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FORTITUDE OF THE PRO-INFLAMMATORY PROFILE OF EXTREMELY OBESE CHILDREN IN LAHORE DIVISION

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Abstract:

Aim: *To assess the status of pro-inflammatory markers in children with obesity.*

Subjects and methods: *The study involved 37 children (20 non-obese and 17 obese). Levels of C reactive protein, α-2 macroglobulin (A2M) and hepatoglobin were measured. Anthropometric results are correlated with biochemical parameters.*

Study Design: *A Cross-Sectional Study.*

Place and Duration: *In the Department of Biochemistry, Services Hospital Lahore for one year period from February 2018 to February 2019.*

Results: *The mean CRP in serum, A2M and hepatoglobin were several folds advanced in the obese group than in the group of control (p <0.05). CRP was significantly correlated with body weight (BW), body physique index (BMI) and A2M (p-value <0.05).*

Conclusion: *Childhood obesity is linked with a pro-inflammatory state before the appearance of co-morbidities of metabolic syndrome, whereas haptoglobin, CRP and A2M are the best indicator of inflammation development.*

Key words: *Obese children, CRP, inflammatory profile.*

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INTRODUCTION:

The incidence of obesity in the global epidemic has reached an alarming level that is considered clinically as more than 300 million overweight adults. Worldwide, less than 5 million adults, 22 million children, as well as 155 million schoolchildren are overweight. This means that one in 10 children around the world is overweight. In 2000, it was estimated that more than half of American adults are overweight, reflecting a 61% increase in 10 years. Pakistan is a developing country where the prevalence of obesity is similar to that in some developed countries. Many local studies have documented complications related to overweight and obesity in Pakistan. However, these studies did not focus on the real image, especially in children.

Recent studies over the past two decades have shown that obesity is a single gene dysfunction (monogenic obesity) or a metabolic disease that can be caused by many genes, each of which contributes little to the definition of phenotype of obesity (multi-gene obesity). Fathers' obesity contributes to obesity in children with their mother's early menstrual bleeding. Obesity due to the accumulation of fat in the visceral part of the body, namely central or abdominal obesity, is more prone to health problems and is associated with a greater risk of metabolic syndrome than peripheral or subcutaneous obesity.

Obesity is a direct consequence of the imbalance between energy consumption and energy expenditure, or the extent to which fat, abnormal or excessive fat accumulation can affect health. This storage is controlled by a variety of complex mechanisms, including environmental interaction with genetic, neuronal and biochemical interactions. Superfluous dynamism is mainly stored in adipose tissue as triglycerides. Even though adipocytes are exactly premeditated for energy storage and easy fat exchange, morphological changes are accompanying with an increase in body fat. In response to the growth of adipocytes during the development of obesity, this affects the functioning of adipose tissue. In recent years, obesity has been found to cause more complications. The relationship between obesity and inflammation was first described as a positive correlation between fat mass and tumor necrosis factor (TNFα) expression. A relationship between obesity and inflammation has been demonstrated in obese individuals by increasing plasma levels of several pro-inflammatory markers, including severe phase proteins such as cytokines and C-reactive protein (CRP). Although the increase in visceral fat deposits and adipose muscle hypertrophy has been associated with a higher degree of fat inflammation, some pathways leading to the pro-inflammatory fat inflammation in humans have recently been identified. Recently, however, much devotion has been paid to the role of macrophages. In 2003, two studies show that food obesity is associated with infiltration of macrophages into white adipose tissue. Infiltrated macrophages, which are then part of the stromal fraction of adipose tissue, are responsible for the production of a wide range of proinflammatory proteins, including CRP, serum amyloid A, TNFα and interleukin-6 (IL-6). CRP releases acute phase hepatocytes such as haptoglobulin and α-2 macroglobulin. The proposed study provides for simultaneous measurement of pro-inflammatory markers (PCR, alpha-macroglobulin and haptoglobin) in children (lean and obese) and uses a multiplex system. Multiplex protein analysis can be used as a tool for advanced diagnosis of metabolic diseases in complex clinical presentations. By recognizing biochemical differences that are prone to obesity through a variety of mechanisms, obese people can be divided into specific groups that can respond to specific diets and / or exercise regimens, medications, or surgery. In summary, obesity is a serious health risk and effective, evidence-based treatments are needed to minimize obesity-related diseases.

MATERIALS AND METHODS:

This Cross-Sectional Study was held in the Department of Biochemistry, Services Hospital Lahore for one year period from February 2018 to February 2019. to The study, which involved 37 children aged 0.5 to 10 years. Informed consent was given by all patients. Patients and / or parents were informed about the possible benefits and risks of this study. The subjects were subjected to detailed medical examinations based on a family history of diabetes and obesity and family history in order to determine their lifestyle and eating habits. The researcher physically examined all individuals for growth and developmental abnormalities and recorded anthropomorphic data. Patients with obesity syndrome (e.g. Cushing's syndrome, Down syndrome, autism, etc.) were not included in the study. This is a cross-sectional prospective study. According to BMI, the entities are divided into the following 2 age groups:

Group I: control: BMI <80 percentage of children (n = 20, average age: 4.64 years) Group II: obesity: BMI> 95 percent (n = 17; average age: 7.46 years) Body weight (total body weight); Body mass index (BMI) was calculated according to the equation: BMI = IM

(kg) / height (m) 2 the percentage of BMI was determined according to WHO height tables. 3-4 ml of venous blood was collected from a vein of the arm after fasting for 12 hours. Blood samples were centrifuged at 5000 rpm and the serum sample was aliquoted and stored at -20 ° C until use. All biochemical parameters were determined using standard procedures. Serum C-reactive protein (PCR), haptoglobin and alpha 2 macroglobin levels were estimated using the Bioplex test system using an acute inflammatory phase determining pane. Collected data was analyzed using SPSS version 17 (SPSS, Inc., Chicago, IL, USA). The significance of the difference between the control group and obesity was analyzed using a two-tailed Student's t-test. The Pearson test was used to calculate the correlation between related variables. P <0.05 was considered statistically significant.

RESULTS:

The physical properties of the subjects are summarized in Table 1 and Figure 1. The average BMI of non-obese children (n = 20, mean age: 4.64 years) was 18.18, 28.71 kg. / m2 in obese people (n = 17, average age: 7.46 years). The average weight and height of non-obese people were 17.58 kg and 0.97 m in the obese group compared with 46.31 kg and 1.23 m.

Table 1: Physical characteristics of children (0.5-10 years). Data are expressed as mean ± SEM (median).

Group	**Non-obese**	**Obese**	**P**
N	20	17	
BMI (kg/m2)	18.18±1.33 (16.97)	28.71±1.80 (26.63)	0.000a
Weight (kg)	17.58±1.79 (15.75)	46.31±6.13 (45)	0.000a
Height (m)	0.97±0.04 (0.98)	1.23±0.06 (1.30)	0.001a

Significantly different from the non-obese group (Student's t-test);

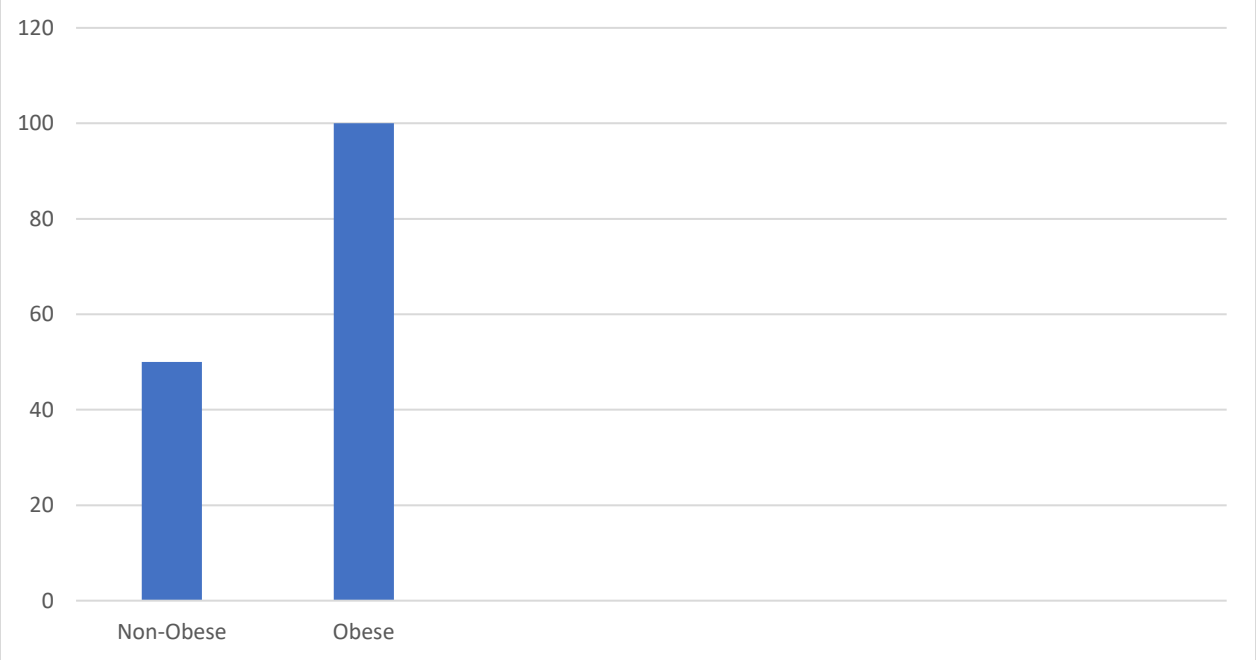

Figure 1: BMI values of non-obese and obese children (0.5-10) age).
Age, weight and height were very high in both groups, correlated with each other (p <0.01).

Biochemical profile: serum PCR levels, α-2 macroglobulin and haptoglobin are shown in Table 2.

Table 2: Serum macroglobulin and PCR concentration in non-obese and obese people (0.5-10 years). Data mean ± SEM (median).

Group	**Non-obese (20)**	**Obese (17)**	**P**
Serum CRP concentration (µg/ml)	0.53±0.16	4.07±0.98	<0.05a
Serum concentration α-2macroglobulin (g/l)	1.15±0.21	2.88±0.72	<0.05a

Significantly different from the non-obese group (student test).

CRP: Average serum concentration in CRP was several times larger than the obese group controls (0.53 vs. 4.07; p <0.05). PCR levels 0–2.75 (0.36) to 0–12 (1.88) respectively obese and obese children. CRP was found to be correlated with BW and BMI in obese people (r = 0.561, p = 0.024; r = 0.513, p = 0.04) and is not obese group (r = 0.687, p = 0.05; r = 0.578, p = 0.024).

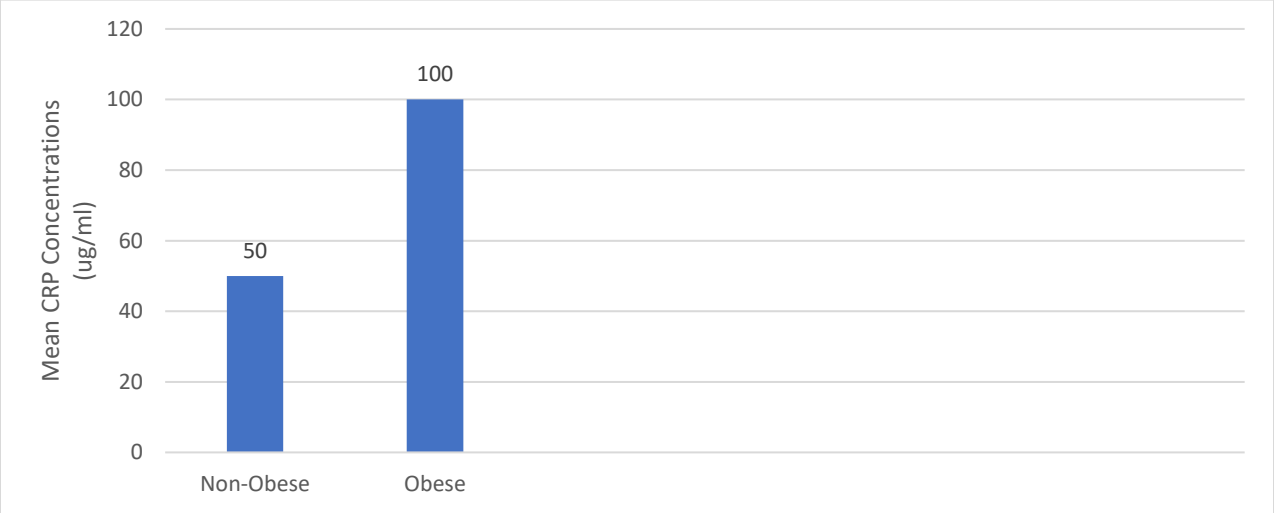

Fig. 2: CRP values in non-obese and obese children (0.5–10 years)

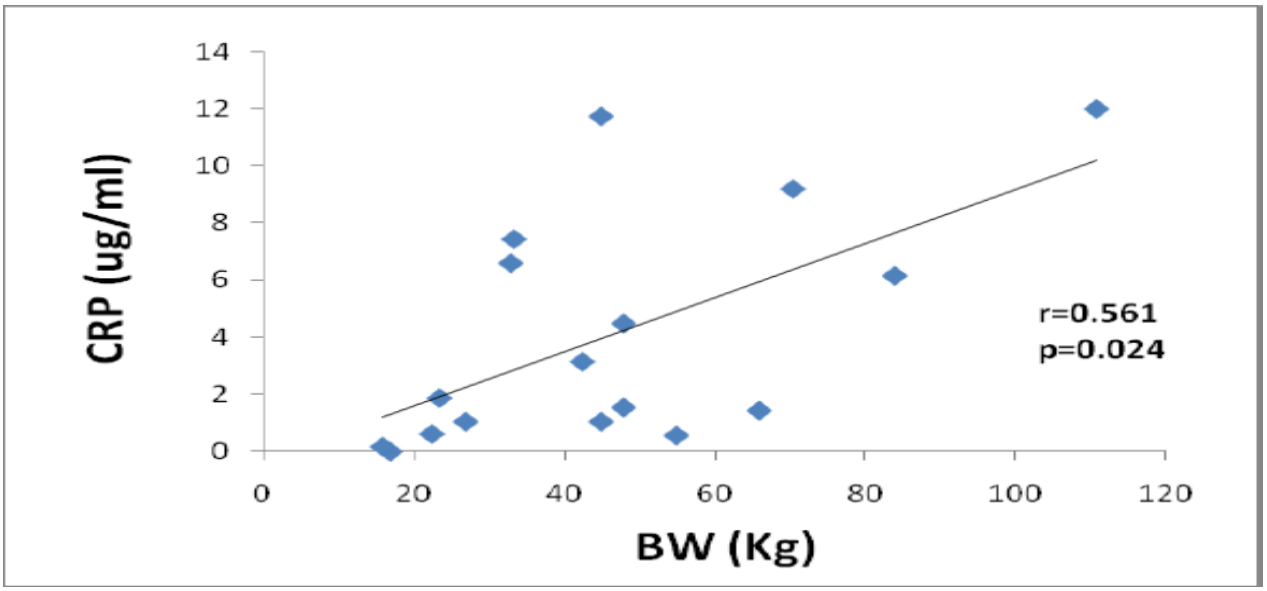

Figure 3: A distribution graph showing the relationship between Pearson PCR and BW levels in the obese group.

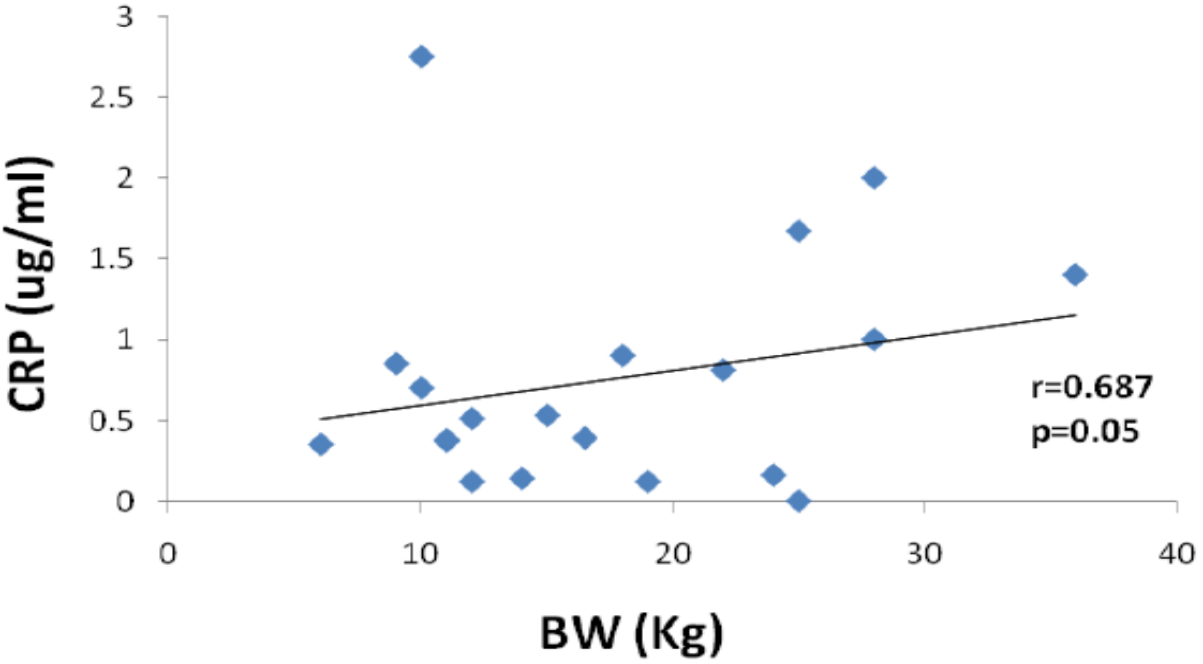

Figure 4: A distribution graph showing the relationship between Pearson PCR and BW levels in the non-obese group.

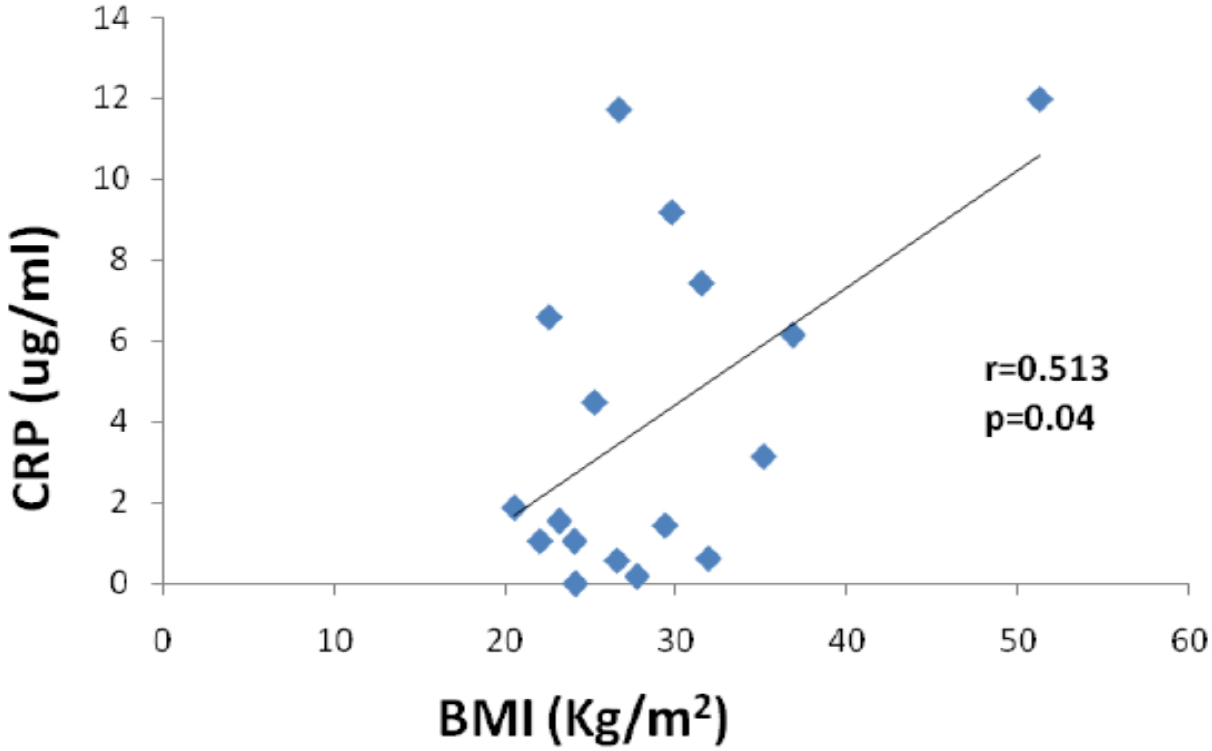

Figure 5: A distribution graph showing the relationship between Pearson's CRP and BMI levels in the obese group.

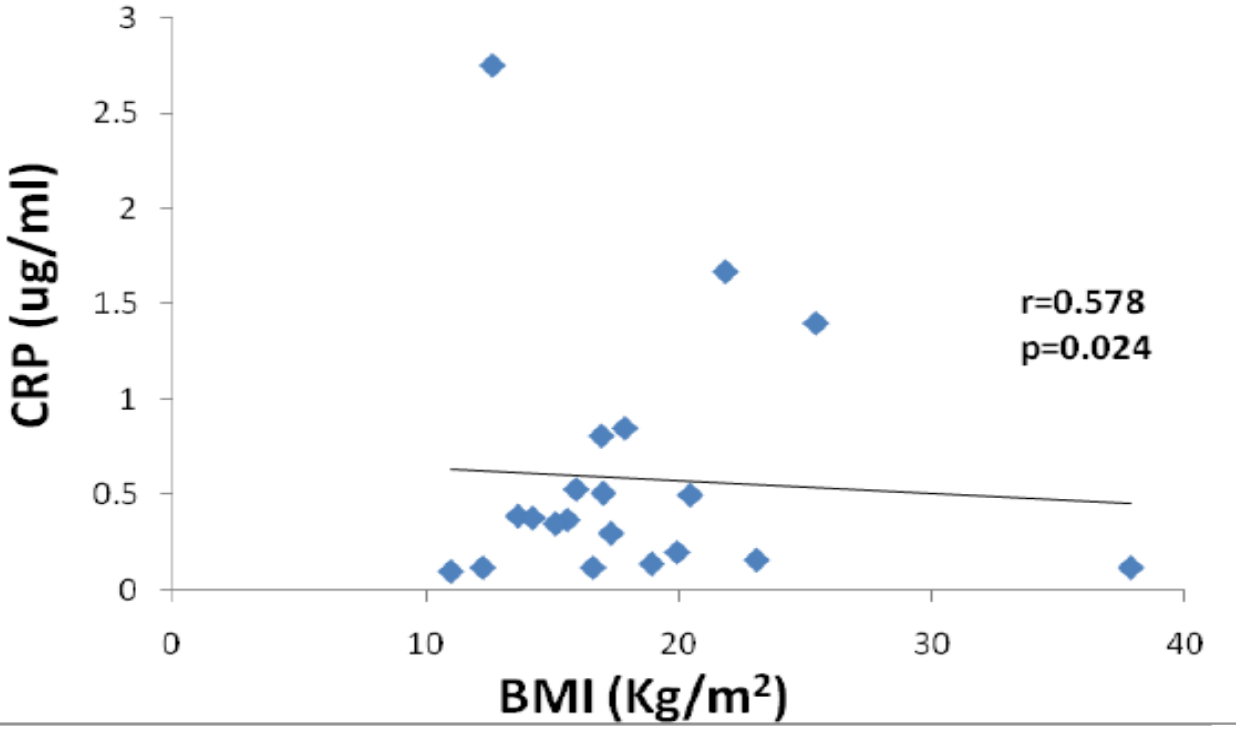

Figure 6: A distribution graph showing the correlation between Pearson's CRP levels and BMI in the non-obese group. A-2 macroglobulin: mean levels of α-2 macroglobulin (2.88 ± 0.72) were expressively higher in the obese group than in non-obese subjects ($p < 0.05$) ($p < 0.05$). Macroglobulin levels in lean individuals ranged between 0.20-3.00 (0.75)

μIU / ml and 0.17-11,00 (1.98) μIU / ml in obese children. It was found that macroglobulin α-2 shows a significant correlation with the level of CRP in the obese group (r = 0.604; p = 0.017).

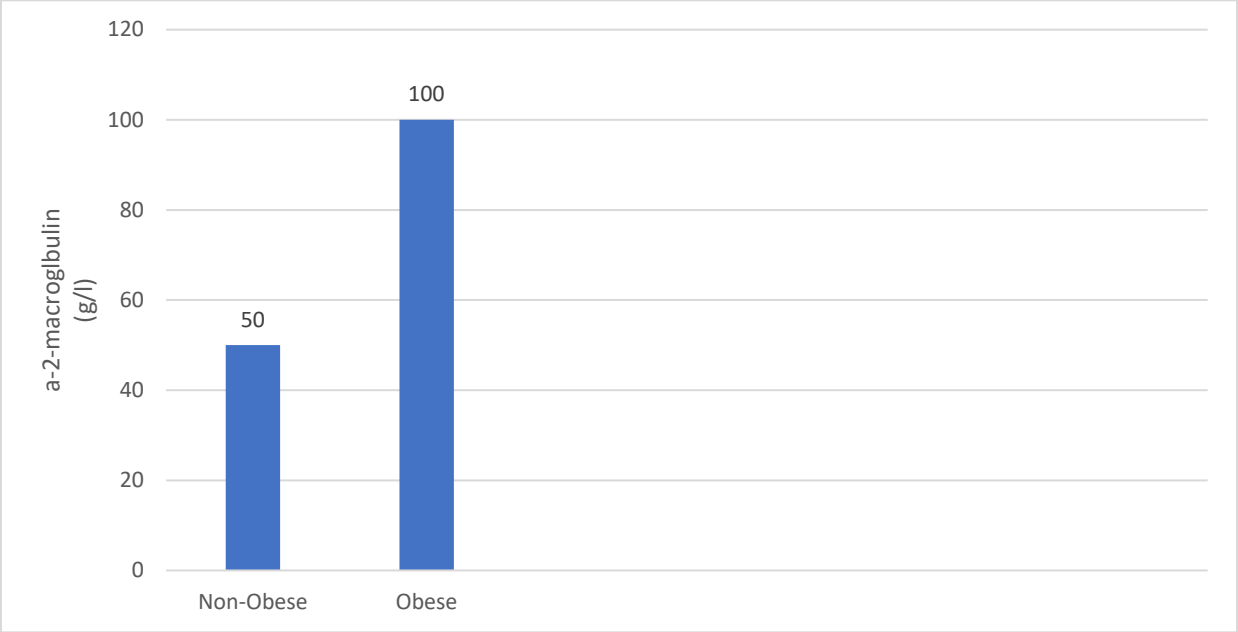

Figure 7: Mean α-2 macroglobulin concentration in obese and non-obese children (0.5-10 years).

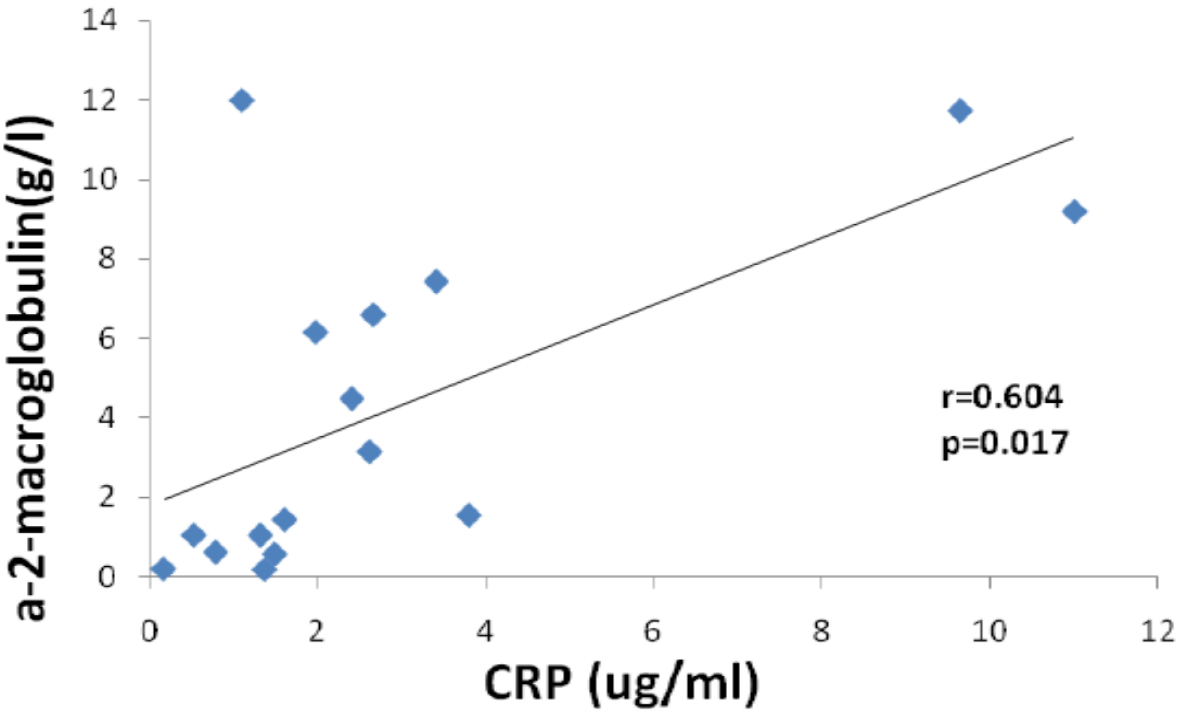

Figure 7: A distribution graph showing the Pearson correlation between α-2 macroglobulin and CRP in the obese group

Haptoglobin: In the obese group, all patients (100%) had elevated hepatoglobin levels, while 50% of non-obese subjects had normal haptoglobin serum levels.

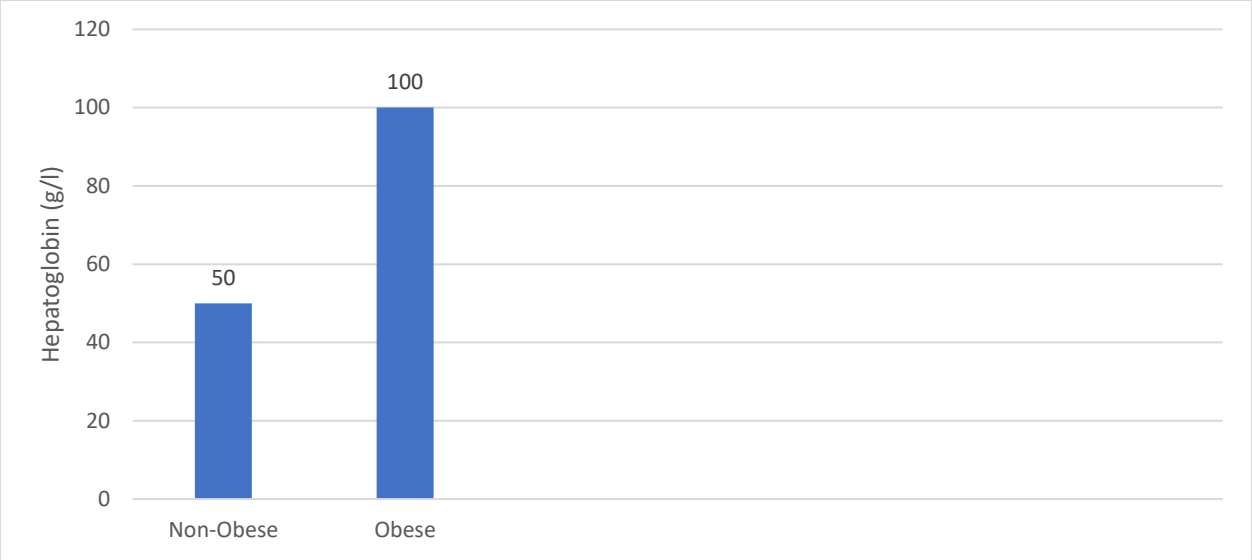

Figure 8: Comparative status of haptoglobin levels in obese and non-obese children (0.5-1010 years).

DISCUSSION:

This study was conducted to examine and analyze markers of the acute pro-inflammatory phase in the rare case of childhood obesity. The relationship between obesity and low-grade inflammation depends largely on the level of obesity. Some studies have associated increases in inflammatory markers with early stages of obesity. Only a few studies show the pathophysiology of childhood obesity.

The children included in the study consisted of two groups according to differences in body weight, height and BMI. Participation criteria were children with BMI << 50-80 in the weak (non-obese) group and children with BMI> 95% in the obese group. In obese children, body weight and height are directly correlated with age in both groups, regardless of the proportion of fat. Despite the previous fact, BMI was strongly associated with age in obese people, but increased significantly compared to the control group. Serum CRP concentration was expressively higher in obese children equated to the control group (4.07 ± 0.98 vs. 0.53 ± 0.16; $p < 0.05$). High levels of CRP have been reported and have been attributed to the development of systemic inflammation in adults, and obesity has often been seen in DM. High CRP levels may be associated with increased fat and cytokine secretion. IL-6 and TNF-α play a key role in this process. We found a positive relationship between CRP and body weight and BMI in both groups. However, other studies may provide information on whether high CRP levels are the cause or consequence of childhood obesity. Haptoglobulin levels were associated with fat mass15. Its role in inflammation was also assessed in people with an increase in BMI. In addition, it is believed that the increase in haptoglobulin synthesis is due to the secretion of IL-6 by adipocytes. All subjects in our study showed very high haptoglobulin values; only 50% of non-obese people had normal range values (0.3–3 g / L). The rest of the weakest children may have some secondary causes, leading to an increase in the level of haptoglobulin reported as an inflammatory marker in altered homeostasis. The mean levels of A2M, another pro-inflammatory protein, were expressively higher in obese people compared to non-obese people of the same age (2.88 ± 0.72 and 1.15 ± 0.21, $p < 0.05$). Some previous studies have tried to measure A2M levels in patients with obesity and metabolic disorder. According to one study, scientists were unable to obtain equivalent results due to low A2M levels in obese patients. We also found a positive relationship between A2M and CRP ($r = 0.604$; $p = 0.017$), indicating an increase in the primary cytokine causing an increase in secondary pro-inflammatory proteins. This study highlights the role of immune markers in the pathogenesis of obesity and insulin resistance, but does not fully explain the causes of obesity and inflammation associated with obesity. Leptin that accompanies him.

CONCLUSION:

The rate of obesity is growing worldwide. Ecological changes continue to arise in established and emerging countries, leading to a worldwide outbreak of morbidity and transience in the coming years. According to this study, obese children at an early age underwent pro-inflammatory stages. Acute phase

stress proteins are also directly related to obesity. We have a polygenic risk of improving obesity and hope that a better considerate of the causes and complex relationships of obesity will lead to better preclusion and treatment.

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