Determination of some biochemical parameters in sera of normotensive and hypertensive obese female in Baghdad

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Abstract: Obesity is a disease describe a case of excessive accumulation of body fats. Obesity is linked to the morbidity of human health, such as the development of hypertension. The study designed to investigate the levels of glucose, urea, creatinine, and lipid profile parameters in sera of obese female and hypertensive obese female. The study included 90 subjects who divided onto three groups equally, in which they are healthy control, normotensive obese, and hypertensive obese. A significant elevation (P<0.01) has observed in the level of glucose, urea, creatinine, triglycerides (TGs), cholesterol, LDL-C, and VLDL-C in obese females compared with control, yet only TGs and VLDL-C were significantly differ in hypertensive obese from normotensive obese. On the other hand, the level of HDL-C was significantly decreased in obese female compared with control. The elevation of TGs and VLDL in hypertensive obese female indicates their role in the complications of hypertension during obesity.

Keywords: Obesity, hypertension, lipid profile, urea, creatinine.

1. Introduction

Obesity is a disease describe a case of excessive accumulation of body fats [1]. This increasing of body fats mainly results from imbalance between the actual calories which the body needs and the uptake calories. Obesity may also involve other etiological factors other than lifestyle such as genetic, metabolic, and environmental factors [2]. The prevalence of obesity has grown in high rates during the last decades to reach (with overweight) to almost a third of the world’s population these days [3]. In recent years, obesity among children and adolescents has emerged as a global epidemic and serious public health problem in the Eastern Mediterranean region. The studies on obesity prevalence in Iraq had been conducted in different cities and revealed a serious percentage of obese among the population [4].

The world health organization (WHO) had defined obesity, by using the term of body mass index (BMI), as people with BMI equal to 30 kg/m² or higher [5]. In addition to BMI, there are some anthropometric
measurements such as waist circumference, waist to hip ratio [6], and waist to height ratio [7] had used as indicators that gave the distribution of body fats, and morbidity risks [8].

Obesity is linked to the morbidity of human health through its involvement in the development of hyperlipidemia, hypertension, insulin resistance, diabetes mellitus, and heart failure [9].

Obese people have shown an increasing of the likelihood of having hypertension by 3.5 folds. On the other hand, it has been estimated that 60-70 percent of hypertension could be attributed to obesity [10]. Obesity induces hypertension through several mechanisms, including adipocytes derivatives adipokines and cytokines mechanism, sympathetic activation mechanism, renal mechanism through activation of renin angiotensin aldosterone system (RAAS), neurohumoral pathways, and metabolic functions mechanisms [11]. The present study is aimed to investigate the level of lipid profile parameters, as well as glucose, urea, and creatinine in sera of obese females and hypertensive obese females.

2. Materials and Methods

2.1. Subjects
The study has included three groups control, normotensive obese, and hypertensive obese. Thirty healthy females were collected as control for the study from Mustansiriyah University, as well as thirty females of each of the remaining groups were collected from AL-Kindy Obesity Research and Therapy Unit. The ages of the samples were between (18-72) years old. The laboratory side of the study was performed at the laboratory of biochemistry research in the department of chemistry science, Mustansiriyah University from Sep 2019 to Feb 2020.

2.2. Samples Collection
Blood samples were collected from the individuals after fasting for 12 hours. A plastic disposable 5mL syringe was used for venipunctures and blood drawn slowly. Then the blood was translocated into gel tube and left for 10 min at room temperature to clot. Blood samples then centrifuged at 3000g for 10 min and the obtained serum stored in three Eppendorf tubes at -20 ºC until analysis. Anthropometric measurements were obtained also for each participant, including height, weight, and waist circumference.

2.3. Methods
The concentration of glucose, urea, creatinine, cholesterol, triglycerides, and HDL-cholesterol were determined by using commercial kits supplied from BIOLABO-France. LDL-cholesterol and VLDL-cholesterol were determined from the equation of Friedewald [12].

3. Results
The results are expressed in the form of mean ± SD, and considered significant at P≤0.05. The age among the three groups was non-significant (P>0.05), whereas highly significant differences (P<0.01) have obtained for anthropometric variables, as in Table 1. The BMI of the hypertensive obese was the highest among the three groups, it was also true for the waist to hip ratio value (both two parameters have non-significant differences between normotensive and hypertensive groups). The highest value of waist to height ratio was for the normotensive obese with non-significant difference between the hypertensive and normotensive obese female.

Table 1. Age and anthropometric variables.
The level of glucose, urea, creatinine, triglycerides, cholesterol, LDL-cholesterol, and VLDL-cholesterol was significantly elevated (P<0.01) in hypertensive and normotensive obese females. On the contrary the level of HDL-cholesterol was significantly (P<0.01) decreased in both obese females, as cleared in Table 2.

A significant differences (P<0.01) have obtained between the normotensive and hypertensive obese subjects in the level of TG, and VLDL-cholesterol, whereas the rest parameters were non-significant between the two groups of obese females.

Table 2. Parameters of the study in mg/dL.

| Parameters | Control, N=30 (mean ± SD) | Normotensive Obese, N=30 (mean ± SD) | Hypertensive Obese, N=30 (mean ± SD) | P-value |
|------------|---------------------------|--------------------------------------|--------------------------------------|---------|
| Glucose    | 94.75 ± 9.18              | 103.10 ± 16.13                      | 105.57 ± 18.25                      | 0.017   |
| Urea       | 25.65 ± 6.71              | 32.40 ± 8.37                        | 32.46 ± 8.12                        | 0.001   |
| Creatinine | 0.63 ± 0.07               | 0.73 ± 0.08                         | 0.75 ± 0.08                         |         |
| TG         | 97.18 ± 17.05             | 146.33 ± 32.38                      | 177.64 ± 35.79                      | <0.001  |
| Cholesterol| 153.23 ± 17.24            | 264.07 ± 62.94                      | 255.08 ± 69.43                      |         |
| HDL-C      | 48.40 ± 4.29              | 42.94 ± 14.27                       | 38.28 ± 6.27                        |         |
| LDL-C      | 85.39 ± 16.4              | 191.81 ± 57.4                       | 181.3 ± 68.85                       |         |
| VLDL-C     | 19.44 ± 3.41              | 29.31 ± 6.30                        | 35.50 ± 7.16                        |         |

4. Discussion
The glucose homeostasis is controlled by several factors such as hormones. The healthy individuals own an efficient production of insulin which promote liver, adipose and muscle cells to absorb glucose at high levels of blood glucose [13]. In obese, a problem of insulin resistance takes a major portion of the elevated fasting glucose concentration in the blood. The insulin resistance in obesity may start as an actual consequence of the fact that hypertrophied adipocytes have no more space for entering glucose and handle it, this may also true for muscle, liver, and β-pancreatic cells under lipids accumulation [14]. Hence, glucose will lose a valuable deposit.

The risk of hypertension becomes greater at high levels of blood glucose [15]. Hyperinsulinemia and hyperglycemia increase renal sodium reabsorption directly. The increased tubular sodium retention results
in an increased cardiac output and vascular stress, with arterial remodeling and vasoconstriction leading to hypertension [16].

Urea is a small molecule results from the metabolism of amino acids through oxidation of ammonia. Urea is produced in the body in a continuous manner mainly by the liver, and found in the blood normally in low concentrations while the major portion eliminated into the urine by the kidney [17]. Creatinine is another small molecule filtered by kidney through renal glomerular and not being reabsorbed in the renal tubular region. Creatinine is produced non-enzymatically from creatine [18]. Obesity and hypertension found to alter the renal functions [19]. Researches have shown that obese people suffer from a decrease in glomerular filtration rate at high significant statistics [20]. This may explain the elevation of blood urea and creatinine levels.

High density lipoprotein (HDL), low density lipoprotein (LDL), and very low density lipoprotein (VLDL) are lipoproteins presented in the plasma, they work collectively to transport the water insoluble TGs and cholesterol to different tissues for storage or utilizing [21]. When the liver receive a payment of TGs from the circulation, hepatocytes resent these TGs to the circulation in the form of VLDL (the highest the level of TGs the highest the secretion of VLDL) [22]. VLDL follows a conversion into LDL through lipolysis process catalyzed by lipase enzymes and further modifications by lipid transfer proteins [23]. The HDLs are complexes act to transfer cholesterol from the peripheral tissues to the liver, result in the clearance of blood from cholesterol [24]. Plasma cholesterylster-transfer-protein (CETP) facilitates the transfer of neutral lipids between lipoproteins. CETP transfers cholesterol esters from HDL to apoB-containing lipoproteins in exchange for TG from apoB-containing lipoproteins to HDL [25].

In obesity, the adipocytes are full of TGs and the hypertrophied adipocytes have no extra room to store more TGs, hence the liver will receive a magnificent amounts of TGs, consequently liver increases the secretion of VLDLs. Also the expression of lipoprotein lipase in adipose tissue is reduced during obesity, which results in deflection of lipolysis. Furthermore, increasing of TG level increases the exchange of cholesterol esters and TG between VLDL and HDL, and LDL by CETP, which leads to decrease the concentration of HDL cholesterol in the plasma, as well as a reduction of the TG content in LDL [26].

5. Conclusions
The increase of TGs and VLDL level in hypertensive obese female declare the role of these two parameters in the severity of hypertension during obesity. TGs elevation comprises an important portion of further complications in the sequence of obesity and hypertension harmful effects such as atherosclerosis.

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