Sex-specific Prevalence of coronary heart disease among Tehranian adult population across different glycemic status: Tehran Lipid and Glucose Study, 2008-2011

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Abstract

Background: Coronary heart disease (CHD) is one of the most common causes of deaths and alarmingly Iranian populations had a high rank of CHD worldwide**.** The object of the current study is to assess the prevalence of CHD, including clinical CHD and silent myocardial infarction (MI) across different glycemic categories.

Methods: This study was conducted on 7,368 Tehranian participants (Men=3312), aged ≥ 30 years from 2008 to 2011. Clinical CHD was defined as hospital records which approved by an outcome committee. Using Minnesota Code ECG classifications, silent MI was defined by appearance of major abnormal Q/QS waves (MC 1.1 or MC 1.2), or minor abnormal Q/QS waves (MC 1.3) plus major abnormal ST-T (MC 4.1, MC 4.2, MC 5.1 or MC 5.2.)

Results: Among the total population, the age-standardized prevalence of CHD was 12.82% [confidence interval (CI) 95%:12.11-13.53]. The age-standardized prevalences of total CHD, clinical CHD and silent MI were 13.42% (12.33-14.5), 9.69% (8.81-10.56) and 3.73% (3.04-4.43) for men and were 12.70% (11.73-13.66), 8.56% (7.79-9.33) and 4.14% (3.50-4.78) for women, respectively. Moreover, 17.67% (13.90-21.44) of the male and 18.94% (14.59-23.30) of the female diabetic population had CHD. Specifically, the prevalence of total CHD among known and newly diagnosed diabetic populations and those with combined impaired fasting glucose (IFG) and impaired glucose tolerance (IGT) were 19.53% (15.22-23.84), 16.12% (12.29-19.94) and 16.84% (11.85-21.83), respectively. Compared to other glycemic categories, the prevalence of silent MI was higher among those with combined IFG and IGT (6.80 (2.84-10.76)), although it didn't reach a significant level.

Conclusion: The high prevalence of CHD, especially among those suffering from both IFG and IGT and diabetic populations, necessitates urgent implementation behavioral interventions among Tehranian populations, evidence based on guidelines for clinical management of diabetic patients.

Background

Coronary heart disease (CHD) is one of the most common causes of deaths worldwide[1–3] and its global fatality rate increased from 7.3 million in 2007 to 8.93 million deaths in 2017[1]. Among residents of Tehran, as a metropolitan city, we previously reported that the overall prevalence of CHD was 21.8% (22.3% among women and 18.8% among men) in 1991–2001[4]. Also the incidence rate of CHD was 10.5 and 6.1 per 1000 person-years among males and females, respectively[5]. Importantly, over 40% of mortality among Tehranian adults, aged ≥ 30 years, is attributed to cardiovascular disease (CVD)[6].

On the other hand, type 2 diabetes mellitus (T2DM) is known to be the major leading factor for CHD and its mortality[7, 8]. Based on national studies in 2011, about 11.4% and 14.6% of Iranian adults had diabetes mellitus (DM) and impaired fasting glucose (IFG), respectively. Furthermore, there was alarming increase of 35.1% in the prevalence of DM from 2005 to 2011[9]. A prediabetes tsunami, which included both impaired glucose tolerance (IGT) and IFG, was also reported among an Iranian population, with ≥

4% of adult individuals developing prediabetes each year[10]. Besides the well-known association between diabetes and CVD, it was also shown that prediabetes status can lead to CHD and CVD[11, 12].

A large number of diabetic patients who live with CHD, have never had any symptoms attributable to clinical CHD (silent coronary artery disease(CAD))[13]. Among non-diabetic populations, the prevalence of silent CAD ranged from 9–57%, but was not as common as those reported among diabetic patients[14]. Furthermore, myocardial infarction (MI), as the most important part of CAD, can occur asymptomatically (i.e. silent MI), constituting up to 50% of MI[15, 16], and usually diagnosed just by existence of an abnormal Q-wave in electrocardiogram (ECG) examinations[16]. Presence of silent MI increased the risk of CVD and all-cause mortality, and there is no significant difference in prognosis, between silent and clinical MI[16]. In the light of differences in ECG diagnostic criteria and study population, there is wide variability in prevalence estimation for silent MI[15]; however, the prevalence of silent MI may reach up to 6.4% in general populations[16], and being more prevalent among diabetic patients. Moreover, diabetes and prediabetes status could be a predictive and associated factors for silent MI[16, 17], but about prediabetes, the relation is less clear and necessitates further studies[18].

The aim of the current study is to report the population based prevalence of clinical CHD and silent MI among Iranian adults, aged ≥ 30 years, in a general population, according to their glycemic status categories in phase IV (2008–2011) of oldest cohort in the Middle East and North Africa (MENA) region, the Tehran Lipid and Glucose Study (TLGS)[19].

Methods

Study design and population study

This study was performed within the framework of TLGS, which is an ongoing community-based cohort study being conducted on a representative sample of citizens of Tehran. TLGS aims at determining the prevalence and incidence of non-communicable diseases and their risk factors and also aimed to prevent them by developing healthier life styles. Further details for TLGS have been described before[19]. Briefly, after the first baseline examination (1999–2001), 3 follow up phases were performed until 2011 at three-year intervals. For this study, 8,400 individuals aged ≥ 30 years, participants of phase IV of TLGS (2008–2011), were enrolled in this study. Exclusions included subjects with missing data on fasting plasma glucose (FPG) or 2-hour post challenge plasma glucose (2 h-PCPG), who have never taken any glucose lowering medications (n = 452) and those with missing ECG data of (n = 580). Finally, 7,368 participants remained for analysis for the current study.

Clinical and laboratory measurements

Using structured questionnaires, a trained nurse collected data which included demographic data, past medical history, drug history, family history of CVD, education and smoking status and levels of physical activity. Physical activity level was assessed by the Modifiable Activity Questionnaire (MAQ), which assessed all three type of activities including leisure time, job, and household activities in the past

year[20]. Details of anthropometric and blood pressure (BP) measurements have been published previously[20]. Body mass index (BMI) was calculated as weight divided by the square of the height (kg/m^2). After 12–14 hours overnight fasting, blood samples were collected between 07:00 AM and 09:00 AM and analyzed the same day. Except for those who had on glucose lowering medication, a standard oral glucose tolerance test with 75 gr glucose was done for all participants. More details of laboratory measurements have been published elsewhere[20]. Using a PC-ECG 1200 machine, two trained nurses recorded 12-lead resting ECGs for all participants, based on the standard protocol of the School of Public Health, University of Minnesota[21]; based on the codes provided by University of Minnesota[22], two trained physicians coded ECGs individually and to guarantee quality, a third trained physician, recoded 10% of ECGs randomly and rechecked all data.

Definition of terms

Participants were categorized into different groups as follows: Normal glucose tolerance (NGT), FPG < 5.6 and 2 h-PCPG < 7.7 mmol/l; isolated impaired fasting glucose (iIFG), 5.6 ≤ FPG ≤ 7 and 2 h-PCPG < 7.7 mmol/l; isolated impaired glucose tolerance (iIGT), 7.7 ≤ 2 h-PG ≤ 11.1 and FPG < 5.6 mmol/l; combined IFG and IGT (IFG/IGT), 5.6 ≤ FPG ≤ 7 and 7.7 ≤ 2 h-PCPG ≤ 11.1 mmol/l[23]. Moreover, in the present study, prediabetes status was defined as the presence of IFG or IGT. Finally, newly diagnosed diabetes (NDM) was defined as FPG ≥ 7.0 or 2 h-PCPG ≥ 11.1 mmol/l among those not on glucose lowering medications and known diabetes (KDM) as subjects with history of taking any glucose lowering medication. Smoking status was defined as current, past and never smoker. Education levels were classified as < 6 years (reference group), 6–12 years, and > 12 years. Low physical activity (inactive) was defined as not achieving a minimum score of 600 MET (metabolic equivalent task)-minutes per week[24]. Positive family history of premature CVD included any history of CHD/stroke in a male first-degree relative aged < 55 years or female first-degree relative aged < 65 years.

Definition of clinical CHD and silent MI

Details of the collection of outcome data have been reported elsewhere[20]. To summarize, each individual was under continuous surveillance for any medical outcome leading to hospitalization. As a part of the cohort data collection, a trained nurse called all participants annually and recorded any medical events experienced, during the last year. Any event reported was followed-up by a home visit and collection of medical data from hospital by a trained physician. Collected data were evaluated by a consulting committee, the outcome committee, included a principal investigator, an internist, an endocrinologist, a cardiologist, an epidemiologist and the physician who collected the outcome data and specific outcomes. Every confirmed event was considered as a non-communicable disease outcome based on ICD-10 criteria[20, 25]. In this study, clinical CHD was defined as ICD-10 rubric I20-I25; silent MI as ECG evidence of new MI without clinical CHD. ECG evidence of MI was defined by appearance of Minnesota Code ECG classifications as a major Q/QS wave abnormality (MC 1.1 or MC 1.2), or minor Q/QS waves abnormality (MC 1.3) plus major ST-T abnormality (MC 4.1, MC 4.2, MC 5.1 or MC 5.2)[26].

Finally, in the current study we also defined CHD as presence of clinical CHD or silent MI.

Statistics

Baseline characteristics are presented as means [standard deviations (SD)], median (interquartile range) and number (frequency) as appropriate. ANOVA and Kruskal-Wallis test, were used for comparison of means and medians, respectively and Chi squared tests were applied for comparison of frequencies.

Crude prevalence estimates of CHD are reported, according to all different glycemic categories, stratified for clinical CHD and silent MI. Regarding differences in the age distributions between the TLGS population from 2008 to 2011 and the Iranian census 2010 (supplementary Table 1), especially in the 30-39-year age group and those aged ≥ 70 years, age standardized prevalences were reported using the Iranian (Tehran province) census 2010.

Statistical analyses were done using STATA version 14. P-values < 0.05 were considered to be statistically significant.

Results

The study sample included 7,368 participants (men = 3,312) aged ≥ 30 years (mean age (SD) 50.2 (13.4) years). Sex-specific baseline characteristics across glycemic status categories are shown in Tables 1 and 2 for men and women, respectively. Generally, participants with NGT, in comparison with IFG/IGT and DM groups, had better cardiometabolic risk profiles, including age, BMI, triglycerides, systolic and diastolic BP for both sexes and total cholesterol, HDL-C and waist/hip ratio only among women. Furthermore, compared with the IFG/IGT and DM groups, participants with NGT had a better education status and lower prevalences of lipid lowering and anti-hypertensive drug usage among both sexes. Moreover, for most of above mentioned factors, participants with IFG/IGT were ranked between NGT and DM groups.

Overall, the age-standardized prevalence of CHD for Tehranian population was 12.82% [CI 95%: (12.11–13.53)] including 8.89% (8.32–9.45) and 3.94% (3.47–4.40) for clinical CHD and silent MI, respectively.

The age-standardized prevalence of total CHD across NGT, iIFG, iIGT, combined IFG and IGT, NDM and KDM groups is shown in Fig. 2. Accordingly, the highest prevalence of CHD was attributed to KDM. Moreover, among prediabetic groups, the prevalence of total CHD was more prominent in the combined IFG and IGT, than among isolated groups. The prevalence of clinical CHD and silent MI, across these 6 above-mentioned glycemic categories are also illustrated in Table 4. The age-standardized prevalences (95% CI) of clinical CHD were 7.47(6.64–8.30), 7.59(6.23–8.96), 7.77(5.37–10.18), 10.04(6.67–13.41), 10.25(8.18–12.33) and 16.06(12.68–19.43) among NGT, iIFG, iIGT, combined IFG and IGT, NDM and KDM groups, respectively. The corresponding values for silent MI among these groups were 3.62 (3.06–4.18), 3.68 (2.41–4.96), 2.61 (0.44–4.79), 6.80 (2.84–10.76), 5.87 (2.53–9.20), 3.47 (0.63–6.31) and 3.94 (3.47–4.40), respectively.

Discussion

In this population-based study (2008–2011) about 13% of urban Tehranian residents were found to have CHD, of whom about 9% and 4% had clinical CHD and silent MI respectively. Generally, in comparison with women, men had higher prevalence of total and clinical CHD. In addition, the prevalences of total CHD and silent MI were about 18% and 3% among male diabetic participants and were about 19% and 6% among their female counterparts, respectively. Despite, the higher prevalence of clinical CHD among diabetic participants, our findings did not show a higher prevalence of silent MI among diabetic participants compared to other glycemic categories in either gender. Finally, the NDM group and those suffering from both IFG and IGT showed similar high prevalences for total CHD (about 16%), which were higher than that of the NGT group.

In our previous study, using self-reported history of CHD and ECG-defined ischemia for defining CHD, a 15.1% prevalence of CHD was reported for Tehranian adults in 1999–2001[4]. The differences between the prior study and the current study might be attributable to the following factors. Firstly, in a previous of ours, the prevalence of silent ischemia was reported to be > 10% in both sexes, significantly higher than those reported in the current study. It should be noted that in our previous study, we included both probable and possible Minnesota coding of CHD, while in the current study, only definite Minnesota coding of MI was included as diagnostic criteria[26, 27]. Secondly, for history of CHD, it was considered positive, only when its hospital records were provided and then confirmed by the outcome committee. Hence despite a strong association between Rose angina and incident CHD events which reported among tehranian population[28], we did not include it in the clinical CHD definition. Hence, in the current study, considering the stringent criteria for definition of clinical CHD and silent MI led to underestimations for prevalence of CHD.

It is important to note that due to different diagnostic criteria for CHD definition and different study age-group categorization, comparing our results with other population-based studies is somewhat difficult. Abbasi et al. reported that among an Iranian population, aged over 20 years, the national prevalence of self-reported CHD was 5.3% (5.6% among urban residents) in 2011[29]; their values for prevalence of self-reported CHD were significantly lower than our reports. Moreover, in comparison with our results for silent MI, in a cross-sectional population-based study in southern Iran, Nabipour et al. reported that 1.4% of their participants (1.9% of men and 1% of women), aged 25–64 years were diagnosed to have ECGs with evidence of previous MI (Minnesota codes 1.1 and 1.2)[30]. Comparing our results with the two studies previously mentioned suggests that Tehranian residents rank high in the prevalence of CHD among Iranian populations.

Compared to developed countries, based on a national study, the American Heart Association reported the total CHD prevalence to be 6.7% among US adults, aged ≥ 20 years (7.4% for men and 6.2% for women) [31]. Furthermore, data from the Quebec Integrated Chronic Disease Surveillance System (QICDSS) indicated (□) that in 2012/2013, the crude prevalence of CHD was 9.4% among an adult Canadian population and (□) that from 2000/2001 to 2012/2013 the age-standardized prevalence had increased by 14%, despite having a slight decreasing trend since 2009/2010[32]. For the United Kingdom (UK), data from the Quality and Outcomes Framework (QOF) indicated that the prevalence of CHD remained

constant at about 3% in England and 4% in Scotland, Wales, and Northern Ireland between 2004/2005 and 2014/2015[33]. Among Asian countries, results of the China National Diabetes and Metabolic Disorders Study showed that the prevalences of self-reported CHD were 0.72% and 0.48% among male and female adult Chinese, respectively[34]. Also the prevalence of CHD from national studies varies from 2–4% in India[35]. Furthermore, in Saudi Arabia, as a Middle Eastern country, the age-adjusted prevalence of CHD was reported to be 5.9% among men and 4.4% among women, aged 30–70 years[36]. Generally, it seems that the estimated prevalence of CHD among Tehranian populations are alarmingly higher than corresponding figures in US[31], Canada[32], UK[33], China[34], India[35] and Saudi Arabia[36], an issue previously addressed in 2015 by Zhu et al.[37] As we reported previously, modifiable risk factors including diabetes, hypertension, smoking and dyslipidemia totally had population attributable fraction of 36.6% and 50.2% of CHD among male and female Tehranian populations, respectively[5]. Other reasons that might justify the high prevalence of CHD among Tehranian populations are related to impact of air pollution[38–40] and stress[41] both of which are common in Tehran.

Focusing on diabetes status, as reported by a national study in 2016, among Iranian diabetic patients, aged ≥ 18 years, the crude prevalences of clinical CHD were 25.1% for men and 23.2% for women[42]; the corresponding values were 27.88% and 25.66% among our diabetic population. It seems that similar to total papulations, among the Tehranian diabetic population, the prevalence of CHD was higher than that reported in nationwide studies. The age-standardized prevalences of clinical CHD were 4.43% and 4.76% among male and female Chinese patients with T2DM, respectively[43]. Moreover, the prevalence of CHD among Thai patients with diabetes was 3.54% in 2013[44]. Also among Swedish diabetic patients, aged 45–74 years, the crude prevalences of CHD were 24.9% for men and 18.0% for women, which were lower than our results, despite the fact that their population was older than ours[45]. In addition, a significant racial difference was reported in the prevalence of CHD between White and African diabetic patients in a hospital-based study[46]. It has been suggested that there is a racial susceptibility for CHD among diabetic patients, which could make Iranian diabetic patients more prone to developing CHD, compared to Asian, African and European ethnicities. Furthermore, although CVD risk factors among Iranian diabetic populations have been controlled to some degree, during recent years, most diabetic participants still have uncontrolled CVD risk factors[47] which could also have led to high prevalence of CHD among our diabetic population.

In the current study as expected the highest prevalence of CHD was shown among diabetic participants; however, we also found that the NDM group showed > 16% prevalence of CHD. We have previously reported that during a 7.6 years follow-up, Tehranian adults with NDM, especially in men, exhibited a CHD risk comparable to non-DM with a prior CHD[48]. Also, regarding prediabetes status, the prevalence of CHD become more prominent among participants with combined IFG and IGT, similar to NDM groups (Fig. 2). Based on angiographic data, among a non-diabetic population, it was reported that participants with combined IFG and IGT had higher prevalence of significant CHD and higher severity of disease; however, there were no significant differences among subjects with NGT, I-IFG, and I-IGT[49].

In the current study, we found that about 25% (4.48/18.18) of total CHD among the diabetic population was attributed to silent MI. Previous studies reported different prevalences of silent MI among diabetic populations, considering the different tools used to assess infarction, ranging from 3.9% (by ECG-criteria) to 37% (by dipyridamole thallium scintigraphy)[16]. We also observed a high prevalence of silent MI reach to 6.8% among combined the IFG and IGT population, although our findings did not support significant difference in the prevalence of silent MI across glycemic status categories, probably due to the limited number of silent MI in each group. Recently among an elderly population it was also shown that subjects with IFG had no increased risk of silent MI than those with NFG. Furthermore, Bhatt et al. found an independent association between Q-waves and the homeostasis model assessment of insulin resistance (HOMA-IR)[50]. To the best of our knowledge, no previous study has examined the prevalence of silent MI among strictly defined glucose tolerance groups, using both FPG and 2 h-PCPG criteria. However two studies showed participants with IGT[51] and IFG[52] had higher prevalence of silent MI than the NGT group. Some authors have speculated that prediabetes and diabetes status cause cardiovascular autonomic neuropathy which could imply some degree of cardiac pain suppression[52]. Therefore, in the light of this mechanism, diabetic and prediabetic populations are more prone to silent CHD and silent MI.

Strengths of this study include using standardized ECG procedures for defining stringent criteria of MI, definite documented CHD ascertained by an outcome committee and finally the determination of CHD prevalence among the whole population, based on glycemic categories, using the glucose challenge test.

Several limitations need to be acknowledged. First, our study shows an optimistic picture of CHD prevalence among our population since inclusion of subjects in an ongoing study can improve the level of attention paid to controlling their health risks (cohort effect). Therefore, the burden of CHD will be much higher in the context of the community. Second, this investigation was conducted among residents of Tehran as a metropolitan city and our results might not be generalizable to rural zones. Third, we have only examined non-fatal CHD, whereas the prevalence of CHD is much higher, by when considering the burden of fatal CHD. Fourth, because of our large population-based sample, it’s not practical and reasonable to apply some modalities including stress test, positron emission tomography and angiography for all participants to detect silent ischemia[53]. Finally, the sensitivity and specificity of ECG criteria for detecting previous MI is limited[27] and patients with acute non-Q-wave MI do not necessarily develop Q waves eventually[15]. Also some of the Q waves disappear after MI[26].

Conclusion

The alarmingly high prevalence of CHD, especially among those with combined IFG and IGT and diabetic populations, necessitates urgent behavioral intervention to be aimed at halting obesity tsunami[54] and physical inactivity[55] among Tehranian populations with following and implementing evidence-based guidelines and interventions for the clinical management of diabetic patients. Last, but not least, the impact of environmental and psychosocial factors on CHD in Tehranians should be investigated in future studies.

Declarations

Ethics approval and consent to participate

This study was approved by the Institutional Review Board (IRB) of the Research Institute for Endocrine Sciences (RIES), Shahid Beheshti University of Medical Sciences, and all participants provided written informed consent.

Consent for publication

Not applicable.

Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

Founding

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Author contributions

Study conception and design: S.S.M and F.H; Analysis and interpretation of data: M.H, D.P and F.H; Drafting of manuscript: S.S.M, H.G and F.H; Critical revision: S.S.M, A.G, H.G, F.A and F.H. All authors read and approved the final manuscript.

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Abbreviations

CHD: Coronary Heart Disease; CVD: Cardiovascular Disease; T2DM: Type 2 Diabetes Mellitus; DM: Diabetes Mellitus; IFG: Impaired Fasting Glucose; IGT: Impaired Glucose Tolerance; CAD: Coronary Artery Disease; MI: Myocardial Infarction; ECG: Electrocardiogram; MENA: Middle East and North Africa; TLGS: Tehran Lipid and Glucose Study; FPG: Fasting Plasma Glucose; 2h-PCPG: 2-hour Post Challenge Plasma Glucose; IRB: Institutional Review Board; RIES: Research Institute for Endocrine Sciences; MAQ: Modifiable Activity Questionnaire; BP: Blood Pressure; BMI: Body Mass Index; NGT: Normal Glucose Tolerance; iIFG: Isolated Impaired Fasting Glucose; iIGT: Isolated Impaired Glucose Tolerance; IFG/IGT: Combined IFG and IGT; NDM: Newly Diagnosed Diabetes; KDM: Known Diabetes Mellitus; SD: Standard deviations; QICDSS: Quebec Integrated Chronic Disease Surveillance System; UK: United Kingdom; QOF: Quality and Outcomes Framework; HOMA-IR: Homeostasis Model Assessment of Insulin Resistance (HOMA-IR)

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Tables

Table 1. Baseline characteristics of male participants across glycemic status categories: Tehran Lipid and Glucose Study (Phase IV: 2008-2011).

Baseline Characteristics Continuous variables	Number of valid Values	NFG and NGT	IFG/IGT	DM	P-value
Age (years)	3312	47.3(12.9)	53.9(13.9)	59.8(12.7)	<0.001
BMI (kg/)	3278	26.7(4)	27.9(4.1)	28.1(4.1)	<0.001
Waist/hip ratio	3278	1(0.1)	1(0.1)	1(0.1)	<0.001
Triglycerides* (mg/dL)	3312	134(97-188)	148(107-206)	153(111-209)	<0.001
Total cholesterol (mg/dL)	3312	189.6(36)	194.3(38.3)	185.5(45.6)	<0.001
HDL-C (mg/dL)	3308	42.6(9.2)	42.2(9.4)	41.9(9.5)	0.25
Systolic BP (mmHg)	3305	117.3(15.6)	124.1(18.5)	129.9(19.2)	<0.001
Diastolic BP (mmHg)	3305	79(10.6)	80.8(10.8)	82.5(11.2)	<0.001
Categorical variables					
Smoking status	3301				<0.001
Never smoker		1018(55.8)	568(57.8)	283(57.3)	
past smoker		334(18.3)	225(22.9)	126(25.5)	
Current smoker		473(25.9)	189(19.2)	85(17.2)	
Education status	3310				<0.001
< 6 years		283(15.5)	258(26.2)	189(38.3)	
6-12 years		999(54.6)	512(52)	228(46.2)	
>12 years		549(30)	215(21.8)	77(15.6)	
Low Physical activity	3112	709(40.2)	392(43.3)	180(40.7)	0.305
Family history of CVD	3312	72(3.9)	43(4.4)	25(5.1)	0.531
Lipid lowering drug use	3312	75(4.1)	96(9.7)	124(25.1)	<0.001
Anti-hypertensive drug use	3312	126(6.9)	124(12.6)	143(28.9)	<0.001

Data are shown as mean (SD) for continuous variables and % for categorical variables; p values are calculated by ANOVA and chi-squared tests, respectively. *Triglycerides are given as median (interquartile range) due to skewed distribution. Family history of CVD is defined as diagnosis of CVD in male or female first-degree blood relatives, aged < 55 years or aged < 65 years, respectively. N, number of valid values for each variable; NFG, normal fasting glucose; NGT, normal glucose tolerance; IFG/IGT, impaired fasting glucose or impaired glucose tolerance; NDM, newly diagnosed diabetes; KDM, known diabetes; BMI, body mass index; HDL-C, high density lipoprotein-cholesterol; BP, blood pressure; CVD, cardiovascular disease.

Table 2. Baseline characteristics of female participants across glycemic status categories: Tehran Lipid and Glucose Study (Phase IV: 2008-2011).

Baseline characteristics	Number of valid values	NFG and NGT	IFG/IGT	DM	P-value
Continuous variables					
Age (years)	4056	45.2(11.6)	53(12.1)	59.2(11)	<0.001
BMI (kg/)	4004	28.6(4.7)	31.4(18.5)	31.3(5.6)	<0.001
Waist/hip ratio	4004	0.9(0.1)	1(0.1)	1(0.1)	<0.001
Triglycerides* (mg/dL)	4055	107(78-149)	146(108-196)	172(126-242.5)	<0.001
Total cholesterol(mg/dL)	4055	191.4(36.3)	206.4(40.7)	201.9(49.4)	<0.001
HDL-C (mg/dL)	4054	52(11.4)	49.5(11.3)	47.9(10.8)	<0.001
Systolic BP (mmHg)	4047	112(16.3)	122.1(19.4)	130.9(22.7)	<0.001
Diastolic BP (mmHg)	4047	74.7(10.4)	78.3(10.9)	79.9(11.8)	<0.001
Categorical variables					
Smoking status	4046				0.46
Never smoker		2267(95.1)	938(95.3)	646(95.1)	
Past smoker		39(1.6)	17(1.7)	17(2.5)	
Current smoker		77(3.2)	29(2.9)	16(2.4)	
Education	4051				<0.001
< 6 years		549(23)	427(43.4)	423(62.3)	
6-12 years		1314(55)	465(47.2)	220(32.4)	
>12 years		524(22)	93(9.4)	36(5.3)	
Low physical activity	3985	592(24.9)	261(27.0)	254(39.3)	<0.001
Family history of CVD	4056	144(6)	75(7.6)	42(6.2)	0.225
Lipid lowering drug use	4056	122(5.1)	128(13.0)	265(38.9)	<0.001
Anti-hypertensive drug use	4056	190(8)	209(21.2)	316(46.3)	<0.001

Data are shown as mean (SD) for continuous variables and % for categorical variables; p values are calculated by ANOVA and chi-squared tests, respectively. *Triglycerides are given as median (interquartile range) due to skewed distribution. Family history of CVD is defined as diagnosis of CVD in male or female first-degree blood relatives, aged < 55 years or aged < 65 years, respectively. N, number of valid values for each variable; NFG, normal fasting glucose; NGT, normal glucose tolerance; IFG/IGT, impaired fasting glucose or impaired glucose tolerance; NDM, newly diagnosed diabetes; KDM, known diabetes; BMI, body mass index; HDL-C, high density lipoprotein-cholesterol; BP, blood pressure; CVD, cardiovascular disease.

Table 3. Prevalence of clinical CHD and silent CHD across glycemic status categories, by gender: Tehran Lipid and Glucose Study (Phase IV: 2008-2011)

			NFG and NGT	IFG/IGT	DM	Total
Male	Clinical CHD	Case/Total	154 /1831	134/986	138/495	426/3312
		Crude prevalence % (95% CI)	8.41 (7.14-9.68)	13.59 (11.45-15.73)	27.88 (23.92-31.83)	12.86 (11.72-14.00)
		Age-standardized prevalence† % (95% CI)	8.21(6.98-9.44)	8.92 (7.38-10.46)	14.85 (11.29-18.41)	9.69 (8.81-10.56)
	Silent MI	Case/Total	61/ 1831	39 /986	18/ 495	118/3312
		Crude prevalence % (95% CI)	3.33 (2.51-4.15)	3.96 (2.74-5.17)	3.64 (1.99-5.29)	3.56 (2.93-4.19)
		Age-standardized prevalence† % (95% CI)	3.35 (2.52-4.18)	4.54 (2.90-6.18)	2.82 (1.37-4.27)	3.73 (3.04-4.43)
Female	Clinical CHD	Case/Total	124/2388	99/986	175/682	398/4056
		Crude prevalence % (95% CI)	5.19 (4.30-6.08)	10.04 (8.16-11.92)	25.66 (22.38-28.94)	9.81 (8.90-10.73)
		Age-standardized prevalence† % (95% CI)	7.09 (5.96-8.22)	7.63 (6.02-9.24)	12.97 (10.97-14.97)	8.56 (7.79-9.33)
	Silent MI	Case/Total	94/2388	37/986	39/682	170/4056
		Crude prevalence % (95% CI)	3.94 (3.16-4.72)	3.75 (2.57-4.94)	5.72 (3.97-7.46)	4.19 (3.57-4.81)
		Age-standardized prevalence† % (95% CI)	3.83 (3.06-4.59)	3.55 (2.12-4.98)	5.98 (2.04-9.92)	4.14 (3.50-4.78)

Age-standardized prevalence is calculated based on †Iranian population distribution data from the National Consensus Bureau for Tehran province (2010). NFG, normal fasting glucose; NGT, normal glucose tolerance; IFG/IGT, impaired fasting glucose or impaired glucose tolerance; DM, diabetes mellitus; CHD, coronary heart disease; MI, myocardial infarction; CI, confidence interval.

Table 4. Sex-adjusted prevalence of clinical CHD and silent MI across glycemic status categories: Tehran Lipid and Glucose Study (Phase IV: 2008-2011)

		NFG and NGT	iIFG	iIGT	IFG and IGT	NDM	KDM	Total
Clinical CHD	Case/Total	278/4219	116/1122	48/397	69/453	88/436	225/741	824/7368
	Crude prevalence % (95% CI)	6.59 (5.84-7.34)	10.34 (8.56-12.12)	12.09 (8.88-15.30)	15.23 (11.92-18.54)	20.18 (16.41-23.96)	30.36 (27.05-33.68)	11.18 (10.46-11.90)
	Age-standardized prevalence[†]% (95% CI)	7.47 (6.64-8.30)	7.59 (6.23-8.96)	7.77 (5.37-10.18)	10.04 (6.67-13.41)	10.25 (8.18-12.33)	16.06 (12.68-19.43)	8.89 (8.32-9.45)
Silent MI	Case/Total	155/4219	41/1122	9/397	26/453	27/436	30/741	288/7368
	Crude prevalence % (95% CI)	3.67 (3.11-4.24)	3.65 (2.56-4.75)	2.27 (0.8-3.73)	5.74 (3.59-7.88)	6.19 (3.93-8.46)	4.05 (2.63-5.47)	3.91 (3.47-4.35)
	Age-standardized prevalence[†] % (95% CI)	3.62 (3.06-4.18)	3.68 (2.41-4.96)	2.61 (0.44-4.79)	6.80 (2.84-10.76)	5.87 (2.53-9.20)	3.47 (0.63-6.31)	3.94 (3.47-4.40)

Age-standardized prevalence is calculated based on [†]Iranian population distribution data from the National Consensus Bureau for Tehran province (2010). NFG, normal fasting glucose; NGT, normal glucose tolerance; iIFG, isolated impaired fasting glucose; iIGT, isolated impaired glucose tolerance; IFG and IGT, both impaired fasting glucose and impaired glucose tolerance; NDM, newly diagnosed diabetes; KDM, known diabetes. CHD, coronary heart disease; MI, myocardial infarction; CI, confidence interval.

Figures

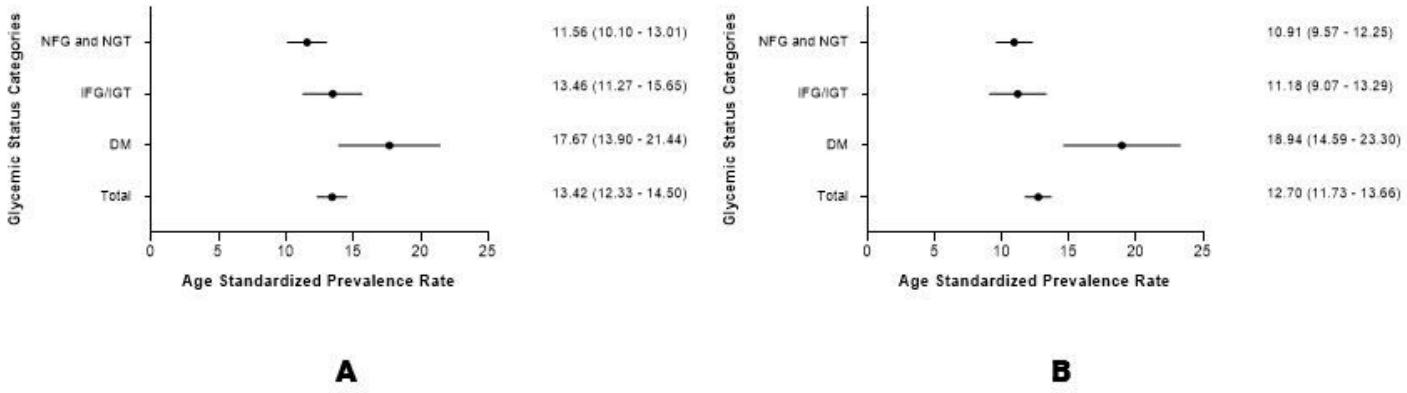

Figure 1

Prevalence of age-standardized CHD across 3 glycemic status categories (NFG, IFG/IGT and DM) among men (A) and women (B): Tehran Lipid and Glucose Study (Phase IV: 2008-2011). Age-standardized prevalence (95% confidence interval (CI)) is calculated based on Iranian population distribution data from the National Consensus Bureau for Tehran province (2010). NFG, normal fasting glucose; NGT, normal glucose tolerance; IFG/IGT, impaired fasting glucose or impaired glucose tolerance; DM, diabetes mellitus. CHD, coronary heart disease;

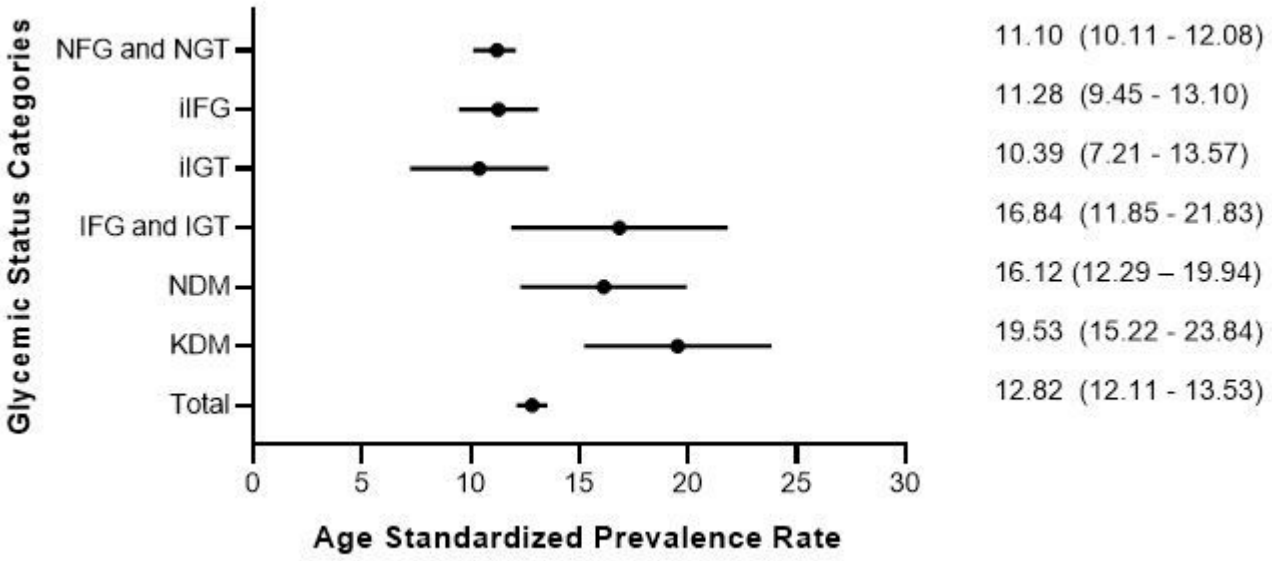

Figure 2

Prevalence of age-standardized CHD across 6 glycemic status categories (NFG, iIFG, iIGT, IFG/IGT, NDM and KDM) among whole population: Tehran Lipid and Glucose Study (Phase IV: 2008-2011). Age-standardized prevalence (95% confidence interval (CI)) is calculated based on Iranian population distribution data from the National Consensus Bureau for Tehran province (2010). NFG, normal fasting glucose; NGT, normal glucose tolerance; iIFG, isolated impaired fasting glucose; iIGT, isolated impaired glucose tolerance; IFG and IGT, both impaired fasting glucose and impaired glucose tolerance; NDM, newly diagnosed diabetes; KDM, known diabetes. CHD, coronary heart disease;

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