Psychopathology and its effect on treatment compliance in pediatric obesity patients

Yasemen Taner[1], Ayça Törel-Ergür[2], Gülbağ Bahçıvan[3], Mahinur Gürdağ[4]

Departments of [1]Child and Adolescent Psychiatry, [2]Pediatric Endocrinology, [3]Psychiatry, and [4]Nutrition and Dietetics, Ufuk University Faculty of Medicine, Ankara, Turkey

SUMMARY: Taner Y, Törel-Ergür A, Bahçıvan G, Gürdağ M. Psychopathology and its effect on treatment compliance in pediatric obesity patients. Turk J Pediatr 2009; 51: 466-471.

Obesity is a common health problem in children and adolescents and has life-threatening physical complications as well as psychological consequences, including negative self-image, low self-esteem and social difficulties. Psychiatric disorders, especially depression and anxiety disorders, are present at higher rates in obese patients. The aim of this study was to investigate the presence and type of psychopathology in a group of obese children and to determine the effect of comorbid psychiatric disorders on treatment compliance. Fifty-four obese patients were evaluated by clinical interviews as well as Schedule for Affective Disorders and Schizophrenia for School-Age Children, Present and Lifetime Version (KIDI-SADS-PL) for psychiatric diagnosis. Fifty percent of the sample was found to have psychopathology and treatment compliance was found to be poor in the group with comorbid psychiatric disorders. This shows that child and adolescent psychiatrists should be included as team members while treating pediatric obese patients.

Key words: obesity, psychopathology, children, adolescents, treatment compliance.

Obesity is defined by the increase in mass and content of fat cells resulting in increase in adipose tissue[1].

Although there is no common consensus on the definition of obesity in terms of amount of adipose tissue, current opinion in determining obesity in children and adolescents is by the use of body mass index (BMI) and the percentiles[1]. BMI is associated with long-term complications related to obesity[2]. To determine obesity in children and adolescents, BMI should be plotted on a graph in relation to age to determine the exact percentile; 85th to 95th percentile is conceptualized as obesity, whereas 95th percentile or higher is morbid obesity[3,4].

Epidemiological data reveal that the prevalence of overweight adolescents is at least 10% for adolescents aged 12 to 17, and nearly 20% more are at risk for becoming overweight. Especially in developed countries, excessive nutrition in children and adolescents is known to be a threat to healthy life[5]. There is evidence that childhood obesity is associated with an increased risk of adult obesity[6,7].

There are various explanations for obesity, including child and family factors. Adoption and twin studies show a genetic tendency for obesity, and the best predictor of childhood obesity is found to be obesity in the biological parents[8,9]. Prader-Willi syndrome[10], mutations in melanocortin receptor gene (MC4R)[11], and congenital leptin deficiency[12] are specific genetic conditions that are found to play a role in the emergence of obesity. Most often, genetic vulnerability is thought to occur through polygenetic mechanisms operating in children in families predisposed to obesity[5]. Children at greater risk include those with difficult temperament, those overfed during infancy and those breastfed for a shorter time[13-15]. Easily available, calorie-dense food combines with an increasingly sedentary lifestyle to produce an alarming increase in obesity and concomitant risk for its medical complications. In some individuals, genetic susceptibility is the fact that leads to vulnerability to obesity. Type 2 diabetes, problems in cholesterol metabolism, hypertension, joint disease, and even increased risk of cancer are the medical complications of obesity[5].

Not only physical but also psychological consequences of obesity have been reported recently. Childhood obesity is reported to result in negative self-image, low self-esteem and social difficulties, especially in the adolescent period[5]. In a Swedish sample composed of 136 girls whose ages ranged from 15 to 20, somatoform disorders, affective disorders, anxiety disorders, and pain disorders were reported to be higher in obese children[16]. In another research investigating the psychiatric morbidity in obese children aged from 5 to 15 years, obese children revealed significantly higher internalized and externalized questionnaire scores and poorer social skills[17]. Fifty-eight percent of this sample had a comorbid psychiatric diagnosis, with anxiety disorders being the most common. Psychiatric disorders were particularly pronounced in those obese children whose parents were disturbed, but there was no correlation between severity of obesity in the child or his (her) parents and frequency of psychiatric disorders. In a study conducted in Turkish obese adolescents, more than half of the clinical obese adolescents (16/30) were found to have a Diagnostic and Statistical Manual of Mental Disorders (4^{th} ed) (DSM-IV) diagnosis, often involving major depressive disorder ($n = 10$)[18].

In other studies conducted in obese children, this population was found to have significantly higher eating-disordered cognitions and more binge-eating problems and problematic behaviors than normal weight children[19,20].

Childhood obesity is not only a risk factor for psychiatric disorders in children and adolescents, but also a predictive variable for future psychological disturbances in obese adult populations. Patients with early onset obesity have a greater frequency and higher levels of emotional distress and psychiatric symptomatology than patients with late-onset obesity[21].

Anxiety disorders and depression are the most studied psychiatric disorders in obese children. Some studies support the higher incidence of depression[22,23] and anxiety disorders[17] in obese children and adolescents, but there are also findings that obese children and adolescents do not present more depressive symptoms, lower self-esteem or anxiety[24-26].

The aim of this study was to investigate the incidence and impact of psychiatric disorders in children and adolescents with obesity.

Material and Methods

This cross-sectional study was performed in an urban university setting in Turkey by the Pediatric Endocrinology and Child and Adolescent Psychiatry Departments.

Subjects

The subjects included 27 girls and 27 boys (age range: 7 to 16 years) who had been admitted to the Pediatric Endocrinology Department with a complaint of obesity and who were defined as obese by the pediatric endocrinologist (A.T.E.) and had been followed up by both the Pediatric Endocrinology Department and the Nutrition and Dietetics Department by an experienced dietician (M.G.) for at least three months. All subjects with a diagnosis of obesity between the ages of 7-16 were consulted with the Child and Adolescent Psychiatry Department. Subjects with another endocrinological disorder, including clinical and subclinical hypothyroidism and those with other chronic medical illnesses or mental retardation were excluded from the study.

Protocol

After parents and subjects received a complete description of the study and were informed about the scope of the psychiatric investigation, parents and children and adolescents who accepted to participate in the study were enrolled. Informed consent was taken orally from all of the subjects. A data form including sociodemographic data and physical measures was completed for each subject by a research assistant of psychiatry (G.B.). After this procedure, the patients and their parents who accepted to join the study were referred to the child and adolescent psychiatrist (Y.T.) who was blind to treatment compliance of the participants. Psychiatric disorders were screened according to DSM-IV-TR[27] criteria and with the Schedule for Affective Disorders and Schizophrenia for School-Age Children: Present and Lifetime Version (K-SADS-PL)[28], which is a semi-structured interview used to further verify the diagnosis. After the psychiatric evaluation, patient compliance with the obesity treatment, which was applied by the Pediatric Endocrinology and Nutrition and Dietetics Departments, was evaluated. Treatment compliance evaluation was based

on the children's presenting for the weekly visits regularly, following their diets given by the Nutrition and Dietetics Department, and the presence of the expected weight loss of 500-700 g/week. Patient treatment compliance was considered as good-excellent for patients who came regularly to weekly visits, followed the diets and as a result had the expected weight loss.

Schedule for Affective Disorders and Schizophrenia for School-Age Children, Present and Lifetime Version

The Schedule for Affective Disorders and Schizophrenia for School-Age Children, Present and Lifetime Version (K-SADS-PL) is a semi-structured instrument developed by Kaufman and colleagues[28] to screen psychopathology in children and adolescents aged between 6-18. It screens psychopathology by gathering information from both parents and their offspring. The psychiatric conditions included in this instrument are: mood, psychotic, anxiety, disruptive behavioral, elimination, eating, and tic disorders, and alcohol and other substance use disorders. The reliability and validity of K-SADS-PL was reported in Turkey in 2004[29].

Data Analysis and Statistics

Comparisons with respect to categorical measures were performed using chi-square tests, or Fisher's exact tests if there were cells with expected frequencies of less than five. Numerical variables were compared by t-test and considered significant if different at the $p<0.05$ level. All statistics are reported two-tailed; standard deviations are reported throughout. Data was evaluated by SPSS-11.0.

Results

Out of 56 obese subjects, 54 were included in the analysis; one mother declined to participate in the study and another patient was excluded due to missing information in the data form.

Subjects had a mean age of 12.25 (SD=2.98), and 50% (n=27) were male. The mean weight and height of the subjects were 63.75 kg (SD=19.91 kg) and 149.29 cm (SD=17.30 cm), respectively. The number of subjects attending primary school, secondary school and high school were 27 (50%), 9 (16.7%) and 18 (33.3%), respectively. School performance was bad and moderate in 21 (31.5%) children and adolescents and good and excellent in 33 (68.5%).

Children and adolescents with a history of obesity and psychiatric illness in the family constituted 53.7% (n=29) and 31.5% (n=17) of the sample, respectively.

Obese children with a psychopathology constituted half of the sample and seven of them had multiple psychopathologies. The distribution of the psychopathology types are presented in Table I.

The group with psychopathology was not statistically different from the group without psychopathology in terms of age, weight, height, sex, family history of psychiatric disorder, and the degree of obesity (obesity/morbid obesity). Obese children without any psychopathology had better treatment compliance and better school success compared to obese children with psychopathology. Patients with a family history of obesity had less psychopathology compared to patients without a family history of psychopathology (Table II).

Table I. Type of Psychopathology in Obese Children

Psychopathology type	Frequency	Percent
Attention deficiency and hyperactivity disorder	3	5.6
Depression	8	14.8
Social phobia	7	13.0
Obsessive compulsive disorder	2	3.7
Attention deficit/hyperactivity disorder, encopresis and enuresis nocturna	1	1.9
Encopresis, enuresis nocturna, simple phobia	1	1.9
Depression, encopresis, enuresis nocturna	1	1.9
Obsessive compulsive disorder, social phobia	2	3.7
Depression, social phobia	2	3.7
No psychopathology	27	50.0

Table II. Group Comparison Based on Presence or Not of Psychopathology in Terms of Age, Weight, Height, Sex, School Success, Family History of Psychiatric Disorder or Obesity, Medication Compliance, and Type of Obesity

Variables		Children with psychopathology N=27		Children without psychopathology N=27		Test (p)
		Mean	(SD)	Mean	(SD)	t-test (p)
Age		12.85	2.82	11.66	3.07	1.47 (0.146)
Weight		67.18	17.38	60.33	21.95	1.27 (0.209)
Height		151.96	15.76	146.62	18.63	1.13 (0.261)
		N	(%)	N	(%)	X^2 (p)
Sex	Girls	15		12		0.667 (0.414)
	Boys	12		15		
School success	Bad-moderate	16		1		19.31 (0.001*)
	Good-excellent	11		26		
Family history of obesity	Present	10		19		6.03 (0.014*)
	Absent	17		8		
Family history of psychiatric illness	Present	11		6		2.14 (0.143)
	Absent	16		21		
Treatment compliance	Bad-moderate	21		13		5.08 (0.024*)
	Good-excellent	6		14		
Obesity type	Morbid obese	21		17		1.42 (0.233)
	Obese	6		10		

*$p<0.05$, SD: Standard deviation. N: Number.

Discussion

This was a study undertaken to examine psychopathology and its effect on treatment compliance in a group of Turkish children and adolescents followed by the Departments of Pediatric Endocrinology and Nutrition and Dietetics for obesity. Psychiatric disorders were evaluated using clinical interviews as well as K-SADS-PL, by gathering data from both the patients and their parents.

Findings of this study revealed that half of the obese children and adolescents in this sample had comorbid psychiatric disorders. This result supports the finding that psychiatric comorbidity is common among obese children and adolescents[17,18]. Although a higher incidence of psychopathology in obese children and adolescents has been documented, disagreement exists regarding the nature of the psychopathology. Depression and social phobia were the two most common psychiatric disorders in this sample of patients, and this is consistent with the results of studies that found depression and anxiety disorders as the most common psychopathologies in obese children and adolescents[17,18,22,23]. Psychological and social factors are known to play an important role in the development of social phobia[30] as well as depressive disorders[31]. Higher incidence of comorbid psychiatric disorders in obese children in this sample may have resulted from negative self-image, low self-esteem and social difficulties reported in obese children in a previous study[5]. Therefore, obesity can be regarded as a risk factor for the development of psychopathologies.

When the patients were grouped in terms of having obesity or morbid obesity, there were no differences in psychiatric diagnoses according to the degree of obesity. This finding is consistent with the study of Vila and colleagues[17], which also found no correlation between the degree of obesity and the presence of psychopathology.

In contrast to former research that reported that the presence of psychopathology in obese youngsters is highly correlated with the parents' psychopathology[17], psychiatric disorders in this sample were not different between the samples grouped in accordance with the presence or not of psychopathology in the parents. Related to the findings of this study, it can be suggested

that obesity might be a more important risk factor for psychopathology than genetic inheritance of the psychiatric disorder.

The high ratio of family history of obesity in the first-degree relatives of children and adolescents was concurrent with the finding that genetic vulnerability plays a role in development of obesity[8,9]. It was an interesting finding that obese children and adolescents with obese family members had less psychiatric disorders compared to obese patients without obese relatives. This distinction reveals that environmental factors are important in perception of the social problems brought by obesity. That is, obese children and adolescents with obese family members seem to adapt better to their physical appearance and social difficulties resulting from obesity than the obese children and adolescents without obese relatives.

Obese children and adolescents with comorbid psychiatric diagnosis had lower school performance. It is a known fact that psychiatric disorders have a negative effect on social functioning as well as school performance[27]. As lower school performance has an additional devastating effect on the self-esteem of children and adolescents with obesity, it becomes important to evaluate those patients for comorbid psychiatric diagnosis.

Treatment compliance is negatively affected by the presence of psychopathology in obese children and adolescents. In addition to the pediatric endocrinologist and dietician, child and adolescent psychiatrists should also be involved in the team management of patients with obesity.

Since the rate of psychopathology is quite high in obese children and adolescents, we suggest a psychiatric consultation and follow-up in this group of patients, not only for the purpose of treatment but also to prevent comorbid psychiatric disorders.

REFERENCES

1. Rodopman Arman A, Yılmaz S. Obesity and child psychiatry. In: Soykan AS, Taner YI (eds). Children and Adolescent Mental Health and Disorders. İstanbul: Golden Print; 2007: 587-601.
2. Barlow SE, Dietz WH. Obesity evaluation and treatment: Expert Committee Recommendations. The Maternal and Child Health Bureau, Health Resources and Services Administration and the Department of Health and Human Services. Pediatrics 1998; 102: E29.
3. Ogden CL, Flegal KM, Carroll MD, Johnson CL. Centers for Disease Control and Prevention 2000 growth charts for the United States: improvements to the 1997 National Center for Health Statistics version. Pediatrics 2002; 109: 45-60.
4. Troiano RP, Flegal KM, Kuczmarski RJ, Campbell SM, Johnson CL. Overweight prevalence and trends for children and adolescents. The National Health and Nutrition Examination Surveys 1963 to 1991. Arch Pediatr Adolesc Med 1995; 149: 1085-1091.
5. Waller DA. Eating disorders. In: Sexson SB (ed). Child and Adolescent Psychiatry (2nd ed). USA: Blackwell Publishing Ltd; 2005: 154-162.
6. Epstein LH, Wing RR. Behavioral treatment of childhood obesity. Psychol Bull 1987; 101: 331-342.
7. Epstein LH, Wing RR, Valoski A. Child obesity. Pediatr Clin North Am 1985; 32: 363-380.
8. Gallaher MM, Hauck FR, Yang-Oshida M, Serdula MK. Obesity among Mescalero preschool children. Am J Dis Child 1991; 145: 1262-1265.
9. Sorensen TI, Price RA, Stankard AJ, Schulsinger F. Genetics of obesity in adult adoptees and their biological siblings. BMJ 1989; 298: 87-90.
10. Mascari MJ, Gottlieb W, Butler MG, et al. The frequency of uniparental disomy in Prader-Willi syndrome. Implications for molecular diagnosis. New Engl J Med 1992; 326: 1599-1607.
11. Farooqi IS, Keogh JM, Yeo GS, Lank EJ, Cheetham T, O'Rahilly S. Clinical spectrum of obesity and mutations in the melanocortin 4 receptor gene. New Engl J Med 2003; 348: 1085-1095.
12. Farooqi IS, Jebb SA, Langmack G, et al. Effects of recombinant leptin therapy in a child with congenital leptin deficiency. New Engl J Med 1999; 34: 879-884.
13. Carey WB, Hegvik RL, McDevitt SC. Temperamental factors associated with rapid weight gain and obesity in middle childhood. J Dev Behav Pediatr 1988; 9: 194-198.
14. Kramer MS, Barr RG, Leduc DG, Boisjoly C, Pless IB. Infant determinants of childhood weight and adiposity. J Pediatr 1985; 107: 104-107.
15. Mogan J. Parental weight and its relation to infant feeding patterns and infant obesity. Int J Nurs Stud 1986; 23: 255-264.
16. Buddeberg-Fisher B, Klaghofer R, Reed V. Associations between body weight, psychiatric disorders and body image in female adolescents. Psychother Psychosom 1999; 68: 325-332.
17. Vila G, Zipper E, Dabbas M, et al. Mental disorders in obese children and adolescents. Psychosom Med 2004; 66: 387-394.
18. Erermis S, Cetin N, Tamar M, Bukusoglu N, Akdeniz F, Goksen D. Is obesity a risk factor for psychopathology among adolescents? Pediatr Int 2004; 46: 296-301.
19. Isnard P, Michel G, Frelut ML, et al. Binge eating and psychopathology in severely obese adolescents. Int J Eat Disord 2003; 34: 235-243.
20. Tanofsky-Kraff M, Yanovski SZ, Wifley DE, Marmarosh C, Morgan CM, Yanovski JA. Eating-disordered behaviors, body fat, and psychopathology in overweight and normal-weight children. J Consult Clin Psychol 2004; 72: 53-71.

21. Mills JK, Andrianopoulos GD. The relationship between childhood onset obesity and psychopathology in adulthood. J Psychol 1993; 27: 547-551.

22. Ericson SJ, Robinson TN, Haydel KF, Killen JD. Are overweight children unhappy? Body mass index, depressive symptoms, and overweight concerns in elementary school children. Arch Pediatr Adolesc Med 2000; 154: 931-935.

23. Wallace WJ, Sheslow D, Hassink S. Obesity in children: a risk for depression. Ann N Y Acad Sci 1993; 699: 301-303.

24. Britz B, Siegfried W, Ziegler A, et al. Rates of psychiatric disorders in a clinical study group of adolescents with extreme obesity and in obese adolescents ascertained via a population based study. Int J Obes Relat Metab Disord 2000; 24: 1707-1714.

25. Cortese S, Cuzzolaro M, Maffeis C, et al. Depressive symptoms and low self-esteem in obese children and adolescents. Minerva Pediatr 2005; 57: 65-71.

26. Pastore DR, Fischer M, Friedman SB. Abnormalities in weight status, eating attitudes, and eating behaviors among urban high school students: correlations with self-esteem and anxiety. J Adolesc Health 1996; 18: 321-329.

27. American Psychiatric Association. Diagnostical and Statistical Manual of Mental Disorders, 4th Edition, Text Revision (DSM-IV-TR). Washington DC: APA; 2000.

28. Kaufman J, Birmaher B, Brent D, et al. Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version (K-SADS-PL): initial reliability and validity data. J Am Acad Child Adolesc Psychiatry 1997; 36: 980-988.

29. Gokler B, Unal F, Pehlivanturk B, Cengel Kultur E, Akdemir D, Taner Y. Reliability and Validity of Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version-Turkish Version (K-SADS-PL-T). Turk J Child Adolesc Ment Health 2004; 11: 109-116.

30. Taneli Y. Specific phobia and social phobia. In: Soykan AS, Taner YI (eds). Children and Adolescent Mental Health and Disorders. İstanbul: Golden Print; 2007: 475-492.

31. Ağaoglu B. Major depressive disorder. In: Soykan AS, Taner YI (eds). Children and Adolescent Mental Health and Disorders. İstanbul: Golden Print; 2007: 361-372.