METABOLIC COMPLICATIONS OF OBESITY IN CHILDREN

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10.1136/archdischild-2019-epa.639

Introduction Children with obesity have high risk of metabolic complications.

Aim To reveal frequency of metabolic complications in children with obesity.

Methods We examined 91 obese children, aged 7–17 (BMI > 95 percentile). Mean age of children was 12.46 ± 3.5 yrs. Obesity in children manifested at 5.3±0.5 yrs. Family anamnesis characterized by obesity in 71%, diabetes type 2 - in 25%, hypertension - in 53% in the first degree relatives. All patients were examined by clinical, biochemical, ultrasound methods. An oral glucose tolerance test (OGTT) accompanied by four point of insulinemia was performed. HOMA index was calculated according to the standard formula. In children with fatty liver chronic hepatitis were excluded. Metabolic syndrome (MS) was diagnosed according to a classical definition (Weiss's criteria).

Results In obese children metabolic complications were found in 39.6%. The prevalence of the metabolic complications was follows: hypertriglyceridemia 38.2%, glucose intolerance 17.6%, hypertension 52.6%. Increase of serum cholesterol was revealed in 24%, low density lipoproteins - in 14%, decrease of high-density lipoproteins – in 32% children. Metabolic syndrome was found in 18 (19.8%) patients with BMI 30.9 ± 3.4 kg/m². Insulin resistance revealed in 25% children. HOMA index was 4.6 ± 3.3 mU/l. Ultrasound signs of fatty liver were shown in 40 patients.

Conclusions This study showed a high prevalence of metabolic complications among obese schoolchildren.

Case Report The index is a 2.5 year old girl with moderate eczema associated with nocturnal itch. She was referred to local services and was trialled on topical Clobetasone and Betamethasone cream. After little improvement this was changed to topical Hydrocortisone (1%) and Betamethasone cream. Her parents applied the steroid creams on a continuous basis for at least four months. They also sourced different homeopathic creams from Pakistan. Upon analysis of their ingredients in the laboratory, Betamethasone and Dexamethasone were identified in one of them. She required a 5 day course of oral Prednisolone for an allergic reaction to nuts. Over time the eczema remitted but all treatments were continued. After six months, she presented with rapid weight gain over 2 month period [weight >98th centile]. This was associated with increased skin pigmentation over ankles and knees, striae over her thighs and marked hypertrichosis.

Biochemical testing confirmed absence of diurnal variation with suppressed 8am cortisol levels. She was switched to maintenance eczema care with emollients and topical Tacrolimus. An ACTH stimulation test confirmed adrenal suppression [peak Cortisol 45nmol/L]. Adjunctive investigations for other causes of hypoaldrenism were reported normal. We educated the family on emergency management of cortisol deficiency in case of emergency use only. After four weeks off all steroid containing medications she was placed on a 5 day course of oral Cortisone with suppressed 8am cortisol levels. She was switched to maintenance eczema care with emollients and topical Tacrolium. An ACTH stimulation test confirmed adrenal suppression [peak Cortisol 550nmol/L]. Adjunctive investigations for other causes of hypoadrenalism were reported normal. We educated the family on emergency management of cortisol deficiency in case of emergency use only.

Discussion The role of corticosteroids in the management of inflammation is well established but has resulted in widespread, prolonged and often unsupervised use. There is inter-individual variation in steroid sensitivity, small doses can result in dramatic adverse effects. We did suspect that the homeopathic medicine was steroid based. This does not rule out the possibility that the other ingredients facilitated steroid increase absorption or reduced metabolism.

Conclusion Adrenal suppression is a rare side effect of topical steroid use. Children on courses of steroids for longer than one week require medical monitoring to ensure that the treatment is appropriate, safe and used correctly.
the effectiveness of physical activity. The presence in patients with obesity and overweight polymorphisms of genes that adversely affect carbohydrate and fat metabolism, dictates the need to organize a special personalized approach to diet therapy and exercise.

**Background**

Neonatal diabetes mellitus is a rare condition with one case per 300,000 to 500,000 live births. It presents with marked hyperglycaemia in the first six months of life and is commonly of genetic origin. Approximately half of cases are transient (TNDM) with the reminder being permanent (PNDM). The majority of Permanent Diabetes Mellitus (PNDM) cases are secondary to genetic mutations in K-ATP channel genes.

**Case**

We report two infants who presented in the neonatal period with hyperglycaemia and were subsequently diagnosed with neonatal diabetes.

The first infant presented at 22 hours of life with blood glucose of 44.3 mmol/L and in ketoacidosis. He was born by LSCS at 34 weeks gestation due to IUGR with a birth weight of 1.36 kg. He received an IV insulin infusion for two months with frequent dose titration due to challenging glycaemic control. He was then successfully transitioned to a subcutaneous insulin infusion pump. Genetic analysis confirmed homozygous INS non sense variant, p(ARf43Ter).

The second infant presented at six weeks of age in severe diabetic ketoacidosis with a blood glucose of 60 mmol/L. She was born at term weighing 2.46 kg following induction of labour for IUGR. She presented with lethargy and vomiting for 24 hours on a background of failure to thrive and chronic candidiasis from birth. She was managed with an IV insulin infusion in the ICU before transitioning to a subcutaneous insulin pump. Genetic analysis revealed *de novo* KCNJ11 gene mutation. She was then successfully transitioned to oral Glibenclamide which optimised her glycaemic control and resulted in complete withdrawal of subcutaneous insulin.

**Discussion**

These are two cases of NDM with identified genetic mutations. The INS mutation is localised to amino acid residues affecting cleavage and/or folding of pro-proinsulin and pro-insulin which leads to prolonged ER stress and subsequent B-cell apoptosis. The KCNJ11 gene mutation identified in our second patient results in K-ATP channel overactivity. The K-ATP channel is at the end of the glycolytic pathway and has a KIR amino terminus responsible for channel gating. Its deletion results in channels that are continuously closed reducing sensitivity to inhibitory ATP and resulting in dramatic hyperglycaemia. Oral glibenclamide inhibits its K-ATP channel activity with resultant normoglycaemia. Early identification is essential for optimal management and targeted therapy.