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Metabolic syndrome and insulin resistance in obese adolescent boys

O. Khyzhnyak[1], M. Minkov[2], R. Ratschmann[2], M. Zauner[2], I. Cherevko[1], Yu. Karachentcev[1]

[1] Institute of Endocrine Pathology Problems Ukrainian Academy of Medical Science, Kharkiv, Ukraine, Department of Age-Related Endocrinology, Kharkiv, Ukraine

[2] Rudolfstiftung Hospital, Department of Pediatrics & Adolescent Medicine & Neonatology, Vienna, Austria

The aim — to investigate the association between some components of metabolic syndrome (MS) and the level of insulin resistance and secretory function of the pancreatic β-cells in adolescent boys.

Materials and methods. 292 adolescent boys (12—17 years old) with overweight and obesity (BMI > 95 p. c.) were examined. Evaluation of anthropometric parameters included measurements of height, weight, waist circumference, blood pressure (BP) and assessment of pubertal development (Tanner stage). Fasting insulin (IRI), plasma glucose (PG), and lipids were collected for analysis with commercial kits (Roche Diagnostics GmbH). Insulin sensitivity (HOMA-%S) and βcell function (HOMA-%B) were evaluated using HOMA Calculator v.2.2. Statistical analysis was performed using the certified program SPSS (version 9.0). Data are presented as mean and 95 % confidence interval. P-value < 0.05 was considered as statistically significant.

Results and discussion. Among 292 patients hypertension was diagnosed in 37.7 % of them, hypertriglyceridemia (TG) in 35.9 %, reduced level of high density lipoproteins (HDL) — in 31.9 % of patients. Impaired glucose tolerance (IGT) was found in 8.9 % and insulin resistance (HOMA-%S < 100 %) — in 91.1 % of patients. Univariate regression analysis revealed nonlinear association (r = 0.67, p = 0.0001) between insulin sensitivity and β-cell function. Insulin resistant individuals (HOMA-%S < 100 %) had an increased level of TG (OR = 16.6 [16.4—16.8]) and arterial hypertension (OR = 2.13 [1.2—6.09]) and decreased level of HDL (OR = 3.36 [1.9—5.2]). Increased fasting glucose (≥ 6.1 mmol/L) in adolescents with obesity is associated with tension of β-cell secretory function (OR = 14.7 [10.3—19.1]).

Conclusions. We found a correlation between insulin sensitivity/β-cell function and MS components (e. g. BP, TG, HDL, PG). In 15.4 % of obese adolescents with insulin resistance and insufficient β-cell function we did not find hypertension and dyslipidemia (that are typical diagnostic criterias rather for prediabetes, not MS). In 35.9 % of those with insulin resistance and obesity and normal βcell function it was found basic diagnostic features of MS, such as abdominal obesity, high BP, high TG and low HDL levels. These patients are predisposed to develop cardiovascular complications in adult age.

Key words: obesity, metabolic syndrome, insulin resistance, puberty.

The global prevalence of obesity and diabetes has increased dramatically in the past quarter century [1]. Childhood obesity is reaching epidemic proportions and represents the most important chronic disease in this age group and is become a worldwide epidemic [2]. The disease is spreading from industrialized countries to the urban settings of developing countries. The global prevalence has increased markedly: more than 42 million overweight children under the age of five in 2010 (WHO website sources). Urbanization, unhealthy diet and increasingly sedentary lifestyle are major contributors to such disorders and have increased the prevalence of childhood obesity, particularly in developing countries. In the USA 15.8 % of children between 6 and 11 years and 16.1 % of adolescents have a body mass index (BMI) in the range of overweight [5, 9]. Similar trends have also been observed in many European countries, where, based on the latest International Task Force criteria, overweight and obesity are present in 31.8 % of school-aged children [6]. Concomitantly, with the increasing prevalence of childhood obesity, the prevalence of metabolic syndrome (MS) is rising among children and adolescents1 and reaches 50 % in severely obese subjects [3, 4]. Worldwide estimates of the prevalence of MS range from 1.2 % to 22.6 % for youth and 9.0 %

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Хижняк Оксана Олегівна, д. мед. н., проф., зав. відділення вікової ендокринології
61002, м. Харків, вул. Алчевських, 10. Тел. (057) 315-11-94
E-mail: zenrost@mail.ru

to 35.0 % for adults, depending on the definition of MS used, the region, the study design, the years of the study, and the age group and study population [7].

The MS is a constellation of cardiometabolic risk factors that are predictive for chronic disease and all-cause mortality in adults [11, 16, 18]. It is estimated that risk of cardiovascular disease (CVD) doubles and the risk of type 2 diabetes mellitus (T2DM) increases fivefold if MS is present [8]. MS is characterized by the presence of different combinations of risk factors including obesity, hypertension, elevated fasting triglycerides, insulin resistance, low total cholesterol, high low-density lipoprotein cholesterol, low high-density lipoprotein cholesterol (HDL-C), apolipoprotein B, C-reactive protein and homocysteine [7]. These clinical features of MS, if present together, tend to suggest a common etiology; the proposed mechanisms underlying MS and its influence on health outcomes are discussed elsewhere.

Childhood obesity is also associated with an increased risk for several metabolic complications, such as insulin resistance, glucose intolerance and T2DM. In particular, insulin resistance is the most common metabolic alteration related to obesity [13], and it represents an important link between obesity and other metabolic as well as cardiovascular complications [14]. As CVD is the first cause of morbidity and mortality in adulthood, the epidemic of childhood obesity will cause a real burden for the future of the society. Most of the health-threatening consequences of juvenile obesity are due to metabolic disturbances induced by an excessive accumulation of fat which leads to chronic diseases like T2DM, hypertension and CVD [10, 12]. Obese children are also at risk of developing psychological problems, orthopaedic and respiratory disorders and also certain malignancies. Furthermore, childhood obesity frequently tracks into adulthood, thus representing a major contributor to the adult obesity epidemic and to the increased cardiovascular morbidity and mortality in adult life. All these data are alarming and underline how obesity is a real threat for the health of children and adolescents. Thus, the prevention of obesity-induced metabolic conditions is a cornerstone of the management of overweight and obesity during childhood [15].

Due to the challenging proportion of children and adolescents affected by obesity in most countries, finding effective ways to identify obese paediatric patients who are at increased risk of developing cardiovascular and metabolic complications has been recognized to be a promising strategy to improve prevention of complications of early obesity and has been a major priority of the research in the field of childhood obesity for several years [17].

Given insulin resistance is the important link between obesity and the associated metabolic abnormalities and cardiovascular risk, clinicians should be aware of high risk groups and treatment options. Treatment for reducing insulin resistance and other obesity-associated comorbidities should focus on changes in health behaviors to achieve effective weight management. Lifestyle interventions incorporating dietary change, increased physical activity, and decreased sedentary behaviors, with the involvement of family and adoption of a developmentally appropriate approach, should be used as the first line treatment. Current evidence suggests that the primary objective of dietary interventions should be to reduce total energy intake and a combination of aerobic and resistance training should be encouraged.

Diagnosing MS among children and adolescents proves particularly challenging given the difficulty in establishing accurate, meaningful and harmonized criteria for this population. As there is no universally accepted biochemical definition of insulin resistance in children and adolescents, identification and diagnosis of insulin resistance usually relies on clinical features such as acanthosis nigricans, polycystic ovary syndrome, hypertension, dyslipidemia, and nonalcoholic fatty liver disease. Consequently, prevalence estimates of MS among children and youth very greatly depending on the adopted definition [6]. In 2007, the IDF released their Consensus Definition of the Metabolic Syndrome in Children and Adolescents [19]. This criterion provides an age- and sex-specific definition for youth aged 10 to 15 years. The IDF definition further stipulates that the worldwide adult definition of MS should be applied for individuals aged 16 years or older and that MS should not be diagnosed in children less than 10 years old [18]. For children age 10 years or older, metabolic syndrome can be diagnosed with abdominal obesity and the presence of two or more other clinical features (i. e. elevated triglycerides, low HDL-cholesterol, high blood pressure, increased plasma glucose). In the absence of contemporary definitive data, the criteria adhere to the absolute values in the IDF adult definition, except that waist circumference percentiles are recommended and one (rather than a sex-specific) cut-off is used for HDL-cholesterol levels. Further research is needed to identify optimum criteria for the definition of the syndrome. Moreover, recent epidemiological study found that adolescent males carry a three times higher risk for metabolic syndrome compared to adolescent females. Although some MS features are unequivocally present in children and adolescents, the diagnostic criteria for this age group are still a matter of debate.

The goal of the present study was to investigate the association obesity and some MS features in adolescent boys.

Subject, inclusion and exclusion criteria: 292 adolescent boys (12—17 years) with overweight or obesity (BMI > 95P) were examined. Boys with fetal

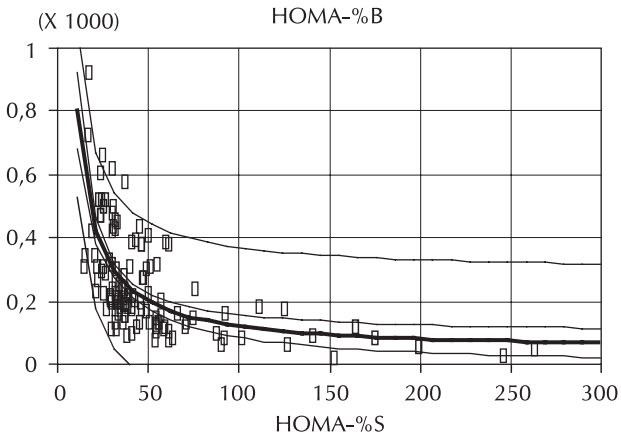

Fig. 1. **Association between insulin sensitivity (HOMA-%S) and β-cell function HOMA-%B) in obese boys (r = 0.67, p = 0.0001)**

or congenital diseases that could affect fetal growth were excluded (TORCH infections: Toxoplasmosis, other (syphilis, varicella-zoster, parvovirus B19), Rubella, Cytomegalovirus (CMV), Herpes infections, and congenital malformation). Gestational age below 34 weeks of gestation (WG) or children presenting with a severe neonatal condition were also not included in the study.

Materials and methods

Physical examination included height, weight, waist circumference and blood pressure (BP) measurements, as well as pubertal development estimation (Tanner stage). Blood samples for insulin (IRI), plasma glucose (FPG), and lipid measurements were taken in fasting state. Insulin sensitivity (HOMA-%S) and β-cell function (HOMA-%B) were estimated by HOMA Calculator v.2.2.

Data analysis. Statistical analysis was performed using SPSS version 9.0. Univariate regression analysis has been performed. Data are given as mean and 95 % CI, and best-fitting model equations.

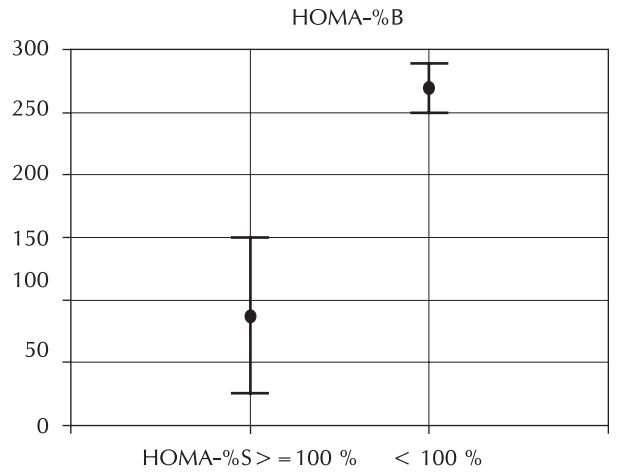

Fig. 2. **Secretory function of pancreas β-cells in obese boys with different insulin sensitivity**

Results and discussion

Among the 292 study objects 37.7 % were hypertensive, 35.9 % had high triacylglycerol (TG) levels, and 31.9 % had low HDL-cholesterol levels. Fasting glucose intolerance was found in 8.9 % and insulin resistance (HOMA-%S < 100 %) in 91.1 % of the patients. Univariate regression analysis revealed nonlinear association (r = 0.67, p = 0.0001) between insulin sensitivity and β-cell function (fig. 1).

Most of the objects are in HOMA2-%S < 100 % on the X-axis, which is evidence that they have different degrees of insulin resistance. Y axis has been allocated three important areas: HOMA2-%B in < 50 %, which reduced secretory activity or decline of pancreas β-cell reserves; HOMA2-%B = 50—100 %, corresponding to a normal pancreas secretory activity; HOMA2-% > 100 % — of it increasing.

Analysis of variance allowed to demonstrate a statistically significant difference (p = 0.0001) pancreas secretory activity at forming of groups of boys on the basis of insulin sensitivity (fig. 2).

In adolescents with clinical manifestations of MS compensation of reduced insulin sensitivity is due by stretching pancreas secretory function. However, according to the data (fig. 1 and 2) secretory function pancreas β-cell varies widely in individuals with normal and, in some cases, and increased sensitivity of insulin — target tissues.

It was analyzed the association of MS components: insulin sensitivity and secretion pancreas β-cell. Insulin resistant individuals — (HOMA-%S < 100 %) had an increased prevalence of high TG, OR = 16.6 [16.4—16.8], HDL-cholesterol, OR = 3.36 [1.9—5.2], and arterial hypertension, OR = 2.13 [1.2—6.09]. Fasting glucose intolerance (≥ 6.1 mmol/L) was associated with β-cell dysfunction, OR = 14.7 [10.3—19.1] (table 1 and 2).

According to data in table 2, increased TG levels (≥ 1.7 mmol/L) and reduced HDL-cholesterol levels (< 1.03 mmol/L), which have the highest OR values, associated with reduced insulin sensitivity and β-cell ability in compensation insulin resistance by secretory stretching pancreas secretory function. Despite the fact that high blood pressure (SBP ≥ 130, DBP ≥ 85 mm Hg) in adolescents with insulin resistance (IR) occurs about 2 times more likely than adolescents with normal insulin sensitivity, there were not statistically significant in association of blood pressure and IR (see table 2). Similarly, we can only talk about trends in the association of high blood pressure with increased secretory activity of pancreas β-cell (see table 1).

The data of the study allowed to set that fasting glucose (≥ 5.6 mmol/L) is statistical significantly associated with a reduced secretory function of β-cells

Table 1. **The risk of developing clinical manifestations of metabolic syndrome in adolescent obese boys depending of the pancreas β-cells secretory function**

Clinical signs	Statistics	HOMA2-%B		
		< 50 %	**50—100 %**	**> 100 %**
Arterial blood pressure: SBP ≥ 130 mm Hg, DBP ≥ 85 mm Hg	OR [95 % CI]		←	1.82 [0—5.45]
			←	1.61 [0.34—2.89]
Fasting plasma glucose ≥ 5.6 mmol/l	OR [95 % CI]	1.71 [0.63—2.80] →		
			14.7* [10.3—19.1] →	
		25.3* [24.1—26.5] →		
Triglycerides ≥ 1.7 mmol/l	OR [95 % CI]		← 2.90* [2.73—3.07]	
				← 2.11 [0—2.13]
			←	6.13* [5.94—6.31]
HDL-cholesterol < 1.03 mmol/l	OR [95 % CI]		← 19.33* [19.15—19.52]	
				← 1.41 [0—5.57]
			←	13.74* [13.55—13.93]

Notes. *P < 0.05 — the level of statistical significance; ← a reference to the comparison group.

Table 2. **The risk of developing clinical manifestations of metabolic syndrome in adolescent obese boys depending on sensitivity of the insulin — targets tissues**

Clinical signs	Statistics	HOMA2-%S	
		< 100 %	**≥ 100 %**
Arterial blood pressure: SBP ≥ 130 mmHg, DBP ≥ 85 mm Hg	OR [95 % CI]	2.13 [0—6.09] →	
Fasting plasma glucose ≥ 5.6 mmol/l	OR [95 % CI]		← 2.68 [0—6.31]
Triglycerides ≥ 1.7 mmol/l	OR [95 % CI]	16.6* [16.4—16.8] →	
HDL-cholesterol < 1.03 mmol/l	OR [95 % CI]	3.36* [1.9—5.2] →	

Notes. *P < 0.05 — the level of statistical significance; ← a reference to the comparison group.

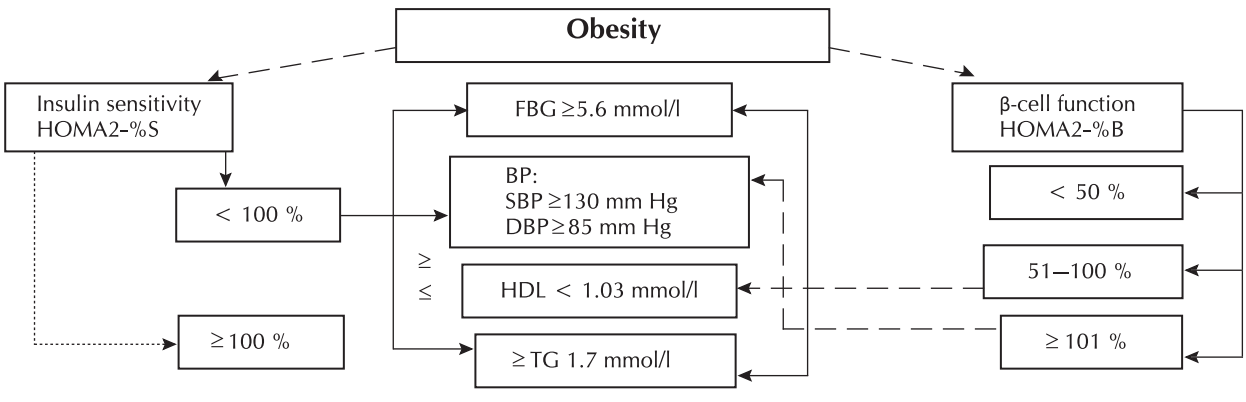

Fig. 3. **The association of clinical and laboratory signs of metabolic syndrome in adolescence obese boys**

(HOMA2-%B < 50 %) and independent of insulin sensitivity (fig. 3).

Thus, the data of the study identified that in obese boys reduced insulin sensitivity plays a key role in evolution of MS and an additional reduction of the β-cell secretory function are the foundation for the early development of atherogenic dyslipidemia and glucose intolerance.

Conclusions

1. Obesity was a major clinical component of the MS manifestation in adolescent boys.

2. We found a correlation between insulin sensitivity/β-cell function and MS features (BP, TG, HDL, FPG).

3. The majority of those with resistance to insulin and still normal β-cell function had a full set of MS features, e. g. abdominal obesity, high BP, high TG and low HDL-cholesterol (35.9 %). They are predisposed to develop metabolic cardiovascular complications.

4. Full set of MS component: abdominal obesity, hypertension, high tricylglicerol level and low HDL-cholesterol level are presented in adolescent boys with resistance to insulin and sufficient β-cell function.

5. Part of the obese adolescent individuals with resistance to insulin and insufficient β-cell function did not have hypertension and dyslipidemia (15.4 %). They fulfill the criteria for prediabetes rather than for MS.

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REFERENTS

1. Bacha F., Saad R., Gungor N., Arslanian S.A. Are obesity-related metabolic risk factors modulated by the degree of insulin resistance in adolescents? // Diabetes Care. — 2006. — Vol. 29. — P. 1599—1604.
2. Barlow S.E. Expert Committee: Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity: summary report // Pediatrics. — 2007. — Vol. 120. — P. S164—S192.
3. DeBoer M.D. Obesity, systemic inflammation, and increased risk for cardiovascular disease and diabetes among adolescents: a need for screening tools to target interventions // Nutrition. — 2013. — Vol. 29. — P. 379—386.
4. Ford E.S. Risks for all-cause mortality, cardiovascular disease, and diabetes associated with the metabolic syndrome: a summary of the evidence // Diabetes Care. — 2005. — Vol. 28 (7). — P. 1769—1778.
5. Franks P.W., Hanson R.L., Knowler W.C. et al. Childhood obesity, other cardiovascular risk factors and premature death // N. Engl. J. Med. — 2010. — Vol. 362. — P. 485—493.
6. Golley R.K., Magarey A.M., Steinbeck K.S. et al. Comparison of metabolic syndrome prevalence using six different definitions in overweight pre-pubertal children enrolled in a weight management study // Int. J. Obes. (Lond). — 2006. — Vol. 30. — P. 853—860.
7. Haffner S.M. The metabolic syndrome: inflammation, diabetes mellitus, and cardiovascular disease // Am. J. Cardiol. — 2006. — Vol. 97 (2A). — P. 3A—11A.
8. Hedley A.A., Ogden C.L., Johnson C.L. et al. Prevalence of overweight and obesity among US children, adolescents, and adults, 1999—2002 // J. Am. Medical Association. — 2004. — Vol. 291. — P. 2847—2850.
9. Isomaa A., Almgren P., Tuomi T. et al. Cardiovascular morbidity and mortality associated with the metabolic syndrome // Diabetes Care. — 2001. — Vol. 24 (4). — P. 683—689.
10. McNeill A.M., Rosamond W.D., Girman C.J. et al. The metabolic syndrome and 11-year risk of incident cardiovascular disease in the Atherosclerosis Risk in Communities study // Diabetes Care. — 2005. — Vol. 28 (2). — P. 385—390.
11. Pacifico L., Anania C., Martino F. et al. Management of metabolic syndrome in children and adolescents // Nutr. Metabol. Cardiovasc. Dis. — 2011. — Vol. 21. — P. 455—466.
12. Park M.H., Falconer C., Viner R.M., Kinra S. The impact of childhood obesity on morbidity and mortality in adulthood: a systematic review // Obes Rev. — 2012. — Vol. 13. — P. 985—1000.
13. Rosenberg B., Moran A., Sinaiko A.R. Insulin resistance (metabolic) syndrome in children // Panminerva Med. — 2005. — Vol. 47. — P. 229—244.
14. Singh R., Shaw J., Zimmet P. Epidemiology of childhood type 2 diabetes in the developing world // Pediatric Diabetes. — 2004. — Vol. 5. — P. 154—168.
15. Wang Y., Lobstein T. Worldwide trends in childhood overweight and obesity // Intern. J. Pediatric Obesity. — 2006. — Vol. 1. — P. 11—25.
16. Weiss R., Dziura J., Burgert T.S. et al. Obesity and the metabolic syndrome in children and adolescents // N. Engl. J. Med. — 2004. — Vol. 350. — P. 2362—2374.
17. World Health Organization, Fight Childhood Obesity to prevent diabetes, say WHO and IDF [Електронний ресурс]. — Режим доступу: http://www.who.int/mediacentre/news/releases/2004.
18. Zimmet P., Alberti K.G., Kaufmann F. et al. The metabolic syndrome in children and adolescents e an IDF consensus report // Pediatr Diabetes. — 2007. — Vol. 8. — P. 299—306.
19. Zimmet P., Alberti K.G., Shaw J. Global and societal implications of the diabetes epidemic // Nature. — 2001. — Vol. 414. — P. 782—787.

Метаболічний синдром та інсулінорезистентність у хлопців з ожирінням

О. Хижняк[1], M. Minkov[2], R. Ratschmann[2], M. Zauner[2], І. Черевко[1], Ю. Караченцев[1]

[1] ДУ «Інститут проблем ендокринної патології імені В.Я. Данилевського НАМН України», Харків, Україна

[2] Rudolfstiftung Hospital, Department of Pediatrics & Adolescent Medicine & Neonatology, Vienna, Austria

Мета роботи — проаналізувати асоціацію деяких компонентів метаболічного синдрому (МС) з рівнем інсулінорезистентності та секреторної функції β-клітин підшлункової залози (ПЗ) у хлопців-підлітків з ожирінням.

Матеріали та методи. Обстежено 292 хлопчиків з надлишковою масою тіла та ожирінням (ІМТ > 95 %) віком 12—17 років. Оцінка антропометричних показників включала вимірювання зросту, маси тіла, обводу талії, артеріального тиску (АТ), визначення статевого дозрівання (стадії за Таннером). Зразки крові брали натще для визначення рівня глюкози в плазмі (FPG), інсуліну (IRI), показників ліпідного обміну в сироватці крові з використанням комерційних наборів (Roche Diagnostics GmbH). Чутливість до інсуліну і функцію β-клітин ПЗ оцінювали з використанням HOMA-калькулятора v.2.2. Статистичний аналіз виконаний з використанням сертифікованої програми SPSS (версія 9.0). Дані наведені у вигляді середнього і 95 % довірчого інтервалу. Р-значення менше 0,05 вважали статистично достовірним.

Результати та обговорення. Серед 292 обстежених підлітків артеріальна гіпертензія виявлена у 37,7 %, гіпертригліцеридемія — у 35,9 %, знижений рівень ліпопротеїнів високої щільності (ЛПВЩ) — у 31,9 %, порушення толерантності до глюкози — у 8,9 %, у 91,1 % хворих встановлено інсулінорезистентність (HOMA-%S < 100 %). За допомогою одномірного регресійного аналізу встановлено нелінійний зв'язок (r = 0,67, p = 0,0001) між чутливістю до інсуліну та функцією β-клітин ПЗ. У підлітків зі зниженою чутливістю тканин-мішеней до інсуліну (HOMA-%S < 100 %) статистично значуще частіше визначали підвищення рівня тригліцеридів (ТГ) — відношення шансів (ВШ) = 16,6 [16,4—16,8]; АТ — ВШ = 2,13 [1,2—6,09]; зниження рівня ЛПВЩ — ВШ = 3,36 [1,9—5,2]. Підвищення глікемії натще (≥ 6,1 ммоль/л) у підлітків з ожирінням асоційовано з напруженням секреторної функції β-клітин ПЗ – ВШ = 14,7 [10,3—19,1].

Висновки. Встановлено кореляційний зв'язок між чутливістю до інсуліну/функцією β-клітин ПЗ з такими компонентами МС, як рівень АТ, ТГ, ЛПВЩ, FPG. У 15,4 % підлітків з ожирінням на тлі інсулінорезистентності та зниженої функціональної активності β-клітин не виявлено підвищеного АТ та дисліпопротеїнемії, що більш притаманно діагностичним критеріям переддіабету, а не МС. У 35,9 % хлопців-підлітків з ожирінням у поєднанні з інсулінорезистентністю та нормальною функцією ПЗ виявлено основні діагностичні ознаки МС, а саме: абдомінальне ожиріння, артеріальну гіпертензію, високий рівень ТГ і низький — ЛПВЩ. Ці пацієнти перебувають у групі ризику розвитку в дорослому віці серцево-судинних ускладнень.

Ключові слова: ожиріння, метаболічний синдром, інсулінорезистентність, пубертат.

Метаболический синдром и инсулинорезистентность у мальчиков с ожирением

О. Хижняк[1], M. Minkov[2], R. Ratschmann[2], M. Zauner[2] , И. Черевко[1], Ю. Караченцев[1]

[1] ГУ «Институт проблем эндокринной патологии имени В.Я. Данилевского НАМН Украины», Харьков, Украина

[2] Rudolfstiftung Hospital, Department of Pediatrics & Adolescent Medicine & Neonatology, Vienna, Austria

Цель работы — проанализировать ассоциацию некоторых компонентов метаболического синдрома (МС) с уровнем инсулинорезистентности и секреторной функции β-клеток поджелудочной железы (ПЖ) у мальчиков-подростков с ожирением.

Материалы и методы. Обследовали 292 мальчиков с избыточной массой тела и ожирением (ИМТ > 95 %) в возрасте 12—17 лет. Оценка антропометрических показателей включала измерение роста, массы тела, окружности талии, артериального давления (АД), определение полового созревания (стадии по Таннеру). Образцы крови брали в состоянии натощак для определения уровня глюкозы в плазме (FPG), инсулина (IRI), показателей липидного обмена в сыворотке крови с использованием коммерческих наборов (Roche Diagnostics GmbH). Чувствительность к инсулину и функцию β-клеток ПЖ оценивали с использованием HOMA-калькулятора v.2.2. Статистический анализ выполнен с использованием сертифицированной программы SPSS (версия 9.0). Данные приведены в виде среднего и 95 % доверительного интервала. Р-значение менее 0,05 считали статистически достоверным.

Результаты и обсуждение. Среди 292 обследованных подростков артериальная гипертензия выявлена у 37,7 %, гипертриглицеридемия — у 35,9 %, сниженный уровень липопротеинов высокой плотности (ЛПВП) — у 31,9 %, нарушение толерантности к глюкозе — у 8,9 %, у 91,1 % больных — инсулинорезистентность (HOMA% S < 100 %). С помощью одномерного регрессионного анализа установлена нелинейная связь (r = 0,67, p = 0,0001) между чувствительностью к инсулину и функцией β-клеток ПЖ. У подростков со сниженной чувствительностью тканей-мишеней к инсулину (HOMA% S < 100 %) достоверно чаще отмечали повышение уровня триглицеридов (ТГ) — отношение шансов (ОШ) = 16,6 [16,4—16,8]; АД — ОШ = 2,13 [1,2—6,09]; снижение уровня ЛПВП — ОШ = 3,36 [1,9—5,2]. Повышение гликемии натощак (≥ 6,1 ммоль/л) у подростков с ожирением ассоциируется с напряжением секреторной функции β-клеток ПЖ — ОШ = 14,7 [10,3—19,1].

Выводы. Установлена корреляционная связь между чувствительностью к инсулину/функцией β-клеток ПЖ с такими компонентами МС, как уровень АД, ТГ, ЛПВП, FPG. У 15,4 % подростков с ожирением на фоне инсулинорезистентности и сниженной функциональной активности β-клеток не выявлено повышения АД и дислипопротеинемии, что в большей степени соответствует диагностическим критериям преддиабета, а не МС. У 35,9 % мальчиков-подростков с ожирением в сочетании с инсулинорезистентностью при нормальной функции ПЖ выявлены основные диагностические критерии МС, а именно: абдоминальное ожирение, артериальная гипертензия, высокий уровень ТГ и низкий — ЛПВП. Эти пациенты находятся в группе риска развития в старшем возрасте сердечно-сосудистых осложнений.

Ключевые слова: ожирение, метаболический синдром, инсулинорезистентность, пубертат.