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Article

Estimation of Some Biochemical Tests in the Serum of Obese Men with Normal Blood Pressure and High Blood Pressure

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ABSTRACT

Obesity is a medical word that refers to a state in which a person's body fat levels are excessively high. Obesity is linked to a variety of health issues in humans, like the emergence of hypertension. This study aimed to look at the blood glucose concentration, urea, creatinine, and lipid profile factors in obese and hypertension-overweight men's sera.

The study included 75 people who were evenly divided into three groups: healthy controls, normotensive obese people, and hypertension-obese people. When comparing obese males to controls, there was a significant increase ($P<0.01$) in blood glucose, urea, creatinine, cholesterol, triglycerides, low-density lipoprotein cholesterol (LDL-C), and very low-density lipoprotein cholesterol (VLDL-C) although only triglycerides (TGs) and VLDL-C were substantially different in hypertension obese versus normotensive obese. Compared to controls, Obese men had significantly lower levels of high-density lipoprotein cholesterol (HDL-C). High levels of triglycerides (TGs) and Very Low-density Lipoprotein cholesterol (VLDL) in hypertensive obese males show they play a role in hypertension problems.

Keywords: Obesity, Hypertension, Urea, Creatinine, lipid profile.

INTRODUCTION

Obesity is a medical word that refers to a state in which a person's body fat levels have accumulated excessively [1]. This increase in body fat is primarily due to a discrepancy between the calories required by the body and the calories consumed. Other etiological factors, like genetic, physiological, and ecologic variables, may play a role in obesity [2]. Obesity has increased at an alarming rate in recent decades, now affecting about a third of the realm's populace (with overweight) [3]. In recent years, childhood and adolescent obesity has become a worldwide problem and a significant health concern in the European Region. Obesity occurrence studies in Iraq were undertaken in various towns and found a significant ratio of fat people in the community [4-6]. The Realm Health Institute classified obesity as those with a body mass index (BMI) of 32 kg/m2 or greater [7].

Aside from BMI, anthropometric Waist circumference, midriff proportion and midriff proportion [8] are all parameters to consider. They have been employed to indicate body fat distribution and morbidity concerns [9]. Obesity influences the progression of cardiovascular disease, hyperlipidemia, hypertension, insulin resistance, and diabetes mellitus, all of which are linked to morbidity in human health[10]. Obesity increases the risk of hypertension by 3.5 times in obese people. Obesity, on the other hand, is thought to be responsible for 60 to 70% of hypertension [11].

Adipocyte products such as leptin and cytokine, sympathetic stimulation, renal stimulation via the renin-angiotensin-aldosterone system (RAAS), neurohormonal processes, and metabolism functioning mechanisms all contribute to hypertension in obesity [12-14]. The focus of this research is to look at lipid level characteristics as well as glucose, urea, and creatinine levels in obese and hypertensive obese men's sera.

MATERIALS AND METHODS

Subjects

The study had three groups: control, obese with normal blood pressure, and obese with high blood pressure. A total of 25 healthy individuals and 25 males from each of the other groups were selected for the study. The samples' ages ranged from 20 to 65

Samples Collection

Individuals' samples were taken after they had fasted for 12 hours. The blood samples were left to coagulate for 15 minutes at room heat. After centrifuging samples at 2800g for 5 minutes, the sera were collected and kept at -20 oC until analysis.

RESULTS

The data are analyzed as mean and SD and are significant at ($P \leq 0.05$). The difference in gender between the different groups was not significant ($P > 0.05$), while anthropometric characteristics showed highly significant changes ($P < 0.01$), as in (Table). Among the three groups, the obese with high blood pressure had the highest BMI.

Parameters	Control (mean ± SD)	Obese with normal blood pressure (mean ± SD)	Obese with high blood pressure (mean ± SD)	P-value
Age	30.58 ± 14.17	32.65 ± 9.31	38.89 ± 13.1	
BMI (kg/m^2)	20.55 ± 2.03	40.52 ± 11.01	39.04 ± 10.85	
WC (cm)	75.45 ± 3.98	117.35 ± 15.25	116.43 ± 14.61	P<0.001
WHpR	0.8 ± 0.04	0.88 ± 0.23	0.89 ± 0.09	
WHtR	0.5 ± 0.03	0.71 ± 0.21	0.73 ± 0.35	

Table 1. The age and anthropometric variables of control obese with normal blood pressure and obese with high blood pressure.

The blood sugar levels, urea, creatinine, triglycerides, cholesterol, low-density lipoprotein cholesterol, and very low-density lipoprotein cholesterol remained considerably higher in obese males with hypertension and normotension (P<0.01). The amount of high-density lipoprotein cholesterol, on the other hand, was considerably (P<0.01) lower among both obese males, as shown in (Table 2). The levels of triglycerides (TG) and VLDL-cholesterol were significantly different (P<0.01) between obese with normal blood pressure and obese with high blood pressure, although the rest of the data have not been.

Parameters	Control (mean ± SD)	Obese with normal blood pressure (mean ± SD)	Obese with high blood pressure (mean ± SD)	P-value
Glucose	96.35 ± 11.07	105.12 ± 17.33	108.19 ± 19.28	0.015
Urea	27.45 ± 7.93	35.42 ± 9.11	36.06 ± 9.15	0.001
Creatinine	0.8 ± 0.06	0.82 ± 0.11	0.8 ± 0.75	
Triglycerides	98.21 ± 16.07	144.27 ± 27.31	168.74 ± 42.91	
Cholesterol	150.81 ± 18.3	245.15 ± 71.29	232.1 ± 70.41	P<0.001
HDL-C	45.5 ± 5.09	40.18 ± 12.13	36.19 ± 5.87	
LDL-C	86.71 ± 14.5	185.71 ± 55.21	172.7 ± 64.25	
VLDL-C	20.17 ± 4.53	31.52 ± 7.22	33.56 ± 8.17	

Table 1. The Parameters study of control, obese with normal blood pressure and obese with high blood pressure.

DISCUSSION

Several factors, including hormones, influence glucose homeostasis. Healthy people produce enough insulin to allow their liver, adiposity, and muscle tissue to absorb glucose even when blood glucose levels are high [15]. Insulin resistance accounts for a large amount of fast glucose concentration in obese blood. Insulin deficiency in obesity could begin as a direct result of overdeveloped adipose cells not having sufficient to accept and process glucose. When lipids build, that might also be relevant to muscle, liver, and pancreatic cells [16]. As a result, a significant amount of glucose will be lost.

When blood glucose levels are high, the risk of hypertension increases [17]. Renal sodium reabsorption is directly increased by hyperinsulinemia and hyperglycemia. Increased tubular sodium retention raises microvascular stress and heart rate, resulting in artery remodel and vascular constriction, which leads to high blood pressure [18]. The byproduct of ammonia being oxidized during the metabolism of amino acids appears to be a small molecule called urea. The kidneys eliminate the bulk of the urea that is continually produced by the body, principally by the liver and is seen in low amounts in the blood. [19]. Creatinine is Another particle passed by the renal and not recycled back into the tubule area. Creatinine is produced without the assistance of enzymes from creatinine. Obesity and hypertension have been linked to changes in renal function. Obese patients have reduced renal blood flow at high meaningful statistics, according to studies [20]. This could explain why blood urea and creatinine concentrations are higher.

Lipoproteins in the bloodstream carry Liquid TG cholesterols to various organs for storage or usage. When the liver gets a repayment of TGs from the blood, cells return them to the bloodstream in the shape of VLDL. VLDL gets fat oxidation catalyzed via lipase enzymes, and lipid transfer protein is modified further. HDLs are compounds that carry cholesterol via periphery tissues to the liver, cleaning cholesterol from the bloodstream [21]. The blood protein cholesteryl ester-transfer-protein assists neutral lipids in transferring throughout lipoproteins. CETP substitutes TG from apoB-containing lipoproteins for cholesterol esters from HDL [22].

The adipose cells of obese individuals are overflowing with TGs, and the overdeveloped adipose cells cannot collect anymore. As a result, the liver absorbs a large quantity of TGs and excrete more VLDLs. Obesity also reduces the production of lipoprotein lipase in fat cells, resulting in lipolysis defection. Furthermore, increasing TG levels enhances the interchange of cholesterol esters and TG among VLDL and HDL, as well as between LDL and CETP, resulting in a drop in HDL cholesterol concentration in the plasma and a decrease in TG contented in LDL [23].

CONCLUSIONS

The role of TGs and VLDL in the severity of hypertension in hypertensive obese males is demonstrated by the increased levels of these two markers. Detrimental effects like atherosclerotic can occur as a result of the combination of high blood pressure and obesity, and TG increase is a significant contributor to additional difficulties.

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