

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-01

Title: Barriers to rapidly effective therapies for major depression in the inpatient setting

Presenting Author: Nadia Anabtawi, MS-4, UAB Heersink School of Medicine

Additional Authors: Matthew Macaluso, DO; Matthew Ehrlich, MD: Department of Psychiatry and Behavioral Neurobiology, UAB Heersink School of Medicine

Introduction/Background: In major depression, earlier symptomatic response to treatment predicts a higher probability of remission (1). Electroconvulsive therapy (ECT) shows rapid response for treatment-resistant depression (TRD) with well-established safety, but it is important to minimize cognitive impairment and hemodynamic disturbances (2). In some cases, alternatives like intranasal esketamine (Spravato) or IV ketamine could be preferred (3). Esketamine has shown significantly reduced depressive symptoms at 24 hours (4), but there are obstacles to providing esketamine inpatient. Health systems must be enrolled in the controlled redistribution Spravato-REMS program (5). Spravato is obtained through the buy-and-bill model, which requires a costly upfront investment by the hospital (6). If obtained through insurance and a specialty pharmacy, approval can take weeks. Moreover, while there is an established institutional protocol for inpatient IV ketamine use in intractable pain, IV ketamine use in depression at our institution is only approved in outpatient clinics via a cash-only model. These barriers to inpatient access to esketamine and ketamine precluded their use in our patient.

Case Description: This is 92-year-old male with COPD and HFpEF who was admitted to the geriatric psychiatry unit with depression and catatonia. Scheduled lorazepam improved catatonic symptoms which included immobility, mutism, and negativism. However, he continued to display worsening hopelessness, apathy, and suicidal ideation with no improvement from escitalopram, mirtazapine, and methylphenidate. He began refusing food with the intent to hasten his death, necessitating a rapid-acting treatment. Esketamine and ketamine were not available inpatient, so ECT was initiated.

Discussion and Conclusion: Esketamine administration requires patient monitoring for two hours after dosing (7)--a requirement easily achievable inpatient. However, in Alabama, there are seven outpatient treatment centers certified in Spravato-REMS but no certified inpatient hospitals (8). Further compounding the barriers to inpatient esketamine access is the suboptimal system for billing, coding, and drug acquisition that needs to be further explored. Additionally, the protocol for IV ketamine in intractable pain, typically managed by anesthesiology and inpatient pain services, does not readily translate to psychiatric use, so development of a dedicated inpatient psychiatric ketamine infusion team should be considered (9). Availability of inpatient esketamine or ketamine would offer patients rapidly effective alternatives to ECT.

References:

1. Martínez-Amorós, E., Goldberg, X., Gálvez, V., de Arriba-Arnau, A., Soria, V., Menchón, J. M., Palao, D. J., Urretavizcaya, M., & Cardoner, N. (2018). Early improvement as a predictor of final remission in major depressive disorder: New insights in electroconvulsive therapy. *Journal of affective disorders*, 235, 169–175. <https://doi.org/10.1016/j.jad.2018.03.014>
2. Hermida, Adriana P. MD*; Mohsin, Mamoon MD†; Marques Pinheiro, Ana P. MD*; McCord, Elizabeth MD*; Lisko, John C. MD‡; Head, Lyndsay W. MD§. The Cardiovascular Side Effects of Electroconvulsive Therapy and Their Management. *The Journal of ECT* 38(1):p 2-9, March 2022. | DOI: 10.1097/YCT.0000000000000802

3. Phillips, J.L., Jaworska, N., Kamler, E. et al. A randomized, crossover comparison of ketamine and electroconvulsive therapy for treatment of major depressive episodes: a Canadian biomarker integration network in depression (CAN-BIND) study protocol. *BMC Psychiatry* 20, 268 (2020). <https://doi.org/10.1186/s12888-020-02672-3>
4. Fu D-J, Ionescu DF, Li X, et al. Esketamine nasal spray for rapid reduction of major depressive disorder symptoms in patients who have active suicidal ideation with intent: double-blind, randomized study (ASPIRE I). *J Clin Psychiatry*. 2020;81(3):19m13191.
5. Access and Reimbursement Guide: Information to Support the Access and Reimbursement Process for SPRAVATO. Retrieved from https://www.spravatohcp.com/sites/www.spravatohcp.com/files/spravato_access_reimbursement_guideline.pdf?v=4
6. DeMarzo, Amanda. “What Is Buy and Bill?” Prior Authorization Training, 22 Mar. 2021, <https://www.priorauthtraining.org/what-is-buy-and-bill/>.
7. Bahr R, Lopez A, Rey JA. Intranasal Esketamine (Spravato™) for Use in Treatment-Resistant Depression In Conjunction With an Oral Antidepressant. *P T*. 2019 Jun;44(6):340-375. PMID: 31160868; PMCID: PMC6534172.
8. Spravato Find a Center. Retrieved from https://www.spravatohcp.com/find-a-center?zipcode=35205&by_distance=all
9. Ithman M, Sobule R, Kundert C, Campbell A, Ehrhardt E. Ketamine Infusions Administered Solely by Psychiatric Staff. *Mo Med*. 2022 Mar-Apr;119(2):164-166. PMID: 36036039; PMCID: PMC9339397.

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-02

Title: Locked Units and Lockup: Delusional Disorder and the Justice System

Presenting Author: Kiley Brady, MS-3, USA College of Medicine

Additional Author: Evan Chavers, MD, Department of Psychiatry, USA College of Medicine

Introduction/Background: Delusional Disorder (DD) is defined by the presence of one or more delusions for at least one month in duration where functional ability is not impaired, and behavior is not odd outside the direct effects of the delusions. There are many different types of delusions and DD can be further specified by type. The most common type is persecutory, but others include grandiose, erotomaniac, jealous, somatic, mixed or unspecified. The high incidence of persecutory delusions in this patient population can lead to difficulties with law enforcement. It has even been found that there is a higher prevalence of individuals with DD in prison systems than in the standard population. Therefore, it is imperative to attempt to help patients gain insight into their illness and limit the bizarrry of their delusions in order to help mitigate future unlawful or negative legal interactions.

Case Description: A 47-year-old white female was petitioned for inpatient hospitalization after the police were contacted by her neighbor after patient left her 10-year-old daughter at home for an extended period of time. EMS arrived at her home where she found to be making bizarre and paranoid statements. She was convinced to go the local ED and transferred to an inpatient facility after 7 days. Patient has a psychiatric history of OUD on methadone, depression, self-reported bipolar disorder, and one prior psychiatric hospitalization where she was diagnosed with psychosis, unspecified. Upon interview she endorsed bizarre paranoid delusions revolving around her neighbor. Collateral information from her partner revealed that she began having delusions for the past 3 years. These delusions became less bizarre following multiple antipsychotic trials, but they persisted over the course of her stay.

Discussion and Conclusion: Individuals with DD have a potentially higher likelihood of negative interactions with the legal system in particular those with paranoid delusions. Paranoid delusion can be associated with violent criminal conduct due to a sense of impending danger; individuals might also report the target of their delusions in an attempt to have an outside agency validate their reality. It has also been shown that police are more inclined to arrest an individual showing bizarre behavior. Therefore, it is important to explore treatment plans that limit the bizarre nature of delusions and special attention be paid to intention to act on delusional thoughts, particularly violent ones.

References:

1. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 5th ed. Washington D.C.: 2013.
2. Bartol CR, Bartol AM. Criminal Behavior: A Psychosocial Approach. 7th ed. Upper Saddle River, NJ: Pearson Prentice Hall; 2005.
3. Bezuidenhout C, Collins CR. Implications of Delusional Disorders and Criminal Behavior for Criminology. *Acta Criminologica*. 2007;20(3).
4. Joseph SM, Siddiqui W. Delusional Disorder. In: StatPearls. Treasure Island (FL): StatPearls Publishing; July 20, 2022.
5. Tamburello A, Bajgier J, Reeves R. The prevalence of delusional disorder in prison. *J Am Acad Psychiatry Law*. 2015;43(1):82-86.

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-03

Title: Methamphetamine Induced Catatonia: Considerations in the ED Setting

Presenting Author: Taran Carasco, MS-3, USA College of Medicine

Additional Authors: Jonathan Lee, MS-3; Evan Chavers, MD: USA College of Medicine

Introduction/Background: Catatonia is a psychomotor syndrome that can present with abnormal motor and behavioral signs that can range from echolalia to mutism and agitation to stupor. While catatonic features are classified in the DSM-5 as a subtype of schizophrenia and specifier for mood disorders, features can be secondary to infectious, immunologic, neurologic, metabolic, and substance-induced etiologies. The majority of reported substance-induced catatonic episodes have been secondary to cannabis, cocaine, and opioid use or alcohol and benzodiazepine withdrawal. Very few cases of methamphetamine-induced catatonia have been published. This case emphasizes the importance of considering, and screening for, methamphetamines in patients who present with catatonic features.

Case Description: A 30-year-old man with a past psychiatric history of Cannabis Use Disorder was brought to the emergency department by his mother for a five day history of insomnia and one day history of mutism and decreased oral intake. His mother described him as intelligent, hardworking, and high-functioning at baseline. She suggested that he used “some bad drugs” prior to the change in his behavior. On exam, he was vitally stable but appeared stuporous. He did not follow commands or speak. His affect was labile and appeared anxious with intermittent tearfulness. He was hypervigilant and scanned the room constantly with his eyes. He appeared to be responding to internal stimuli. He exhibited marked psychomotor agitation and nearly continuous purposeless movements. Bush-Francis Catatonia Rating Scale was 13 at that time. His labs were remarkable for amphetamines and cannabinoids on his urine drug screen and an elevated creatinine kinase and ammonia. All imaging was unremarkable.

Discussion and Conclusion: Catatonia is a syndrome associated with abnormal movements and reactions to the environment with subtypes including retarded catatonia, excited catatonia, and malignant catatonia. Due to the broad symptoms and severity of catatonia, many cases go unrecognized and are difficult to differentiate from delirium, encephalopathy, and postictal states. This case illustrates the importance of thorough history taking and use of investigations to complete assessment and formulate a management plan. It is important to recognize that while catatonia is traditionally associated with psychiatric disorders, medical and substance-induced causes should always be assessed for.

References:

1. Bestha DP, Padala P. A case of catatonia secondary to polysubstance abuse and acetaminophen overdose. *Prim Care Companion J Clin Psychiatry*. 2010;12(1):PCC.09100790. doi: 10.4088/PCC.09100790ecr. PMID: 20582301; PMCID: PMC2882818.
2. Bush, G., Fink, M., Petrides, G., Dowling, F., & Francis, A. (1996). Catatonia. I. Rating scale and standardized examination. *Acta psychiatrica Scandinavica*, 93(2), 129–136. <https://doi.org/10.1111/j.1600-0447.1996.tb09814.x>
3. Jiang, S., & Sullivan, G. (2022). Methamphetamine-Induced Catatonia. The primary care companion for CNS disorders, 24(5), 21cr03211. <https://doi.org/10.4088/PCC.21cr03211>
4. Walther, S., Stegmayer, K., Wilson, J. E., & Heckers, S. (2019). Structure and neural mechanisms of catatonia. *The Lancet Psychiatry*, 6(7), 610–619. [https://doi.org/10.1016/S2215-0366\(18\)30474-7](https://doi.org/10.1016/S2215-0366(18)30474-7)

5. Yeoh, S. Y., Roberts, E., Scott, F., Nicholson, T. R., David, A. S., & Rogers, J. P. (2022). Catatonic Episodes Related to Substance Use: A Cross-Sectional Study Using Electronic Healthcare Records. *Journal of dual diagnosis*, 18(1), 52–58.
<https://doi.org/10.1080/15504263.2021.2016342>

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract: 22-1-04

Title: Depakote Augmentation in the Treatment of Catatonia

Presenting Author: Amanda Davis, PGY-1, USA College of Medicine, Department of Psychiatry

Additional Authors: Shane Stephenson, MS-3, USA College of Medicine; Miranda Crowell, MD; Praveen Narahari, MD: AltaPointe Health Systems and USA College of Medicine, Department of Psychiatry

Introduction/Background: Catatonia, first described by Dr. Karl Kahlbaum in the 1800s, is a syndrome of various psychomotor and behavioral signs associated with psychiatric, neurological, and medical disorders.[1] Up to 15% of patients admitted to an acute psychiatric service meet diagnostic criteria, but patients with mood disorders are most commonly affected.[2,3] Benzodiazepines and electroconvulsive therapy (ECT) are first-line treatment options and often dramatically improve symptoms.[4,5] However, the response to benzodiazepines varies among patient subgroups, and ECT is not always accessible.[2] As a result, alternative treatment strategies have been investigated, such as atypical antipsychotics and anti-epileptic drugs.[6]

Case Description: The patient is a 62-year-old female with a history of Bipolar I Disorder with psychotic features who presented due to mania and psychosis. She was previously stable on Fluphenazine (Prolixin) for 18 years. Multiple medications were added to her regimen at a local facility before she arrived at the inpatient psychiatric unit. Her mania and psychosis improved with titration of Prolixin, but she began to display signs of catatonia. She responded positively to a Lorazepam (Ativan) challenge test, but her catatonia minimally improved with Ativan monotherapy at various doses. To further target catatonia, Depakote was added and titrated. Approximately two weeks after initiating Depakote, her symptoms significantly improved.

Discussion and Conclusion: This case report highlights using Depakote as an alternative treatment strategy for catatonia in a patient with a mixed mood and psychotic disorder. ECT was not accessible, and minimal symptom improvement occurred with Ativan monotherapy. Labs and imaging were unremarkable for medical causes of catatonia. Her symptoms of catatonia resolved with treatment and did not regress during the taper of Ativan. The initiation of Aripiprazole may have also played a role in symptom improvement.

References:

1. Francis A. Catatonia: diagnosis, classification, and treatment. *Curr Psychiatry Rep.* 2010;12(3):180-185. doi:10.1007/s11920-010-0113-y
2. Rosebush PI, Mazurek MF. Catatonia and its treatment. *Schizophr Bull.* 2010;36(2):239-242. doi:10.1093/schbul/sbp141
3. Taylor MA, Fink M. Catatonia in psychiatric classification: a home of its own. *Am J Psychiatry.* 2003;160(7):1233-1241. doi:10.1176/appi.ajp.160.7.1233
4. Fricchione GL, Cassem NH, Hooberman D, Hobson D. Intravenous lorazepam in neuroleptic-induced catatonia. *J Clin Psychopharmacol.* 1983;3(6):338-342.
5. Beach S, Francis A, Fricchione G. Treatment algorithm for catatonia. *Psychiatric Times.* 2019. Accessed February 13, 2023. <https://www.psychiatristimes.com/view/neuropsychiatry-catatonia-clinical-implications>.
6. Beach SR, Gomez-Bernal F, Huffman JC, Fricchione GL. Alternative treatment strategies for catatonia: A systematic review. *Gen Hosp Psychiatry.* 2017;48:1-19. doi:10.1016/j.genhosppsy.2017.06.011

7. Vijayvargiya R. Subtypes of Catatonia. Psychiatric Times. 2023. Accessed February 13, 2023. <https://www.psychiatrictimes.com/view/the-many-faces-of-catatonia-an-under-recognized-clinical-syndrome>

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-05

Title: Valbenazine for Treating Tardive Dyskinesia

Presenting Author: Kenneth Davis, MS-3, UAB Heersink School of Medicine

Additional Authors: Desalyn Johnson; Anupama Yedla, MD; Clinton Martin MD: UAB Heersink School of Medicine

Introduction/Background: Tardive Dyskinesia (TD) is a condition defined by involuntary movements of the face, lips, trunk, tongue and extremities that may vary in severity, from a slight tremor to uncontrollable movement of the whole body. TD may occur from long-term use of antipsychotic drugs (APDs) such as dopaminergic antagonist medications. Approximately 20%-50% of patients taking APDs develop TD. There is currently no cure for TD, but numerous treatment options are available such as clozapine, propranolol, clonazepam, and most notably Valbenazine.

Description: 69-year-old woman with Bipolar I disorder that developed TD after long-term treatment with APDs. Her medications included Keppra, Seroquel, and Invega. Initially, her AIMS score was 7. She began treatment for TD with Valbenazine 80mg. After 9 weeks at follow-up, her AIMS score was 2 with her TD symptoms much improved and no notable side effects.

Discussion and Conclusion: Tardive Dyskinesia is due to upregulation of dopamine receptors secondary to medication induced blockade. The resulting increase of receptors on the presynaptic membrane leads to an exaggerated dopaminergic response and the clinical symptoms of TD. When treating patients with ADPs, medical providers should be aware of comorbid risk factors for TD present in their patient. Studies have suggested that post-menopausal women are at increased risk for TD. This is caused by a decrease in estrogen, which regulates dopamine activity. Caution should also be taken in patients with bipolar disorder and epilepsy. As certain mood stabilizer and antiepileptics have been associated with increased TD risk. Vesicular Monoamine Transporter 2 (VMAT2) is responsible for the intracellular uptake of neurotransmitters such as dopamine into vesicles to allow for synaptic release. Valbenazine functions by inhibiting VMAT2, decreasing the amount of dopamine available for release. Valbenazine has a limited adverse reaction profile and functions as a pro-drug, leading to an increased half-life. This allows for once daily dosing, thus improving patient adherence. This is in stark comparison to other VMAT2 inhibitors, such as tetrabenazine, whose mechanism of action is performed by the metabolite R,R,R-DHTBZ. Overall, Valbenazine has shown to be an effective treatment option for patients suffering with the hyperkinetic symptoms of Tardive Dyskinesia.

References:

1. Harriott, N. D., et al. (2018). Chapter Two - VMAT2 Inhibitors and the Path to Ingrezza (Valbenazine). *Progress in Medicinal Chemistry*. D. R. Witty and B. Cox, Elsevier. 57: 87-111. <https://doi.org/10.1016/bs.pmch.2017.12.002> Vasan S, Padhy RK. Tardive Dyskinesia. [Updated 2022 Aug 16]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK448207/?report=classic>
2. Cornett EM, Novitch M, Kaye AD, Kata V, Kaye AM. Medication-Induced Tardive Dyskinesia: A Review and Update. *Ochsner J*. 2017 Summer;17(2):162-174. PMID: 28638290; PMCID: PMC5472076 Robert A. Hauser, M.D., M.B.A. , et al. (2017). "KINECT 3: A Phase 3 Randomized, Double-Blind, Placebo-Controlled Trial of Valbenazine for Tardive Dyskinesia." *American Journal of Psychiatry* 174(5): 476-484. <https://ajp.psychiatryonline.org/doi/abs/10.1176/appi.ajp.2017.16091037>

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-06

Title: Catatonia: A Case Report

Presenting Author: Steven Gaffin, MS-4, UAB Heersink School of Medicine

Additional Authors: Senthil Rajaram Manoharan, MD; Clinton Martin, MD: UAB Heersink School of Medicine, Department of Psychiatry

Introduction/Background: Catatonia is described as a behavioral syndrome characterized by abnormal movements despite capacity which occurs more commonly in patients with underlying unipolar major depression or bipolar disorder [1]. The most common clinical features of catatonia are: hypokinesia, mutism, stupor, posturing, negativism, waxy flexibility, staring, and echophenomena [2,3,4].

Case Description: A 25-year-old female with past medical history of anxiety and depression, presented to the ED with her mother for increased paranoia, unusual posturing, and odd behavior for 24 hours prior to arrival. She was initially diagnosed with psychosis and started on Risperdal 2mg twice a day. On day two, patient was showing signs of verbigeration, stereotypy, withdrawal, refusal to eat, and stupor. Risperdal was discontinued and benzodiazepine was initiated. Catatonic symptoms resolved the next day and delusional and paranoid thoughts resolved on day 12. Patient was discharged on Fluoxetine, Olanzapine, and a follow-up with behavioral health.

Discussion and Conclusion: In this case presentation, symptoms of psychosis and catatonia were presenting synchronously which effectively masked catatonic features. It wasn't until the administration of Risperdal that catatonia was apparent and was then addressed appropriately. Antipsychotics are contraindicated in catatonic patients as it can exacerbate the condition [5]. In a study of 148 catatonic patients, nearly 75% were also psychotic [6]. The presence of psychosis should alert the clinician to the possibility of underlying catatonia to reduce the possibility of exacerbation.

References:

1. Taylor MA, Fink M. Catatonia in psychiatric classification: a home of its own. *Am J Psychiatry* 2003; 160:1233.
2. Fink M, Taylor MA. The catatonia syndrome: forgotten but not gone. *Arch Gen Psychiatry* 2009; 66:1173.
3. Fink M, Taylor MA. The catatonia syndrome: forgotten but not gone. *Arch Gen Psychiatry* 2009; 66:1173.
4. Francis A. Catatonia: diagnosis, classification, and treatment. *Curr Psychiatry Rep* 2010; 12:180.
5. Lee JW. Neuroleptic-induced catatonia: clinical presentation, response to benzodiazepines, and relationship to neuroleptic malignant syndrome. *J Clin Psychopharmacol* 2010; 30:3.
6. Rosebush PI, Mazurek MF. Catatonia and its treatment. *Schizophr Bull* 2010; 36:239.

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-07

Title: Delusional Ideation Resulting from Long-Term PTSD: A Case Study

Presenting Author: Sela Gavan, MS-3, USA College of Medicine

Additional Author: Maridith Hollis, MD, USA College of Medicine, Department of Psychiatry

Introduction/Background: Post-Traumatic Stress Disorder is a commonly recognized diagnosis in veterans, victims of abuse, and those affected/displaced by natural disasters. There are specific criteria by the DSM-V which must be met including exposure to death or serious harm and the individual must persistently re-experience the traumatic event through intrusive symptoms and subsequently develop avoidance behaviors and negative outlook on self or the world.¹ Some patients also experience new onset of psychotic symptoms in relation to their PTSD diagnosis but do not meet criteria for a psychotic spectrum disorder.

Case Description: A 27 year old Caucasian female presents voluntarily for inpatient psychiatric admission due to suicidal ideation and erratic behaviors with concern for harm to self or others. Her psychiatric history includes documented Adjustment Disorder with concern for PTSD, childhood abuse, and self reported ADHD. The patient endorsed prior symptoms of hypervigilance, avoidance, difficulty trusting others, and increased irritability. Of late, the patient has been under increased stress with new jobs and relocation, which have worsened preexisting symptoms to the point of paranoid ideations and ideas of reference, which have limited her occupational and interpersonal functioning. While receiving inpatient treatment, the patient's Zoloft dose was increased to 150mg QHS and the patient was started on Seroquel, which was titrated to therapeutic dose of 250mg QHS to target mood stabilization and sleep disturbances.

Discussion and Conclusion: The research worthy part of this patient's case was the escalation to delusional content. A recent Danish study of refugees seeking treatment demonstrated that up to 30% of those diagnosed with PTSD had secondary psychotic symptoms (hallucinations or persecutory delusions) with comorbid depression increasing the risk of psychotic symptoms.² Further research is needed to determine the etiology and pharmacotherapy needed to treat these symptoms most effectively, and the hypothesis of elevated dopamine beta hydroxylase in patients with PTSD and comorbid psychotic symptoms could provide evidence for the reason Seroquel, an anti-dopaminergic drug, was an effective treatment for this patient.³

References:

1. American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.). <https://doi.org/10.1176/appi.books.9780890425596>
2. Rathke H, Poulsen S, Carlsson J, Palic S. PTSD with secondary psychotic features among trauma-affected refugees: The role of torture and depression. *Psychiatry Res.* 2020 May;287:112898. doi: 10.1016/j.psychres.2020.112898. Epub 2020 Feb 24. PMID: 32179211.
3. Hamner MB, Gold PB. Plasma dopamine beta-hydroxylase activity in psychotic and non-psychotic posttraumatic stress disorder. *Psych Res* 1998;77(3):175-181.

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-08

Title: Delirium Due to Tianeptine Withdrawal: A Case Report

Presenting Author: Kevin Gallagher, MS-4, Alabama College of Osteopathic Medicine

Additional Authors: Luke Engeriser, MD, DFAPA; Frederick Brown, DO: USA College of Medicine, Department of Psychiatry

Introduction/Background: Tianeptine is an atypical antidepressant, similar in structure to tricyclics, approved for use for major depressive disorder in some European and Asian countries. It has never been approved by the Food and Drug Administration in the United States. Due to its agonism at μ -opioid receptors, withdrawal from tianeptine is manifested by typical opioid withdrawal symptoms, and opioid agonist medications have been used successfully for tianeptine withdrawal (1,3,4). It has been used both recreationally and to self-treat opioid withdrawal since around the year 2000, and poison control centers have noticed an increase in calls related to tianeptine use (1,2). Using a case example, we describe atypical delirium from a patient withdrawing from tianeptine.

Case Description: We describe the case of a 24-year-old female who presented to our psychiatric crisis center for tianeptine withdrawal. The patient had been taking approximately 9 tianeptine capsules every 5 hours. Prior to admission, the patient had tried to taper off of tianeptine but was unsuccessful due to symptoms of anxiety, nausea, vomiting, and elevated heart rate. On day 3 of withdrawal at the crisis center, the patient began experiencing waxing and waning confusion, auditory hallucinations, visual hallucinations, and agitation consistent with delirium.

Discussion and Conclusion: This case demonstrates that withdrawal from tianeptine can cause an atypical delirium along with the more common withdrawal profile of opioids. Given the increasing frequency of tianeptine use, it is important for clinicians to be aware of both the potential for opioid withdrawal syndrome upon discontinuation of tianeptine as well as the potential for withdrawal associated delirium.

References:

1. El Zahran T, Schier J, Glidden E, Kieszak S, Law R, Bottei E, Aaron C, King A, Chang A. Characteristics of Tianeptine Exposures Reported to the National Poison Data System - United States, 2000-2017. *MMWR Morb Mortal Wkly Rep.* 2018 Aug 3;67(30):815-818.
2. Rushton W, Whitworth B, Brown J, Kurz M, Rivera J. Characteristics of tianeptine effects reported to a poison control center: a growing threat to public health. *Clin Toxicol (Phila).* 2021 Feb;59(2):152-157.
3. Gassaway MM, Rives ML, Kruegel AC, et al. The atypical antidepressant and neurorestorative agent tianeptine is a μ -opioid receptor agonist. *Transl Psychiatry* 2014;4:e411.
4. Trowbridge P, Walley AY. Use of Buprenorphine-Naloxone in the Treatment of Tianeptine Use Disorder. *J Addict Med.* 2019 Jul/Aug;13(4):331-333.

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-09

Title: Psychiatric Manifestations of Porencephaly: The Holes in Our Knowledge

Presenting Author: Katrina Hatch, MS-3, UAB Heersink School of Medicine - Huntsville

Additional Author: Briana Mayberry, MS-3, UAB Heersink School of Medicine; Janaki Nimmagadda, MD, UAB Heersink School of Medicine - Huntsville, Department of Psychiatry

Introduction/Background: Porencephaly is an extremely rare disorder (3.5/100,000 births) of the central nervous system that causes a cyst or cavity filled with cerebrospinal fluid to develop in the brain antenatally or prenatally.[1] It is commonly thought to be on a continuum with schizencephaly.[2] Possible risk factors for porencephaly are: thrombophilias, maternal cocaine use, maternal abdominal trauma, vascular injury in the third trimester, pre-eclampsia, COL4A mutation.[1,2,3] Diagnosis is typically made before age one but has been an incidental finding in adults above the age of 40.[1] Children with porencephaly typically suffer from: spastic contractures or weakness, seizures/epilepsy, hydrocephalus, cognitive and developmental difficulties.

Case Description: Patient is a 20-year-old male with a past medical history of right-sided hemi-cerebral palsy from 70% porencephaly, anxiety, autism spectrum disorder, oppositional defiant disorder, and attention deficit hyperactivity disorder. He was born to a 41-year-old female with pre-eclampsia. When he was around six months old, brain MRI showed 70% of his left hemisphere consisted of a cyst without brain matter. The patient has behavioral problems mostly at home. His outbursts have escalated to physical altercations with his parents and destruction of property at home. He will use intimidation and ultimatums to manipulate situations in his favor. In school, he has an Individualized Education Program. He also experiences anxiety when his routine is changed or disrupted. His parents have noticed that he lacks impulse control and awareness to danger. This has improved as he has aged, but he will still act without thinking of the consequences.

Discussion and Conclusion: While neurological and psychiatric involvement can widely vary, it is common to see developmental delay and cognitive difficulties. Patients often suffer from poor emotional control and aggressive behaviors. No specific treatment for porencephaly is currently available, so treatment is usually aimed at symptom control: surgically removing the cysts, placing a shunt, antiepileptics, rehabilitation and physical therapies. The prognosis of porencephaly depends on location and extent of the cyst. When considering treatment modalities for these patients, psychiatry involvement has shown to improve wellness. Treatment with medication or therapy should be considered to increase overall quality of life.

References:

1. NAEF. (1958). Clinical features of porencephaly; a review of thirty-two cases. *A.M.A. Archives of Neurology & Psychiatry.*, 80(2), 133–147.
2. Monteagudo, A, M.D.; Timor-Tritsch, I E, M.D. (2022, June 15). Prenatal diagnosis of CNS anomalies other than neural tube defects and ventriculomegaly. UpToDate.
3. Neuroepithelial Cysts, Porencephaly, and Perivascular Spaces. (2016, Jan 22). Radiology Key. <https://radiologykey.com/neuroepithelial-cysts-porencephaly-and-perivascular-spaces/>
4. Bernstein, E.L. (1939) Psychiatric Aspects of Porencephaly. *American Journal of Insanity*, 96 (3) 723-731. <https://doi.org/10.1176/ajp.96.3.723>
5. Eller, K. & Kuller, J. (1995). Fetal Porencephaly. *Obstetrical & Gynecological Survey*, 50 (9), 684-687.

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract: 23-1-10

Title: Agitation with Frontotemporal Dementia

Presenting Author: Caleb Hood, MS-3, UAB Heersink School of Medicine

Additional Author(s): Sheridan Rose, MS-3, Edward Via College of Osteopathic Medicine - Auburn; Clinton Martin MD, UAB Heersink School of Medicine, Department of Psychiatry

Introduction/Background: Frontotemporal dementia (FTD) is a form of dementia, most commonly diagnosed in individuals less than 65 years of age. FTD is an umbrella clinical term that encompasses a group of neurodegenerative diseases characterized by progressive deficits in behavior, executive function, or language. Behavioral and personality changes occur frequently over the course of many neurodegenerative diseases, but they are among the most prominent symptoms in FTD. Agitation is a common behavioral disturbance featuring exaggerated motor activity and verbal and/or physical aggressiveness, that can be very distressing for patients and caregivers.

Case Description: This patient is a 75-year-old male diagnosed with frontotemporal dementia five years ago. Since his diagnosis he has had significant worsening of his mood and impulsivity with symptoms described as anger, frustration, and constant pacing. Neurology started him on quetiapine, but he ran out at some point, after which his agitation and sleep problems worsened. He also takes citalopram, which the patient feels helps his anxiety and agitation. He was restarted on the quetiapine and continued the citalopram. However, potential triggers and how to deal with changes in his emotions were also discussed. A follow-up was scheduled to further discuss his overall treatment plan and goals.

Discussion and Conclusion: Agitation is observed in up to 40% of patients with FTD and its incidence is higher in moderate to severe stages of the disease. Agitation occurs because of the interplay between neurobiological and environmental determinants, and adversely impacts cognitive performance, functional status, and patients' quality of life and enhances caregiver's distress. Recent studies have investigated a wide range of non-pharmacological approaches to chronic dementia-related agitation. The person-centered care system uses background, personality, and lifestyle of the patient to promote a positive environment for the best adherence and outcome. Music and socialization can be used for many patients to decrease boredom, which has shown to significantly reduce agitation. Non-pharmacological treatments represent first-line options but are often of limited efficacy and require skilled caregivers. This may explain why various categories of psychotropic drugs are used for treatment of agitation in dementia. These include typical (promazine) and atypical antipsychotics, antidepressants, anticonvulsants, antihistaminergic drugs (hydroxyzine), and herbal preparations.

References:

1. Carrarini C, Russo M, Dono F, Barbone F, Rispoli MG, Ferri L, Di Pietro M, Digiovanni A, Ajdinaj P, Speranza R, Granzotto A, Frazzini V, Thomas A, Pilotto A, Padovani A, Onofri M, Sensi SL, Bonanni L. Agitation and Dementia: Prevention and Treatment Strategies in Acute and Chronic Conditions. *Front Neurol.* 2021 Apr 16;12:644317. doi: 10.3389/fneur.2021.644317. PMID: 33935943; PMCID: PMC8085397.
2. Ooi CH, Yoon PS, How CH, Poon NY. Managing challenging behaviours in dementia. *Singapore Med J.* 2018 Oct;59(10):514-518. doi: 10.11622/smedj.2018125. PMID: 30386856; PMCID: PMC6199192. Hedman R, Sandman PO, Edvardsson D. Enacting person-centred care in home care

services for people with dementia. *J Clin Nurs*. 2022 Jun;31(11-12):1519-1530. doi: 10.1111/jocn.16004. Epub 2021 Aug 15. PMID: 34396618.

3. Cohen-Mansfield J, Dakheel-Ali M, Marx MS, Thein K, Regier NG. Which unmet needs contribute to behavior problems in persons with advanced dementia? *Psychiatry Res*. 2015 Jul 30;228(1):59-64. doi: 10.1016/j.psychres.2015.03.043. Epub 2015 Apr 14. PMID: 25933478; PMCID: PMC4451402.
4. Magierski R, Sobow T, Schwertner E, Religa D. Pharmacotherapy of Behavioral and Psychological Symptoms of Dementia: State of the Art and Future Progress. *Front Pharmacol*. 2020 Jul 31;11:1168. doi: 10.3389/fphar.2020.01168. PMID: 32848775; PMCID: PMC7413102.

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-11

Title: Examining Etiologies for Personality Change in a Patient with Epilepsy

Presenting Author: Michael Huber, MS-3, UAB Heersink School of Medicine

Additional Authors: Kenneth Davis, MS-3; Ryan Zaniewski, MS-3: UAB Heersink School of Medicine

Introduction/Background: Personality changes are a complex cascade that have various etiologies. They can originate from common primary psychiatric disorders, such as schizophrenia, bipolar disorder, or depression. Neurological issues like epilepsy and side effects of anti-epileptic drugs like Topiramate, in addition to electrolyte imbalances like hyperglycemia and hypocalcemia can also affect personality. As medical students, we may typically think of personality changes as a separate, stand alone issue with clear-cut distinction from other medical issues. We discuss a patient experiencing a personality change with a history of epilepsy, a condition that is also known to cause post-ictal personality changes and what to consider when making a diagnosis.

Case Description: A 44 year old female with a history of type 1 diabetes and epilepsy on Topiramate and Primidone who presented to the ED after being found unresponsive for 30 minutes. She had been seizure-free for the past 20 years. She was initially treated for DKA in the ICU, but upon transfer to the medical floor, she displayed flattened affect, psychomotor retardation, bizarre behavior, delusions of grandeur, and wandered the hospital at night. The patient's mother noticed progressive flattening of her affect with short term memory and focus problems for the past two years. She had also exhibited bizarre behavior like sneaking into her neighbor's garage and experienced auditory hallucinations. An EEG report showed right temporal high amplitude paroxysmal sharp waves. She was started on Abilify but had no improvement in her affect or behavior.

Discussion and Conclusion: This case poses a diagnostic challenge as her personality changes could have multiple etiologies including electrolyte imbalances, psychiatric disorders, medication adverse effects, and post epileptic personality changes. Electrolyte-based personality changes resolve once imbalances are corrected making this less likely given the patient's status did not improve following correction of electrolytes. Similarly, the patient did not respond to Abilify making an underlying psychiatric disorder less likely. Topiramate-induced psychosis should also be considered; however, given the patient's acute on chronic psychiatric presentation coupled with the EEG findings suggests post-epileptic personality changes as the likely etiology.

References:

1. Geschwind N. (2009). Personality changes in temporal lobe epilepsy. *Epilepsy & behavior* : E&B, 15(4), 425–433.
2. Goldenberg M. M. (2010). Overview of drugs used for epilepsy and seizures: etiology, diagnosis, and treatment. *P & T : a peer-reviewed journal for formulary management*, 35(7), 392–415.
3. Kober, D., & Gabbard, G. O. (2005). Topiramate-induced psychosis. *The American journal of psychiatry*, 162(8), 1542.
4. Webb, W. L., Jr, & Gehi, M. (1981). Electrolyte and fluid imbalance: neuropsychiatric manifestations. *Psychosomatics*, 22(3), 199–203.

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-12

Title: ASD Diagnosis in A Previously Undiagnosed Adult

Presenting Author: Amy Hudson, PGY-2, USA College of Medicine

Additional Authors: Samantha Lee, PGY-2, USA College of Medicine; Adryanna Tucker OMS-3, Edward Via College of Osteopathic Medicine - Auburn; Edgar W. Finn, MD, USA College of Medicine, Department of Psychiatry

Introduction/Background: Approximately 1% of the global population carries a diagnosis of autism spectrum disorder (ASD)¹. ASD is characterized by impairment in social communication and restricted and repetitive behaviors and interests. In this case, a psychiatric patient with likely ASD had gone undiagnosed for years due to his previous schizophrenia diagnosis, in which previous providers had likely attributed his symptoms to psychosis but were rather indications of underlying autism. Diagnosis of ASD in adulthood is complicated due to comorbidities, sensitivities, and repetitive behaviors being seen as psychotic, such as this patient. Obtaining reliable collateral from someone who is able to elaborate on details of the patient's developmental history as well as a timeline of current and past symptoms and behaviors the patient exhibits is critical. His case also highlights the lack of resources available for adults with autism.

Case Description: This patient is a 33-year-old male with a history of schizophrenia and multiple previous hospitalizations who was admitted to an involuntary inpatient psychiatric facility due to an increase in aggressive behaviors, an exacerbation of disorganized thought, and suspected medication nonadherence. Although the patient manifests significant thought and speech disorganization, he also demonstrates perseverative speech such as naming various roads, locales, and public officials in his hometown. He engages in repetitive behaviors such as repetitive exhibitionism in significantly inappropriate places. Collateral information was obtained from his sister. In childhood, he was very quiet and often communicated through gestures. He displayed repetitive behavior during childhood. He did not tolerate variation in his daily routine. His social skills were poor, and teachers reported he had no interest in interacting with school staff or peers. Teachers also described learning delays in academic subjects and life skills. However, there is no medical record of the patient being tested for possible autism spectrum disorder. At the time of publication, the patient remains at this inpatient psychiatric facility awaiting placement to an ID group home.

Discussion and Conclusion: The diagnostic instruments ADI-R and ADOS are currently considered the best tools to diagnose ASD, which are not always readily available in underserved regions. The relative lack of psychiatric resources for children with mental health and developmental diagnoses makes it more likely that individuals such as our patient will never receive the proper diagnosis, the proper treatment, or the proper educational interventions during critical developmental stages in childhood and adolescence. The main treatment of ASD is education to the patient and those who interact with them, such as family and school staff. Patients that are diagnosed in late adulthood are more likely to fail to have appropriate resources and placement, ending up in inpatient psychiatric hospitals, group homes, and even homeless. Resources are scarce in underserved areas for children and even less resources for when those children reach adulthood. Due to poor social communication skills and rigid repetitive and ritualistic behaviors, these patients may have difficulties in employment and social relationships. These patients experience profound social and economic exclusion as they are not only overlooked by the community but healthcare providers as well². Training programs for social learning, life skills, structured leisure activity, anger management, and employment programs have shown to improve

social outcomes and community inclusion³. This case emphasizes the misdiagnosis and late diagnosis of autism in adults and the lack of resources available for these patients.

References:

1. World Health Organization. (2022, March 30). Autism. World Health Organization. Retrieved February 19, 2023, from <https://www.who.int/news-room/fact-sheets/detail/autism-spectrum-disorders>
2. Murphy CM, Wilson CE, Robertson DM, Ecker C, Daly EM, Hammond N, Galanopoulos A, Dudley I, Murphy DG, McAlonan GM. Autism spectrum disorder in adults: diagnosis, management, and health services development. *Neuropsychiatr Dis Treat*. 2016 Jul 7;12:1669-86. doi: 10.2147/NDT.S65455. PMID: 27462160; PMCID: PMC4940003.
3. Gray KM, Keating CM, Taffe JR, Brereton AV, Einfeld SL, Reardon TC, Tonge BJ. Adult outcomes in autism: community inclusion and living skills. *J Autism Dev Disord*. 2014 Dec;44(12):3006-15. doi: 10.1007/s10803-014-2159-x. PMID: 24915930.

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-13

Title: Treating Concurrent Obsessive Compulsive Disorder and Autism Spectrum Disorder

Presenting Author: Mia Jetsu, MS-3, USA College of Medicine

Additional Authors: Lauren Mussell, MS-3; Tyeler Rayburn, MD; Robert Detrinis, MD: USA College of Medicine, Department of Psychiatry

Introduction: Patients with autism spectrum disorder (ASD) and concurrent obsessive compulsive disorder (OCD) represent a unique therapeutic challenge. ASD is one of the most common comorbidities with OCD. Seeming overlap includes fixed repetitive actions, anxiety, and lack of insight. While symptoms appear similar at the phenotypic level of expression, the causative psychological and neurochemical nidus of these maladaptive behaviors are distinct for each illness, demonstrating a considerable dilemma for treatment in the outpatient and inpatient setting. Here we present an illustrative case of a patient with ASD and OCD for further discussion of best practices in management.

Case description: A 38 years old man with OCD and ASD was admitted to an inpatient psychiatric hospital for destabilization following the loss of his primary caregivers. He presented with severe anxiety, depression, uncontrollable compulsions of proselytizing, incessant repetition of religious scripture, and behavioral dysregulation. While the patient's mood attenuated with treatment; despite multiple medication trials, the patient's uncontrollable need for compulsory scripture recitation persisted. The patient's thoughts remained saturated with themes of religion, and fixated on his own shame. Additionally, the patient was so thoroughly convinced as to the irreverent and blasphemous nature of peers and staff that he required frequent as-needed sedative-hypnotic-antipsychotics for intractable hostility, and mandatory self-hygiene due to deeply-embedded shame and obsessiveness with remaining modest. The patient lacked any insight into his illness, and was consistently unable to connect with his own subjective experience of emotion. This patient is was involuntarily committed for provision of ongoing psychiatric treatment.

Discussion: Treating comorbid ASD and OCD requires understanding the distinct origins of symptoms. For example, as seen clinically with this patient, repetitive behaviors in autism serve as a pleasurable or anxiolytic release, whereas in OCD alone are often self-identified as disabling and distressing. The ASD/OCD diathesis is difficult to manage with therapy, due to the patient's lack of reciprocity and inability to access emotional valence within themselves. Successful treatment (which we would define as restoration to daily functioning), requires "medicating what can be medicated" and "therapizing what can be therapized."

References:

1. Ivarsson, T., Melin, K., & Wallin, L. (2008). Categorical and dimensional aspects of co-morbidity in obsessive-compulsive disorder (OCD). *European child & adolescent psychiatry*, 17, 20-31.
2. Zandt, F., Prior, M., & Kyrios, M. (2007). Repetitive behaviour in children with high functioning autism and obsessive compulsive disorder. *Journal of autism and developmental disorders*, 37, 251-259.
3. Zandt, F., Prior, M., & Kyrios, M. (2009). Similarities and differences between children and adolescents with autism spectrum disorder and those with obsessive-compulsive disorder: Executive functioning and repetitive behavior. *Autism*, 13(1):43-57.
4. Bejerot, S. (2007). An autistic dimension: A proposed subtype of obsessive-compulsive disorder. *Autism*, 11(2), 101-110.

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-14

Title: Impulse Control Disorders and Prader-Willi Syndrome

Presenting Author: Desalyn Johnson, MS-3, UAB Heersink School of Medicine

Additional Authors: Janaki Nimmagadda, MD; Clinton Martin, MD: UAB Heersink School of Medicine, Department of Psychiatry

Introduction/Background: Prader Willi Syndrome (PWS) is a rare genetic disorder that is associated with neonatal hypotonia, delayed sexual development, and intellectual delay. The genetic basis for PWS is related to imprinting via methylation of the maternal copy of chromosome 15q11. Since the paternal copy is exclusively expressed, a paternal microdeletion in this region can lead to this unique clinical presentation. PWS is associated with various facial abnormalities including narrow nose bridge, thin upper lip and strabismus of the eyes. Diabetes, hypogonadism and cryptorchism are also common features. In addition to the effect PWS has on metabolic, sexual, and endocrine function, PWS can also lead to a variety of psychiatric manifestations.

A key feature of is obesity secondary to hyperphagia. Due to this extreme desire for food and limited satiation, patients may resort to hoarding food or eating discarded food. Hyperphagia can be associated with impulse control disorder, obsessive compulsive disorder and addiction. Naltrexone and bupropion have been sought as potential treatments to increase satiety and decrease impulsivity in patients with PWS. Anxiety is also a common presentation in these patients. Although many symptoms overlap with generalized anxiety disorder, patients with PWS tend to have unique worries that relate to food security, food planning and changes in scheduling.

Case Description: The patient is a 21-year-old male with Prader-Willi Syndrome, Major Depressive Disorder, Generalized Anxiety Disorder, hyperphagia and impulse control disorder. He is currently taking clonazepam 1mg for agitation, 600mg of Lithium Carbonate in the morning, 750mg of Lithium Carbonate in the evening, 300mg of Oxcarbazepine twice daily and 20 mg capsule of Fluoxetine three times a day.

Discussion and Conclusion: Prader-Willi Syndrome can be associated with a plethora of psychiatric conditions, including impulse control disorder, major depressive disorder, generalized anxiety disorder, and deficits in social skills. Medical professionals caring for patients with Prader-Willi syndrome must pay special attention to their psychiatric needs. Although emphasis is often placed on the metabolic effects secondary to hyperphagia, emphasis should also be placed on the psychiatric components contributing to the presentation.

References:

1. Puri MR, Sahl R, Ogden S, Malik S. Prader-Willi Syndrome, Management of Impulsivity, and Hyperphagia in an Adolescent. *J Child Adolesc Psychopharmacol*. 2016 May;26(4):403-4. doi: 10.1089/cap.2015.0240. Epub 2016 Mar 30. PMID: 27028699.
2. Schwartz, L., Caixàs, A., Dimitropoulos, A., Dykens, E., Duis, J., Einfeld, S., Gallagher, L., Holland, A., Rice, L., Roof, E., Salehi, P., Strong, T., Taylor, B., & Woodcock, K. (2021). Behavioral features in prader-willi syndrome (PWS): Consensus paper from the International PWS Clinical Trial Consortium. *Journal of Neurodevelopmental Disorders*, 13(1). <https://doi.org/10.1186/s11689-021-09373-2>

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-15

Title: Examination of Quetiapine in the Role of Delirium

Presenting Author: Cara King, MS-3, USA College of Medicine

Additional Authors: Jeanetta Malone, MD; Maridith Hollis, MD: USA College of Medicine, Department of Psychiatry

Introduction/Background: Delirium is a serious but not uncommon complication of many hospital admissions. Statistics suggest in the acute care setting delirium rates can reach up to 87% (Maldonado). Along with the immediate complications of delirium including increased mortality, morbidity, and hospital stays, studies show that patients who experience delirium are at risk for long term sequelae such as cognitive decline (Goldberg). With such troubling consequences to consider, the goal of physicians should be to reduce the incidence of delirium by limiting risk factors. Quetiapine has been researched in the past as a pharmacological agent in the treatment of agitation in delirium (Devlin). However, some studies suggest that quetiapine may be able to induce delirium outside the presence of other risk factors (Almeida). Below we discuss the case of a patient with sudden onset delirium related to initiation of quetiapine.

Case Description: Patient L is a 20-year-old female with a past psychiatric history of PTSD and unspecified depressive disorder who presented voluntarily to an inpatient psychiatric hospital for homicidal ideations towards her mother. Upon evaluation, patient appeared to be endorsing homicidal ideation in relation to trauma induced symptoms including intrusive thought processes, nightmares, and hypervigilance. The patient was initiated on sertraline and quetiapine to target the above symptoms. Sertraline was titrated to 100mg daily without apparent side effects. Quetiapine was initiated at 50mg nightly and titrated up to 200mg over a 5 day period. After titration, patients began to exhibit symptoms concerning for delirium including new onset delusions, hallucinations, and fluctuating mental status. There was some concern that quetiapine was at least partially responsible for the patient's change in status therefore the medication was discontinued. The patient showed rapid improvement after discontinuation and soon thereafter was discharged to outpatient follow up.

Discussion and Conclusion: The prevention and management of delirium continues to be a significant topic for research and progress. Given both the immediate and long-term risks of delirium clinicians primary focus should be on prevention. The research behind the use of antipsychotics in delirium has been limited and somewhat controversial. Current studies suggest that quetiapine may be one of the preferred agents to manage agitation in delirium. However, it appears that quetiapine in itself may be a pro-delirium agent. Further research into the risk and benefits of quetiapine in delirium would be helpful in considering prevention and management in the future.

References:

1. Almeida, F., Albuquerque, E., & Murta, I. (2019). Delirium Induced by Quetiapine and the Potential Role of Norquetiapine. *Frontiers in neuroscience*, 13, 886. <https://doi.org/10.3389/fnins.2019.00886>
2. Devlin, J. W., Roberts, R. J., Fong, J. J., Skrobik, Y., Riker, R. R., Hill, N. S., Robbins, T., & Garpestad, E. (2010). Efficacy and safety of quetiapine in critically ill patients with delirium: a prospective, multicenter, randomized, double-blind, placebo-controlled pilot study. *Critical care medicine*, 38(2), 419–427. <https://doi-org.libproxy.usouthal.edu/10.1097/CCM.0b013e3181b9e302>
3. Goldberg, T. E., Chen, C., Wang, Y., Jung, E., Swanson, A., Ing, C., Garcia, P. S., Whittington, R. A., & Moitra, V. (2020). Association of Delirium With Long-term Cognitive Decline: A Meta-

analysis. *JAMA neurology*, 77(11), 1373–1381. <https://doi.org.libproxy.usouthal.edu/10.1001/jamaneurol.2020.2273>

4. Maldonado J. R. (2017). Acute Brain Failure: Pathophysiology, Diagnosis, Management, and Sequelae of Delirium. *Critical care clinics*, 33(3), 461–519. <https://doi.org.libproxy.usouthal.edu/10.1016/j.ccc.2017.03.013>

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-16

Title: Cornelia de Lange Syndrome – Psychiatric Manifestations

Presenting Author: Alyssa Lambert, MS-3, UAB Heersink School of Medicine

Additional Author: Janaki Nimmagadda, MD, UAB Heersink School of Medicine - Huntsville, Department of Psychiatry

Introduction/Background: Cornelia de Lange Syndrome (CdLS) is a rare genetic disorder involving multiple organ systems with varying physical, cognitive, and behavioral characteristics present in 1/10,000 live births.(2,3) The syndrome is caused by a mutation in the genes that code for proteins in the cohesin complex, which regulates cohesion between sister chromatids and is essential in the mitosis process.(1)

Case Description: A 5yo female with a past medical history notable for Cornelia de Lange syndrome (CdLS), seizures, attention deficit hyperactivity disorder (ADHD), and disruptive behavior disorder presents for continuing behavioral issues at home and school including aggression, temper tantrums, and head banging during anger fits. She was diagnosed with CdLS at 3yo and her mother states she has many characteristic CdLS features including high arching palate, micrognathia, typical facial characteristics, and global developmental delay with an estimated emotional age of a 2-year-old. She started speaking when she was 3.5 years old. On exam, the patient appears younger than her stated age, made intermittent eye contact, has up turned nose, high arching brows, low set ears, small hands, short digits, and hirsutism localized to her back. She plays with toys in the office and follows instructions. She responds to questions with 2-3 word sentences. She does show some impulsivity by turning off the lights during the exam and running into the hallway.

Discussion and Conclusion: Patients with CdLS are often misdiagnosed, so it is important to consider this syndrome when a patient's presentation is suspicious for a genetic disorder to avoid delaying treatment.(3) Our patient has GERD, intestinal malrotation, cardiac defects, and respiratory problems which are all commonly seen in this syndrome. Patients with CdLS have characteristic behaviors including self-injury, anxiety, negative affect, autism spectrum disorder (ASD), ADHD, and sleep problems.(2,3) ASD is highly prevalent in this syndrome, with 51-67% of patients with CdLS meeting the score for diagnosis.(2) About 56% of patients have self-injurious behavior which seems to be related to severity of intellectual disability, Communication difficulty, and impulsivity.(4) Early diagnosis, treatment, and involvement of a multidisciplinary medical team leads to a better quality of life for these patients.(6)

References:

1. Grados MA, Alvi MH, Srivastava S. Behavioral and psychiatric manifestations in Cornelia de Lange syndrome. *Curr Opin Psychiatry*. 2017 Mar;30(2):92-96. doi: 10.1097/YCO.0000000000000311. PMID: 28125439; PMCID: PMC7451083.
2. Groves L, Oliver C, Moss J. Behaviour across the lifespan in Cornelia de Lange syndrome. *Curr Opin Psychiatry*. 2021 Mar 1;34(2):112-117. doi: 10.1097/YCO.0000000000000671. PMID: 33278154.
3. Cascella M, Muzio MR. Cornelia de Lange Syndrome. 2022 Feb 5. In: *StatPearls* [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. PMID: 32119471.
4. Selicorni A, Mariani M, Lettieri A, Massa V. Cornelia de Lange Syndrome: From a Disease to a Broader Spectrum. *Genes (Basel)*. 2021 Jul 15;12(7):1075. doi: 10.3390/genes12071075. PMID: 34356091; PMCID: PMC8307173.

5. Huisman S, Mulder P, Kuijk J, Kerstholt M, van Eeghen A, Leenders A, van Balkom I, Oliver C, Piening S, Hennekam R. Self-injurious behavior. *Neurosci Biobehav Rev*. 2018 Jan;84:483-491. doi: 10.1016/j.neubiorev.2017.02.027. Epub 2017 Jul 8. PMID: 28694012.
6. Mulder PA, Huisman SA, Hennekam RC, Oliver C, van Balkom ID, Piening S. Behaviour in Cornelia de Lange syndrome: a systematic review. *Dev Med Child Neurol*. 2017 Apr;59(4):361-366. doi: 10.1111/dmcn.13361. Epub 2016 Dec 18. PMID: 27988966.

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-17

Title: ADHD treatment in children- Less is more

Presenting Author: Pedram Maleknia, MS-3, UAB Heersink School of Medicine

Additional Author: Janaki Nimmagadda, MD: UAB Heersink School of Medicine - Huntsville, Department of Psychiatry

Introduction/ Background: Attention-deficit hyperactivity disorder (ADHD) is defined as age-inappropriate and impairing levels of inattention, hyperactivity, and/or impulsivity. The estimated percentage of children affected by ADHD is between 5% to 12%. The pharmacological treatments for this condition in children consist of psychostimulants (methylphenidate and amphetamines) and non-psychostimulants (atomoxetine and alpha2-agonists). The efficacy and safety of ADHD medications continues to be controversial despite the recommendations in clinical guidelines. Current guidelines are not consistent in their treatment recommendations as some recommend methylphenidate over amphetamines while others do not distinguish between these two.

Case Description: A 6y/o black male presents with a PMH of severe hyperactivity, impulsivity and easy distractibility. He has been diagnosed by his previous provider as ADHD, Hyperactive type. He was started on long-acting Amphetamine derivative, lisdexamfetamine 20mg. Patient was referred to our clinic for possible depression and ongoing ADHD treatment. Parents reported that he has lost 5 pounds in the last six months. They also noticed that he wakes up every night to eat snacks and he hoards food under his bed. He has recently started seeing a Gastroenterologist for stomach pain, vomiting, poor appetite and weight loss for which he was prescribed omeprazole and ondansetron. Parents also reported that they are now giving melatonin 10mg to help him sleep but it does not always work. On examination, he appeared younger than his stated age, thin, maintained poor eye contact, and sat quietly beside his parents. He did not answer when asked about his mood but his affect was guarded and blunt. After further clarification of history, we determined that the majority of symptoms became a concern after starting lisdexamfetamine. After discussing with parents, we changed the stimulant to short acting methylphenidate 5mg and also added clonidine 0.1mg for better control of hyperactivity and impulsivity. Patient started sleeping better, his appetite improved significantly, he did not have any more headaches and started to gain some weight. During follow up visits, he appeared bright, played well in the office, maintained fair eye contact and had a full range of affect.

Discussion and Conclusion: A systematic review and meta-analysis illustrated that methylphenidate was more efficacious than placebo and more tolerated than placebo in a study consisting of 133 double-blind randomized controlled trials. Amphetamines were more efficacious than placebo but were less tolerable than placebo in this same trial. In this same study, methylphenidate was the only drug with better acceptability than placebo. The adverse events associated with amphetamines as opposed to methylphenidate include increased anorexia, weight loss, insomnia, and changes in blood pressure. These adverse events are not seen, or if seen are far less detrimental when methylphenidate is used. Amphetamine-based medications have more dopamine and norepinephrine availability, which increases their likelihood of medicinal divergence and misuse, illustrated in figure 1. In addition, when clinicians use methylphenidate, it allows for more potent medicinal treatment to be considered if first-line treatment fails. More research to create an algorithm that is followed by pediatricians and psychiatrists will ease the burden of refractory ADHD treatment for all involved.

References:

1. Cortese S, Adamo N, Del Giovane C, Mohr-Jensen C, Hayes AJ, Carucci S, Atkinson LZ, Tessari L, Banaschewski T, Coghill D, Hollis C, Simonoff E, Zuddas A, Barbui C, Purgato M, Steinhausen HC, Shokraneh F, Xia J, Cipriani A. Comparative efficacy and tolerability of medications for attention-deficit hyperactivity disorder in children, adolescents, and adults: a systematic review and network meta-analysis. *Lancet Psychiatry*. 2018 Sep;5(9):727-738. doi: 10.1016/S2215-0366(18)30269-4. Epub 2018 Aug 7. PMID: 30097390; PMCID: PMC6109107.
2. Sayal K, Prasad V, Daley D, Ford T, Coghill D. ADHD in children and young people: prevalence, care pathways, and service provision. *Lancet Psychiatry*. 2018 Feb;5(2):175-186. doi: 10.1016/S2215-0366(17)30167-0. Epub 2017 Oct 9. PMID: 29033005.
3. Chai G, Governale L, McMahon AW, Trinidad JP, Staffa J, Murphy D. Trends of outpatient prescription drug utilization in US children, 2002-2010. *Pediatrics*. 2012 Jul;130(1):23-31. doi: 10.1542/peds.2011-2879. Epub 2012 Jun 18. PMID: 22711728.
4. Bolea-Alamañac B, Nutt DJ, Adamou M, Asherson P, Bazire S, Coghill D, Heal D, Müller U, Nash J, Santosh P, Sayal K, Sonuga-Barke E, Young SJ; British Association for Psychopharmacology. Evidence-based guidelines for the pharmacological management of attention deficit hyperactivity disorder: update on recommendations from the British Association for Psychopharmacology. *J Psychopharmacol*. 2014 Mar;28(3):179-203. doi: 10.1177/0269881113519509. Epub 2014 Feb 12. PMID: 24526134.
5. Storebø OJ, Ramstad E, Krogh HB, Nilausen TD, Skoog M, Holmskov M, Rosendal S, Groth C, Magnusson FL, Moreira-Maia CR, Gillies D, Buch Rasmussen K, Gauci D, Zwi M, Kirubakaran R, Forsbøl B, Simonsen E, Gluud C. Methylphenidate for children and adolescents with attention deficit hyperactivity disorder (ADHD). *Cochrane Database Syst Rev*. 2015 Nov 25;2015(11):CD009885. doi: 10.1002/14651858.CD009885.pub2. PMID: 26599576; PMCID: PMC8763351.
6. Maleknia P, Chalamalla AR, Arynchyna-Smith A, Dure L, Murdaugh D, Rocque BG. Prevalence of attention-deficit/hyperactivity disorder and intellectual disability among children with hydrocephalus. *J Neurosurg Pediatr*. 2022 May 27:1-7. doi: 10.3171/2022.4.PEDS2249. Epub ahead of print. PMID: 35623365.
7. Heal DJ, Smith SL, Gosden J, Nutt DJ. Amphetamine, past and present--a pharmacological and clinical perspective. *J Psychopharmacol*. 2013 Jun;27(6):479-96. doi: 10.1177/0269881113482532. Epub 2013 Mar 28. PMID: 23539642; PMCID: PMC3666194.
8. Amphetamine Vs. Methylphenidate.; 2021. Accessed September 1, 2022. <https://www.youtube.com/watch?v=XfrF1nOsOhk>
9. Ritalin vs Adderall - Difference and Comparison | Diffeen. Accessed September 1, 2022. https://www.diffeen.com/difference/Adderall_vs_Ritalin
10. Cortese S, Newcorn JH, Coghill D. A Practical, Evidence-informed Approach to Managing Stimulant-Refractory Attention Deficit Hyperactivity Disorder (ADHD). *CNS Drugs*. 2021 Oct;35(10):1035-1051. doi: 10.1007/s40263-021-00848-3. Epub 2021 Aug 17. PMID: 34403134.
11. Verghese C, Abdijadid S. Methylphenidate. 2022 Jan 12. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan--. PMID: 29494058.

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-18

Title: Mutism as the Presenting Symptom of Psychosis and Identifying First Episode Psychosis

Presenting Author: Jeanetta Malone, PGY-2, USA College of Medicine

Additional Author: William Burns, MD, USA College of Medicine

Introduction/Background: Approximately 1% of the world's population is affected by schizophrenia, however schizophrenia remains one of the leading causes of disability worldwide.³ Therefore, it's important to identify those who may be at risk of developing the disease and initiating early treatment. Studies have shown that the duration of time with untreated psychosis can lead to worse response to pharmacologic intervention and poorer prognosis.⁴ Despite this, almost 40% of patients who go on to develop a primary psychotic disorder were not given a diagnosis at their initial episode of care.¹ Below we review the case of a patient with sudden onset mutism concerning for first episode psychosis.

Case Description: Patient is an 18-year-old male with past psychiatric history of MDD who presented to an inpatient psychiatric hospital for mutism and minimal oral intake. The patient stopped talking 6 months prior to hospitalization but was brought in for evaluation after he began to refuse to eat or drink. On evaluation, patient refused to verbally participate, but responded with hand gestures. Patient presentation was initially concerning for depression, and he was started on mirtazapine. Mirtazapine was titrated without any perceivable effect and with patient requiring IV fluids due to severe dehydration, treatment was escalated quickly. Patient was administered lorazepam 2mg IM to rule out possible catatonia without any improvement. Due to the bizarre nature of the patient's presentation, he was started on risperidone and titrated up to 1mg twice daily. After initiation of risperidone, patient began to write responses to questions and a week after he spoke for the first time in 6 months. Patient was discharged on risperidone and unfortunately lost to follow up.

Discussion and Conclusion: Mutism can be appreciated in a variety of conditions. In psychiatry, the differential includes psychotic, dissociative, and affective disorders with catatonia being the most common cause.² With mutism seen in such a wide range of disorders, it is especially important to keep psychosis in one's mind when given this presentation. Given the prevalence of delayed diagnosis of first episode psychosis as well as the associated poor outcomes further research should be conducted on the evaluation and recognition of this patient population.

References:

1. Albin, K., Albin, C., Jeffries, C. D., & Perkins, D. O. (2021). Clinician Recognition of First Episode Psychosis. *The Journal of adolescent health : official publication of the Society for Adolescent Medicine*, 69(3), 457–464. <https://doi-org.libproxy.usouthal.edu/10.1016/j.jadohealth.2020.12.138>
2. Grover, S., Naskar, C., & Sharma, P. (2022). Treatment-resistant non-catatonic mutism in schizophrenia: A case report and review of literature. *Asian journal of psychiatry*, 71, 103067. <https://doi.org/10.1016/j.ajp.2022.103067>
3. Mueser, K. T., & McGurk, S. R. (2004). Schizophrenia. *Lancet (London, England)*, 363(9426), 2063–2072. [https://doi-org.libproxy.usouthal.edu/10.1016/S0140-6736\(04\)16458-1](https://doi-org.libproxy.usouthal.edu/10.1016/S0140-6736(04)16458-1)
4. Perkins DO, Gu H, Boteva K, et al. Relationship between duration of untreated psychosis and outcome in first-episode schizophrenia: A critical review and meta-analysis. *Am J Psychiatry* 2005;162:1785e804

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-19

Title: Neither Here nor There: A Case of Reduplicative Paramnesia

Presenting Author: Tucker McCaleb, MS-3, UAB Heersink School of Medicine

Additional Authors: Alyssa Lambert MS-3; Tarak Vasavada, MD: UAB Heersink School of Medicine - Huntsville, Department of Psychiatry

Introduction/Background: Reduplicative paramnesia (RP) is a type of spatial delusion characterized by the firm belief that a typically familiar location has been replicated and now exists simultaneously in two or more distinct places [1]. It is similar to delusional misidentification syndromes (DMS), in which patients believe certain persons, objects, or the self have been replaced or transformed [2]. DMS have strong comorbid psychiatric concurrence (60% to 75%) with psychotic disorders [2,3]. However, RP's presumed etiology is believed to be almost entirely neural-based, with most literature correlating its occurrence with structural brain damage [4,5].

Case Description: A 52-year-old male with no contributing past medical history and family history of Huntington's disease presented to the ED with his wife for a suicide attempt (SA). Two weeks before arrival, he presented to the same ED for choreiform movements. He received a head CT, MRI, and MRA that showed no significant abnormality and was informed that his symptoms might be due to Huntington's disease. He was discharged home and was referred to an outpatient neurologist. Shortly after, the patient began experiencing worsening derealization, confusion, and increased difficulty performing ADLs. His upcoming SA entailed him placing a firearm in his mouth in front of his wife and stating, "how can [he] die if [he's] already dead". He was diagnosed with MDD with psychotic features and involuntarily committed. During his hospital stay, he experienced a feeling that he was simultaneously back in the hospital room and at home. While his wife was visiting him, he would hallucinate that she was at home as well. He also experienced auditory hallucinations of voices telling him to remain calm and that everything would be okay.

Discussion and Conclusion: There is a paucity of literature describing RP; most research thus far is from a neuroanatomy perspective correlating RP with structural brain abnormalities. Our patient's presentation differs as his neuroimaging was essentially normal. His acute onset of RP could be a manifestation of his MDD with psychotic features with replication of a familiar setting serving as an unconscious defense mechanism to help console the patient. Further research should be conducted to explore other psychiatric etiologies of RP.

References:

1. Gerace, C., & Blundo, C. (2013). Reduplicative paramnesia: not only one. *The Journal of neuropsychiatry and clinical neurosciences*, 25(3), E16–E18. <https://doi.org/10.1176/appi.neuropsych.12030072>
2. Jovic, M.D., Zeljko (1992) "Delusional Misidentification Syndromes," *Jefferson Journal of Psychiatry*: Vol. 10: Iss. 1, Article 4. DOI: <https://doi.org/10.29046/JJP.010.1.001> Available at: <https://jdc.jefferson.edu/jeffjpsychiatry/vol10/iss1/4>
3. Politis, M., & Loane, C. (2012). Reduplicative paramnesia: a review. *Psychopathology*, 45(6), 337–343. <https://doi.org/10.1159/000337748>
4. Borghesani, V., Monti, A., Fortis, P., & Miceli, G. (2019). Reduplicative paramnesia for places: A comprehensive review of the literature and a new case report. *Clinical neurology and neurosurgery*, 181, 7–20. <https://doi.org/10.1016/j.clineuro.2019.03.022>

5. Alves, P. N., Fonseca, A. C., Silva, D. P., Andrade, M. R., Pinho-E-Melo, T., Thiebaut de Schotten, M., & Martins, I. P. (2021). Unravelling the Neural Basis of Spatial Delusions After Stroke. *Annals of neurology*, 89(6), 1181–1194. <https://doi.org/10.1002/ana.26079>

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-20

Title: Observation of Blood Dyscrasias in Antipsychotic Non-naive Patient During Medication Transition: A Case Report

Presenting Author: Kimberly McWilliams, PGY-2, UAB Heersink School of Medicine

Additional Author: Bidari Birur, MD, UAB Heersink School of Medicine

Introduction/Background: It is well known in psychiatry and pharmacology that Clozapine carries with it the risk of agranulocytosis. However, similar risk has also been noted in the structurally similar olanzapine and other typical and atypical antipsychotics. Patients who have a history of drug-induced blood dyscrasias have an increased chance of developing a second generation antipsychotic induced blood dyscrasia. Overlapping treatment, change in medication, and augmentation strategies should be considered when treating patients with antipsychotics. Patients should also be routinely screened for pre-existing Benign Neutropenia of Ethnicity as well as anemia and neutropenia before starting any antipsychotics to establish an appropriate baseline before therapy. Follow-up labs should be obtained at regular intervals to ensure patients are being appropriately screened for neutropenia and other blood dyscrasias.

Case Description: Mr. H is a 35 year-old Black Male with an established psychiatric history of schizophrenia presented to the emergency department from Jail. Patient had uncontrolled symptoms of psychosis evidenced by coprophagia and scotolia as well as charging at other inmates, officers, and declaring that he is the Messiah. Patient explains that his feces is “made of gold” and that is why he put it in his hair. He had been treated prior to his imprisonment with paliperidone Trinzta. While in jail, the patient was treated with haloperidol 10mg BID for three months and olanzapine 20mg nightly for two months. While being screened for medical clearance for admission to the psychiatric unit, the patient was noted to have pancytopenia. As a previous patient of the health system, it was compared to prior labs and determined to be new onset. Subsequently, hematology was consulted and conducted a few additional tests and determined that the most likely cause of the patient’s pancytopenia was his antipsychotic medication. The recommendation was to hold the current medication and change to a different medication so the patient’s blood counts could recover. However, when the patient transitioned to paliperidone after his blood counts began to recover, his blood counts dropped yet again. This was attempted twice before the decision was made to place the patient on lithium in an effort to support his white blood cell counts. While there was recovery in neutrophil count and platelets with lithium and vitamin b12, there was still an overall leukopenia present. For this reason the paliperidone was suspended indefinitely. The patient was not placed on an antipsychotic due to continued blood dyscrasias, and was released due to lack of overt psychosis warranting admission. However, the patient was brought back to the emergency room two weeks later with increased psychosis. He was admitted to the psychiatric hospital after obtaining medical clearance and was started on aripiprazole with no drop in blood counts. Patient was later transitioned to risperidone again with no drop in blood counts.

Discussion and Conclusion: While it is currently the standard of care to check CBCs with clozapine patients, it should also be considered standard of care when switching patients from one antipsychotic to another, or when prescribing multiple antipsychotics concomitantly. This is due to the known risk of antipsychotics to cause leukopenia and that it can lead to agranulocytosis which can be fatal. While it is true that risks and benefits must be weighed, it is important not to overlook the hematologic implications these medications can have. If a patient has shown to have any blood dyscrasia due to the administration of an antipsychotic, the clinician should consider waiting a short time after full recovery

of blood counts before the initiation of a new antipsychotic to ensure stability of cell counts and clearance of other antipsychotics as not to increase risk of recurrence or progression.

References:

1. Wasti, Afshan & Zahid, Saadia & Ahmed, Nikhat. (2013). Antipsychotic drugs induced iron deficiency anemia in schizophrenic patients.. International Journal of Advanced Research. 1. 111-118.
2. Carol A. Tamminga (2000) Treatment mechanisms: traditional and new antipsychotic drugs, Dialogues in Clinical Neuroscience, 2:3, 281-286, DOI: 10.31887/DCNS.2000.2.3/ctamminga
3. Kattalai Kailasam V, Chima V, Nnamdi U, Sharma K, Shah K. Risperidone-induced reversible neutropenia. Neuropsychiatr Dis Treat. 2017;13:1975-1977 <https://doi.org/10.2147/NDT.S141472>
4. Jose Angel Mendez Sanchez (2021) Antipsychotics and Neutropenia: An Update. J Men Hea Psy Dis 2: 1-7.
5. Vasanth Kattalai Kailasam, Victoria Chima, Uchechukwu Nnamdi, Kavita Sharma & Kairav Shah (2017) Risperidone-induced reversible neutropenia, Neuropsychiatric Disease and Treatment, 13:, 1975-1977, DOI: 10.2147/NDT.S141472

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-21

Title: Managing Behavioral Symptoms in Autoimmune Limbic Encephalitis

Presenting Author: Lauren Mussell, MS-3, USA College of Medicine

Additional Authors: Mia Jetsu, MS-3; Tyeler Rayburn, MD; Sandra Parker, MD: USA College of Medicine, Department of Psychiatry

Introduction/Background: Patients with autism spectrum disorder (ASD) and concurrent obsessive compulsive disorder (OCD) represent a unique therapeutic challenge. ASD is one of the most common comorbidities with OCD. Seeming overlap includes fixed repetitive actions, anxiety, and lack of insight. While symptoms appear similar at the phenotypic level of expression, the causative psychological and neurochemical nidus of these maladaptive behaviors are distinct for each illness, demonstrating a considerable dilemma for treatment in the outpatient and inpatient setting. Here we present an illustrative case of a patient with ASD and OCD for further discussion of best practices in management.

Case Description: A 38 years old man with OCD and ASD was admitted to an inpatient psychiatric hospital for destabilization following the loss of his primary caregivers. He presented with severe anxiety, depression, uncontrollable compulsions of proselytizing, incessant repetition of religious scripture, and behavioral dysregulation. While the patient's mood attenuated with treatment; despite multiple medication trials, the patient's uncontrollable need for compulsory scripture recitation persisted. The patient's thoughts remained saturated with themes of religion, and fixated on his own shame. Additionally, the patient was so thoroughly convinced as to the irreverent and blasphemous nature of peers and staff that he required frequent as-needed sedative-hypnotic-antipsychotics for intractable hostility, and mandatory self-hygiene due to deeply-embedded shame and obsessiveness with remaining modest. The patient lacked any insight into his illness, and was consistently unable to connect with his own subjective experience of emotion. This patient is was involuntarily committed for provision of ongoing psychiatric treatment.

Discussion and Conclusion: Treating comorbid ASD and OCD requires understanding the distinct origins of symptoms. For example, as seen clinically with this patient, repetitive behaviors in autism serve as a pleasurable or anxiolytic release, whereas in OCD alone are often self-identified as disabling and distressing. The ASD/OCD diathesis is difficult to manage with therapy, due to the patient's lack of reciprocity and inability to access emotional valence within themselves. Successful treatment (which we would define as restoration to daily functioning), requires "medicating what can be medicated" and "therapizing what can be therapized."

References:

1. Ivarsson, T., Melin, K., & Wallin, L. (2008). Categorical and dimensional aspects of co-morbidity in obsessive-compulsive disorder (OCD). *European child & adolescent psychiatry*, 17, 20-31.
2. Zandt, F., Prior, M., & Kyrios, M. (2007). Repetitive behaviour in children with high functioning autism and obsessive compulsive disorder. *Journal of autism and developmental disorders*, 37, 251-259.
3. Zandt, F., Prior, M., & Kyrios, M. (2009). Similarities and differences between children and adolescents with autism spectrum disorder and those with obsessive-compulsive disorder: Executive functioning and repetitive behavior. *Autism*, 13(1):43-57.
4. Bejerot, S. (2007). An autistic dimension: A proposed subtype of obsessive-compulsive disorder. *Autism*, 11(2), 101-110.

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-22

Title: Imposters Among Us: Amphetamine-Induced Misidentification Syndrome (Capgras Delusions)

Presenting Author: Michael Pettit, MS-3, UAB Heersink School of Medicine

Additional Authors: Tarak Vasvada MD; Clinton Martin MD: UAB Heersink School of Medicine - Huntsville, Department of Psychiatry

Introduction/Background: Capgras Syndrome (CS) is a delusion of misidentification, usually that a familiar person or even one's own self has been replaced by a duplicate (1). As the presentation is variable, so are the etiologies. Chiefly, psychiatric and neurodegenerative disorders have been implicated (2). However, we present a case of what we believe to be amphetamine-induced CS.

Case Description: The patient is a 49-year-old female with a history significant for diagnosis of bipolar disorder. She was evaluated in the ER for agitation and increasing paranoia. After her husband became profoundly ill 5yr prior, she believed his eyes and behavior changed and that he might have been a replacement. She had not yet harmed him but believed that the only way to find out would be to kill him so her real husband would return. She also believed that an identical double had replaced her son-in-law and that she might have been in the Illuminati. These beliefs had intensified over the preceding 6mo, leading to greater social and interpersonal dysfunction. Additionally, she began using recreational amphetamines 5yr ago, which she continued to use regularly. She denied other illicit substances. She initially denied the report of these delusions but confirmed them after admission. Ziprasidone treatment was initiated, and as her agitation improved, she was discharged with outpatient commitment.

Discussion and Conclusion: In addition to the afore-mentioned psychiatric and neurodegenerative etiologies for CS, illicit substance use has been implicated. However, these reports are often in the context of unspecified or combined substance use (3, 4). Nonetheless, some studies have specified certain substances. Notably, amphetamines have demonstrated this association, but detailed reports of clinical course in the setting of amphetamine use alone was not noted (4, 5). Evaluation of CS should not be limited to determination of etiology but should include anticipation of potential adverse events, such as violence. Both the affected and misidentified individuals are often in harm's way (3). The potential for physical harm in the context of CS should not be understated. Hopefully, careful management of patients with illicit substance use can help mitigate the stressors and risks they experience and reduce overall harm potential.

References:

1. Vasavada, T, Masand, P. An unusual variant of Capgras syndrome. *Percept Mot Skills* (1992) 75(3 Pt 1):971-974. doi:10.2466/pms.1992.75.3.971
2. Silva, JA, Leong, GB, Weinstock, R, Sharma, KK, Klein, RL. Delusional misidentification syndromes and dangerousness. *Psychopathology* (1994) 27(3-5):215-219. doi:10.1159/000284872
3. Pandis, C, Agrawa, I N, Poole, N. Capgras' delusion: A systematic review of 255 published cases. *Psychopathology* (2019) 52(3):161-173. doi:10.1159/000500474
4. Sampathi, BR, Sofine, A, Alvarez, J, Bota R. Capgras syndrome in substance-induced psychosis. *Ment Illn* (2018) 10(2):7807. doi:10.4081/mi.2018.7807
5. Josephs, KA. Capgras syndrome and its relationship to neurodegenerative disease. *Arch Neurol* (2007) 64(12):1762-1766. doi:10.1001/archneur.64.12.1762

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-23

Title: The Ketamine Lifeline: A Hope for Treatment Resistant Depression?

Presenting Author: Michael Pettit, MS-3, UAB Heersink School of Medicine

Additional Authors: Clinton Martin MD; Richard Shelton MD; Janaki Nimmagadda MD; Anupama Yedla, MD; UAB Heersink School of Medicine - Huntsville, Department of Psychiatry

Introduction/Background: Treatment-Resistant Depression (TRD) is persistent depressive dysfunction despite adequate treatment from 2 different antidepressant classes. TRD has been estimated to comprise 30.9% of medicated Major Depressive Disorder (MDD) in the US (1). Much effort has been put forth to combat this pervasive effect. One such effort has been the study of ketamine and esketamine, NMDA receptor antagonists that could play an important role in this rise to challenge against TRD.

Case Description: The patient is a 35-year-old female with a history of treatment-resistant depression, borderline personality disorder, and anxiety with comorbid hypertension, chronic migraines, and chronic pain. After having tried multiple other antidepressant medications with other psychiatric providers, the patient presented to psychiatry clinic with increasing depression, hopelessness, and passive suicidal ideations. They received IV ketamine which she tolerated well with rapid improvement in somatization, energy, mood, and suicidal ideation. Intranasal maintenance esketamine was initiated with persistence of antidepressant effects but with diminished effect relative to initial response.

Discussion and Conclusion: Ketamine has demonstrated the ability to rapidly and significantly reduce depressive symptoms in TRD with sustained effect after a single dose (2). Intranasal esketamine has demonstrated similar sustained effect as an adjunct in TRD. This effect of esketamine was shown to have dose-dependent duration and magnitude. Doses as low as 28mg have shown significant effect, and some doses have shown sustained effect with administration as infrequent as every other week (3). Despite these merits, long-term ketamine treatment has the potential for drawbacks. The development of tolerance has been reported, and questions also remain regarding the ability to consistently sustain treatment effects (4, 5). Despite these challenges in the study of ketamine and TRD, benefits have been demonstrated. Hopefully through more study, we can better characterize the pharmacologic activity of ketamine and develop treatment regimens that sustain positive effects for our patients with mitigation of unwanted adverse effects in TRD.

References:

1. Zhdanova M, Pilon D, Ghelerter I, et al. The prevalence and national burden of treatment-resistant depression and major depressive disorder in the United States. *J Clin Psychiatry* (2021) 82(2):20m13699 doi:10.4088/JCP.20m13699
2. Zarate CA, Jr., Singh JB, Carlson PJ, et al. A randomized trial of an N-methyl-D-aspartate antagonist in treatment-resistant major depression. *Arch Gen Psychiatry* (2006) 63(8):856-864 doi:10.1001/archpsyc.63.8.856
3. Daly, EJ, Singh JB, Fedgchin M, et al. Efficacy and safety of intranasal esketamine adjunctive to oral antidepressant therapy in treatment-resistant depression: A randomized clinical trial. *JAMA Psychiatry* (2018) 75(2):139-148 doi:10.1001/jamapsychiatry.2017.3739
4. Schak KM, Vande Voort JL, Johnson EK, et al. Potential risks of poorly monitored ketamine use in depression treatment. *Am J Psychiatry* (2016) 173(3):215-218 doi:10.1176/appi.ajp.2015.15081082
5. Siegel AN, Di Vincenzo JD, Brietzke E, et al. Antisuicidal and antidepressant effects of ketamine and esketamine in patients with baseline suicidality: A systematic review. *J Psychiatr Res* (2021) 137:426-436 doi:10.1016/j.jpsychires.2021.03.009

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-24

Title: Normoprolactinemic galactorrhea in the setting of pregnancy delusions; a discussion of pseudocyesis

Presenting Author: Madeleine Powell, OMS-3, Edward Via College of Osteopathic Medicine - Auburn

Additional Author: Christiana Wilkins, MD, East Alabama Health, Department of Psychiatry

Introduction/Background: Delusions of pregnancy are not uncommon in women with psychosis and represent a fixed, false belief with no physical signs of pregnancy. This is in contrast to pseudocyesis which is the false belief of pregnancy with physical signs of pregnancy [1]. The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) categorizes pseudocyesis under Other Specified Somatic Symptoms and Related disorders [2]. The Internet-based clinical reference Uptodate defines pseudocyesis as a non-psychotic illness, whereas the DSM5 does not distinguish pseudocyesis as a psychotic versus non-psychotic disease. This is a rare phenomenon that has been scarce in the literature, typically only reported in case reports and case series. According to The Journal of Nursing Practitioners, roughly 550 cases have been reported with the majority occurring in ages ranging from 20-44 years old [3]. Most cases occur in developing countries and even 1% of reported cases present with symptoms of labor [4]. Often described as “phantom pregnancy”, pseudocyesis typically occurs in cultures that expect women to bear a child. Pseudocyesis has been described in times of infertility, relationship stress, and even after the death of a child [3][4]. The “non-psychotic” criteria makes it a tough diagnosis for those who are acutely psychotic with delusions of pregnancy and physical symptoms of pregnancy. It is important to note that physical symptoms of pregnancy in a patient with psychosis could potentially further induce delusions of pregnancy. Some authors suggest not differentiating between pseudocyesis and pregnancy delusions [5].

Case Description: A 20 year old nulliparity white female, Ms. X, with known bipolar I disorder presented with symptoms of mania to the Emergency Department via officers from the local jail. Ms. X was admitted to the inpatient psychiatric unit with a diagnosis of bipolar I disorder, current episode manic, severe with psychotic features. Throughout her admission she displayed delusions of pregnancy and often complained of abdominal growth and pain. On day 21 of her inpatient stay, Ms. X complained of galactorrhea. Urine and serum hCG were both negative, TSH and free T4 were within normal limits, prolactin level was within normal limits, and a brain computerized tomography scan without contrast yielded no abnormalities. Ms. X medications at this time were Aripiprazole 15mg, Olanzapine 10mg, and as needed Quetiapine 100mg and Chlorpromazine 50-100mg. The breast discharge continued for two weeks while the patient was titrated to Aripiprazole 30mg. All of these medications have the potential to raise prolactin levels and cause galactorrhea. Ms. X’s galactorrhea further induced her pregnancy delusions and she was never able to be redirected from these thoughts during her 54 day inpatient admission.

Discussion and Conclusion: Normoprolactinemic galactorrhea is a rare symptom of antipsychotic medication use. For nulliparous women it is very rare for galactorrhea to occur in the setting of normal prolactin levels, especially while taking antipsychotics [6]. Since prolactin elevation has been associated with symptoms similar to pregnancy, the literature has grown to display pseudocyesis and delusion of pregnancy on a continuum that occurs in women of all mental statuses [7]. Whereas delusions of pregnancy occur in the absence of pregnancy symptoms, pseudocyesis is physical symptoms misinterpreted as pregnancy as happened in our patient [1]. This rarity raises the concern of how physical symptoms can further induce somatic delusions as has been reported in many case studies

[7]. This case provides an example of the overlap between pseudocyesis and delusions of pregnancy and furthers the question whether there should be a distinction between the two.

References:

1. Tarín JJ, Hermenegildo C, García-Pérez MA, Cano A. Endocrinology and physiology of pseudocyesis. *Reprod Biol Endocrinol*. 2013;11:39. Published 2013 May 14. doi:10.1186/1477-7827-11-39
2. *Diagnostic and Statistical Manual of Mental Disorders: DSM-5*. Arlington, VA: American Psychiatric Association; 2017.
3. Campos SJ, Link D. Pseudocyesis. *The journal for nurse practitioners*. 2016;12(6):390-394. doi:10.1016/j.nurpra.2016.03.009
4. Ibekwe PC, Achor JU. Psychosocial and cultural aspects of pseudocyesis. *Indian J Psychiatry*. 2008;50(2):112-116. doi:10.4103/0019-5545.42398
5. Anvi-Barron O, Gupta R, Miller LJ. Pseudocyesis. UpToDate. https://www.uptodate.com/contents/pseudocyesis?search=pseudocyesis&source=search_result&selectedTitle=1~44&usage_type=default&display_rank=1. Accessed December 12, 2022.
6. Wieck A, Haddad PM. Antipsychotic-induced hyperprolactinemia in women: pathophysiology, severity and consequences: Selective literature review. *British Journal of Psychiatry*. 2003;182(3):199-204. doi:10.1192/bjp.182.3.199
7. Seeman MV. Pseudocyesis, delusional pregnancy, and psychosis: The birth of a delusion. *World J Clin Cases*. 2014;2(8):338-344. doi:10.12998/wjcc.v2.i8.328

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-25

Title: Gas Station Heroin: Tianeptine Withdrawal Symptom Presentation, Risk Factors for Use and Withdrawal Management

Presenting Author: Ashton Prestage, OMS-3, Edward Via College of Medicine -Auburn

Additional Authors: Amy Hudson, PGY-2, USA College of Medicine; Luke Engeriser, MD, USA College of Medicine, AltaPointe Health

Introduction/Background: Tianeptine is an atypical antidepressant marketed in Europe. Described as a mu-opioid receptor agonist, it is theorized that its antidepressant effect may be related to its mu-opioid receptor activity.¹ Tianeptine is not approved for use in the United States by the Food and Drug Administration. However, tianeptine can be found online marketed as a nootropic. Although it is illegal in Alabama, tianeptine can be purchased in gas stations in nearby states such as Mississippi and Florida under the names of ZaZa Red, TD Red, and Tianna.² A study documented that tianeptine users were interested in the drug for opioid substitution or withdrawal alleviation as well as self-medication for psychiatric symptoms such as anxiety.³ However, patients can develop a physiologic tolerance for tianeptine, requiring supratherapeutic doses to prevent distressing withdrawal symptoms that may include psychosis.⁴ In this case study, two patients are profiled who presented to a community mental health center for tianeptine withdrawal. We later discuss risk factors for tianeptine use and management strategies for withdrawal symptoms.

Case Description: Patient #1 is a 37-year-old male with a history of GAD and alcohol use disorder who presented to the behavioral health crisis center for tianeptine withdrawal. Patient had last used tianeptine two weeks ago. Withdrawal was characterized by depersonalization/derealization, clouded sensorium, muscle spasticity, aches, pains, nausea, diarrhea, and severe fatigue. This was his third withdrawal attempt. Previous withdrawals had been managed with Suboxone 16 mg, which was eventually tapered off. Patient #2 is a 46-year-old male who presented to the behavioral health crisis center for co-occurring alcohol and tianeptine withdrawal. He self-medicated with alcohol to relieve anxiety and associated panic attacks. He found alcohol no longer relieved his symptoms, so he started taking tianeptine two months prior after he saw the supplement in a gas station. He reports anxiety was relieved immediately with “4-6 pills”. He quickly built a tolerance, and he increased his pill intake weekly. The day prior to presentation, patient had consumed 25-30 pills. Patient was placed on CIWA and COWS protocol for alcohol and tianeptine withdrawal, respectively. Patient was not initiated on Suboxone due to lack of moderate withdrawal symptoms, and he was discharged to an inpatient rehabilitation facility. Prior to discharge, the patient was provided with a prescription for low-dose clonazepam to treat acute panic attacks, which he had previously come to rely on tianeptine to treat.

Discussion and Conclusion: This is a case series depicting tianeptine addiction and withdrawal. Tianeptine is a mu-opioid agonist that has been shown to have anti-depressant effects. It also has demonstrated activity at glycine-B site in the NMDA receptor and GluA1 subunit of the AMPA receptor, which may contribute to its anxiolytic effects⁵. Both patients have a history of alcohol use disorder and anxiety, which we suspect may be risk factors for tianeptine use disorder. We also relate our patients’ experiences to the public’s perception of tianeptine, while also dissecting how it is marketed to the public. Finally, we discuss established treatment options for tianeptine withdrawal. Physicians should ask their patients about nootropics use and be able to identify tianeptine’s clinical presentation as its availability and subsequent usage increases.

References:

1. Lauhan, R., Hsu, A., Alam, A., & Beizai, K. (2018). Tianeptine abuse and dependence: Case report and literature review. *Psychosomatics*, 59(6), 547–553. <https://doi.org/10.1016/j.psym.2018.07.006>
2. Krishnan, M. (2022, December 12). 'gas station heroin' is causing intense withdrawals. it's legal in most states. Tianeptine, or 'Gas Station Heroin,' Causes Intense Withdrawals And It's Mostly Legal. Retrieved February 19, 2023, from <https://www.vice.com/en/article/88q3va/tianeptine-gas-station-heroin-legal-in-most-states>
3. Smith, K. E., Rogers, J. M., Strickland, J. C., & Epstein, D. H. (2021). When an obscurity becomes trend: Social-media descriptions of tianeptine use and associated atypical drug use. *The American Journal of Drug and Alcohol Abuse*, 47(4), 455–466. <https://doi.org/10.1080/00952990.2021.1904408>
4. Karim, A., & Ioannou, C.(2020). Tianeptine abuse leading to an episode of Psychosis: A case report and literature review. *Journal of Psychiatric Practice*, 26(2), 146–148. <https://doi.org/10.1097/prs.0000000000000448>
5. Michienzi, A. E., & Borek, H. A. (2022). Emerging agents of substance use/misuse. *Emergency Medicine Clinics of North America*, 40(2), 265–281. <https://doi.org/10.1016/j.emc.2022.01.001>
6. Alamo, C., García-García Pilar, Lopez-Muñoz Francisco, & Zaragoza Cristina. (2019). Tianeptine, an atypical pharmacological approach to depression. *Revista De Psiquiatría Y Salud Mental (English Edition)*, 12(3), 170–186. <https://doi.org/10.1016/j.rpsmen.2018.09.001>
7. Samuels, B. A., Nautiyal, K. M., Kruegel, A. C., Levinstein, M. R., Magalong, V. M., Gassaway, M. M., Grinnell, S. G., Han, J., Ansonoff, M. A., Pintar, J. E., Javitch, J. A., Sames, D., & Hen, R. (2017). The behavioral effects of the antidepressant tianeptine require the mu-opioid receptor. *Neuropsychopharmacology*, 42(10), 2052–2063. <https://doi.org/10.1038/npp.2017.60>
8. García-García P. Tianeptina, antide- presivo con perfil beneficio/riesgo positivo. *Rev Psiquiatr Salud Ment (Barc)*. 2016;9:234-235. 9. Nabil, Syed; Chong Guan, Ng; Abd Rashid, Rusdi. Tianeptine Dependence: A Case Report. *Malaysian Journal Of Psychiatry* 27(1):p 47-51, June 2018.

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-26

Title: Managing Psychiatric Symptoms in a Patient with Autoimmune Limbic Encephalitis

Presenting Author: Tyeler Rayburn, PG-1, USA College of Medicine

Additional Author: Sandra Parker, MD, USA College of Medicine, Department of Psychiatry

Introduction: Limbic encephalitis is an autoimmune, usually paraneoplastic, inflammation of the limbic system with neuropsychiatric implications. Symptoms include mood changes, circadian irregularities, hallucinations, delusions, disorganized thought, neurocognitive dysfunction, seizures, among others. It rarely occurs without concurrent neoplasm, though when it does, psychiatric manifestations are often the sole symptoms of encephalitis flare. Here we present an expository case of autoimmune encephalitis with cardinal psychiatric symptoms.

Case Description: A 12 years old boy with a history of autoimmune limbic encephalitis was consulted to our psychiatric service for violent ideations, auditory hallucinations, behavioral dysregulation, and physical aggression. The patient had been admitted one week prior for inpatient provision of the first of three treatments of rituximab for encephalitis flare and was discharged shortly thereafter. Pregabalin was also started for neuropathic pain. In the interim, the patient's psychiatric symptoms persisted and worsened, culminating in him eloping from home and being found by law enforcement hours later, demonstrating incoherent and erratic behavior. He was transported to a tertiary medical center for urgent evaluation. On exam, the patient was irritable, hyperactive, and violent, though could not articulate why and was frustrated that he could control himself. MRI was performed demonstrating stable basal ganglia FLAIR signal representing unchanged if mildly improved encephalitis. Urine drug screen was negative. The patient was admitted for continued treatment of encephalitis exacerbation. Sublingual dexmedetomidine was considered due to level of aggression, though the patient was adequately managed with as-needed sedative-hypnotic-antipsychotic until his previously-prescribed paliperidone was reintroduced. As of the publishing of this abstract, this patient is continuing to be managed psychiatrically. Full treatment course to be discussed with poster presentation.

Discussion: Many patients with autoimmune encephalitis present first to a psychiatrist. The natural history of the illness frequently begins with psychiatric symptoms, with neurologic symptoms developing with disease progression. Often these patients will present with agitation, aggression, and mood instability. According to the most comprehensive review, a new onset, otherwise inexplicable mixed mood-psychosis syndrome should prompt further diagnostic evaluation. Diagnosis of autoimmune encephalitis requires neuroimaging and cerebrospinal fluid antibody testing. Treatment is prompt immunotherapy and symptomatic management.

References:

1. Finke, C. (2019). A transdiagnostic pattern of psychiatric symptoms in autoimmune encephalitis. *The Lancet Psychiatry*, 6(3), 191-193.
2. Dalmau, J., & Graus, F. (2018). Antibody-mediated encephalitis. *New England Journal of Medicine*, 378(9), 840-851.
3. Vernino, S., Geschwind, M., & Boeve, B. (2007). Autoimmune encephalopathies. *The neurologist*, 13(3), 140-147.
4. Gultekin, S. H., Rosenfeld, M. R., Voltz, R., Eichen, J., Posner, J. B., & Dalmau, J. (2000). Paraneoplastic limbic encephalitis: neurological symptoms, immunological findings and tumour association in 50 patients. *Brain*, 123(7), 1481-1494.

5. Nevels, R. M., Dehon, E. E., Alexander, K., & Gontkovsky, S. T. (2010). Psychopharmacology of aggression in children and adolescents with primary neuropsychiatric disorders: a review of current and potentially promising treatment options. *Experimental and clinical psychopharmacology*, 18(2), 184.

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-27

Title: Antibiotics as Novel Therapy to Treat Valproic Acid Overdose

Presenting Author: Sheridan Rose, MS-3, Edward Via College of Osteopathic Medicine - Auburn

Additional Author: Caleb Hood, MS-3, UAB Heersink School of Medicine

Introduction/ Background: Valproic Acid (VPA) is one of the most commonly prescribed anti-epileptic drugs, used for both seizure prophylaxis and as a antimanic in patients with bipolar disorder. VPA toxicity is a life-threatening event that may result in severe CNS depression and hepatotoxicity. Our case report highlights the use of a novel therapy for VPA toxicity, carbapenem antibiotics. When used in combination with standard of care treatment, carbapenems can provide a positive outcome for patients with VPA toxicity.

Case Description: A 24-year-old male presented to the emergency department and reported he intentionally ingested twenty 500 mg delayed release Depakote tablets. The patient had a past medical history significant for bipolar disorder with psychotic features and extrapyramidal side effects. Initial vital signs were within normal limits. The patient's lab tests in the emergency department showed a serum valproic acid level of 547.0 and an ammonia blood level of 76. The patient was given a loading dose of levocarnitine at 100 mg/kg, thereafter, to be continued at 50mg/hr for eight hours. Ertapenem 1 g to be infused at a rate of 200 mL/hr. The patient was admitted to the intensive care unit for close observation. A repeat valproic acid level six hours after initial doses of levocarnitine and ertapenem was 284.0. The patient's valproic acid level returned to baseline 48 hours after admission. The patient was discharged into an inpatient psychiatric care facility.

Discussion and Conclusion: The standard of care for VPA toxicity has been long been supportive measures and carnitine supplementation (1). In addition to standard therapies, there have been increasingly positive outcomes in patients with VPA toxicity who were treated with carbapenem antibiotics. While the exact mechanisms of action are unknown. Carbapenems are proposed to both decrease intestinal absorption (2) and inhibit glucuronidation of VPA in hepatocytes, leading to a decrease in blood levels (3).

References:

1. Lheureux PE, Hantson P. Carnitine in the treatment of valproic acid-induced toxicity. *Clin Toxicol (Phila)*. 2009 Feb;47(2):101-11. doi: 10.1080/15563650902752376. PMID: 19280426.
2. Dreucean D, Beres K, McNierney-Moore A, Gravino D. Use of meropenem to treat valproic acid overdose. *Am J Emerg Med*. 2019 Nov;37(11):2120.e5-2120.e7. doi: 10.1016/j.ajem.2019.158426. Epub 2019 Sep 4. PMID: 31500925.
3. Al-Quteimat O, Laila A. Valproate Interaction With Carbapenems: Review and Recommendations. *Hosp Pharm*. 2020 Jun;55(3):181-187. doi: 10.1177/0018578719831974. Epub 2019 Feb 22. PMID: 32508355; PMCID: PMC7243600.

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-28

Title: Child and Adolescent Psychiatry Training Recruitment: Does Exposure in Medical School Correlate with Knowledge About the Career Specialty and Confidence in Patient Interactions?

Presenting Author: Moiqua Tillman, PGY-4, UAB Heersink School of Medicine

Additional Authors: Ethan Atwood, PGY-4; Michael Falola, MD, MPH; Blessing Falola, MD: UAB Heersink School of Medicine, Department of Psychiatry

Introduction/Background: National Pediatric Organizations and the United States Surgeon General recently declared an ongoing national emergency in mental health for the child and adolescent population (1,2). There has been an increasingly significant workforce shortage in child and adolescent psychiatry (CAP). In 2019, the Substance Abuse and Mental Health Services Administration (SAMHSA) reported 8,000-9,000 practicing providers with the necessity of an additional 48,000-49,000 to adequately meet the needs of pediatric patients with serious mental illness (3). According to AACAP Workforce Map by state, there are no child and adolescent psychiatrist in 55 out of 67 Alabama counties (4). The National Resident Matching Program data for the past five years shows a high percentage trend of unfilled training positions in the subspecialty (5). To gain additional insight into this challenge, this project investigated if exposure to CAP in medical school is likely to result in increased knowledge about the subspecialty and confidence in handling CAP patients and their families.

A survey was designed to assess residents' exposure to CAP in medical school, knowledgeability regarding CAP specialty career, and the confidence level in interacting with CAP patients and families. The career knowledgeability and confidence level were measured using Likert scale ranging from 1 to 5. The survey was administered to general psychiatry residents via Qualtrics link. The overall response rate was 74%. Of the 26 psychiatry residents that completed survey, 15 (57.7%) were PGY-1, 5 (19.2%) were PGY-2, 3 (11.5%) PGY-3 and 2 (7.7%) were PGY-4. Data was analyzed using percentages and chi-square test.

Data analysis revealed 17 (65%) had CAP exposure, while 9 (35%) had no exposure. There was a large percentage difference in knowledge of CAP specialty between the CAP-exposed vs. CAP non-exposed group; 65% vs. 33%; $p=0.13$ respectively. There is a marked percentage difference between the confidence level of the CAP- exposed vs. CAP non-exposed group, 47% vs. 11%, $p=0.067$, respectively.

Discussion and Conclusion: These findings suggest that CAP exposure in medical school is associated with greater knowledge about the CAP specialty and higher level of confidence in CAP patient/family interactions. Future studies can include multiple sites with geographical regions representation.

References:

1. Aacap. (n.d.). Pediatricians, caps, and children's hospitals declare national emergency. Retrieved, from https://www.aacap.org/AACAP/zLatest_News/Pediatricians_CAPs_Childrens_Hospitals_Declare_National_Emergency_Childrens_Mental_Health.aspx
2. Shoemaker, E.Z., Brenner, A.M. Making More Child and Adolescent Psychiatrists: Responding to the National Emergency in Mental Health in Children and Adolescents. *Acad Psychiatry* 46, 1-5 (2022). <https://doi.org/10.1007/s40596-022-01597-1>

3. Aacap. (n.d.). Workforce maps by State. Retrieved February 25, 2023, from https://www.aacap.org/AACAP/Advocacy/Federal_and_State_Initiatives/Workforce_Maps/Home.aspx
4. Fellowship Data & Reports. NRMP. (n.d.). Retrieved, from <https://www.nrmp.org/match-data-analytics/fellowship-data-reports/>.

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-29

Title: Observation of Blood Dyscrasias in Antipsychotic Non-naive Patient During Medication Transition: A Case Report

Presenting Author: Kimberly McWilliams, PGY-2, UAB Heersink School of Medicine

Additional Author: Bidari Birur, MD, UAB Heersink School of Medicine

Introduction/Background: It is well known in psychiatry and pharmacology that Clozapine carries with it the risk of agranulocytosis. However, similar risk has also been noted in the structurally similar olanzapine and other typical and atypical antipsychotics. Patients who have a history of drug-induced blood dyscrasias have an increased chance of developing a second generation antipsychotic induced blood dyscrasia. Overlapping treatment, change in medication, and augmentation strategies should be considered when treating patients with antipsychotics. Patients should also be routinely screened for pre-existing Benign Neutropenia of Ethnicity as well as anemia and neutropenia before starting any antipsychotics to establish an appropriate baseline before therapy. Follow-up labs should be obtained at regular intervals to ensure patients are being appropriately screened for neutropenia and other blood dyscrasias.

Case Description: Mr. H is a 35 year-old Black Male with an established psychiatric history of schizophrenia presented to the emergency department from Jail. Patient had uncontrolled symptoms of psychosis evidenced by coprophagia and scotolia as well as charging at other inmates, officers, and declaring that he is the Messiah. Patient explains that his feces is “made of gold” and that is why he put it in his hair. He had been treated prior to his imprisonment with paliperidone Trinzta. While in jail, the patient was treated with haloperidol 10mg BID for three months and olanzapine 20mg nightly for two months. While being screened for medical clearance for admission to the psychiatric unit, the patient was noted to have pancytopenia. As a previous patient of the health system, it was compared to prior labs and determined to be new onset. Subsequently, hematology was consulted and conducted a few additional tests and determined that the most likely cause of the patient’s pancytopenia was his antipsychotic medication. The recommendation was to hold the current medication and change to a different medication so the patient’s blood counts could recover. However, when the patient transitioned to paliperidone after his blood counts began to recover, his blood counts dropped yet again. This was attempted twice before the decision was made to place the patient on lithium in an effort to support his white blood cell counts. While there was recovery in neutrophil count and platelets with lithium and vitamin b12, there was still an overall leukopenia present. For this reason the paliperidone was suspended indefinitely. The patient was not placed on an antipsychotic due to continued blood dyscrasias, and was released due to lack of overt psychosis warranting admission. However, the patient was brought back to the emergency room two weeks later with increased psychosis. He was admitted to the psychiatric hospital after obtaining medical clearance and was started on aripiprazole with no drop in blood counts. Patient was later transitioned to risperidone again with no drop in blood counts.

Discussion and Conclusion: While it is currently the standard of care to check CBCs with clozapine patients, it should also be considered standard of care when switching patients from one antipsychotic to another, or when prescribing multiple antipsychotics concomitantly. This is due to the known risk of antipsychotics to cause leukopenia and that it can lead to agranulocytosis which can be fatal. While it is true that risks and benefits must be weighed, it is important not to overlook the hematologic implications these medications can have. If a patient has shown to have any blood dyscrasia due to the administration of an antipsychotic, the clinician should consider waiting a short time after full recovery

of blood counts before the initiation of a new antipsychotic to ensure stability of cell counts and clearance of other antipsychotics as not to increase risk of recurrence or progression.

References:

1. Wasti, Afshan & Zahid, Saadia & Ahmed, Nikhat. (2013). Antipsychotic drugs induced iron deficiency anemia in schizophrenic patients.. International Journal of Advanced Research. 1. 111-118.
2. Carol A. Tamminga (2000) Treatment mechanisms: traditional and new antipsychotic drugs, Dialogues in Clinical Neuroscience, 2:3, 281-286, DOI: 10.31887/DCNS.2000.2.3/ctamminga
3. Kattalai Kailasam V, Chima V, Nnamdi U, Sharma K, Shah K. Risperidone-induced reversible neutropenia. Neuropsychiatr Dis Treat. 2017;13:1975-1977 <https://doi.org/10.2147/NDT.S141472>
4. Jose Angel Mendez Sanchez (2021) Antipsychotics and Neutropenia: An Update. J Men Hea Psy Dis 2: 1-7.
5. Vasanth Kattalai Kailasam, Victoria Chima, Uchechukwu Nnamdi, Kavita Sharma & Kairav Shah (2017) Risperidone-induced reversible neutropenia, Neuropsychiatric Disease and Treatment, 13:, 1975-1977, DOI: 10.2147/NDT.S141472

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-30

Title: PCOS and Psychiatric Disorders

Presenting Author: Miranda Worley, MS-3, UAB Heersink School of Medicine - Huntsville

Additional Author: Clinton Martin, MD, FAPA, UAB Heersink School of Medicine - Huntsville

Introduction/Background: Polycystic ovarian syndrome (PCOS) is the most common endocrine disorder in reproductive age women.¹ Diagnosis is made by identifying 2 out of 3 Rotterdam criteria: oligo/anovulation, clinical and/or laboratory evidence of hyperandrogenism, and polycystic ovaries. Hyperandrogenism leads to side effects including hirsutism and acne, which can cause low self-esteem. Tay et al. demonstrated that women with PCOS are more likely to have issues with self-esteem along with psychological distress compared to controls.² In addition, people with PCOS often develop insulin resistance, infertility, and obesity. To date, most guidelines on PCOS focus only on gynecologic and endocrine aspects of the condition and do not address accompanying psychological disorders.

Case Description: Patient is an 18-year-old with a past medical history of ADHD, gender dysphoria, generalized anxiety disorder, major depression disorder, and comorbid PCOS. PCOS symptoms began at age 12 with pain and mood swings along with irregular cycles. Psychiatric symptoms also began at age 12 with multiple hospitalizations for suicidal ideation and self-harming behavior requiring multiple psychiatric medications. At age 16, their OB/GYN started oral contraception pills and PCOS symptoms improved. Now, the patient's psychiatric symptoms are maintained well with desvenlafaxine 50mg and methylphenidate ER 27 mg.

Discussion and Conclusion: Currently, the American College of Obstetrics and Gynecology (ACOG) does not have recommendations regarding screening for depression and anxiety in patients with PCOS. A meta-analysis revealed that women with PCOS have three times the odds of depression symptoms and five times the odds for anxiety symptoms when compared with women without PCOS.³ The etiology of comorbid psychiatric disorders with PCOS is unclear, but insulin resistance, hyperandrogenism, and obesity are theorized to play a role. The Australian Endocrine Society recommends that women diagnosed with PCOS be screened for anxiety and depression due to the high prevalence of comorbidity.⁴ No guidelines have been released that discuss treating patients with PCOS and psychiatric disorders compared to patients without PCOS. PCOS-related treatments such as lifestyle modifications, oral contraceptive pills, and insulin resistance management can help decrease depression and anxiety symptoms.¹ Future studies should explore if specific antidepressants/anxiolytics are more effective in treating comorbid PCOS and psychiatric illness.

References:

1. Cooney, L. G., & Dokras, A. (2017). Depression and Anxiety in Polycystic Ovary Syndrome: Etiology and Treatment. *Current psychiatry reports*, 19(11), 83. <https://doi.org/10.1007/s11920-017-0834-2>
2. Tay, C. T., Teede, H. J., Hill, B., Loxton, D., Anju E. Joham, A. E. (2019). Increased prevalence of eating disorders, low self-esteem, and psychological distress in women with polycystic ovary syndrome: a community-based cohort study. *Fertility and Sterility*, 112(2), 353-361. <https://doi-org.uab.idm.oclc.org/10.1016/j.fertnstert.2019.03.027>
3. Cooney, L. G., Lee, I., Sammel, M. D., & Dokras, A. (2017). High prevalence of moderate and severe depressive and anxiety symptoms in polycystic ovary syndrome: a systematic review and meta-analysis. *Human Reproduction*, 32(5), 1075–1091. <https://doi.org/10.1093/humrep/dex044>

4. Legro, R. S., Arslanian, S. A., Ehrmann, D. A., Hoeger, K. M., Murad, M. H., Pasquali, R., Welt, C. K., & Endocrine Society (2013). Diagnosis and treatment of polycystic ovary syndrome: an Endocrine Society clinical practice guideline. *The Journal of clinical endocrinology and metabolism*, 98(12), 4565–4592. <https://doi.org/10.1210/jc.2013-2350>

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-31

Title: Vagal Nerve Stimulation in Treatment-Resistant Depression

Presenting Author: Ryan Zaniewski, MS-3, UAB Heersink School of Medicine

Additional Author: Clinton Martin, MD, UAB Heersink School of Medicine - Huntsville, Department of Psychiatry

Introduction/Background: Major Depression disorder is a major challenge to global mental health and is detrimental to quality of life. Although current treatments involving antidepressant medications are effective, one third to one half of patients do not respond to multiple trials of antidepressants. (2) Vagal nerve stimulation (VNS) is an FDA approved therapy for treatment-resistant depression (TRD) for those who have not had an adequate response to two or more antidepressant trials. VNS has been found to be beneficial in improving quality of life and suicidality among unipolar TRD patients and improved depression in bipolar TRD patients. (6) Studies have also shown patients receive longterm benefit and lower rates of relapse with one study showing 42% of participants having a response to treatment with remission rates of 22% at 2 years. (7) Comparatively, one study examining the longterm effects of pharmacotherapy, ECT, and psychotherapy showed response rates of 18.2% at 2 years with a 7.8% remission rate. (4)

Case Description: Our patient is a 57 year old woman who has failed several trials of SSRI, SNRI, MAOI, ECT and TMS. Her depression was intractable and associated with suicidal ideation, complete lack of motivation, loss of sleep, tearfulness, rejection sensitivity and extreme guilt. Prior to VNS treatment she was severely depressed for over 30 years. Since receiving her VNS, she has noticed about 65% remission of her symptoms which has made a significant difference in her quality of life.

Discussion and Conclusion: VNS works via an implantable device that sends a low current electrical pulse (0.25-2.5milliAmp) through the afferent fibers of the left cervical vagus nerve and via the nucleus tractus solitarius to various regions of the brain. (5) Increased activity of serotonergic and noradrenergic systems have also been observed. (4) Metabolic changes have been observed in areas involved in mood regulation. (1) VNS is well tolerated with the most common side with minor side effects. (6,7) Current studies aim to further evaluate longterm efficacy and mechanism of action. With more information being elucidated about VNS therapy for TRD patients, more clinicians and patients may choose VNS therapy as another option in the treatment of this complicated disease..

References:

1. Chae, J. H., Nahas, Z., Lomarev, M., Denslow, S., Lorberbaum, J. P., Bohning, D. E., & George, M. S. (2003). A review of functional neuroimaging studies of vagus nerve stimulation (VNS). *Journal of psychiatric research*, 37(6), 443–455.
2. Cipriani, A., Furukawa, T. A., Salanti, G., Chaimani, A., Atkinson, L. Z., Ogawa, Y., Leucht, S., Ruhe, H. G., Turner, E. H., Higgins, J. P. T., Egger, M., Takeshima, N., Hayasaka, Y., Imai, H., Shinohara, K., Tajika, A., Ioannidis, J. P. A., & Geddes, J. R. (2018). Comparative Efficacy and Acceptability of 21 Antidepressant Drugs for the Acute Treatment of Adults With Major Depressive Disorder: A Systematic Review and Network Meta-Analysis. *Focus (American Psychiatric Publishing)*, 16(4), 420–429.
3. Dorr, A. E., & Debonnel, G. (2006). Effect of vagus nerve stimulation on serotonergic and noradrenergic transmission. *The Journal of pharmacology and experimental therapeutics*, 318(2), 890–898.

4. Dunner, D. L., Rush, A. J., Russell, J. M., Burke, M., Woodard, S., Wingard, P., & Allen, J. (2006). Prospective, long-term, multicenter study of the naturalistic outcomes of patients with treatment-resistant depression. *The Journal of clinical psychiatry*, 67(5), 688–695.
5. Kamel, L. Y., Xiong, W., Gott, B. M., Kumar, A., & Conway, C. R. (2022). Vagus nerve stimulation: An update on a novel treatment for treatment-resistant depression. *Journal of the neurological sciences*, 434, 120171.
6. Nahas, Z., Marangell, L. B., Husain, M. M., Rush, A. J., Sackeim, H. A., Lisanby, S. H., Martinez, J. M., & George, M. S. (2005). Two-year outcome of vagus nerve stimulation (VNS) for treatment of major depressive episodes. *The Journal of clinical psychiatry*, 66(9), 1097–1104.
7. Nierenberg, A. A., Alpert, J. E., Gardner-Schuster, E. E., Seay, S., & Mischoulon, D. (2008). Vagus nerve stimulation: 2-year outcomes for bipolar versus unipolar treatment-resistant depression. *Biological psychiatry*, 64(6), 455–460.

APPA 2023 Spring Meeting Student/Resident Poster Presentation

Abstract 23-1-32

Title: A Case of Phenibut Withdrawal

Presenting Author: Tamara Zaza, UAB Heersink School of Medicine - Huntsville, Department of Psychiatry

Additional Authors: Hunter Soleymani MS-3; Oseyime Okoeguale, MD; Tarak Vasavada, MD: UAB Heersink School of Medicine - Huntsville, Department of Psychiatry

Introduction/Background: Phenibut is a neuropsychiatric drug widely used in Russia for its reported anxiolytic, tranquilizing, and cognition-enhancing properties (1). It is not approved for use in the United States but can be purchased online. It has been shown to have abuse potential and withdrawal syndromes (2-4). We present a patient admitted to the ICU for suspected phenibut withdrawal.

Description: A 48-year-old female with a history of hypertension and anxiety presented as a transfer to the ICU due to accelerated hypertension up to 251/142. The patient was drowsy with flat affect, paranoia, and oriented to self only. CT head was negative for acute findings. In the ICU, her blood pressure was stabilized until acceptable for the inpatient floor. She had altered mental status four days after admission and reported auditory and visual hallucinations. A psychiatric consult was called, and a detailed history was obtained. She admitted to taking phenibut powder for the past several months to self-treat her anxiety, working up to five times the daily recommended dose. Her last dose was two days before admission. She was placed on a chlordiazepoxide taper starting at 25mg TID, baclofen 10mg TID, and gabapentin 300mg TID. On day six of admission, the patient was alert, could hold a conversation, and denied visual/auditory hallucinations. She felt markedly improved and was at baseline mood and mentation. She was discharged with a chlordiazepoxide taper over the next eight days.

Discussion and Conclusion: Phenibut was initially synthesized in Russia in the 1960s as a GABA analog with an additional phenyl ring for increased permeability across the blood-brain barrier (1). It has a similar structure to baclofen and gabapentin and exhibits effects on the GABA-A, GABA-B, and dopamine receptors (1,5). Withdrawal symptoms include agitation, insomnia, anxiety, pain/burning under the skin, and tachycardia (2-4). While there are no guidelines for treating phenibut withdrawal, case reports have shown promising outcomes with baclofen, gabapentin, and benzodiazepines (2-4). Knowledge of the signs and symptoms of phenibut withdrawal and potential treatment options is essential amongst psychiatrists so that the appropriate treatments can be initiated and further complications can be avoided.

References:

1. Lapin I. Phenibut (beta-phenyl-GABA): a tranquilizer and nootropic drug. *CNS Drug Rev.* 2001 Winter;7(4):471-81.
2. Sethi R, Ravishankar DA, Nagireddy R. Phenibut Dependence and Withdrawal. *Prim Care Companion CNS Disord.* 2021 Feb 18;23(1):19102457.
3. Magsalin RM, Khan AY. Withdrawal symptoms after Internet purchase of phenibut (β -phenyl- γ -aminobutyric acid HCl). *J Clin Psychopharmacol.* 2010 Oct;30(5):648-9.
4. Hardman MI, Sprung J, Weingarten TN. Acute phenibut withdrawal: A comprehensive literature review and illustrative case report. *Bosn J Basic Med Sci.* 2019 May 20;19(2):125-129.
5. Zvejniece L, Vavers E, Svalbe B, Veinberg G, Rizhanova K, Liepins V, Kalvinsh I, Dambrova M. R-phenibut binds to the α - δ subunit of voltage-dependent calcium channels and exerts gabapentin-like anti-nociceptive effects. *Pharmacol Biochem Behav.* 2015 Oct;137:23-9.
6. Nedzlek C D, Michaelis A. An Unusual Presentation of an Uncommon Drug: A Case Report on Phenibut Overdose. *Cureus.* 2022 April 7;14(4): e23913.