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Case Report

Successful management of type 2 diabetes, obesity and urinary incontinence in a 45-year-old female patient

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Abstract

This case report presents a 45-year-old female patient with uncontrolled Type 2 Diabetes Mellitus (T2DM), with obesity, and stress urinary incontinence (SUI) requiring frequent diaper use. The patients underwent a stepwise treatment approach, starting with glycemic regulation and weight reduction using GLP1 receptor agonist (GLP-1RA) combined with insulin therapy and weight reduction strategies. After achieving significant metabolic improvement and weight loss, the patient successfully underwent surgical intervention with an artificial urinary sphincter (AUS) implantation, leading to complete resolution of incontinence. This case highlights the importance of a comprehensive, multidisciplinary approach in managing complex metabolic and urological conditions.

Keywords: type 2 diabetes mellitus, obesity, stress urinary incontinence, GLP-1 1receptor agonist, metabolic control, weight loss, artificial urinary sphincter

Introduction

T2DM and obesity frequently co-exist, increasing the risk of complications such as diabetic neuropathy, nephropathy and urinary incontinence due to multiple factors, increased intra-abdominal pressure and impaired pelvic floor function [1]. Obesity and insulin resistance contribute to poor metabolic control [2], while stress urinary incontinence (SUI) is exacerbated by increased intra-abdominal pressure and potential autonomic neuropathy affecting bladder function [3, 4]. The coexistence of these conditions poses therapeutic challenges, requiring a comprehensive, phased management strategy to address both metabolic dysfunction and urinary complications [5].

Conventional management of T2DM includes lifestyle interventions, oral antidiabetics, and insulin therapy, but newer treatments such as GLP-1 receptor agonists (GLP-1Ras) [6] offer benefits in weight loss and glycemic control [7, 8]. Recent therapeutic advances, particularly the introduction of glucagon-like peptide-1 receptor agonists (GLP-1RAs), have provided clinicians with tools that can address multiple disease mechanisms simultaneously. In recent years, GLP-1 receptor agonists (GLP-1RAs) have demonstrated superior efficacy in improving glycemic control, facilitating weight loss [9], and reducing cardiovascular risks [10–12]. In cases where conservative and metabolic optimization measures fail to resolve persistent incontinence [13], surgical intervention, such as artificial urinary sphincter (AUS)

implantation, remains the gold standard for achieving continence and restoring quality of life.

This case highlights how GLP-1RA therapy [14] weight reduction and AUS placement successfully addressed both metabolic and urinary issues.

Case presentation

Patient profile

The patient, a 45-year-old woman, presented with a long-standing history of Type 2 Diabetes Mellitus (T2DM), obesity, and severe urinary incontinence. Her diabetes had been diagnosed a decade earlier and had remained poorly controlled despite previous insulin therapy, with progressive deterioration of metabolic parameters over the years. Her weight had steadily increased, resulting in a body mass index (BMI) of 36 kg/m^2 at the time of presentation. Hypertension was also present but was well controlled with amlodipine. The urinary incontinence, which had persisted for three years, was severe enough to require continuous use of diapers, significantly affecting her mobility, daily activities, and self-esteem. Pelvic floor exercises and bladder training had been attempted without success, and the condition had worsened over the preceding year.

Upon examination, she appeared obese but showed no signs of acute distress. Vital signs were stable, and cardiovascular and respiratory examinations were unremarkable. Pelvic examination revealed no obvious pelvic organ prolapse but confirmed poor pelvic floor tone. Initial laboratory investigations revealed a marked metabolic derangement, with glycated hemoglobin (HbA1c) at 9.5%, fasting blood glucose at 10 mmol/L, and postprandial glucose at 14.0 mmol/L. Dyslipidemia was evident, with total cholesterol at 5.68 mmol/L, LDL cholesterol at 3.62 mmol/L, HDL cholesterol at 0.98 mmol/L, and triglycerides at 2.82 mmol/L. Renal function was preserved, with a serum creatinine level of 79.58 µmol/L and an estimated glomerular filtration rate (eGFR) of 85 mL/min/1.73 m^2. Urinary assessment revealed microalbuminuria, with a urinary albumin-to-creatinine ratio of 60 mg/g, alongside glycosuria and mild proteinuria on dipstick testing (Table 1).

Treatment strategy

Management proceeded in two phases, beginning with metabolic control and weight reduction, followed by surgical correction of urinary incontinence. The initial priority was to optimize glycemic regulation and reduce the patient's body weight, as both factors were contributing not only to poor metabolic health but also to the severity of her urinary incontinence. Pharmacologic therapy was initiated with a GLP-1 receptor agonist, specifically semaglutide, at a dose of 1 mg weekly, chosen for its dual benefits in lowering blood glucose

Table 1: Diagnostic assessment.

Parameter	Value	Reference range
HgA1c	9.5%	<7%
Fasting blood glucose (FBG)	9.99 mmol/l	4.1–5.9 mmol/l
BMI	36 kg/m^2	18.5–24.9 kg/m^2 (normal)
Total cholesterol (TC)	5.68 mmol/l	4.1–5.2 mmol/L
LDL-C	3.62 mmol/L	2.2–3.7 mmol/L
HDL	0.98 mmol/L	1.0–2.0 mmol/L
Triglycerides (TG)	2.82 mmol/L	0.3–1.7 mmol/L
Serum creatinine	79.58 umol/L	49–115 umol/L
eGFR	85 ml/min/1.73 m^2	>90 ml/min/1.72 m^2
UACR	60 mg/g	<30 mg/g (normal)
Urinalysis	Glycosuria (+), proteinuria (+)	-

Note: HgA1c – Hemoglobin A1C; FBG – Fasting blood glucose; BMI – body mass index; TC – total cholesterol; LDL-C – low density lipoprotein cholesterol; HDL – high density lipoprotein cholesterol; TG – triglycerides; e GFR – estimated glomerular filtration rate; UACR – urinary; albumin-to-creatinine ration.

and promoting weight loss. Basal–bolus insulin therapy was also introduced, using insulin glargine for steady background control and insulin aspart to manage postprandial glucose spikes. Metformin at 1000 mg twice daily was continued to improve insulin sensitivity.

Alongside medication, a structured lifestyle program was implemented, focusing on a calorie-restricted, low-carbohydrate, high-protein diet tailored to her preferences and nutritional needs. This was paired with a supervised exercise regimen consisting of moderate-intensity aerobic activities and resistance training for approximately 45 minutes a day, five days a week. Over a six-month period, the patient achieved a 12% reduction in body weight, with her BMI decreasing from 36 to 31.5 kg/m^2. Glycemic control improved markedly, with HbA1c decreasing from 9.5% to 6.8%, fasting glucose dropping from 10mmol/L to 6.1mmol/L, and postprandial readings falling within near-target range. Lipid parameters also improved, particularly LDL cholesterol and triglycerides.

Despite these significant metabolic and anthropometric improvements, the patient continued to experience daily episodes of urinary incontinence, with no substantial relief from prior conservative measures. Given the persistence of symptoms and the impact on her quality of life, surgical intervention was considered. After a thorough urological evaluation, she underwent implantation of an artificial urinary sphincter (AUS) using the AMS 800 system under general anesthesia. The postoperative course was uneventful, and bladder retraining commenced soon after recovery. Within three months, the patient reported complete continence, eliminating the need for diapers, and described substantial improvements in mobility, confidence, and social participation. This phased approach—prioritizing glycemic control and weight loss before surgical intervention—ensured that she entered surgery in an optimized metabolic state, reducing perioperative risk and enhancing the likelihood of durable postoperative success.

Discussion

The objective of this case report was to demonstrate the efficacy, safety and metabolic benefits of GLP-1 RAs in an obese patient with T2DM inadequately controlled on insulin therapy (±OADs) [7, 15, 16]. GLP-1RA improves glycemic control and promotes significant weight loss (6–15%) through appetite suppression and delayed gastric emptying [6, 14]. It also reduces insulin requirements while improving HgA1c, and cardio-renal-metabolic risk in high-risk diabetic patients [17].

Obesity is a major contributor to stress urinary incontinence due to increased intra-abdominal pressure [2, 3]. Even a weight reduction of 5–10% can lead to a 50% reduction in incontinence episodes [13]. AUS implantation is the gold standard for severe stress urinary incontinence in patients who fail conservative measures. In this case, AUS completely resolved the patient's incontinence, significantly improving her quality of life.

Learning points

- Stepwise Approach: A phased treatment strategy combining medical and surgical intervention is crucial for patients with multifactorial health conditions like T2DM, obesity, and urinary incontinence;
- GLP-1RA Efficacy: GLP-1 receptor agonists are highly effective in achieving glycemic control and weight reduction, which can alleviate incontinence symptoms in obese patients;
- AUS for severe SUI: Artificial urinary sphincter implantation remains the gold standard for severe stress urinary incontinence when conservative measures fail;
- Multidisciplinary care: Successful outcomes require collaboration between endocrinologists, urologists, and dietitians to address both metabolic and urological needs.

Conclusion

This case highlights the synergistic effects of GLP-1RA therapy, insulin optimization, and weight loss, followed by surgical intervention (AUS implantation), in successfully managing a complex case of uncontrolled T2DM, obesity, and urinary incontinence. This approach led to successful glycemic regulation, weight reduction, and complete resolution of urinary incontinence. A phased multidisciplinary approach incorporating advanced diabetes therapies, lifestyle intervention, and urological procedures can lead to optimal metabolic and functional outcomes.

Conflict of interest

The authors declare no conflict of interest

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Consent to participate

Written informed consent was obtained from all the participants.

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