EQ 5D-5L utility in the full Cohort 2 (-0.021, P<0.001) and limited Cohort 2 (-0.024, P<0.001). A significant association was also found with patient rated severity in limited Cohort 2 (P<0.05), but not with clinician-rated severity. Similar results were found for SDS total score.

CONCLUSIONS: RE-KINECT patients were consistent in evaluating the severity and impact of TD, whether based on subjective assessments or standardized patientreported instruments (EQ-5D-5L, SDS). Clinician-rated severity of TD may not always correlate with patient perceptions of the significance of TD. Patient selfassessments (focused on symptom impact) can be clinically relevant; incorporating such measures into everyday practice may provide a more comprehensive approach to TD assessment and management.

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106 "Random Twitching" - A Case Presentation

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ABSTRACT: Purpose of the study: To present a case about a 12 year old with a movement disorder to educate the community about an uncommon side effect of a commonly utilized class of psychiatric medications. Simple statement of methods: Patient was seen in the context of a consultation-liaison psychiatry capacity during the first author's general psychiatry residency. Information was obtained from an electronic medical record and interviews with other physicians that treated the patient. Research about the patient's supposed diagnosis was conducted using a PubMed + OneSearch searches and articles were obtained under the guidance of a certified hospital librarian.

RESULTS/DISCUSSION: Withdrawal Emergent Dyskinesia is an uncommon, but debilitating condition that can occur after a rapid discontinuation/dosage change of a neuroleptic. This condition has been documented sparsely in the literature; more literature exists regarding its presence in children than in adults. The condition lasts for 2-3 months and resolves spontaneously in ~90% of cases. The literature that is available suggests (1) avoiding neuroleptic use in children if possible, (2) tapering off antipsychotics slowly, (3) using benzodiazepines and/or beta-blockers to treat symptoms of this condition, and (4) restarting the neuroleptic if symptoms do not improve. **CONCLUSION:** Withdrawal Emergent Dyskinesia is an uncommon, poorly studied, debilitating condition that can occur after a rapid discontinuation/dosage change of a neuroleptic. Future research efforts could be focused on (a) the prevalence of neuroleptic withdrawal symptoms in both adults and children, (b) the complete neurochemical and neurobiological pathogenesis of WED, and (c) the differences in terms of diagnosis and treatment between dyskinesias associated with both neuroleptic use and/or withdrawal. In addition, the existence of such a condition is yet another reason to reconsider off-label use of neuroleptics to treat behavioral symptoms in the absence of clear psychiatric indications for their use.

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Examining Real World Treatment Pathways in Parkinson Disease Psychosis: Initial Findings from the INSYTE Observational Study

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ABSTRACT: Study Objectives: The INSYTE study provides an understanding of the management of Parkinson disease psychosis (PDP) in actual practice settings, including use of antipsychotic (APs) and their impact on clinical, economic, and humanistic outcomes. Treatment paradigms or the benefits/consequences of various "real world" PDP treatment strategies have not been evaluated. Thus, providers may be using a wide range of AP treatment strategies that contrast with consensus recommendations.

METHOD: The INSYTE study is enrolling up to 750 patients from up to 100 sites in the US. Data are compiled at the baseline (BL) visit and from standard-of-care follow up visits over 3 years. PDP treatment pathways are defined from 3 BL cohorts reflecting (1) no AP medication, (2) use of pimavanserin (PIM), or (3) other AP treatment. Information about APs used is collected at each follow-up visit: history, duration, dose, adjustment, and rationale for adjustment of treatment. Outcomes assessments (clinical, quality of life, disease burden) by the physician, patient, and caregiver are also collected. AP medication and outcomes data are analyzed for patients completing a BL and 1 follow up visit (FU1).

RESULTS: For 404 patients with BL and FU1 visits (mean 120.7 days from BL), 56.8% used no AP medications, 26.0% used PIM, and 13.6% used other APs at BL. The No Medication group was noted to be less severe in key BL disease parameters. Considering primary PDP treatments at BL and FU1 (including no treatment), 26 - distinct pathways were being employed. 12.6% of patients had AP medication adjustments between BL and FU1 visits, most frequently from the non-PIM group. Adjustments of APs occurred in many forms: introduction of a single AP (64.7%), introduction of multiple APs (5.9%), switching to another AP (3.9%), decreasing the number of APs (5.9%), and discontinuation (19.6%).

CONCLUSIONS: Multiple, divergent AP treatment strategies for PDP exist in actual practice. No identifiable BL characteristics correlated with the broad range of AP treatment pathways. The numerous distinct AP treatment pathways utilized (n=26) reflect discordance with the updated 2019 MDS evidence-based recommendations, which recognize only 2 APs as "efficacious" and "clinically useful": pimavanserin and clozapine. Education of healthcare professionals remains a priority for PDP management.

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108 Warning: Generic Suboxone Not Equal to Name Brand

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ABSTRACT: Introduction: On June 14, 2018, the FDA approved generic buprenorphine/naloxone, as an alternative to the brand Suboxone (FDA,2018). A patient who developed acute withdrawal symptoms when switched from Suboxone to generic buprenorphine/naloxone at the same dosage, with resolution with replacement with brand name Suboxone, is presented. Induction of withdrawal with generic buprenorphine/naloxone has not heretofore been described.

METHODS: Case Study: A 39-year-old right handed single male presented with a past medical history of intravenous heroin dependence. He was relapse free for 5 years and without change on Suboxone film 8mg/2mg twice daily, and was provided with prescriptions for the same, which was substituted to generic brand Dr. Reddy's Lab SA buprenorphine HCl/naloxone HCl 8mg/2mg film. After two days on this, one hour after taking generic buprenorphine/naloxone film, symptoms of withdrawal began as manifest by hot flashes, diaphoresis, cold chills, leg cramping, and nausea without vomiting. These were the same symptoms he experienced during his past inpatient withdrawal from opioids. These symptoms recurred every day for an entire week until switching back to brand name Suboxone, whereupon his withdrawal symptoms resolved.

DISCUSSION: The mechanism whereby the generic buprenorphine/naloxone combination induced withdrawal symptoms is unclear. It appears that this generic version was either not effectively blocking the mu receptors or the naloxone was inducing a withdrawal state. Possibly the porous nature of the film was such that less of the buprenorphine was absorbed through the mucosa. As a result, less was transferred into the bloodstream, across the blood brain barrier, to the nucleus accumbens, and ultimately on kappa opioid/mu receptor (Centerwatch, 2002). Alternatively, a greater amount of naloxone may have been absorbed transmucosally, thus inducing withdrawal. The absorption may have been normal, but the exact milligram dosage may not be accurate with either too little buprenorphine or too much naloxone. On the other hand, this buprenorphine compound may have been pH sensitive, such that it became inactivated upon exposure to the mildly acidic salivary pH. He could have been malingering this response. Again this is unlikely since he was not given a higher dose of buprenorphine/ naloxone, rather the same dose of Suboxone as previously prescribed. It is important that physicians be aware of the possibility for acute withdrawal and increased cravings, which can lead to relapse while using this agent. Further investigation of the efficacy of the generic variant and Suboxone as replacement therapy is warranted.

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Hyperthyroidism-induced Psychosis

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OBJECTIVE: To describe the presence of psychotic symptomatology in a patient with hyperthyroidism