

ABSTRACT: Introduction: Choreaform movements provoked by opiates is an infrequent adverse event. Buprenorphine induction of chorea has not heretofore been described. Such a case is presented.

METHOD: Case Study: A 38-year-old female presented with a decade long history of alcohol, cocaine, benzodiazepine, and heroin abuse. The patient was insufflating 1.5 grams of heroin daily. On presentation, she was actively withdrawing, scoring 17 on the Clinical Opioid Withdrawal Scale. Urine toxicology screening was positive for opiates, cocaine, and cannabinoids. Buprenorphine 4 mg sublingual was initiated. Within one hour, she observed, "My legs were moving uncontrollably as if I was a marionette." These dance-like movements were isolated to both legs and gradually resolved after discontinuation of buprenorphine: most of the movements manifested in the first 8 hours, and dissipated over the next 2 days. She did have similar movements after treatment with quetiapine during a previous hospitalization, years earlier.

RESULTS: Abnormalities in physical examination: General: goiter, bilateral palmar erythema. Neurological examination: Cranial Nerve (CN) Examination: CN I: Alcohol Sniff Test: 2 (anosmia). Motor Examination: Drift testing: mild right pronator drift. Reflexes: 3+ bilateral lower extremities. Neuropsychiatric Examination: Clock Drawing Test: 3 (abnormal). Animal Fluency Test: 18 (normal). Go-No-Go Test 6/6 (normal).

DISCUSSION: Buprenorphine induced chorea could be a result of partial mu-opioid agonism, or kappa and delta receptor antagonism (Burke, 2018; Cowan, 1977). Mu-opioid receptor activation causes increased dopamine turnover in the nigrostriatum, which is responsible for locomotor sensitization (Campos-Jurado, 2017). With the addition of mu-opioid receptor modulation of dopamine release, kappa-opioid receptor alters various neurotransmitters in the basal ganglia, potentiating hyperkinetic movements. Buprenorphine's choreiformogenic action may be due to kappa-opioid receptors ability to augment neurotransmission in the striatum (Escobar, 2017; Bonnet, 1998). The combination of simultaneous activity of these three opioid receptors may cause chorea, since they act to modulate dopamine, glutamate, and GABA in the direct and indirect pathways within the basal ganglia (Abin, 1989; Cui, 2013; Allouche, 2014; Trifilieff, 2013). This patient's history of heroin and cocaine use may have caused supersensitization of dopamine receptors (Memo, 1981), provoking hyperkinesia. Involvement of substance-induced sensitization with concurrent kappa-opioid receptor neurotransmitter augmentation in direct and indirect pathways in the basal ganglia may

have primed our patient to the development of chorea after buprenorphine administration. Further investigation for the presence of extrapyramidal movements in those undergoing buprenorphine treatment is warranted.

132 Treating Comorbid Childhood Bipolar Disorder and ADHD

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ABSTRACT: Objectives: According to Nemeth et al. (2011), pediatric mania is difficult to distinguish from childhood hyperactivity. Both share 3 common symptoms: distractibility, motoric hyperactivity, and talkativeness (Biederman, 2000). Oftentimes, children are referred from their pediatrician due to a lack of appropriate response to stimulant medication. Pediatricians have learned that merely raising the dose or changing the stimulant does not work. A thorough neuropsychological evaluation often reveals Bipolar Mania. They may have comorbid Bipolar Disorder and ADHD. This poster paper will examine measures that can assist in this important differential diagnosis as well as offer treatment options, including medication management.

METHODS: This case study includes three pediatric patients diagnosed with Childhood Bipolar Disorder and ADHD. A comprehensive psychoeducational assessment was conducted for each of the patients, which resulted in this comorbid diagnosis.

RESULTS: One of the most helpful measures was the TOVA. When a child's attention and impulsivity scores are normal, and response time and variability scores are abnormal, both on and off medication, that is an indication of a mood disorder (Nemeth et al., 2007). These children also performed poorly on measures of processing speed, and verbal learning and interference tasks (Henin et al., 2007). Measures of affect and personality were important diagnostically. A combination of Amantadine and either Clonidine HCL ER or Propranolol, as prescribed by a medical psychologist, were found to be effective in controlling the symptoms of this comorbid diagnosis.

CONCLUSIONS: An evaluation of children's intellectual, attentional, behavioral, mood, and personality functioning is crucial for a differential diagnosis. In cases of

comorbidity, ADHD and Childhood Bipolar Disorder, the sooner the child is on appropriate medications, the better. When just the surface diagnosis of ADHD is medicated, the outcome is often problematic. There may be a poor response to treatment and a higher rate of suicide.

133 Toxic Psychosis: Follow-up After One Year of Treatment

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ABSTRACT: Introduction: Anti-NMDA (N-methyl-D-aspartate) receptor encephalitis often presents itself in psychiatric settings as first-break psychosis. I present a case of a 31-year-old female who returned to the clinic one year after being treated for NMDA receptor antibody encephalitis.

CASE REPORT: Ms. C is a 31 y/o female who returned to the clinic after one year of being discharged from the hospital for NMDA-receptor encephalitis with positive serological NR1 antibodies. She was initially admitted to our inpatient psychiatric facility for an unspecified psychotic disorder complicated with seizure-like episodes. She was given various psychotropic medications without any improvement. She was moderately responsive to olanzapine and lorazepam. Her condition gradually worsened; she stopped communicating and became mute. Neurology consultation prompted work-up for encephalitis and the probable diagnosis of NMDA receptor encephalitis. She was subsequently treated with steroids, IVIG and then intrathecal rituximab and bortezomib. In addition to these aforementioned medications, she underwent a prophylactic oophorectomy and 10 ECT treatments for life threatening catatonia. After three weeks of this regimen, Ms. C recovered completely and was discharged home.

DISCUSSION: This case adds to literature that suggests prompt diagnosis and management of NMDA receptor encephalitis significantly improves prognosis. Treatment should be initiated if the patient meets probable diagnostic criteria for NMDA receptor encephalitis. Similar to other cases in the literature, our patient's symptom of catatonia improved with ECT administration. During Ms. C one year follow-up, no evidence of psychotic symptoms were appreciated. Family members reported that she had returned to her baseline cognitive function.

134 Long-Term Deutetrabenazine Treatment Is Associated with Sustained Treatment Response in Tardive Dyskinesia: Results from an Open-Label Extension Study

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ABSTRACT: Background: In the 12-week ARM-TD and AIM-TD studies evaluating deutetrabenazine for the treatment of tardive dyskinesia (TD), the percentage of patients achieving $\geq 50\%$ response was higher in the deutetrabenazine-treated group than in the placebo group. These studies also showed low rates of overall adverse events (AEs) and discontinuations associated with deutetrabenazine. The current open-label study evaluated the long-term efficacy and safety of deutetrabenazine in patients with TD.

METHODS: Patients with TD who completed ARM-TD or AIM-TD could enroll in this open-label, single-arm extension study, titrating up over 6 weeks to a maximum total daily dose of deutetrabenazine 48 mg/day on the basis of dyskinesia control and tolerability. The proportion of Abnormal Involuntary Movement Scale (AIMS; items 1-7) responders was assessed based on response rates for achieving $\geq 50\%$ improvement from baseline in the open-label extension study. AIMS score was assessed by local site raters for this analysis.

RESULTS: 343 patients enrolled in the extension study. At Week 54 (n=249; total daily dose [mean \pm standard error]: 38.6 \pm 0.66 mg), the mean percentage change from baseline in AIMS score was -40%; 48% of patients achieved a $\geq 50\%$ response and 59% of those had already achieved a $\geq 50\%$ response at Week 15. Further, 34% of those who had not achieved a $\geq 50\%$ response at Week 15 achieved a $\geq 50\%$ response at Week 54. At Week 106 (n=169; total daily dose: 39.6 \pm 0.77 mg), the mean