Q wave T wave vector discordance in hypertrophic cardiomyopathy: septal hypertrophy and strain pattern

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SUMMARY Hypertrophic cardiomyopathy is a common cause of prominent non-infarctional Q waves. A retrospective analysis of previously published cases confirmed a characteristic Q wave T wave vector discordance in hypertrophic cardiomyopathy. In 41 of 44 cases with predominant Q waves (as part of QS or Qr complexes where Q wave amplitude exceeded R wave height), the T wave was positive, and in all cases with QS type complexes the T wave was positive. This characteristic electrocardiographic sign probably represents a pattern of septal hypertrophy and strain (Q waves with positive T waves and ST segment elevation) which is the inverse of the classical pattern of left ventricular hypertrophy and strain (tall R waves with inverted T waves and ST segment depression).

Hypertrophic cardiomyopathy, including the classic syndrome of idiopathic hypertrophic subaortic stenosis, is an important cause of pseudoinfarct electrocardiographic patterns (Estes et al., 1963; Braudo et al., 1964). Q waves simulating myocardial infarction may appear in multiple leads, mimicking anteroseptal, anterolateral, and/or inferior wall injury. In addition, tall R waves in the right chest leads may lead to the mistaken diagnosis of true posterior myocardial infarction (Goldberger, 1975). In Frank and Braunwald’s (1968) series of patients with classic idiopathic hypertrophic subaortic stenosis, pseudoinfarct Q waves were noted in 56 per cent of 123 cases.

Although the high prevalence of non-infarct Q waves in hypertrophic cardiomyopathy is widely known, criteria for distinguishing these patterns from actual infarction have received only limited attention. The morphology of the Q waves may be of some diagnostic help. The Q waves of hypertrophic cardiomyopathy may show a relatively distinctive, lance-like appearance, with a narrow, deep, and sharply inscribed contour. In other cases, the Q waves have a jagged, W-shaped morphology (Fig. 1). While such Q wave patterns may be suggestive of hypertrophic cardiomyopathy, particularly in younger patients, these patterns are by no means diagnostic. Deep, narrow Q waves are commonly seen with Duchenne muscular dystrophy (Perloff et al., 1966) and bizarre QS complexes may occur with myocardial infarction or other types of cardiomyopathy. Furthermore, the abnormal initial depolarisation forces seen in the vectorcardiogram in cases of hypertrophic cardiomyopathy are generally indistinguishable from true infarct patterns (Estes et al., 1963; Stein et al., 1968).

In certain cases, the polarity of the T wave may be of additional help in the differential diagnosis of hypertrophic cardiomyopathy vs. myocardial infarction. It has been observed that the T wave vector in hypertrophic cardiomyopathy is characteristically oriented away from the direction of the Q wave vector (Goldberger, 1975). As a result, leads showing predominant Q waves, as part of QS or Qr type complexes, will show positive T waves. In contrast, infarct Q waves are often associated with negative T waves, reflecting primary ischaemic repolarisation abnormalities. The purpose of this study was to investigate further the validity of the discordant Q wave T wave sign in hypertrophic cardiomyopathy and to discuss the concept of a ‘septal hypertrophy and strain’ pattern.

Methods

The electrocardiograms of patients with hypertrophic cardiomyopathy documented primarily by catheterisation were reviewed in a retrospective fashion. Forty-four cases of hypertrophic cardiomyopathy with deep pseudoinfarct Q waves were

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found among the published reports\(^1\). These electrocardiograms were evaluated specifically to test the hypothesis that the T waves would be positive in leads showing predominant Q waves, as part of QS or Qr type complexes. Q wave patterns meeting these criteria in one or more of leads I, II, aVL, aVF, V2 to V6 were studied.

Results

In 41 of 44 cases (93\%), all leads showing predominant Q waves (QS or Qr complexes) also had positive T waves. Furthermore, all leads showing QS or W shaped complexes had positive T waves. Two partial exceptions to this Q wave T wave discordance pattern were noted. In 1 case reported by Leachman and Leatherman (1965, Fig. 2), the electrocardiogram showed an atypical intraventricular conduction delay with QS waves in leads I and II, and Qr waves in leads V5 to V6, all associated with positive T waves. However, lead aVL showed a Qr complex with slight T wave inversion. In another case (Morgan and Forker, 1972, case 10), an incomplete right bundle-branch block was present, and not all of the leads with Qr type complexes showed positive T waves. Finally, Flowers and Horan (1973) also reported a case with prominent Q waves (as part of QR complexes) and inverted T waves.

It should be emphasised that this discordant Q wave T wave sign applies only in leads showing predominant Q waves (QS or Qr patterns where the Q wave exceeds the R wave amplitude). In cases where QR complexes are present, with the R wave equalling or exceeding the Q wave, the T wave may be upright or inverted. Fig. 1 shows a typical example of Q wave T wave discordance in leads with predominant Q waves.

Discussion

The Q waves in hypertrophic cardiomyopathy probably represent augmented depolarisation forces from the hypertrophied septum (Braudo et al., 1964). Normally, the left-to-right direction of septal forces

\[ V_5 \]

LVH/STRAIN

\[ V_5 \]

SEPTAL HYPERTROPHY/STRAIN

Fig. 2 Classical left ventricular hypertrophy and strain pattern (ST segment depression with inverted T waves in leads with prominent R waves) is the inverse of septal hypertrophy and strain pattern (positive T waves with slight ST segment elevation in leads with predominant Q waves, for example V5).

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\(^1\) Hollman et al., 1960 (1); Benchimol et al., 1963 (1); Estes et al., 1963 (1); Prescott et al., 1963 (3); Braun et al., 1964 (6); Cohen et al., 1964 (1); Welch and Scott, 1965 (1); Coyne, 1968 (3); Frank and Braunwald, 1968 (3); Klein et al., 1968 (3); Leachman and Leatherman, 1968 (3); Peter et al., 1968 (2); Stein et al., 1968 (1); Bahl et al., 1970 (1); Marriott, 1972 (1); Morgan and Forker, 1972 (2); Flowers and Horan, 1973 (1); Chung, 1974 (2); Goldberger, 1975 (5); Hansen et al., 1975 (1); Witham, 1975 (1); Friedman, 1977 (1).
depolarisation produces small positive (r) waves in the right chest leads, and small septal q waves in the left chest leads and one or more of the limb leads. Septal hypertrophy may magnify these q waves, resulting in inferior and/or anterior pseudo-infarct Q wave patterns. In some cases of hypertrophic cardiomyopathy, QS waves may appear in the right praecordial leads (V1 to V3), simulating anteroseptal myocardial infarction. It is not certain whether the Q waves in these leads reflect posteriorly directed septal forces (Wigle and Baron, 1966) or whether they are the result of concomitant hypertrophy of the left ventricular free wall. Coyne (1968) suggested that the abnormal depolarisation forces in hypertrophic cardiomyopathy might also result from an alteration in the normal sequence of ventricular activation.

The characteristic Q wave T wave discordance in hypertrophic cardiomyopathy can be explained by analogy with the QRS-T pattern seen with classical left ventricular hypertrophy. The electrocardiographic pattern of concentric or free wall left ventricular hypertrophy generally shows discordant QRS and T wave vectors: leads with tall R waves are associated with T wave inversions, as part of the left ventricular hypertrophy and 'strain' pattern (Fig. 2). It is not certain whether these 'strain' T waves reflect primary left ventricular ischaemia or whether they are secondary to an alteration in the repolarisation sequence of hypertrophied muscle.

In cases of predominant septal hypertrophy, an analogous septal 'strain' pattern might occur. However, since septal depolarisation is oriented to the right, the septal hypertrophy and strain pattern (deep Q waves and positive T waves) seen in the lateral chest leads will be the inverse of the classical free wall left ventricular hypertrophy and strain pattern (tall R waves with T wave inversions) (Fig. 2). In addition, these deep septal Q waves are often followed by ST segment elevation, reciprocal to the ST segment depression typically seen with left ventricular hypertrophy and strain.

With myocardial infarction, leads showing prominent Q waves may also have positive T waves. However, in many cases, distinctive ischaemic T wave inversions will appear. The presence of predominant Q waves (as part of QS or Qr type complexes) with negative T waves makes the diagnosis of pure hypertrophic cardiomyopathy unlikely. On the other hand, the presence of deep Q waves (particularly with a lance or W shape) with positive T waves is compatible with, but not diagnostic of, hypertrophic cardiomyopathy.

In some cases, hypertrophic cardiomyopathy and coronary artery disease may co-exist. However, the effect of transmural infarction on the pseudo-infarct electrocardiographic patterns of hypertrophic cardiomyopathy has not been specifically described. In cases of hypertrophic cardiomyopathy with deep Q waves, acute myocardial infarction might have different effects, depending on the location of the infarct. For example, septal infarction might lead to a paradoxical diminution in Q wave magnitude, since the Q waves reflect septal enlargement. Lateral infarction, on the other hand, should augment these septal Q waves, because of loss of counterbalancing free wall depolarisation forces. Acute myocardial infarction superimposed on the pattern of hypertrophic cardiomyopathy might also produce diagnostic ischaemic repolarisation changes.

Discordance of the Q wave T wave vector may thus be a helpful clue in the differential diagnosis of hypertrophic cardiomyopathy. The positive T waves in leads with predominant Q waves (as part of QS or Qr complexes) probably represent a pattern of septal hypertrophy and strain.

References

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