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Frequency of Metabolic Syndrome in Subfertile Female Population in Mosul

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Abstract

Background Metabolic syndrome is associated with obesity, which is a common condition in subfertlie women population. For that reason, the infertile women will be more predisposed for cardiac diseases.

Objective To know the prevalence of the metabolic syndrome among subfertile women in Mosul, a city in the north of Iraq.

Methods Seventy subfertile females aged >17 years were selected randomly. Adult Treatment Panel-III (ATP-III) guidelines was used for diagnosing metabolic syndrome. The diagnosis was done if any three of the following were present: central obesity, raised triglycerides ≥150 mg/dl, low high-density lipoprotein (HDL-C) cholesterol, blood pressure ≥130/≥85 mmHg, and diabetes or fasting serum glucose (FSG) ≥100 mg/dl.

Results Metabolic syndrome was diagnosed in 16 (22.85%) women. The prevalence was 15.8% in the population younger than 30 years and 55.5% in ages more than 30 years. Fifty percent of women with class II obesity had metabolic syndrome. The most common abnormality was the abnormal waist circumference (100%). Patients having metabolic syndrome had three components of the syndrome and none had four or five components at the same time.

Conclusion The prevalence of metabolic syndrome is high among subfertile women. Focus of cardiovascular prevention should be undertaken for these subjects.

Keywords Metabolic syndrome, infertility, polycystic ovary syndrome, diabetes, obesity

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List of abbreviations: ATP-III = Adult Treatment Panel-III, BMI = Body mass index, CO = Celsius or Centigrade, FSG = fasting serum glucose, HDL-C = High-density lipoprotein cholesterol, 11β-HSD = 11β-hydroxysteroid dehydrogenase type 1, HPA = Hypothalamic-pituitary-adrenal, IL-6 = Interleukin-6, QC = quality control, rpm = round per minute, SD = standard deviation, TG = triglycerides, TNF-α = Tumor Necrosis Factor –α

Introduction

Subfertility (or commonly referred as to infertility) is a failure of conception after 12 months of unprotected regular intercourse, could be primary in which the women has never get pregnant before or could be secondary when the women has delay in conception after a previous pregnancy [(1)]. The association between infertility and obesity is a well-known relation as one of the most important causes of infertility is the polycystic ovary syndrome which is characterized by central obesity [(2)].

Metabolic syndrome, is an association between at least three of the five following medical conditions: abdominal obesity, hyptriglyceridemia, hyperglycemia, hypertension and low high-density lipoprotein (HDL) levels [(3)]. The prevalence of the metabolic syndrome has varied markedly between different studies; because of the different

criteria for the definition of the syndrome [4,5]. The syndrome is common in the United States [6] and its prevalence is increasing in Asia [7]. Metabolic syndrome increases the risk of developing cardiovascular disease and type 2 diabetes [8].

The Ministry of Health in Iraq, reported that death from cardiovascular diseases and diabetes constitute more than 40% of causes of death among females in the 15-49 years age [9]. The aim of this study was to determine the prevalence of the metabolic syndrome in subfertile female in Mosul by the use of the ATP-III guidelines [10].

Methods

Study Population

The study design is a cross sectional study. The place of the study is the private clinic of the author between 2010 and 2011 in Mosul, the capital of the Iraqi governorate Ninevah. The anthropometric parameters were assessed. Random sampling was used and a total of 70 women aged between 17-43 years were included in the study. Consent was taken from all the subjects after they were given verbal information about the study. Approval of the Ethical Committee of Mosul and Ninevah Colleges of Medicine was achieved. The study participants were without medications for hypertension, diabetes, or dyslipidemia and without clinical diabetes.

Measurements

Waist circumferences were measured with flexible tape at the level of umbilicus between the lowest rib and the iliac crest in duplicate to the nearest mm.

The blood pressure was measured two times on the left upper arm and the average used. Systolic (Korotkoff phase I) and diastolic (Korotkoff phase V) blood pressure was measured in sitting position with the use of random zero sphygmomanometer. Hypertension was diagnosed when the average systolic blood pressure was more than 130 mmHg or diastolic blood pressure more than 85 mmHg.

Biochemical investigations were analyzed at the laboratory postgraduate studies in the Department of Biochemistry of Mosul College of Medicine. Overnight fasting blood samples were obtained from all subjects included in this study subjects by antecubital venipuncture.

Five milliliters (5 ml) of venous blood sample from each patient were collected in a plain tube, allowed to clot for 15 minutes in a water bath at 37 °C. Serum was separated by centrifugation at 3000 rpm for 15 minutes to ensure complete separation, and then the serum was divided into 2 parts: 0.5 ml was taken in a plain tube for glucose estimation which was measured at once by standard kit method. The remaining was used for the estimation of lipid profile parameters, particularly serum TG and HDL-C.

The sample was stored at –20 °C until determination was done on daily basis. For accuracy and reproducibility internal quality control (QC) of pooled serum was used within the run and within the batch throughout the work.

Fasting serum glucose (FSG) was estimated using a kit supplied by Bio-con Company, Germany (by Glucose-oxidase-peroxidase colorimetric enzymatic method). FSG more than 100 mg/dl was considered abnormal in this study.

Serum triglycerides (TG) was also estimated by enzymatic method by Fossti in 1982, using a kit supplied by Biomerieux Company, France [11].

Serum high density lipoprotein cholesterol (HDL-C) was determined by enzymatic method followed by Lopez in 1977 using a kit supplied by Biomerieux Company, France [12].

Statistical analyses

The data are presented as frequencies and percentages. Risk ratio is calculated at confidence interval of 95%.

Results

Of the 70 women involved in the study there were 16 having the criteria of metabolic syndrome (22.85%). The mean age of the study group was 27.2 years (SD 6.56), range between

17-43 years. Table 1 showed the distribution of age in the study population.

Table 1. The distribution of age in the study population

Age groups in years	Number	Percentage
<20	10	14.3%
21-30	38	54.3%
31-40	18	25.7%
>40	4	5.7%
Total	70	100%

Table 2 showed the frequency of metabolic syndrome according to age group. The prevalence was 15.8% in the population younger than 30 years and 55.5% in ages more than 30 years. Risk ratio is 4.41 (1.46 at confidence interval of 90%).

Table 2. The frequency of metabolic syndrome according the age groups

Age group in years	Number	Percentage
Less than 20 (n=10)	0	0%
21-30 (n=38)	6	15.8%
31-40 (n=18)	10	55.6%
41-50 (n=4)	0	0%
Total (n=70)	16	22.8%

The mean BMI was 27.6 kg/m^2 (SD 4.85), range from 19-38. Table 3 showed the distribution of BMI. About 75% of the study population was overweight or obese class I and II.

Table 3. The distribution of BMI among the study group (n=70)

BMI in kg/m^2	No.	%
Not overweight BMI <24.9	18	25.7%
Overweight BMI 25-29.9	32	45.7%
Class I obesity BMI 30-34.9	12	17.1%
Class II obesity BMI 35-39.5	8	11.4%
Class III obesity BMI >40	0	0%

Fifty percent of the eight women with class II obesity have the metabolic syndrome (table 4). The prevalence of metabolic syndrome increased with increasing BMI. Risk ratio was 2. 59 (1.74 at 90% confidence interval).

The most common abnormality was the abnormal waist circumference (100%), then the low high density lipoprotein (87.5%), hypertension (56.3), high fasting serum glucose (43.8%), and the least is the high triglycerides (12.5%) (Table 5).

Table 4. The frequencies of metabolic syndrome in different BMI groups

BMI	Number of cases with metabolic syndrome	Percentage
Not overweight (n=18)	2	1.1%
Overweight (n=32)	6	18.7%
Class I obesity (n=12)	4	30.0%
Class II obesity (n=8)	4	50.0%
Class III obesity (n=0)	0	0%
Total (n=70)	16	22.8%

Table 5. The frequencies of abnormalities in women with metabolic syndrome (n=16)

Abnormality	Number	Percentage
Abnormal waist circumference	16	100%
Low high density lipoprotein	14	87.5%
High blood pressure	9	56.3%
High fasting serum glucose	7	43.8%
High triglycerides	2	12.5%

Twenty women have two abnormalities (28.5%), eight had abnormal waist circumference and hypertension and 2 had low high density lipoprotein and high triglyceride level. Twenty-four women had one abnormality (34.2%), (table 6).

Table 6. The frequencies of other abnormalities

Abnormalities	Two abnormalities (n=20)	One abnormality (n=24)
Low high density lipoprotein	2	20
Waist circumference more than 88 cm	8	2
High triglyceride level	2	0
Hypertension	8	2
High fasting serum glucose	0	0

Discussion

The prevalence of metabolic syndrome among United States adult population was 21.8%, about the same prevalence of this study [6]. This may reflect similarities of eating habits and low physical activity between the two communities. Because of the population growth; the total people of metabolic syndrome would increase in Mosul.

Infertility may predict metabolic syndrome and the development of cardiometabolic disease [13]. Disruptions in the hypothalamic-pituitary-adrenal (HPA) axis in women with infertility is implicated in the development of metabolic syndrome [14].

The prevalence of metabolic syndrome increases with age and affects women more than men [15]. In this study also the main age

group affected by the metabolic syndrome is (31-40) year age (table 2).

In this study, 1.1% of women have metabolic syndrome without obesity (table 4). Elevated liver 11β-hydroxysteroid dehydrogenase type 1 (11β-HSD1) activity may be relevant to the metabolically obese, normal-weight individual [16]. This enzyme has a role in the development of metabolic syndrome via intracellular steroid reactivation of inert circulating 11-dehydrocorticosterone (cortisone in humans) into active corticosterone (cortisol), and so increasing tissue glucocorticoids and causes hypertension [17]. This enzyme can also enhance hepatic lipid deposition [18].

The association of metabolic syndrome with obesity has been shown in other studies. Weiss et al. found that 49.7% of severely obese has metabolic syndrome [19].

The most common abnormality is waist circumference which is due to visceral adiposity. Visceral adiposity plays a role in the pathophysiology of insulin resistance [20]. Insulin resistance is thought to be an underlying feature of metabolic syndrome [21]. In a study done in Baghdad, it was found that insulin resistance is caused by inflammatory cytokines (interleukin-6 'IL-6' and Tumor Necrosis Factor –α 'TNF-α') [22]. TNF-α is also associated with endometriosis which may exacerbate the condition of subfertility in these patients [23].

The presence of hypertriglyceridemia, low HDL-C concentrations, and high TG/HDL-C ratios almost never occurred as isolated disorders, and were nearly always associated with insulin resistance because insulin affects TG and HDL-C metabolism [24]. Low level of serum HDL-C cholesterol is a risk of ischemic stroke [25].

This study concluded that metabolic syndrome is a common disorder in women especially in association with infertility. Metabolic syndrome is a predictor of cardiovascular disease, for that reason more attention has to be made to prevent the development of this disease in infertile women

Author contribution

Dr. Jarjees: sample collection and analysis of the results and writing the paper. Hasan and Al Dabbagh did the biochemical tests.

Conflict of interest

The authors have no conflict of interest to declare.

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References

1. Larsen U. Research on infertility: Which definition should we use? Fertil Steril. 2005; 83(4): 846-52. doi: https://doi.org/10.1016/j.fertnstert.2004.11.033.
2. Cooney LG, Dokras A. Beyond fertility: polycystic ovary syndrome and long-term health. Fertil Steril. 2018; 110(5): 794-809. doi: 10.1016/j.fertnstert.2018.08.021.
3. Miranda PJ, DeFronzo RA, Califf RM, et al. Metabolic syndrome: definition, pathophysiology, and mechanisms. Am Heart J. 2005; 149(1): 33-45. doi: 10.1016/j.ahj.2004.07.013.
4. Bonora E, Kiechl S, Willeit J, et al. Prevalence of insulin resistance in metabolic disorders: the Bruneck Study. Diabetes. 1998; 47(10): 1643-9. doi: 10.2337/diabetes.47.10.1643.
5. Rantala AO, Kauma H, Lilja M, et al. Prevalence of the metabolic syndrome in drug-treated hypertensive patients and control subjects. J Intern Med. 1999; 245(2): 163-74.
6. Ford ES, Giles WH, Dietz WH. Prevalence of the metabolic syndrome among US adults: findings from the Third National Health and Nutrition Examination Survey. JAMA. 2002; 287(3): 356-9. doi: 10.1001/jama.287.3.356.
7. Meigs JB. Invited commentary: insulin resistance syndrome? Syndrome X? Multiple metabolic syndrome? A syndrome? Factor analysis reveals patterns in the fabric of correlated metabolic risk factors. Am J Epidemiol. 2000; 152(10): 908-11; discussion 912. doi: 10.1093/aje/152.10.908.
8. Kaur J.A comprehensive review on metabolic syndrome. Cardiol Res Pract. 2014; 2014: 943162. doi: 10.1155/2014/943162.
9. Annual statistical report 2016. Ministry of Health/Environment, Republic of Iraq.
10. Stone NJ, Robinson J, Lichtenstein AH, et al. 2013 ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol. 2014; 63(25 Pt B): 2889-934. doi: 10.1016/j.jacc.2013.11.002.

11. Fossti P, Prencipe L. Serum triglycerides determined colorimetrically with an enzyme that produces hydrogen peroxide. Clin Chem. 1982; 28(10): 2077-80.
12. Grundy SM, Cleeman JI, Daniels SR, et al. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute scientific statement: Executive Summary. Crit Pathw Cardiol. 2005; 4(4): 198-203.
13. Gleason JL, Shenassa ED, Thom ME. Self-reported infertility, metabolic dysfunction, and cardiovascular events: a cross-sectional analysis among U.S. women. Fertil Steril. 2019; 111(1): 138-46. doi: 10.1016/j.fertnstert.2018.10.009.
14. Brunner, EJ. Social factors and cardiovascular morbidity. Neurosci Biobehav Rev. 2017; 74(Pt B): 260-8. doi: 10.1016/j.neubiorev.2016.05.004.
15. Eckel RH. The metabolic syndrome. In: Kasper DL, Fauci AS, Hauser SK (eds). Harrison's principles of internal medicine. McGraw-Hill Medical; 2015. p. 2449.
16. Paterson JM, Morton NM, Fievet C, et al. Metabolic syndrome without obesity: Hepatic overexpression of 11β-hydroxysteroid dehydrogenase type 1 in transgenic mice. Proc Natl Acad Sci U S A. 2004; 101(18): 7088-93. doi: 10.1073/pnas.0305524101.
17. Jamieson PM, Chapman KE, Seckl JR. Tissue- and temporal-specific regulation of 11beta-hydroxysteroid dehydrogenase type 1 by glucocorticoids in vivo. J Steroid Biochem Mol Biol. 1999; 68(5-6): 245-50.
18. Kong S, Ruan J, Zhang K, et al. Kill two birds with one stone: making multi-transgenic pre-diabetes mouse models through insulin resistance and pancreatic apoptosis pathogenesis. PeerJ. 2018; 6: e4542. doi: 10.7717/peerj.4542.
19. Weiss R, Dziura J, Burgert T S, et al. Obesity and the metabolic syndrome in children and adolescents. N Engl J Med. 2004; 350: 2362-74. doi: 10.1056/NEJMoa031049.
20. Lebovitz HE. Insulin resistance: definition and consequences. Exp Clin Endocrinol Diabetes. 2001; 109 Suppl 2: S135-48. doi: 10.1055/s-2001-18576.
21. Grundy SM. Hypertrigyceridemia, insulin resistance, and the metabolic syndrome. AM J Cardiol. 1999; 83(9B): 25F-29F. doi: 10.1016/s0002-9149(99)00211-8.
22. Qasim QA, Fareed YY, Hassan JK. Evaluation of inflammatory state in diabetic patients by measuring of interleukin-6 and tumor necrosis factor-α in obese and non-obese type 2 diabetes mellitus patients as compared with control subjects. Iraqi JMS. 2017; 15(3): 297-304. doi: 10.22578/IJMS.15.3.12.
23. Al-Azawy VSA. Levels of tumor necrosis factor alpha and interleukin-17 in fertile and infertile women with endometriosis. Iraqi JMS. 2017; 15(2): 159-64. doi: 10.22578/IJMS.15.2.8.
24. Kawamoto R, Tabara Y, Kohara K, et al. Relationships between lipid profiles and metabolic syndrome, insulin resistance and serum high molecular adiponectin in Japanese community-dwelling adults. Lipids Health Dis. 2011; 10: 79. doi: 10.1186/1476-511X-10-79.
25. Al-Khazraji AK, Mudhahir DA, Hassan WF. Serum HDL in Patients with Ischemic Stroke a case control study. Iraqi JMS. 2016; 14(2): 153-8.

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