**Type 2 diabetes remission in obesity: a weight-centric approach**

**Background:**

Obesity affects one in three Australian adults and most patients with type 2 diabetes mellitus (T2DM) are obese, highlighting how both disorders share an underlying pathophysiological defect. Clinical studies have demonstrated that marked weight loss, particularly when early and >15% of total bodyweight, often induces T2DM remission and exerts benefits that extend beyond glycaemic control. Until recently, bariatric surgery was the only intervention that could provide and sustain weight loss of this magnitude. However, current anti-obesity pharmacotherapy affords substantial weight loss, which should represent the foundation of T2DM treatment.

**Case Study:**

A 44-year-old female weighing 169.5 kg (BMI 67.9 kg/m2) was recently diagnosed with T2DM with an HbA1c of 11.9% and fasting blood glucose (FBG) of 17.6 mmol/L, complicated by comorbid obstructive sleep apnoea, gastro-oesophageal reflux disease, hypertension, dyslipidaemia, and depression. Following the introduction of a very-low energy diet, daily physical activity, low dose metformin (500 mg daily), and incretin therapy she lost 20.3 kg (-12% total bodyweight) over a period of 6 months. In addition, her HbA1c normalised to 5.4% with a FBG of 5.1 mmol/L. Furthermore, she reported a marked improvement in quality of life due to better sleep and improved stamina, allowing increased participation in outdoor activities with her family. With ongoing lifestyle interventions and multidisciplinary involvement, she was able to lose a further 54.2 kg over the next 30 months (total weight loss -74.5 kg, -44%) with additional improvements in lipid profile (triglycerides, HDL) and physical functioning, whilst remaining in T2DM remission.

**Discussion:**

This case emphasises the evident glycaemic and cardiometabolic benefits of implementing a weight-centric approach in patients living with T2DM and obesity. Substantial weight loss, as a disease modifying upstream intervention, can potentially disrupt or even reverse the underlying pathophysiology of T2DM and therefore should be the primary treatment goal in these patients.