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ORIGINAL ARTICLE

Metabolic monitoring of obese children born small for gestational age

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KEYWORDS
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Summary
Introduction: The ''catch-up growth'' phenomenon in children born small for gestational age (SGA) has been linked to early onset obesity with the subsequent emergence of metabolic syndrome (MetS) or its components. It has been postulated that the prevalence of MetS and its components increases strongly with age.
Materials and methods: A retrospective study was carried out over a 5 year period (2007—2011) to determine long-term metabolic complications in obese children born with normal weight for gestational age (appropriate for gestational age: AGA) and SGA. 517 patients were qualified into the study. According to birth weight and gestational age they were first divided into SGA (107 patients — 20%) and AGA (410 patients — 80%) and then by age into three subgroups: prepubertary group, pubertary group and adolescents. Blood pressure, lipids and glucose were measured. Oral glucose tolerance tests (oGTT) were performed in all subjects.
Results: Prepubertary patients showed no significant differences between SGA and AGA; 4.8% met the framing criteria (according to Weiss) for MetS. Pubertary patients showed a slightly increased prevalence of MetS among SGA patients 10.8%, compared to AGA patients 7.3%. MetS prevalence was significantly higher in obese adolescents born SGA 26.3% compared to AGA 15.7%.
Conclusion: MetS or its components develop progressively with age. Increased prevalence of MetS in SGA patients indicates that being born SGA appears to be an additional risk factor in the development of MetS starting with puberty.

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Introduction

About 3—5% of neonates are born small for gestational age (SGA). 90% of babies born SGA catch up in growth by the age of 2. The rapid ''catch up'' growth during the cell division period up to 2 years of age leads to hyperplasic obesity [1,2]. These children have an increased risk for later obesity and metabolic syndrome (MetS).

ESPE/L.W.P.E.S February 2006 — Consensus Manchester defines SGA <—2 standard deviations (SD) for weight and/or size. Accurate knowledge of gestational age, proper auxological parameters at birth, reference data for the target population and prenatal growth charts are essential for SGA diagnose.

SGA etiology should be identified whenever possible. Pathogenetic mechanisms are diverse and may influence prognosis and treatment efficacy. Intrauterine growth restriction may be caused by a number of fetal, maternal, placental and demographic factors. Thus, children born SGA are not a homogeneous group but consist of several subgroups with different etiologies. The etiology of prenatal growth restriction remains unidentified in approximately 40% of cases.

In 90% of the cases, catch-up growth occurs until the age of 2—4 years, whilst 10% of children born SGA do not recover target size and remain with short stature. The catch-up growth group have a high risk of developing MetS with all its components: obesity, impaired glucose tolerance, insulin resistance with subsequent development of diabetes, arterial hypertension, dyslipidemia. There is also a risk of developing adrenal pathology and reproductive pathology [3—5]. These changes have been related to intrauterine life environment and linked to epigenetic fetal programming.

Aim of the study

The study proposes 3 main objectives.

To determine the prevalence of SGA among obese children; to identify whether SGA is an additional risk factor for MetS and its components beside obesity; to establish the onset age of MetS in obese children born SGA.

Materials and methods

A retrospective descriptive study was conducted over a period of five years, between January 2007 and December 2011 at the Emergency Hospital for Children ''Louis Ţurcanu'' Timişoara in the departments of Diabetes and Nutritional Diseases, Endocrinology and Cardiology.

Table 1 Weiss criteria of MetS in children and adolescents; it is necessary to fulfill at least 3 of 5 criteria [6].

Criteria	Component
Obesity: AC or BMI	BMI Z score $\geq$ 2.0
Fasting Glucose or oGTT	Baseline glucose >5.6 mmol/l Glucose at 2 h >7.8 mmol and <11.1 mmol/l
Systolic blood pressure or diastolic blood pressure	>95th percentile for age, sex and height[a]
HDL cholesterol (mg/dL)	<5th percentile[b]
Triglycerides (mg/dL)	>95th percentile[b]

AC, abdominal circumference; BMI, body mass index; OGTT, oral glucose tolerance test; HDL, high density lipoproteins.
[a] National High Blood Pressure Education Program [13].
[b] Guidelines of the German working group on obese children and adolescents, 2008 [14].

Children were considered obese on the basis of age specific BMI reference guidelines from Centers for Disease Control and Prevention Child Growth Standards 2000 (above 95th percentile) [7]. When defining SGA, growth charts proposed by Niklasson [8] were used; newborns weighing less than 2 SD from the average for gestational age were considered as being SGA. Birth weight and height were obtained from patients' medical files as recorded at delivery.

The criteria we used to diagnose MetS were those from Weiss et al. [6] (Table 1).

We calculated Z score using the formula: measured value — average value in the reference population/standard deviation of the reference population [7].

Statistical processing of data on the distribution of obese AGA and SGA children according to age and diagnosis, using ANOVA — two factor without replication is illustrated in Table 3. By applying ANOVA — two factor without replication, the objective was to determine whether the differences between the sought data are significant enough, in order to draw pertinent conclusions.

Exclusion criteria were syndromal, chromosomal or infectious etiology of low birth weight, endocrine or syndromal disorders, systemic disease or acute illness.

We analysed 517 patients diagnosed with obesity, including 410 patients AGA and 107 patients SGA. Obese AGA and SGA patients were distributed into subgroups by age, namely prepubertal (5—10 years), pubertal (11—14 years) and adolescent