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# ESTIMATION OF SERUM BIOCHEMICAL MARKERS IN OBESITY

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**Abstract**

**Background: Estimation** of Biochemical Parameters of Obese aged 20-40 yrs with Normal controls visiting OPD in Government General Hospital, Kurnool.

**Methods :** We included 35 participants (n=35 cases and n=35 controls) who attended Medicine OPD at Government General Hospital ,Kurnool from March 2023 to August 2023.The study aimed to examine blood markers that may help in determining obesity severity. The markers of interest were serum cholesterol, triglycerides, CRP, Vitamin D and Vitamin B12, levels.

**Results:** The study showed that 40.02% of individuals had a total cholesterol level, 36.08% had a high-density lipoprotein cholesterol level, 20.09% had a low-density lipoprotein level, 29.05% had a Triglyceride, 45.08% had a fasting glucose level, and 45.2% had a postprandial glucose level that was greater than the threshold values.45% had increased CRP Levels. 35% had decreased levels of Vitamin D and Vitamin B 12 levels. Comparative study shows Significant rise in serum cholesterol, Triglyceride ,LDL ,VLDL ,Plasma Glucose and serum CRP in Obese With P value <0.0001. Significant decrease in vitamin D and Vitamin B 12 in Obese with P value <0.0001.

**Conclusion**: Significant Differences were found in Biochemical parameters compared to Control group. The finding suggest that Biochemical Markers are Potential Biomarkers for Obesity. The biological markers of obesity that have been found are helpful early markers that can be used to identify distinct groups of patients who are at risk for cardiovascular disease.

**Key words: Obesity, Total Cholesterol, CRP, Vitamin B12, Vitamin D.**

**Introduction:**

Obesity is characterized by a surplus of body weight that exceeds the necessary physical requirements, caused by an excessive buildup of fat. The primary cause of excessive body weight is the accumulation of fat, namely triacylglycerol. Other forms of energy storage, such as glucose glycogen or protein in the liver and muscles, do not have the capacity to exceed the necessary limitations like adipose tissue does. The ability of anabolic steroids to enhance

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lean body mass and overall body mass has been observed exclusively in individuals who are already malnourished.[1]

Obesity is a very significant problem that must be addressed on a global scale. There are a significant number of persons who are afflicted by it in several developing nations, such as India. A growing number of urban Indians are experiencing obesity, abdominal obesity, and other comorbidities.[2]

There is evidence that an unfavorable biochemical profile is independently related with cardiovascular disease. It is believed that dyslipidemia, poor glucose tolerance, insulinemia, hypertension, and vascular abnormalities are significant surrogate markers for the development of cardiovascular disease in the future.[3]

In light of the growing prevalence of obesity in India, it is of the utmost importance to add biochemical markers into surveillance in order to evaluate reliable data for the purpose of estimating the present risk of cardiovascular disease.

Physicians must understand obesity comorbidities and their effects on clinical biomarkers and obese patients' health. Few research have examined how obesity affects these biomarkers Triglyceride, HbA1c, cholesterol, Glucose, CRP, Vitamin D and Vitamin B12.

**Materials and Methods:**

The present cross-sectional study was conducted in the Department of Biochemistry, Kurnool Medical College Kurnool. we included 35 participants (n=35 cases and n=35 controls) who attended Medicine OPD at Government General Hospital ,Kurnool from March 2023 to August 2023 .The study was approved by the Institutional Ethical Committee, and all participants gave informed consent.

The subjects are categorised in to two groups

Control Groups: The 35 subjects chosen as a control for the study. The individuals were having no evidence of any infection .No history of physiological and pathological illness, not taking any kind of treatment which likely to affect the lipid profile levels.

Study Groups: Study was compromising 35 subjects.

The complete History about name ,age, Height ,weight ,BMI ,BP, Pulse, Dietary Habit, socioeconomic status, Menstrual History ,Occupation ,Smoking/Alcohol habits ,Life style were taken.

About 7 ml of fasting blood sample from antecubital vein was withdrawn in a perfectly cleaned disposable syringe and about 5 ml blood transferred to a red tube and kept for 30 minutes at room temperature and 1ml blood was transferred into fluoride tube. The serum was separated by centrifugation at 3000.rpm for 10 minutes. The sample was analysed for Total cholesterol, HDL-cholesterol, Triglyceride, LDL, VLDL, The total cholesterol (TC), triglyceride (TG) and HDL (high density lipoprotein) were measured by automated enzymatic method. The LDL (low density lipoprotein) was obtained using FRIELDWALD calculation.[4]. Blood glucose was analysed by colorimetric enzymatic method. Vitamin D and Vitamin B 12 by CLIA Method. For the classification of serum glucose concentration, we used the Standards of Medical Care in Diabetes reference, issued by the American Diabetes Association (ADA).

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**STATISTICALANALYSIS:**

All of the information was entered into the 2013 edition of Microsoft Excel. Statistical Software for the Social Sciences version 13.0 was utilised for the categorization and analysis of the data (SPSS Inc., Chicago, IL). Means and standard deviations were used to describe the quantitative variables, whilst frequency distributions, proportions, and percentages were utilised to explain the qualitative variables. Observed values are expressed as mean ± SD. The significance of mean difference between groups were assessed by student. "t" test and distribution of t probability (P) and degree of freedom.. A 'p' value of less than 0.05 was considered to indicate a significant.

**Results:**

**Table no.1: Mean Values for Anthropometry of Normal (Control) and Obese (cases) subjects,**

| S.no | Variable | Control | Cases |
|---|---|---|---|
| 1 | Age (yrs) | 38..67±5.2 | 38.3±2.5 |
| 2 | Height(cm) | 155±3.26 | 153±5.3 |
| 3 | Weight (kgs) | 53.5±3.2 | 84±7.6 |
| 4 | BMI ($Kgm^2$) | 23±1.35 | 34.8±2.4 |

**Table 2: Statistical Analysis of Biochemical parameters in Normal (Control )and Obese (cases )subjects ,**

| S.no | Variable | Control | Cases | difference | Standard error | t statistic | DF | P value |
|---|---|---|---|---|---|---|---|---|
| 1. | Total cholesterol | 167±15.32 | 203.5±53.2 | 36.5 | 5.06 | 7.20 | 68 | <0.0001* |
| 2. | HDL cholesterol | 54.5±8.5 | 45.37±6.3 | -9.13 | 1.78 | -5.10 | 68 | <0.0001* |
| 3. | Triglyceride | 118.8±34 | 243.0±45.1 | 124 | 9.54 | 13.00 | 68 | <0.0001* |
| 4. | LDL | 102.5±5.3 | 115.3±9.5 | 12.8 | 1.83 | 6.96 | 68 | <0.0001* |
| 5. | VLDL | 26.5±5.8 | 42.3±14.5 | 15.8 | 2.64 | 5.98 | 68 | <0.0001* |
| 6. | Plasma Glucose | 99.5±13.5 | 114.5±15.2 | 15.0 | 3.43 | 4.36 | 68 | <0.0001* |
| 7. | CRP | 8.35±3.5 | 49.2±26.3 | 40.85 | 4.48 | 9.10 | 68 | <0.0001* |
| 8. | Vitamin D(ng/ml) | 23.95±4.5 | 10.95±6.34 | -12.4 | 1.31 | -9.43 | 68 | <0.0001* |
| 9. | Vitamin B12(pmol/L) | 250.6±56.25 | 100.5±44.8 | 150 | 12.15 | -12.34 | 68 | <0.0001* |

**Present study was carried out in 35 Normal subjects and 35 obese. Participants are aged between 25-40 yrs .Table 1 shows Mean and SD of Anthropometry of Cases and controls.**

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**Table 2 Depicts Statistical Analysis of Biochemical parameters in Normal (Control) and Obese (cases) subjects. Comparative study shows Significant rise in serum cholesterol, Triglyceride ,LDL ,VLDL ,Plasma Glucose and serum CRP in Obese With P value <0.0001*.**

**Significant decrease in vitamin D and Vitamin B 12 in Obese with P value <0.0001*.**

**Discussion:**

**One of the most prevalent conditions that negatively impacts people's health and quality of life, as well as contributing to the development of depression, is obesity. Atherosclerosis and cardiovascular illnesses are also potential outcomes of this syndrome, in addition to other risk factors. A low-grade inflammatory response is also considered to be a risk factor, in addition to being overweight.[5]**

**Based on the present study, we found that increase in Lipid profile ,Blood glucose and Serum CRP in obese and inverse , significant association between Vitamin D, Vitamin B12 and Obesity.**

Various irregularities contribute to the dyslipidemia observed in obese patients. The presence of these abnormalities is caused by the simultaneous occurrence of increased transport of free fatty acids to the liver due to higher levels of total and visceral fat, insulin resistance, and an inflammatory state triggered by the infiltration of macrophages into adipose tissue. An essential factor in the increase of serum TG levels is the excessive synthesis of VLDL particles by the liver . The secretion rate of very low-density lipoprotein (VLDL) particles is strongly influenced by the availability of triglycerides (TG), which is determined by the quantities of fatty acids accessible for the synthesis of TG in the liver. The presence of a large amount of TG inhibits the breakdown of Apo B-100 within the liver, leading to an increase in the production and release of VLDL.[6,7,8,9,10]

Obese patients experience elevated levels of Apo C-III. Insulin inhibits the expression of Apo C-III, hence the insulin resistance observed in obese patients may explain the rise in Apo C-III levels. Apo C-III functions as a suppressor of lipoprotein lipase activity, which consequently hinders the elimination of lipoproteins that are rich in triglycerides. If insulin resistance is severe, it can lead to a reduction in the activation of lipoprotein lipase by insulin, resulting in a decrease in the clearance of lipoproteins that are rich in triglycerides. Hence, a reduction in the elimination of triglyceride-rich lipoproteins also plays a role in the increase of blood triglyceride levels in obese patients.[11]

Obesity is characterized by the presence of macrophages that invade adipose tissue, leading to an inflammatory response. Macrophages create cytokines, while fat cells produce adipokines, both of which have an impact on lipid metabolism. [12]

Adipokines, such as adiponectin and resistin, control the process of lipid metabolism. Obese individuals exhibit reduced amounts of adiponectin in their bloodstream. Reduced adiponectin levels are linked to increased blood triglyceride (TG) levels and decreased high-density lipoprotein cholesterol (HDL-C) levels.[13]

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Pro-inflammatory cytokines have an impact on the metabolism of HDL. Initially, they reduce the synthesis of Apo A-I, the primary protein component of HDL. Furthermore, within macrophages, the presence of pro-inflammatory cytokines results in a reduction in the expression of ABCA1 and ABCG1. Consequently, this reduction leads to a decrease in the transport of phospholipids and cholesterol from the cell to HDL. Furthermore, pro-inflammatory cytokines exert a suppressive effect on the synthesis and functionality of LCAT, hence restricting the transformation of cholesterol into cholesterol esters inside HDL. This phase is essential for the creation of a typical spherical HDL particle and enhances the capacity of HDL to carry cholesterol. Collectively, these alterations caused by pro-inflammatory cytokines may lead to a reduction in HDL-C and Apo AI concentrations.[14,15]

Elevations in pro-inflammatory cytokine levels will promote the synthesis of lipoproteins rich in triglycerides (TG) and hinder the removal of these TG-rich lipoproteins. This combined effect leads to the elevation of blood TG levels observed in obese patients.[16,17]

The linkage between obesity and impaired glucose metabolism is intricate. Initially, certain research have indicated that there is a correlation between a genetic inclination towards central obesity and an increased susceptibility to type 2 diabetes.The presence of insulin resistance in children with early-stage obesity was found to be connected with increased expression of genes related to central obesity and type 2 diabetes.These findings partially elucidate the strong correlation between central adiposity and insulin resistance as well as type 2 diabetes. Furthermore, obesity is characterized by a persistent state of inflammation. Elevated levels of inflammatory adipokines, such as progranulin, procalcitonin, and interleukin-34, were seen in obese children and were found to be linked to insulin resistance. Furthermore, malnutrition frequently occurs in individuals who are obese. [18,19,]

Obese children had reduced levels of total 25-hydroxy vitamin D, vitamins A, C, and E, as well as zinc and magnesium, in comparison to non-obese children. Malnutrition in children was linked to increased systemic inflammation and decreased insulin sensitivity.In obesity, there is an excess of branched-chain amino acids and metabolites. This excess facilitates the transit of fatty acids in the blood vessels and leads to insulin resistance.Insulin resistance in obesity can be exacerbated by decreased blood flow to both subcutaneous and visceral fats, as well as impaired glucose absorption in resting skeletal muscle. [20]

Furthermore, there is strong evidence indicating that low-grade inflammation plays a crucial role in linking obesity and insulin resistance, along with endothelial dysfunction. Elevated levels of serum inflammatory markers, such as C-reactive protein (CRP), have been observed in individuals who are obese. These markers have been linked to the severity of insulin resistance (IR) and dysfunction of the endothelium. Several mechanisms have been proposed to explain the mild inflammation associated with obesity. Initially, adipose tissue serves as a significant reservoir of proinflammatory cytokines, including TNFα and IL-6, as well as anti-inflammatory cytokines, such as adiponectin. Furthermore, infrared radiation (IR) can induce inflammation by impeding the insulin's anti-inflammatory properties. Obesity is primarily caused by oxidative stress, which is triggered by the excessive consumption of

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macronutrients or an elevated metabolic rate. This oxidative stress can contribute to the development of inflammation. Indeed, the introduction of glucose or fat into the bodies of both healthy and overweight individuals results in heightened inflammation and reduced vascular responsiveness.[21,]

The primary mechanism seems to be the sequestration of VD. Obesity may provide a larger storage space for VD and/or 25(OH)D due to the excess fat tissue. This could result in lower levels of 25(OH)D in the blood. However, Drincic et al. disputed this idea and suggested that 25(OH)D is merely diluted in a larger volume in individuals with obesity, supporting the volumetric dilution hypothesis. Alternative theories propose alterations in VD metabolism, particularly in adipose tissue, where the levels of CYP2J2 mRNA were observed to be lower in obese women compared to lean women. In a recent analysis, it was suggested that several factors may influence vitamin D levels in older individuals with obesity. These factors include the storage or dilution of vitamin D in fatty tissue, increased breakdown of vitamin D in fatty tissue, decreased conversion of vitamin D to its active form, and reduced exposure to sunlight.[22,23]

Multiple possible explanations may account for these correlations. A possible explanation is that decreased levels of vitamin B12 in the blood would result in the accumulation of folate as 5-methyltetrahydrofolate, hindering the conversion of homocysteine into methionine. Consequently, this would lead to a decrease in protein synthesis and the deposition of lean tissue. The potential cause could potentially be attributed to the malfunctioning of adipocytes, which is associated with reduced amounts of vitamin B12 and subsequent cellular inflammation. Obesity may potentially reduce serum vitamin B12 levels due to factors such as reduced intake or absorption of the vitamin, increased breakdown of the vitamin, storage of the vitamin in fat tissue, or alterations in the composition of gut bacteria that might impact vitamin B12 metabolism. Remarkably, a recent study conducted on the Danish population revealed a strong correlation between lower serum vitamin B12 levels and greater BMI. However, it was found that a genetic risk score, which is related to variations associated with vitamin B12 levels, did not demonstrate any association with BMI. Additional research is needed to explore the cause-and-effect relationship between vitamin B12 levels and obesity.[24]

Conclusion: In the present study, there is Significant association of lipid profile, plasma Glucose, Serum CRP, vitamin B12, vitamin D and Obesity. The findings underscore the significance of evaluating lipid profile, plasma Glucose, Serum CRP, vitamin B12, vitamin D in individuals with obesity and a confirmed to enhance their health condition and prevent complications.

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