A 18-year-old white female with a body mass index of 28 kg/m² presented with a 6-month history of intermittent esophageal dysphagia, dizziness, and heavy vomiting associated with frequent premature ventricular complexes (PVCs). Her echocardiogram was normal, and serial 24-hour ambulatory Holter monitoring documented between 100 and 5000 monomorphic PVCs. A 12-lead ECG obtained during an emergency department visit revealed monomorphic single PVC, as well as pairs and triplets with inferior axis and left bundle-branch block pattern. Upper endoscopy and chest x-ray did not reveal any abnormalities. Videofluoroscopic modified barium swallow study with simultaneous ECG monitoring was performed with the patient in a supine position in the catheterization laboratory. The patient was asked to swallow 15 mL of barium, and subsequently fluoroscopy was performed twice, first during sinus rhythm and then during frequent PVC. During sinus rhythm, no symptoms of dysphagia and no abnormalities in barium passage through the esophagus were documented (Movie I). During frequent PVC, however, the patient reported difficulty swallowing and intraesophageal reflux, and prolonged barium deposits in the upper esophagus were documented (Movie II). In esophageal manometry during sinus rhythm, no significant abnormalities were found; however, during frequent PVC, esophageal spasm and abnormal motility were documented. Motility disorders (“corkscrew and nutcracker esophagus”) were excluded with manometry and follow-up. After ineffective treatment with metoprolol, propafenone, and verapamil, the patient was referred for radiofrequency (RF) ablation. Coronary angiography excluded coronary artery abnormalities. Intracardiac mapping catheterization documented the earliest ventricular activation in the left aortic cusp. Local potentials preceded the QRS complex during PVC by 35 ms. Two RF applications were delivered, and after the second application, the arrhythmia disappeared and was no longer inducible by pacing applications were delivered, and after the second application, the arrhythmia disappeared and was no longer inducible by pacing applications were delivered, and after the second application, the arrhythmia disappeared and was no longer inducible by pacing applications were delivered, and after the second application, the arrhythmia disappeared and was no longer inducible by pacing applications were delivered, and after the second application, the arrhythmia disappeared and was no longer inducible by pacing applications were delivered, and after the second application, the arrhythmia disappeared and was no longer inducible by pacing applications were delivered, and after the second application, the arrhythmia disappeared and was no longer inducible by pacing applications were delivered, and after the second application, the arrhythmia disappeared and was no longer inducible by pacing. Serial Holter monitoring showed no PVCs, and control esophageal manometry did not reveal any abnormalities.

The present case report illustrates a simple technique for the evaluation of a new cause of dysphagia, so-called “PVC-associated dysphagia.” This syndrome has been recently found in ~5% of patients with frequent PVCs without organic heart disease. Although it has been known for many years that esophageal disorders can cause brady- and tachyarrhythmias, it has not been previously reported that frequent PVCs can also cause dysphagia. We suspect that a cardioesophageal reflex arc could be the mechanism responsible for PVC-associated dysphagia. It would be valuable to study patients with symptoms of arrhythmia and unexplained or functional dysphagia by simultaneous evaluation with esophageal motility and ECG monitoring during sinus rhythm and arrhythmia. Furthermore, this course of study could help electrophysiologists find a reversible cause of dysphagia and an effective treatment.

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**Disclosures**

None.

**References**


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