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ABSTRACT: Introduction: Catatonia is an underrecognized neuropsychiatric syndrome affecting approximately 10% of individuals hospitalized on inpatient psychiatric units. First-line treatments for this condition include benzodiazepines (BZD) and/or electroconvulsive therapy (ECT). However, 20-40% of individuals do not respond to BZD alone and ECT is not always accessible. Second generation antipsychotics (SGA) have been used to treat catatonia in these circumstances. Here, we review the literature pertaining to the efficacy and safety of SGA in the treatment of catatonia.

METHODS: We conducted a PubMed search for articles linking catatonia to antipsychotics, under the search heading "catatonia" or "kahlbaum" and "risperidone", "amisulpride", "iloperidone", "olanzapine", "aripiprazole", "paliperidone", "clozapine", "brexpiprazole", or "cariprazine". Reports commenting on SGA treatment efficacy and/or their role in the development of catatonia were included in the analysis. Selected articles were reviewed for patient demographics, psychiatric/medical history, symptoms, cause of catatonia and treatment, and co-administered agents. For each SGA, we calculated the number of cases in which catatonia was likely improved with antipsychotic treatment, and the number of cases in which catatonia was precipitated or worsened with antipsychotic treatment (improved/worsened ratio). Case data was assessed using the Naranjo Adverse Drug Reaction Probability Scale. Descriptive statistics were used to analyze the data.

RESULTS: At the time this abstract was written, we reviewed 480 of the original 507 articles. One hundred and seventeen of the 480 met inclusion criteria. There was one randomized controlled trial (RCT), five prospective studies, four retrospective studies and 107 case reports. Of all reviewed literature quetiapine (34:3, 92%), aripiprazole (16:2, 89%), amisulpride (18:1, 95%), andclozapine (19:1, 95%) had the highest improved/worsened ratio, conversely paliperidone (0:5, 0%) had the lowest improved/worsened ratio.

CONCLUSION: Of the available literature quetiapine, amisulpride, aripiprazole, and clozapine were found to be relatively safe andeffective as treatment options in catatonia, while palipderidone was found to have reports pointing to its role in the development/worsening, but none on the improvement, of catatonia. These results need to be interpreted with caution. In the majority of cases where SGA's were effective, patients were co- treated with other pharmacologic agents (most frequently benzodiazepines), making it difficult to assess the role of the antipsychotic alone. Also, given that the preponderance of studies were case reports, publication bias may be an important limitation. Further studies are needed to examine the safety and efficacy of SGA in treating catatonia.

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Hospital Utilization Rates Following Antipsychotic Dose Reductions Among Patients With Schizophrenia

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ABSTRACT: Introduction: Tardive dyskinesia (TD), an often-irreversible movement disorder, develops in patients treated withantipsychotics. Although antipsychotic dose reduction has been utilized in the management of TD, the benefits and risks of lowering doses have not been well studied and could cause additional burden to patients.

OBJECTIVE: To analyze the healthcare burden of antipsychotic dose reduction in patients with schizophrenia.

METHODS: Medical claims from six US states spanning 6 years are retrospectively analyzed for ≥10% or ≥30% antipsychotic dosereductions and compared with those from patients receiving stable doses. Outcomes measured include inpatient admissions and emergency room (ER) visits for schizophrenia, all psychiatric disorders, and all causes.

RESULTS: Baseline analysis revealed 17,984 patients with ≥10% and 14,029 patients with ≥30% dose reduction. Patients with ≥ 10% dose reduction and matched controls

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were similar in age (mean 45.5 years), gender (51% male) and healthcare plan type. Preliminary analyses indicate that ≥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 ≥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 glycolimiaand 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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Buspirone-Induced Somnambulism

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

BACKGROUND: Buspirone 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,

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