

were similar in age (mean 45.5 years), gender (51% male) and healthcare plan type. Preliminary analyses indicate that $\geq 10\%$ dose reduction is associated with increased risk of admission or ER visit for schizophrenia (hazard ratio [HR] 1.26; 95% confidence interval [CI] 1.18, 1.35; $P < 0.001$) and all psychiatric disorders (HR 1.18; 95% CI 1.11, 1.25; $P < 0.001$) versus controls, which may be even greater with $\geq 30\%$ dose reduction. Final updated results of ongoing analyses will be presented at the meeting.

CONCLUSIONS: Patients with antipsychotic dose reductions may be at risk for significant increases in hospital utilization rates. This suggests that dose reductions may increase overall healthcare burden in some schizophrenia patients, and highlights the need for alternative strategies in the management of TD.

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Buprenorphine/Naloxone (Suboxone and Bunavail)-Induced Glycolimia, an Indication of Undermedication?

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ABSTRACT: INTRODUCTION: Buprenorphine/Naloxone combination drugs such as Suboxone and Bunavail have not been reported to induce glycolimia. Two such cases are presented.

METHODS: CASE STUDY: Case 1: A 30-year-old, right-handed, white female with a history of opioid abuse was started on 4.2 mg buprenorphine/0.7 mg naloxone (Bunavail) BID and began sweet cravings and consumption of sweet foods. In a typical day she would eat 16 strawberry pop-tarts and 2 boxes of Little Debbie cookies. This may have provoked the 10 pound weight augmentation in the first two weeks of treatment. She denied any craving for opioids and no evidence of opioid withdrawal was present. Her Clinical Opiate Withdrawal Scale (COWS) score = 4 (normal).

Case 2: A 51-year-old, right-handed, male with opiate dependence, four days following the initiation of Suboxone (8 mg buprenorphine/2 mg naloxone) BID, developed strong cravings for sugary foods including donuts and ice cream, of which he was previously never

inclined to eat and gained 10 pounds in one month. His COWS score = 7 (mild symptoms).

DISCUSSION: There are myriad mechanisms that may be acting to induce sugar cravings with buprenorphine/naloxone. Humans and rats acutely withdrawing from opiates, such as heroin, develop strong urges for consumption of sugary substances (Lieblich et al., 1991; Sapira, 1968; Weiss, 1982). Glycolimia in the above cases may reflect early or subclinical withdrawal, which if becoming more severe, would manifest as opioid craving. If the value of the reward system induced by sweets doesn't meet the threshold invoked by the opioid stimulation, this "withdrawal" may lead to further sugar cravings in an attempt to reach the same reward level. In animals, certain foods and drugs share the same neurological pathway involved in the "reward system" potentially explaining why opioids influence food palatability in humans (Pelchat, 2002).

Alternatively, it is possible that buprenorphine induces hypoglycemia at high doses (Bullingham et al., 1981) such that hypoglycemia may paradoxically act to enhance sugar craving similar to the Somogyi effect in insulin dependent diabetics. Another possible mechanism of action is that since buprenorphine acts to decrease glucose metabolism in the brain (Walsh et al., 1994), this may lead to a neural compensatory response by increasing sugar access to the brain behaviorally via glycolimia and somatically reducing insulin release, thus explaining the high hemoglobin A1c observed in opioid addicts (Giugliano, 1984). Given the above presentation, complaints of sugar craving may indicate consideration to increase buprenorphine dosing and trial of this in those with glycolimia without opioid dependence may be warranted.

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Buspiron-Induced Somnambulism

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ABSTRACT: Objective: Buspiron has not heretofore been reported to trigger somnambulism. Such a case is described.

BACKGROUND: Buspiron is a partial 5HT1A agonist, which acts to suppress REM sleep and increase sleep fragmentation (Ware, 1994).

DESIGN/METHODS: A 36-year old right handed woman presented with one-year of constant anxiety and panic attacks with epochs of dyspnea, tachycardia, diaphoresis,

paresthesias of both hands, and out of body sensations. She affirmed déjà vu and PM insomnia, and vivid dreams. When in high school she had one sleepwalking event, without recurrence. The patient was begun on buspirone, initially 2.5 mg and raised to 5 mg QHS. Within a day of increasing the dose, she experienced an episode whereby in the early hours in the morning, she removed her pajamas, folded them neatly into a stack next to the bed, and returned to bed, sans clothes. She was amnesic for this event, but based this history on her husband's report. She discontinued the buspirone and for over 1 year, there has been no recurrence of such events.

RESULTS: Abnormalities in her neurological examination: Mental Status

EXAMINATION: Anxious. Memory: immediate recall: 7 digits forwards and 4 digits backwards: Cranial Nerve Examination: Cranial Nerve I: Alcohol Sniff Test: 8 cm (hyposmia), Reflexes: 3+ throughout. Neuropsychiatric testing: Clock Drawing test: 4 (Normal). Animal Fluency test: 20 (normal).

CONCLUSIONS: Buspirone induced somnambulism may relate directly to its mechanism of action, as a 5HT_{1A} partial agonist. Since other 5HT_{1A} agonists, to cause noctambulism (Raja2012). Buspirone increases sleep fragmentation (Wilson, 2005), and thus may act to disrupt slow wave sleep, promoting susceptibility to somnambulism. Alternatively, her somnambulism may be a nocturnal variant of Buspirone induced dissociative state (Bystritsky, 2013). Given the above, it is worthwhile to query those who are undergoing therapy with buspirone for the development of somnambulism.

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Emetophobia: The Specific Phobia of Vomiting: 2 Case Studies With 1-Year Follow-up

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ABSTRACT: EDUCATIONAL OBJECTIVES: At the conclusion of the session, the participant should be able: 1) To recognize emetophobia & how it can lead to significant impairment & reduce quality of life; 2) To appreciate the prevalence of emetophobia among the general population, 3) To learn that the selective serotonin reuptake inhibitor sertraline can be effective for emetophobia; 4) Need for more research.

INTRODUCTION: Emetophobia is an intense, irrational fear of vomiting. The prevalence rate of emetophobia in a community sample has been estimated to 8.8 % with a female to male ratio 4:1: It can occur at any age and can have a chronic course affecting one's academic/career, family, and social life.

METHODS: Case 1: B. was a 7 year old female referred by her pediatrician for psychiatric evaluation for her intense fear of vomiting. Mother shared that B's overwhelming fear of vomiting started when she was 6 years old and it may have stemmed from an incident when one of B's cousins threw up inside their van. At school, B constantly monitored whether or not anybody was getting sick around her. If she found out, someone was sick she began screaming and crying. B's academic performance was negatively affected due to her intense irrational fear.

Case 2: P was a 12 year male patient referred by his pediatrician for psychiatric evaluation after receiving 4 days inpatient treatment on the pediatric unit for dehydration. Patient reported that he was afraid of vomiting and gradually stopped eating and drinking & became so dehydrated that he was hospitalized. Several months prior to his hospitalization he had suffered from the flu & during that time he had intense vomiting and since then he has been fearful of a recurrence of the vomiting.

After a complete psychiatric evaluation, a diagnosis of emetophobia was established for each patient. Both patients were treated with sertraline.

RESULTS: B and P both started with initial dose of sertraline 12.5mg daily and then increased gradually over a period of next few months. They responded well with sertraline. B and P continued sertraline 50mg daily and 25mg daily as a maintenance treatment respectively. At 1 year follow up visit both of them were symptoms free.

CONCLUSIONS: Emetophobia is a common and chronic debilitating mental illness. Still there are no treatment protocols and randomized controlled trials for the treatment of emetophobia. CBT/Exposure-based therapies are the most commonly used approaches for emetophobia per literature. More studies are needed for a better understanding of emetophobia, which is relatively deserted illness although it can cause as much suffering as other major psychiatric disorders do and any patient presenting with these symptoms deserve to be evaluated and managed with scientific understanding and guideline.

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