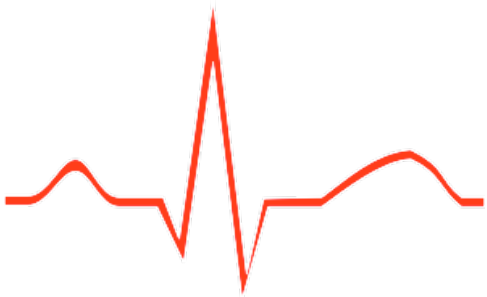
AALBORG UNIVERSITY
DENMARK

Abnormal and normal Q waves in inferior ECG leads



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Abstract

Background Identifying pathologic Q wave is made by measurement of amplitude and duration and to be pathological both should exceed a determined extent. However, it has not been investigated how the amplitude and/or duration contribute in the prediction of prognosis when present in the inferior leads.

Objectives We sought the association between Q wave´s duration and amplitude in inferior leads and prognosis.

Methods By taking advantage of digitalized ECGs; we could identify three exclusive populations to investigate different Q wave morphology in the inferior leads (II, III and aVF) and their prognostic value after a follow-up period of 5 years. Danish registries were used to obtain data on diagnosis and outcomes. We used multivariate-adjusted Cox proportional hazards model to estimate the risk of mortality of Q waves when present with only pathologic duration, only pathologic amplitude and both in inferior leads. Individuals with normal Q waves in the investigated lead were used as a reference.

Results We identified 36,645 individuals for lead II, 8,129 for lead III and 42, 892 for lead aVF. The mortality rates in the study population for lead III, II and aVF were 15.7%, 9.1% and 10.2%, respectively. Multivariate-adjusted risk for mortality was statically significant for individuals with Q waves with only pathologic duration in lead II [hazard ratio (HR): 1.48, confidence interval (95% CI): 1.08-2.02], and for lead aVF (HR: 1.17, CI: 1.05-1.29) and only pathologic amplitude (HR: 1.36, CI: 1.20-1.55 and HR: 1.52, CI: 1.35-1.71) when present in lead II and aVF respectively.

Q waves with only pathologic duration in lead III were also associated with increased mortality (HR: 2.52, CI: 1.44-4.42).

Conclusions This study showed that presence of Q wave with only pathologic duration (≥ 30 msec) or only pathologic amplitude (≥ 100 mV) in one inferior lead (II or aVF) accompanied with presence of Q wave with amplitude ≥ 100 mV and duration ≥ 30 msec in another inferior lead were associated with increased mortality.

Resume

Introduktion Den inferiore ventrikelvæg er et hyppig sted for myokardieinfarkt (MI), og udgør ca. 40-50% af alle MI. Tidligere studier viser at inferior MI er associeret med bedre prognose i forhold til patienter med anterior MI, dog får ca. halvdelen af de med inferior MI, komplikationer som forværrer deres prognose. Forekomst af patologiske Q-takker anses være et tegn på et gammel MI, og der har blevet igennem tiden forskellige udviklet nye klassifikationssystemer til identificering af patologiske Q-takker. Disse klassifikationssystemer er opbygget gennem måling af længden og bredden af en Q-tak, og for at være patologiske skal disse målinger overstige en vis størrelse. De gamle klassifikationssystemer er kendte for at være avancerede og komplekse og for at skabe et fælles kriterium for diagnosering af akutte og gamle MI, Third Universal Definition of Myocardial Infarction kriterier blevet udviklet i 2012. Kriterium for gamle inferior MI tage udgangspunkt i patologiske Q-takker i de inferiore afledninger (II, III og aVF). Patologiske Q-takker ifølge det kriterium skal være tilsted i to afledninger samtidig, længden skal være ≥100 millivolt (mV) og bredden ≥30 millisekund (ms). Det er stadig ikke velundersøgt hvad bredden alene og længden alene i en Q-tak har for en betydning i at forudsige prognosen.

Undersøgelse af Third Universal Definition of Myocardial Infarction for de inferior afledninger og betydning af Q-takker længde og bredde vil bidrage til mere akkurat tolkning af EKG og efterfølgende bedre diagnosering af MI.

Formålet med dette studie er at undersøge relationen mellem Q-takkers længde og bredde i de inferiore afledninger og prognosen ved tilstedeværelse af Q-takker med unormal morfologi i en afledning samtidig som tilstedeværelse af Q-takker i en anden inferior afledning ifølge Third Universal Definition of Myocardial infarction.

Metode Gennem at bruge mere end 500,000 digitaliserede EKG registrerede i Københavns Praktiserende Lægers Laboratorium mellem 2001 og 2011, vi kunne identificere tre eksklusive studiepopulationer til at undersøge forskellig morfologi af Q-takker i de inferiore afledninger (II, III og aVF) og deres prognostisk værdi efter en opfølgningsperiode på 5 år. Administrative danske registre blevet brugt til at samle data vedrørende diagnoser og mortalitet. Multivariat-justeret Cox proportionel hazards model var anvendt til at estimere risikoen for mortalitet når Q-takker er tilstede med kun patologisk længde, kun patologisk bredde og når begge er patologiske samtidig. Reference gruppen bestod af individer som har normale Q-takker i den undersøgte afledning.

Resultater Vi identificerede 36,645 individer for at undersøge Q-takker i afledning II, 8,129 individer i afledning III og 42,892 individer i afledning aVF. Efter 5års opfølgningsperiode mortalitet i studiepopulation for Q-takker i afledning III var 15.7%, 9.1% i studiepopulation for afledning II og 10.2% i studiepopulationen for afledning aVF. Gennem den multivariat-justerede model fandt vi at risikoen for mortalitet var statistik signifikant for individer med Q-takker med kun patologisk bredde i afledning II [hazard ratio (HR): 1,48, konfidensinterval (95% CI): 1.08-2.02], og i afledning aVF (HR: 1.17, CI: 1,05-1,29), og kun patologisk længde (HR: 1,36, CI: 1,20-1,55 og HR: 1,52, CI: 1,35-1,71) når disse er tilstede i afledning II og aVF hhv. Q-tak med kun patologisk bredde i afledning III var også associeret med øget mortalitet (HR: 2,52, CI: 1,44-4,42).

Konklusion Denne undersøgelse viste, at tilstedeværelse af Q-takker med kun patologisk bredde (≥ 30 ms), eller kun patologisk længde (≥100 mV) i en inferior afledning (II eller aVF) ledsaget med tilstedeværelse af Q-takker med patologisk længde og bredde samtidig i en anden inferior afledning var associeret med øget mortalitet.

Introduction

The inferior wall of the left ventricle is a common site for myocardial infarction (MI) and represent up to 40-50 % of all myocardial infarctions[1]. The majority of previous studies suggest that inferior MI is associated with a better prognosis than anterior wall MI. However, about 50% of patients with inferior MI will face post MI complications like heart block, which will change their otherwise better prognosis[1–3].

Traditionally the inferior MI was defined as follow: Q waves with duration of 40 msec or more and Q/R ratio of 0.25 or more in leads II, III or aVF[4]. Several studies suggest that when Q waves are present in two or more leads of the same group, the accuracy in prediction of prior MI will be higher than when present in isolated leads[5,6].

It has been assumed that the more leads showing a Q wave and the greater the size of the Q wave, i.e. deeper amplitude and wider duration, the higher probability of prior MI and the larger the infarction area is[7,8]. Yet, studies that examine the different aspects of QRS-complex and their correlation with MI burden, do not address if Q wave morphology i.e. duration and amplitude solely can act as a prognostic marker for the previous MI[9]. Therefor further knowledge about the Q wave morphology will benefit better estimates of Q wave´s prognostic value.

Different criteria have been used to define prior MI by using pathologic Q wave, and the newest one is the Third Universal Definition of Myocardial infarction, which have been developed in 2000 as a consensus between American and European guidelines. These criteria have been redefined several times, most recent in 2012. Q waves in any two leads of the inferior lead group (II, III and aVF) with a duration ≥30msec and amplitude ≥ 100mV or QS complexes are considered as pathologic following the new criteria for prior inferior wall MI[10].

Examining the current criteria for the inferior leads and testing the significance of the Q wave´s

duration and amplitude will contribute to a more accurate reading of the ECG and eventually better diagnosing of MI.

In the current study, we aim to investigate the relation between Q wave´s duration and amplitude in inferior leads and the prognosis when having abnormal Q wave´s morphology in one lead and at the same time having Q waves in another inferior lead following the Third Universal Definition of Myocardial infarction.

Methods

Databases

Copenhagen General Practitioners' Laboratory is a facility to which most general practitioners in the greater region of Copenhagen, Denmark, refer their patients for clinical tests such as biochemistry and ECG recordings. Those ECGs were recorded digitally and processed by using the Marquette 12 SL algorithm, version 21[11–13]. ECGs that were recorded between 2001-2011 were used along with other Danish administrative registries to perform our study, by use of the personal and unique identification number allocated to all individuals with permanent residence in Denmark. The Danish National Population Registry was utilized to gather information about the date of birth and sex. Date of death and causes of death were collected from the Danish Register of Causes of Death. Information about diagnoses was gathered from the Danish National Patient Register, which contains data about hospitalizations, outpatient visits since 1978. Both the 8th and 10th revisions of the International Classification of Disease have been used.

Study population

This study population included all individuals who have taken ECGs at Copenhagen General Practitioners' Laboratory between 2001 and 2011. Individuals were censored on December 31, 2013, or after 5 five years follow-up. The primary outcome of this study was all-cause mortality. In

the Danish Register of Causes if Death, information about cardiovascular mortality, in general, were available and it was chosen as the secondary outcome of the study.

The aim of this study was to examine each inferior lead alone (II, III, aVF) when Q waves appeared in the lead of interest and at the same time in one other inferior lead. To make this possible, three different study populations were defined. The study population for examining Q waves in lead II contained ECGs with pathologic Q waves either in lead III or aVF. Population for studying lead III included ECGs with pathologic Q waves either in lead II or aVF. The last population for aVF comprised ECGs with pathologic Q waves in either lead II or III. To ensure that Q waves were not present at the same time in the two other inferior leads than the lead of interest, in each study population, ECGs with Q waves present in the two other leads besides the lead of interest were excluded. The purpose of the exclusion was to ensure that the estimation of mortality was only made from the lead of interest plus one additional contiguous inferior lead.

Electrocardiography

The 12 SL algorithm contains statements coded uniquely for each finding in the ECG such as a sinus rhythm or atrial fibrillation[11]. Some findings may affect the correct reading of ECG measurement and influence the Q wave size other than prior MI and should, therefore, be excluded. Consequently, ECGs with poor data quality, different degrees of AV block, atrial fibrillation, atrial flutter, different tachyarrhythmias, junctional rhythms, pacemaker spikes, premature ventricular or aberrantly conducted complexes, intraventricular conduction and pre-excitation like Wolff-Parkinson-White and ventricular pre-excitation WPW pattern type A, wide QRS rhythm (> 120 milliseconds (msec)), right bundle branch block (RBBB), left bundle branch block (LBBB), left anterior fascicular block and complete heart block were all excluded. ECGs, where information regarding sex or age of the patient was missing, were also excluded. If the patient had more than one ECG, only the first was considered in the analyses.

In addition, ECGs, where the age of the patient was less than 16 years, were excluded.

The Third Universal Definition criteria were used to identify Q waves consistent with prior myocardial infarction in the inferior leads (II, III, aVF). A Q wave is considered pathologic when the duration is ≥ 30 msec and amplitude ≥ 0.1 mV. Similarly appearance of a QS complex was considered pathologic[10]. A QS complex were defined as Q amplitude >0 and R amplitude=0.

Estimation of Q wave´s duration and amplitude contribution to the mortality risk for each population was achieved as follows: when studying lead III, either lead II or aVF, having a duration of ≥ 30 msec and amplitude ≥ 0.1 mV were considered as a pathologic Q wave. Q waves in lead III were split to when the amplitude was ≥ 0.1 mV alone and when the duration ≥ 30 msec alone and finally when both duration ≥ 30 msec and amplitude ≥ 0.1 mV were present simultaneously. In the same way, when studying lead II, lead III or aVF should have Q wave that have both pathologic duration and amplitude. The Q waves in lead II were split to Q waves with only pathologic duration and only pathologic amplitude and when both were pathologic at the same time. When examining Q waves in lead aVF, then Q waves with both pathologic duration and amplitude would be present either in lead II or III and the Q waves in aVF would be studied when there were only pathologic duration, only pathologic amplitude and when both were present at the same time.

Statistical analysis

Kaplan-Meier curves were plotted to demonstrate the cumulative mortality. Cox proportional hazard regression was used to calculate the hazard ratio (HR) for Q waves, with only pathologic duration or only pathologic amplitude or both, in lead II, III and aVF and all-cause mortality, the model were adjusted for age and gender. P value < 0.05 were seen as statistically significant. Cox proportional hazard regression assumptions: linearity and proportionality were checked and found valid. The analyzes were done by SAS version 9.4 (SAS Institute Inc., Cary, NC, USA) and R statistics (version 3.2.2, Development Core Team).

Ethics

Permission for using registry data for this study were obtained from Danish Data Protection Agency (GEH-2014-014/ I-Suite no: 02732), and the Danish laws do not demand approval from ethics committee to perform registry-based studies.

Results

Baseline characteristics

Of a total of 309,824 individuals that were not excluded, three study populations were identified after the different criteria for each group we investigated. A total of 8,129 individuals had pathologic Q waves in lead II or aVF and were used to investigate the duration and amplitude of Q waves in lead III. The reference group was those that did not fulfill any of the criteria for Q waves in lead III. The second group was for studying the duration and amplitude of Q waves in lead II and included totally 36,645 individuals. Finally a total of 42, 892 individuals were identified for investigating Q waves in lead aVF and those had Q waves either in lead II or III.

The demographics of the study populations are presented in Table 1. In the whole study population the average age was 53.9 years (interquartile range [IQR] 41.1-65.9 years). In the study population for Q waves in lead III 60.5% were female and 13.5% among them had a known MI diagnosis. While in the study population of lead II 52.0% were female and 4.5% had a previous MI. Finally, 50.3% were female of the population for Q waves in lead aVF and among them 6.2% had a history of MI.

		Total	Female	Male	Previous MI
Age – yrs. Median {IQR}		53.9 {41.1 to 65.9}	55.1 {41.4 to 68.4}	52.6 {40.8 to 63.3}	69.2 {59.6 to 78.2}
Lead III N=8,129					
	No pathological Q waves	1,710 (21.0)	1,092 (22.2)	618 (19.3)	153 (14.0)
	Duration ≥ 30 msec and amplitude <100 mV	84 (1.0)	60 (1.2)	24 (0.7)	3 (0.3)
	Duration < 30 msec and amplitude ≥100 mV	3 (0.0)	3 (0.1)	0 (0.0)	1 (0.1)
	Duration ≥ 30 msec and amplitude ≥100 mV	6,332 (77.9)	3,764 (76.5)	2,568 (80.0)	937 (85.6)
Lead II N=36,645					
	No pathological Q waves	33,111 (90.4)	17,458 (91.7)	15,653 (88.9)	1456 (84.4)
	Duration ≥ 30 msec and amplitude<100 mV	239 (0.7)	45 (0.2)	194 (1.1)	40 (0.7)
	Duration < 30 msec and amplitude ≥100 mV	3,000 (8.2)	1,463 (7.7)	1,537 (8.7)	161(9.3)
	Duration ≥ 30 msec and amplitude ≥100 mV	295 (0.8)	73 (0.4)	222 (1.3)	68 (3.9)
Lead aVF N=42, 892					
	No pathological Q waves	27,145 (63.3)	15,194 (70.4)	11,951 (56.1)	1060 (39.9)
	Duration ≥ 30 msec and amplitude <100 mV	4,697 (11.0)	1,689 (7.8)	3,008 (14.1)	308 (11.6)
	Duration < 30 msec and amplitude ≥100 mV	3,289 (7.7)	1,639 (7.6)	1,650 (7.7)	203 (7.6)
	Duration ≥ 30 msec and amplitude ≥100 mV	7,761 (18.1)	3,066 (14.2)	4,695 (22.0)	1,085 (40.9)

Table 1 Baseline Characteristics of the Study Population. Median and inter quartile range [IQR] presented for age. The number of individuals and percentage presented for the different groups. MI= Myocardial infarction.

Univariate analysis of survival

The outcomes (all-cause and cardiovascular mortality) after the follow-up period of five years are shown in Table 2 and the Kaplan-Meier survival curves are presented in Figure1-3. All-cause mortality in the three populations were highest in the group with Q waves in lead III with 15.7% (N=1,278) in comparison to lead II population 9.1% (N=3,324) and lead aVF population 10.2% (N=4,375).

Death of cardiovascular cause were responsible for about the half of all death in all three populations, 53.0% (N=677), 46.8% (N=1,555) and 48.4% (N=2,119) in the population for Q waves in lead III, II and aVF, respectively.

There was only 3 individuals with isolated pathologic amplitude in lead III at the same time as other Q waves in either in lead II or in lead aVF, therefor we have chosen to exclude this group from our analysis.

		All-cause mortality	Cardiovascular mortality
Lead III N=8,129			
	No pathological Q waves	204 (16.0%)	98 (14.5)
	Duration ≥ 30 msec and amplitude <100 mV	13 (1.0)	5 (0.7)
	Duration < 30 msec and amplitude ≥100 mV	0 (0.0)	0 (0.0)
	Duration ≥ 30 msec and amplitude ≥100 mV	1061 (83.0)	574 (84.8)
Lead II N=36,645			
	No pathological Q waves	2,956 (88.9)	1,355 (87.1)
	Duration ≥ 30 msec and amplitude <100 mV	40 (1.2)	23 (1.5)
	Duration < 30 msec and amplitude ≥100 mV	261 (7.9)	139 (8.9)
	Duration ≥ 30 msec and amplitude ≥100 mV	67 (2.0)	38 (2.4)
Lead aVF N= 42, 892			
	No pathological Q waves	2,363 (54.0)	1,080 (51.0)
	Duration ≥ 30 msec and amplitude <100 mV	450 (10.3)	219 (10.3)
	Duration < 30 msec and amplitude ≥100 mV	324 (7.4)	164 (7.7)
	Duration ≥ 30 msec and amplitude ≥100 mV	1,238 (28.3)	656 (31.0)

Table 2 Outcome from both the primary (all-cause mortality) and secondary (cardiovascular mortality) endpoints. Data presented as a number of death and percentage.

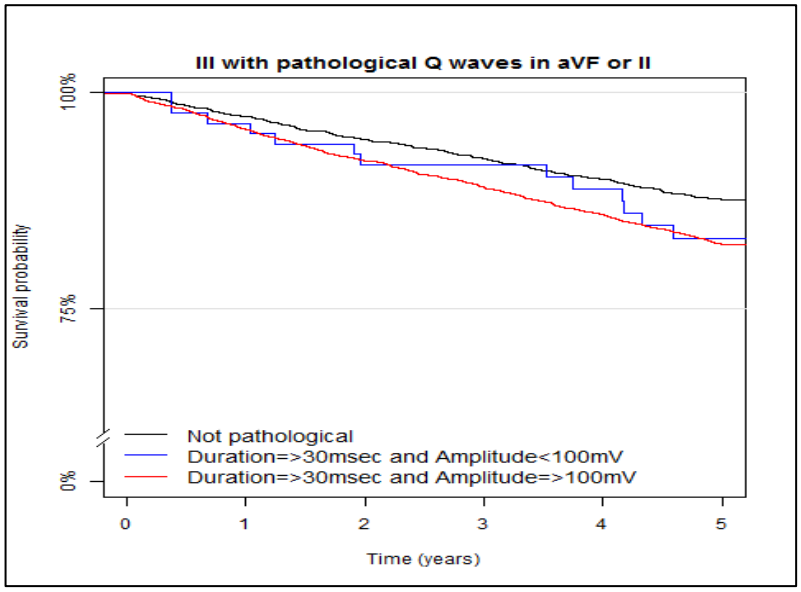

Figure 1 Kaplan-Meier cumulative survival analysis for the different combination of Q wave morphology in lead III present at the same time with pathologic Q waves in lead aVF or II. The Not pathological Q waves (Normal duration and amplitude in lead III) used as a reference.

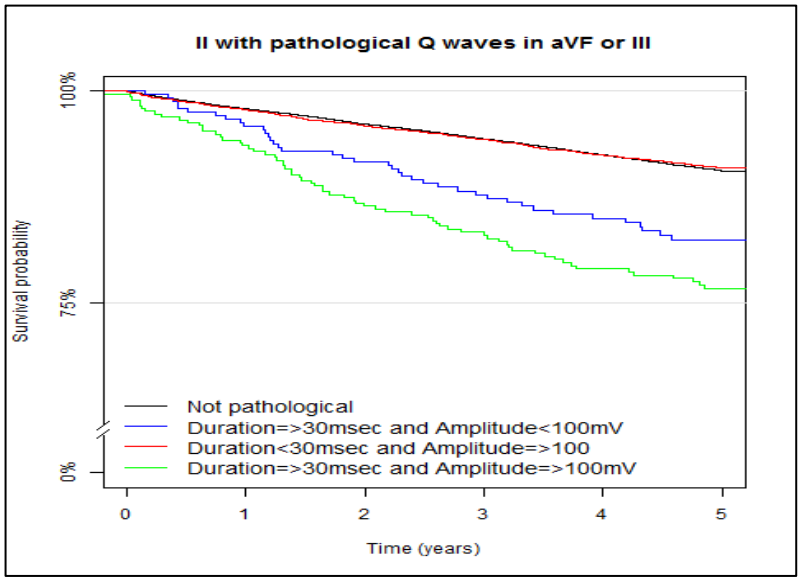

Figure 2 Kaplan-Meier cumulative survival analyzes for the different combination of Q wave morphology in lead II present at the same time with pathologic Q waves in lead III or aVF. The Not pathological Q waves (Normal duration and amplitude in lead II) used as a reference.

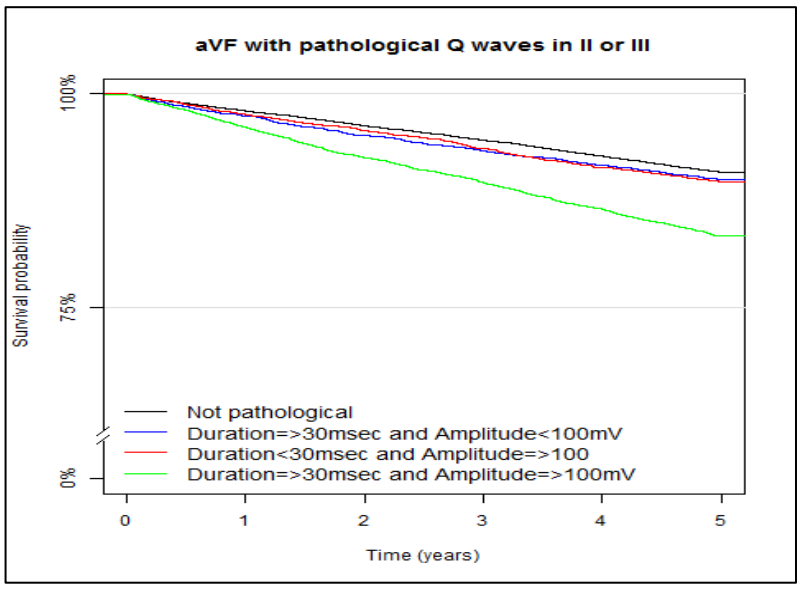

Figure 3 Kaplan-Meier cumulative survival analyzes for the different combination of Q wave morphology in lead aVF present at the same time with pathologic Q waves in lead II or III. The Not pathological Q waves (Normal duration and amplitude in lead aVF) used as a reference.

Multivariate analysis of survival

The results of the multivariate analyzes for all-cause mortality are presented in Figure 4. Individuals in each study population that did not have any of the inclusions criteria were used as the reference. The model was adjusted for age and gender.

The study showed that if the individual have Q waves (both duration ≥30 msec and amplitude ≥100 mV) in one inferior lead and at the same time have Q waves with only pathologic duration ≥30 msec in another inferior lead, then the mortality risk was significantly increased. This pattern was observed in the three combinations for all inferior leads III, II and, aVF. The mortality risk was highest for Q waves with pathologic duration in lead III (HR: 2.52, 95% CI: 1.44-4.42).

In the study population for Q waves in lead II and aVF, there were adequate individuals to estimate the mortality risk for when the individual only have a pathologic amplitude (≥100mV). We found also a significant increase in the mortality risk.

We tested also the mortality risk for when an individual fully meet the Third Universal Myocardial Infarction criteria for inferior prior MI i.e. having Q waves with both pathologic duration and amplitude in two leads at the same time and found that all the inferior leads

accompanied with another lead had significantly high mortality in comparison with the reference. Q waves in lead II and one of the other two leads (III or aVF) were associated with the highest risk (HR: 1.81, 95%CI: 1.42-2.30). We found also that male sex was associated with a higher mortality than female in all groups.

The multivariate analysis was also performed with cardiovascular mortality as the endpoint. The results did not differ considerably between the two endpoints. Almost all groups had marginally higher risk of cardiovascular death. There were only two groups that differed slightly from the results of all-cause mortality, which were the group of those with only pathologic duration in lead III (HR: 2.04, 95% CI: 0.83-5.01, P=0.120) and those with only pathologic duration in lead aVF (HR: 1.25, 95% CI: 1.08-1.44, P=0.120).

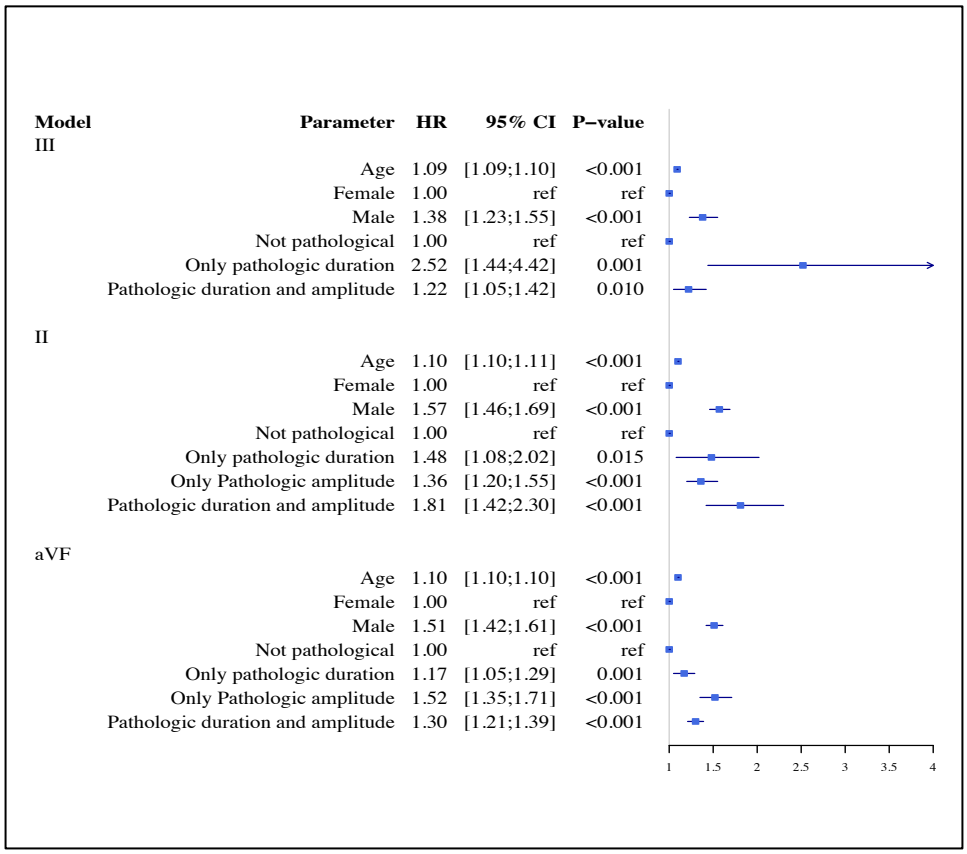

Figure 4 All-cause mortality in individuals with Q waves in the three study populations. Model for lead III N=8,129 contains ECGs with Q waves in lead II or aVF and Q waves in III with either pathologic duration or amplitude or both. Model for lead II N= 36, 645 includes ECGs with Q waves in lead III or aVF and Q waves in lead II of different morphology combinations. Finally model for lead aVF N= 42,892 includes ECGs with Q waves in lead II or III and Q waves of different morphology in lead aVF. All the models are adjusted for age and gender. The not pathological Q waves in the lead of interest were used as a reference.

Discussion

This study examined the risk of mortality in individuals with pathologic Q waves in the inferior leads (II, III, aVF). The pathologic Q waves were defined utilizing the Third Universal Myocardial Infarction criteria of prior myocardial infarction that include both a pathologic amplitude and duration in two leads simultaneously. Further, we studied the risk of mortality when only duration and only amplitude of a Q wave were pathologic. When investigating either amplitude or duration we demanded coexistence of another inferior lead with pathologic Q waves defined after the Third Universal Myocardial Infarction criteria.

The main results of our study showed that if the Q wave in inferior leads was partly pathologic then it was associated with an increase in all-cause and cardiovascular mortality when present with another lead that fully meet the Third Universal Myocardial Infarction criteria. This applied to Q waves in lead II and aVF. Due to the sample size (N=3) for isolated pathologic amplitude in lead III, we were not able to find the same pattern for lead III as for lead II and aVF.

Previous studies

In a study from 2012, Godsk et al.[2] found that larger Q waves defined according to the Minnesota Code Classification system (MC) most stringent criteria, resulted in worse prognosis compared with small Q waves, but that even small Q waves were responsible for increased risk for mortality. The small Q waves were also defined after the same system, but with less stringent criteria. Their findings support the suggestion that the size of a Q wave can describe the extent of myocardial damage after an infarct and consequently the prognosis.

The most stringent criteria in Minnesota Code criteria requires that the Q wave ≥40 msec or ≥ 30 msec if also Q/R <1/3, expect in lead III or aVF where the Q wave should be ≥ 50 msec to be pathologic, while the less stringent criteria require smaller durations i.e. ≥30 msec and Q/R <1/3

and >20 msec and Q/R <1/5[14]. Godsk study[2] applied to our findings that Q waves ≥30 msec present with another pathologic lead were associated with higher mortality.

Kochav et al. [9] studied 152 patients with acute MI if ECG parameters can describe the size of MI in left ventricle by using serial ECG records and delayed enhancement cardiac magnetic resonance (DE-CMR) and found that patients with pathologic Q waves have larger infarcts when controlled by DE-CMR or enzymes. Furthermore, they found that early post-acute MI that the total Q wave amplitude independently associated with infarct size while the total Q wave duration did not. But when they investigated the follow-up ECG about 29 days after the MI, they found that Q wave duration independently associated with the size of infarct, whereas Q wave amplitude did not correlate with the size. The authors suggest that the reason for this was that when the infarct area shrinks with time, it may reduce the amplitude and in a lesser degree also the duration. In our study, we found both that the amplitude and the duration respectively were associated with higher mortality on the long-term.

In other study made by Roark et al.[15] that included 31 patients, evaluating a previously developed scoring system, based on QRS complex of ECG for estimation the size of MI, correlated to autopsy findings. Those patients had MI in the inferior third of the left ventricle. In this study, the authors mean that duration of Q wave that is 30 msec rather than 40 msec in lead aVF was a sensitive indicator of infarction in the inferior third of left ventricle. They found that 90% of the infarcts could be recognized by a Q wave with a duration ≥ 30 msec in lead aVF. Other studies also suggested that Q waves in an isolated aVF were as sensitive as when Q waves were present in leads II, III, and aVF[15]. In comparison with our study we did not investigate the isolated aVF but in combination with II or III, it did show that pathologic duration or amplitude or both do increase the mortality risk. Also, our findings did apply to that duration of ≥ 30 msec do predict higher mortality.

Conversely, Bao et al.[16] found that as Q wave duration increased, the mortality rose in a 90-day follow-up in patients with ST-elevation MI (STEMI). They also found that in individuals with inferior MI, the relation between the Q wave duration and mortality stayed flat until a duration ≥40 msec was reached, and 90-day mortality in those with Q waves < 40 msec were 2.3% while in those ≥40 msec was 5.7%. Because of those findings they did suggest that in inferior MI that the threshold for Q wave duration in the Third Universal Myocardial Infarction criteria, should be augmented to ≥40 msec to provide better prognostic value. In our study, we did find that either Q wave with duration ≥ 30 msec or amplitude ≥0.1mV simultaneously with another lead in the inferior group do increase the risk for mortality. Our and Bao et al.[16] findings suggest that the demands to define a pathologic Q wave in the inferior leads by the Third Universal Definition of Myocardial infarction criteria need further investigation to reach a better prognostic value.

Conclusions

This study showed that presence of Q wave with only pathologic duration ≥ 30 msec or only pathologic amplitude ≥ 100 mV in one inferior lead (II or aVF) accompanied with a presence of Q wave with amplitude ≥ 100 mV and duration ≥ 30 msec in another inferior lead were associated with increased mortality. The present findings indicated that the current criterion for pathologic Q waves´ duration and amplitude in inferior leads might need further investigation.

Clinical implications

Our findings show that presence of Q waves with pathologic amplitude and duration in one lead and simultaneously with either pathologic duration or and amplitude in another lead increased the risk of death. Those results do not apply fully to the Third Definition of Myocardial Infarction criteria, and normally patients with such findings are found to be normal. Our findings indicate a need for

further investigation of the Third Universal Definition of Myocardial infarction and validation of the morphology of a pathologic Q wave in the inferior leads. This will benefit physician in diagnosing patients with prior MI and eventually improve survival, but also help in epidemiologic studies that are based on ECG findings.

Study limitations

Our study is a historical retrospective, and as other epidemiologic studies, there was no possibility to investigate the biologic background to our findings. Although our study population is large and we believe it is fairly representative of the background population. Yet, it is worth naming that the ECGs were recorded by a referral of general practitioner and those were taken obviously for a reason, therefore, it may lead to some selection bias. However, all factors that were considered to be confounders were included in our multivariable analysis. Danish registries were used to bring data about diagnoses and mortality, and the diagnose used in our study i.e. MI have been validated[17], which can be seen as a strength of the study.

Prospective

Next we will investigate the eventual interaction between the Q waves and MI by analyzing individuals with Q waves with and without a known MI diagnosis.

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