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Pathological Q waves as an indicator of prior myocardial infarction in patients with coronary artery disease and diabetes mellitus: a comparison of the prevalence and diagnostic accuracy according to present and former criteria

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ABSTRACT

Introduction. Electrocardiography (ECG) is a widely used diagnostic method for identification of patients with previous myocardial infarction (MI). The ECG manifestation of prior MI is the presence of the pathological Q waves. Patients with coronary artery disease (CAD) and diabetes are at high risk of MI. The aim of this study was to compare the prevalence and diagnostic accuracy of the pathological Q waves as an indicator of prior MI in patients with CAD and diabetes according to the present and former criteria.

Methods. A cross-sectional, multi-centre study was conducted in outpatient clinics across Poland. Family physicians performed physical examinations, registered ECGs, and collected relevant information about onset of CAD and diabetes, presence and onset of hypertension, dyslipidaemia, heart failure, diabetic complications, history of MI, and pharmacotherapy. Centralised manual assessment of the obtained ECG tracings was performed. Two definitions of the pathological Q-waves were used — a present one according to the Universal Definition of MI and a former one based on the definition of MI developed by the World Health Organization.

Results. We enrolled 796 patients (48.1% women, mean age 67.5 ± 10.2 years, and 51.9% men, mean age 64.3 ± 10.3 years) into the study. There were 158 patients (19.8%) — 102 men (24.7%) and 56 women (14.6%), who met the present definition of the pathological Q waves and 106 patients (13.3%) — 74 men (17.9%) and 32 women (8.4%), who met the former definition of the pathological Q waves. The prevalence of the pathological Q waves varied due to the certain group of leads. It was highest in the inferior leads — 104 and 75 according to the present and former definitions, respectively. Of note, the rate of the pathological Q waves increased up to 2.6 times in the lateral leads after the introduction of the less restrictive present definition. Sensitivity of prior MI detection by means of the present and former criteria was 26.8% and 19.8%, and specificity was 87.0% and 92.8%, respectively. The application of the present and former definitions detected prior MI with 65.6% and 71.6% positive predictive value, and with 56.3% and 55.6% negative predictive value, respectively.

Conclusions. In the era of reperfusion therapy, ECG appears to be a poor diagnostic tool for detection of previous MI due to its low sensitivity. However, it may identify individuals without previous MI with rather high specificity. In diabetics with CAD, the present definition of the pathological Q waves increases sensitivity of prior MI detection by 7%, with a decrease in specificity by 6% as compared with the former definition.

Key words: electrocardiography, pathological Q waves, coronary artery disease, diabetes mellitus

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Introduction

Electrocardiography (ECG) is a simple and widely available method for identification of patients with prior myocardial infarction (MI). Although the definition of MI has evolved in time, the presence of the pathological Q waves still remains significant for previous MI [1, 2]. However, due to the recent improvements in the treatment of acute coronary syndromes, we can observe a decrease in the incidence of Q-wave MI and an increase in the incidence of non-Q-wave MI [3, 4]. In 2007, the Joint European Society of Cardiology, American College of Cardiology Foundation, American Heart Association, and World Heart Federation Task Force introduced a new pathological Q waves definition, which is less restrictive than the previous one, so the amount of diagnosed MI based on ECG might have increased [5].

Patients with coronary artery disease (CAD) and diabetes are at high risk of MI [6, 7]. Several studies suggested that diabetes itself is an equivalent for CAD and is related with as high risk of MI as in non-diabetic patients with previous MI [8–10]. The presence of diabetes or CAD is connected with an increased risk of cardiovascular morbidity and mortality [11–14]. Importantly, the coexistence of diabetes and CAD increases cardiovascular risk geometrically [15].

The aim of this study was to compare the prevalence and diagnostic accuracy of the pathological Q waves as an indicator of prior MI in patients with CAD and diabetes according to the present and former criteria.

Methods

Study design and conduction

A cross-sectional, multi-centre study was conducted among patients with CAD and diabetes by family physicians in primary care centres in Poland in 2009.
Inclusion criteria were:
— previous angiographically proven clinical diagnosis of CAD;
— previous diagnosis of type 2 diabetes mellitus according to the American Diabetes Association criteria, and
— provision of informed consent for the study participation.
Exclusion criteria consisted of:
— presence of ventricular pacing;
— presence of left or right bundle branch block;
— presence of left anterior or posterior hemiblock;
— presence of left ventricular hypertrophy (recognised with at least one of seven ECG criteria used, as described in our previous publication [16]);
— presence of pre-excitation;

— inability of adequate ECG assessment and interpretation due to technical reasons.

For each patient, a thorough history was taken and detailed physical examination was performed. Blood pressure was measured by a physician with a mercury sphygmomanometer or an automated device in the outpatient clinic in a quiet and relaxed environment, with the subject seated for at least 10 minutes. The average of two measurements was used for the analysis. Information collected from the medical history included: history of hypertension, dyslipidaemia, gout, heart failure, diabetic complications (diabetic foot, retinopathy, nephropathy, and neuropathy), previous MI, and stroke, as well as duration of hypertension, diabetes, and CAD. Hypertension was defined as systolic blood pressure $\geq$ 140 mm Hg and/or diastolic blood pressure $\geq$ 90 mmHg or treatment with anti-hypertensive drugs. Measured laboratory parameters included: fasting plasma glucose, glycated haemoglobin (HbA_{1c}), total cholesterol, high-density lipoprotein (HDL) cholesterol, low-density lipoprotein (LDL), cholesterol, and triglycerides. The aforementioned results were collected from patients' medical documentation when measured within 12 months. Detailed information on participants' pharmacological treatment during the last six months was recorded, including the use of metformin, sulfonylurea derivates, insulin, acetylsalicylic acid, clopidogrel, statins, fibrates, angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, beta-adrenolytics, diuretics, and calcium channel antagonists.

The study protocol was approved by the Local Ethics Committee and was performed in accordance with the Declaration of Helsinki.

Electrocardiography

Standard supine 12-lead ECGs were registered at a 25 mm/s speed and 10 mm/mV gain. They were analysed according to the American Heart Association, American College of Cardiology Foundation, and Heart Rhythm Society Recommendations for the Standardisation and Interpretation of the Electrocardiogram [17]. Centralised evaluation was performed in all ECGs manually, questionable cases were resolved by an independent, experienced cardiologist. Segment duration was the average of three measurements of consecutive complexes. In order to detect the pathological Q waves we used two definitions: the present one based on the Universal Definition of MI endorsed by the Joint European Society of Cardiology, American College of Cardiology Foundation, American Heart Association, and World Heart Federation Task Force for the Redefinition of MI, and the former one based on the definition of MI developed by the World Health Organization [5].

Table 1. Prevalence of the pathological Q waves depending on the location in the electrocardiogram

Location	Qp n = 158	Qf n = 106	Difference Qp vs. Qf	Qp Men n = 102	Qf Men n = 74	Difference Qp vs. Qf Men	Qp Women n = 56	Qf Women n = 32	Difference Qp vs. Qf Women
Lateral (leads I, aVL, V6)	52 (32.9)	20 (18.9)	32 (160.0)	27 (26.5)	11 (14.9)	16 (145.5)	25 (44.6)	9 (28.1)	16 (177.8)
Inferior (leads II, III, aVF)	104 (65.8)	75 (70.8)	29 (38.7)	71 (69.6)	55 (74.3)	16 (29.1)	33 (58.9)	20 (62.5)	13 (65.0)
Anterior (leads V1–V5)	27 (17.1)	21 (19.8)	6 (28.6)	16 (15.7)	13 (17.6)	3 (23.1)	11 (19.6)	8 (25.0)	3 (37.5)

Data are presented as numbers (percentages). Qf — former definition of the pathological Q waves; Qp — present definition of the pathological Q waves

According to the present definition, a pathological Q-wave is any Q-wave of duration ≥ 0.02 s or QS complex in leads V2–V3 or a Q-wave of duration ≥ 0.03 s and amplitude ≥ 0.1 mV or QS complex in leads I, II, aVL, aVF, or V4–V6 in any two leads of a contiguous lead grouping (I, aVL, V6; V4–V6; II, III, aVF). The former criteria defined a pathological Q-wave as a Q-wave of duration ≥ 0.04 s and amplitude ≥ 0.1 mV or QS complex in any two leads of a contiguous lead grouping.

Statistical analysis

The results are shown as mean $\pm$ standard deviation (SD) for continuous variables and as number and percentage for categorical variables. Continuous data were checked for normal distribution by the use of the Shapiro-Wilk W test. Differences in clinical characteristics between patients meeting the present and former definitions of the pathological Q waves were assessed using the Chi-square test for categorical variables and the Student's t-test for normally distributed continuous variables. The non-parametric Mann-Whitney U test was applied for comparisons of continuous variables for which distribution deviated from normality. A two-sided difference was considered significant at $p < 0.05$. The statistical analysis was carried out using the Statistica 9.1 package (StatSoft, Tulsa, OK, USA).

Results

Out of 1116 potentially eligible patients, 796 subjects — 383 women (48.1%), mean age 67.5 $\pm$ 10.2 years, and 413 men (51.9%), mean age 64.3 $\pm$ 10.3 years, were enrolled into the study. We excluded 320 participants due to: paced rhythm in the assessed ECGs (n = 18), the presence of left or right bundle branch block (n = 76), the presence of left ventricular hypertrophy (n = 200), the presence of ventricular pre-excitation (n = 5), or inability of adequate ECG assessment and interpretation caused by technical reasons (n = 21).

There were 158 patients (19.8%), 102 men (24.7%) and 56 women (14.6%), who met the present definition of the pathological Q waves, and 106 patients (13.3%), 74 men (17.9%) and 32 women (8.4%), who met the former definition of the pathological Q waves. We observed a significant increase of the pathological Q wave recognition amounting 49.1% comparing patients meeting the former definition (n = 106) and the present definition (n = 158). The prevalence of the pathological Q waves varied due to the certain groups of leads. It was the highest in the inferior leads (II, III, aVF) — 104 according to the present definition and 75 according to the former definition. Of note, the rate of the pathological Q waves increased up to 2.6 times in the lateral leads, after the introduction of the less restrictive present definition (Tab. 1).

The statistical analysis of the whole study population did not show any differences between patients meeting the present and former criteria of the pathological Q waves (Tab. 2). We also did not reveal any differences comparing men or women meeting the present and former definitions (Tab. 3).

We also compared patients meeting the present definition of the pathological Q waves but not fulfilling the former criteria with patients meeting the former definition only. Patients meeting only the former definition more often had previous MI, had higher concentration of triglycerides, and were more often treated with beta-blockers as compared with those meeting only the present definition. There was also a trend showing that patients meeting only the former definition were more often treated with metformin and less often with sulfonylurea derivates as compared with those meeting the present definition (Tab. 4).

Using information from the anamnesis concerning previous MI, we calculated sensitivity, specificity, and predictive values for prior MI detection by means of the present and former definitions of the pathological Q waves. The sensitivity was 26.8% and 19.8%, while the specificity was 87.0% and 92.8% for the present and former criteria, respectively. Because of the relatively low

Table 2. Clinical characteristics of patients meeting the present and former definitions of the pathological Q waves

Clinical feature	Qp n = 158	Qf n = 106	p
Age (years)	65.5 ± 9.6	65.5 ± 9.2	0.96
Height [cm]	170.5 ± 7.8	170.7 ± 7.7	0.83
Weight [kg])	90.2 ± 15.2	91.3 ± 14.7	0.69
BMI [kg/m^2]	31.1 ± 5.4	31.4 ± 5.4	0.78
Waist circumference [cm]	102.2 ± 12.8	103.5 ± 11.7	0.55
Hips circumference [cm]	106.2 ± 11.1	106.1 ± 11.7	0.91
Heart rate [beats/min]	73.3 ± 12.8	73.5 ± 13.3	0.97
Systolic blood pressure [mm Hg]	140.3 ± 17.1	139.1 ± 16.6	0.38
Diastolic blood pressure [mm Hg]	82.7 ± 10.4	81.9 ± 10.0	0.59
Previous MI	99 (62.7%)	73 (68.9%)	0.30
Previous stroke	20 (12.7%)	11 (10.4%)	0.57
Duration of CAD (years)	5.8 ± 5.4	5.8 ± 5.7	0.78
Duration of diabetes (years)	8.5 ± 6.7	8.5 ± 6.2	0.78
Duration of hypertension (years)	12.6 ± 8.3	12.4 ± 8.1	0.92
Duration of dyslipidaemia (years)	9.9 ± 6.2	10.0 ± 5.9	0.72
Duration of heart failure (years)	4.6 ± 4.3	4.4 ± 4.5	0.75
Duration of microangiopathy (years)	5.1 ± 4.3	4.2 ± 3.3	0.40
Duration of heart failure (years)	4.6 ± 4.3	4.4 ± 4.5	0.75
Laboratory tests results:			
Fasting plasma glucose [mg/dL]	124.7 ± 32.7	121.8 ± 25.8	0.61
HbA$_{1c}$ (%)	7.2 ± 1.0	7.2 ± 1.0	0.80
Total cholesterol [mg/dL]	203.2 ± 49.0	203.4 ± 48.4	0.88
HDL cholesterol [mg/dL]	48.7 ± 17.4	46.2 ± 14.7	0.50
LDL cholesterol [mg/dL]	119.0 ± 40.4	116.2 ± 36.3	0.87
Triglycerides [mg/dL]	164.0 ± 96.4	173.0 ± 107.4	0.38
Treatment:			
ACEI	129 (81.6%)	89 (84.0%)	0.63
ARB	31 (19.6%)	22 (20.8%)	0.82
Acetylsalicylic acid	134 (84.8%)	92 (86.8%)	0.65
Clopidogrel	32 (20.3%)	25 (23.6%)	0.52
Statins	141 (89.2%)	97 (91.5%)	0.54
Fibrates	35 (22.2%)	27 (25.5%)	0.53
Beta-adrenolytics	141 (89.2%)	99 (93.4%)	0.25
Diuretics	85 (53.8%)	59 (55.7%)	0.77
CCA	42 (26.6%)	26 (26.4%)	0.98
Metformin	108 (68.4%)	77 (72.6%)	0.46
Sulfonylurea derivates	62 (39.2%)	36 (34.0%)	0.38
Insulin	48 (30.4%)	34 (32.1%)	0.77

Data are presented as mean values ± SD or numbers (percentages). ACEI — angiotensin-converting enzyme inhibitors; ARB — angiotensin II receptor blockers; BMI — body mass index; CAD — coronary artery disease; CCA — calcium channel antagonists; HbA$_{1c}$ — glycated haemoglobin; HDL — high-density lipoprotein; LDL — low-density lipoprotein; MI — myocardial infarction; Qf — former definition of the pathological Q waves; Qp — present definition of the pathological Q waves

Table 3. Sex-related clinical characteristics of patients meeting the present and former criteria of the pathological Q waves

Clinical feature	Men Qp n = 102	Men Qf n = 74	p	Women Qp n = 54	Women Qf n = 32	p
Age (years)	65.0 ± 9.3	65.2 ± 9.0	0.84	66.4 ± 10.2	66.7 ± 9.8	0.93
Height [cm]	174.6 ± 5.9	174.2 ± 5.7	0.63	163.1 ± 4.5	162.0 ± 4.0	0.33
Weight [kg]	94.0 ± 13.5	94.0 ± 13.5	0.87	83.5 ± 15.9	85.6 ± 15.9	0.60
BMI [kg/m²]	30.9 ± 4.8	31.1 ± 5.0	0.96	31.5 ± 6.4	32.7 ± 6.4	0.46
Waist circumference [cm]	105.0 ± 11.8	105.0 ± 11.5	0.99	97.3 ± 13.2	100.8 ± 11.3	0.28
Hips circumference [cm]	104.9 ± 10.7	105.1 ± 11.3	1.00	108.6 ± 11.4	109.0 ± 8.5	0.75
Heart rate [beats/min]	72.4 ± 1.0	72.7 ± 1.0	0.09	75.1 ± 12.2	76.1 ± 10.7	0.57
Systolic blood pressure [mm Hg]	139.3 ± 17.0	138.0 ± 17.0	0.60	142.2 ± 17.3	142.8 ± 15.6	0.65
Diastolic blood pressure [mm Hg]	82.8 ± 10.3	82.3 ± 10.5	0.71	82.5 ± 10.7	82.0 ± 9.4	0.94
Previous MI syndrome	66 (64.7%)	51 (68.9%)	0.56	33 (58.9%)	22 (68.8%)	0.81
Previous stroke	12 (11.8%)	7 (9.5%)	0.63	8 (14.3%)	4 (12.5%)	0.81
Duration of CAD (years)	5.2 ± 5.2	5.6 ± 5.5	0.56	7.1 ± 5.6	6.3 ± 5.9	0.39
Duration of diabetes (years)	8.1 ± 6.6	8.4 ± 6.2	0.66	9.1 ± 6.9	9.5 ± 7.0	0.70
Duration of hypertension (years)	11.9 ± 8.0	11.4 ± 7.7	0.76	14.0 ± 8.6	15.3 ± 8.7	0.43
Duration of dyslipidaemia (years)	9.6 ± 6.4	9.8 ± 6.1	0.81	10.5 ± 6.0	11.5 ± 6.4	0.51
Duration of heart failure (years)	3.8 ± 3.7	3.6 ± 3.6	0.86	6.2 ± 5.0	6.4 ± 5.8	0.98
Duration of microangiopathy (years)	4.8 ± 4.0	4.1 ± 2.7	0.76	5.6 ± 4.9	5.2 ± 4.4	0.91
Laboratory rests results:						
Fasting plasma glucose [mg/dL]	119.6 ± 20.9	119.4 ± 22.1	0.86	132.8 ± 44.7	128.7 ± 32.9	0.78
HbA$_{1c}$ (%)	7.2 ± 1.0	7.3 ± 1.0	0.72	7.2 ± 1.1	7.3 ± 1.3	0.89
TC [mg/dL]	205.3 ± 47.8	209.4 ± 50.2	0.65	199.8 ± 51.4	191.7 ± 39.7	0.79
HDL [mg/dL]	46.8 ± 17.7	44.6 ± 14.6	0.66	52.1 ± 16.5	53.4 ± 17.0	0.72
LDL [mg/dL]	123.1 ± 34.3	122.8 ± 32.3	0.97	111.5 ± 49.3	102.8 ± 40.0	0.72
TG [mg/dL]	169.9 ± 107.1	181.0 ± 121.5	0.52	153.1 ± 72.7	154.6 ± 50.5	0.52
Treatment:						
ACEI	82 (80.4%)	60 (81.1%)	0.91	47 (83.9%)	29 (90.6%)	0.38
ARB	25 (24.5%)	20 (19.6%)	0.71	6 (10.7%)	2 (6.3%)	0.48
Acetylsalicylic acid	85 (83.3%)	62 (83.8%)	0.94	49 (87.5%)	30 (93.8%)	0.35
Clopidogrel	24 (23.5%)	18 (24.3%)	0.90	8 (14.3%)	7 (21.9%)	0.36
Statins	93 (91.2%)	68 (91.9%)	0.87	48 (85.7%)	29 (90.6%)	0.50
Fibrates	24 (23.5%)	19 (25.7%)	0.74	11 (19.6%)	8 (25.0%)	0.56
Beta-adrenolytics	91 (89.2%)	69 (93.2%)	0.17	50 (89.3%)	30 (93.8%)	0.48
Diuretics	58 (56.9%)	42 (56.8%)	0.99	27 (48.2%)	17 (53.1%)	0.66
CCA	24 (23.5%)	18 (24.3%)	0.90	18 (32.1%)	10 (31.3%)	0.93
Metformin	63 (61.8%)	51 (68.9%)	0.33	45 (80.4%)	26 (81.3%)	0.92
Sulfonylurea derivates	34 (33.3%)	25 (33.8%)	0.95	28 (50.0%)	11 (34.4%)	0.16
Insulin	35 (34.3%)	25 (33.8%)	0.72	13 (23.2%)	9 (28.1%)	0.61

Data are presented as mean values ± SD or numbers (percentages). ACEI — angiotensin-converting enzyme inhibitors; ARB — angiotensin II receptor blockers; BMI — body mass index; CAD — coronary artery disease; CCA — calcium channel antagonists; HbA$_{1c}$ — glycated haemoglobin; HDL — high-density lipoprotein; LDL — low-density lipoprotein; MI — myocardial infarction; Qf — former definition of the pathological Q waves; Qp — present definition of the pathological Q waves

Table 4. Clinical characteristics of patients meeting the present criteria of the pathological Q waves but not fulfilling former criteria, and those meeting the former criteria only

Clinical feature	Patients meeting Qp but not Qf n = 52	Patients meeting only Qf n = 106	p
Age (years)	65.2 ± 10.5	65.7 ± 9.2	0.73
Height [cm]	170.5 ± 8.0	170.5 ± 7.7	0.88
Weight [kg]	87.6 ± 16.2	91.5 ± 14.7	0.26
BMI [kg/m^2]	30.1 ± 5.2	31.6 ± 5.5	0.33
Waist circumference [cm]	99.1 ± 14.6	103.7 ± 11.6	0.07
Hips circumference [cm]	106.2 ± 12.1	106.3 ± 10.6	0.87
Heart rate [beats/min]	72.6 ± 12.3	73.6 ± 13.1	0.66
Systolic blood pressure [mm Hg]	142.2 ± 18.0	139.4 ± 142.2	0.55
Diastolic blood pressure [mm Hg]	83.7 ± 11.1	82.2 ± 10.1	0.44
Previous MI	26 (50.0%)	73 (68.9%)	<0.05
Previous stroke	9 (17.3%)	11(10.4%)	0.22
Duration of CAD (years)	5.9 ± 5.1	5.8 ± 5.6	0.75
Duration of diabetes (years)	7.9 ± 7.2	8.7 ± 6.4	0.25
Duration of hypertension (years)	12.6 ± 8.4	12.6 ± 8.2	0.82
Duration of dyslipidaemia (years)	8.9 ± 6.4	10.3 ± 6.2	0.18
Duration of microangiopathy (years)	6.6 ± 5.9	4.5 ± 3.4	0.34
Duration of heart failure (years)	5.5 ± 3.5	4.4 ± 4.5	0.33
Laboratory tests results:			
Fasting plasma glucose [mg/dL]	129.1 ± 42.9	122.5 ± 26.3	0.41
HbA$_{1c}$ (%)	7.0 ± 0.8	7.3 ± 1.1	0.35
TC [mg/dL]	202.1 ± 51.8	203.9 ± 47.7	0.57
HDL [mg/dL]	51.8 ± 20.6	47.3 ± 15.8	0.42
LDL [mg/dL]	123.8 ± 48.8	116.6 ± 35.8	0.87
TG [mg/dL]	146.0 ± 74.0	172.7 ± 105.0	<0.05
Treatment:			
ACEI	40 (76.9%)	89 (84.0%)	0.28
ARB	9 (17.3%)	22 (20.8%)	0.61
Acetylsalicylic acid	42 (80.8%)	92 (86.8%)	0.32
Clopidogrel	7 (13.5%)	25 (23.6%)	0.14
Statins	44 (84.6%)	97 (91.5%)	0.19
Fibrates	8 (15.4%)	27 (25.5%)	0.15
Beta-adrenolytics	42 (80.8%)	99 (93.4%)	<0.05
Diuretics	26 (50.0%)	59 (55.7%)	0.50
CCA	14 (26.9%)	28 (26.4%)	0.95
Metformin	31 (59.6%)	77 (72.6%)	0.098
Sulfonylurea derivates	26 (50.0%)	36 (34.0%)	0.052
Insulin	14 (26.9%)	34 (32.1%)	0.51

Data are presented as mean values ± SD or numbers (percentages). ACEI — angiotensin-converting enzyme inhibitors; ARB — angiotensin II receptor blockers; BMI — body mass index; CAD — coronary artery disease; CCA — calcium channel antagonists; HbA$_{1c}$ — glycated haemoglobin; HDL — high-density lipoprotein; LDL — low-density lipoprotein; MI — myocardial infarction; Qf — former definition of the pathological Q waves; Qp — present definition of the pathological Q waves.
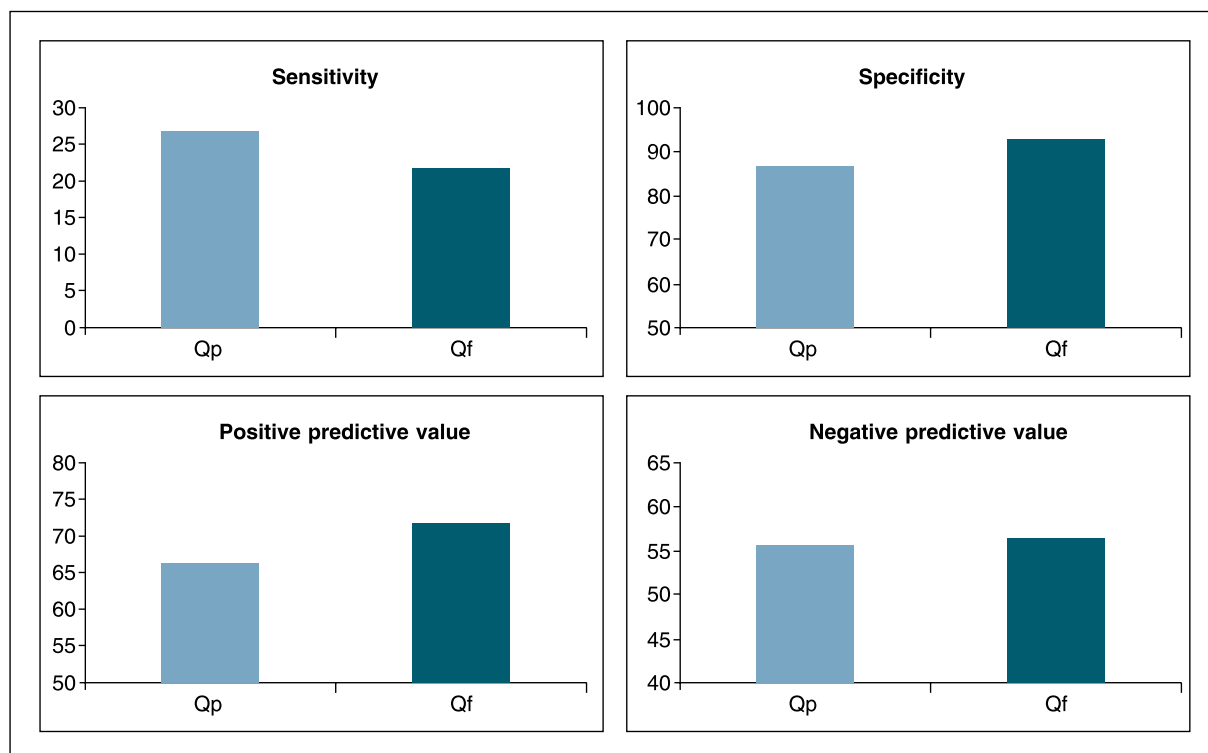
Figure 1. Diagnostic accuracy of the presence of the pathological Q waves according to the present and former criteria for the detection of prior myocardial infarction. Qp — present definition of the pathological Q waves; Qf — former definition of the pathological Q waves

false positive rate (present = 13.0%, former = 7.2%), the predictive positive value of the pathological Q waves presence was 65.6% and 71.6% for the present and former definitions, respectively. Because of the high false negative rate (present = 73.2%, former = 80.2%), their absence truly reflected the absence of previous MI in only 56.3% and 55.6% for the present and former criteria, respectively (Fig. 1).

Discussion

In our study, the prevalence of the pathological Q waves according to the present and former criteria was 158 (20%) and 106 (13%), respectively. We observed a 49% increase in the rate of the pathological Q wave recognition after the introduction of the present definition, resulting in a 49% increase in the ECG recognition of prior MI. Parikh et al., while analysing the Framingham Heart Study data, noticed a significant decrease in MI fatality rates with the prevalence rate at a similar level; it was the so-called "the biomarker effect" [18]. The number of MIs diagnosed based on ECG decreased, but simultaneously the number of MIs diagnosed based on biomarkers increased.

As a result, the total MI prevalence was much higher than expected, due to the introduction of more accurate and more sensitive cardiac necrosis biomarkers [18].

Focusing on the prevalence of the pathological Q waves depending on the location, we observed a 160% increase in the rate of the pathological Q waves in lateral leads, a 29% increase in anterior leads, and a 39% increase in inferior leads when the present criteria were applied. According to the present ECG standards, small Q waves are acceptable in leads I, aVL, aVF, and V4–V6 [1], which may explain the significant increase of the pathological Q wave diagnosis in lateral leads, and we may expect some of them to be false positive.

The major finding of this study was very low sensitivity of the pathological Q waves for detecting prior MI, according to the present definition, which was approximately 27%, while according to the former definition it was 20%. In our study, specificity was 87% and 93% for the present and former criteria, respectively. According to older papers based on autopsy as the reference method by Horan et al. and Lindvall et al., corresponding values for the sensitivity of the pathological Q wave presence were 61% and 76%, and for their specificity were 89% and 76%, respectively [19, 20].

In more recent papers by Al-Mohammad et al., Sandler et al., Asch et al., and Nadour et al., based on positron emission tomography, scintigraphy, and magnetic resonance imaging (the two last studies) as the reference methods, respectively, the sensitivity of the pathological Q waves was 41–66%, while the specificity ranged from 75% to 85% [21–24]. Furthermore, in recent years, according to the significant progress in treatment of acute coronary syndromes, we can observe a decreasing proportion of patients diagnosed with Q-wave MIs (ranging from 38% to 67%) together with a corresponding increase in the proportion of non-Q-wave MIs [4, 25]. This fact seems to be crucial for the interpretation of our findings.

Interestingly, Godsk et al. hypothesised that the presence of any Q waves in individuals without known cardiovascular disease is a predictor of poor prognosis [26]. They followed 5381 subjects, of whom 114 (2.1%) had Q waves in ECG. In their study, the primary endpoint, a composite of death and hospitalisation for CAD, occurred more often in patients with Q waves as compared with participants without Q waves in ECG (44% vs. 18%; p < 0.001). The authors underlined that the presence of even small Q waves is a prognostic factor of poor outcome (hazard ratio 1.4; 95% confidence interval 1.0–2.0; p < 0.05).

There are some limitations of the current study that deserve attention. First of all, ECGs were recorded in our study using analog, not digital, systems and therefore they were analysed manually. Secondly, we were not able to use the same electrocardiographic devices for all patients in different regions of Poland. Thirdly, the information about previous MI was based on the patient's history, not on echocardiography or magnetic resonance imaging.

Conclusions

In the era of reperfusion therapy, ECG appears to be a poor diagnostic tool for detection of previous MI due to its low sensitivity. However, it may identify individuals without previous MI with rather high specificity. In diabetics with CAD, the present definition of the pathological Q waves increases the sensitivity of prior MI detection by 7%, with a decrease in specificity of 6% as compared with the former definition.

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References

1. Thygesen K, Alpert JS, Jaffe AS et al. Third universal definition of myocardial infarction. Eur Heart J 2012; 33: 2551–2567.
2. Roberts R, Pratt CM, Alexander RW. Pathophysiology, recognition and treatment of acute myocardial infarction and its complications. In: Schlant RC, Alexander RW, eds. Hurst's the Heart: Arteries and Veins. McGraw-Hill, New York 1994: 1107–1184.
3. Abdulla J, Brendorp B, Torp-Pedersen C, Kober L. Does the electrocardiographic presence of Q waves influence the survival of patients with acute myocardial infarction? Eur Heart J 2001; 22: 1008–1014.
4. Furman MI, Dauerman HL, Goldberg RJ, Yarzebski J, Lessard D, Gore JM. Twenty-two year (1975 to 1997) trends in the incidence, in-hospital and long-term case fatality rates from initial Q-wave and non–Q-wave myocardial infarction: a multi-hospital, community-wide perspective. J Am Coll Cardiol 2001; 37: 1571–1580.
5. Thygesen K, Alpert JS, White HD et al. Universal definition of myocardial infarction. Eur Heart J 2007; 28: 2525–2538.
6. Kannel WB, McGee DL. Diabetes and glucose tolerance as risk factors for cardiovascular disease: the Framingham Study. Diabetes Care 1979; 2: 120–126.
7. Pyörälä K, Laakso M, Uusitupa M. Diabetes and atherosclerosis: an epidemiologic view. Diabetes Metab Rev 1987; 3: 463–524.
8. Whiteley L, Padmanabhan S, Hole D, Isles C. Should diabetes be considered a coronary heart disease risk equivalent? Diabetes Care 2005; 28: 1588–1593.
9. Juutilainen A, Lehto S, Ronnemaa T, Pyorala K, Laakso M. Type 2 Diabetes as a "Coronary Heart Disease Equivalent". An 18-year prospective population-based study in Finnish subjects. Diabetes Care 2005; 28: 2901–2907.
10. Haffner SM, Lehto S, Ronnemaa T, Pyorala K, Laakso M. Mortality from Coronary Heart Disease in Subjects with Type 2 Diabetes and in Nondiabetic Subjects with and without Prior Myocardial Infarction. N Engl J Med 1998; 339: 229–234.
11. Kannel WB, McGee DL. Diabetes and cardiovascular disease: the Framingham study. JAMA 1979; 241: 2035–2038.
12. Nathan DM, Meigs J, Singer DE. The epidemiology of cardiovascular disease in type 2 diabetes mellitus: how sweet it is or is it? Lancet 1997; 350: 14–19.
13. Pekkanen J, Linn S, Heiss G et al. Ten-year mortality from cardiovascular disease in relation to cholesterol level among men with and without preexisting cardiovascular disease. N Engl J Med 1990; 322: 1700–1707.
14. Rosengren A, Hagman M, Wedel H, Wilhelmsen L. Serum cholesterol and long-term prognosis in middle-aged men with myocardial infarction and angina pectoris: a 16-year follow-up of the Primary Prevention Study in Göteborg, Sweden. Eur Heart J 1997; 18: 754–761.
15. Oguz A, Damci T, Pehlivanoglu S et al. The impact of diabetes and coronary artery disease on mortality and morbidity in patients with hypertension. Turk Kardiyol Dern Ars 2009; 37: 221–225.
16. Ostrowska M, Bellwon J, Koziński M et al. Prevalence of electrocardiographic left ventricular hypertrophy among patients with coronary artery disease and diabetes mellitus. Med Res J 2016; 1: 1–9.
17. Hanock EW, Deal BJ, Mirvis DM et al. AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram: Part V: Electrocardiogram Changes Associated With Cardiac Chamber Hypertrophy A Scientific Statement From the American Heart Association Electrocardiography and Arrhythmias Committee, Council of Clinical Cardiology; The American College of Cardiology Foundation; and the Heart Rhythm Society Endorsed by the International Society for the Computerized Electrocardiology. J Am Coll Cardiol 2009; 53: 992–1002.
18. Parikh NI, Gona P, Larson MG et al. Long-term trends in myocardial infarction incidence and case fatality in the National Heart, Lung, and Blood Institute's Framingham Heart Study. Circulation 2009; 119: 1189–1191.
19. Horan LG, Flowers NC, Johnson JC. Significance of the diagnostic Q wave of myocardial infarction. Circulation 1971;43: 428–436.
20. Lindvall K, Erhardt L, Sjogren A. Echo- and electrocardiographic findings in relation to autopsy in myocardial infarction. Clin Cardiol 1982; 5: 51–61.
21. Al-Mohammad A, Norton MY, Mahy IR et al. Can the surface electrocardiogram be used to predict myocardial viability? Heart 1999; 82: 663–667.

22. Sandler LL, Pinnow EE, Lindsay J. The accuracy of electrocardiographic Q waves for the detection of prior myocardial infarction as assessed by a novel standard of reference. Clin Cardiol 2004; 27: 97–100.

23. Asch FM, Shah S, Rattin C, Swaminathan S, Fuisz A, Lindsay J. Lack of sensitivity of the electrocardiogram for detection of old myocardial infarction: A cardiac magnetic resonance imaging study. Am Heart J 2006; 152: 7422–7428.

24. Nadour W, Doyle M, Williams RB et al. Does the presence of Q waves on the EKG accurately predict prior myocardial infarction when compared to cardiac magnetic resonance using late gadolinium enhancement? A cross-population study of noninfarct vs infarct patients. Heart Rhythm 2014; 11: 2018–2026.

25. Abdulla J, Brendorp B, Torp-Pedersen C, Kober L. Does the electrocardiographicpresence of Q waves influence the survival of patients withacute myocardial infarction? Eur Heart J 2001; 22: 1008–1014.

26. Godsk P, Jensen JS, Abildstrom SZ, Appleyard M, Pedersen S, Mogelvang L. Prognostic significance of electrocardiographic Q-waves in a low-risk population. Europace 2012; 14: 1012–1017.