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Brochure of Published Abstracts

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From Awareness to Action: The Role of Opioid REMS Education in Safer Pain Management and Opioid Prescribing

Kathleen J. Harb, PharmD Candidate; Sarah A. Nisly, PharmD, MEd, BCPS, FCCP;
Lisa B. Phipps, PharmD, PhD

1. CP, healthcare professional; NP, nurse practitioner; PA, physician associate.

Introduction/Background:

In 2021, 75.4% of drug overdose deaths in the United States involved an opioid, underscoring the need for education in pain management and opioid safety¹

The Opioid Analgesic REMS program offers education on proper treatment and monitoring of pain through multiple methods²

Methods:

Study Design:

The longitudinal program consisted of 6 modules anchored by live webinars with corresponding recorded videos, content-related emails, podcasts, and downloadable slides

Participants could engage in as many or as few of the webinars and corresponding recorded webcasts as desired. For each topic, identical questions were asked prior to the educational content and immediately following the content.

The educational content was designed to cover the FDA Blueprint for Healthcare Providers Involved in the Treatment and Monitoring of Patients with Pain³

Content was designed using P-E-R principles⁴

Data Collection:

Demographic data from participants in 6 live and 6 online recorded webcasts were included.

As each participant moved throughout the longitudinal curriculum, participation in the FDA Blueprint components was tracked.

For each educational topic, responses were matched by participant and aggregated for a pooled analysis.

Primary Outcome:

Analyze the impact on participant knowledge and competence changes throughout a REMS-compliant longitudinal continuing education program.

Statistical Analysis:

Descriptive statistics were used to describe demographics and FDA Blueprint completion.

Microsoft Excel was used to analyze the answers to the preactivity and postactivity questions.

Results:

Questions in the "Managing Patients on Opioid Analgesics" module showed the most growth (average of 17.9%) between preactivity and postactivity testing.

The question about prescribing naloxone had the smallest growth rate (1.2%) and had only ~50% correctness in both preactivity and postactivity testing.

Questions in the "Designing Individualized and Effective Treatments" module showed the second-smallest growth rates (4.3% on average) but had high averages overall of correct answers (~80%).

Case-based questions were among those with the highest absolute increases, ranging from 19.3% to 30.3% from preactivity to postactivity.

Conclusions:

More robust education on naloxone prescribing needs to be included in future education given low growth rates and low averages.

High correct responses in some items for designing treatment plans allow for deeper discussion in these areas.

Case-based questions are useful for assessing multiple domains of information and can address >1 learning objective.

GLP-1 Agonists in the Reduction of Alcohol Use Disorder

Olivia Hill MS-III, Sarah Hughes MS-III, Aakanksha Singh MBBS DNB,
Michael Ang-Rabanes MD, Raja Mogallapu MD

Introduction:

Research has shown that glucagon-like peptides (GLP-1) agonists, which were originally developed as diabetes medications, have gained popularity due to their weight loss properties such as suppressing appetite and reducing the speed of gastric emptying. Additionally, these drugs have been found to mitigate substance use disorders due to their effects on the brain-gut axis. This makes them useful for treating coexisting comorbidities and reducing healthcare costs. In a case report, a 44-year-old male with morbid obesity and metabolic syndrome who had an inconsistent eating pattern and consumed significant amounts of alcohol (90 beers per month) was prescribed Dulaglutide. Within three months, he lost 25 pounds and reduced his alcohol use to 4-8 beers per month. This case report adds to the evidence supporting the positive effects of GLP-1 agonists on both weight loss and alcohol use reduction.

Case Presentation:

The patient was a 44-year-old male who came to the outpatient clinic seeking help for his weight gain and lifestyle management. During the visit, he weighed 275 pounds, had a body mass index of 41.8, and a waist circumference of 62 inches. He had a history of hypertension and was noted to be prediabetic, with a hemoglobin A1c of 5.8%. The patient's family had a history of obesity. He had undergone back surgery recently, which increased his weight. He reported consuming processed foods and doing less exercise. Additionally, he mentioned drinking approximately 90 beers per month and was in precontemplation about his alcohol use. The patient's AUDIT score, a 10-question screening tool for AUD, was 19.

He was prescribed Semaglutide 0.25 mg for weight loss and prediabetes, and lab tests for insulin and lipids were ordered. The patient received counseling on nutrition and lifestyle changes, including daily exercise of 20-30 minutes and restricting his food intake after dinner and before breakfast each morning. A preliminary laboratory study revealed that his insulin level was 27.4 mIU/mL, his HDL was 43, his triglycerides were 294, and his VLDL was 48. The patient was prescribed Dulaglutide 0.75 mg/0.5 mL since his insurance did not cover Semaglutide. In his first follow-up visit seven weeks later, he reported no adverse side effects and had lost ten pounds. He expressed that his drinking habits had changed, and he had reduced his beer consumption to only 30 beers per month. The patient reported feeling full after drinking and reduced urges and cravings to drink. During this visit, the patient agreed to increase his Dulaglutide dose to 1.5 mg for weight loss and prediabetes, and a follow-up appointment was scheduled in 5 weeks. In the second follow-up visit, the patient's waist circumference was from 47 inches, and he was drinking four to eight beers per month, with a new AUDIT score of 6. His weight had decreased to 250 pounds, with a body mass index of 37.3. His repeat laboratory tests showed a decrease in his triglycerides from 294 to 213, but no other significant changes were noted in his lipid panel. His HbA1c was at 5.4, and his Insulin level was at 13.7.

Discussion:

Alcohol use disorder (AUD) is one of the top causes of avoidable deaths, resulting in the loss of 3.3 million people annually to automobile accidents, liver disease, pancreatitis, and many other complications (7). Additionally, its costs are devastating, both on an individual and national level, contributing to nearly 12% of health-care costs in the US (12).

Current treatments for AUD are limited and have varying success rates, and many do not receive or seek treatment. Antidepressants, disulfiram, acamprosate, and naltrexone are the main pharmacological assets for AUD (17). One study showed that naltrexone and sertraline together were helpful, but when used on their own, they were not more helpful than the placebo (9). Naltrexone and acamprosate are the first line, and both have been shown to be mildly helpful but can be associated with unpleasant side effects. Literature reviews found that acamprosate and naltrexone have a NNT of 18 and 11, respectively, in order to have an effect, and one study found that naltrexone was able to reduce heavy drinking but not sobriety (17, 10). Furthermore, the hepatic metabolism of naltrexone can be contraindicated for those with existing liver disease commonly associated with AUD (9). Additionally, patients with concurrent opioid use also are unable to use naltrexone for AUD due to precipitation of withdrawal (14). Acamprosate has mixed data, and has been shown to be better at preventing any alcohol use as opposed to helping those with existing AUD; it also cannot be used in those with severe renal disease (10, 17). Disulfiram has very undesirable side effects and most patients will not

maintain use (17).

Not surprisingly, substance use disorders, including AUD, have been shown to be related to binge eating; one study found that in females, excess alcohol use correlated with binge eating (9). GLP-1 agonists, originally a diabetes medication due to their insulin-releasing properties, have recently been utilized for weight loss, even without diabetes (5). The agonists have multiple mechanisms, one of which slows gastric emptying to reduce appetite and increase fullness, including for drinks, and the other impacts and influences receptors in reward-seeking areas in the CNS (5).

The left insula, hypothalamus, and orbitofrontal cortex are involved with food anticipation and satiety, and GLP-1 receptors are found in these areas (5). Studies have also shown that GLP-1 decreases extracellular dopamine release and increases dopamine turnover, which decreases the reward aspect of food and alcohol (4). Multiple studies on mice have found that treatment with Exendin-4, a GLP-1 agonist, reduced alcohol consumption and reinforcement via these dopamine pathways (2, 4, 5, 15, 16). Importantly, following one of the studies, subjects did not have significant relapse following treatment cessation, which is very clinically and financially significant (16). It is also worth mentioning that liraglutide, which is marketed under the brand name 'Victoza,' has been shown to decrease alcohol consumption in rats by impacting the mesolimbic dopamine centers similarly (12).

Single nucleotide polymorphisms (SNP) in the GLP-1 receptor genes have shown clinically significant differences. One study showed that humans and mice with a GLP1R 168Ser non-wild type allele had a higher IV alcohol intake, as well as an increased signal intensity in the Globus Pallidus during a reward-seeking task (13). Similarly, mice with less expression of the 168Ser wild-type allele were found to respond less to GLP-1 agonists (13). While the current evidence is limited, the existing data on SNPs in the GLP-1 receptor genes and their role in alcohol and other substance use disorders shows promise and warrants future research.

Insulin plays a significant role in regulating blood sugar levels and controlling the metabolism of glucose and lipids. Insulin resistance in the brain can lead to an increase in alcohol intake and addiction; in our case, it was observed that after 12 weeks of treatment, there was a significant decrease in insulin levels, which may have contributed to the reduction in cravings for alcohol (2). Insulin resistance in the hypothalamus can affect the brain's reward circuitry, leading to an increased desire for alcohol and other substances of abuse (2). Therefore, the decrease in insulin levels may have played a role in reducing the cravings for alcohol.

Our patient presented with both weight gain and excess alcohol use. Even though his insurance would only cover Dulaglutide and not Semaglutide, his results were remarkable.

Conclusion:

This case report demonstrates a case of weight loss and significant alcohol use reduction in the setting of GLP-1 agonist use, drugs originally used for diabetes. There has been growing literature on the benefits of GLP-1 agonists not only on weight loss but also on AUD. The widespread effects of GLP-1 in the CNS, especially dopaminergic regions, and the GI tract work together to reduce cravings and urges for both food and substances. Our patient's case of an approximately 93% reduction in alcohol consumption over only 12 weeks provides additional support for a new and superior pharmacological treatment of AUD. While the results of this case report are promising, larger studies with more participants and longer follow-up periods are needed to confirm these findings and establish the efficacy of GLP-1 agonists as a standard treatment for AUD. Additionally, more research is required to understand the underlying mechanisms of GLP-1 agonists in reducing cravings and urges for both food and substances. Despite these limitations, the findings of this study provide a significant step forward in the development of new and improved pharmacological treatments for AUD.

Unraveling the Diagnostic Dilemma: A Case Report on Challenges in Early Parkinson's Disease Diagnosis

Aakanksha Singh MBBS DNB, Sarah Hughes MS-III, Olivia Hill MS-III,
Emily Gibson RN BSN student PMHNP, Michael Ang-RabanesMD,
RajaMogallapuMD ¹

Introduction/Background:

Neurodegenerative diseases like Parkinson's disease (PD) encompass both nonmotor symptoms as well as motor symptoms. A study by A.Maillet et al., explored the significance of serotonergic degeneration alongside the hypodopaminergic state in PD. Serotonin depletion causes prodromal non-motor symptoms such as loss of smell, constipation, urinary dysfunction, orthostatic hypotension, excessive daytime sleepiness, rapid eye movement sleep behavior disorder, apathy, anxiety, and depression. These symptoms may be specific to PD, and if they co-occur, there is a greater likelihood of a PD diagnosis. Additionally, corticolimbic dysfunction causes reduced activation in the ventral striatum and heightens activity in the dorsal striatum, which can lead to impulsive behaviors like pathological gambling in PD.

Aim of the study: To highlight the early occurrence of various non-motor symptoms, such as anhedonia and impulsivity leading to behaviors like gambling and hypersexuality, in Parkinson's disease. These symptoms can present initial challenges in the diagnosis and treatment of PD.

Evaluation & Workup

Positive for impulsivity but had no other symptoms of bipolar disorder

No history of Parkinson's psychosis or dementia and it was ruled out.

Negative family history

Psychological Testing: Mood disorder scale was negative for bipolar disorder

Physical & Neurological Examinations: Asymmetric bilateral rigidity in both hands, rigidity in his right leg, and reduced hand swing while walking. His muscle strength and other neurological examination was within normal limits

Mental Status Examination: Psychomotor retardation, flat affect, and poor insight about his health

Treatment: Amantadine 100mg TID for motor symptoms, buspirone 5mg TID for anxiety

A gradual tapering of olanzapine from 10mg to 5mg to rule out drug-induced parkinsonism (extra-pyramidal symptoms) and probably diagnoses of early PD associated with anhedonia.

Neurology Consultation: Ruled out all other causes and confirmed diagnoses of Parkinson's disease unspecified

Case Presentation:

A 58-year-old married male patient, evaluated two years ago for his first depressive episode after suffering a significant business loss, presented with symptoms of anxiety, racing thoughts, insomnia, decreased libido and significant weight loss. His wife noted a change in his demeanor from charming and easygoing to withdrawn and less motivated, spending most of his time watching TV.

Over the past four years, he has experienced financial losses due to gambling. Additionally, he has reported symptoms such as constipation,

handwriting difficulties, and trouble swallowing

The patient reported a lack of pleasure in previously pleasurable activities and easy fatigability in his daily chores

He was prescribed paroxetine, and olanzapine 10mg as a working diagnosis of bipolar disorder, by his previous

care provider. Olanzapine helped with his sleep and appetite without any improvement in his mood

Discussion:

Parkinson's Disease (PD) is the most prevalent neurodegenerative disease occurring typically in the 5th to 6th decade of life

Non-motor symptoms precede motor symptoms by 4-6 years and are linked to depletion of dopamine and serotonin, affecting emotional processing.

Common non-motor symptoms include gastrointestinal issues, apathy, anxiety, anhedonia, loss of smell, psychosis, and REM sleep disorders.

Early-stage symptoms like compulsive gambling, binge eating, and hypersexuality are attributed to dysregulation in brain pathways responsible for learning, decision-making, and reward processing

Mesolimbic and mesocortical pathways play crucial roles: mesolimbic for impulsive behavior and mesocortical for executive function

Dopamine agonists (e.g., carbidopa-levodopa) commonly cause these symptoms, and reducing dosage often leads to improvement

In this case study, isolated gambling and hypersexuality were features of impulsive-compulsive disorder in PD

Carbidopa-levodopa effectively treated non-motor symptoms of hypersexuality and gambling, improved anhedonia, fatigue and PD motor symptoms

Conclusion:

Late diagnosis of ICD with anhedonia along with movement disorders is not uncommon, as seen in this case

Early diagnosis and treatment of PD can improve the patient's prognosis and overall quality of life

Impulsivity symptoms like gambling and hypersexuality could be prodromal indicators of non-motor symptoms in Parkinson's, not just side effects of dopamine agonists

Pathological gambling and hypersexuality are commonly associated with males; females can present with compulsive shopping and binge eating