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Idiopathic premature ventricular contractions in a pregnant woman – A case report

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Abstract

The cause of premature ventricular contractions remains unknown. PVCs are usually benign in patients with structurally normal heart. A detailed clinical history and physical examination maybe enough to diagnose PVCs as the pulse rate will have skipped beats. A 12 lead ECG can diagnose PVCs. 2D ECHO a non-invasive procedure can be used as a diagnostic tool to rule structural heart defects. Maternal and fetal outcomes are good in asymptomatic PVCs. PVCs can be treated with beta blockers to make the patient comfortable. Many patients with PVCs may require only reassurance. Family history plays a major role in aiding the diagnosis of PVCs in asymptomatic patients by knowing about the family members who died suddenly or at an early age. Many more works need to be done in the community-based settings to make aware and to identify the risk population.

KEYWORDS: Premature ventricular contractions**,** skipped beats, PVC burden

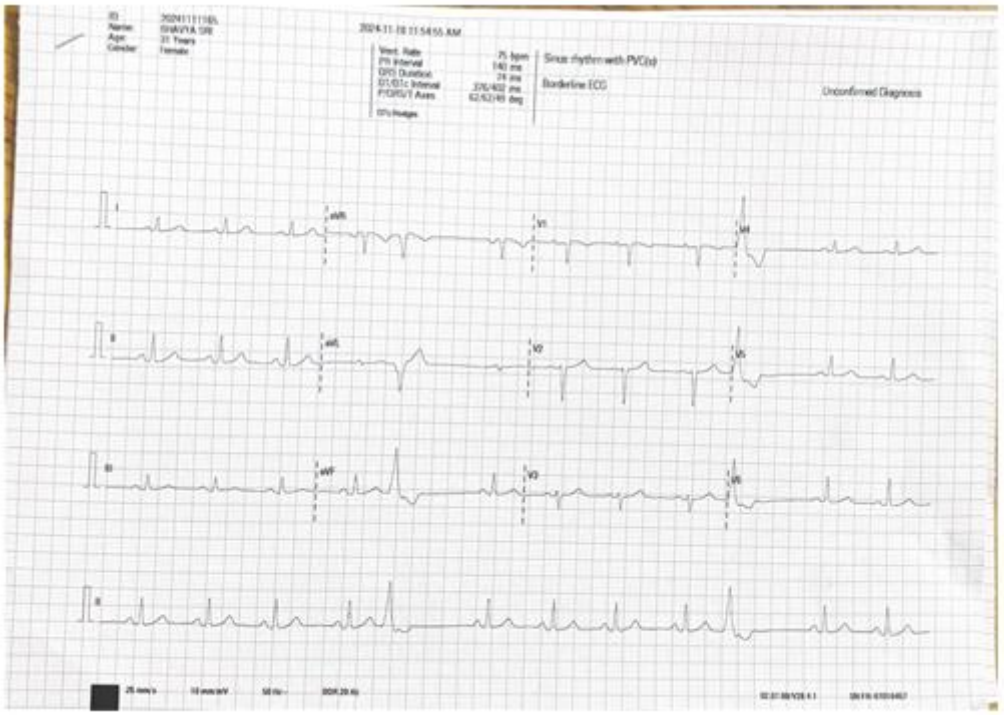
INTRODUCTION

- Idiopathic premature ventricular contractions (PVCs) are relatively benign in cases without structural heart diseases but may signal as increased risk of sudden death in cases with structural heart disease (SHD) and maybe markers of underlying pathology.
- An estimated prevalence of 1-4%is found in the general population on standing 12-lead ECG.
- The physiological alterations during pregnancy result in hemodynamic changes, including increased heart rate, intravascular blood volume and cardiac output, and reduced systemic vascular resistance. All of these may provoke or exacerbate maternal arrhythmias.
- In pregnant patients with CVD,arrhythmias and heart failure are the most common complications that arise.
- Heart failure also has variable manifestations in pregnant patients ranging from mild left ventricular impairment to patients with advanced heart failure with acute decompensated heart failure.
- Premature atrial and ventricular contractions are common due to hormonal and volume changesapart from these,the most common maternal arrhythmias in pregnancy in the order of observed prevalence are supraventricular arrhythmias, atrial fibrillation and ventricular arrhythmias.
- Symptoms include palpitations,presyncope,chest pain,and syncope,especially in the setting of ventricular arrhythmias.

CASE REPORT:

A 31, year old female, G2A1, 37 weeks of GA came to regular antenatal checkup.On general examination while recording the pulse rate skip beats were noted. She had similar complaints in the past (5years ago, in previous pregnancy), no significant family history. The patient has no history of arrhythmias. There is no known history of cardiovascular disease in her immediate family. All the trimesters were uneventful. On further investigation, her ECG and 2D ECHO revealed premature ventricular contractions with ectopic beats. Cardiologist opinion was taken and patient was prescribed TAB.MET XL 12.5 mg /po/od for one week and patient was advised admission for safe confinement. Her

vitals signs were within normal limits. Her physical examination was unremarkable with regular cardiac rate and rhythm noted in auscultation and no lower extremity edema. At Admission laboratory tests results were notable for hemoglobin level of 12.4 gm/dl, platelet count of 2.3 lakhs/cumm,and thyroid level,potassium and ionized calcium were within normal limits. A cardiology opinion was taken and mode of delivery was advised to be ELECTIVE LSCS. Case was shifted to cardiac ICU at 3pm on 18/11/24 for better care and management and postoperatively routine antibiotics, analgesics were given. ECG was advised every 6th hourly. ECG was repeated which showed sinus rhythm with frequent premature ventricular contraction



Post operatively on day 0 serum electrolytes were repeated which showed decreased serum calcium and magnesium. Then Inj. Magnesium sulfate 4 gms IV In 100 ML NS given over 15 mins. Correction of calcium was done with Inj. Calcium gluconate 1 amp in 100 ml NS over 15 mins. ECG were repeated every 6th hourly until 24 hrs which showed decrease in premature ventricular contraction rate. Patient vitals were stable on post operative day 1, early ambulation, routine antibiotics and analgesics were given. Breast feeding was encouraged. ECG on day 1 showed decreased PVCs.

DISCUSSION

Idiopathic PVCs are benign in structurally normal hearts. Patients with PVCs typical come to clinical attention for one of two reasons:

1. Symptoms
2. An incidental finding on medical examination

Symptoms include palpitations which may manifest as chest discomfort, sensation of skipped or irregular heartbeats, presyncope, dyspnea and fatigue. The proportion of patients with asymptomatic versus symptomatic is not known. A family history is important to elucidate any possible inherited disorders that may be associated with PVCs and risks for sudden death. As initial screening it is useful to recommend asking about any first-degree family members who either died suddenly or at an early age. In the absence of symptoms PVCs may manifest as irregular pulse or as an incidental finding on ECG. Both patients and doctors should become alert by apparent bradycardia by a palpable pulse alone occurring because of intermittent, poorly perfused PVCs. Beta blockers or calcium channel blockers are helpful in making the patient comfortable.PVCs can be diagnosed only by 12 lead ECG and it is non invasive to determine PVC location. 24hour Holter monitoring is gold standard in assessing PVC frequency. Recent evidence supports that monitoring maybe required before the maximum daily PVC frequency is observed. Daily monitoring of ECG can also be done. An Echocardiogram is indicated for everyone because of PVCs to exclude structural heart diseases. As a routine practice of advising ECG in clinical scenario may help to identify the patients of asymptomatic PVCs and they can be treated with beta blockers.Many patients with PVCs do not develop systolic dysfunction or clinical heart failure. It is interesting to identify these patients by a routine ECG. Many more works needto be done in the community-based settings to make aware and to identify the risk population. Many of the patients with PVCs can be managed by giving reassurance.

For patients requiring further management apart from reassurance following has to be looked into

1. Information regarding symptoms
2. PVC burden (which is the percentage of all beats in PVCs)
3. Absence or presence of structural heart disease

If the PVC burden is low, ejection fraction normal, with no structural heart diseases reassurance with low dose beta blockers are helpful.

CONCLUSION

Pregnancy and labour are believed to place a women at higher risk of tachyarrhythmias. The risk of ventricular events in pregnancy remains unknown. Idiopathic premature preventricular contractions are benign without structural heart disease. PVCs are best evaluated with detailed clinical history, physical examination, family history and 12 lead ECG. PVC burden is most reliable predictor of incident heart failure. Although many patients with PVCs will require only reassurance, beta blockers. Additional research is needed to identify the fundamental etiologies of PVCs, the differential PVC frequencies among the individuals that are observed and development of prevention strategies to find out the root cause of PVC. Maternal and neonatal outcomes are usually good with frequent PVC and structurally normal heart.

REFERENCES

1. Tong C, Kiess M, Deyell MW, et al. Impact of frequent premature ventricular contractions on pregnancy outcomes. Heart. 2018;104(16):1370-1375.https://pubmed.ncbi.nlm.nih.gov/29463610/.
1. H. Roukoz, W. Saliba, Dofetilide: a new class III antiarrhythmic agent, Expert. Rev. Cardiovasc. Ther. 5 (2007) 9–19.
2. Nguyen KT, Vittinghoff E, Dewland TA, Dukes JW, Soliman EZ, Stein PK, Gottdiener JS, Alonso A, Chen LY, Psaty BM, et al. Ectopy on a single 12-lead ECG, incident cardiac myopathy, and death in the community. J AmHeart Assoc. 2017;6: e006028.
3. Moss AJ, Davis HT, DeCamilla J, Bayer LW. Ventricular ectopic beats
4. and their relation to sudden and non-sudden cardiac death after myocardial infarction. Circulation. 1979; 60:998–1003. Doi:10.1161/01.cir.60.5.998
5. S. Niwano, Y. Wakisaka, H. Niwano, H. Fukaya, S. Kurokawa, M. Kiryu, et al., Prognostic significance of frequent premature ventricular contractions originating from the ventricular outflow tract in patients with normal left ventricular function, Heart. 95 (2009) 1230–1237.
6. Y.G. Kim, Y.Y. Choi, K.D. Han, K.J. Min, H.Y. Choi, J. Shim, et al., Premature ventricular contraction increases the risk of heart failure and ventricular tachyarrhythmia, Sci. Rep. 11 (2021) 12698.
7. Baman TS, Lange DC, Ilg KJ, Gupta SK, Liu TY, Alguire C, Armstrong W, Good E, Chugh A, Jongnarangsin K, et al. Relationship between burden of premature ventricular complexes and left ventricular function. Heart Rhythm. 2010; 7:865–869. doi: 10.1016/j.hrthm.2010.03.036
8. Latcham Setty R, Bogun F. Premature ventricular complexes and premature ventricular complex induced cardiomyopathy. Curr Probl Cardiol. 2015; 40:379–422. doi: 10.1016/j.cpcardiol.2015.03.002