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Use of semaglutide in a 54-year-old patient with cocaine abuse and weight loss: a case report

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Abstract

Context This case report is interesting because it highlights a direction for the treatment of comorbid obesity and cocaine use disorder, which is an increasing clinical condition from an epidemiological point of view, and allows us to identify the possibility of a new strategy to address the problem of substance craving, particularly for cocaine.

Case presentation This case report discusses the efficacy of semaglutide in a 54-year-old Caucasian patient with a history of cocaine abuse and obesity. Subcutaneous semaglutide was administered, as per guidelines, with a progressive weekly increase for a total of 12 weeks. The patient was monitored with respect to clinical parameters, as well as psychodiagnostic ones. The patient demonstrated significant weight loss and a marked reduction in cocaine craving.

Conclusion The action of semaglutide on the hunger and reward centers offers a new approach to the treatment of patients with obesity and concomitant substance use disorders. By targeting glucagon-like peptide-1 receptors involved in both metabolic regulation and reward processing, semaglutide could potentially reduce both food intake and drug craving, thereby improving outcomes for these patients. The findings suggest that semaglutide may be a promising therapeutic option for the management of substance abuse in patients with comorbid obesity.

Keywords Obesity, Cocaine abuse, Semaglutide, Craving, Case report

Introduction

Obesity and substance abuse, particularly cocaine use, represent significant public health challenges. Recent studies have indicated that medications used primarily for metabolic disorders, such as semaglutide—a glucagon-like peptide-1 (GLP-1) receptor agonist—may have potential benefits beyond weight management, including the modulation of reward pathways associated with substance use disorders.

Obesity—a global health challenge

Obesity has become a significant global health issue, with its prevalence nearly tripling since 1975. As of 2021, more than 1.9 billion adults are classified as overweight, and over 650 million adults are obese, which is defined as having a body mass index (BMI) of 30 kg/m^2 or higher [45]. Obesity is linked to a wide range of comorbidities, including type 2 diabetes, cardiovascular diseases, and cancer, contributing to increased morbidity and mortality. It also imposes a heavy financial burden on healthcare systems [17]. The development of obesity is complex, involving genetic, behavioral, environmental, and metabolic factors. Hormones such as leptin and ghrelin, which

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regulate appetite and satiety through the hypothalamus, play a critical role in energy balance [30].

Cocaine abuse and its neurological underpinnings

Cocaine is a highly addictive substance with significant public health implications, especially in Western countries. It works by blocking the reuptake of dopamine, norepinephrine, and serotonin, leading to elevated neurotransmitter levels and stimulating postsynaptic receptors, producing euphoria and addiction [38]. Chronic use leads to neuroadaptive changes in the brain's reward system, particularly in the mesolimbic dopamine pathway, involving the ventral tegmental area (VTA) and nucleus accumbens (NAc). These changes drive compulsive drug-seeking behavior and dependence [20]. Cocaine addiction is also characterized by intense cravings that can persist long after cessation, leading to relapse [25].

The intersection of obesity and substance use disorders

There is a substantial overlap between obesity and substance use disorders, both of which involve shared neurobiological pathways in the brain's reward system. The concept of "food addiction" explains compulsive eating behaviors in obesity, where high-calorie foods trigger similar cravings to drugs [11]. Both conditions are associated with reduced dopamine D2 receptor availability in the striatum, leading to diminished sensitivity to natural rewards and prompting compensatory overeating or drug use [39, 42]. Individuals with obesity are more likely to develop substance use disorders and vice versa. For example, individuals with higher BMI are more prone to substance misuse, and individuals recovering from addiction may overeat to compensate for drug abstinence [4, 27].

The role of GLP-1 in metabolism and behavior

GLP-1 is an incretin hormone secreted by the L-cells of the small intestine in response to food intake. It regulates glucose metabolism by enhancing insulin secretion, inhibiting glucagon, and slowing gastric emptying [16]. GLP-1 also influences food intake and body weight, making it a target for obesity treatments. Recent studies have revealed its role in the central nervous system, where it affects reward processing. GLP-1 receptors in brain regions such as the nucleus accumbens, VTA, and hypothalamus modulate the rewarding properties of food and drugs, suggesting potential in treating obesity and substance use disorders [1, 9].

Semaglutide—a GLP-1 receptor agonist with dual potential

Semaglutide, developed initially for type 2 diabetes, improves glycemic control by enhancing insulin secretion, suppressing glucagon, and promoting satiety [16]. It has shown superior efficacy in weight loss compared with other treatments, primarily by increasing satiety and decreasing appetite, particularly for high-fat foods [2, 44]. Preclinical studies suggest semaglutide may reduce the rewarding effects of addictive substances such as alcohol and cocaine, showing potential for treating substance use disorders [37]. GLP-1 receptors in brain areas associated with addiction, such as the nucleus accumbens and VTA, underscore its broader therapeutic potential [1, 9, 20].

Case presentation

The patient is a 54-year-old man with a 15-year history of cocaine abuse and a diagnosis of obesity (BMI: 35.4 kg/m^2, weight: 105 kg). The patient had previously attempted various weight loss programs and substance abuse treatments without sustained success. He presented to me seeking assistance with both weight management and substance use. The decision to initiate semaglutide therapy in this patient was influenced by several critical factors, including the patient's dual diagnosis of obesity and chronic cocaine use. The intersection of these conditions presents a unique therapeutic challenge, as each condition can exacerbate the other. Given this overlap, a treatment that addresses both metabolic dysregulation and abnormal reward processing was deemed most appropriate.

Patient background and initial assessment

A 54-year-old Caucasian man presented with primary concerns of obesity and chronic cocaine use, which had impacted his physical and mental health. With a 15-year history of cocaine abuse, marked by frequent binges, intense cravings, and failed attempts to quit, the patient struggled with shame and relapse. Despite this, he had not pursued long-term addiction treatment owing to skepticism and fear of withdrawal. At the time of presentation, he had a BMI of 35.4 kg/m^2 (105 kg) and led a sedentary lifestyle with poor dietary habits, contributing to significant weight gain over the past decade.

He also suffered from depression and anxiety, partially managed with a selective serotonin reuptake inhibitor (SSRI), but his symptoms worsened during periods of increased drug use or attempted abstinence. He had no history of cardiovascular disease or diabetes, although his family had a history of hypertension and type 2 diabetes. His motivation for treatment was driven by the desire to improve his physical health, reduce stigma, and address the psychological toll of cocaine use. The clinical team implemented a comprehensive and integrative treatment plan to address both his obesity and the neurological and psychological aspects of cocaine addiction.

Intervention—initiation of semaglutide

We initiated a 12-week treatment with semaglutide, a GLP-1 receptor agonist, on the basis of evidence supporting its efficacy in promoting weight loss and potentially reducing drug cravings [37, 44]. The treatment began with a weekly dose of 0.25 mg, gradually increased to 1.0 mg by week 8, following standard titration protocols to minimize gastrointestinal side effects [6, 28]. Throughout the treatment, the patient's weight, BMI, and cocaine cravings were monitored weekly using the Cocaine Craving Questionnaire-Brief (CCQ-Brief) to track craving intensity [35, 36]. The patient's mood was also closely observed using the Hamilton Depression Rating Scale (HAM-D) and Hamilton Anxiety Rating Scale (HAM-A) to detect any mental health changes that could necessitate adjustments to psychiatric treatment [13, 14]. Given the ineffectiveness of SSRIs, they were gradually withdrawn to optimize treatment outcomes.

Weight and psychiatric outcomes

Outcome measures

1. Weight and BMI:
2. Baseline: the patient's initial weight was 105 kg, with a BMI of 35.4 kg/m^2.
3. Week 12: the final recorded weight was 92 kg, corresponding to a BMI of 31.0 kg/m^2.

This reflects a total weight loss of 13 kg (12.4% of initial body weight).

2. Cocaine craving:
3. Cocaine craving was assessed using the Cocaine Craving Questionnaire-Brief (CCQ-Brief), which measures craving intensity on a scale from 1 to 7, with higher scores indicating greater craving.
4. Baseline CCQ-Brief score: 5.6
5. Week 12 CCQ-Brief score: 2.3

This represents a significant reduction in craving, corresponding to a decrease of 58.9% in the CCQ-Brief score.

By the end of the 12-week treatment, the patient experienced a significant reduction in body weight, dropping from 105 to 92 kg, which corresponded to a decrease in BMI from 35.4 kg/m^2 to 31.0 kg/m^2 (Fig. 1) and represented a 12.4% reduction in weight [5, 44]. The patient reported improved physical health, including increased energy and reduced joint pain, and expressed satisfaction with his progress, which enhanced his self-esteem and motivation.

A notable outcome was the significant reduction in cocaine cravings. The patient's CCQ-Brief score decreased from 5.6 to 2.3 over 12 weeks, a 58.9% reduction (Fig. 1), suggesting semaglutide's potential influence

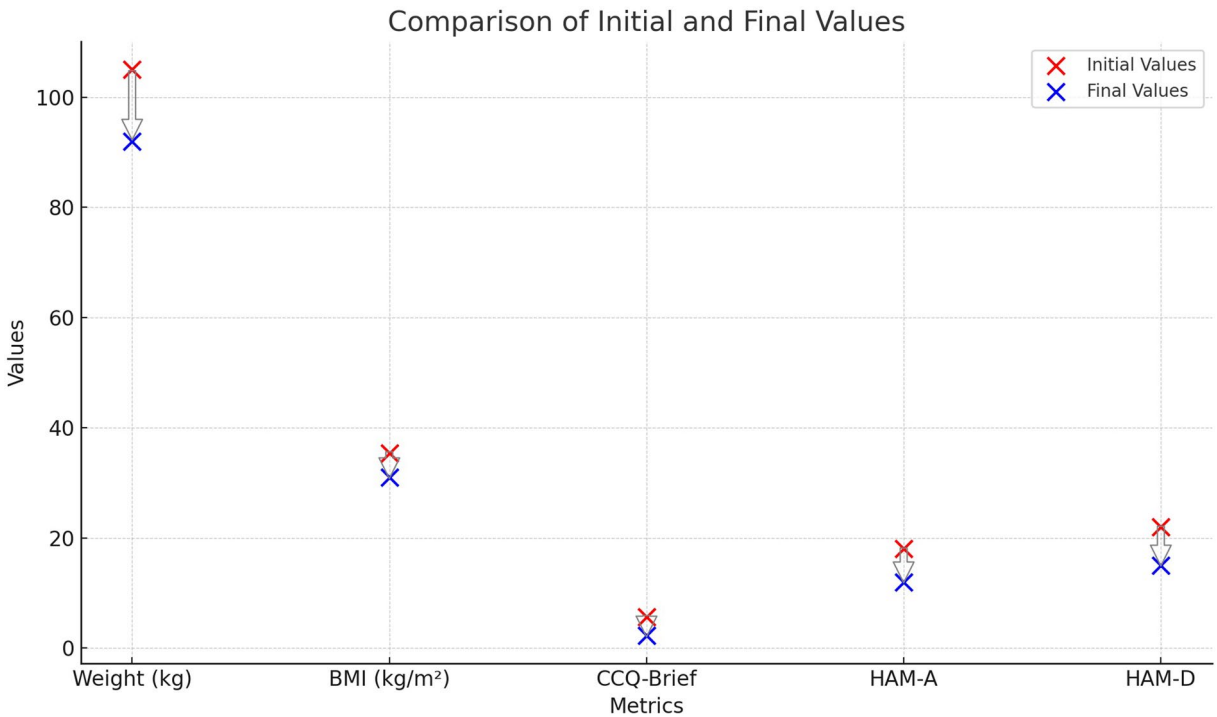

Fig. 1 Comparison of initial and final outcomes of weight and psychiatric values

on craving and reward circuits, though the mechanisms remain unclear [19]. As cravings decreased, the patient shifted focus to healthier activities such as exercise, aided by behavioral counseling that reinforced positive lifestyle changes.

Psychiatrically, his mood and anxiety remained stable, with slight improvements in HAM-D and HAM-A scores, likely reflecting the psychological benefits of weight loss and reduced cravings [10]. Overall, the patient felt more in control and optimistic about his future, marking significant progress in both physical and psychological health.

Adverse effects and safety profile

The patient tolerated the semaglutide treatment well, with no serious adverse effects reported. Mild gastrointestinal symptoms, including nausea and occasional diarrhea, were noted during the initial weeks of treatment, particularly during dose escalation. These symptoms were managed with dietary adjustments and did not require discontinuation of the drug [6].

Throughout the treatment period, the patient's vital signs and laboratory parameters remained stable, with no evidence of hypoglycemia or other metabolic disturbances. Regular monitoring of liver function tests and renal function showed no abnormalities, indicating that semaglutide was safe and well tolerated in this patient [22].

Discussion

This case highlights semaglutide's potential as a dual-action treatment for patients with co-occurring obesity and substance use disorders, suggesting it influences both metabolic and reward pathways. The patient's significant weight loss (13 kg, or 12.4% of initial body weight) and marked reduction in cocaine craving align with previous findings from clinical trials such as the STEP program, where semaglutide led to weight reductions of 10–15% over 68 weeks [44]. The rapidity of weight loss in this case, achieved in just 12 weeks, suggests that semaglutide may be particularly effective in individuals with comorbid conditions such as substance use disorders.

The reduction in cocaine craving, with a 58.9% decrease in the patient's CCQ-Brief score, is consistent with preclinical studies showing that GLP-1 receptor agonists reduce drug-seeking behavior and the reinforcing effects of addictive substances such as cocaine and alcohol [9, 37]. The exact mechanism by which semaglutide influences cravings is unclear but likely involves modulation of dopaminergic pathways in the brain's reward circuitry [19]

Semaglutide's weight loss effects are tied to its ability to reduce appetite, delay gastric emptying, and increase satiety, contributing to a negative energy balance [2]. Beyond weight management, GLP-1 receptor activation in brain regions such as the nucleus accumbens can reduce the reinforcing properties of drugs, suggesting semaglutide's potential in treating substance use disorders [1]. The neuromodulatory role of GLP-1, influencing synaptic plasticity and neurotransmitter release, may alter the brain's response to drug-related stimuli and reduce relapse risk [15, 43].

Moreover, the interaction between GLP-1 signaling and other neurotransmitter systems, such as glutamate and GABA, might further explain how semaglutide decreases cravings and drug-seeking behaviors [15]. The case suggests that semaglutide may exert effects beyond weight management, reinforcing the growing body of evidence that GLP-1 receptor agonists hold promise in the treatment of substance use disorders [19, 33]. Given the influence of GLP-1 on neural circuits involved in addiction, semaglutide could be especially useful in patients with comorbid obesity and substance use disorders, where these pathways may be particularly dysregulated. However, further research is needed to confirm these findings and better understand the mechanisms involved.

Conclusion

The case of a 54-year-old man with obesity and cocaine dependence provides valuable insights into semaglutide's potential for treating complex conditions involving both metabolic and neuropsychiatric aspects. The patient's significant weight loss and reduction in cocaine cravings suggest that semaglutide may offer an effective approach to addressing both issues simultaneously. However, these findings are preliminary, and further robust clinical trials, mechanistic studies, and long-term research are required to fully understand semaglutide's efficacy and safety in treating addiction. As addiction medicine evolves, semaglutide and other GLP-1 receptor agonists may become crucial components of personalized and integrated treatment strategies for patients with obesity and substance use disorders.

Implementing semaglutide in clinical practice requires careful patient selection, monitoring, and exploring the potential for combination therapies. In addition, it is essential to ensure equitable access to this treatment and reduce stigma surrounding pharmacological interventions for addiction. Traditional weight loss interventions, such as lifestyle changes and bariatric surgery, often fail to address the neurobiological factors underlying obesity, leading to difficulties in maintaining long-term weight loss [21]. Similarly, pharmacotherapies for substance use disorders rarely address associated metabolic complications [40].

Semaglutide has the potential to revolutionize the treatment of obesity and addiction, but achieving this will require coordinated efforts in research, clinical practice, and policy. As we refine the use of GLP-1 receptor agonists, we move toward more comprehensive care for patients with these interconnected conditions, potentially involving targeted combination therapies [24].

Limitations of the case and current evidence

Despite the promising results of this case, the limitations of a single case report must be acknowledged. Findings, while suggestive, cannot be generalized without further research, as individual patient factors, concomitant medications, and clinical context may influence outcomes [41]. The off-label use of semaglutide in treating cocaine dependence remains experimental, with early-stage clinical trials and preclinical evidence supporting its influence on drug cravings [9, 32]. Clinicians should approach semaglutide cautiously, integrating it into comprehensive treatment plans alongside behavioral therapy.

Short-term follow-up (12 weeks) limits understanding of long-term sustainability in weight loss and craving reduction. Longitudinal studies are needed to assess the durability of semaglutide's effects and long-term safety, including potential risks such as pancreatitis and thyroid C-cell hyperplasia [8, 21, 29].

Semaglutide's dual effects on weight loss and cravings raise questions about its mechanisms. While its peripheral metabolic effects are well understood [16, 23], its modulation of dopamine signaling in the nucleus accumbens and ventral tegmental area (VTA) suggests broader neuropsychiatric applications [7, 19]. Further research is needed to clarify how GLP-1 receptor activation influences addiction-related behaviors.

Future research directions

The outcomes of this case highlight several research directions. Randomized controlled trials (RCTs) are needed to evaluate semaglutide's efficacy in reducing drug cravings and promoting abstinence, comparing it wit other addiction treatments such as naltrexone, acamprosate, and disulfiram [18]. Mechanistic studies using neuroimaging, such as functional magnetic resonance imaging (fMRI) and positron emission tomography (PET), could offer insights into semaglutide's impact on brain regions linked to reward and impulse control, identifying biomarkers for treatment response [31, 40]. Longitudinal studies are critical for assessing the long-term effects on weight loss, craving reduction, relapse rates, and overall health [26]. In addition, combining semaglutide with behavioral interventions such as cognitive-behavioral therapy (CBT) or mindfulness could enhance treatment outcomes [3]. Further research should also explore semaglutide's potential in treating other addictions such as alcohol, opioid, and nicotine use [12, 34, 37].

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Author contributions

As the sole author, I declare that the research process is outlined in accordance with the guidelines for research publications.

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Availability of data and materials

I confirm that the data supporting the findings of this study are available and can be provided upon reasonable request. Any restrictions regarding data sharing have been noted.

Declarations

Ethics approval and consent to participate

I assure that all ethical standards required for this type of research have been met. No ethics committee approval was required. Patient consent for treatment was requested and obtained.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests

I declare that there are no competing financial or nonfinancial interests that could have influenced the outcomes of this research.

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