PREMATURE VENTRICULAR COMPLEX-INDUCED CARDIOMYOPATHY: A CASE REPORT AND REVIEW OF LITERATURE

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ABSTRACT

Introduction: Frequent premature ventricular complexes (PVCs) have theability to cause or contribute to cardiomyopathy and heart failure symptoms in long-term.
Case Illustration: A 46 years old male presented with recurrent palpitations, the latest since 6 hours before admission. PMH of hypertension, diabetes and heart disease were denied. BP: 120/80 mmHg, HR: 138 bpm, RR 22x/minute. ECG: LV focus multifocal PVC bigeminy. **Lab:** within normal limits. **CXR:** cardiomegaly. Echocardiography: MR and LVH. The patient was administered diltiazem IV in the ED. Bisoprolol was given during discharge.
Discussion: PVC-induced cardiomyopathy's mechanisms are not entirely clear; a more likely explanation is abnormal ventricular activation resulting in mechanical dyssynchrony, >10.000 PVCs/day has high risk and this patient's PVC burden should be calculated. It is a diagnosis of exclusion andmade after exploring possible causes and excluding them.Pharmacotherapy to suppress PVCs includes β-Blockers, CCB, and other antiarrhythmic drugs. Results of the use of β-Blockers and CCB are modest with reported efficacy rates in the 20% range.In patients with PVC-induced cardiomyopathy, successful elimination of PVCs with ablation frequently restores ventricular function acutely and has aprocedural success rate of PVC elimination of 84%. Unfortunately, this patient is not a suitable candidate for catheter ablation. Hence, β-Blockers was chosen for long-term therapy.
Conclusion: Frequent PVCs with high burden has potential to cause adverse cardiovascular events and should be treated to prevent its deleterious consequences. Suppression of PVCs using either antiarrhythmic pharmacological agents or emerging catheter ablation techniques appears to reverse the LV dysfunction.
Keywords: PVC, Cardiomyopathy, burden

ABSTRAK

Pendahuluan: Kompleks ventrikular prematur (KVP) memiliki kemampuan untuk menyebabkan kardio miopati atau gejala gagal jantung dalam jangka panjang.
Ilustrasi Kasus: Seorang laki-laki berusia 46 tahun datang dengan keluhan berdebar-debar sejak 6 jam sebelum masuk rumah sakit. Riwayat penyakit hipertensi, diabetes, dan gagal jantung disangkal. Tekanan darah 120/80 mmHg, detak jantung 138 kali/menit, laju nafas 22x/menit. EKG menunjukan KVP bigemini multifocal. Pemeriksaan laboratorium dalam batas normal. Ro thoraks: kardiomegali. Ekokardiografi: MR dan LVH. Pasien diberikan diltiazem IV saat di IGD. Pasien diberikan bisoprolol saat pulang.
Diskusi: Mekanisme mengapa kardiomiopati yang disebabkan oleh VKP masih belum sepenuhnya jelas; penjelasan yang paling memungkinkan adalah aktivasi abnormal pada ventrikel yang menyebabkan fungsi mekanis menjadi tidak sinkron, VKP >10.000/hari memiliki risiko yang tinggi. Hal tersebut merupakan diagnosis eksklusi setelah menyingkirkan kemungkinan penyebab lain. Farmako terapi untuk mengurangi VKP adalah dengan penyekat beta, penyekatk anal kalsium, dan obat antiaritmik lainnya. Pengunaan penyekat beta dan kanal kalsium memiliki efikasi hanya sekitar 20%. Ablasi kateter yang berhasil dapat memperbaiki fungsi ventrikel dengan angka keberhasilan 84%, namun, pasien ini bukan merupakan kandidat yang cocok untuk ablasi kateter. Oleh karena itu penyekat beta merupakan pilihan untuk terapi jangka panjang.
Kesimpulan: Kontraksi ventrikular prematur yang sering berpotensi untuk menyebabkan adverse cardiovascular events dan harus ditangani untuk mencegah komplikasi. Mengurangi VKP dengan obat anti aritmik atau ablasi kateter dapat mengembailkan fungsi ventrikel.
Kata kunci: VKP, kardiomiopati, beban

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Kesimpulan: Kontraksi ventrikular prematur yang sering berpotensi untuk menyebabkan *adverse cardiovascular events* dan harus ditangani untuk mencegah komplikasi. Mengurangi VKP dengan obat anti aritmik atau ablasi kateter dapat mengembailkan fungsi ventrikel.

Kata kunci: *VKP, kardiomiopati, beban*

INTRODUCTION

Premature ventricular complex (PVCs) without underlying cardiac disease were longed thought to be benign, it is true in most cases. However, recent evidence showed that frequent PVCs have theability to cause or contribute to cardiomyopathy and heart failure symptoms in long term.[1]Premature ventricular contractions-induced cardiomyopathy is reversible cardiomyopathy due to PVCs.[2] In this paper we will present a patient without other cardiovascular risk factors developed a left ventricular hypertrophy accompanied with frequent PVCs.

CASE ILLUSTRATION

A 46 years old male presented with recurrent palpitations, with the latest was since 6 hours before admission. He frequently complained of palpitations for most days of the week, however, he felt that the symptoms were more severe at that time. He did not experience chest pain, shortness of breath, nausea, vomiting or sweating. He did not complaint of paroxysmal nocturnal

dyspne a, orthopnea, syncope or edema. Past medical history of hypertension, diabetes and heart disease were denied. The patient did not smoke and avoid caffeine. On presentation, to emergency department the patient was fully conscious with blood pressure of 120/70 mmHg, heart rate of 138 bpm, and respiratory rate of 22x/minute.

Cardiac examination revealed a grade II/VI systolic murmur at the apex radiating to axilla suggestive of mitral regurgitation with apex displaced downward and leftward. There was no hepatomegaly, edema or enlarged thyroid glands. Electrocardiography showed frequent multifocal PVC (bigeminy) originating from left ventricular focus**(Fig 1)**. Complete blood counts, glucose, electrolytes, renal and liver function werewithin normal limits. Chest X-Ray showed cardiomegaly**(Fig 2).** The patient was admitted and given intravenous diltiazem. Echocardiography showed mitral regurgitation (MR) and left ventricular hypertrophy (LVH)**(Fig 3)**. The patient was diagnosed with tachycardia-induced cardiomyopathy with differential diagnosis of primary valvular heart disease. The patient was given bisoprolol and discharged from the hospital.

DISCUSSION

Premature ventricular complex-induced cardiomyopathy's mechanisms are not entirely clear; a more likely explanation is abnormal ventricular activation resulting in mechanical dyssynchrony, >10.000 PVCs/day has high risk and this patient's PVC burden was >10.000 PVCs/day.[3]It is a diagnosis of exclusion and made after exploring possible causes and excluding them.This patient denies previous history and other risk factors for cardiovascular disease. Blood pressure, glucose,and electrolytes were also within normal limits. Electrocardiography, chest x-ray, and echocardiography in this patient showed LVH with MR. Frequent palpitation and evidence of frequent PVCs without other cardiovascular risk factors or history may explain the cause of LVH. Mitral regurgitation may be due to LVH or primary valvular disease, hence, a differential diagnosis.

Further investigations should be performed, including 24-hour Holter monitor to quantify PVC burden and can detect any related arrhythmias. In those with exercise-inducedPVCs, treadmill test may be ordered. Testing for sleep apnea is appropriate if there is high nocturnal PVCs burden. Typically, electrophysiological studies may not be needed but may be

helpful to detect potential causes of PVCs such as arrhythmogenic right ventricular dysplasia and infiltrative cardiomyopathies.[2,4]

In healthy people with PVC, adjusted hazard ratio for cardiac death is 3,98 & 0,95 in male and female, respectively.[5]Indications to initiate therapy for patients with PVCs include bothersome symptoms, the presence of complications (cardiomyopathy with decreased EF or ventricular dilatation and PVC induced tachycardia), PVCs triggering malignant ventricular arrhythmias, and PVCs limiting optimal biventricular pacing.[3]Management in asymptomatic patients with normal LV function and a very frequent PVCs (>20%) is still controversial. Whether it should be managed with medication or ablation to prevent future risk of cardiomyopathyor follow-up assessment of left ventricular function is still undetermined.[3]No treatment other than reassurance needed in patients without inherited arrhythmia syndromes who have asymptomatic or mildly symptomatic PVCs. In those with asymptomatic PVCs and a normal LV function without accompanying structural heart disease,consider follow-up in those with PVC burden >10,000 in 24 hours or in patients with PVC burden <10,000 in 24 hours if symptoms worsen. Medications are reported to suppress PVCs in 10%-40% of patients.Pharmacotherapy to suppress PVCs includes β-Blockers (1st line), CCB, and other antiarrhythmic drugs.[6,7] Results of the use of β-Blockers and CCB are modest with reported efficacy rates in the 20% range. In CHF-STAT trial, amiodarone decreased hourly PVCs (44 ± 145 vs 254 ± 370, $P < .001$) with 69% of patients experiencing an 80% decrease in PVC burden at 3 months. However, long-term use is limited by its adverse effect profile.

Catheter ablation has emerged as a relatively safe and effective option to pharmacotherapy for PVC elimination. It is recommended in right ventricular outflow tract (RVOT) PVC or VT with symptoms, failed pharmacotherapy or decline in LV function. It is also indicated in PVCs that trigger recurrent ventricular fibrillation leading to implantable cardioverter defibrillation (ICD) shock or lead to electrical storms.[2,6,7]This patient fulfilled neither of those criteria. In patients with PVC-induced cardiomyopathy, successful elimination of PVCs with ablation frequently restores ventricular function and has anacute procedural success rate of PVC elimination of 84% (80%-90%) of patients with complications reported in 0%-8% of

patients.[2] Predictors of success were RVOT PVC location and monomorphic as opposed to multiple PVC morphologies. Unfortunately, both were not present in this patient, the likelihood of success and clinical improvement vs the potential risk should be weighted. Factors to evaluate include PVC frequency, anticipated PVC location, the number of PVC morphologies, pharmaceutical alternatives, and patient age and comorbidities.We chose pharmacotherapy over ablation withβ-Blockers(1st line medication) was chosen as the initial therapy.

CONCLUSION

Frequent PVCs with high burden has potential to cause adverse cardiovascular events and should be treated to prevent its deleterious consequences. Suppression of PVCs using either antiarrhythmic pharmacological agents or emerging catheter ablation techniques appears to reverse the LV dysfunction.

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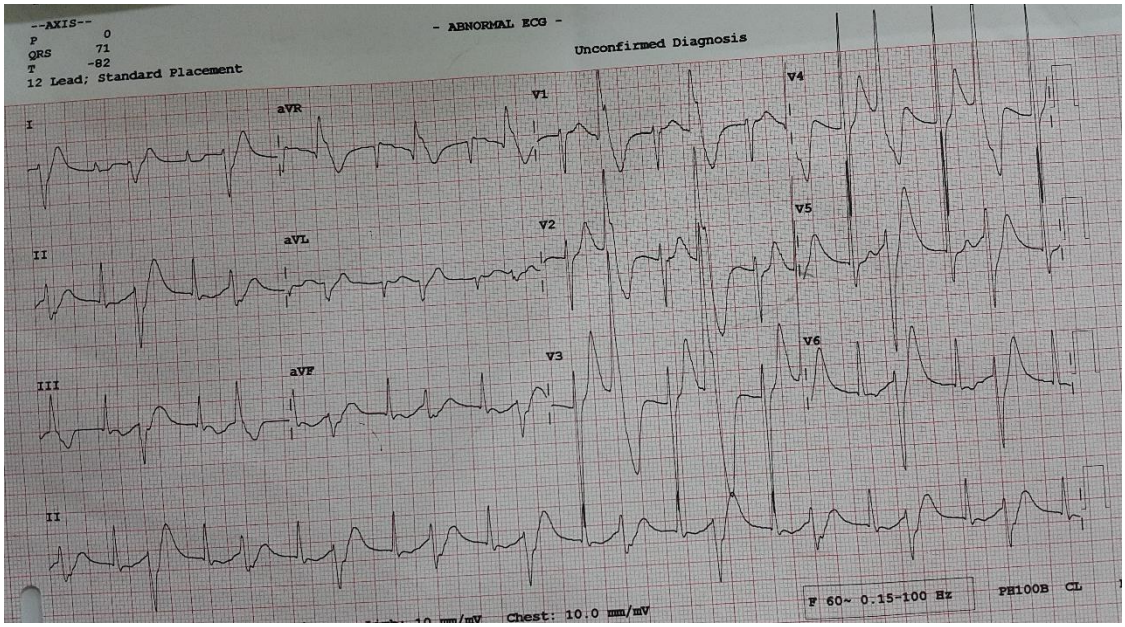

Fig 1. Patient's ECG

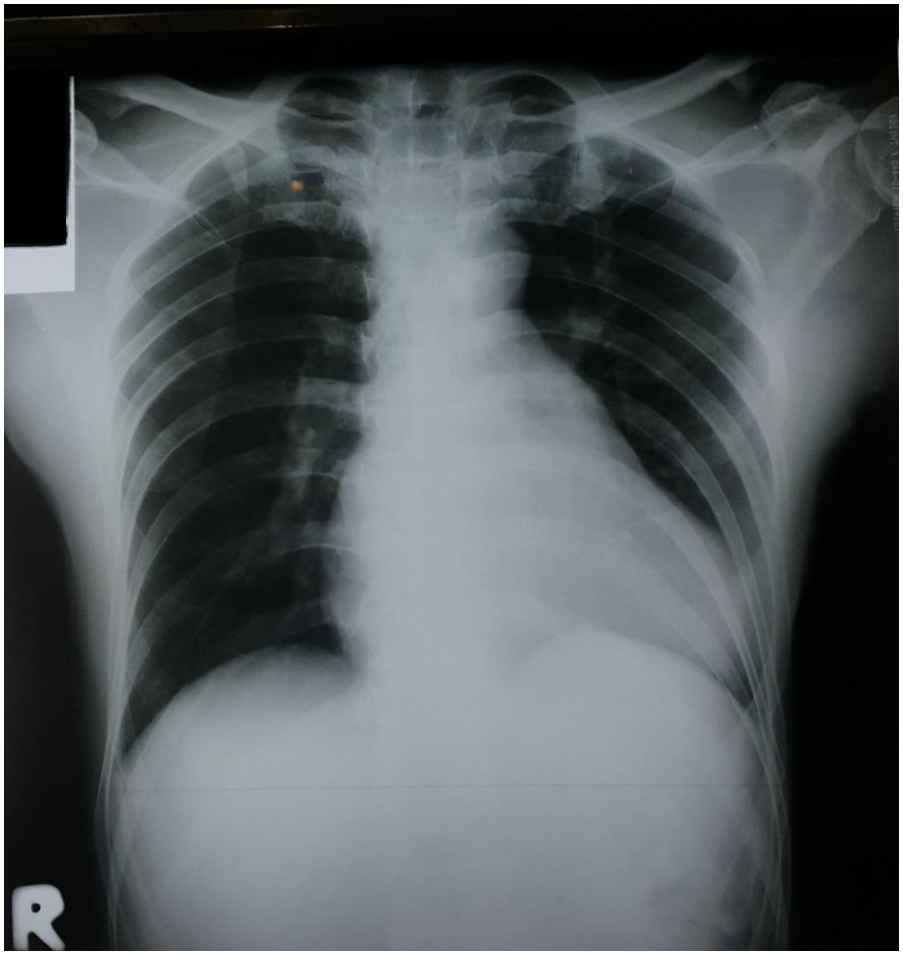

Fig 2. Patient's Chest X-Ray

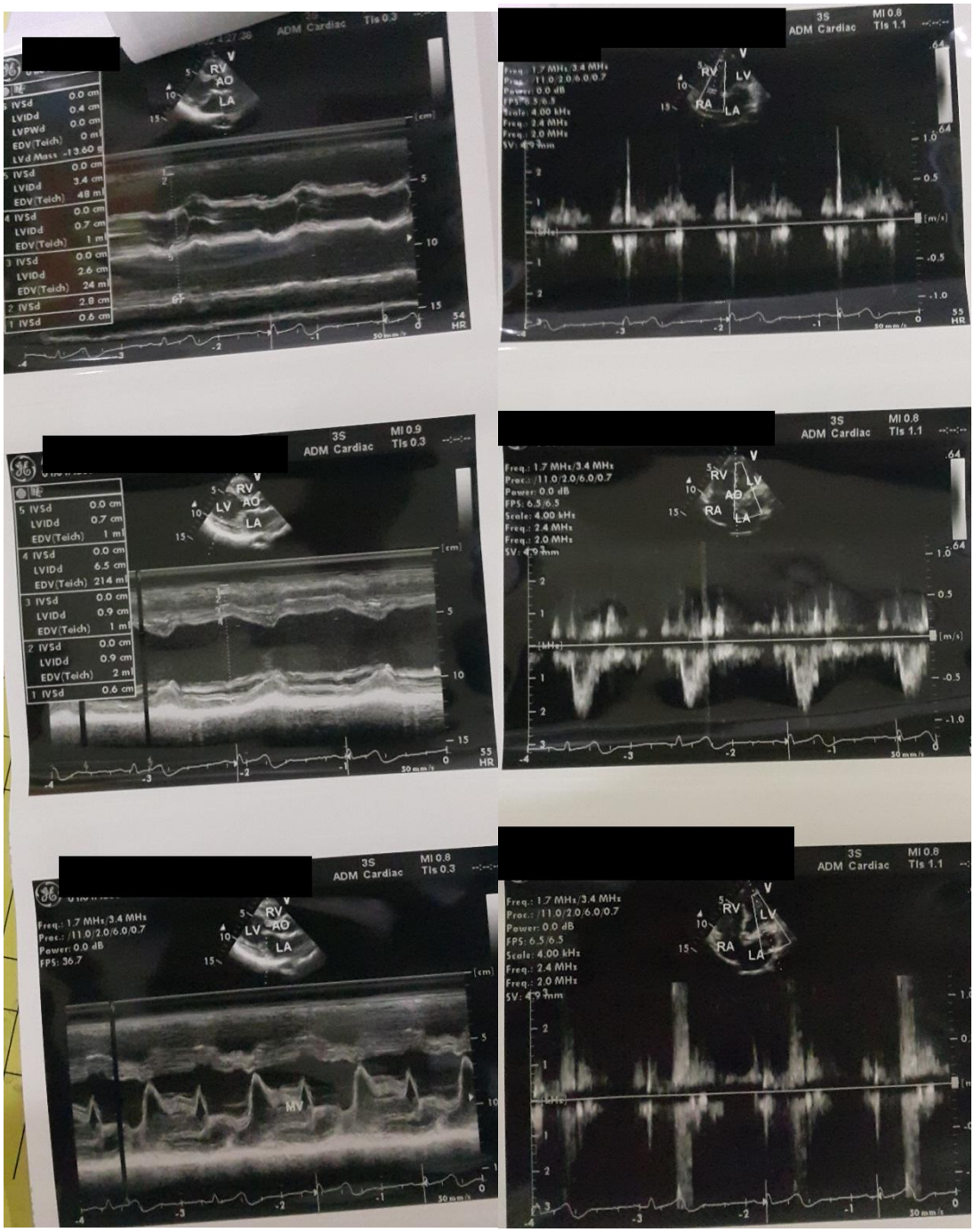

Fig 3. Patient's Echocardiography