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Triglyceride-Glucose Index: A Practical but Partial Surrogate of Insulin Resistance in Children with Obesity

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Introduction:

Childhood obesity is strongly associated with insulin resistance (IR) and the early development of cardiometabolic complications. However, direct assessment of IR using gold-standard methods is impractical in large paediatric population studies. The triglyceride–glucose (TyG) index, derived from fasting triglyceride and glucose concentrations, has emerged as a simple surrogate marker of IR in adults, but evidence in children, particularly those with obesity, remains limited. This study aims to determine the correlation between the TyG index and HOMA-IR and other IR-related markers in children with obesity.

Methods:

A total of 524 children with obesity aged 10–16 years were included in this cross-sectional analysis. Anthropometric measurements and fasting biochemical parameters, including glucose, lipid profile, insulin, leptin, and adiponectin, were assessed. Obesity status was defined using BMI z-score: overweight ($>+1.0$ SD), obesity ($>+2.0$ SD), and morbid obesity ($>+3.0$ SD). IR was defined as HOMA-IR ≥ 4.0 . Participants were stratified into tertiles of TyG index. Triglyceride and glucose concentrations were converted to mg/dL prior to TyG calculation using the formula: $TyG = \ln [TG (mg/dL) \times FPG (mg/dL)]/2$. Higher TyG values indicate lower insulin sensitivity. Correlations between TyG index and IR-related parameters were assessed using Pearson correlation coefficients.

Results:

Children with IR had a significantly higher mean TyG index compared with insulin-sensitive children (4.50 ± 0.22 vs 4.36 ± 0.21). The TyG index showed moderate correlations with HOMA-IR ($r = 0.374$, $p < 0.001$) and fasting insulin ($r = 0.366$, $p < 0.001$), and weak correlations with HbA1c ($r = 0.103$, $p < 0.001$) and leptin:adiponectin ratio ($r = 0.104$, $p < 0.001$). Children in the highest TyG tertile exhibited greater waist circumference, higher total cholesterol, triglyceride:HDL-C ratio, fasting insulin, HbA1c, and leptin:adiponectin ratio. Furthermore, those in the highest TyG tertile had a higher frequency of IR, abdominal obesity, and metabolic syndrome compared with children in the lowest tertile.

Conclusion:

The TyG index demonstrates moderate association with established IR markers in children with obesity. However, its limited correlation with glycaemic control and adipokine balance suggests that TyG primarily reflects insulin–glucose–lipid interactions. Therefore, the TyG index should be used in combination with other metabolic markers to more comprehensively assess IR and cardiometabolic risk in paediatric populations.