GLP-1RA in Hospital Sultanah Nur Zahirah Terengganu, a tertiary centre in East Coast Malaysia. Clinical outcomes assessed were weight, BMI, total daily dose of insulin (TDD), HbA1c reduction and adverse events after six months of treatment initiation.

RESULTS

A total of 12 patients were eligible for this study, with a median age of 48 years (24.3). The majority were Malays (91.7%), with 1 Indian. There were equal numbers of males and females. Ten patients had diabetes, with four diagnosed for more than ten years. Nine (75%) were on insulin treatment prior to GLP1-RA initiation, with a median baseline TDD of 66 IU/day (74.0). Two patients were started on GLP1RA for obesity. Eight patients were on injectables, and the remaining were on oral GLP1RA. At baseline, the median weight, BMI and HbA1c were 123.3 kg (49), 48 kg/m^2 (18.9), and 8.5% (3.8), respectively. After six months, there were significant reductions in median weight and BMI, 111 kg (47.3) and 41.3 kg/m^2 (16.2) (P-value = 0.012, 0.018, respectively). A median weight reduction of 5% from baseline was observed. There was a reduction in median HbA1c to 7.7% (4.0), however, this was not statistically significant (P-value = 0.34). No change in TDD was observed (P-value = 0.85). Three patients (25%) experienced mild gastrointestinal symptoms, which did not require discontinuation of GLP1RA.

CONCLUSION

GLP1-RA is effective for weight loss even with a shorter treatment duration, while the effect on HbA1c and TDD reduction may require a longer treatment duration.

EP_A061

DECIPHERING THE PUZZLE: GLP-1 AGONIST-INDUCED ACUTE KIDNEY INJURY UNRELATED TO MEDICATION SIDE EFFECT

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INTRODUCTION/BACKGROUND

GLP-1 agonists are crucial treatments for type 2 diabetes and obesity due to their positive impact on glucose control and weight. However, concerns have arisen about their potential to cause pre-renal acute kidney injury (AKI), typically attributed to gastrointestinal side effects. This case presents a notable anomaly: AKI occurring without gastrointestinal symptoms, prompting further investigation into the mechanisms behind GLP-1 agonist-associated AKI.

CASE

A 35-year-old Malay male was seen with morbid obesity class II, young-onset hypertension and stable chronic kidney disease stage 3. He was started on semaglutide at 0.25 mg weekly, with plans to up titrate to 0.5 mg weekly. Upon initiation, renal function showed a concerning decline in eGFR from 54 to 35 mL/min/1.73 m^2. Despite ruling out gastrointestinal side effects or dehydration and obstructive uropathy through ultrasound, semaglutide was temporarily withheld. After a month without the medication, his eGFR improved to 53 mL/min/1.73 m^2. We then cautiously initiated Liraglutide at 0.6 mg OD which resulted in a weight reduction from 98 kg to 94 kg within a month. However, renal function deteriorated further, with eGFR dropping to 34 mL/min/1.73 m^2 and creatinine levels rising to 204 mmol/L. Understanding the importance of preserving renal function, we subsequently discontinued GLP-1 agonist therapy.

CONCLUSION

Despite the absence of gastrointestinal side effects, GLP-1 receptor agonists can still be associated with acute kidney injury. Hence, it is important to monitor renal profile regularly, especially when starting treatment with these medications.

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INTERTWINING FATE OF PROLACTIN AND METABOLISM: THE OVERLOOKED CAUSAL EFFECT RELATIONSHIP

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INTRODUCTION/BACKGROUND

Hyperprolactinaemia has always been regarded as an excess of hormones resulting in metabolic abnormalities. However, recent studies have shown that prolactin has a protective value against metabolic disorders in obese individuals.[1] Increased circulating prolactin inhibits adipocyte hypertrophy, downregulates expression of inflammatory cytokines in visceral adipose tissue and alleviates insulin resistance.[1] However, chronically high prolactin influences orexigenic-anorexigenic hormones, resulting in hyperphagia, weight gain and obesity.[2] We present a patient with Metabolically-Healthy-Obesity [MHO] with hyperprolactinemia.