Frequent Premature Ventricular Contractions

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Premature ventricular contractions (PVCs) are considered normal in many circumstances but can act as markers to other cardiac disease such as cardiomyopathy and ischemic heart disease. This case is presented to illustrate frequent PVCs as a marker for serious heart disease.

CASE PRESENTATION

The proposed insured is a 42-year-old male who applied for a large amount of life insurance with our company for a buy-sell arrangement. He gave no medical history. His screening lab work and a paramed exam were normal. His electrocardiogram (EKG), however, showed a bigeminal rhythm. The native EKG was otherwise normal. Due to the risk involved, and perhaps a suspicious mood was prevailing, the case was declined until further evaluation.

The following information was sent in after the client visited a doctor: “Saw Dr. — because of the PVCs and nonsustained ventricular tachycardia which was of a very mild degree and mild LV [left ventricular] dysfunction.” “The cardiac cath did not reveal any coronary disease.” “He has asymptomatic mild cardiomyopathy which will respond to Accupril [an angiotensin converting enzyme inhibitor].”

Since the office note did not allay the suspicion, the other cardiologist’s catheterization report was requested and revealed “Preop Diagnosis: High grade ventricular ectopy with severely abnormal thallium scan.” Left ventricular enlargement, significant hypokinesia including moderately severe basilar wall hypokinesia, distal anterior wall hypokinesia, and severe inferior wall hypokinesis, were found. Ejection fraction was 48%, measured in a post-PVC beat. Coronary arteries were normal.

DISCUSSION

In essence, this applicant knew of no adverse medical history but had asymptomatic ventricular bigeminy. While his doctor tried to downplay the obvious risk of a cardiomyopathy that was discovered, we found that the proposed insured had a significant cardiomyopathy of unknown cause, and his case
was declined. What is the significance and risk of frequent PVCs?

PVCs are common. About 80% of the normal population will have some PVCs found on 24-hour monitoring. The frequency of PVCs increases with age and in some benign conditions such as in mitral valve prolapse. Mortality risk of PVCs in asymptomatic healthy patients is controversial. However, 3 main cardiac diseases are also responsible for frequency, complexity, and lethality of PVCs. These are left ventricular hypertrophy (LVH), coronary artery disease (CAD), and cardiomyopathy. We will explore the significance of PVCs in normal subjects, LVH, CAD, and left ventricular dysfunction.

**PREMATURE VENTRICULAR CONTRACTIONS IN APPARENTLY NORMAL SUBJECTS**

A study by Kennedy followed normal patients with frequent or complex ventricular ectopy for 10 years, showing a less-than-expected death rate in a group of normal subjects with the ectopy. The caveats here, though, were that the subjects were extensively screened for heart disease, the sample size was small for a mortality study, and the mortality comparison was in the “healthy United States population.”

A part of the Framingham study followed 6000 men and women for an average of 5 years, screened with a 1-hour monitoring session. Groups were also divided by gender and whether clinically evident heart disease was present. Twelve percent of normal men had frequent or complex PVCs, compared with 33% in men with CAD. This compares with 12% in normal women and 26% in women with CAD. The study indicated there was a twofold increase in mortality in men with asymptomatic frequent or complex PVCs and no evident coronary disease, while the presence of frequent or complex PVCs did not increase risk of death when CAD was already evident. Women did not seem to have an adverse effect from PVCs in this study compared with expected mortality. Frequent or complex PVCs, in this study, were associated with increased deaths and served as a marker for coronary artery disease.

**PREMATURE VENTRICULAR CONTRACTIONS IN LVH**

The frequency and complexity of PVCs in LVH are correlated to left ventricular mass. Left ventricular hypertrophy is associated with increased ventricular ectopy and an increase in sudden death.

Patients with hypertension with LVH have more frequent PVCs and more complexity of PVCs. There is also a high incidence of nonsustained ventricular tachycardia in hypertensives who show LVH on EKG. Twenty-eight percent with LVH on EKG had nonsustained ventricular tachycardia versus 8% with hypertension and no LVH and 2% of normal controls. The ventricular tachycardia was especially associated with LVH and ST-T wave changes on EKG.

It is difficult to correlate the impact of PVCs in the mortality of LVH due to the confounding presence of hypertension and coronary artery disease in these patients. It stands to reason, however, that an increase in electrical instability (frequency of PVCs) would contribute to the doubling of mortality from sudden death seen in LVH.

**PREMATURE VENTRICULAR CONTRACTIONS IN CORONARY ARTERY DISEASE**

PVCs are more prevalent and complex in patients with coronary artery disease (CAD). PVCs in coronary artery disease are related to amount of coronary obstructions, bouts of ischemia, previous myocardial infarction (MI), and level of left ventricular dysfunction. In a study of 124 patients undergoing cardiac catheterization, persistence of ectopy was 3 times more likely in patients with CAD, and complexity of ventricular ectopy was correlated to number of vessels involved. Single-vessel CAD had no difference in prevalence or complexity of PVCs compared with normal subjects.
PREMATURE VENTRICULAR CONTRACTIONS IN CARDIOMYOPATHY

Over 80% of patients with cardiomyopathy produce frequent and complex ventricular arrhythmias. The frequency of PVCs appears to be related to the ventricular size rather than function, suggesting myocardial stretch as a cause. It is uncertain what separate contribution ventricular arrhythmias make in the mortality of congestive heart failure. In the case presented above, the frequent PVCs (bigeminy) reflected a dilated cardiomyopathy and served as a marker for the disease. The PVCs may not be a direct threat to mortality but were able to index the case as a hazard due to cardiomyopathy.

Mortality in LV dysfunction is related to the complexity of PVCs and extent of LV dysfunction. Gradman's study confirmed that reduced ejection fractions (below 40%) are especially risky, with a large percentage (50%) of deaths being sudden and related to PVC frequency, ventricular tachycardia frequency, and lowest ejection fractions. Holmes reported on ambulatory testing of patients with LV dysfunction in an attempt to stratify mortality risk by level of ventricular arrhythmias. Peak complexity of ventricular arrhythmias was indicative of high mortality not explainable by left ventricular filling pressures alone.

Due to proarrhythmias produced by antiarrhythmic drugs, treatment of asymptomatic ventricular arrhythmias has been abandoned except for use of beta blockers post-myocardial infarction and, in some cases, of congestive heart failure. The mainstay of treatment, not known to reduce arrhythmic deaths independent of benefit from improved left ventricular function, is to improve LV function with afterload reduction. To this end, angiotensin converting enzyme inhibitors and beta blockers have reduced deaths in congestive heart failure.

UNDERWRITING IMPLICATIONS

In this case, PVCs were serving as a marker for cardiomyopathy. The important factors here were the unexpected frequency of PVCs and the large amount of insurance.

CONCLUSIONS

While, by far, most PVCs and even episodes of bigeminy are benign in younger individuals, occasionally one will get lucky and discover a case like this one that unnerves us a bit. When evaluating risk in PVCs, look at the underlying EKG findings and the risk of CAD in the applicant. The EKG may show evidence of prior MI or of ischemia, and the CAD risk profile can give you an idea of pretest probability of CAD. The presence of frequent or complex PVCs may serve as a marker in dilated cardiomyopathies. Your level of risk ultimately may play a role in accepting “benign” PVCs with or without further medical evaluation.

REFERENCES
