

Evaluation of ophthalmic manifestations according to insulin resistance, lipid and pubertal status in obese and healthy children

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Abstract

Purpose: We aimed to evaluate the ophthalmic manifestations in terms of insulin resistance (IR), lipid and pubertal status in obese and healthy children.

Methods: Subjects (aged from 5 to 17 years) were divided into 2 groups according to their body mass index (BMI) percentiles as obese and non-obese and 4 subgroups according to absence or presence of insulin resistance, hyperlipidemia and pubertal status. Sixty obese children (BMI >95th percentile), 58 healthy control subjects with the same age range (BMI <85th percentile) were admitted to this prospective study. Demographic features and laboratory measurements of subjects were recorded. Ophthalmological examination, including ocular surface parameters, intraocular pressure, central corneal thickness (CCT), and biometric measurements were assessed among subgroups.

Results: The subjects in obese group were significantly more hypertensive than control group [13 patients (21.7%), and 1 patient (1.7%) respectively, ($p=0.001$)]. Incidence of hyperinsulinism and the homeostasis model assessment for IR (HOMA-IR) levels and of obese children were significantly higher than control group ($p=0.003$, and $p=0.034$, respectively). The rate of hypertensive retinopathy was significantly higher in obesity (+) hyperlipidemia (+) subgroup ($p=0.002$). The mean CCT values were significantly lower in obesity (+) IR (+) subgroup ($p=0.023$). Schirmer test scores were significantly lower in obesity (-) subgroups regardless of IR, lipid and pubertal status.

Conclusions: Our findings suggest that decreased CCT levels may be related to presence of IR in obese children. The coexistence of hypertensive retinopathy with hyperlipidemia may occur in childhood obesity.

Introduction

During the last 20 years obesity has become a major public health problem in advanced and developing countries [1,2]. Obesity is one of the most pressing challenges that humanity faces today [3]. Insulin resistance (IR) is one of the most concerning complications of childhood obesity (CO) [4]. Both environmental and genetic factors are involved in the pathogenesis of IR. IR is the state of reduced tissue sensitivity to insulin [4]. IR is more frequent in children with very high BMI [5]. IR is considered to be a chronic low-grade inflammation [6,7]. We hypothesized that dry eye, as an inflammatory disease, may be correlated with obesity in these children based on the role of this inflammation leading to overweight and obesity.

Hyperlipidemia is another risk factor related to CO [4]. It is estimated that approximately 42% of obese children have lipid abnormalities [4]. The most common lipid abnormality pattern consists of elevated triglycerides, decreased high-density lipoprotein cholesterol and normal to mildly elevated low-density lipoprotein cholesterol [4]. Ophthalmological complications of hypertriglyceridaemia include eruptive, and other forms of xanthomata including xanthelasmata, corneal arcus, lipaemia retinalis, decrease in retinal blood flow, and lipid emboli affecting vision [8].

Puberty leads to the maturation of secondary sex characteristics, height increase, and attainment of reproductive capacity. CO also contributes to early onset of puberty [9]. Pubertal growth spurt may lead to the changes in ophthalmic examination in both genders. We are interested in the influence of pubertal status on ophthalmic findings in obese children.

Although obesity is becoming a major cause of morbidity in children with effects later on in adult life, as far as we know, there is no data about comparison of ocular surface parameters, and limited data on intraocular pressure, central corneal thickness, and biometric measurements in terms of IR, lipid and pubertal status between obese and healthy children. Therefore, we aimed to investigate this issue.

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Materials and methods

This study was undertaken as a prospective analysis between January 2016 and April 2016, in Training and Research Hospital. A total of 118 patients who were referred by our pediatric endocrinology department to our ophthalmology outpatient clinic, were enrolled in this study. The inclusion criteria for the study groups were primary obesity (body mass index (BMI) >95th percentile for sex and age) and healthy children within the same age range with normal BMI (<85th percentile). Study subjects also were divided into 4 subgroups according to absence or presence of insulin resistance, hyperlipidemia and pubertal status. Exclusion criteria included over-weight children (85th-95th percentiles for BMI), previous glaucoma, orbital masses, severe myopia (>6D), diseases of the cornea, the presence of cardiovascular, renal, neurological, thyroid, mental, metabolic disorders and genetic syndromes.

Each subject underwent full ophthalmologic examination including best-corrected visual acuity, biomicroscopic anterior segment and fundus examination, and IOP measurement by noncontact tonometer (Reichert 7 CR Corneal Response Technology, USA). Three consecutive IOP measurements were taken for each eye and the mean value was recorded. Visual acuity test results, detection of significant refractive errors following cycloplegia, and ocular alignment findings based on cover-uncover, alternate cover, and Hirschberg testing were recorded for each subject. Snellen or HOTV testing was used to determine visual acuity. Cycloplegic retinoscopy and fundus examination following dilation of the pupils with cyclopentolate 1 % were also performed. The refractive status of each subject was assessed using a hand-held automated refractometer (SureSight™ autorefractor, Welch Allyn, Skaneateles Falls, NY, USA). The ultrasonic pachymeter and A-mode biometry probe (Nidek US-4000 Echoscanner, Japan) were used to determine CCT and axial length, respectively. After administering topical proparacaine hydrochloride 0.5% (Alcaine ophthalmic solution, Alcon, Turkey), measurements were taken with the tip of the probe targeting the center of the pupil and perpendicular to the cornea while the subject was looking at a fixed target. The probe was sterilized with alcohol after each subject was examined. At least 5 consecutive measurements were obtained for each eye and the mean value was recorded. Tear break up time (TBUT) and Schirmer 1 test with topical anesthesia were performed to all patients 30 minutes after full ophthalmologic examination. The TBUT was measured after fluorescein staining. Subjects were instructed to blink, and the tear film was examined using the cobalt blue filter of a slit-lamp biomicroscope. The time interval in seconds between the instillation of fluorescein and the appearance of the first randomly distributed dry spot was enrolled as the TBUT. This method was repeated three times for each eye, and the average of the results was registered as the mean TBUT. TBUT of less than 10 seconds was determined as abnormal. The Schirmer 1 test was applied by placing a standardized strip of filter paper in the 1/3 lateral tarsal conjunctiva away from the cornea. Outcomes were expressed in millimeters after 5 minutes of wetting [10].

Prevalence of obesity was defined using BMI percentiles for age and sex. Obesity in children was defined when a BMI was greater than the 95th percentile for age and sex according to the reference values for the Turkish pediatric population [11]. BMI was calculated based on the formula: $BMI = [\text{weight/height}^2 (\text{kg/m}^2)]$ [4].

Pubertal stage was based on Tanner's classification and divided into prepubertal (Tanner stage 1) and pubertal (Tanner stage 2-5) subjects including stage 2 thelarche in females, >4ml testis volume in males [12].

Laboratory findings were recorded from the patients' data. All children had undergone a 12-hour fast prior to blood sampling. Blood lipid and fasting glucose levels were evaluated by Beckman Coulter DXC 800/USA biochemical analyzer. Hyperlipidemia was diagnosed as >110 mg/dL for low-density lipoprotein cholesterol, <40 mg/dL for high-density lipoprotein cholesterol, >170 mg/dL for total cholesterol and >150 mg/dL for triglyceride levels [13]. Insulin levels were determined by ADVIA Centaur XP Immunoassay System (Siemens, Germany) analyzer. Hyperinsulinism was diagnosed as fasting insulin levels >15 for pubertal stage 1, >20 for pubertal stage 2-3 and >30 for pubertal stage 5. In clinical practice, the homeostasis model assessment-insulin resistance (HOMA-IR) index is used to diagnose IR [4]. HOMA-IR index was calculated based on the formula: $HOMA-IR = \text{insulin (mU/L)} \times \text{glucose (mmol/L)} / 22.5$, taking 2.6 for prepubertal male and 2.2 for prepubertal female, and 5.2 for pubertal male and 3.8 for pubertal female as the cutoff value for the diagnosis of IR [14].

This study was carried out with the Institutional Review Board/Ethics Committee approval. The research adhered to the tenets of the Declaration of Helsinki. Informed consent was obtained from all parents after explaining the nature and purpose of the study.

Statistical analysis

All analyses were performed using SPSS for Windows 18.0 (version 18.0, SPSS, Chicago, IL, USA). Continuous variables are presented as mean \pm standard deviation (SD). Continuous variables were compared using Student's unpaired t tests or Mann-Whitney nonparametric U tests. Categorical variables were compared using chi-square statistics. Comparison of parametric values among groups was performed by One-Way ANOVA. A two-tailed p value <0.05 was considered as significant.

Results

The subjects in obese group were significantly more hypertensive than control group ($p=0.001$). Incidence of hyperinsulinism and HOMA-IR levels and of obese children were significantly higher than control group ($p=0.003$, and $p=0.034$, respectively). The baseline demographic features and laboratory measurements of study subjects were shown in (Table 1). Spherical values were between -1.75 D and +3.00 D in obese group, -3.50 D and +2.00 D in control group. All astigmatism values were recorded in plus cylinder; cylindrical values were between +0.50 D and +2.75 D in both groups. Comparison of ophthalmic pathologies of study subjects according to insulin resistance, lipid and pubertal status were shown in (Table 2, 3, 4). The mean central corneal thickness values were significantly lower in obesity + and insulin resistance + subgroup ($p=0.023$) (Table 2). Schirmer test scores were significantly lower in obesity – subgroups regardless of insulin resistance, lipid and pubertal status (Table 2, 3, 4). The rate of hypertensive retinopathy was significantly higher in obesity + hyperlipidemia + subgroup ($p=0.002$) (Table 3).

Discussion

To the best of our knowledge, this is the first study that demonstrates the comparison of ocular surface clinical parameters in terms of insulin resistance, lipid and pubertal status between obese and normal weight healthy children.

The frequency of IR occurrence is increasing dramatically in developed countries. IR occurs physiologically during puberty, but it is also a pathological condition predisposing child to develop abnormal glucose tolerance, diabetes, hypertension and polycystic ovary

Table 1. Baseline demographic features and laboratory measurements of study subjects.

Variable	Obese (n = 60)	Non-Obese (n = 58)	p
Age	11.0±3.0	11.8±3.0	0.146
Gender (boy)	33 (55%)	33 (55%)	33 (55%)
Body Mass Index	28.5±5.4	22.3±3.9	<0.001
Hypertension	13 (21.7%)	1 (1.7%)	0.001
Total cholesterol	166±33	163±26	0.595
High density lipoprotein	48±11	52±13	0.094
Low density lipoprotein	102±26	94±23	0.076
Triglyceride	103±44	101±42	0.802
Glucose	91±8.0	91±7.0	0.681
Insulin	18.3±9.8	14.5±7.1	0.019
Hyperinsulinism	29 (48.3%)	13 (22.4%)	0.003
Insulin Resistance	35 (58.3%)	24 (41.4%)	0.066
HOMA-IR	4.13±2.5	3.27±1.6	0.034
Prepuberty (Tanner Stage 1)	23 (38.3%)	15 (25.9%)	0.321
Puberty (Tanner Stage 2-5)	37 (61.7%)	43 (74.1%)	0.321
Precocious puberty	2 (3.3%)	0 (0%)	0.161
HOMA-IR: Homeostasis model assessment for insulin resistance			

Table 2. Ophthalmic pathologies and clinical measurements of study subgroups according to insulin resistance status.

Ophthalmic findings	Obesity – IR – n=34	Obesity – IR + n=24	Obesity + IR – n=25	Obesity+ IR + n=35	p
Strabismus	0 (0%)	0 (0%)	1 (4%)	0 (0%)	0.29
Lens opacities	0 (0%)	1 (4%)	0 (0%)	1 (2.9%)	0.556
Refractive errors	6 (21.2%)	6 (24%)	10 (40%)	5 (24.3%)	0.139
Amblyopia	3 (9.1%)	1 (4%)	3 (12%)	2 (5.7%)	0.699
Central Corneal Thickness	555±31	544±42	550±31	530±27	0.023
Intraocular Pressure	15.4±2.3	15.8±2.8	15.8±2.8	14.7±2.4	0.327
Schirmer test (mm)	7.7±4.6	12.3±7.4	15.5±8.8	15.5±8.8	<0.001
Break-up time (sec)	10.6±1.3	10.6±1.3	10.0±2.9	9.8±2.3	0.551
Axial length (mm)	22.8±1.2	23.2±0.8	23.3±0.7	22.8±1.1	0.262
Lens thickness (mm)	3.6±0.3	3.4±0.2	3.5±0.2	3.5±0.2	0.17
Anterior chamber depth (mm)	3.6±0.3	3.6±0.2	3.5±0.2	3.5±0.2	0.097
Vitreous chamber depth (mm)	15.6±1.1	16.0±0.8	15.5±2.8	15.9±1.9	0.573
Hypertensive retinopathy	0 (0%)	0 (0%)	1 (4%)	1 (2.9%)	0.556
IR: Insulin resistance					

Table 3. Ophthalmic pathologies and clinical measurements of study subgroups according to lipid status

Ophthalmic findings	Obesity – Hyperlipidemia – n=42	Obesity – Hyperlipidemia + n=16	Obesity + Hyperlipidemia – n=41	Obesity + Hyperlipidemia + n=19	p
Strabismus	0 (0%)	0 (0%)	1 (2.6%)	0 (0%)	0.595
Lens opacities	0 (0%)	1 (6.3%)	1 (2.4%)	0 (0%)	0.365
Refractive errors	9 (21.4%)	4 (25%)	12 (29.3%)	3 (15.8%)	0.684
Amblyopia	3 (7.1%)	1 (6.3%)	5 (12.2%)	0 (0%)	0.417
Central Corneal Thickness	533±37	545±36	535±24	544±40	0.153
Intraocular Pressure	15.7±2.6	15.0±2.3	14.8±1.9	15.2±2.6	0.399
Schirmer test (mm)	9.4±6.1	10.4±7.3	16.3±8.9	15.4±6.7	<0.001
Break-up time (sec)	10.6±1.7	9.9±1.6	9.6±2.7	10.3±2.3	0.304
Axial length (mm)	23.0±1.0	22.7±1.2	23.2±0.8	22.5±1.3	0.237
Lens thickness (mm)	3.5±0.2	3.5±0.3	3.5±0.1	3.5±0.3	0.962
Anterior chamber depth (mm)	3.6±0.2	3.6±0.3	3.5±0.2	3.4±0.3	0.133
Vitreous chamber depth (mm)	15.8±1.1	15.6±0.9	15.7±2.2	15.8±0.8	0.953
Hypertensive retinopathy	0 (0%)	0 (0%)	1 (2.4)	1 (5.3%)	0.002

syndrome among girls [1]. In our study incidence of IR between groups was not significantly different. But HOMA-IR levels and incidence of hyperinsulinism were significantly higher in obese children. CO and IR are also characterized by impaired immunity and a low-grade inflammation status depending on the multicellular release of cytokines,

adipokines, and reactive oxygen species [1, 6-8, 15-17]. We thought that dry eye may be correlated with obesity in these children based on the role of this inflammation leading to overweight and obesity. However, unexpectedly, we have found significantly lower scores in control group. It may be because of the small size of the study groups.

Table 4. Ophthalmic pathologies and clinical measurements of study subgroups according to pubertal status

Ophthalmic findings	Obesity – Puberty – n=15	Obesity – Puberty + n=43	Obesity + Puberty – n=23	Obesity + Puberty + n=37	p
Strabismus	0 (0%)	0 (0%)	1 (3.2%)	0 (0%)	0.419
Lens opacities	1 (6.3%)	0 (0%)	0 (0%)	1 (3.4%)	0.286
Refractive errors	3 (19.7%)	10 (23.8%)	6 (19.4%)	9 (31%)	0.705
Amblyopia	0 (0%)	4 (9.5%)	3 (9.7 %)	2 (6.9%)	0.628
Central Corneal Thickness	551±42	550±34	539±29	535±30	0.259
Intraocular Pressure	15.35±3.0	15.6±2.3	15.0±2.2	14.8±2.0	0.55
Schirmer test (mm)	9.5±7.2	9.8±6.1	13.3±7.7	19.3±7.1	<0.001
Break-up time (sec)	9.8±2.2	10.6±1.4	9.4±3.1	10.4±1.7	0.123
Axial length (mm)	22.5±1.0	23.1±1.1	22.8±1.1	23.2±0.9	0.216
Lens thickness (mm)	3.4±0.1	3.6±0.3	3.6±0.2	3.4±0.1	0.126
Anterior chamber depth (mm)	3.5±0.2	3.6±0.3	3.5±0.2	3.4±0.2	0.094
Vitreous chamber depth (mm)	15.5±1.1	15.9±0.9	15.3±2.4	16.2±0.9	0.235
Hypertensive retinopathy	0 (0%)	0 (0%)	1 (3.2%)	1 (3.4%)	0.578

Some epidemiological studies have described an association between obesity and IOP in adults [18,19]. Some authors argue that obesity increases IOP due to an excessive intraorbital adipose tissue deposit, leading to a rise in blood viscosity and episcleral venous pressure, and a consequent decrease in the facility of aqueous outflow [5]. For other authors, obesity only increases IOP when it is associated with IR [5]. The autonomic dysfunction and the osmotic gradient induced by hyperglycemia with a consequent fluid shift into the intraocular space have been proposed to explain the association between IOP and IR. A recent review concluded that there is an association between higher BMI and higher IOP in adults [2]. Akinci *et al* found that obesity was an independent risk factor for increased IOP [20]. In contrast to these results, Allb LL *et al* do not show a correlation between body mass index and IOP in children [5]. In the study of Koçak N, there were no significant differences in IOP measurements, central corneal thicknesses, cup/disc ratios and visual field parameters between obese and normal children [21]. In our study the difference among subgroups were not significantly different in terms of IOP. However, CCT values were significantly lower in obesity + and insulin resistance + subgroup. CCT could be affected with the hormonal variations of insulin.

There is no evidence that IOP influences retinal vascular caliber in healthy young children [22]. However, increasing BMI is associated with increasing retinal venular caliber over time in children [6]. In adolescents, greater body fat deposition is related to narrower retinal arterioles and wider retinal venules [23]. Progressive retinal venular widening could be a manifestation of an adverse microvascular effect of obesity early in life [6]. Young adolescents with elevated blood pressure and obesity have changes in retinal vessel caliber that are associated with ocular and systemic vascular diseases in adulthood [24]. A strong and independent association between adiposity and blood pressure was present during early childhood. These data have important public health implications because elevated blood pressure at a young age may be associated with increased cardiovascular risk in later life [25]. CO could induce some risk factors for cardiovascular disease including serum lipid abnormalities, hypertension, and atherosclerosis [26]. In our study population hypertension rate was significantly higher in obese group than control group.

The central obesity, as well as its combination with overweight or general obesity is more frequent in boys than in girls. Higher IOP was associated with the nonocular parameters of female sex, higher body mass index, younger age, maternal myopia and with the ocular parameters of longer axial and smaller corneal horizontal diameter [27]. Variations in the onset and peak progression of myopia may be associated with height spurts [28]. We have not found a significant

difference in terms of refractive errors, biometric measurements or gender among subgroups.

In conclusion; our findings suggest that decreased CCT levels may be related to presence of IR in obese children. CCT is important for diagnostic and therapeutic purposes such as assessment before refractive surgery, monitoring corneal changes after extended contact lens wear, and determining the reliability of IOP measurements. In addition, hypertensive retinopathy can accompany hyperlipidemia in obese children. The role of IR, lipid and pubertal status should be further investigated with more subjects to compare the ophthalmic changes of obese population with non-obese ones.

Conflict of interest

No potential conflict of interest relevant to this article was reported.

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