Risk factors of ISCHEMIC HEART DISEASE in females in Babylon

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Introduction Chronic heart disease is the leading cause of death in both men and women. It caused one of every six U.S. deaths in 2006; CHD mortality was 425,425, and myocardial infarction mortality was 141,462. Approximately every 25 seconds, an American will experience a coronary event, and approximately every minute a death will be attributed to a coronary event. Approximately every 34 seconds, an American will have an MI and 15 percent will die of it (1) . Because cardiovascular disease (CVD) is the most important cause of death in women in the United States, it is imperative that the main risk factors for CVD in women be identified and modified. The risk factors that have the strongest impact on the incidence of CVD in women are not necessarily the same as those for men. The risk for women increases at menopause, most likely because of the decrease in levels of circulating estrogen. The classic risk factor for CVD is altered lipid levels. In middle-aged women, elevated low-density lipoprotein cholesterol lev- els are somewhat less important relative to lowered levels of high-density lipoprotein cholesterol and ele□vated triglyceride levels as independent risk factors. The metabolic syndrome, which encompasses a range of conditions known to be CVD risk factors, also has a greater impact on the incidence of CVD in women than in men. Various emerging risk factors appear to be important indicators for vascular disease in women, including C-reactive protein, homocysteine, and lipopro□tein levels. Many of these risk factors are affected by hormone replacement therapy, which may diminish CVD risk in postmenopausal women. Because of the complex origin of CVD, it is important to target the full array of risk factors for modification, rather than focusing on a single factor or treatment to the exclusion of other important markers (2) . Cardiovascular disease (CVD) has been perceived as a disease mainly affecting men. It is now be- coming more accepted that CVD is also the leading cause of death in women. Over a lifespan, approxi- mately the same proportion of the female population as the male population dies of complications resulting from CVD in the United States (3) . However, there are sex-specific differences, both in the way that CVD presents clinically and in the risk factors that predis- pose individuals to the underlying atherosclerosis. In women, the age-related increase in CVD tends to lag behind that of men by approximately 10 years; it increases after menopause, leading to the idea that endogenous estrogen is protective in premenopausal women.Smoking is a prominent risk factor for both sexes. Other significant factors are age, hypertension, family history, obesity, diabetes, and inactivity. Smoking is not as strong a risk indicator for women as it is for men, whereas other factors, such as low levels of high-density lipoprotein cholesterol (HDL-C), ele- vated triglyceride levels, and diabetes, have more of an impact on the risk picture for women than for men. The metabolic syndrome (also known as insulin resis- tance or syndrome X) is highly associated with CVD risk in women, especially in middle age. Emerging risk factors, such as C-reactive protein, homocysteine, and lipoprotein(a) also have recently been found to play a role in coronary artery disease, particularly in postmenopausal women. The aim of this article is to review established and emerging risk factors for cor- onary artery

disease in women, and to discuss how these markers are affected by estrogen replacement therapy (ERT) and hormone replacement therapy (HRT) (4) . Methodology Study was done in Babylon ،It was done between first of April 2022and first of May 2022. A structured questionnaire was designed and translated in Arabic language. This study was conducted in Merjan teaching hospital and in Imam Al□Sadiq teaching hospital . We have collected 70 sample. Included patients any female has ischemic heart disease at any age group. The objective of the study were explained for patient before asking them. The questionnaire consisted of 28 questions. The questionnaire included nine important domains: socio□demographics information, medical history, Type of IHD, history of coronary artery, history of coronary intervention, Gynecological history, family history, investigations and also about drug history. Socio-demographics contain generally questions and characteristics about the patient in addition to BMI calculation And there are questions about history of coronary artery disease with interventions, gynecological history and the family history. The last domain contain questions about the investigation of the condition of patient. Smoking state Frequency Percent No smoking 51 72.9 Smoking 19 27.1 Total 70 100.0 BMI range Frequency Percent 18.5 - 24.9 7 10.0 25 - 29.9 27 38.6 > 30 36 51.4 Total 70 100.0 Results : A total of 70 sample Of ischemic heart disease in femal were included. Table 1 : About (51.4%) of women with obesity that they have BMI above 30 , while there about (38.6 %) were overweight, and finally about (10%) were within the normal range of BMI . Table 2 : we found that (72.9%) of women non -smokers and(27.1%) were still smoking , not a significant result but has an association between smoking and ischemic heart disease

Discussion of the Risk factors Obesity Obesity is a complex metabolic disorder that afflicts 35% of the adult population in the United States. As an important risk factor for ischemic heart disease (IHD) and its metabolic precedents, it has become one of the most serious health problems in many parts of the world (6) . Currently,we found 51.4% of females that Body mass index equal or more than 30 , while 38.6% have 25-29.9 , and 10% was within normal range 18.5-24.9 . Age Once a woman reaches the age of 50 to 52 (about the age of natural menopause), the risk of heart disease increases dramatically . Smoking : The prevalence of current smoking was 80.8% in male cases and 53.8% in male controls and 59.5% of female cases were smokers compared with 35.8% of controls. No interaction was found between current smoking and gender on myocardial infarction risk (P = 0.401). A dose-effect response was present, the odds favoring myocardial infarction reaching an eight-fold increase for those who smoked >25 cigarettes/day compared with never smokers (5). High blood pressure High blood pressure can lead to coronary artery diseases because it adds force to the artery walls. Over time, this can damage these blood vessels and lead to more plaque buildup. The narrowed artery limits or blocks the flow of blood to the heart muscle, which means it might not get enough oxygen. In time, this may cause chest pain (angina). Plaque can also break off or damage a blood vessel, possibly leading to a heart attack or stroke. Family history of cardiovascular disease If your parents have/had cardiovascular disease (especially if they were diagnosed before age 50), you have an increased risk of developing it. Ask your doctor when it’s appropriate for you to start screenings for cardiovascular disease so it can be detected and treated early . Diabetes Diabetes mellitus is associated with an increased risk of cardiovascular death and a higher incidence of cardiovascular diseases including coronary artery diseases (CAD), congestive heart failure (CHF) , and atrial fibrillation . The mechanisms underlying the association between glucose homeostasis and each of myocardial dysfunction and atrial fibrillation remain mostly

speculative. In contrast the relationship between abnormal glucose homeostasis and coronary artery disease has been the center of extensive basic science, epidemiological, and therapeutic research studies. Diabetes mellitus has been well described as a cardiovascular risk factor in developed countries (9) . Conclusion Premenopausal women are relatively protected from CVD compared with men, but this protection wanes with advancing age and with the onset of meno- pause. Levels of LDL-C, HDL-C, and triglycerides, which are established risk markers for atherosclerosis, are unfavorably altered with increased age and at menopause in association with the decrease in circu- lating estrogen levels. The profile of risk markers for CVD is somewhat different for women than for men. Elevated total cholesterol levels are important in both sexes and may in fact have even more of an effect on elderly women's risk. Whereas elevated LDL-C is perhaps the most important risk indicator for men, for women, lowered HDL-C and elevated triglycerides are notable independent risk factors for the develop- ment of CVD in middle age. Women are also more strongly affected than men by the presence of diabetes and by the presence of the combination of risk factors embodied in the metabolic syndrome. Among the newer risk factors, homocysteine and lipoprotein(a) levels have been found to be independent risk factors in both men and women. For women therefore (and increasingly so for men), it is believed that risk mod□ification should focus on LDL-C first and then on triglycerides and HDL-C as well, using non–HDL-C goals as a guide. Therapy should aim to reduce the risk associated with multiple factors, both lipid and nonlipid (8) . Many of the risk factors discussed are exaggerated when a woman goes through menopause, and many are ameliorated by HRT after menopause. Almost all aspects of the lipid profile are improved by exogenous estrogen. Although progestins oppose the benefits of estrogen, this does not appear to be equally true for all HRT combinations. The lipid-altering profile of a given formulation should be taken into account when choosing an HRT. Postmenopausal HRT has a posi- tive effect on several emerging risk factors, lowering both homocysteine and lipoprotein(a) levels. HRT has also been associated with increased levels of C-reac- tive protein, although the clinical significance of this effect remains to be determined and weighed against the many known benefits of HRT on cardiovascular and noncardiovascular systems (7). References : 1. Excerpta Medica, Inc. Am J Cardiol 2002;89(suppl):28E–35E Excerpta Medica, Inc. Am J Cardiol 2002;89(suppl):28E–35E 2. American Heart Association. 2001 Heart and stroke statistical update. Avail- able at: http://www.americanheart.org/statistics. Accessed March 19, 2001. 3. Abbott, J., and N. Berry. 1991. Return to work during the year following first myocardial infarction. British Journal of Clinical Psychology 30(Pt 3):268–270. 4. Alpert JS, Thygesen K, Antman E, Bassand JP. Myocardial infarction redefined-a consensus document of The Joint European Society of Cardiology/American College of Cardiology Committee for the redefinition of myocardial infarction. J Am Coll Cardiol. 2000;36:959–69. 5. Nordestgaard BG, Palmer TM, Benn M, Zacho J, Tybjaerg-Hansen A, Davey Smith G, Timpson NJ. The effect of elevated body mass index on ischemic heart disease risk: causal estimates from a Mendelian randomisation approach.PLoS Med. 2012; 9:e1001212. doi: 10.1371/journal.pmed.1001212. 6. Mendelsohn ME, Karas RH. The time has come to stop letting the HERS tale wag the dogma. Circulation 2001;104:2256 –2259. 7. Knopp RH. Drug treatment of lipid disorders. N Engl J Med 1999;341:498 –511