

CONCLUSION

Repaglinide remains an important armamentarium in personalized diabetes management. Its pharmacokinetics and flexible dosing allow intensification of glycemic control while minimizing the risk of hypoglycemia. It also offers a viable strategy for selected patients struggling with complex insulin regime.

EP_A067

SUBARACHNOID HEMORRHAGE: A RARE BUT DEVASTATING COMPLICATION OF DIABETIC KETOACIDOSIS

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INTRODUCTION

Diabetic ketoacidosis (DKA) is a common complication of type 1 diabetes mellitus (T1DM). Neurological complications of DKA are rare in adults. We report a case of young lady with T1DM who presented with severe DKA and complicated with subarachnoid hemorrhage (SAH).

CASE

A 19-year-old female was diagnosed with T1DM following an admission for DKA 2 months earlier. Her hemoglobin A1c was 17.2%, and autoimmune markers showed positive anti-glutamic acid decarboxylase (anti-GAD) antibodies (71 IU/mL) with negative anti-insulinoma-associated antigen 2 (anti-IA2) and islet cell antibodies. Her C-peptide level was low at 69 pmol/L. She was discharged with basal bolus insulin analogues, but she defaulted on her medications. She presented to the hospital 2 months later with a history of epigastric pain, vomiting, and breathlessness for 2 days, and was then found unconscious at home. There was no history of trauma. She was intubated for a poor Glasgow Coma Scale (E1V1M1). Blood results showed severe DKA with glucose 25 mmol/L, blood gas pH 6.572, bicarbonate 2.6 mmol/L, and ketone more than 8.8 mmol/L. She was started on insulin infusion and fluid resuscitation. DKA resolved after 24 hours. Computed tomography (CT) of the brain showed SAH in the right frontal and left frontotemporal regions. CT angiography of the brain reported no evidence of an intracranial aneurysm. She was extubated on day 4 of admission. Subsequent cerebral digital subtraction angiography was unremarkable. The patient achieved complete neurological recovery on follow-up.

CONCLUSION

This case highlights that severe DKA may rarely be associated with spontaneous SAH, which increases morbidity and mortality. Proposed mechanisms include vascular injury, oxidative stress from severe ketoacidosis, and endothelial dysfunction related to inflammatory mediators. Clinicians should have a high suspicion and consider neuroimaging in DKA patients with unexplained reduced consciousness or atypical neurological findings.

EP_A068

ASSOCIATION OF TESTOSTERONE WITH INSULIN RESISTANCE AND BETA-CELL FUNCTION IN TYPE 2 DIABETES MELLITUS

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INTRODUCTION

Insulin resistance and progressive beta-cell dysfunction are key components of the pathophysiology of type 2 diabetes mellitus (T2DM). The triglyceride-to-high-density lipoprotein cholesterol (TG/HDL) ratio has been widely used as a marker of insulin resistance. In addition, testosterone deficiency has been associated with adverse metabolic profiles and T2DM. However, the relationship between testosterone levels, insulin resistance, and beta-cell function in patients with T2DM remains incompletely understood.

METHODOLOGY

A cross-sectional study was conducted involving 25 male patients with T2DM attending the diabetic clinic at Dr. Mohammad Hoesin General Hospital. Serum testosterone, C-peptide, TG, and HDL levels were obtained from routine clinical data. The TG/HDL ratio was calculated as an index of insulin resistance. Normality was assessed using the Shapiro-Wilk test, and due to the non-normal distribution of key variables, Spearman correlation analysis was applied.

RESULTS

A strong inverse correlation was observed between testosterone levels and TG/HDL ratio ($r = -0.860$, $p < 0.001$), indicating that higher insulin resistance was associated with lower testosterone levels. In addition, TG/HDL ratio showed a strong negative correlation with C-peptide levels ($r = -0.741$, $p < 0.001$), suggesting reduced beta-cell function in the presence of increased insulin resistance.

CONCLUSION

In patients with T2DM, increased insulin resistance is strongly associated with lower testosterone levels and reduced beta-cell function. These findings highlight the interaction between metabolic dysfunction and hormonal status in middle-aged and elderly patients with T2DM.

EP_A069

WHOLE-BODY PHASE ANGLE AS A MARKER OF CELLULAR HEALTH IN A WORKPLACE LIFESTYLE INTERVENTION

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INTRODUCTION

Phase angle (PhA) derived from bioelectrical impedance analysis reflects cellular integrity and has emerged as a potential marker of metabolic health. Its role in detecting early physiological changes during lifestyle intervention remains underexplored in workplace obesity programmes.

METHODOLOGY

This longitudinal study included 30 adults with complete paired measurements enrolled in a 12-month workplace lifestyle programme. Body composition was assessed using multifrequency bioelectrical impedance analysis. Measurements included whole-body PhA, body cell mass (BCM), skeletal muscle index (SMI), and extracellular water to total body water ratio. Paired comparisons and correlation analyses were performed.

RESULTS

Mean PhA increased modestly from $5.59 \pm 0.70^\circ$ to $5.67 \pm 0.69^\circ$, although this did not reach statistical significance ($p = 0.17$). Changes in PhA were not significantly associated with changes in BCM ($r = -0.03$, $p = 0.89$). However, baseline PhA demonstrated a moderate positive association with SMI ($r \approx 0.51$, $p < 0.01$), suggesting a link between cellular integrity and muscle status. Individual trajectories revealed heterogeneous responses despite minimal weight change.

CONCLUSION

Whole-body phase angle provides insight into cellular health beyond conventional anthropometric measures. While short-term changes were modest, PhA may serve as a clinically useful adjunct for monitoring physiological response in obesity management programmes.

EP_A070

INTEGRATING BODY COMPOSITION AND BIOCHEMICAL MARKERS FOR METABOLIC RISK STRATIFICATION IN LIFESTYLE INTERVENTION

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INTRODUCTION

Obesity is a heterogeneous metabolic disease in which individuals with similar body weight may exhibit markedly different physiological risk profiles. Conventional monitoring using weight alone may fail to capture underlying metabolic and cellular changes during lifestyle intervention. Integrating body composition and biochemical markers may improve risk stratification in clinical practice.

METHODOLOGY

A prospective observational analysis was conducted among participants enrolled in a structured lifestyle programme. Baseline and follow-up assessments included biochemical markers (fasting glucose, renal function, lipid profile, and liver enzymes) alongside body composition parameters derived from bioelectrical impedance analysis, including percent body fat, visceral fat area, skeletal muscle index (SMI), and phase angle (PhA). Changes over time and associations between metabolic and body composition variables were analyzed.

RESULTS

Metabolic responses varied substantially despite similar anthropometric profiles. Renal function improved significantly, with increased estimated glomerular filtration rate (eGFR) observed over time ($p = 0.01$), and was inversely associated with fasting glucose ($r = -0.41$, $p < 0.01$). Lipid abnormalities persisted, although reductions in low-density lipoprotein cholesterol were noted ($p = 0.04$). Total cholesterol remained elevated and correlated positively with fasting glucose ($r = 0.36$, $p = 0.02$). Improvements in aspartate aminotransferase were observed ($p = 0.03$), while other liver markers remained stable. Notably, body composition parameters, including PhA and SMI, demonstrated variability independent of weight change, reflecting heterogeneous physiological adaptation.