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Case Report

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Reduction in Patient-Reported Food-Related Preoccupation During Low-Dose Oral Semaglutide Treatment in Anorexia Nervosa, Binge-Eating/Purging Subtype

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Abstract

Background: Anorexia nervosa, binge-eating/purging subtype (AN-BP), is characterized by restriction of energy intake, intense fear of gaining weight, and distorted body image, with binge eating and/or purging behaviors as a potential feature. Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) act on gastrointestinal motility, appetite regulation, and food reward salience, and have been associated with reduced binge-eating frequency and a reported symptom called food noise (persistent, intrusive thoughts related to food consumption). Existing literature on GLP-1 RAs in anorexia-spectrum disorders has largely focused on misuse and associated weight loss.

Case Presentation: We present a 30-year-old woman with AN-BP, PTSD, generalized anxiety disorder, and major depressive disorder, with a decade-long history of restrictive eating, multiple hospitalizations, and persistent food noise. Oral semaglutide was previously used off-label to control food noise while minimally impacting weight. Food noise and eating behavior improved markedly within seven days of initiation; by eight weeks, PHQ-9 and GAD-7 scores had improved substantially, and weight increased to a stable, appropriate range, and her body mass index (BMI) remained above 18.5 at the eight-week follow-up.

Discussion: This case illustrates a potential pharmacological paradox, as GLP-1 agonists are usually associated with weight loss; it was temporally associated with weight gain and/or maintenance and reduction of food noise, supporting more consistent oral intake in an AN-BP patient. Given contrasting reports of misuse of semaglutide worsening restrictive pathology in atypical AN, response may depend on individual patient factors, underscoring the need for further study before generalizing this approach.

Keywords: anorexia nervosa; low-dose; oral semaglutide; binge-eating; purging subtype

Introduction

Anorexia nervosa (AN) is an eating disorder characterized by restriction of energy intake resulting in significantly low body weight, an intense fear of gaining weight or persistent behaviors precluding weight gain, and distorted body image. In the binge-eating/purging subtype (AN-BP), recurrent binge eating and/or purging behaviors occur in the preceding 3 months [1]. Current treatment guidelines emphasize nutritional rehabilitation, weight restoration, psychotherapy, and medical monitoring. In contrast, pharmacological options targeting the core psychopathology of AN are limited [2].

Glucagon-like peptide-1 receptor agonists (GLP-1 RAs), are incretin mimetic medications used to treat type 2 diabetes mellitus and obesity [3]. GLP-1 signaling is implicated in gastrointestinal motility, satiety, appetite regulation, and food reward salience [3-5]. Emerging evidence suggests that altered GLP-1 signaling is involved in the psychopathology of binge-eating disorders, and preliminary evidence suggests that GLP-1 RAs are associated with a reduction in binge-eating frequency in patients with binge-eating

disorder [6,7]. However, to our knowledge, there are no published reports describing physician-supervised GLP-1 RA treatment in patients with anorexia nervosa, binge-eating/purging subtype. Existing literature has instead focused on the misuse of GLP-1 RAs, primarily in atypical anorexia nervosa, where treatment was associated with significant weight loss rather than therapeutic management of the eating disorder [8,9].

The concept of "food noise" is an emerging, patient-centered construct that has recently been used to describe persistent food-cue reactivity that often leads to intrusive food-related thoughts and disordered eating behaviors [10,11]. As GLP-1 RAs may reduce food cravings, improve satiety, and inhibit food reward salience, these effects may plausibly attenuate food noise [12,13]. Nonetheless, their use in anorexia-spectrum disorders raises significant clinical concern, as their weight-lowering effects are in direct conflict with the primary therapeutic goal of prevention of further weight loss.

We present a case of anorexia nervosa, binge-eating/purging subtype, in which physician-supervised treatment with oral semaglutide was associated with a marked patient-reported reduction in food noise, while body weight remained largely stable in an appropriate range. Thus, this case illustrates a potential pharmacological paradox in which a medication typically associated with weight loss was utilized with apparent symptomatic benefit in a patient whose treatment fundamentally relied on maintaining a healthy body weight.

Case Presentation

Our patient is a 30-year-old woman with a history of post-traumatic stress disorder (PTSD), generalized anxiety disorder (GAD), AN-BP, and major depressive disorder (MDD).

The patient also has a 10-year-long history of attempts to lose weight with unknown medications, herbal remedies, and excessive aerobic exercise; she adopted a progressively restrictive approach, such as one-meal-per-day diets or no food intake for entire days at times. From a baseline of 120 pounds (BMI 22), the patient dropped to 100 pounds (BMI 18.3) in one episode five years ago, which resulted in her admission to a psychiatry unit. Over the following four years, she required five further hospitalizations for similar episodes.

Alongside attempts to lose weight, the patient complained of incessant food noise, described by her as intolerable food cravings and intrusive thoughts related to different tastes or recipes. After dietary counselling, the patient's BMI recovered to 19; semaglutide 3 mg orally daily was then commenced at age 27 by her psychiatrist, after the initial recommendation by her dietician, as an off-label attempt to reduce food noise, for which the patient gave informed consent. The stopping criteria implemented were withdrawal of semaglutide if her weight dropped below 94 lb. She was closely monitored weekly for detection of adverse effects, progress in symptoms, and weight; she was continued on semaglutide thereafter. Over the course of approximately three years, semaglutide was temporally associated with control of the patient's self-reported food noise with no major impact on her weight.

However, following a major psychosocial stressor six months ago (the end of a romantic relationship), the patient began to report symptoms of depression. Her PHQ-9 score was 20, consistent with severe depression [14], and her GAD-7 score was 15, consistent with severe anxiety [15], thus escitalopram (20 mg daily) was commenced alongside cognitive behavioral therapy. Following four weeks of adequate pharmacological and psychotherapeutic interventions, with consistent adherence, no clinical improvement was noted, and she admitted to engaging in purging behaviors such as purposeful induction of vomiting. On follow-up two months later, her body weight had decreased from 110 pounds (BMI 20.1) to 94 pounds (BMI 17.2). At this point, semaglutide was promptly discontinued and psychotherapy, alongside mirtazapine (30 mg daily), was initiated.

Mirtazapine was up-titrated to 45 mg daily after the fourth week, and by eight weeks, her PHQ-9 was still 14 (consistent with moderate depression), and the same concerns for her weight and restrictive eating behavior were present. Her weight at that time was 103 pounds (BMI 18.8), so olanzapine (5 mg daily) was added. Following a four-week trial, the patient reported no improvement of her food noise. Given that her BMI had increased to 18.8 kg/m2, and food-related preoccupation remained, oral semaglutide (3 mg daily) was reintroduced.

On one-week follow-up following the initiation of semaglutide (3 mg daily), her self-reported levels of food-related preoccupation and disordered eating habits had markedly reduced. She reported absence of

these intrusive and persistent food-related thoughts, and was adherent to a three-meal-per-day routine, stating that she felt less anxious about increasing oral intake as she believed semaglutide would reduce the likelihood of excessive weight gain. On eight-week follow-up, her mood additionally displayed marked improvement with a PHQ-9 score of 7 points (down 7 points), and a GAD-7 score of 5 points, consistent with mild anxiety [15]; a weight of 109 pounds (BMI 20) was additionally recorded.

Currently, she is maintained on oral semaglutide (3 mg daily), while olanzapine and mirtazapine were discontinued due to a lack of perceived clinical benefit. Clonazepam (2 mg as needed) was reserved for severe episodes of anxiety. She has remained adherent to individual and family behavior therapy.

Discussion

This case raises the hypothesis that, in a highly selected patient with AN-BP, a reduction in "food noise" may have indirectly supported adherence to nutritional rehabilitation despite the appetite- and weight-reducing effects associated with GLP-1 RAs. This should not be interpreted as semaglutide being an effective treatment for AN-BP nor as evidence that semaglutide facilitates weight gain; the patient's improvement occurred during concurrent psychotherapy and recent pharmacological treatment, thus substantially limiting causal inferences.

AN is associated with substantial medical and psychiatric morbidity and elevated premature mortality related to medical complications and suicide [16]. The patient consistently denied any suicidal ideation; however, the patient was at high risk due to her AN-BP and psychiatric comorbidities, thus, close monitoring was strictly followed.

Nutritional rehabilitation is often complicated by the patient's resistance and fear of gaining weight [17], as well as food-related intrusive thoughts [10,11]. Current treatment guidelines emphasize the combination of nutritional rehabilitation, psychotherapy, and medical monitoring, while pharmacological options only support treatment rather than cure the illness directly [2].

While GLP-1 treatment is conventionally used for weight-loss indications, it was largely associated with weight maintenance and gain in this case. GLP-1 RAs mimic the effect of endogenous glucagon-like peptide-1 (GLP-1), a form of incretin. Common drugs belonging to this class of medications include semaglutide, exenatide, and liraglutide. GLP-1 RAs lower blood sugar and promote weight loss through various properties, including stimulation of glucose-dependent insulin secretion [3], modulation of central reward pathways involved in food-reward salience, and attenuation of hedonic responses to food [5,13]. This reward-modulating property has generated interest in GLP-1 RAs as a treatment for binge eating disorder and bulimia nervosa, where the reduction of intrusive food cravings and binge urges has been observed in earlier studies [6,7]. It is possible that this property of modulation of central reward pathways, which may be involved in the concept of food noise, may have been implicated in the reduction in food noise observed in the patient despite weight maintenance. In this case, this property may have allowed for more consistent oral intake, both due to the reduction of food noise and the psychological reassurance that the patient's weight would not rise excessively, thus being associated with a seemingly paradoxical weight gain or maintenance with use of semaglutide.

Several limitations must be considered when interpreting this case report. The effects of semaglutide in Populations are not uniformly beneficial, and existing case literature suggests the response may not generalize. A reported case of semaglutide misuse in atypical AN highlighted contradictory exacerbation of restrictive pathology [8], while another described worsening of atypical AN in an adolescent female following semaglutide initiation [9]. These contrasting reports emphasize that the direction of the effect may depend heavily on individual factors, such as baseline eating psychopathology and psychiatric comorbidities. Additionally, the patient's weight had already increased from 94 lb to 103 lb before semaglutide was reintroduced, indicating other factors contributing to recovery. Furthermore, a significant limitation of the study is that this observational report does not constitute evidence sufficient to establish causation. Critically, the patient's clinical trajectory was not entirely stable; a major psychosocial stressor (a relationship breakup) triggered a depressive episode that necessitated temporary withdrawal of semaglutide due to substantial weight loss. This highlights a significant vulnerability where the patient's baseline psychological stability may play a role in treatment effect with GLP-1 RAs. Further large-scale, controlled studies are necessary to fully investigate this potential pharmacological paradox.

Conclusion

This case describes a temporal association between the initiation of semaglutide in a patient with AN-BP and marked reduction in patient-reported "food noise". This intervention was associated with mitigation of the patient's food noise symptoms and with psychological reassurance that excessive weight gain was unlikely, and was temporally associated with restoration of the patient's BMI to approximately 20 kg/m2 during follow-up.

Declarations

Authors Responsibility

All the authors independently verified all clinical information, interpretations, and references and take full responsibility for the final content

Conflict of Interest Statement

The authors have no conflicts of interest to disclose.

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Data Availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request. Data are not publicly available because they contain information that could compromise participant confidentiality.

Statement of AI Usage

No Artificial Intelligence tools were used in the creation, research, or drafting of this work. All content, data, and analysis are the sole product of human authorship and effort.

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