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.
Funding Acknowledgements: ACADIA Pharmaceuticals Inc.

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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

https://doi.org/10.1017/S1092852920000279 Published online by Cambridge University Press

ABSTRACT: Psychiatric-spectrum symptoms associated with thyrotoxicosis has been well reported in the past. However, psychosis in a patient with a thyroid nodule is a rare find. Here, the case of twenty four-year-old, single, unemployed, Albanian American male with self-reported history of Attention Deficit Disorder (ADD) and Cannabis use disorder was brought in to the Comprehensive Psychiatric Emergency Program (CPEP) due to new onset psychosis. Patient was paranoid, disorganized with labile mood. He had poor insight, judgement and impulse control. The event coincided with a period of unemployment in his life and new onset of hallucinations from past few days. He was brought in to the hospital after he was found pacing and having lack of sleep. Patient was treated with antipsychotic for acute psychosis. He was started on Risperidone initially to which he did not respond to. Blood work showed low TSH and elevated T4. Physical exam was noted for palpable thyroid nodule. Further labs resulted in high thyroid peroxidase antibody. Ultrasound of thyroid with color flow showed single nodule in the left lobe and iodine uptake activity localized to the left lobe. Patient was started on Methimazole 5 mg along with Haldol 5mg orally twice a day which improved his symptoms tremendously. Patient was stabilized and after 1 week was discharged on Haldol 5 mg by mouth two times a day for Psychosis, Cogentin 1 mg by mouth two times a day for extrapyramidal system (EPS), and Methimazole 5 mg by mouth daily for overactive thyroid nodule.

CONCLUSION: Psychosis associated with thyroid nodule is rare but possible. The onset of psychotic syndrome is an important clinical element whose underlying medical cause must be promptly clarified. Psychosis can present in a number of ways and can have different causes. Apart from psychiatric causes, underlying medical causes should always be considered. In this case it was important to get a full clinical history of the patient as well as complete physical examination. The differential diagnosis of a psychotic disorder in light of a medical disease should always be considered in order to promptly diagnose and treat the underlying cause to reduce the morbidity and possibly the mortality associated with it.

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Implementation of Personalized Medicine in a Community Psychiatry Practice

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OBJECTIVE: To describe the initial results of implementing pharmacogenomics testing in a community-based psychiatry practice and potential impacts on medication management.

METHOD: Retrospective chart review of prospectively maintained medical records of all adult patients with pharmacogenomics results from 9/01/2017 to 6/30/2019 under the care of psychiatrist and clinical pharmacist.

RESULTS: A total of 51 patients met inclusion criteria. A total of 7 pharmacokinetic genes and, due to changes in the test report over time, a range of 6-10 pharmacodynamic genes relevant to psychotropic medications were evaluated per patient. Every patient had genetic variations, with an average of 6.1 per patient (range 3-9; SD= 1.5). Patients were taking an average of 3.6 (range 1-8; SD=1.7) psychiatric medications at the time of the genetic test, to treat an average of 5 psychiatric conditions (range 1-9; SD=2.2). An average of 1.2 (range 0-4; SD=1.0) gene-drug interactions were uncovered per patient. Following review by psychiatrist and pharmacist, medication adjustments resulted in patients remaining on an average of 3.6 psychiatric medications, but decreasing the average number of gene-drug interactions per patient to 0.8 (range 0-3, SD=0.8).

DISCUSSION: The large number of genetic variations observed per patient is consistent with previous findings 1-2. The decrease in number of gene-drug interactions following testing demonstrates the practical utility of pharmacogenomics information to guide medication therapy. This study did not examine outcomes such as improvement in psychiatric condition or reduction in medication adverse effects; however, these endpoints have been evaluated in other trials 3-4.

CONCLUSIONS: Pharmacogenomics testing presents an opportunity for a personalized medicine approach in a community-based psychiatry practice.

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https://doi.org/10.1017/S1092852920000279 Published online by Cambridge University Press