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Original Research

Association between night eating syndrome in overweight and obese children 10-17 years of age and dyslipidemia

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Abstract

Background and Aims: Night eating syndrome **(**NES) is one of the reasons for increased prevalence of obesity. Nevertheless, there is lack of knowledge about the influence of NES on lipid metabolism in children. The objective of the study was to determine the influence of NES on the lipid profile in overweight and obese children 10–17 years of age. **Material and Method:** A population of 110 children with excess weight and obesity was enrolled in the study. NES was diagnosed based on existing criteria. Anthropometric measurements were performed for all participants. Lipid profile assessments included detection of TC, HDL-C, and TG with an enzymatic method and formula assessment for LDL-C, VLDL-C, and non-HDL-C levels. **Results:** NES was diagnosed in 21.82% of children. Dyslipidemia was detected in 70.83% of children with NES and in 44.19% subjects in the group without NES ($p = 0.04$). Atherogenic level of HDL-C was diagnosed in 45.83% subjects with NES compared with children without NES 15.12% ($p = 0.001$). Multivariate regression analysis has found significant association between NES and evidence of dyslipidemia and HDL-C level in the population of obese and overweight children ($p < 0.05$). **Conclusions:** NES is one of the reasons for changes in lipid profile in children; therefore, screening for eating disorders in overweight and obese children must be implemented in routine practice to reduce the risk of dyslipidemia and its consequences.

Keywords: children, lipids, night eating syndrome, obesity.

Background and Aims

Night eating syndrome (NES), as a type of eating disorder, has been studied for more than 60 years among different weight groups [1]. NES is characterized by abnormal timing of food intake, which could be accompanied by changes in sleep patterns [1]. NES was found to be genetically determined; the chances of having such type of eating disorder are 5 times higher in children who have relatives with the same problem [1, 2, 3]. The prevalence of NES varies. It depends on the age and the weight of the patient, and his socioeconomic status [3]. Nevertheless, the exact cause is still unknown. It is also unknown what is primary, i.e., whether NES causes obesity or obesity causes NES [1, 3]. Notably, eating disorders in children and adolescents are difficult to recognize and diagnose; many of them can be described as Eating Disorders Not Otherwise Specified [4]. Despite extensive research on the subject, the problem of NES as a type of eating disorder in pediatric population is still ambiguous and debatable.

It should be emphasized, that both NES and lipid metabolism has circadian patterns [5]. Disturbances of the rhythm of daily activity causes failure of circadian rhythm, which is associated with the occurrence of metabolic syndrome, components of which are abdominal type of obesity and dyslipidemia [6]. Night eating and sleep disorders as the components of NES could lead to changes in circadian secretion of melatonin

and leptin and, as a result, all disturbances in metabolic activity [5]. Notably, lipids make up 80% from 15% of all metabolites with circadian rhythm [5]. At the same time, it should be emphasized that the diurnal metabolic rhythms are influenced not only by internal factors, but also by a number of external factors, in particular, exposure to light at night, duration and quality of sleep, food intake and physical activity [2, 5].

Therefore, NES could be a potential risk factor of metabolic disorders. The influence of eating disorders on the lipid profile has been studied in adult population while pediatric subjects were not taken into account [7]. However, studies on dyslipidemia and NES are still lacking. This is why the research community is deeply interested in dyslipidemia and its connections with eating disorders. Quite recently, manifestations of this problem in children have received considerable attention. Therefore, it has become the topic of our research.

The aim of the study was to determine the impact of NES on lipid metabolism in overweight and obese children 10–17 years of age.

Material and Method

Study design and patients

A population of 90 obese (81.82%) and 20 overweight (18.18%) children, 10–17 years of age, was recruited by the study. All children were assessed in Ternopil Regional Hospital, Ukraine. Informed consent was obtained from each child's legal representative(s). The study protocol conformed to the ethical guidelines of Helsinki's Declaration and was approved by the Bioethics Commission of I. HorbachevskyTernopil State Medical University (Protocol 24, dated 27.08.2014).

Laboratory, anthropometric and clinical data collection

Physical examination was performed in all study subjects: weight, height, waist circumferences (WC), and hip circumferences (HC) were measured in internationally accepted units (kg, m). Body mass index (BMI) and waist/hip ratio (W/H ratio) were also calculated.

The diagnosis of NES was based on the following criteria [8, 9]:

1. Increased food intake in the evening and/or at night, which was manifest by one or both of the following: a) at least 25% of the total amount of food consumed after the evening meal; b) at least two episodes of food intake at night per week.
2. Being aware of eating too late.
3. At least three of the following characteristics should be present: a) lack of appetite in the morning and/or skipping breakfast four or more times during a week; b) a strong craving for food between dinner and bedtime and/or during the night; c) presence of a sleep disorder (trouble falling asleep and/or episodes of insomnia) on four or more nights per week; d) believing/feeling like one should eat before going to bed; e) presence of depression and/or low mood in the evening.
4. Presence of an eating disorder associated with significant distress and/or impaired body function.
5. Minimal duration of disrupted eating patterns of three months.
6. Presence of an eating disorder unrelated to certain medical disorders, abuse, or dependence on taking psychoactive substances, the effects of certain medications, and mental disorders.

Total cholesterol (TC), high density lipoprotein (HDL-C), and triglycerides (TG) were determined in the serum using an enzyme method (reagents "Human GmbH", Germany). The following formulas were used to find the levels of low density lipoprotein cholesterol (LDL-C), very low density lipoprotein (VLDL-C), and non-HDL-cholesterol (non-HDL-C):

$$\text{LDL-C (mmol/l)} = \text{TC(mmol/l)} - (\text{TG(mmol/l)}/2.2 + \text{HDL-C[mmol/l]});$$
$$\text{VLDL-C (mmol/l)} = \text{TG(mmol/l)}/2.2;$$
$$\text{non-HDL-C (mmol/l)} = \text{TC(mmol/l)} - \text{HDL-C (mmol/l)}.$$

According to expert recommendation, acceptable, borderline, and high/low (for

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HDL-C) levels of lipids have been identified [10]. The following lipid profile parameters were seen as acceptable lipid levels: total cholesterol (TC) lower than 4.40 mmol/l (170 mg/dl), low density lipoproteins (LDL-C) less than 2.85 mmol/l (110 mg/dL), non-HDL-C lower than 3.11 mmol/l (120 mg/dL), triglycerides lower than 1.0 mmol/l (90 mg/dL), high density lipoproteins (HDL-C) higher than 1.17 mmol/l (45 mg/dL). The following values were seen as borderline: TC: 4.40—5.17 mmol/l (170–199 mg/dL); LDL-C: 2.85–3.36 mmol/l (110–129 mg/dL); non-HDL-C: 3.11-3.75 mmol/l (120-144 mg/dL); triglycerides: 1.02–1.46 mmol/l (90–129 mg/dL); HDL-C: 1.04—1.17 mmol/l (40—45 mg/dL). The levels of lipids equal to or higher than 5.18 mmol/l (200 mg/dL) for TC, 3.37 mmol/l (130 mg/dL) for LDL-C, 3.76 mmol/l (145 mg/dL) for non- HDL-C, 1.47 mmol/l (130 mg/dL) for triglycerides and lower than 1.04 mmol/l (40 mg/dL) for HDL-C were regarded as atherogenic and dyslipidemia was diagnosed.

Statistical analysis

Data management and analysis was performed using STATISTICA 7.0 software. Comparison of two independent samples with normal distribution was carried out using Student's t-test; Mann-Whitney U-test was used for parameters with abnormal distribution. The differences were considered significant at $p < 0.05$. Two-tailed Fisher exact test and Pearson Chi-square test were used to analyze the frequency tables. Odds ratio (OR) and its 95% confidence interval (95% CI) were calculated to evaluate the impact of the factor on the outcome. Logistic regression was performed to find the association between NES and parameters of lipid metabolism.

Results

Night-eating syndrome was diagnosed in 24 children (21.82 % [95% CI 13.98; 32.46]). There were no gender-specific differences concerning the frequency of NES: the disorder was found in 17.65% (95% CI 9.88; 29.11) boys and in 36% girls (95% CI 16.46; 68.34) (Fisher exact test, two-tailed $p = 0.06$): the male/female ratio in the NES group was 1.67/1 (62.50% and 37.50%, respectively).

Socio-demographic and clinical characteristics of children recruited in the study are presented in Table 1. Anthropometric parameters (body weight, BMI, WC, HC) were significantly higher in group of children with NES (table 1) (Student's t-test, $p < 0.05$), despite absence in morbidity rate (overweight/obesity) in the compared groups (Fisher exact test, two-tailed $p > 0.05$). The prevalence of NES was 20% (95% CI 5.45; 51.21) among overweight children and 22.22% (95% CI 13.57; 34.32) among their peers with obesity (Fisher exact test, two-tailed $p = 0.99$). Taking into account that WC was significantly higher among night eaters, we have investigated the impact of NES on abdominal obesity. In the group with NES, AO was found in 75.00% of children versus 83.72% in the non-NES group (Fisher exact test, two-tailed, $p = 0.37$).

Dyslipidemia was diagnosed in 70.83% of children with NES (95% CI 41.26; 100.00) and in 44.19% subjects in the group without NES (95% CI 31.27; 60.65): Fisher exact, two-tailed, $p = 0.04$; OR = 3.07; 95% CI 1.15–8.15; $p = 0.02$ (NES = exposure, dyslipidemia = outcome) (table 2).Among serum lipid levels only HDL-C has significant differences across the groups with and without NES (table 2). It should be emphasized that HDL-C levels were higher in the group without NES: 1.25 (1.14; 1.38) versus 1.10 (0.98; 1.31) in the group of subjects diagnosed with NES ($p = 0.01$ for Mann-Whitney U test).

Analysis of serum lipid levels depending on cardiovascular risk has shown that levels of TC, LDL-C, non-HDL-C and TG were not significantly different across the groups with and without NES ($p > 0.05$ for Pearson Chi-square test) (table 3). Nevertheless, acceptable levels of HDL-C were registered in 33.33% of children with NES (95% CI 14.39; 65.68) and in 68.60% of subjects without NES (95% CI 52.23; 88.49) ($p = 0.001$). A reverse pattern was found for low level of HDL-C: it was diagnosed in 45.83% subjects in the group with NES (95% CI 22.88; 82.01); in the group without NES low levels of HDL-C were detected less often: in 15.12% of children (95% CI 8.05; 25.85) ($p = 0.001$). There were no statistical differences in borderline levels of HDL-C (table 3). The

Table 1: Socio-demographic and clinical variables of overweight and obese children, 10-17 years of age, depending on the presence of night eating syndrome.

Parameters		Groups		P
		NES(+)	NES(–)	
age, years		16.17 ± 0.64	14.98 ± 1.68	0.002*
gender	boys	15 (17.65%)	71 (82.35%)	0.060
	girls	9 (36.00%)	17 (64.00)	
weight status	overweight	4 (20.00 %)	16 (80.00%)	0.999
	obesity	20 (22.22%)	70 (77.78)	
Weight, kg		99.63 ± 23.17	89.31 ± 15.63	0.012*
Height, m		1.72 ± 0.10	1.72 ± 0.11	0.932
BMI, kg/m^2		33.54 ± 5.84	30.02 ± 3.27	<0.001*
WC, cm		102.83 ± 13.63	96.41 ± 9.37	0.009*
HC, cm		111.06 ± 11.20	106.73 ± 6.59	0.020*
W/H ratio		0.93 ± 0.07	0.90 ± 0.05	0.068

NOTES. NES+ - group of children with diagnosed NES; NES- - group of children without NES, BMI – body mass index; WC – waist circumference; HC – hip circumference; W/H ratio – waist/hip ratio;
* = statistically significant results.

Table 2: Lipid profile in children 10–17 years of age depending on the presence of night eating syndrome (Me [Q25; Q75]).

Indices (mmol/l)	Groups		Mann-Whitney U test, p
	NES(+)	NES(–)	
Total cholesterol	3.90 (3.42; 4.97)	4.08 (3.41; 4.52)	0.93
LDL-cholesterol	2.29 (1.76; 2.94)	2.13 (1.65; 2.71)	0.36
VLDL-cholesterol	0.47 (0.36; 0.66)	0.52 (0.41; 0.70)	0.20
Non-HDL cholesterol	2.80 (2.24; 3.69)	2.71 (2.15; 3.25)	0.43
Triglycerides	1.03 (0.79; 1.52)	1.17 (0.90; 1.55)	0.20
HDL cholesterol	1.10 (0.98; 1.31)	1.25 (1.14; 1.38)	0.01*

NOTES. NES(+) - group of children with diagnosed NES; NES(-) - group of children without NES
* = statistically significant results.

calculation of odds ratio has shown that NES may influence HDL-C levels. For low HDL-C levels, the following values were obtained: OR = 4.75; 95% CI 1.75–12.87 and p = 0.002. For acceptable HDL-C levels, the following values were obtained: OR = 0.23; 95% CI 0.09–0.60 and p = 0.003. For borderline HDL-C levels, the following values were obtained: OR = 1.35; 95% CI 0.43–4.23 and p = 0.602.

One of the NES criteria is the time of last food intake before going to sleep. Therefore, we have analyzed the influence of time between last food intake and bedtime and parameters of lipid status. Our study has found that HDL-C levels were closely related to the time of food intake before going to sleep (r = 0.20; p = 0.04) (table 4).

Results of regression analysis (univariate and multivariate [after adjustment for weight status]) have confirmed association between NES and evidence of dyslipidemia in the population of obese and overweight children: AOR = 3.09; 95 %

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Table 3: Serum lipid levels depending on the presence of night eating syndrome.

Indices	NES +		NES –		p-value
	n	%	n	%	
Dyslipidemia	17	70.83	38	44.19	0.04*
Total cholesterol					
Acceptable	16	66.67	61	70.93	0.47
Borderline	3	12.50	15	17.44	
High	5	20.83	10	11.63	
LDL-cholesterol					
Acceptable	17	70.83	66	76.74	0.83
Borderline	4	16.67	11	12.79	
High	3	12.50	9	10.47	
Non-HDL cholesterol					
Acceptable	13	54.17	57	66.28	0.46
Borderline	5	20.83	16	18.60	
High	6	25.00	13	15.12	
Triglycerides					
Acceptable	12	50.00	33	38.37	0.35
Borderline	4	16.67	27	31.40	
High	8	33.33	26	30.23	
HDL-cholesterol					
Acceptable	8	33.33	59	68.60	0.01*
Borderline	5	20.83	14	16.28	
Low	11	45.83	13	15.12	

Notes. * – statistically significant result.

Table 4: The correlation between last food intake before going to bed and serum lipid levels in groups with and without night eating syndrome.

Parameters	Total (n = 110)	
	Spearman, r	*p*-value
Total cholesterol	0.12	0.20
LDL cholesterol	0.08	0.41
VLDL cholesterol	0.05	0.59
Non-HDL cholesterol	0.08	0.40
Triglycerides	0.05	0.60
HDL cholesterol	0.20	0.04*

NOTES. * – statistically significant result.

CI 1.14–8.41 ($p < 0.05$). Among all lipids only HDL-C was significantly related to the presence of NES: AOR = 6.39; 95 % CI 2.06–19.87 ($p < 0.05$) (table 5).

Discussion

The data in the available literature highlights the impact of eating behaviors on obesity in children [11, 12]. It should be emphasized that many previous studies involved adult subjects of different age groups, sex, and ethnicity [13, 14]. The issue of NES in pediatric populations is a topic of ongoing discussion and research.

According to our results, the prevalence of NES was 21.82% (95% CI 13.98; 32.46), which is consistent with the data obtained by scholars in the US, where NES occurs at a rate of 6.5–21% among young individuals 9–19 years of age [14]. At the same time, as reported by investigators in the UK, this eating disorder occurs with a frequency of 8.9–14% among persons with obesity [9]. The latter differences in the prevalence of NES are

Table 5: Association between NES and parameters of lipidogram.

Indices	Univariate regression analysis			Multivariate regression analysis		
	unadjusted OR	95% CI	p	adjusted OR	95% CI	*p*
Dyslipidemia (presence/absence)	3.07	1.14–8.25	0.027*	3.09	1.14–8.41	0.027*
TC (high/acceptable)	0.52	0.15–1.78	0.297	0.53	0.16–1.80	0.305
LDL-C (high/acceptable)	0.77	0.18–3.22	0.594	0.81	0.19–3.46	0.773
non-HDL cholesterol (high/acceptable)	0.49	0.16–1.56	0.227	0.50	0.16–1.59	0.236
TG (high/acceptable)	1.05	0.36–3.03	0.925	1.05	0.36–3.04	0.925
HDL-C (low/acceptable)	6.24	2.06–18.86	0.001*	6.39	2.06–19.87	0.002*

NOTES: adjusted OR was calculated after adjustment for weight status (overweight/obesity).
* – statistically significant result.

attributable to the lack of representativeness of the samples selected for the study. In particular, this reflects the different age demographics of study subjects, as well as the differences in nutrition habits and food cultures in people of different national and ethnic backgrounds [14]. In addition, some scientists exclude children under the age of 13 years from evaluation for NES because the diet of children in this age group is mostly defined and controlled by their parents [14].

The results of our study have not found any gender differences concerning NES. Nevertheless, the literature reports that eating disorders are more typical for girls compared with boys; their frequency is 6 times higher in girls and three times higher in women [15, 16]. The absence of gender-specific differences concerning NES in our study can be explained by a small population of study subjects diagnosed with NES. Sex-dependent differences in eating behaviors are the result of hypothalamic-pituitary-gonadal axis function and the influence of other hormones and neuropeptides, such as ghrelin, cholecystokinin, glucagon-like peptide-1, glucagon, insulin, amylin, apolipoprotein A-IV, fatty-acid oxidation, leptin, serotonin, melanocyte stimulating hormone, neuropeptide Y, agouti-related peptide, melanin-concentrating hormone, and dopamine [16]. Both estrogens and androgens stimulate food intake: estrogens increase daily food intake through more frequent eating, while androgens increase meal sizes; the validity of these findings was supported by data obtained in gonadectomy [16].

In our study, NES was diagnosed in every fifth overweight child or child with obesity. The findings in this study are similar to the results obtained in an adult population study, where NES was considered a risk factor for obesity [9, 17, 18, 19]. However, studies by R.H. Striegel-Moore et al. and Calugi S. et al. negated the relationship between eating at night and being overweight in children [20, 21]. Such differences could be attributable not only to the different age, gender, and ethnic composition of study groups, but also to the time of assessment, because during the school vacation, which is typically three months long, the nutritional patterns of children change significantly [14, 18]. Other factors that could make a difference include dietary patterns in the families, as well as parental education and socio-economic status of the family [14, 18].

The data in literature suggests NES to be closely related to metabolic syndrome [19]. One of the core components of metabolic syndrome includes HDL-C levels. Similar findings were obtained in our study, i.e., the level of HDL-C was closely associated with NES in children 10–17 years of age. We have found children with NES to have a potential threefold increase in the risk of dyslipidemia compared to children without this eating disorder ($p = 0.02$). Other authors reported night snacking to increase TC and LDL-C levels [22]. This has been attributed to the changes in the circadian rhythm of cholesterol metabolism found in night eating. Changes in meal times impact the expression of cholesterol

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7-alpha-hydroxylase and 3-hydroxy-3-methylglutaryl-coenzyme Areductase, leading to hypercholesterolemia [22].

Previous researches have studied that cholesterol level and LDL-C have diurnal variations. In the same time data about circadian patterns of HDL-C level is still debatable; some of the studies deny this pattern, another has written about a very small-amplitude rhythm (2–4%) for HDL-C [5]. Notably, changes in food intake for several hours have significant effect on the cholesterol and leptin level [5].

Researches of the adult population emphasized that night work increased the risk of dyslipidemia, in particular, increased triglycerides level, free fatty acid level and decreased level HDL-C, which could be explained by changes in the circadian rhythm [23]. Noteworthy, sleep duration, obesity, and physical inactivity are the leading causes of dyslipidemia [5, 23].

In addition, it was found that having a meal before sleep contributes to decreasing in HDL-C level ($p < 0.05$). Recent studies have shown some advantages in eating at bedtime, in that it could help maintain protein synthesis and glycogen re-synthesis when a small, nutrient-dense and high-protein meal is taken in close connection with physical activity [24]. However, such circumstances are not typical for the examined cohort of children: the participants in our study were not healthy and physically active subjects; they were overweight or obese and typically had low level of physical activity, especially in evening and morning hours.

Lipid screening is recommended at the age of 9–11 years and at the age of 17–21 years (before and after puberty); nevertheless, if risk factors are present, selective screening may be performed [25]. That being said, the results of our study should be taken in the account when considering risk factors of dyslipidemia in children, which are not limited to obesity as a principal risk factor, but also include NES.

Conclusions

NES may have significant health implications in young people due to its contribution to excess weight or obesity and disorders of lipid metabolism. Our study has found NES to be one of the reasons for changes in the lipid profile in children; therefore, screening for eating disorders should be implemented as a routine practice to reduce the risk of dyslipidemia and the associated consequences.

Acknowledgments

Authors' contributions: All authors contributed to the study conception and design. Halyna Pavlyshyn designed the study, participated in discussion of the obtained results, revised and edited the paper. Kateryna Kozak provided the examination of the patients, data collection, analyzed the results. Mariya Marushchak participated in discussion of the obtained results, revised, and edited the paper. All authors read and approved the final manuscript.

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Conflict of Interest

The authors declare no conflict of interest.

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