METABOLIC FACTORS CONTRIBUTING TO THE DEVELOPMENT OF INSULIN RESISTANCE IN CHILDREN

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Annotation

Childhood obesity has become a pressing global epidemic, closely linked to the early onset of metabolic syndrome and insulin resistance (IR). This thesis investigates the specific metabolic factors driving IR in school-aged children. Based on a cross-sectional clinical and laboratory analysis of 131 children, the study reveals severe metabolic disruptions, including hyperinsulinemia, atherogenic dyslipidemia, and hyperleptinemia, highlighting the urgent need for early targeted interventions.

Key words: insulin resistance, metabolic syndrome, pediatric obesity, lipid profile, leptin, glucose tolerance.

Introduction

The global prevalence of pediatric overweight and obesity has increased exponentially, representing a major public health crisis. According to the World Health Organization, 1.9 billion people worldwide are overweight or obese. In the pediatric demographic, excess adiposity is the primary trigger for insulin resistance, which serves as the pathophysiological core of metabolic syndrome, type 2 diabetes, and cardiovascular diseases. The development of IR is a complex, multifactorial process influenced by genetic, hormonal, and environmental factors. Understanding specific metabolic alterations—such as changes in lipid profiles and adipokine secretion—is crucial for developing effective preventive strategies for children.

Material and methods

A cross-sectional clinical and laboratory study was conducted involving 131 school children aged 10-11 years. Participants underwent comprehensive anthropometric and biochemical evaluations. Based on the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) index and clinical screening, a main investigative group demonstrating clear signs of IR (23 children) and a normo-metabolic control group (20 children) were identified for detailed physiological comparison. Biochemical measurements included fasting glucose, oral glucose tolerance test (OGTT), fasting insulin, lipid profile (total cholesterol, LDL, HDL, triglycerides), and serum leptin levels, evaluated using automated biochemical analyzers and Enzyme-Linked Immunosorbent Assays (ELISA).

Result and discussion

The investigation revealed profound metabolic deviations contributing to the pathogenesis of IR. Both urban and rural cohorts exhibited marked basal hyperglycemia (6.2 and 6.1 mmol/L, respectively, vs. a 4.8 mmol/L baseline) and severe hyperinsulinemia. Fasting insulin levels in both groups reached 11.83 μU/mL, representing a pathological 1.69-fold increase over the normal baseline of 7.0 μU/mL. The OGTT demonstrated significant impairment in glucose clearance, particularly at the 3-hour mark (reaching up to 15.0 mmol/L in rural children), indicating early beta-cell exhaustion and compromised peripheral insulin sensitivity.

Furthermore, atherogenic dyslipidemia was highly prevalent as a contributing metabolic factor. Compared to the healthy baseline, LDL (bad cholesterol) surged by 45.6% in urban and 61.8% in rural children. Concurrently, protective HDL cholesterol dropped significantly by 22.8% and 32.4%, respectively, while triglycerides saw massive elevations up to +68.9%.

Finally, serum leptin—a critical adipokine—was pathologically elevated by 103.8% in urban children and 176.3% in rural children compared to controls. This massive hyperleptinemia indicates severe leptin resistance, driven by visceral fat accumulation and nutritional imbalances, further exacerbating the metabolic collapse.

Conclusion and recommendation

The development of insulin resistance in children is aggressively driven by a triad of metabolic factors: continuous hyperinsulinemia, severe atherogenic dyslipidemia, and profound hyperleptinemia. To combat this growing epidemic, it is strongly recommended to implement mandatory, comprehensive metabolic screening in schools that goes beyond basic BMI measurements to detect early lipid and glycemic abnormalities. Public health policies must focus on rigorous nutritional education, improving school meal standards by reducing simple sugars and animal fats, and creating accessible infrastructure for daily physical activity to restore energy balance in the pediatric population.

References

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