Original Article

Chronic Inflammatory Markers in Overweight and Obese Children: A Cross-sectional Analytical Study

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Abstract

Introduction: Childhood obesity is associated with chronic low-grade systemic inflammation, which results in obesity-related comorbidities. This study compared the inflammatory markers between obese and normal children and assessed obesity-related comorbidities. **Methods:** In this cross-sectional analytical study, 40 obese children between 5-18 years of age were recruited as cases, and an equal number of age and gender-matched normal children as the control. The inflammatory markers-high sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), interleukin-10 (IL-10), and adiponectin were compared between the groups. Hypothyroidism, dyslipidemia, insulin resistance, hypertension, and nonalcoholic fatty liver disease (NAFLD) were screened among obese children. **Results:** We observed a male-female ratio of 1.5:1 in each group. The median hs-CRP between obese and normal children were 2.53 mg/L (0.94,6.85) and 0.77 mg/L (0.19,7.19), and the median IL-6 levels were 3.56 pg/ml (2.17,5.48) and 3.76 pg/ml (1.08,7.91) respectively. The median IL-10 levels between obese and control groups were 2.06 pg/ml (0.35,6.3) and 1.82 pg/ml (0.41,6.5), and the median adiponectin levels between the groups were 8.6 mcg/ml (6.65,16.04) and 9.79 mcg/ml (8.45,11.91) respectively. We didn't observe significant differences in the markers between the groups. Dyslipidemia, insulin resistance, and metabolic syndrome were seen in 80%, 52.5%, and 45% of obese children, respectively. Other comorbidities-NAFLD, hypertension, and hypothyroidism, were observed in 27.5%, 25%, and 7.5% of obese children, respectively. IL-6 had a significant positive correlation with total cholesterol (r = 0.40), LDL levels (r = 0.50), and HDL (r = 0.32). **Conclusion:** There was no difference in inflammatory markers between obese and normal children. Dyslipidemia and insulin resistance were the most common comorbidities.

Keywords: Children, co-morbidity, inflammatory markers, obesity

Introduction

Childhood obesity has become an epidemic in the twenty-first century. The prevalence has increased from 5.4% to 7.8% among children aged 3–15 during the COVID-19 pandemic due to social restrictions, decreased physical activity, reduced access to healthy foods, increased stress, screen time and consumption of processed foods and sugary drinks.[1] Obesity is associated with low-grade systemic inflammation, which activates the immune system and alters the secretion of inflammatory markers. The development of adipose tissue hypertrophy in obese individuals results in intracellular oxidative stress and hypoxia, triggering the release of proinflammatory cytokines like high sensitivity C-reactive protein (hs-CRP), tumour necrosis factor (TNF) and interleukin-6 (IL-6) and decreased production of anti-inflammatory markers like interleukin-13 (IL-13), interleukin-10 (IL-10), interleukin-4 (IL-4) and adiponectin, thus resulting in metabolic syndrome.[2,3] Also, CRP and IL-6 levels were significantly related to the degree of obesity and tend to increase with the number of components of metabolic syndrome.[4] Studies have reported the beneficial effect of anti-interleukin-1 therapy on reducing the HbA1c in type 2 diabetes.[5] Thus, identifying these inflammatory markers in children would help us to use targeted therapy against these

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markers in the future and halt the progression of metabolic syndrome. Several adult studies observed the relationship between inflammation and obesity. However, there is a lack of literature that discusses the inflammatory markers of childhood obesity in the Indian population. This study was designed to compare proinflammatory markers such as hs-CRP and IL-6 and anti-inflammatory markers like IL-10 and adiponectin in obese and overweight children. It also assessed obesity-related comorbidities.

Materials and Methods

This cross-sectional analytical study was done in a tertiary care teaching institute in South India between July 2021 and May 2023. Children aged 5 to 18 years with body mass index (BMI) ≥23 to <27 adult equivalents were defined as overweight, BMI ≥27 adult equivalents was categorised as obesity according to Indian Academy of Pediatrics (IAP) growth charts and enrolled as cases.[6] Age and gender-matched children ±6 months with normal BMI attending the general paediatric outpatient clinic for minor ailments were recruited as controls after resolution of illness. The primary outcome was to compare the proinflammatory markers (hs-CRP, IL-6) and anti-inflammatory markers (IL-10, adiponectin) between case and control groups. The secondary outcomes were to study the comorbidities and correlation between inflammatory markers and comorbidities. Informed consent was obtained from the parents or guardians of the participating children, and verbal assent from children over seven years. We excluded drug-induced obesity, proven genetic and syndromic forms of obesity, and children with recent (within one month) acute febrile illness from our study.

Basic demographic details like age, sex and socioeconomic status (SES) of the recruited children were obtained. Anthropometric measures like BMI, waist circumference (WC), waist-to-height ratio (WHtR) and waist-to-hip ratio (WHR) were measured between the groups. Food intake measurements were calculated based on the 24-hour dietary recall method. Risk factors of obesity, such as daily calorie and protein intake, type of food intake, sleep duration, duration of physical activity and screen time, were calculated based on the parents' history and compared between the groups. We also screened for complications like polycystic ovary syndrome (PCOS), obstructive sleep apnoea (OSA), acanthosis nigricans and psychological disturbances between the groups. Both cases and control underwent aseptic blood withdrawal for estimation of hs-CRP, IL-6, IL-10 and adiponectin. Only obese children were subjected to a comorbid screening like thyroid function tests, homeostatic model assessment for insulin resistance (HOMA-IR), fasting lipid profile and liver enzymes. Ultrasonography of the abdomen was performed to diagnose non-alcoholic fatty liver disease (NAFLD). Metabolic syndrome was defined when three or more of the following criteria are met: Waist circumference ≥90th centile, BP ≥90th centile, Triglycerides ≥110 mg/dl, HDL ≤40 mg/dl and fasting plasma glucose ≥110 mg/dl.[7] Hs-CRP was measured by enzyme immunoassay with DBC kits (Diagnostics Biochem Canada Inc.) with a sensitivity of 10 ng/ml, and a value >3 mg/L was considered elevated. IL-6 levels were measured using 'Abbkine-Human IL-6 ELISA kit' with a two-site sandwich ELISA method, and the detection range of the kit was 3.13 pg/ml–200 pg/ml. IL-6 level >10 pg/ml was considered elevated. IL-10 levels were quantified using the 'Abbkine-Human IL-10 ELISA Kit' employing a two-site sandwich ELISA method with a detection range of 2.35 pg/ml–150 pg/ml. IL-10 level of <4.8 pg/ml was considered reduced. Adiponectin levels were quantified using the 'Abbkine-Human Adiponectin ELISA Kit' employing a two-site sandwich ELISA method. The detection range of the kit was 2 mcg/ml–500 mcg/ml, and the value <3 mcg/ml was considered reduced. Fasting insulin levels were quantified using 'Calbiotech Inc., Insulin ELISA kit' employing a solid phase sandwich ELISA method. The other laboratory parameters were quantified using 'DNA Healthcare ELISA kits.'

Statistical analysis: The sample size was estimated using the mean difference of IL-6 between the two groups, which was 1.1 (±0.6) pg/ml and 0.8 (±0.3) pg/ml in a study conducted by Utsal *et al*.[8] With a power of 80%, an α-error of 5%, and an allocation ratio of 1:1, the estimated sample size was 80 (40 cases and 40 controls) (OpenEpi Version 3). The normality of data was checked using the Shapiro–Wilk test. Continuous variables were compared using the student *t*-test if normally distributed and the Mann–Whitney U test if non-normally distributed. The categorical variables were compared using the Chi-square test (or Fisher's exact test if cell frequency <5). Pearson or Spearman correlation was performed based on the normality of distribution between inflammatory markers and comorbidities. Two-tailed tests were used, and a P value < 0.05 was considered statistically significant. SPSS (Version 16.0. Chicago, SPSS Inc.) was used for data analysis.

Ethical aspects

This study was approved by the Institute Ethics Committee, Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER), Puducherry, India-605006. The approval number was JIP/IEC/2021/106, dated 05.07.2021. Informed consent was obtained from the parents or guardians of the participating children, and verbal assent from children over seven years. This study was conducted in accordance with the ethical standards of the Helsinki Declaration of 1964, as revised in 2013.

Results

Seventy-seven children in the obesity (case group) and 64 in the control group were assessed for eligibility (13-not eligible, 14 refused: 10-met exclusion in the case group; 12-not eligible: 10-refused: 2-met exclusion in the control group), 40 in case group and 40 in the control group were enrolled. Out of 40 cases, 38 (95%) were obese and two (5%) overweight. Children with normal BMI in the case group and abnormal BMI in controls were considered as not eligible. Nineteen (47.5%)

children belonged to 5–10 years, 20 (50%) children belonged to 11–15 years, and one child (2.5%) had an age >15 years. The baseline characteristics are enumerated in Table 1. Among the obesity risk factors, daily calorie ($P < 0.001$), protein intake ($P < 0.001$) and sleep duration per day ($P < 0.001$) were statistically significant between the groups [Table 1]. The average duration of physical activity per day was 0.2 hours in obese children and 0.1 hours in normal children ($P = 0.107$). Only seven (18%) children in the case group had one hour of physical activity, and three (8%) in the control group met the criteria for activity. One child had OSA in our study. None of the children had PCOS or psychological disturbances.

The most common comorbidity was dyslipidemia (80%), followed by insulin resistance (52.5%) and metabolic syndrome (45%) [Table 2]. Eighteen (45%) children with obesity had elevated hs-CRP compared to 16 (40%) children in the control group. IL-6 levels were elevated in 5 (12.5%) obese children and 7 (17.5%) normal children. Twenty-six (65%) children in both the obese and control group had reduced IL-10 levels. None of the children in the groups had low adiponectin levels. There was no significant difference in inflammatory markers between the groups [Table 3]. IL-6 had a moderate degree of positively correlated with total cholesterol (r = 0.40; $P = 0.01$) and with HDL level (r = 0.32; $P = 0.039$). IL-6 had a strong positive correlation with LDL levels (r = 0.50, $P = 0.001$). There was no significant difference in inflammatory markers and anthropometry parameters between children with and without metabolic syndrome.

Discussion

Obese children have hypertrophied adipose tissue, which triggers chronic low-grade inflammation. The inflammatory cytokines induce the comorbidities of obesity. Vazquez *et al*.[9] observed a positive correlation between obesity and socioeconomic status in developing countries, attributed to the early introduction of solid foods, excess consumption of junk foods, reduced physical activity and parental behaviours. Our study has a similar finding, as almost 75% of obese children belonged to upper and middle socioeconomic status.

The multicentre cross-sectional study by Khadilkar *et al*.[10] defined WC >70th centile as a cut-off to screen for the risk of metabolic syndrome, which has a sensitivity of 0.84 and a specificity of 0.85 in boys, a sensitivity of 0.82 and a specificity of 0.85 in girls respectively. 70th centile WC has a positive predictive value of 95% and a negative predictive value of 80.8% for metabolic syndrome in both boys and girls. In our study, 36 (90%) children in obese had WC >70th centile, 15 (37.5%) children had WC >90th centile and 18 (45%) obese children had metabolic syndrome. Among children with metabolic syndrome, 15 (83.3%) had WC >70th centile and 8 (44.4%) had WC >90th centile.

Table 1: Baseline characteristics of the study groups

Parameter	Case ($n=40$)	Control ($n=40$)	*P*-value
*Age, years	10.6 (2.7)	10.6 (2.7)	1.000
Male: Female, *n* (%)	24 (60): 16 (40)	24 (60): 16 (40)	1.000
Socio-economic status, *n* (%) (Modified Kuppuswamy scale)			
I-Upper	1 (2.5)	3 (7.5)	0.514
II-Upper middle	14 (35)	9 (22.5)	
III-Lower middle	15 (37.5)	15 (37.5)	
IV-Upper lower	10 (25)	12 (30)	
V-Lower	0 (0)	1 (2.5)	
Weight for age, *n* (%)			
Above normal (>2z)	20 (50)	0	-
Height for age, *n* (%)			
Tall stature (>2z)	3 (7.5)	0	0.114
Stunting (<-2z)	3 (7.5)	1 (2.5)	
Waist circumference, *n* (%)			
>70th centile	36 (90)	0	-
>90th centile	15 (37.5)		
Waist-to-height ratio (>0.5), *n* (%)	35 (87.5)	0	-
Waist-to-hip ratio (>0.9), *n* (%)	37 (92.5)	1 (2.5)	-
**Calorie intake (kcal/kg/day)	53.61±7.66	36.35±8.03	<0.001
**Protein intake per day (g/kg/day)	4.6 (4.3–5.3)	4.1 (3.8–4.5)	<0.001
Non-vegetarian, *n* (%)	38 (95)	33 (82.5)	0.077
**Sleep duration/day (hours)	8 (7.75–8.25)	7 (7–8)	<0.001
**Screen time/day (hours)	3 (2–4)	3.5 (3–4)	0.107
Acanthosis nigricans, *n* (%)	5 (12.5)	0	0.021
Obstructive sleep apnoea, *n* (%)	1 (2.5)	0	1.000

All data is presented in *mean (SD), **median (IQR) or as stated *n* (%)

Table 2: Comorbid conditions and laboratory parameters in the obese (case) group

Parameter	Case (*n*=40)
Comorbid conditions*, *n* (%)	
Dyslipidaemia	32 (80)
Insulin resistance	21 (52.5)
Metabolic syndrome	18 (45)
Non-alcoholic fatty liver disease	11 (27.5)
Hypertension	10 (25)
Hypothyroidism	3 (7.5)
Laboratory parameters	
#TSH, µIU/mL	2.89 (1.96)
TSH >6.2 µU/L, *n* (%)	3 (7.5%)
#Free T4, ng/dL	1.04 (0.22)
#Free T3, pg/mL	3.56 (1.18)
Total cholesterol, mg/dL	157.60 (37.38)
Total cholesterol ≥200 mg/dL, *n* (%)	6 (15%)
#LDL, mg/dL	107.9 (29.3)
LDL ≥130 mg/dL, *n* (%)	6 (15%)
#HDL, mg/dL	35.03 (8.50)
HDL <40 mg/dL, *n* (%)	28 (70%)
##Triglycerides, mg/dL	116.50 (94–145)
Triglycerides ≥150 mg/dL, *n* (%)	8 (20%)
##Fasting blood glucose, mg/dL	91 (82.5 – 106.5)
Fasting Insulin, µU/mL	33.7 (16.5–80.3)
##HOMA-IR	5.63 (3.06–21.68)
##ALT, IU/L	26 (22.5–35)
ALT >40 IU/L, *n* (%)	5 (12.5%)
##GGT, IU/L	20.5 (17–26)
GGT >30 IU/L, *n* (%)	4 (10%)

All data presented as *n* (%), other than #mean (SD) or ##median (IQR). Abbreviations: TSH – Thyroid stimulating hormone; T4 – Thyroxine; T3 – Triiodothyronine; LDL – Low-density lipoprotein; HDL – High-density lipoprotein; HOMA-IR - Homeostatic model assessment of insulin resistance; ALT - Alanine transaminase; GGT – Gamma glutamyl transferase. *The same child may have one or more comorbid conditions. Hence, the number may not equal to 40

Table 3: Inflammatory markers between the study groups

Parameter	Case (*n*=40)	Control (*n*=40)	*P*-value
High-sensitivity C-reactive protein, mg/L	2.53 (0.94–6.85)	0.77 (0.19–7.19)	0.108
Interleukin-6, pg/mL	3.56 (2.17–5.48)	3.76 (1.08–7.91)	0.850
Interleukin-10, pg/mL	2.06 (0.35–6.3)	1.82 (0.41–6.5)	0.810
Adiponectin, mcg/mL	8.60 (6.65–16.04)	9.79 (8.45–11.91)	0.290

All data is presented as median (IQR)

Similarly, WHtR and WHR are considered tools to measure central obesity. Sarna *et al*.[11] suggested that WHtR >0.5 indicates central obesity, which is linked to childhood morbidity more than BMI, as the latter measures general adiposity. Another study proposed that WHR >0.9 can also be used to define central obesity.[12] 87.5% and 92.5% of children had a WHtR >0.5 and WHR >0.9, in our study, respectively. In a systematic review, Mistry *et al*.[13] observed that risk factors like decreased physical activity, prolonged screen time and excess consumption of a calorie-dense diet were significantly higher in obese children compared to normal children. We also observed a significant difference in calorie intake and sleep duration in the obese group compared to the control group. In contrast, the duration of physical activity was longer and the median screen time was less in the obese group than in the control group. This may be because of parental counselling in the obese group.

Narang *et al*.[14] described that OSA occurs in up to 60% of obese children, and it independently increases the cardiovascular burden of these children. We observed OSA in only one child. The low incidence was attributed to not using polysomnography for diagnosis. Clinical manifestations of insulin resistance like PCOS and acanthosis nigricans are commonly observed in obesity. Obesity at 14 years of age is associated with a 61% higher risk of having PCOS symptoms at a later age,[15] and the prevalence of acanthosis nigricans ranges from 49.2% to 58.2%.[16] None of the children in our study had PCOS, and only five obese children (12.5%) had acanthosis nigricans.

In our study, 45% of obese children had elevated hs-CRP compared to 40% of the control group, which was not statistically significant. Chakraborty *et al*.[17] revealed that CRP and leptin levels increased, whereas adiponectin levels were reduced with adiposity. Jain *et al*.[18] observed that hs-CRP and IL-6 were elevated in half of the obese children. Serum adiponectin was low in 16.5% of children and was found to have a significant inverse correlation with waist circumference. IL-6 levels were elevated in 5 (12.5%) obese children and 7 (17.5%) control group in this study. This was comparable to Galcheva *et al*.,[19] where IL-6 levels are not significantly associated with adiposity variables in prepubertal children.

Kulshrestha *et al*.[20] concluded that IL-10 levels are significantly lower in obese children with metabolic syndrome. On the contrary, Tam *et al*.[21] observed an elevation of IL-10 levels in overweight and obese girls compared to normal-weight girls. We observed no significant difference in IL-10 and serum adiponectin levels between the groups.

We analysed the correlation between the levels of inflammatory markers and comorbid obesity parameters. In this study, IL-6 levels positively correlated with total cholesterol and LDL levels. Chang *et al*.[22] also observed that IL-6 levels correlate positively with LDL levels and negatively with HDL levels. Zamudio *et al*.[23] described that 48.5% of adult patients with extreme obesity had hypothyroidism, and TSH levels significantly correlated with the increase in IL-6, leptin and E-selectin. Juliaty *et al*.[24] reported that hs-CRP had a significant positive correlation with triglyceride and a negative correlation with HDL levels. Tawfic *et al*.[25] also observed a significant positive correlation between adiponectin and HDL levels, but there was no correlation between adiponectin and other lipid parameters. There was a positive correlation between IL-6 and HDL in this study, which needs to be explored in further studies.

We observed that 80% of obese children had dyslipidemia, 52.5% had insulin resistance, 27.5% had ultrasonographic

evidence of fatty liver changes, and 7.5% had hypothyroidism. Elmaoğulları *et al*.[26] described the prevalence of dyslipidemia as 43%, with hypertriglyceridemia being the most common abnormality. This differs from our study, in which 70% had low HDL, whereas only 20% of obese children had hypertriglyceridemia. They described a vicious cycle between insulin resistance and dyslipidemia that alters lipid metabolism, resulting in NAFLD. The increased proinflammatory cytokines in obesity increase deiodinase activity, causing hypothyroidism. Thus, among children with dyslipidemia, 63% had NAFLD and 28.95% had insulin resistance. Hypertension was observed in 25% of obese children in our study. Mohan *et al*.[27] concluded a high prevalence of sustained hypertension and obesity among urban school children, with a prevalence of obesity and hypertension in urban children at 11% and 8.4%, respectively.

The limitation of the study was the small sample size, which may not power enough to measure the difference in all inflammatory markers. We did not use a fibro scan or ultrasound-based grading to follow up on the fatty liver changes. Also, this study did not investigate other novel obesity-related inflammatory markers like calprotectin.[28] To our knowledge, this is the first study that explored the level of inflammatory markers in the South-Indian population. We also recruited normal children for comparison. However, we did not find any difference in inflammatory markers. A similar study in a larger population with a larger sample size might help us identify significant differences in these markers.

Conclusion

To conclude, there was no difference in levels of inflammatory markers between obese and normal children. Dyslipidemia and insulin resistance are the most commonly observed comorbidities, followed by NAFLD and hypertension. Almost half of the obese children had metabolic syndrome.

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Nil.

Authors' contributions

CGD had full access to all the data in the study and took responsibility for the integrity of the data and the accuracy of the data analysis: CGD–Conceptualized the study. CGD, RGK, GPS, JS, RR–Study concept, design and protocol development. RGK–Acquisition, analysis and first draft of the manuscript. JS, GPS–Supervision of biochemical analysis of samples. All authors contributed critical revision of the manuscript and approved the final version of the manuscript.

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Conflicts of interest

There are no conflicts of interest.

Data availability

Authors declare that data related to the study will be made available upon request.

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