



## APPA 2020 Fall Meeting Resident Poster Presentation

### Abstract 20-2-01

**Title:** Nausea in an antidepressant with 5HT-3 Antagonism

**Authors:** Ryan Salazar, MS3; Janaki Nimmagadda, MD

**Introduction:** About 17.3 million adults in the US have suffered from a major depressive episode in 2017 (1). The main stays to treat depression are with SSRIs and SNRIs which are used to increase the amount of serotonin within the synaptic cleft. GI disturbances are commonly seen in patients on SSRIs and according to a study: 17% to 26% of patients had experienced nausea or stomach upset while on SSRIs (8). Nausea and vomiting has been known to cause treatment discontinuation of SSRIs (2). The highest rate of GI side effects has been associated with the SSRI fluvoxamine, however, newer antidepressants such as Vortioxetine have also been frequently reported with GI side effects like nausea (2). Most side effects of SSRIs occur due to the extensive number of serotonin receptors in the body, especially in the gut. Typically, many side effects of SSRIs resolve within a few days to weeks.

**Case Presentation:** Patient is a 44-year-old Caucasian female with a history of hypertension, obesity, and migraines. Psychiatric history includes depression which was diagnosed when she was in college. Her symptoms of depression include apathy, hypersomnia, feelings of guilt, self-isolation when depressed and endorses passive suicidal ideation without plan. She has tried multiple different anti-depressants, but mostly stayed on Escitalopram (Lexapro) and Citalopram (Celexa) for the past 20-25 years due to fewer side effects compared to other medications. A few months ago, she was switched to the newer anti-depressant Vortioxetine (Trintellix)10mg. She noted decreased depressive symptoms with the medication, however she had severe nausea and vomiting for which she had to start taking Ondansetron (Zofran). Afterwards, she noted no problems and felt that her mood was amazing with the new regimen. Five months after starting Vortioxetine, she had to be hospitalized for some surgical complications and during her hospital stay, she was given Vortioxetine without Ondansetron and it resulted in her feeling nauseous again. After receiving Ondansetron, the nausea resolved.

**Discussion:** The mechanism of action of Vortioxetine includes serotonin transporter (SERT) blockade, agonism of 5HT-1A and 5HT-1B receptors, and antagonism of 5HT-1D,

5HT-7 and 5HT-3 receptors all of which increase the amount of serotonin within the synaptic cleft. 5-HT<sub>3</sub> antagonists like Ondansetron are known to be potent anti-emetics. However, blockade of 5-HT<sub>3</sub> receptors on interneurons in the brain can increase serotonin, dopamine, norepinephrine, acetylcholine, and histamine (5). Vortioxetine is known to cause downstream effects on these neurotransmitters, as well as glutamate and GABA. 5-HT<sub>3</sub> antagonists decrease afferent visceral and chemoreceptor trigger zone stimulation of the medullary vomiting center by decreasing the action of serotonin (6). Other known treatments for nausea include antihistamines, anticholinergics, and dopamine antagonists (6). Nausea from Vortioxetine could be due to its effect on interneurons in the brain or from downstream changes in other neurotransmitters associated with nausea and vomiting. These mechanisms may overshadow any hypothetical anti-nausea effect from antagonism of 5HT-3.

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## **Abstract 20-2-02**

**Title:** Mental Health and Sarcoidosis

**Authors:** Amer Babi, MS4; Jennifer Lamar, MS4; Kevin Narang, MS4; Janaki Nimmagadda, MD

### **Learning Objectives:**

1. Characterize Sarcoidosis as a disease
2. Understand the impact Sarcoidosis has on mental health
3. Consider the rationale and pertinence of screening for Sarcoidosis in select patients

### **Case Presentation:**

Patient is a 15-year-old Caucasian female with history significant for anemia presented to clinic for possible anxiety and depression. The patient states that she has been feeling "off" over the past year and that it has been gradually worsening. She attributes these mood changes to feeling like she has not adjusted to starting high school as well as her peers. She complains of feeling low on energy, losing interest in activities she was previously passionate about, changes in sleep and appetite and frequently feeling like a burden on her family. She also complains of new onset episodes of feeling excessively anxious around 3 times per week but denies symptoms related to panic attacks. The patient has a maternal family history of sarcoidosis, anxiety, and depression. Two of her family members have officially been diagnosed with sarcoidosis, her maternal great grandmother in her early 60s as well as her maternal aunt in her 40s. Family reports that few other family members may also have sarcoidosis but have not been officially diagnosed. The family members who have been diagnosed with depression and anxiety are exclusively female, reportedly have symptom onset at a younger age, and have struggled with it throughout their lives. The family members who have been diagnosed with sarcoidosis also reportedly have more severe cases of anxiety and depression in comparison to other family members who have not displayed any sarcoid related symptoms.

### **Discussion:**

Sarcoidosis is an inflammatory and immunological disease characterized by non-caseating granulomas with multiple organ system involvement. There is thought to be a genetic component to sarcoidosis as higher rates of the disease are seen in families especially with mother-child relationships. However, it is challenging to diagnose due to vague complaints such as fatigue. Furthermore, it is more mystifying to know when the disease actually manifests. Can one be asymptomatic for years without any overt somatic complaints? Some studies report up to 65% prevalence of anxiety/depression in patients with asymptomatic or symptomatic sarcoidosis. While the cause is not known,

those with the inflammatory condition had significant psychosocial stress prior their diagnosis. This is a reasonable observation considering the interplay between the immune system and stress is well documented. Is it possible for anxiety/depression to be present in a large population of Sarcoidosis patients prior to their diagnosis? With vague complaints, patients suffer on average of 5 years before being diagnosed. Is it valid to screen patients presenting with anxiety or depression who also have a strong family history for Sarcoidosis? We need more research in this area to guide clinical practice.

## **Abstract 20-2-03**

**Title:** A Case of Clozapine-Induced Hypothermia

**Authors:** Samantha Lee, MS4; Mazen Omar, MD; J. Luke Engeriser, MD

### **Summary:**

The adverse drug reaction of hyperthermia from neuromalignant syndrome is more well-known and more commonly investigated than hypothermia. We present a case of a 54-year-old African American female with past medical history of type 2 diabetes, hypertension, schizophrenia, and seizure disorder admitted to a psychiatric hospital who developed generalized weakness, garbled speech, urinary incontinence, and altered mental status. She was transferred to an emergency department as a stroke code and was admitted for further work-up. For the patient's acute encephalopathy, a head CT scan showed no acute intracranial pathology ruling out a stroke, the blood cultures were negative ruling out an infection, and the patient neurologically returned to baseline within a day ruling out an ongoing neurological cause of encephalopathy. The patient was hypothermic at 27.1 C (80.78 F) on admission but otherwise hemodynamically stable. Medical work-up for hypothermia resulted in negative blood cultures ruling out infection and TSH/T4 levels within normal limits ruling out hypothermia or myxedema coma. Her temperature was stabilized with a Bair hugger. The patient was already on clozapine 250 mg before admission to the inpatient psychiatric facility and was known to be on this current regimen for longer than 3 months. She was also currently taking topiramate 100 mg at bedtime for her seizure disorder. She had had recent medication changes a week prior to the onset of these symptoms with discontinuation of benzotropine and initiation of clonazepam 1 mg twice daily. Due to continued somnolence and dysarthric speech, her clonazepam was decreased to 0.5 mg twice daily. In suspicion of clozapine-induced hypothermia, her clozapine was discontinued and quetiapine 50 mg at bedtime was initiated to treat her chronic schizophrenia. Her temperature stabilized within a few days.

### **Discussion:**

This case highlights hypothermia as a possible adverse reaction to clozapine and illustrates the importance of monitoring the body temperature of patients, especially when initiating treatment or increasing the dosage. Common medical causes of hypothermia include hypoglycemia, sepsis, hypothyroidism, and stroke, and these were all ruled out. This patient was calculated to have a score of 4 on Naranjo Scale for the probability of an adverse drug reaction.

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## Abstract 20-2-04

**Title:** Persistent Genital Arousal Disorder

**Authors:** Cayla Van Alstine, MS2; Maridith Hollis, MD; Darshana Pai, MD; Serena Nimityongskul, MD; William Brooks, MD

**Summary:** A 47yo woman presents to an outpatient clinic complaining of unwanted, distressing genital hyperarousal sensations (sometimes reaching orgasm and sometimes not) with non-intimate physical stimulation. It appears the patient has persistent genital arousal disorder (PGAD), a rather uncommon disorder first described in the early 2000s. Through this unique case study, the authors seek to further elucidate the multiple etiologies of PGAD and standards of treatment.

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## Abstract 20-2-05

**Title:** Gender Dysphoria in Adults with Autism Spectrum Disorder

**Authors:** Dominique Moreno, DO; Logan Rogers, DO; Lucas Boone, MD; Lori Lowthert, MD

**Summary:** The patient that will be discussed is a 51-year-old, assigned female at birth (AFAB). During their inpatient hospitalization within our facility, they were diagnosed with Autism Spectrum Disorder (ASD); this diagnosis was confirmed with extensive Psychological Testing. ASD may present in many ways, depending on the individual. This developmental disorder can impact how one perceives and interacts with others. This may lead to difficulty socializing and communicating with their peers.

Of importance, the patient's preferred pronouns are they/theirs. They expressed gender dysphoria, specifically claiming to be non-binary. Per the American Psychiatric Association, gender dysphoria is the mismatch between an individual's assigned physical gender, and the gender with which they/she/he identifies. To identify as non-binary, means that one's gender identity is neither exclusively male or female- it may be between or beyond.

The combination of this patient's gender dysphoria and Autism Spectrum Disorder has led to multiple misclassifications of patient's true diagnosis, over the 40 plus year history of their treatment. We will explore the increasing association of gender dysphoria in the setting of Autism spectrum disorder and express the need for further literature/studies to be performed.

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## Abstract 20-2-06

**Title:** Olanzapine Induced Hair Loss – A Case Series

**Authors:** Darshana S. Pai, MD; Ashish Srivastava, MD; J. Luke Engeriser, MD

**Summary:** Several psychotropic medications have been implicated in hair loss—most commonly, valproic acid and lithium. The reported dermatological side effects associated with antipsychotic agents are rash, pruritus, photosensitivity, skin pigmentation, fixed drug eruptions, and alopecia (1,2,3). However, the number of studies which report atypical antipsychotic induced alopecia is quite limited to a few case reports. Olanzapine is an atypical antipsychotic and has been widely used in the treatment of psychosis and mood disorders. Although it is generally well tolerated, it has been associated with the adverse effect of metabolic syndrome. We present a case series in which several adult female patients reported hair loss following administration of olanzapine which resolved after cessation or decrease of dosage of the drug.

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## Abstract 20-2-07

**Title:** Neurosarcoidosis and Psychosis, a Case Study

**Authors:** Michael L. Marshall, MD; J. Luke Engeriser, MD

**Summary:** Background: Sarcoidosis is a systemic disease characterized by noncaseating granulomatous inflammation with an estimated incidence of 35.5 per 100,000 person-years in African Americans and 10.9 per 100,000 person-years in Americans of European descent. Nervous system involvement occurs in 5-16% of cases and common presenting signs often involve the optic nerve, other cranial nerves, the spinal cord, or brainstem. However, less common presenting signs include involvement of the meninges, cognitive decline, hydrocephalus, and hypothalamic-pituitary-adrenal axis dysfunction. Delirium and psychosis are estimated to manifest in 1% of individuals with sarcoidosis.

**Case:** We describe a 35-year-old African-American male with a history of obstructive hydrocephalus, seizure disorder, obstructive sleep apnea, and morbid obesity who presented with a two month history of intermittent paranoia and visual hallucinations progressing in intensity, as well as drowsiness. His psychotic symptoms were reported to onset shortly after a shunt revision procedure, though there was no evidence of shunt malfunction or seizure activity on evaluation. The patient exhibited signs of hypercapnic respiratory failure and subsequent CT of the thorax revealed significant mediastinal and hilar adenopathy. Mediastinal lymph node biopsy showed granulomatous processes indicative of sarcoidosis, and high dose intravenous corticosteroids were administered with gradual improvement of psychosis and agitation.

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