Interesting Electrocardiogram

Pre-excitation and Cardiomyopathy (with a note on Myocardial Bridges)

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These tracings were among 22 electrocardiograms submitted on a 63-year old woman applying for life insurance. They were sent to us by Dr. W. S. Clough of the Southeastern Head Office. This woman, who has had many investigations, including a workup at the Cleveland Clinic, began having chest pains in 1969 relieved by nitroglycerin. She was hospitalized in 1970 for recurring chest pains. In 1973 she developed “cardiac insufficiency.” In 1975 she had tachycardia and was reported to have left ventricular failure. At the time of application for insurance in 1983 she was better, taking diuril, lanoxin and inderal, and had no chest pain. A current chest x-ray was said to show no cardiomegaly or congestive lung changes.

The first electrocardiogram taken at the onset of her symptoms (9/24/69) shows pre-excitation of the W-P-W type. Note the short PR (0.08 sec.), long QRS (0.12 sec.) and delta waves best seen in leads I, aVL and V6. In the presence of ventricular pre-excitation the ST-T abnormalities have no primary meaning. Following this tracing, two more (8/1/72 and 4/14/83) fail to show any form of pre-excitation. Since there is not W-P-W pattern present (which distorts the record) the ST-T abnormalities in leads II, III, aVF and V3-V6 emerge as 8/1/72 (on no digitalis) differs very little from that of 4/14/83 taken eleven years later. Her workup revealed the diagnosis of idiopathic cardiomyopathy.

The disappearance from the electrocardiogram of all signs of pre-excitation in the second and third electrocardiograms is noteworthy. At least 50% of patients with W-P-W form of accelerated AV conduction have intermittency,—i.e., show the pattern only occasionally. However, this patient never had it again and it is tempting to assume that the pathologic process of her myopathy obliterated the bypass tract. This permitted normal AV conduction and the discovery of her abnormal ST-T waves.

Well over 80% of cases of pre-excitation of W-P-W form have no associated heart disease. When pre-excitation is seen in coronary or hypertensive disease (neither being present here) it is an incidental and unrelated finding. There are, however, certain congenital lesions seen in patients with pre-excitation. These include Ebstein’s anomaly, tricuspid and mitral atresia, transposition of great arteries, ventricular septal defects, patent ductus arteriosus, Tetralogy of Fallot, atrial septal defects, pulmonic stenosis, dextrocardia, aortic valve atresia, coarctation of the aorta and cardiomyopathies.

It would appear, therefore, that this woman had two congenital abnormalities—pre-excitation and cardiomyopathy. Of course, the latter explained her symptoms.

One is also tempted to invoke an additional problem here. It has recently been shown1 that patients with obstructive hypertrophic myopathy have a high prevalence of significant myocardial bridges. These act to squeeze the coronary arteries in systole and may produce ischemia. They can even completely occlude a vessel temporarily. Angiographic studies have identified this mechanism. Perhaps it was operative in this woman and could have explained the ST-T abnormalities.

Reference

Tracing 3
(4/14/83)