Journal of Saidu Medical College 2011; 1(1)

PREVALENCE OF OBESITY IN THE ADOLESCENT POPULATION IN MINGORA CITY, DISTRICT SWAT.

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ABSTRACT:

Background: The dramatic increase in the prevalence of overweight and obesity and its resultant co morbidities are associated with significant health and financial burdens, warranting strong and comprehensive prevention efforts. The objective of the present study was to evaluate the prevalence of obesity in the adolescent population of Mingora city, district Swat.

Methodology: In the present study, both boys and girls between 10-20 years of age were included. The prevalence of obesity was detected in 200 subjects by using body mass index. Anthropometric parameters of obese subjects were compared with non-obese group.

Results: The prevalence of obesity in the adolescent boys was 6.7 % and that in adolescent girls was 8.35. The prevalence was higher in female subjects, compared to male subjects. Obese subjects had significantly higher weight, body mass index, compared to non-obese group.

Conclusion: Prevalence of obesity is higher in female subjects which is related to body mass index..

KEYWORDS Weight (Wt), Height (Ht), Body mass index (BMI).

INTRODUCTION:

Obesity is defined as body mass index (BMI) greater than or equals to the 95th percentile for age and gender, overweight as a BMI greater than or equal to the 85th percentile but less than the 95th percentile, and normal weight as BMI less than the 85th percentile[1].

BMI is recognized as one of the most useful indices for adiposity both in children and adults. BMI is determined by dividing weight (wt) in kilogram by height (ht) in meters squared. It is highly correlated with weight (0.8-0.9). BMI is also highly correlated with body fat (0.7-0.8). Based on data for children 6-15 years, World Health Organization (WHO) has suggested the following cut-off points for BMI as broad guidelines for defining obesity in both sexes[2].

Age (years)	BMI (kg/m^2)
≤14	19-20
15	25
≥16	28

While Sardinha et al recommends optimum Body Mass Index threshold values to define obesity as follows[3].

BMI threshold	Boys	Girls
Age 10-11	19.0	19.6
Age 12-13	19.4	21.2
Age 14-15	24.0	21.9

In normal individuals, the percentage of body tissue that is adipose tissue varies by gender (greater in post pubertal females than males) and age (about 12% at birth, increasing to 25% at 5 months, then decreasing to 15% to 18% during puberty)[4].

The changes in body composition that occur during adolescence have been well described. In boys, fat-free mass tends to increase, body fat as a percentage of body weight decreases, and fat tends to be deposited abdominally. In girls, both fat and fat-free mass increase, fat-free mass as a percentage of body weight decreases, and fat tends to be deposited in the buttocks. In both sexes, factors that contribute to the quantity of body fat and abdominal fat distribution appear to increase

The risk of subsequent complications[5]. Obesity is a significant health problem, with medical and psychological consequences for children and adolescents[6].

Only a small percentage of childhood obesity is associated with a hormonal or genetic defect, with the remainder being idiopathic in nature. An endogenous cause for obesity can be either suspected or eliminated from the differential diagnosis in virtually all children based on a careful history and physical examination. In most cases, this should negate the need for expensive and unnecessary laboratory evaluations[7].

Growth failure characterizes endogenous obesity. Children with an associated genetic or hormonal syndrome are short, usually at or under the 5th percentile of height for age. Conversely, children with idiopathic obesity are taller, usually above the 50th percentile. Even the onset of a hormonal abnormality in a previously tall child will be marked by a significantly slower rate of growth compared with the child's previous growth curve.[7] Parental obesity is the most important risk factor for childhood obesity. Twin, adoption, and family studies indicated that inheritance is able to account for 25% to 40% of inter-individual difference in adiposity[8].

It is possible that the functional organization of the hypothalamic or other neurogenic feeding centers in an obese person is different from that of a non-obese person. Also, there may be abnormalities of neurotransmitters or receptor mechanism in the neural pathways of the hypothalamus that control feeding[9].

The hormone, Leptin is made almost exclusively in adipose tissue. Low plasma levels of leptin and insulin (e.g. during fasting and weight loss) increase food intake and decrease energy expenditure by stimulating neuropeptide Y (NPY) synthesis, and perhaps by inhibiting sympathetic activity and other catabolic pathways. High leptin and insulin concentrations (e.g., during feeding and weight gain) decrease food intake and increase energy expenditure through release of melanocortin and corticotrophin-releasing hormone. The absence of a functional hormone (or its receptor) leads to uncontrolled food intake and resulting obesity[10].

Widespread reports indicate that the prevalence of obesity among children and adolescents has been increasing in recent years, just as it has in adults. A recent report of the initial results of 1999 National Health and Nutrition Examination Survey indicates that prevalence rates have increased even further, to 13% of children aged 6 to 11years and 14% of adolescents aged 12 to 19 years[11].

Obese children under three years of age without obese parents are at low risk for obesity in adulthood, but among older children, obesity is an increasingly important predictor of adult obesity, regardless of whether the parents are obese. Parental obesity increases the risk of adult obesity among both obese and non-obese children less than 10 years of age[12].

SUBJECTS AND METHODS:

Two hundred subjects were randomly selected to detect the prevalence of obesity. Out of these, obese subjects were taken and their anthropometric parameters were compared with those of non-obese (age and sex matched) subjects.

Both males and females adolescent between ages 10-20 years were included in the study.

Subjects were excluded from study participation that had a medical history of disease other than overweight or were taking any medication known to affect metabolism.

Health Scale was used to measure weight and height. Weight was assessed at 2 different points during interview, and the 2 were averaged for these analyses. It was measured to the nearest 0.5 kg. Participants were advised to wear normal clothing without shoes, socks, and belts. Height was also assessed at 2 different points during interview, and the two readings were averaged for these analyses. It was measured to the nearest 0.1 cm[13].

Body mass index (BMI) was determined by

by dividing weight (wt) in kilograms by hight (ht). in Meters squared (BMI=kg/m^2). Body mass index percentile charts were used to determine obesity in boys and girls.[14]

RESULTS:

Table I: Anthropometric parameters and prevalence of obesity in adolescent boys.

Age group yrs	Age (mean)	Weight (kg)	Height (m)	BMI (kg/m^2)	Prevalence
10-15 (n=50)	12.5 ± 1.74	39.77 ± 12.48	1.45 ± 0.11	18.64 ± 3.68	6.67 %
16-20 (n=50)	18 ± 1.44	60.54 ± 9.90	1.71 ± 0.033	20.71 ± 3.56	6.67 %

Graph I: Anthropometric parameters and prevalence of obesity in adolescent boys.

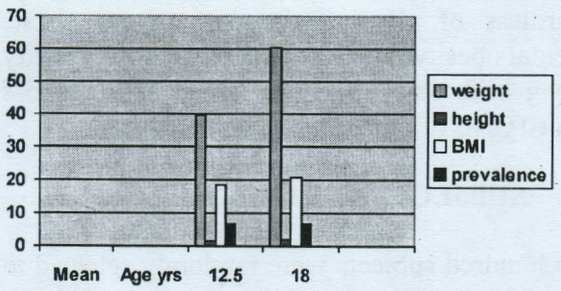


Table I, Graph I: Show anthropometric parameters and the prevalence of obesity in adolescent boys. Mean age (10 15 years) 12.5 1.74: weight, 39.77 12.48, height, 1.45 0.11, BMI, 18.64 3.68, and the prevalence was 6.67 %. Mean age (16-20 years) 18 1.44, weight, 60.54 9.90, height, 1.71 0.033, BMI, 20.71 3.56 and the prevalence was 6.67 %. As shown, the prevalence remained the same from age 10 through 20. The values are expressed as mean SD (standard deviation).

Table II: Anthropometric parameters and prevalence of obesity in adolescent girls.

Age group (years)	Age (mean)	Weight (kg)	Height (m)	BMI (kg/m^2)	Prevalence
10-15 (n=50)	12.5 ± 1.74	40.57 ± 11.21	1.48 ± 0.78	18.29 ± 3.85	6.67 %
16-20 (n=50)	18 ± 1.44	53.17 ± 11.81	1.57 ± 0.027	21.57 ± 4.40	10 %

Graph II: Anthropometric parameters and prevalence of obesity in adolescent girls.

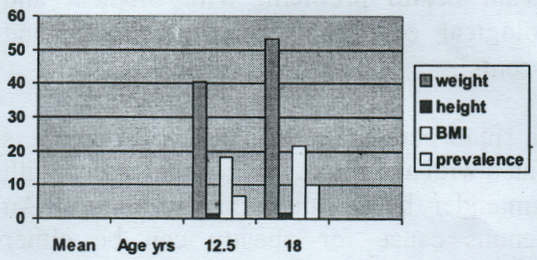


Table II, Graph II: Show anthropometric parameters and the prevalence of obesity in adolescent girls. Mean age (10 15 years) 12.5 1.74: weight, 40.57 11.21, height, 1.48 0.78, BMI, 18.29 3.85, and the prevalence was 6.67 %. Mean age (16-20 years) 18 1.44, weight, 53.17 11.81, height, 1.57 0.027, BMI, 21.57 4.40 and the prevalence was 10 %. The values are expressed as mean SD (standard deviation).

As shown, the prevalence of obesity in children and adolescents was the same for both sexes till the age of 15 years. In the late adolescents, the prevalence increased with increase in age and was higher in female than male.

Table III: Comparison of anthropometric parameters of non-obese group with obese group.

Parameters	Group "A" (non-obese)	Group "B" (obese)
Weight	40 ± 7.27	62 ± 8.43 **
Height	1.45 ± 0.08	1.44 ± 0.07
BMI	19± 1.29	29.9± 1.41 **

Values are giver as mean SD,* p0.05 (significant), ** p 0.001 (highly significant).

Graph III: Comparison of anthropometric parameters of non-obese group with obese group.

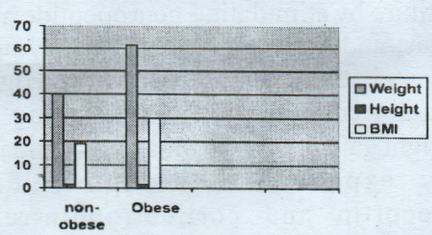


Table III, Graph III: The anthropometric parameters such as weight, height, BMI of group "A" (non-obese) were compared with group B (obese). The mean weight of group "A" was 40 7.27 and that of group "B" was 62 8.43. The p-value was 0.001 (highly significant). The mean height of group "A" was 1.45 0.08 and that of group "B" was 1.44 0.068. The difference in height of two groups were statistically non significant. The mean BMI of group "A" was 19 1.29 and that of group "B" was 29.9 1.41. The p-value was 0.001 (highly significant).

DISCUSSION

Prevalence of obesity varies amongst countries depending upon environmental and behavioral changes brought about by economic development, modernization and urbanization. The variation in prevalence of obesity epidemic in various races and communities of the world may be attributed to heredity, age, sex, and diet, eating patterns, life style and/or behavior.[15]

Obesity tracks from childhood to adulthood.[16] At least 30% of obesity begins in childhood. Conversely 50 to 80% of obese children become obese adults.[17]

The present study showed that obesity was prevalent in both adolescent boys and girls in Mingora city of Swat district. The prevalence of obesity in children and adolescents was the same for both sexes till the age of 15 years. In the late adolescents, the prevalence increased with increase in age and was higher in female than male.

Sheila et al, also observed higher rates than the present study and reported that prevalence of overweight amongst Australian children has increased from 11% in 1985 to 20% in 1995. According to WHO, at least 50% of adults and 20% of children in U.K. and U.S.A. are currently overweight.[18]

Roberts et al reported that the United States has one of the highest obesity rates in the world and is the first nation to have people who are both impoverished and obese. The obesity epidemic is also increasing in Europe, Asia and throughout the Americas: as the Indian newspaper The Tribune states:

Obesity also plagues Middle Eastern countries, with 35 per cent of Egyptians considered obese, a greater proportion than the population in the USA at 20 per cent.[19] Evidence from the National Health and Nutrition Examination survey (NHANES) in the United States and the national study of health and growth in the United Kingdom shows an increasing prevalence of overweight and obesity in young children and adolescents.[20,21]

It is concluded that the significant prevalence of childhood obesity emerges in school-aged children. It is, therefore, suggested that early recognition of excessive weight gain relative to linear growth should become routine in pediatric ambulatory care settings. BMI should be calculated and plotted periodically and intervention should begin in the first years of school because obesity appears to be well established by then.

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