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A cardiac magnetic resonance imaging study of electrocardiographic Q waves in type 2 diabetes: The Fremantle Diabetes Study

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ABSTRACT

To investigate the evolution and significance of Q waves in type 2 diabetes, we studied 36 patients of mean (±S.D.) age 69.9 ± 7.1 years from the longitudinal observational Fremantle Diabetes Study (FDS). All had (i) neither history/symptoms of coronary heart disease (CHD) nor pathological Q waves at FDS recruitment between 1993 and 1996, (ii) five consecutive annual assessments by FDS close-out in 2001, and (iii) contrast-enhanced cardiac magnetic resonance imaging in 2005. At this latter assessment, there were (i) 9 with no history of CHD or Q waves during follow-up (Group 1), (ii) 13 with Q waves on ≥1 electrocardiogram but no CHD history/symptoms (Group 2), and (iii) 14 with CHD history/symptoms irrespective of electrocardiographic status (Group 3). Of 20 episodes of new Q waves in 17 Group 2 or Group 3 patients during FDS follow-up, 17 (85%) resolved within 2 years. A myocardial infarction (MI) was detected by CMR in three patients (8.3%; one subendocardial in Groups 1 and 3, one non-full-thickness in Group 3) but these did not correlate with electrocardiographic appearances. Q waves may have unreliable pathological significance in type 2 diabetes, including as a marker of silent MI.

1. Introduction

Although the presence of Q waves on a 12-lead resting electrocardiogram (ECG) has been used as a simple marker of previous myocardial infarction (MI) in many epidemiological studies, there is evidence that this abnormality does not always indicate transmural necrosis [1,2] and that pathological Q waves can regress over a period of up to several years [3]. In a previous community-based study in type 2 diabetes, the Fremantle Diabetes Study (FDS) [4], we found that patients with 'silent' MI, Q waves consistent with definite/probable MI but without a history or symptoms of coronary heart disease (CHD), had subsequent risks of cardiac and all-cause mortality that were similar to those in patients with no clinical or electrocardiographic evidence of CHD. This apparently favourable prognosis could be explained by the fact that Q waves do not always reflect myocardial damage and/or that there are asymptomatic patients without Q waves who have undetected but significant CHD.

Contrast-enhanced cardiac magnetic resonance imaging (CMR) is a highly accurate method of identifying the site and extent of MI [5–7]. A number of recent studies have examined the relationship between Q waves and myocardial damage using CMR and other imaging modalities including myocardial scintigraphy and positron emission tomography. Most have suggested that Q waves have relatively high specificity but low

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sensitivity [8–10], but there is also evidence that Q waves are specific and sensitive for only anterior [11] or large [12] MIs. All these studies were, however, cross-sectional, and none included control subjects, patients with silent MI or diabetic sub-groups. In order to investigate the evolution and pathological significance of Q waves in type 2 diabetes, we have carried out a longitudinal pilot study of 36 well-characterised patients from the FDS who were free of clinical or ECG evidence of CHD at study entry, were followed for the next 5 years and who underwent subsequent CMR.

2. Materials and methods

2.1. Patients

The FDS was a longitudinal observational cohort study of patients from a postcode-defined community of 120,097 people. Descriptions of recruitment, sample characteristics including classification of diabetes type and details of non-recruited patients have been published elsewhere [13]. Of 2258 diabetic patients identified between 1993 and 1996, 1426 (63%) were recruited to the FDS and 1294 had type 2 diabetes. Eligible patients who declined participation were a mean of 1.4 years older than participants, but their sex distribution, the proportion with type 2 diabetes and the distribution of treatment modalities were similar [13]. The FDS protocol was approved by the Human Rights Committee at Fremantle Hospital and all subjects gave informed consent before participation.

At FDS study entry and annual reviews, patients were classified as having CHD if there was a self-reported history of MI, angina (including use of medication specific for this diagnosis), coronary artery bypass grafting, and/or angioplasty/stenting. In addition, regularly updated hospitalisation data from the Western Australian Hospital Morbidity Database [14] was searched during and after the FDS for separations with a primary diagnosis of ischaemic heart disease as defined by International Classification of Disease (ICD) principal diagnosis codes 410-414 (ICD-9-CM) and I20-I22, I24, I25 (ICD-10-AM).

For the purposes of the present sub-study, we retrospectively identified the 303 type 2 patients who had no history or symptoms of CHD and no pathological Q waves at baseline who had completed five subsequent consecutive annual reviews up to FDS close-out at the end of June 2001. These patients were further divided into three groups: (i) those without clinical evidence of MI and with no Q waves on ECGs taken at each annual review ($n = 227$; Group 1), (ii) those with a history of silent MI on an ECG taken at any of the five annual reviews but no history or symptoms of CHD) ($n = 43$; Group 2), and (iii) those with a history or symptoms of CHD during follow-up irrespective of ECG status ($n = 33$; Group 3). We invited >30 randomly selected patients from the three groups (total $n = 108$) to attend for reassessment in 2005, with a target of 10–15 patients in each group. Those with an absolute contraindication to CMR such as pacemakers or defibrillators were excluded, as were patients with claustrophobia and with a known sensitivity to gadolinium contrast. The present sub-study protocol was approved by the Southern Metropolitan Human Research Ethics Committee and all subjects gave informed consent before participation.

2.2. Clinical assessment and CMR study

Each patient underwent a full clinical assessment in which demographic, anthropometric, clinical and biochemical data were collected [13]. A standard 12-lead ECG was recorded at 25 mm/s chart speed and 10 mm/l mV sensitivity both on inspiration and expiration. A trained technician who was blinded to the clinical data manually interpreted all the ECGs using the Minnesota coding system [15], and a medically trained investigator (SK) verified abnormal findings. Traces with Q wave codes 1.1 or 1.2 were identified as consistent with definite or probable MI [4]. This pair of codes has the highest specificity for silent MI of all ECG abnormalities associated with CHD [16].

Contrast-enhanced CMR was performed using a Siemens AVANTO 1.5 T CMR. All patients received a single injection of commercially available gadolinium chelate at a dose of 0.2 mmol/kg. Five to 15 min after contrast administration, standard inversion recovery flash (T1 weighted) images with the TI (time for inversion) set to null normal myocardium, were acquired. Using repeated breath-holds for each slice, short axis views were obtained from just above the level of the mitral valve to beyond the apex with no gaps between the slices. Each slice had an in-plane resolution of about 2 mm × 1.5 mm with a slice thickness of 6 mm. Standard radial views (2-chamber, 3-chamber and 4-chamber) were acquired, as were extra radial views through any regions of hyper-enhancement seen on the short axis views. The CMR data were assessed by a cardiologist (LD) and an independent physician for the presence and location of MIs and other abnormalities using a standard 16-segment model. All interpretations were done without prior knowledge of the silent MI status of the patients.

2.3. Statistical analysis

The computer packages SPSS for Windows (version 14.0; SPSS Inc., Chicago, IL, USA) was used for data analysis. Data are presented as proportions, mean (±S.D.), geometric mean (S.D. range), or, in the case of variables which did not conform to a normal or log-normal distribution, median [interquartile range (IQR)]. For multiple comparisons of independent samples, the Chi-squared test or the Freeman–Halton extension of Fisher's exact test for a contingency table was used for proportions, ANOVA for normally distributed continuous variables, and the Kruskal–Wallis H-test for non-normally distributed variables. The level of statistical significance was taken as $p < 0.05$.

3. Results

3.1. Sample characteristics

We recruited 36 FDS patients with type 2 diabetes. After clinical assessment, review of the ECG taken specifically for the purposes of the present sub-study and re-categorisation if