

case control study on 52 obese children (body mass index (BMI)>95th percentile) aged 4 to 16 years undertaken at the outpatient endocrine clinic of the Children Hospital at Tabriz University between 2009–2011. This study was conducted to compare the prevalence of vitamin D deficiency and secondary hyperparathyroidism in obese children compared with 57 non obese (BMI< 85th percentile). 109 children including 52 (50.5%) boys and 57 (49.5%) girls were studied. Most of case (76.9%) and control (42.1%) groups suffered from degrees of vitamin D deficiency. There was meaningful statistical difference between two groups considering to vitamin D deficiency and parathyroid hormone ($p = 0.001$). A negative relations was found between iPTH and vit D level ($p < 0.001$, $r = -0.2$), BMI and 25-OH vit D ($p < 0.001$, $r = -0.2$). A positive relation was observed between parathyroid hormone and BMI ($p = 0.009$, $r = 0.1$). Obese children are at high risk at vitamin D deficiency and secondary hyperparathyroidism. BMI appears to be an important risk factor for vitamin D deficiency.

1434 CLINICAL-LABORATORY PECULIARITIES IN CHILDREN WITH OBESITY AND METABOLIC SYNDROME

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Aim To determine clinical-laboratory peculiarities in children with obesity (O) and metabolic syndrome (MS).

Methods 119 children with O and MS were examined in the endocrinological department of University hospital (Minsk) over 2011 year. Group1 patients with O 90(75.6%) (boys/girls=50/40), mean±SD age 12.2±3, group2 MS 29(24.4%) (boys/girls=16/13), mean±SD age 14.2±2.2 yrs ($\delta = 0.04$). Insulin(Ins); total cholesterol(TC); triglycerides(TG); high-density(HDLc), low-density(LDLc) lipoprotein cholesterol; atherogenic coefficient(AC); OGTT with HOMAIR index were defined to all patients. The results were processed using the Statistica 6.1.

Results BMI boys group1 28.1±5.4 kg/m², group2 33.8±4.4 ($\delta = 0.3$); group1 girls 31.8±5.6, group2 36±5.5 ($\delta = 0.6$). The average levels of TC were in normal limits, gender and intergroup differences weren't noted ($\delta = 0.1$). TG boys and girls with MS were 1.73±0.93 and 2.02±0.6 (0.45–1.7 mmol/L), the reliable difference weren't noted ($\delta = 0.8$ and $\delta = 0.3$ respectively). HDLc was norm in all groups regardless of gender ($\delta = 0.2$). LDLc was upgraded in girls group2 3.43±0.79 (< 3.3 mmol/L) ($\delta = 0.4$). AC in boys and girls group2 was 3.31±1.16 and 3.81±0.88 (2–3) ($\delta = 0.1$). Basal and postprandial plasma glucose levels by conducting OGTT didn't exceed normal limits in group1 and group2 regardless of gender ($\delta = 0.08$). Ins boys group1 24.2±18mU/ml (2.1–22), group2 40.1±23.2 ($\delta = 0.1$); girls group1 20.8±14, group2 37.8±16.1 ($\delta = 0.6$). HOMAIR boys group1 5.16±3 (< 2.77), group2 10.87±5.9 ($\delta = 0.1$); girls group1 4.3±3, group2 8.5±3.4 ($\delta = 0.8$).

Conclusions Dyslipidemia was typical to group with MS. Insulin resistance with maintaining the basal and postrandial normoglycemia was noted by conducting OGTT in all patients regardless of gender.

1435 BIOMARKERS OF OXIDATIVE/ANTIOXIDATIVE BALANCE IN MACROSOMIA

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Background To investigate whether the anomalies affecting the antioxidant defenses could start at birth and to check the decrease in antioxidant defenses in macrosomic newborns.

Material and Methods Thirty macrosomic and 30 sex-matched control newborns were recruited for a retrospective case-control study at the Maghnia Maternity Hospital of Tlemcen Department (Algeria).

Results The serum plasma ORAC, albumin, vitamin E, SOD, CAT and GSH-Px levels were significantly decreased in macrosomic than in control newborns, yet no difference was observed after adjustment for weight. Additionally, serum concentrations of malondialdehyde and xanthine oxidase were significantly higher in macrosomic than in controls before adjustment for weight. Moreover, macrosomia was significantly associated with low levels of ORAC (OR = 4.96, 95%CI 1.2–20.55), vitamin E (OR = 4.5, 95%CI 1.29–15.68), SOD (OR = 6.88, 95%CI 1.35–35.11) and CAT (OR = 5.67, 95%CI 1.37–23.46), and with high levels of MDA (OR = 10.29, 95%CI 2.02–52.36).

Conclusions Excessive weight could be a potential factor for decreased anti-oxidative capacity and increased oxidative stress.

1436 MATERNAL WEIGHT GAIN DURING PREGNANCY AND NEONATAL BIRTH WEIGHT: MOROCCAN DATA

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Objective The objective of this study was to investigate the influence of maternal weight gain on birth weight of a population of newborns.

Patients and Methods Study including all patients who delivered in the service of the Maternity Hospital Provincial BENSLIMANE between 1 October 2010 and October 1, 2011. Three groups of patients were formed according to weight gain: less than 8kg, between 8 and 16kg and over 16kg normal. The epidemiological characteristics, obstetric complications and neonatal outcomes were analyzed. The survey is conducted on the basis of a questionnaire, for parturients and obstetric records analysis.

Results The mean birth weight was higher in the group “weight gain” ≥ 16 kg “(3782.9±595 g $p < 0.05$) and the rate of newborns weighing more than 3800g (45.5%, $p < 0.05$), unlike those weighing less than 2600g (hypotrophy) whose percentage was higher in the group “weight gain” < 8 kg (6.2% $p < 0.05$), weight gain greater than 16 kg represented a risk factor for dystocia (34.7%).

Conclusion An excessive weight gain during pregnancy has deleterious effects on neonatal trophicity. It promotes macrosomia. These data point out the interest to follow the recommendations of weight gain during pregnancy.

1437 MARKERS OF THE METABOLIC SYNDROME AND PHYSICAL ACTIVITY IN TEENAGE CHILDREN BORN PRETERM

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Background and Aims The worldwide increase in the Metabolic Syndrome is associated with adverse health outcomes and significant healthcare costs. Early life exposures are key factors in determining later health. Children born preterm appear to be at higher risks of developing insulin resistance. We aimed to determine the prevalence of novel metabolic biomarkers in a cohort of teenage children who were born preterm (< =34 weeks gestation) and correlate these with physical activity.

Methods We studied 24 children using standard techniques including auxology, body composition (BODPOD™), insulin resistance