Post-paroxysmal Tachycardia Syndrome

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Inversion of the T wave of the electrocardiogram may persist for a long time after an episode of paroxysmal tachycardia in the absence of detectable heart disease. Mattingly (1959) has stressed the existence of "spurious heart disease" as a result of erroneous diagnosis. This may lead to unnecessary restriction of physical activity and diet, expensive diagnostic procedures and therapy, and loss of time from work. The false diagnosis may result from misinterpretation of ST segment and T wave changes in the electrocardiogram. ST segment and T wave changes may be caused by digitalis, quinidine, hyperventilation, drinking ice-water, electrolyte imbalance, and exercise. Likoff, Segal, and Dreifus (1962) reported 6 asymptomatic men, aged 16 to 24 years, with low intensity systolic murmurs but pronounced multiple T wave inversion suggestive of myocardial ischaemia in the presence of normal coronary arteriograms. Levine and White (1962) described prolonged QT interval and multiple T wave inversion in a patient with subarachnoid haemorrhage and without abnormal cardiac findings at necropsy.

In the case reported here several episodes of supraventricular paroxysmal tachycardia were followed by prolonged T wave inversion without clinical or laboratory evidence of cardiac disease.

Case Report

A 21-year-old man was admitted on December 16, 1967, with an attack of rapid heart action. He had a history of four similar episodes during the previous four years. His most recent attack had started suddenly, with a tight sensation across the upper abdomen. He was not conscious of his heart's action and did not complain of dyspnoea.

On admission he had a regular tachycardia of 170 a minute. The blood pressure was 120/70 mm. Hg and no cardiac murmurs were audible. The jugular venous pressure was not raised. The electrocardiogram (Fig.

1a) showed a regular ventricular rate of 170 a minute and P waves with a PR interval of 0.16 sec. The ventricular complexes had the configuration of complete right bundle-branch block. The findings were indicative of paroxysmal atrial tachycardia with aberration of ventricular conduction.

The arrhythmia failed to revert with direct current countershock. He was treated with lanatoside C 0.25 mg. 6-hourly for 24 hours. The tachycardia persisted for 24 hours and then reverted to regular sinus rhythm at a rate of 54 a minute. After reversion to sinus rhythm the electrocardiogram (Fig. 1b) showed conspicuous T wave inversion in leads II, III, aVF, V3 to V6, and associated ST segment depression of 3 mm. The T wave inversion was maximal in lead V3 with an amplitude of 13 mm. There were no abnormal Q waves. The serum enzymes (serum aspartate aminotransferase and lactic acid dehydrogenase) were normal. Chest x-ray showed a normal cardiac silhouette. The serum cholesterol was 152 mg./100 ml. Gradual regression of the T wave abnormality occurred. Ten days later T wave inversion had diminished to 2 mm. in lead V4 and the ST segment was isoelectric (Fig. 2A). The electrocardiogram was normal 19 days after admission (Fig. 2B).

Episodes of paroxysmal tachycardia had been previously recorded in May 1964, August 1966, January 1967, and May 1967. On each of these occasions the electrocardiogram during the episode showed supraventricular tachycardia and ventricular complexes of right bundle-branch block configuration. Each attack of tachycardia was followed by T-wave inversion and ST segment depression in the same leads as in the episode of December 1967.

The patient was investigated in the cardiovascular unit of the Belfast City Hospital 4 days after the episode in August 1966, when conspicuous T wave abnormality was present. Right heart catheterization and pulmonary angiography were performed. Pulmonary artery, right ventricular, and right atrial pressures were normal. The angiogram revealed no evidence of any anomalous coronary artery. There was no evidence of cardiomyopathy, coronary artery disease, or other cardiac abnormality.

There has been no recurrence since his last episode in December, 1967, and he leads a normal active life.

FIG. 1.—The electrocardiogram, (A) during the paroxysm of tachycardia, and (B) immediately after reversion to sinus rhythm two days later.

Comment

Persistence of T wave inversion for days or weeks after paroxysmal tachycardia may lead to an erroneous diagnosis of myocardial infarction. The true diagnosis of post-tachycardia syndrome may be established by the absence of symptoms during the period of electrocardiographic abnormality, normal blood enzyme levels, absence of pathological Q waves, multiple T wave inversions without circumscribed anatomical localization, and recurrence of the T wave inversion in identical character and form each time the patient has a paroxysm of tachycardia.

Graybiel and White (1935) stressed the need for caution in the diagnosis of serious heart disease when T wave inversion is the only abnormal finding. They also emphasized the importance of cor-

relation of electrocardiographic findings with the patient's history and physical examination. In this connexion they cited two cases of paroxysmal *ventricular* tachycardia in young robust adults followed by T wave inversion in leads I, II, and III and with gradual return to a normal cardiogram; there was no sign of organic disease. Geiger (1943) later reported electrocardiograms simulating those of coronary thrombosis after cessation of paroxysmal *supraventricular* tachycardia of 48 hours' duration; electrocardiographic pattern was characterized by prominent T wave inversion in leads I, II, and III which persisted for 35 days, depression of ST segments in the leads with inverted T waves, and absence of abnormal Q waves. Smith (1946) reported a further example of the cardiographic

FIG. 2.—The electrocardiogram, (A) 10 days after, and (B) 17 days after tachycardia had ceased.

syndrome occurring after *ventricular* tachycardia, characterized by broad-base T wave inversion in one or more leads, with depression of ST segment in the leads with the most prominent T wave inversion. He believed this to be the nineteenth case reported and noted that only two cases of electrocardiographic abnormality occurring after *supraventricular* tachycardia had been described. Further cases of prolonged benign T wave inversion after paroxysmal ventricular tachycardia were described by Rakov in 1964, and after supraventricular tachycardia by Sargin and Demirkol in 1965.

The mechanism of production of the post-tachycardia syndrome is obscure. It is important, however, in order to avoid an erroneous diagnosis of coronary thrombosis, to recognize that the electrocardiographic changes which may follow an episode

of paroxysmal tachycardia may be benign. It is not known how frequently this syndrome follows paroxysmal atrial tachycardia. Though paroxysmal atrial tachycardia is much more common than paroxysmal ventricular tachycardia, most reported cases of post-tachycardia syndrome have occurred after paroxysmal ventricular tachycardia.

Summary

A case of recurrent supraventricular paroxysmal tachycardia in a 21-year-old man is described. Prolonged, deeply inverted T waves occurred after each paroxysm, with spontaneous reversion to a normal electrocardiogram. There was no evidence of organic heart disease.

Misinterpretation of the electrocardiogram after

paroxysmal tachycardia may lead to spurious heart disease and unnecessary invalidism.

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