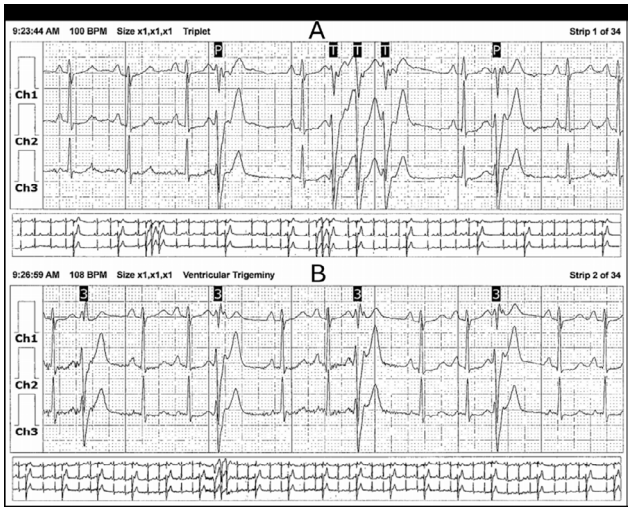

Figure 4. Ventricular events on 24-hour Holter monitor. A – Premature ventricular complex (PVC) triplets, B - Ventricular trigeminy.

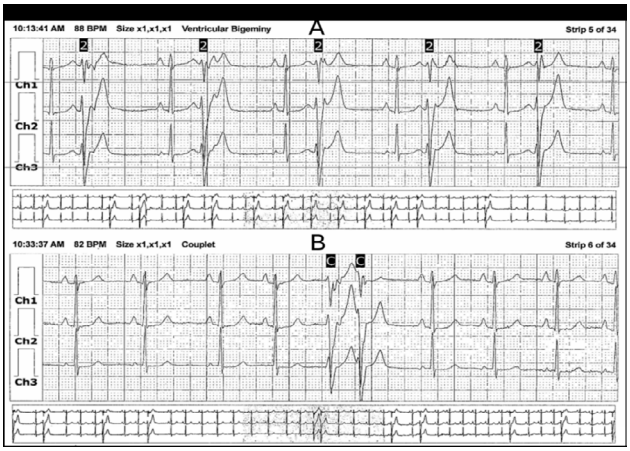

Figure 5. Ventricular events on 24-hour Holter monitor. A - Ventricular bigeminy, B - Premature ventricular complex (PVC) couplets.

DISCUSSION

Cough can both result from and contribute to cardiac arrhythmias.[3] This case highlights an uncommon but significant etiology of chronic cough, PVCs. While chronic cough typically is attributed to pulmonary or upper airway disorders, this patient's lack of response to standard therapies and the correlation between symptom severity and PVC burden suggested a cardiac origin.

The exact mechanism of PVC-induced cough remains uncertain. Proposed explanations include phrenic nerve irritation from the stronger post-extrasystolic contraction and vagal-mediated reflexes due to shared vagal innervation of the heart and airways.[3] In this case, the patient's symptom resolution with beta-blocker therapy and reduced PVC burden supports a likely causal relationship.

Notably, she lacked hallmark features of bronchiectasis, such as a productive cough, wheezing, and frequent exacerbations. Despite a diagnosis of bronchiectasis, she had a 15-year history of a dry, treatment-resistant cough with no documented exacerbations, features inconsistent with classical bronchiectasis.

Compared with the limited literature, most reported cases of PVC-induced cough were diagnosed within months to a few years.[2,5] This case stands out due to the extended duration of misdiagnosis and the presence of multiple confounding comorbidities, including GERD, postnasal drip, and mitral regurgitation. Although mitral regurgitation may contribute to cough via pulmonary congestion or airway irritation, her imaging showed no signs of vascular congestion, making this an unlikely cause.

Furthermore, her cough was unresponsive to treatments targeting GERD and postnasal drip, but improved with cardiac therapy, further implicating PVCs as the primary etiology. The recurrence of both cough and PVCs in 2024 raises the possibility of evolving beta-blocker resistance or new triggers.

This case emphasizes the diagnostic challenges posed by atypical or overlapping clinical features. Misattribution of symptoms to more common pulmonary conditions can lead to years of ineffective treatment and patient frustration. Holter monitoring and exercise testing can reveal occult arrhythmias that may otherwise go undetected.

CONCLUSIONS

PVC-induced chronic cough, though rare, should be considered in patients with unexplained cough, especially when standard treatments fail, or symptoms are accompanied by palpitations or exertional dyspnea. The temporal relationship between PVC burden and cough, along with improvement following antiarrhythmic therapy, supports a causal link in this case.

Clinicians should maintain a broad differential when evaluating chronic cough. A multidisciplinary approach, integrating pulmonology and cardiology, is essential, particularly when initial assessments are inconclusive. With increased use of wearable technology, patients may independently detect arrhythmias, offering additional diagnostic insight. Early recognition of PVCs as a potentially reversible cause of chronic cough can lead to more effective management, reduce unnecessary interventions, and improve patient outcomes.

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