Incidence of the coexistence of left ventricular false tendons and premature ventricular contractions in apparently healthy subjects

MICHIHIRO SUWA, M.D., YUZO HIROTA, M.D., HIKARU NAGAO, M.D., MASAYA KINO, M.D., AND KEISHIRO KAWAMURA, M.D.

ABSTRACT  The incidence of the coexistence of left ventricular false tendons and premature ventricular contractions (PVCs) was evaluated prospectively. Over 14 months, left ventricular false tendons were found in 71 (6.4%) of 1117 consecutive patients examined echocardiographically. Two types of false tendons were observed: longitudinal, from the ventricular septum to the posteroapical wall (n = 62), and transverse, between the septum and the lateral wall (n = 9). Among 62 patients with PVCs and no underlying heart disease, false tendons were detected in 35 (56%); 28 had unifocal and seven had bifocal PVCs. Episodes of ventricular tachycardia were documented in one of the 28 patients with unifocal PVCs and in one of the seven patients with bifocal PVCs. These PVCs were poorly controlled by antiarrhythmic drugs but easily suppressed by exercise. Left ventricular false tendons were detected in 36 patients on routine echocardiographic examinations performed in the other 1055 subjects, and 10 of these patients were judged to have no underlying heart disease. PVCs were detected in two (20%) of these 10 patients. Although a definite conclusion that left ventricular false tendons are arrhythmogenic cannot be derived from these results, the unexpectedly high incidence of the coexistence suggests that left ventricular false tendons may be an etiologic factor in the development of PVCs, especially the rate-dependent and medically uncontrollable PVCs seen in apparently healthy individuals.


The present study was performed prospectively to evaluate the incidence of the coexistence of false tendons and PVCs in apparently healthy individuals who visited our cardiovascular clinic over a 14 month period.

Methods

Study protocols. We designed two study protocols to evaluate the incidence of the coexistence of left ventricular false tendons and PVCs. In the first study, patients with PVCs on routine electrocardiograms (ECGs) or continuous 24 hr ambulatory ECG monitoring but without significant heart disease were sent to the echocardiographic laboratory to be evaluated for left ventricular false tendon. In the second study, patients without organic heart disease and with left ventricular false tendons found on routine echocardiographic examination were evaluated by ambulatory ECG monitoring.

Selection of patients. From November 1982 until January 1984, echocardiographic examinations were performed in 1117 consecutive patients. Sixty-two patients underwent echocardiographic examinations in a search for left ventricular false tendons, since they had PVCs without apparent organic heart diseases. Routine echocardiographic examinations were performed in the other 1055 patients for diagnostic purposes. The presence of organic heart disease was excluded by physical examination, routine ECG, chest x-ray, and exercise stress tests in addition to echocardiographic examination. Special attention was paid to exclude patients with mitral valve prolapse. Diag-
Results

Overall prevalence of left ventricular false tendons. In the series of 1117 consecutive patients, left ventricular false tendons were seen in 71 (6.4%). The tendons were of two types: longitudinal (or diagonal) in the left ventricular cavity in 62 patients (87%) and transverse from the ventricular septum to the free wall in nine (13%). The longitudinal type could be divided into two subtypes morphologically: one with the ends of the tendon attached to the base of the septum and the posterior wall near the apex (figure 3) (this can be called the long longitudinal type, which was seen in 33 patients), and the other with the ends attached to the lower portion of the septum and the posterior wall (figure 4) (this can be called the short longitudinal type or diagonal type, which was seen in 24 patients). Coexistence of these two longitudinal types was seen in five patients. The transverse tendons are situated horizontally from the midportion of the ventricular septum to the lateral wall (figures 2 and 5). There were, however, some false tendons of the longitudinal type that could be interpreted as transverse tendons on the transverse section of the two-dimensional echocardiogram.

Prevalence of false tendons in subjects with PVCs. The incidence of the coexistence of left ventricular false tendons and PVCs is shown in figure 6. Among the 62 patients with PVCs, false tendons were detected in 35 (56%). The age range of these 35 patients (20 male and 15 female) was 12 to 70 years (mean 39). The frequency of PVCs was 10 beats to 30,300 beats (mean 8544 beats) per 24 hr. The focus of PVCs was single (unifocal PVCs) in 28 patients, seven with a right bundle branch block pattern (suggesting left ventricular origin) and 21 with a left bundle branch block pattern (suggesting right ventricular origin) in the precordial leads of the surface ECG. Seven patients had two foci (bifocal PVCs) on standard ECG or ambulatory ECG monitoring. Bifocal PVCs seemed to originate from both right and left ventricles in six patients and from two foci in the left ventricle in one. Ventricular tachycardia was detected in two of the 35 patients, and in both it seemed to originate in the left ventricle. One of them had bifocal PVCs originating in the left ventricle, and two types of ventricular tachycardias were documented corresponding to these PVCs. These two patients had long longitudinal false tendons.

Comparison of PVCs in subjects with and without false tendons. The age range of the 27 patients (nine male and 18 female) with PVCs and without false tendons was 13 to 58 years (mean 43). The frequency of their PVCs averaged 12,410 beats per 24 hr. The focus in all cases was single; four patients showed a right bundle branch block pattern and 23 a left bundle branch block pattern in the precordial leads of the surface ECG. Treadmill exercise tests were performed by 30 patients with left ventricular false tendons and 13 patients without false tendons. PVCs disappeared at heart rates of about 90 to 150 beats/min in both groups and were more easily suppressed at lower heart rates during exercise in patients with false tendons than in those without false tendons (112 ± 21 vs 147 ± 11/min; p < .001). There were no episodes of chest pain or isch-
FIGURE 2. M mode and two-dimensional echocardiograms of the left ventricular false tendon in the patient described in figure 1. Two-dimensional echocardiograms of the left ventricle from the parasternal short-axis (upper right) and apical four-chamber views (lower right). A false tendon (white arrows) is seen traversing the center of the left ventricle between the ventricular septum and the lateral free wall, pulling the ventricular septum inward in diastole to make a figure eight-shaped cavity. The tendon appears in the center of the cavity in the M mode tracing (left, black arrow) and is immobile during the cardiac cycle. This case suggested that false tendons might be a cause of PVCs. IVS = interventricular septum; RV = right ventricle; LV = left ventricle.

FIGURE 3. M mode and two-dimensional echocardiograms of a long, longitudinal left ventricular false tendon. Two-dimensional echocardiograms of the left ventricle from the apical long-axis (upper right) and apical four-chamber views (lower right). One end of the tendon (white arrows) is attached to the base of the ventricular septum and the other to the posteroapical wall of the ventricle. On the M mode echocardiogram (left) the tendon (black arrows) moves with the ventricular septum. AO = aorta; LA = left atrium; RA = right atrium.
These two patients had false tendons of the longitudinal type.

Discussion

Since about a century ago, false tendons in the left ventricular cavity have been noted as anatomic variants at autopsy. Now they can be detected easily by two-dimensional echocardiography. It has frequently been emphasized that caution must be exercised in the identification of the left ventricular endocardium of the ventricular septum and in the differential diagnosis of mural thrombus, since the echo from the tendon might mimic these structures. Moreover, they have been reported to be a cause of functional ejection murmurs. There are no descriptions, however, of other clinically significant features of false tendons. We examined a young man who was referred to us because of frequent PVCs and who had no cardiac abnormalities.

FIGURE 4. Two-dimensional echocardiograms of a short longitudinal left ventricular false tendon. Top, Apical long-axis view; bottom, apical four-chamber view. The false tendon (white arrows) is attached to the lower part of the ventricular septum and the posteroapical wall of the cavity. Abbreviations as in figures 2 and 3.

em ST-T changes during the exercise stress tests. PVCs could be more easily controlled by medical treatment in patients without false tendons than in those with false tendons.

Prevalence of PVCs in subjects with false tendons. Left ventricular false tendons were detected in 36 patients in 1055 routine echocardiographic examinations. Organic heart disease was ruled out by noninvasive diagnostic techniques in 10 patients. PVCs were detected in two of the 10 patients (20%) on repeat ECGs or ambulatory ECG monitoring. The frequency of PVCs was 10 beats per 24 hr in one, and the other had bifocal PVCs (107 beats per 24 hr) on ambulatory ECG monitoring. These two patients had false tendons of the longitudinal type.

FIGURE 5. A left ventricular transverse false tendon (white arrow) is observed in the cavity on parasternal long-axis (top) and short-axis views (bottom) of two-dimensional echocardiograms. On the short-axis view the deformity of the interventricular septum is apparent at the site of attachment of the false tendon, which gives the cavity a figure eight appearance.
except false tendons on cardiac examination, including coronary cineangiography (figure 1). The transverse section of the left ventricle had a figure eight appearance during late diastole because of the tension of the tendon (figure 2), so we thought this mechanical force might be the cause of PVCs. Therefore this study was performed prospectively to determine whether left ventricular false tendons might be a cause of PVCs.

The frequency of left ventricular false tendons has already been reported by some investigators using two-dimensional echocardiography, and the incidence varies from 0.5% to 46%. Because these studies were performed in selected groups of patients, the true incidence of false tendons in the general population is not known. In our laboratory, the incidence of false tendons was 6.4%, but our observations were limited to those patients who were referred to the echocardiographic laboratory and this incidence does not reflect that in the general population.

Relationship between PVCs and false tendons. Among 62 patients with PVCs without organic heart diseases, left ventricular false tendons were detected in 35 (56%); PVCs were detected in two of 10 patients (20%) with left ventricular false tendons without organic heart disease. Recently, Perry et al. described three cases of PVCs and no significant heart disease among 31 pediatric patients with left ventricular false tendons.

PVCs originated from a single focus in 29 and from two foci in eight of the 37 patients with PVCs and left ventricular false tendons. Sustained ventricular tachycardia was detected in two patients. There was no definite association between PVCs and the types or subtypes of false tendons. Unifocal PVCs were seen in 23 patients with longitudinal and six with transverse false tendons, and bifocal PVCs were seen in eight with longitudinal and none with transverse false tendons. Both patients with ventricular tachycardia had longitudinal false tendons.

Mechanisms of PVCs in subjects with false tendons. Two hypotheses can be considered in the study of possible mechanisms of the arrhythmogenesis of false tendons. Tendons of the canine right ventricle have frequently been used in electrophysiologic studies of the conduction system, since they contain Purkinje fibers. It is well known that the automaticity of Purkinje cells is increased by mechanical stretching. Furthermore, histologic examinations have shown that human left ventricular false tendons contain specific conduction cells. These might become an automatic focus of PVCs. Another possibility is that the mechanical stretch of the left ventricular wall where the tendon is attached triggers PVCs. A figure eight deformity of the ventricular cavity during late diastole caused by tension due to the false tendon was frequently observed (figures 2 and 5), especially with the transverse type of tendon. The disappearance of PVCs on exercise might be related to reduction of tension due to decreased left ventricular dimension with tachycardia. Recent studies with endocardial mapping techniques have shown that
PVCs with the left bundle branch block pattern can originate from either the right or left ventricle. In 23 of 27 patients with PVCs with a left bundle branch block pattern, the PVCs had upright QRS complexes in the precordial leads, suggesting that these PVCs might have originated from the ventricular septum. Some patients had two foci corresponding to two adhesions in the endocardium, suggesting that the mechanical force or tension on the left ventricular wall adjacent to the tendon might have caused the PVCs.

A definite conclusion that false tendons in the left ventricular cavity are a cause of PVCs cannot be derived from our results. Further investigations, such as histologic examinations of the false tendons, electrophysiologic studies of the arrhythmias, surgical intervention, and surveys of PVCs, false tendons, and their coexistence in the general population, are necessary to establish the correlation between them. However, the surprisingly high incidence of this coexistence (35/62 false tendons in patients with PVCs and 2/10 PVCs in patients with false tendons) and the different responses to exercise and medical treatment of PVCs with false tendons and those without false tendons strongly suggest that left ventricular false tendons are an etiologic factor in the genesis of ventricular arrhythmias in apparently healthy subjects.

References

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