

APPA 2022 Fall Conference Student/Resident Abstracts

Abstract 22-2-01

Title: Psychogenic Polydipsia induced Hyponatremia and consequent seizure in a schizophrenic patient: A Case Report

Presenting Author: Shawn Chakraborty, MS-4, Alabama College of Osteopathic Medicine

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Introduction/Background: Psychogenic polydipsia (PPD) is described as polyuria and polydipsia, commonly seen in patients with psychiatric illness. While a clear etiology has not been established, excessive water intake leading to hyponatremia in psychiatry patients is not a rare event. Symptomatic hyponatremia has a variety of symptoms including psychosis, fatigue, seizures, coma and death; therefore it is imperative that psychiatrists and all physicians managing the care of psychiatric patients are considering a psychogenic cause of hyponatremia when establishing a differential diagnosis (1).

Description: Ms. M is a 30-year-old Caucasian female with past medical history of schizophrenia, PTSD, OCD, ADHD and eating disorder who presented to the inpatient psychiatric unit in a catatonic, non-verbal state. The patient was treated with therapeutic dose of Ativan and medically stabilized. On the 20th day of her admission, the patient experienced a generalized tonic-clonic seizure and was admitted to the local hospital for workup. Per staff, prior to the incident the patient was seen drinking several cups of water to the point of vomiting. The patient was found to be severely hyponatremic with a sodium level less than 120mmol/L and endorses a history of excessive water intake both prior to and while admitted to the psychiatric inpatient service. The patient was readmitted to the inpatient psychiatric hospital 2 days after the event with CMP within normal limits. The patient was put on 1:1 watch and fluid restriction to 2 liter was initiated. On interview, the patient noted that the urge to drink was a part of her obsessive-compulsive habits with inability to control impulses. The patient did not have further recurrent seizure episodes during the rest of her hospital course.

Discussion and Conclusion: It is imperative that clinicians evaluate seizures in their psychiatric patients with an open mind, keeping both physiologic and psychogenic causes in mind. First line treatment should consist of strict fluid restriction with close supervision and behavioral modification while being sure to avoid rapid correction due to increased risk for central pontine myelinolysis (3). Polydipsia in schizophrenic patients is associated with decreased life-expectancy and proper conservative management can aid the recovery process for psychiatric patients (2).

References:

1. Kotagiri R, Kutti Sridharan G. Primary Polydipsia. 2022 Apr 30. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan -. PMID: 32965922.
2. Sharma P, Shah B, Sangroula M, Jirel R. Psychogenic Polydipsia Complicated to Hyponatremia Induced Seizure in Schizophrenia: A Case Report from Nepal. *Case Rep Psychiatry*. 2019 Nov 16;2019:6021316. doi: 10.1155/2019/6021316. PMID: 31827965; PMCID: PMC6885238.
3. Vieweg WV. Treatment strategies in the polydipsia-hyponatremia syndrome. *J Clin Psychiatry*. 1994 Apr;55(4):154-60. PMID: 8071260.

APPA 2022 Fall Conference Student/Resident Abstracts

Abstract 22-2-02

Title: Voltage Gated Channel Blockage by Antidepressants in Management of Fibromyalgia

Presenting Author: Bayley Atkins, MS-3, University of Alabama at Birmingham Heersink School of Medicine

Additional Authors: Janaki Nimmagadda M.D.; Anupama Yedla M.D.; Richard Shelton M.D.; Clinton Martin M.D.: UABSOM Huntsville Campus, Department of Psychiatry, University of Alabama at Birmingham Heersink School of Medicine

Introduction/Background: Duloxetine has been used as a treatment for both depression and fibromyalgia for some time. The mechanism of action of duloxetine has been known to be inhibition of serotonin and norepinephrine reuptake in the central nervous system. However, in recent years, another mechanism of action has been studied that better explains duloxetine's effectiveness as a treatment for fibromyalgia. One study showed that duloxetine, as well as tricyclic antidepressants, can inhibit voltage gated sodium channels in the dorsal root ganglia more potently than in the central nervous system (Horishita et al). A second study furthers this notion, showing that duloxetine exhibits a concentration-dependent resting block on channels NaV1.3 and NaV1.7, which are key in initiating and propagating pain signals (Rolland et al). These studies illustrate that duloxetine exhibits many similarities to a classic sodium channel blocker and give further insight to how duloxetine exerts its analgesic effect.

Description: This patient is a 59-year-old female with a long history of recurrent major depressive disorder, fibromyalgia, generalized anxiety disorder, binge eating disorder, arthritis, and insomnia. In the past, she was prescribed gabapentin to treat her pain, but found it to be intolerable due to it making her "not feel well".

After trials of other medications proved to be unsatisfactory for the patient, she was started on duloxetine 30 mg twice daily. She was determined to be a good candidate for duloxetine due to her treatment-resistant depression and concomitant fibromyalgia that also persisted despite treatment.

Discussion and Conclusion: Duloxetine has been found to have a $\geq 30\%$ reduction in the Brief Pain Inventory average pain severity score (Arnold, et al). "The 11-item Brief Pain Inventory was developed by the Pain Research Group of the World Health Organization Collaborating Centre for Symptom Evaluation in Cancer Care for assessment of pain intensity, functional interference and pain relief with analgesics" (From: Encyclopedia of Cancer [Third Edition], 2019). When compared to other treatment options such as pregabalin for concomitant treatment of depression and fibromyalgia, duloxetine was shown to have increased efficacy for the treatment of pain (Bidari, et al). Studies have shown that duloxetine is a safe and tolerable option for patients dealing with depression (Hudson, et al), fibromyalgia (Mease, et al), or both. Duloxetine has also been shown to have a similar efficacy to the tricyclic antidepressant drug class. However, amitriptyline was found to be the most potent at blocking sodium channels involved in initiating and propagating pain, particularly NaV1.7 with an IC50 of 4.6 compared to duloxetine's IC50 of 11.7.

Although duloxetine is not as potent as amitriptyline, its side effect profile may be more tolerable for patients and is a viable option for treating patients with depression, fibromyalgia, or both.

References:

1. Horishita, T. et al. Antidepressants inhibit Nav1.3, Nav1.7, and Nav1.8 neuronal voltage-gated sodium channels more potently than Nav1.2 and Nav1.6 channels expressed in *Xenopus* oocytes. *Naunyn-Schmiedeberg's Arch Pharmacol* (2017) 390:1255–1270 DOI 10.1007/s00210-017-1424-x.
2. Rolland, J. et al. Biophysical Characterization Of Duloxetine Activity On Voltage-gated Sodium Channels Involved In Pain Transmission. 2009. [online] Available at: <[https://www.cell.com/biophysj/pdf/S0006-3495\(08\)01453-7.pdf](https://www.cell.com/biophysj/pdf/S0006-3495(08)01453-7.pdf)> [Accessed 9 May 2022].
3. L.M. Arnold, et al. A randomized, double-blind, placebo-controlled trial of duloxetine in the treatment of women with fibromyalgia with or without major depressive disorder. *Pain*, 119 (1–3) (2005), pp. 5-15.
4. Bidari, A., Moazen-Zadeh, E., Ghavidel-Parsa, B. et al. Comparing duloxetine and pregabalin for treatment of pain and depression in women with fibromyalgia: an open-label randomized clinical trial. *DARU J Pharm Sci* 27, 149–158 (2019). <https://doi.org/10.1007/s40199-019-00257-4>.
5. Hudson, J.I. et al. (2005), Safety and tolerability of duloxetine in the treatment of major depressive disorder: analysis of pooled data from eight placebo-controlled clinical trials. *Hum. Psychopharmacol. Clin. Exp.*, 20: 327-341. <https://doi.org/10.1002/hup.696>.
6. Philip J. Mease, et al. Long-Term Safety, Tolerability, and Efficacy of Duloxetine in the Treatment of Fibromyalgia. *Seminars in Arthritis and Rheumatism*, Volume 39, Issue 6, 2010, Pages 454-464, ISSN 0049-0172, <https://doi.org/10.1016/j.semarthrit.2008.11.001>.
7. Hameed S. NaV1.7 and NaV1.8: Role in the pathophysiology of pain. *Mol Pain* 2019; 15: 1744806919858801

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Abstract 22-2-03

Title: L-Methylfolate Augmentation in the Treatment of Refractory Depression

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Additional Authors: Katherine Vintson, DO; Anupama Yedla, MD; Janaki Nimmagadda, MD; Richard Shelton, MD; Clinton Martin, MD: UABSOM Huntsville Campus, Department of Psychiatry

Introduction/Background: Over 60% of patients with major depressive disorder (MDD) fail to experience a complete remission of symptoms following their first antidepressant treatment [1]. Increased body weight and obesity are positively associated with an increased risk of MDD and poorer response to antidepressant treatment [2]. Papakostas et al. shows that adjunctive treatment with L-methylfolate 15 mg/day has shown significantly greater efficacy compared to placebo with continued SSRI therapy plus placebo in response rate and degree of change in depression symptom score [3]. Prior research has shown that the response rate to L-methylfolate augmented with SSRIs was moderated by baseline obesity. Obese patients (BMI \geq 30) with MDD had a greater response than patients who were not obese [4 & 5].

Description: Patient is a 61-year-old female with MDD, GAD, and insomnia with comorbid obesity. She denies suicidal ideation, but states she has difficulty functioning and performing activities of daily living. She has treatment resistant depression due to failing cognitive behavior therapy, \geq 2 antidepressant medications, and transcranial magnetic stimulation. Current treatment regimen includes Abilify 5 MG, Bupropion 300 MG, Duloxetine 60 MG BID, and Ambien 12.5 MG. Patient was started on 7.5 MG L-methylfolate due to treatment resistant depression and BMI \geq 30.

Discussion and Conclusion: L-Methylfolate augmentation with SSRIs can be a useful adjunctive treatment strategy for patients with major depressive disorder who have not responded to SSRIs and have a BMI \geq 30. In Papakostas et al. study, the number needed to treat for response was 6 in favor L-methylfolate. Other augmentation strategies for treatment resistant depression are 31 for atypical antipsychotics and 32 for lithium [3]. L-methylfolate is relatively well tolerated. There is no statistically difference in change in weight, heart rate, or blood pressure between L-methylfolate and placebo [3]. Using L-methylfolate in patients who have a BMI \geq 30 could lead to personalize medicine when addressing treatment resistance depression. More studies need to be conducted to explore if L-methylfolate would be an effective adjunctive therapy for other classes of antidepressant and whether higher dosages of L-methylfolate would result in different outcomes compared to 15 mg/day.

References:

1. Rush AJ, Trivedi MH, Wisniewski SR, et al. Acute and longer-term outcomes in depressed outpatients requiring one or several treatment steps: a STAR*D report. *Am J Psychiatry*. 2006;163(11):1905–1917. doi:10.176/api.ajp.163.1.1905PubMed
2. Kloiber S, Ising M, Reppermund S, et al. Overweight and obesity affect treatment response in major depression. *Biol Psychiatry*. 2007;62(4):321–326. doi:10.1016/j.biopsych.206.10.

3. Papakostas GI, Shelton RC, Zajecka JM, et al. L-methylfolate as adjunctive therapy for SSRI-resistant major depression: results of two randomized, double-blind, parallel-sequential trials. *Am J Psychiatry*. 2012;169(12):1267-1274. doi:10.1176/appi.ajp.2012.11071114
4. Shelton RC, Pencina MJ, Barrentine LW, Ruiz JA, Fava M, Zajecka JM, Papakostas GI. Association of obesity and inflammatory marker levels on treatment outcome: results from a double-blind, randomized study of adjunctive L-methylfolate calcium in patients with MDD who are inadequate responders to SSRIs. *J Clin Psychiatry*. 2015
5. Papakostas GI, Shelton RC, Zajecka JM, Bottiglieri T, Roffman J, Cassiello C, Stahl SM, Fava M. Effect of adjunctive L-methylfolate 15 mg among inadequate responders to SSRIs in depressed patients who were stratified by biomarker levels and genotype: results from a randomized clinical trial. *J Clin Psychiatry*. 2014 Aug;75(8):855-63. doi: 10.4088/JCP.13m08947. PMID: 24813065.

APPA 2022 Fall Conference Student/Resident Abstracts

Abstract 22-2-04

Title: Paradoxical Depression with Phentermine

Presenting Author: Zoey Duncan, MS-3, UAB Heersink School of Medicine

Additional Authors: Anupama Yedla M.D.; Janaki Nimmagadda, M.D.; Richard Shelton M.D.; Clinton Martin, M.D.: UABHSOM Psychiatry Department

Introduction/Background: Phentermine is an FDA approved medication for the treatment of obesity. It is a sympathomimetic amine that acts similarly to amphetamines that increase release of dopamine and norepinephrine. It has no known effects on serotonin. [1] It is approved for short-term, 12-week course of therapy due to the potential for addiction. Results vary from mild to modest reduction in weight with Phentermine alone. It has also been studied in combination with Topiramate and Orlistat. [1] Studies with the combination of Phentermine and Topiramate have shown statistically significant weight reduction in comparison to placebo. [2] In a double-blind, placebo-controlled study of 68 individuals the only statistically significant side effects of Phentermine were dry mouth ($p = 0.001$) and insomnia ($p = 0.004$). Reports of mood changes were equal in both groups. [3]

Description: 58-year-old male with no previous known psychiatric history, with a genetic vulnerability of bipolar illness in his biological mother, developed new onset depressive symptoms after being treated with phentermine. He also has associated anxiety and uses alprazolam as needed. He is very hesitant to come off of his medications bupropion and oxcarbazepine due to the severity of his depressive episode. He is no longer on phentermine.

Discussion and Conclusion: In current understanding of depression, there is decreased serotonin, norepinephrine, and dopamine, which has been used as a target of medications. SNRIs, Bupropion, and Mirtazapine are antidepressants that have an effect of increasing NE concentration in the treatment of depression [4] The use of amphetamines in treatment of depression has been studied, especially in the elderly population. Lavrensky, et. al demonstrated that the use of methylphenidate, alone and in combination with citalopram decreased depression scores in the elderly quicker than citalopram alone. [5] Additional research is needed to determine the role of Phentermine in rates of depression as well as mechanism, which is not currently well understood. Further research could warrant an additional advisory to patients regarding potential mood changes as a result of the medication.

References:

1. Cosentino G, Conrad AO, Uwaifo GI. Phentermine and topiramate for the management of obesity: a review. *Drug Des Devel Ther.* 2011 Apr 5;7:267-78. doi: 10.2147/DDDT.S31443. PMID: 23630412; PMCID: PMC3623549.
2. Son JW, Kim S. Comprehensive Review of Current and Upcoming Anti-Obesity Drugs. *Diabetes Metab J.* 2020 Dec;44(6):802-818. doi: 10.4093/dmj.2020.0258. Epub 2020 Dec 23. PMID: 33389955; PMCID: PMC7801751.

3. Kim KK, Cho HJ, Kang HC, Youn BB, Lee KR. Effects on weight reduction and safety of short-term phentermine administration in Korean obese people. *Yonsei Med J.* 2006 Oct 31;47(5):614-25. doi: 10.3349/ymj.2006.47.5.614. PMID: 17066505; PMCID: PMC2687747.
4. Taylor C, Fricker AD, Devi LA, Gomes I. Mechanisms of action of antidepressants: from neurotransmitter systems to signaling pathways. *Cell Signal.* 2005 May;17(5):549-57. doi: 10.1016/j.cellsig.2004.12.007. PMID: 15683730; PMCID: PMC3581018.
5. Lavretsky H, Reinlieb M, St Cyr N, Siddarth P, Ercoli LM, Senturk D. Citalopram, methylphenidate, or their combination in geriatric depression: a randomized, double-blind, placebo-controlled trial. *Am J Psychiatry.* 2015 Jun;172(6):561-9. doi: 10.1176/appi.ajp.2014.14070889. Epub 2015 Feb 13. PMID: 25677354; PMCID: PMC4451432.
6. Nelson JC. The role of stimulants in late-life depression. *Am J Psychiatry.* 2015 Jun;172(6):505-7. doi: 10.1176/appi.ajp.2015.15030356. PMID: 26029800.

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Abstract 22-2-05

Title: Application of Pharmacogenetics in Pediatric Psychiatry

Presenting Author: Bethany Langner, MS-3, UABHSOM

Additional Authors: Gabrielle Willhelm MS-4, Janaki Nimmagadda, MD: UABHSOM

Introduction/Background: The use of pharmacogenetics in clinical psychiatry practice has become increasingly common, especially in treatment-resistant patients. Prior studies in children have shown that certain gene polymorphisms are linked to a higher incidence of undesirable side effects.¹ In this case report, we will discuss a case of utilizing pharmacogenetics in a pediatric patient with a history of treatment resistance and severe side effects.

Description: A 19-year-old otherwise healthy female initially presented two years ago for symptoms of anxiety and depression. Her symptoms were resistant to treatment despite multiple medication trials. She was also later diagnosed with ADHD and Tic Disorder.

Her results showed mutations in the Solute Carrier Family 6 member 4 (SLC6A4) gene, which encodes an integral membrane protein that transports the neurotransmitter serotonin from synaptic spaces into presynaptic neurons. The patient also had reduced activity of Cytochrome P450 Family 2 subfamily B member 6 (CYP2B6*6) allele, resulting in an intermediate metabolizer phenotype. She had a duplication of the Cytochrome P450 Family 2 subfamily D member 6 (CYP2D6) gene, classifying her as an “ultrarapid metabolizer,” which occurs in roughly 1-2% of the population.²

The CYP2D6 gene product is responsible for metabolism of many different psychiatric medications, including SSRIs, tricyclic antidepressants, antipsychotics, and anxiolytics. This left the patient with limited therapeutic options.

Discussion and Conclusion: Clinicians should keep in mind that while pharmacokinetic genes like CYP2D6 affect drug metabolism, lack of drug-gene interaction does not necessarily predict clinical response.³ For example, Oxcarbazepine and Desvenlafaxine did not offer any positive effects even though they were in the “Use as Directed” category in this patient’s gene studies. More research should be done to determine clinical efficacy of pharmacogenetics in pediatric populations.

References:

1. Wehry AM, Ramsey L, Dulemba SE, Mossman SA, Strawn JR. Pharmacogenomic Testing in Child and Adolescent Psychiatry: An Evidence-Based Review. *Curr Probl Pediatr Adolesc Health Care*. 2018;48(2):40-49. doi:10.1016/j.cppeds.2017.12.003
2. Pratt VM, Scott SA, Pirmohamed M, Esquivel B, Kane MS, Kattman BL, Malheiro AJ, editors. Medical Genetics Summaries [Internet]. Bethesda (MD): National Center for Biotechnology Information (US); 2012–. Codeine Therapy and CYP2D6 Genotype. 2012 Sep 20 [updated 2021 Mar 30]. PMID: 28520350.
3. Poolsup N, Li Wan Po A, Knight TL. Pharmacogenetics and psychopharmacotherapy. *J Clin Pharm Ther*. 2000 Jun;25(3):197-220. doi: 10.1046/j.1365-2710.2000.00281.x. PMID: 10886465.

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Abstract 22-2-06

Title: Semaglutide as a Possible Therapy in Binge Eating Disorder?

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

Introduction/Background: Binge eating disorder (BED) is the most common eating disorder among adults in the United States[1]. The multifactorial etiology of BED makes it the most frequent eating disorder associated with significant physical and psychological comorbidities[2,3]. Despite this fact, less than half of those diagnosed with BED seek treatment[1]. First line treatment for BED is cognitive behavioral therapy[3]. First line pharmacotherapy is topiramate, followed by lisdexamfetamine[3]. The optimal treatment regimen for BED is currently unknown and likely highly individualized due to the complex etiology of the illness.

Description: The patient is a 50-year-old female with a history of binge eating disorder, generalized anxiety disorder, insomnia, major depressive disorder, attention-deficit/hyperactivity disorder, and morbid obesity (BMI > 40). Treatment attempts to control weight and binge eating included sleeve gastrectomy, Adderall, then Vyvanse (lisdexamfetamine). Her biggest stressor is work and maintaining boundaries associated with her work hours. After work, she struggles to maintain motivation to care for her children. Vyvanse has aided her in curbing day-time binges, however, when it wears off in the evenings she experiences significant amounts of anxiety, resulting in uncontrollable binges. In March 2022, the patient began Wegovy (semaglutide) weight loss medication. She reported that the medication has helped curb her binge eating episodes and she had gained little weight since beginning the regimen. When she presented 3 months later, she reported she had lost 50lbs, was better managing work related stress, and even enjoyed a weekend out with her family. The patient attributes this significant improvement to the semaglutide.

Discussion and Conclusion: Few options are available for patients struggling with binge-eating disorder. First line treatment is psychotherapy. For those with little access to psychotherapy, pharmacotherapy becomes the mainstay of treatment. The ideal treatment for BED also addresses a patient's individual and environmental risk factors. Semaglutide is a GLP-1 (glucagon-like peptide 1) receptor agonist, which works on the brain to reduced appetite, palatability, and food intake[4]. The SUSTAIN-6 (Semaglutide Unabated Sustainability in Treatment of Type 2 Diabetes) trial demonstrated that semaglutide lowered the risk of death from cardiovascular causes by 26%[5]. This could prove highly beneficial in a population suffering from multiple comorbidities. Semaglutide can be used for long term weight management[4]. Similar medications could prove to be efficacious in managing BED, however, more research is needed in this population.

References:

1. Hudson, J. I., Hiripi, E., Pope, H. G., Jr, & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biological psychiatry*, 61(3), 348–358. <https://doi.org/10.1016/j.biopsych.2006.03.040>

2. Hilbert A. Binge-Eating Disorder. *Psychiatr Clin North Am.* 2019 Mar;42(1):33-43. doi: 10.1016/j.psc.2018.10.011. Epub 2018 Dec 22. PMID: 30704638.
3. Agüera, Z., Lozano-Madrid, M., Mallorquí-Bagué, N. et al. A review of binge eating disorder and obesity. *Neuropsychiatr* 35, 57–67 (2021). <https://doi.org/10.1007/s40211-020-00346-w>
4. Singh G, Krauthamer M, Bjalme-Evans M. Wegovy (semaglutide): a new weight loss drug for chronic weight management. *J Investig Med.* 2022 Jan;70(1):5-13. doi: 10.1136/jim-2021-001952. Epub 2021 Oct 27. PMID: 34706925; PMCID: PMC8717485.
5. Husain M, Birkenfeld AL, Donsmark M, Dungan K, Eliaschewitz FG, Franco DR, Jeppesen OK, Lingvay I, Mosenzon O, Pedersen SD, Tack CJ, Thomsen M, Vilsbøll T, Warren ML, Bain SC; PIONEER 6 Investigators. Oral Semaglutide and Cardiovascular Outcomes in Patients with Type 2 Diabetes. *N Engl J Med.* 2019 Aug 29;381(9):841-851. doi: 10.1056/NEJMoa1901118. Epub 2019 Jun 11. PMID: 31185157.

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Abstract 22-2-07

Title: Transcranial Magnetic Stimulation Treatment for Medication Resistant Depression

Presenting Author: Audrey Driver, 3, UAB Heersink School of Medicine

Additional Authors: Peyton Robison; Janaki Nimmagadda, M.D.; Anupama Yedla, M.D.; Clinton Martin M.D.: UAB Heersink School of Medicine, UABSOM Huntsville Campus, Department of Psychiatry

Introduction/Background: Approximately 30% of patients with Major Depressive Disorder have treatment-resistant depression (TRD). TRD is defined as depression that fails to respond to at least 2 or more pharmacological therapeutic trials. Several alternatives have been implicated for TRD. ECT, TMS, Nasal Ketamine, and others have shown efficacy in leading to remission of major depression. Of the many, Transcranial Magnetic Stimulation proves to be a very effective option with a 70 percent remission rate, minimal safety concerns, and more recently widespread affordability

Description: 45-year-old woman with chronic depression, that has been treatment resistant to SSRIs, newer antidepressants combination with mood stabilizers. She received 24 treatments of TMS and is being managed on maintenance treatment of combination SSRI medication and mood stabilizer.

Discussion and Conclusion: TMS modulates neural circuit activity in the prefrontal cerebral cortex. It works in an excitatory or inhibitory fashion by providing magnetic pulses of high or low frequencies to a specific area of the brain. For depression, an excitatory, higher frequency such as 10 Hz is used; a 1 Hz inhibitory frequency can be used to treat anxiety. For example, a full TMS treatment session for depression may consist of a 10 Hz frequency where 40 rapid pulses are delivered over 4 seconds repeatedly for 75 times every 11 seconds. While the functions and settings for TMS frequencies and pulses can vary, the goal is to stimulate deeper neurons in the brain which have been under functioning and contributing to depression. Contrary to ECT, TMS procedures do not require general anesthesia and only take approximately 30 minutes per session; thus, TMS can fit more easily into one's daily schedule. Compared to ECT, TMS has a milder side effect profile, mostly limited to mild tapping scalp pain or discomfort during treatment.

Standard patients achieve response, and often remission, after 30 sessions over the course of 6 weeks, but treatment is customizable to extend beyond that timeframe. In patients with concurrent depression and anxiety, TMS can be applied bilaterally and at varying frequencies to target both problems during each session. TMS is a cost-effective method for managing treatment-resistant depression and is covered by most insurances.

References:

1. Kverno KS, Mangano E. Treatment-Resistant Depression: Approaches to Treatment. *J Psychosoc Nurs Ment Health Serv.* 2021 Sep;59(9):7-11. doi: 10.3928/02793695-20210816-01. Epub 2021 Sep 1. PMID: 34459676.

2. Janicak PG, Dokucu ME. Transcranial magnetic stimulation for the treatment of major depression. *Neuropsychiatr Dis Treat*. 2015 Jun 26;11:1549-60. doi: 10.2147/NDT.S67477. PMID: 26170668; PMCID: PMC4492646.
3. Holtzheimer, MD, P. E. (2022, May 16). Technique for performing transcranial magnetic stimulation (TMS). [uptodate.com](https://www.uptodate.com).
4. Arns, M., Iseger, T., Spronk, D. B., Brown, T., & Fitzgerald, P. B. (2011). Repetitive Transcranial Magnetic Stimulation (rTMS) in Depression: Protocols, Mechanisms and New Developments. *Neurofeedback and Neuromodulation Techniques and Applications*. <https://doi.org/10.1016/B978-0-12-382235-2.00010-X>
5. Rossi, S., Hallett, M., Rossini, P. M., & Pascual-Leone, A. (2009). Safety, ethical considerations, and application guidelines for the use of transcranial magnetic stimulation in clinical practice and research. *Clinical Neurophysiology*, 120(12), 2008-2039.
6. <https://tmstherapyhuntsville.com/depression-treatments/>

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Abstract 22-2-08

Title: A case of Parkinson's Disease Psychosis treated with Pimavanserin, novel 5-HT_{2A/C} receptor inverse agonist

Presenting Author: Matthew Raymond, MS-3, University of Alabama in Birmingham Heersink School of Medicine

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Introduction/Background: Parkinson's disease related psychosis (PDP) can occur early or later in the course of the disease and is comprised of well-formed visual hallucinations, presence hallucinations, persecution delusions, and preservation of insight. It is important to differentiate PDP from other primary psychiatric disorders, specifically if the patient is experiencing auditory hallucinations. Initial treatment involves withdrawing or reducing dopaminergic medications and assuming the risk of worsening motor symptoms with this reduction. As second-generation antipsychotics, clozapine and quetiapine have been considered due to their lack of interaction with the D₂ receptor, despite the risk of extrapyramidal symptoms. However, the risk of agranulocytosis when treated with clozapine is considered too high and quetiapine has not been proven more efficacious than placebo. Pimavanserin was developed to be a specific 5-HT_{2A/C} receptor inverse agonist with no activity at the dopamine receptors.

Description: A 75-year-old woman was diagnosed with Parkinson's disease after family noticed the classic signs of shuffling gait, resting tremor, and bradykinesia. She was started on Sinemet 25-100 TID at this time. 8 years after this diagnosis, she began seeing children running throughout her house that she did not recognize. These occasional episodes increased to daily frequency over the course of months, and she was then started on Pimavanserin 34mg qd. After being on this medication for the last 3 years, her husband states that these episodes will occur at most once per month.

Discussion and Conclusion: PDP is a major sequela of Parkinson's disease with poorly understood pathophysiology. Targeting of the serotonergic system has shown benefit in PDP and other patients with psychotic symptoms. Second generation antipsychotics such as clozapine and quetiapine have been used in the past, despite the potential for worsening motor symptoms. Until recently, clozapine has been the best choice for PDP due to its effectiveness at lower doses. However, frequent blood monitoring remains a stumbling block for most patients. Pimavanserin is the only novel drug developed specifically to treat psychosis through the 5-HT_{2A/C} receptor. Pimavanserin has demonstrated efficacy in randomized, placebo-controlled trials without worsening of motor function.

References:

1. ACADIA Pharmaceuticals Inc. (2019, October 25). A Phase 2, Multicenter, Randomized, Double-blind, Placebo-controlled, Study to Evaluate the Efficacy and Safety of Adjunctive Pimavanserin in Major Depressive Disorder. Clinicaltrials.gov. <https://clinicaltrials.gov/ct2/show/NCT03018340?term=03018340&draw=2&rank=1>
2. Fava, M., Dirks, B., Freeman, M. P., Papakostas, G. I., Shelton, R. C., Thase, M. E., Trivedi, M. H., Liu, K., & Stankovic, S. (2019). A Phase 2, Randomized, Double-Blind, Placebo-Controlled Study of

Adjunctive Pimavanserin in Patients With Major Depressive Disorder and an Inadequate Response to Therapy (CLARITY). *The Journal of Clinical Psychiatry*, 80(6).
<https://doi.org/10.4088/jcp.19m12928>

3. Panchal, S. C., & Ondo, W. G. (2018). Treating Hallucinations and Delusions Associated With Parkinson's Disease Psychosis. *Current Psychiatry Reports*, 20(1). <https://doi.org/10.1007/s11920-018-0869-z>
4. Patel, R. S., Bhela, J., Tahir, M., Pisati, S. R., & Hossain, S. (2019). Pimavanserin in Parkinson's Disease-induced Psychosis: A Literature Review. *Cureus*. <https://doi.org/10.7759/cureus.5257>

APPA 2022 Fall Conference Student/Resident Abstracts

Abstract 22-2-09

Title: A Holistic Approach to Addressing Social Determinants of Health in the Pediatric Population

Presenting Author: Chase Craven, MS-3, University of Alabama Heersink School of Medicine - Huntsville

Additional Authors: Janaki Nimmagadda, M.D., University of Alabama Heersink School of Medicine - Huntsville

Introduction/Background: The Social Determinants of Health (SDoH) are defined by the World Health Organization as “the conditions by which people are born, grow, live, work and age.” [1] Most studies on the Social Determinants of Health are performed with adult populations in mind, with results extrapolated to the pediatric population. Strong evidence exists worldwide that individuals in poverty, with poor education, and with increased Adverse Childhood Experiences (ACE) have more health problems and die earlier than those with more income, more education, and fewer ACE’s[2,3] . In this report, we will discuss the use of clinic-based psychosocial screening tools and referral systems to assess the Social Determinants of Health in a pediatric population.

Description: An 18 YOF with diagnosis of Autism Spectrum Disorder comes into clinic with her mother for behavioral concerns. Mother is an unemployed single parent. Father died several years ago secondary to mental illness. Mother has had difficulty with transportation. She has quit her job to take care of her daughter due to multiple behavioral concerns which have led to mother picking daughter up from school multiple times. The patient has recently qualified to receive disability benefits, which has helped the patient have access to better food and plan a few activities outside the home.

Discussion and Conclusion: Increased number of ACE’s are associated with premature mortality[3]. The simple screening question “Do you ever have difficulty making ends meet at the end of the month?” is 98% sensitive and 64% specific for identifying patients living below the poverty line[4].

Clinic-based referral systems have been shown to increase mothers’ access to childcare, fuel assistance programs, and employment[5]. Research indicates that the SDoH have an 80% higher impact on population health than healthcare. Data shows that a higher ratio of social service spending versus healthcare spending results in improved population health.[4] Screening and referring to appropriate resources indirectly improves patient’s health outcomes by assessing the SDoH.

In conclusion, psychosocial screening tools and referral systems are an effective method to assist providers in addressing SDoH in the pediatric population. More research should be performed on health outcomes of clinic-based social intervention in the pediatric population.

References:

1. “Social Determinants of Health.” World Health Organization, World Health Organization, https://www.who.int/health-topics/social-determinants-of-health#tab=tab_1
2. “Closing the Gap in a Generation: Health Equity through Action on the Social Determinants of Health.” Child: Care, Health and Development, vol. 35, no. 2, 2009, pp. 285–286.,

3. Brown, David W., et al. "Adverse Childhood Experiences and the Risk of Premature Mortality." *American Journal of Preventive Medicine*, vol. 37, no. 5, 2009, pp. 389–396.,
4. Brcic V, Eberdt C, Kaczorowski J. Development of a tool to identify poverty in a family practice setting: a pilot study. *Int J Family Med*. 2011;2011:812182. doi: 10.1155/2011/812182. Epub 2011 May 26. Erratum in: *Int J Family Med*. 2015;2015:418125. PMID: 22312547; PMCID: PMC3268233.
5. Hood CM, Gennuso KP, Swain GR, Catlin BB. County Health Rankings: Relationships Between Determinant Factors and Health Outcomes. *Am J Prev Med*. 2016;50(2):129-135. doi:10.1016/j.amepre.2015.08.024

APPA 2022 Fall Conference Student/Resident Abstracts

Abstract 22-2-10

Title: The Chicken and the Egg: Overlapping Major Depressive Disorder, Vascular Dementia, and Obstructive Sleep Apnea

Presenting Author: Danielle Glinka, OMS-3, Alabama College of Osteopathic Medicine

Additional Authors: Aneri Desai, OMS-3, ACOM; Mark Haygood, DO, ACOM

Introduction/Background: Major Depressive Disorder (MDD) can have multiple etiologies leading to the disease. In particular, 25-30% of ischemic strokes have been linked to immediate or delayed vascular dementia with depression being one of the most common psychological symptoms.^{1,2} Obstructive sleep apnea has also demonstrated significant overlap with depressive symptoms and decreased attention, working memory, and executive functions.³

When a patient presents with multiple etiologies that could contribute to MDD, it becomes difficult to find a treatment protocol. The literature lacks an approach to these patients, which makes treatment options reliant on anecdotal and case series reports.

Description: In this case, a 76-year-old male presented to the clinic with concerns of excessive sleep greater than sixteen hours per day, weight loss, severe anhedonia, guilt, poor appetite, lack of motivation, and fatigue. His past medical history was significant for a transient ischemic attack, vascular type degenerative dementia, obstructive sleep apnea, hypertension, and recurrent major depressive disorder. PHQ-9, GAD-7, and MMSE scores were 16/27, 6/21, and 27/30, respectively.

Mental status examination revealed a well-nourished and appropriately dressed male. Muscle strength and tone were both 5/5. Patient was alert and oriented to person, place, time and situation, except for date and day of the week. Patient's speech was slow with sparse content. He reports his mood as "mad". He had restricted affect with mood congruence. Attention, concentration, immediate memory, and remote memory were intact.

Results from an MRI without contrast demonstrated extensive white matter disease in both cerebral hemispheres. Results from a sleep study demonstrated moderate sleep apnea with no REM sleep. The patient was started on Trintillex 5mg daily and given education on sleep hygiene. He also obtained a CPAP machine.

Discussion and Conclusion: In the case of multifactorial depression, the literature lacks a treatment algorithm to follow. This case report highlights a documented treatment approach in these patients who have multiple etiologies contributing to the disease. Further reporting of treatment outcomes associated with OSA and vascular dementia would be helpful to evaluate effective treatment strategies for multifactorial disease presentations.

References:

1. Kalaria RN, Akinyemi R, Ihara M. Stroke injury, cognitive impairment and vascular dementia. *Biochim Biophys Acta.* 2016;1862(5):915-925. doi:10.1016/j.bbadis.2016.01.015

2. Deardorff WJ, Grossberg GT. Behavioral and psychological symptoms in Alzheimer's dementia and vascular dementia. *Handb Clin Neurol.* 2019;165:5-32. doi:10.1016/B978-0-444-64012-3.00002-2
3. Vanek J, Prasko J, Genzor S, et al. Obstructive sleep apnea, depression and cognitive impairment. *Sleep Med.* 2020;72:50-58. doi:10.1016/j.sleep.2020.03.017

APPA 2022 Fall Conference Student/Resident Abstracts

Abstract 22-2-11

Title: Lance-Adams Syndrome in a psychiatric patient

Presenting Author: Hunter Soleymani, MS-3, UAB Heersink School of Medicine - Huntsville

Additional Author: Tamara Zaza, MS-3; Tarak Vasavada, MD; Clinton Martin, MD: UAB Heersink School of Medicine - Huntsville

Introduction/Background: Post-hypoxic myoclonus (PHM) is characterized by myoclonic jerks following an episode of hypoxia. The myoclonic jerks may be mistaken for antipsychotic-induced extrapyramidal symptoms (EPS) in patients taking these medications. While EPS can present as myoclonus, chronic PHM, or Lance-Adams Syndrome (LAS), presents days to weeks following hypoxia and cardiopulmonary resuscitation. We present a patient who was initially evaluated for antipsychotic-induced EPS, but was ultimately diagnosed with LAS.

Description: A 64-year-old male with vocal cord near-paralysis and delusion disorder was admitted to the medical floor for worsening stridor and dyspnea. He was also noted to have bizarre delusions, therefore treatment with risperidone was initiated. On hospital day 3, he experienced cardiac arrest secondary to laryngospasm. Hours after resuscitation, lip smacking and facial twitching were noted, which progressed over the next few hours to intermittent rhythmic extension of bilateral upper and lower extremities. Psychiatry and neurology were consulted to evaluate for EPS/seizures/PHM. Antipsychotic treatment was discontinued due to the patient being sedated with no psychiatric disturbances and to evaluate for antipsychotic-induced EPS. Continuous EEG showed diffuse slow background activity intermixed with burst suppression activity, but no obvious seizure activity. MRI showed no acute findings. Over the next few days, the patient's movements progressed to generalized action myoclonus suggestive of LAS. When the patient was weaned off sedation severe bizarre delusions were noted, so antipsychotic treatment was resumed.

Discussion and Conclusion: LAS is a chronic form of post-hypoxic myoclonus that typically occurs days to weeks following a hypoxic episode. The myoclonic jerks are triggered by purposeful movement, tactile stimulation, and startle, and they disappear with relaxation of the body and sleep (1). The neurotransmitters involved include serotonin and gamma-aminobutyric acid (GABA) (2,3). Drugs typically used for symptomatic control of LAS include sodium valproate, clonazepam, piracetam, levetiracetam, and zonisamide (4). Trials of valproate, clonazepam, and levetiracetam mildly improved our patient's myoclonus, but were ineffective in eliminating the jerks. Since EPS was ruled out, reinitiation of antipsychotic therapy was crucial to avoid worsening delusions. Knowledge of LAS is important amongst psychiatrists to avoid discontinuation of necessary antipsychotic treatment in patients with myoclonic jerks mistaken for EPS.

References:

1. Zhang YX, Liu JR, Jiang B, Liu HQ, Ding MP, Song SJ, Zhang BR, Zhang H, Xu B, Chen HH, Wang ZJ, Huang JZ. Lance-Adams syndrome: a report of two cases. *J Zhejiang Univ Sci B*. 2007 Oct;8(10):715-20.

2. Welsh JP, Placantonakis DG, Warsetsy SI, Marquez RG, Bernstein L, Aicher SA. The serotonin hypothesis of myoclonus from the perspective of neuronal rhythmicity. *Adv Neurol.* 2002;89:307–29.
3. Matsumoto RR, Truong DD, Nguyen KD, Dang AT, Hoang TT, Vo PQ, et al. Involvement of GABA(A) receptors in myoclonus. *Mov Disord.* 2000;15(Suppl1):47–52.
4. Malhotra S, Mohinder K. Lance-Adams syndrome: Difficulties surrounding diagnosis, prognostication, and treatment after cardiac arrest. *Anesth Essays Res* 2012;6:218-22

APPA 2022 Fall Conference Student/Resident Abstracts

Abstract 22-2-12

Title: Naltrexone Use in Adolescents to Decrease Incidences of Self-Harm

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

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

Introduction/Background: Nonsuicidal self-injury (NSSI) is associated with a wide variety of externalizing and internalizing conditions.¹ The NSSI rate in adolescents is around 13% to 23%.^{1,2} NSSI most commonly includes cutting and/or burning oneself without suicidal intent³. It is Generally seen in mid-adolescent years and is often impulsive, with no planning involved.⁴ NSSI can have addictive characteristics, making it difficult for patients to discontinue once started.⁵ It is primarily associated with developmental disabilities, eating disorders, and borderline personality traits/disorder.⁶ NSSI is a common psychiatric symptom that is seen in many different psychiatric diagnoses.⁷ Patients with NSSI have been noted to have elevated rates of emotional reactivity, intensity, and hyperarousability.¹ It has been hypothesized that Naltrexone can inhibit endorphins and decrease addictive potential of recurrent NSSI.⁸

Description: A 15-year-old female presents for a new-patient visit. She has been diagnosed with Bipolar disorder, Disruptive Mood Dysregulation Disorder, and Borderline Personality disorder by an outside institution. She is currently taking Lithium (300mg) once a day at night, Amitriptyline (25mg) once a day at night.

She began taking Naltrexone after her first hospitalization for self-injurious behaviors. She tried a maximum dose of 50mg for one and a half years. According to patient and mother, they did not see any significant difference using Naltrexone for these self-injurious behaviors. However, she continued to have ongoing stressors during this time and was hospitalized 3 other times over the past year.

After her initial visit, our working diagnoses are Attention Deficit Hyperactivity Disorder (ADHD), Disruptive behavior disorder, and sleep disturbances. We have discontinued her Lithium due to noncompliance and lack of response. Our plan is to start stimulants to help with her ADHD symptoms at her next visit along with substituting her Amitriptyline with Clonidine to help with her sleep disturbances. Naltrexone can still be considered as an option in the future depending on her treatment response and compliance during subsequent visits.

Discussion and Conclusion: The primary mechanism of action for naltrexone involves opioid receptor antagonism.⁹ One recent study shows that the Naltrexone opioid blockade decreased the most predominant type of self-injurious behavior, which was head-banging in this study, without having a significant effect on heart rate or blood pressure.¹⁰ Another study consisting of 155 children showed that 98 of the 155 children (77%) had a statistically significant improvement in irritability and hyperactivity with the use of Naltrexone.¹¹ Since patients with NSSI have been noted to have elevated rates of emotional reactivity, intensity, and hyperarousability, Naltrexone is a plausible solution to this issue.¹ It has been shown that beta-endorphins immediately before NSSI injury are significantly lower than after.¹² By antagonizing the opioid receptor, the beta-endorphins that are produced from self-harm cannot create the necessary euphoric effect to reinforce NSSI, thus leading to less self-harming behavior.

References:

1. Peterson J, Freedenthal S, Sheldon C, Andersen R. Nonsuicidal Self injury in Adolescents. *Psychiatry (Edgmont)*. 2008 Nov;5(11):20-6. PMID: 19724714; PMCID: PMC2695720.
2. Jacobson CM, Gould M. The epidemiology and phenomenology of non-suicidal self-injurious behavior among adolescents: a critical review of the literature. *Arch Suicide Res*. 2007;11(2):129-47. doi: 10.1080/13811110701247602. PMID: 17453692.
3. Muehlenkamp JJ, Gutierrez PM. An investigation of differences between self-injurious behavior and suicide attempts in a sample of adolescents. *Suicide Life Threat Behav*. 2004 Spring;34(1):12-23. doi: 10.1521/suli.34.1.12.27769. PMID: 15106884.
4. Rodham K, Hawton K, Evans E. Reasons for deliberate self-harm: comparison of self-poisoners and self-cutters in a community sample of adolescents. *J Am Acad Child Adolesc Psychiatry*. 2004 Jan;43(1):80-7. doi: 10.1097/00004583-200401000-00017. PMID: 14691363.
5. Nixon MK, Cloutier PF, Aggarwal S. Affect regulation and addictive aspects of repetitive self-injury in hospitalized adolescents. *J Am Acad Child Adolesc Psychiatry*. 2002 Nov;41(11):1333-41. doi: 10.1097/00004583-200211000-00015. PMID: 12410076.
6. Kahng S, Iwata BA, Lewin AB. Behavioral treatment of self-injury, 1964 to 2000. *Am J Ment Retard*. 2002 May;107(3):212-21. doi: 10.1352/0895-8017(2002)107<0212:BTOSIT>2.0.CO;2. PMID: 11966334.
7. Gollust SE, Eisenberg D, Golberstein E. Prevalence and correlates of self-injury among university students. *J Am Coll Health*. 2008 Mar-Apr;56(5):491-8. doi: 10.3200/JACH.56.5.491-498. PMID: 18400660.
8. Campbell M, Anderson LT, Small AM, Adams P, Gonzalez NM, Ernst M. Naltrexone in autistic children: behavioral symptoms and attentional learning. *J Am Acad Child Adolesc Psychiatry*. 1993 Nov;32(6):1283-91. doi: 10.1097/00004583-199311000-00024. PMID: 8282676.
9. Wang ZY, Guo LK, Han X, Song R, Dong GM, Ma CM, Wu N, Li J. Naltrexone attenuates methamphetamine-induced behavioral sensitization and conditioned place preference in mice. *Behav Brain Res*. 2021 Feb 5;399:112971. doi: 10.1016/j.bbr.2020.112971. Epub 2020 Oct 17. PMID: 33075396.
10. Herman BH, Hammock MK, Egan J, Arthur-Smith A, Chatoor I, Werner A. Role for opioid peptides in self-injurious behavior: dissociation from autonomic nervous system functioning. *Dev Pharmacol Ther*. 1989;12(2):81-9. PMID: 2714161.
11. Roy A, Roy M, Deb S, Unwin G, Roy A. Are opioid antagonists effective in attenuating the core symptoms of autism spectrum conditions in children: a systematic review. *J Intellect Disabil Res*. 2015 Apr;59(4):293-306. doi: 10.1111/jir.12122. Epub 2014 Mar 4. PMID: 24589346.
12. Störkel LM, Karabatsiakos A, Hepp J, Kolassa IT, Schmahl C, Niedtfeld I. Salivary beta-endorphin in nonsuicidal self-injury: an ambulatory assessment study. *Neuropsychopharmacology*. 2021 Jun;46(7):1357-1363. doi: 10.1038/s41386-020-00914-2. Epub 2021 Jan 4. PMID: 33398083; PMCID: PMC8134499.
13. Pew Charitable Trusts. (2016, November). Medication-assisted treatment improves outcomes for patients with opioid use disorder. Retrieved from <http://www.pewtrusts.org/en/research-and-analysis/fact-sheets/2016/11/medication-assisted-treatment-improves-outcomes-for-patients-with-opioid-use-disorder>

APPA 2022 Fall Conference Student/Resident Abstracts

Abstract 22-2-13

Title: Race Against Time: A Case Report of Multidisciplinary Treatment of Functional Dysphagia

Presenting Author: Jonathan Black, PGY2, UAB Psychiatry

Additional Authors: Melissa Greenfield, UAB; Andrea Arevalo, UAB; Pamela Parker, UAB

Introduction/Background: Functional Neurological Symptom Disorder (FNSD), or Conversion Disorder, is characterized by symptoms of altered voluntary movement or sensation without explanation. The etiology of FNSD is unclear, and treatments are limited. Studies have examined neuroimaging, neuropsychological mechanisms, and biopsychosocial factors to better understand FNSD. Progress in treatments have centered on behavioral programs and quality interdisciplinary care. In this report, we describe the multidisciplinary treatment of a patient with severe functional dysphagia.

Description: A 37-year-old female with MDD, PTSD, hypothyroidism, and a spinal cord injury was hospitalized for odynophagia with intolerance of PO intake, which started after being briefly intubated for anaphylaxis several months prior. She reported being unable to eat for several days. The patient was initially managed with fluids and intravenous opioids, and she used wall suction to eliminate saliva. The patient was evaluated by multiple medical services, and she underwent extensive imaging and several diagnostic procedures, all of which were normal. The patient reported increasing pain and requested higher doses of opioids. Psychiatry was then consulted, by which point the patient had not tolerated PO intake for 9 days.

Psychiatric evaluation revealed a significant need for control, alexithymia, and a history of working as a nurse. We offered olanzapine, which provoked anaphylaxis. Psychology developed a behavioral plan to target her symptoms. The patient demanded placement of a PEG tube, and she continued to deteriorate. On day 12 without PO intake, a sitter was added, and psychology worked with speech therapy to perform FEES and implement the behavioral plan. Within 24 hours, the patient improved and was discharged.

Discussion and Conclusion: The patient improved with a behavioral plan implemented by a multidisciplinary team, which required careful communication and coordination. The plan was designed to uncouple conditioned responses by eliciting unconscious physiological reflexes. Successful management was time-sensitive to avoid medical deterioration, unnecessary procedures, and over-utilization of resources. The diagnosis of FNSD in this patient was complicated by events that raised suspicion for Factitious Disorder. However, literature suggests that features of somatoform disorders overlap and that the sick role and secondary gain can reinforce symptoms of FNSD.

References:

1. American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.). <https://doi.org/10.1176/appi.books.9780890425596>
2. Fobian, A. D., & Elliott, L. (2019). A review of functional neurological symptom disorder etiology and the integrated etiological summary model. *Journal of Psychiatry & Neuroscience*, 44(1), 8–18. <https://doi.org/10.1503/jpn.170190>

3. Hallett, M., Aybek, S., Dworetzky, B. A., McWhirter, L., Staab, J. P., & Stone, J. (2022). Functional neurological disorder: New subtypes and shared mechanisms. *The Lancet Neurology*, 21(6), 537–550. [https://doi.org/10.1016/S1474-4422\(21\)00422-1](https://doi.org/10.1016/S1474-4422(21)00422-1)
4. Suntrup, S., Teismann, I., Wollbrink, A., Warnecke, T., Winkels, M., Pantev, C., & Dziewas, R. (2014). Altered Cortical Swallowing Processing in Patients with Functional Dysphagia: A Preliminary Study. *PLoS ONE*, 9(2), e89665. <https://doi.org/10.1371/journal.pone.0089665>
5. Fobian, A. D., Long, D. M., & Szaflarski, J. P. (2020). Retraining and control therapy for pediatric psychogenic non-epileptic seizures. *Annals of Clinical and Translational Neurology*, 7(8), 1410–1419. <https://doi.org/10.1002/acn3.51138>
6. Kim, Y., Han, S.H., Shin, Y.B., Yoon, J.A., & Kim, S.H. (2021). Diagnosis and successful visual biofeedback therapy using fiberoptic endoscopic evaluation of swallowing in a young adult patient with psychogenic dysphagia: a case report. *Journal of Yeungnam Medical Science*, <https://doi.org/10.12701/yujm.2021.01543>

APPA 2022 Fall Conference Student/Resident Abstracts

Abstract 22-2-14

Title: AMS as a Manifestation of MELAS: A Case Report

Presenting Author: Thomas Kozar, MS-3, UAB Heersink School of Medicine

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Introduction/Background: Altered mental status (AMS) has a broad etiology including metabolic, infectious, toxic, structural, and psychiatric causes. Occasionally, investigations into these etiologies will fail to identify the cause of AMS requiring psychiatrists to consider less common sources of AMS, including mitochondrial disorders. This case report aims to illustrate the presentation and treatment challenges of one AMS patient with Mitochondrial Encephalopathy, Lactic Acidosis, and Stroke like Episodes (MELAS).

Description: The patient is a 55-year-old female who was admitted to Huntsville Hospital due to AMS. Prior to this admission, she displayed signs of confusion and agitation on an international flight and was evaluated for seizures at an outside facility before being discharged. Three days later, she had worsening encephalopathy and presented for re-evaluation. Her medical history is significant for stroke three years prior, resulting in intermittent confusion and language difficulties. Family history revealed that her daughter was diagnosed with MELAS one year ago. The patient was found to have an anion gap metabolic acidosis secondary to lactic acidosis and was empirically started on L-arginine and L-carnitine infusions. During her 11-day admission, she became increasingly disoriented, agitated, and combative with multiple elopement attempts. She would not answer questions during interviews, instead becoming echolalic or silent. Subsequently, she began refusing oral intake and medications, and her family ultimately decided to pursue hospice care.

Discussion and Conclusion: Management of AMS secondary to MELAS presents several challenges. Since the underlying etiology of AMS in these cases is a mitochondrial disorder, the AMS is not curable with traditional antipsychotics. Both first- and second-generation antipsychotics have been shown to worsen mitochondrial function via disruption of the electron transport chain and production of oxygen free-radicals, leading to further cellular damage. Therefore, the approach for treatment of AMS in this population should focus on maximizing cognitive ability. The best therapies to achieve this are L-arginine and L-carnitine infusions to resolve lactic acidosis, low-dose clonazepam for acute agitation, and stable, structured environments to aid with delirium. Though these measures allow patients to safely receive care, they do not significantly improve AMS, underscoring the challenge these patients present to behavioral health physicians.

References:

Casademont J, Garrabou G, Miró O, López S, Pons A, Bernardo M, Cardellach F. Neuroleptic treatment effect on mitochondrial electron transport chain: peripheral blood mononuclear cells analysis in psychotic patients. *J Clin Psychopharmacol.* 2007 Jun;27(3):284-8. doi: 10.1097/JCP.0b013e318054753e. PMID: 17502776.

El-Hattab AW, Adesina AM, Jones J, Scaglia F. MELAS syndrome: Clinical manifestations, pathogenesis, and treatment options. *Mol Genet Metab.* 2015 Sep-Oct;116(1-2):4-12. doi: 10.1016/j.ymgme.2015.06.004. Epub 2015 Jun 15. PMID: 26095523.

Smith AT, Han JH. Altered Mental Status in the Emergency Department. *Semin Neurol.* 2019 Feb;39(1):5-19. doi: 10.1055/s-0038-1677035. Epub 2019 Feb 11. PMID: 30743288.

APPA 2022 Fall Conference Student/Resident Abstracts

Abstract 22-2-15

Title: Patient Selection for L-Methylfolate Supplementation in Treatment Resistant Depression

Presenting Author: Thomas Kozar, MS-3, UAB Heersink School of Medicine

Additional Authors: Zoey Duncan, BS¹, Hope Cain Lackey, BS¹, Anupama Yedla, M.D.², Janaki Nimmagadda, M.D.², Richard Shelton, M.D.², Clinton Martin, M.D.²

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Introduction/Background: Nearly, 30% of patients with depression will not experience remission after multiple trials of selective serotonin reuptake inhibitors (SSRIs), termed treatment resistant depression (TRD). In specific populations, those with methylenetetrahydrofolate reductase (MTHFR) mutations and obesity, it has been theorized that a deficiency in folate, a precursor in monoamine synthesis, could play a role TRD. Obesity increases inflammatory cytokines that stimulate nitric oxide synthase depleting stores of tetrahydrobiopterin (BH4) a downstream product of L-methylfolate in monoamine synthesis. This case report aims to illustrate the experience of one obese patient with TRD who responded positively to folate supplementation and characterize the current literature around patient selection for folate augmentation in TRD.

Description: A 47-year-old female with past medical history significant for MDD, GAD, colon cancer postresection, hypertension, hypercholesterolemia, and asthma failed to respond to almost all SSRI medications, several mood stabilizers including lithium, and several non-SSRI antidepressants. She also had a BMI of greater than 50. After augmenting her treatment regimen of desvenlafaxine and low-dose T3 with 7.5mg of L-Methylfolate daily, the patient reports a significant decrease in depressive symptoms without any major adverse effects.

Discussion and Conclusion: L-methylfolate has shown to be an effective supplemental treatment option for TRD in patients with folate deficiencies. The treatment has a mild side-effect profile mirroring that of placebo as well as a significantly lower cost than other TRD modalities such as transcranial magnetic stimulation. Additionally, literature has demonstrated that the treatment is effective in patients with a high likelihood of response: those with treatment failure with SSRI/SNRI medications and either ≥ 2 of MTHFR gene mutation, BMI ≥ 30 , or inflammatory conditions. Clinical trials have demonstrated a dose response with 15 mg/day of L-methylfolate outperforming 7.5 mg/day. Further studies are needed to determine optimal dosing parameters for clinical practice.

References:

Kverno KS, Mangano E. Treatment-Resistant Depression: Approaches to Treatment. J Psychosoc Nurs Ment Health Serv. 2021 Sep;59(9):7-11. doi: 10.3928/02793695-20210816-01. Epub 2021 Sep 1. PMID: 34459676.

Papakostas GI, Shelton RC, Zajecka JM, Etemad B, Rickels K, Clain A, Baer L, Dalton ED, Sacco GR, Schoenfeld D, Pencina M, Meisner A, Bottiglieri T, Nelson E, Mischoulon D, Alpert JE, Barbee JG, Zisook S, Fava M. L-methylfolate as adjunctive therapy for SSRI-resistant major depression: results of

two randomized, double-blind, parallel-sequential trials. *Am J Psychiatry*. 2012 Dec;169(12):1267-74. doi: 10.1176/appi.ajp.2012.11071114. PMID: 23212058.

Shelton RC, Pencina MJ, Barrentine LW, Ruiz JA, Fava M, Zajecka JM, Papakostas GI. Association of obesity and inflammatory marker levels on treatment outcome: results from a double-blind, randomized study of adjunctive L-methylfolate calcium in patients with MDD who are inadequate responders to SSRIs. *J Clin Psychiatry*. 2015 Dec;76(12):1635-41. doi: 10.4088/JCP.14m09587. PMID: 26613389.

Jain, R., Manning, S., & Cutler, A. (2020). Good, better, best: Clinical scenarios for the use of L-methylfolate in patients with MDD. *CNS Spectrums*, 25(6), 750-764. doi:10.1017/S1092852919001469

Dartois LL, Stutzman DL, Morrow M. L-methylfolate Augmentation to Antidepressants for Adolescents with Treatment-Resistant Depression: A Case Series. *J Child Adolesc Psychopharmacol*. 2019 Jun;29(5):386-391. doi: 10.1089/cap.2019.0006. Epub 2019 May 6. PMID: 31058543.

APPA 2022 Fall Conference Student/Resident Abstracts

Abstract 22-2-16

Title: Delta-8 induced Psychotic Exacerbation: A Case Series with a Review of Literature

Presenting Author: Chelsea Miller, PGY2, UAB Department of Psychiatry and Neurobiology

Additional Authors: Bradley G. Burk (UAB Department of Psychiatry and Neurobiology), Rachel E. Fargason (UAB Department of Psychiatry and Neurobiology), Badari Birur (UAB Department of Psychiatry and Neurobiology)

Introduction/Background: Cannabis ($\Delta 9$ -THC) is the most consumed illicit drug. The Agricultural Improvement Act of 2018 removed hemp, a strain of *Cannabis sativa*, as a controlled substance. This law allowed for the plant to be processed into its components, as long as these products contain less than 0.3% $\Delta 9$ -THC. As a result, a legal psychoactive substance, Delta-8-Tetrahydrocannabinol ($\Delta 8$ -THC), emerged in popularity in late 2020 and is readily available and on display in stores. An increasing number of patients admitted for psychiatric hospitalization report use, with limited literature on the effects.

Description: This case series describes three individual cases of patients who required admission to a university psychiatric hospital after regular use solely of $\Delta 8$ -THC. All three patients developed psychotic and paranoid symptoms concurrently with the use of $\Delta 8$ -THC and a severity exceeding their previous historical presentations. The presenting psychotic symptoms were also atypical for all three patients. New-onset violence and visual hallucinations were noted in two of the patients, one patient with no previous psychiatric history and one patient while on a therapeutic dose of his antipsychotic. In the third case, new onset of bizarre fixed delusions of puppies dissolving in the bathtub developed.

Discussion and Conclusion: A strong body of evidence correlates the use of $\Delta 9$ -THC with psychosis. Based on limited studies and increasing reports of temporal association between $\Delta 8$ -THC use and the development of psychotic symptoms, it appears $\Delta 8$ -THC may act at both the cannabinoid CB1 and CB2 receptors and have similar adverse psychiatric effects as $\Delta 9$ -THC. Physicians should be prepared to gather a specific history of $\Delta 8$ -THC use (which may be considered by patients harmless) and treat patients with $\Delta 8$ -THC-related intoxication and symptoms.

References:

1. Mehmedic Z, Chandra S, Slade D, et al. Potency trends of $\Delta 9$ -THC and other cannabinoids in confiscated cannabis preparations from 1993 to 2008. *Journal of forensic sciences*. 2010;55(5):1209-1217.
2. Abernethy A. Hemp Production and the 2018 Farm Bill. US Food and Drug Administration. 2019.
3. Johnson-Arbor K, Smolinske S. The current state of delta-8 THC. *The American journal of emergency medicine*. 2022;56:259-261.
4. Kruger JS, Kruger DJ. Delta-8-THC: Delta-9-THC's nicer younger sibling? *Journal of cannabis research*. 2022;4(1):1-8.
5. Volkow ND, Swanson JM, Evins AE, et al. Effects of cannabis use on human behavior, including cognition, motivation, and psychosis: a review. *JAMA psychiatry*. 2016;73(3):292-297.
6. Tagen M, Klumpers LE. Review of delta-8-tetrahydrocannabinol ($\Delta 8$ -THC): Comparative pharmacology with $\Delta 9$ -THC. *British journal of pharmacology*. 2022.

APPA 2022 Fall Conference Student/Resident Abstracts

Abstract 22-2-17

Title: Whose Personality is it Anyway? A Case Report on Dissociative Identity Disorder

Presenting Author: Taylor Carter, MS-3, Virginia College of Osteopathic Medicine at Auburn

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Introduction/Background: According to the DSM-5, dissociative identity disorder is characterized by a disruption of identity characterized by two or more distinct personality states and gaps in the recall of events and personal information beyond ordinary forgetting. The symptoms cannot be due to a broadly accepted culture or religious practice (1). Dissociative identity disorder (DID) has been highly correlated to traumatic life experiences, with about 90% of the people diagnosed with DID having experienced various types of abuse and neglect during childhood (1). DID is highly associated with suicide and suicide attempts, and studies show that over 70% of patients diagnosed with DID attempt suicide at some point during their life (1). The current treatment guidelines for dissociative identity disorder may be inefficient by neglecting the psychosocial aspect of treatment (2) (3).

Description: The patient is a 23-year-old female with long standing history of mental illness who presented for psychiatric care at a behavioral health crisis center for suicidal ideation. The patient has a history of trauma and multiple previous psychiatric diagnoses, including dissociative identity disorder, PTSD, anxiety with panic attacks, bipolar disorder, major depressive disorder, and borderline personality disorder. The patient presented after increased suicidal ideation and recent attempt due to the passing of her father. She stated that having dissociative identity disorder is sometime distressing but is mostly protective as the patient “switches” between her multiple personalities in order to modify distressing mood states. The patient self-reported at least 6 identities, all with associated names, personalities, antics, patterns of speech, and specific behaviors. The patient logs these “switches” in her journal, and stated that they occur 2-3 times a day, every day. She also endorsed that the personalities can talk to each other, and that she will internally respond occasionally. The patient denies lapses in memory during any of these episodes. The patient has been resistant to various medications and was maintained on escitalopram, buspirone, and hydroxyzine at time of evaluation, but she reported that none of the medications were efficacious. We began safety planning with the patient on the day of admission, and the next day the patient left against medical advice due to feeling that her mood had improved and that she no longer had suicidal thoughts.

Discussion and Conclusion: Dissociative identity disorder (DID), previously referred to as multiple personality disorder, is a rare dissociative disorder characterized by at least two distinct personalities, and studies have shown that DID is highly correlated with traumatic life experiences (1). Dissociative identity disorder is a disorder with limited prevalence data (1), and therefore more research should be done to more accurately describe typical characteristics of this disorder. However, when a patient presents with dissociative identity disorder, the main focus in treatment should be aimed at addressing the underlying trauma with psychotherapy, reducing and monitoring suicide risk, and treating comorbid conditions with pharmacotherapy (2).

References:

1. American Psychiatric Association. (2013). Dissociative Disorders. In Diagnostic and statistical manual of mental disorders (5th ed.), 330-337.

2. Gleaves, D. H. (1996). The sociocognitive model of dissociative identity disorder: A reexamination of the evidence. *Psychological Bulletin*, 120(1), 42–59.
3. Bethany L. Brand, Richard J. Loewenstein & David Spiegel (2014) Dispelling Myths About Dissociative Identity Disorder Treatment: An Empirically Based Approach, *Psychiatry*, 77:2, 169-189.

APPA 2022 Fall Conference Student/Resident Abstracts

Abstract 22-2-18

Title: Renal Cell Carcinoma in Patients Treated with Second Generation Antipsychotics

Presenting Author: Morgan Malone, MD, PG2, Department of Psychiatry, Whiddon College of Medicine at the University of South Alabama

Additional Authors: Tyeler Rayburn, MD, Department of Psychiatry, Whiddon College of Medicine at the University of South Alabama; Mary Cohen-Colson, MD, Department of Psychiatry, Whiddon College of Medicine at the University of South Alabama

Introduction/Background: Renal cell carcinoma (RCC) is a rare neoplasm arising in the renal cortex. The incidence is of approximately 80,000 cases annually in the United States. Despite the advancements made in treatment over the past two decades, the 5-year survival is 76%. Some recent research has shown a rising incidence of CKD in patients treated with second generation antipsychotics which is a known risk factor to the development of RCC. Here we represent a case series of patients with RCC who have been treated with second generation antipsychotics.

Description: In this case series we reviewed six patient with renal cell carcinoma who had concurrent serious mental illness and prior and current treatment with second-generation antipsychotics. The patients were drawn from a psychiatric clinic with a census of approximately 1000 patients. Diagnoses included schizoaffective disorder, schizophrenia, and bipolar disorder. These patients were managed with varying second generation antipsychotics paliperidone, olanzapine, brexpiprazole, and clozapine. Additionally, patients demonstrated numerous risk factors for RCC prior to their diagnosis.

Discussion and Conclusion: It is certainly a possibility based on the amount of RCC cases found in such a small census size that there is a relationship between second generation antipsychotic use and increased risk of developing RCC. However, of the patients reviewed many demonstrated known risk factors for RCC. It is well known that psychiatric patients, due to the nature of their illness and side effects associated with medications, have a higher tendency to develop comorbid health conditions, of which many are additional risk factors for developing RCC. Data on the end-organ effects of long-term antipsychotic use is limited, and while we identify increased incidence of RCC in patients on long-term antipsychotics, research is necessary to reveal a causative link.

References:

Højlund, M., Lund, L. C., Herping, J. L. E., Haastrup, M. B., Damkier, P., & Henriksen, D. P. (2020). Second-generation antipsychotics and the risk of chronic kidney disease: a population-based case-control study. *BMJ open*, 10(8), e038247.

Wang, H. Y., Huang, C. L. C., Feng, I. J., & Tsuang, H. C. (2018). Second-generation antipsychotic medications and risk of chronic kidney disease in schizophrenia: population-based nested case-control study. *BMJ open*, 8(5), e019868.

Ljungberg, B., Campbell, S. C., Cho, H. Y., Jacqmin, D., Lee, J. E., Weikert, S., & Kiemeny, L. A. (2011). The epidemiology of renal cell carcinoma. *European urology*, 60(4), 615-621.

Felker, B., Yazel, J. J., & Short, D. (1996). Mortality and medical comorbidity among psychiatric patients: a review. *Psychiatric Services*.

Walker, E. R., McGee, R. E., & Druss, B. G. (2015). Mortality in mental disorders and global disease burden implications: a systematic review and meta-analysis. *JAMA psychiatry*, 72(4), 334-341.

APPA 2022 Fall Conference Student/Resident Abstracts

Abstract 22-2-19

Title: Selective Mutism in the Pediatric Population

Presenting Author: Evan Dixon, MS-4, University of South Alabama College of Medicine

Additional Authors: Sarah Bouslog, MS-4, Ingram Easter, MS-4, Shanthi Gatla, MD: University of South Alabama College of Medicine

Introduction/Background: Children with selective mutism are commonly diagnosed before age five. These patients often share characteristics with those who are diagnosed with social phobia and social anxiety disorder, such as shyness, anxiety, and social withdrawal. The overlap between diagnosis of selective mutism and other anxiety disorders in young children, coupled with the tendency for these disorders to be overlooked and undertreated, poses difficulty for families and clinicians to adequately identify characteristics of selective mutism.

Description: A six year old girl presented to the clinic with her mother for an initial psychiatric evaluation for chronic anxiety with accompanying mutism starting 10 months ago. The patient has a history of delayed developmental milestones, delayed speech, and asthma. On exam, the patient was mute, anxious with a flat affect, avoided eye contact, and did not engage with the examiner. Diagnosis of selective mutism and social anxiety by an outside psychologist one week prior to evaluation is reinforced. She was started on fluoxetine 5mg daily and tapered to 10mg daily, and she was encouraged to continue previously initiated psychotherapy. Three months after initiation of pharmacotherapy, the patient became verbal in multiple settings. On exam she had clear speech, was euthymic with full affect, and displayed appropriate eye contact.

Discussion and Conclusion: Selective mutism is defined as absent communication in one or more situations where the patient is expected to communicate in the absence of ASD, intellectual disability, knowledge deficit, deafnesses, and fluency issues in a person that is known to be able to successfully communicate. This patient began communicating at home and at school within two months of treatment. Healthcare providers may use clinical judgment in the risk-benefit analysis for use of medication to treat selective mutism in young patients, either alone or following failure of or in combination with psychotherapy.

References:

American Psychiatric Association. (2013). Anxiety Disorders. Diagnostic and statistical manual of mental disorders (5th ed.). https://doi.org/10.1176/appi.books.9780890425787.x05_Anxiety_Disorders

Letamendi AM, Chavira DA, Hitchcock CA, Roesch SC, Shipon-Blum E, Stein MB. Selective Mutism Questionnaire: measurement structure and validity. *J Am Acad Child Adolesc Psychiatry*. 2008 Oct;47(10):1197-1204. doi: 10.1097/CHI.0b013e3181825a7b. PMID: 18698268; PMCID: PMC2925837.

Chavira DA, Shipon-Blum E, Hitchcock C, Cohan S, Stein MB. Selective mutism and social anxiety disorder: all in the family? *J Am Acad Child Adolesc Psychiatry*. 2007 Nov;46(11):1464-72. doi: 10.1097/chi.0b013e318149366a. PMID: 18049296.

APPA 2022 Fall Conference Student/Resident Abstracts

Abstract 22-2-20

Title: Managing Sexual Violence in the Inpatient Psychiatric Setting

Presenting Author: Sarah Bouslog, MS-4, University of South Alabama College of Medicine

Additional Author: Tyeler Rayburn, PGY-1, University of South Alabama College of Medicine

Introduction/Background: Patients with mania and concurrent sexual disinhibition can be both victims and perpetrators of sexual violence. This represents disparate grounds to implement comprehensive policy to ensure the safety of both patients and staff. Only half of psychiatric institutions have formal policy for managing inpatient sexual violence. Likewise, there remains only a modest corpus of literature on this topic compared to other acts of aggression in the inpatient psychiatric setting. Here we present a case of a sexually-aggressive individual demonstrating an amalgam of clinical, ethical, and medico-legal questions.

Description: A 38 year old man with schizoaffective disorder was involuntarily committed to an inpatient psychiatric hospital for a manic episode with salient features of sexual indiscretions and grandiose and erotomanic delusions. Despite multiple medication trials, clozapine included, and through disparate hospitalizations over the last decade, this patient's delusions and disinhibition remained. While most of the presenting symptoms attenuated with time, the patient's sexual aggression, paucity of insight, and erroneous conviction of polyamory with non-consensual partners persisted, and indeed escalated to a psychologic and physically violent extent eliciting near constant pharmacologic restraint, isolation, and intensive supervision. This patient is awaiting placement in a long-term psychiatric facility for further evaluation and treatment.

Discussion and Conclusion: Adequate management of sexually violent individuals requires thorough supervision and a high staff to patient ratio, both of which are in short supply across a majority of the nation's psychiatric institutions; the mere presence of such patients on a ward generates pervasive anti-therapeutic countertransference among staff. Formal policies should be widespread, and should include protocol for documentation, reporting, prevention, and management of sexual violence. Meticulous documentation is crucial. Special incident reports should be thorough and customized for sexual violence events. And although a patient is hospitalized, and while staff may be habituated to a higher threshold of reaction to anomalous patient behavior, sexually violent acts always warrant law enforcement contact. Instances of sexual aggression and violence in the inpatient setting, both alleged and evident, should be unquestioned and seriously considered.

References:

Barnett, B. (2020). Addressing sexual violence in psychiatric facilities. *Psychiatric services*, 71(9), 959-961.

Lawn, T., & McDONALD, E. L. I. Z. A. B. E. T. H. (2009). Developing a policy to deal with sexual assault on psychiatric in-patient wards. *Psychiatric Bulletin*, 33(3), 108-111.

Wright, E. R., McCabe, H. A., & Koorman, H. E. (2012). Institutional capacity to respond to the ethical challenges of patient sexual expression in state psychiatric hospitals in the United States.

Weltens, I., Bak, M., Verhagen, S., Vandenberg, E., Domen, P., van Amelsvoort, T., & Drukker, M. (2021). Aggression on the psychiatric ward: Prevalence and risk factors. A systematic review of the literature. *PloS one*, 16(10), e0258346.

APPA 2022 Fall Conference Student/Resident Abstracts

Abstract 22-2-21

Title: The Trilemma of Today's Aging Population in the Time of Pandemic: A Case Study of Pre-existing Psychiatric Illness and Cognitive Deficits, COVID-19, and Further Cognitive Decline

Presenting Author: Mack Bozman, MS-4, UABSOM - Huntsville

Additional Authors: Senthil Vel Rajan Rajaram Manoharan, M.D., Assistant Professor, Psychiatry - Geriatric Psychiatry, UABSOM – Huntsville; Tarak Vasavada, MD, Professor, Psychiatry, UABSOM – Huntsville

Introduction/Background: According to the WHO, as of March 19, 2022, COVID-19 has infected 464 million people worldwide and taken the lives of six million people [1]. Much attention and resources have been devoted to understanding this virus; we have developed novel treatments and vaccines in record time. However, we know that immunocompromised patients, older patients, and those with numerous comorbidities are at the most significant risk of infection and death from COVID-19 [2]. This article presents case series of patients already affected by chronic psychiatric illness with baseline cognitive deficits who develop COVID-19 and the cognitive decline they face.

Description: The patient is a 65-year-old Caucasian female with a PMH of hypertension, myasthenia gravis, COPD, and a 30+ year history of medically stable paranoid schizophrenia who presented to the ED from a rehab facility due to recurrent falls. No family history of mental illness or substance use. She was medically stabilized over three weeks and discharged home only to return a day later due to aggressive behaviors, and she then tested positive for COVID-19. On this admission, she was delirious and incoherent, responding to internal stimuli and requiring soft physical restraints. She required the use of three antipsychotics, initially olanzapine and haloperidol, which were eventually tapered and replaced with clozapine. She had severe executive dysfunction, disinhibited behavior throughout her stay, and perseverated on religion. There was a subsequent decline in her functioning that accompanied poor oral intake. She required tube feeding and was briefly intubated due to respiratory collapse related to COVID-19. She had a prolonged recovery course but remained delirious and was eventually discharged to long-term nursing care.

Discussion and Conclusion: The patient presented here had multiple medical comorbidities, chronic psychosis, and some level of baseline cognitive decline or dementia. The patient dealt with schizophrenia for many years and too had a rapid decline after contracting COVID-19, and we portend that this drop is associated with infection of SARS-CoV-2 causing COVID-19. Due to the level of agitation and confusion the patient wasn't amenable to full cognitive evaluation, though bedside evaluation showed that severe attentional deficits were present and that their sensoriums were severely altered.

References:

WHO Coronavirus (COVID-19) Dashboard. (2022). Accessed: March 19, 2022: <https://covid19.who.int/>.

Vetrano DL, Tazzeo C, Palmieri L, Marengoni A, Zucchelli A, Lo Noce C, Onder G: Comorbidity status of deceased COVID-19 in-patients in Italy. *Aging Clin Exp Res.* 2021, 33:2361-5. 10.1007/s40520-021-01914-y

Alzheimer's Association: 2019 Alzheimer's disease facts and figures. *Alzheimers Dement.* 2019, 15:321-87. 10.1016/j.jalz.2019.01.010

Killin LO, Starr JM, Shiue IJ, Russ TC: Environmental risk factors for dementia: a systematic review. *BMC Geriatr.* 2016, 16:175. 10.1186/s12877-016-0342-y

Tomažič T, Čelofiga AK: The role of different behavioral and psychosocial factors in the context of pharmaceutical cognitive enhancers' misuse. *Healthcare (Basel).* 2022, 10:972. 10.3390/healthcare10060972

Sutin AR, Stephan Y, Luchetti M, Terracciano A: Loneliness and risk of dementia. *J Gerontol B Psychol Sci Soc Sci.* 2020, 75:1414-22. 10.1093/geronb/gby112

Watson CJ, Thomas RH, Solomon T, Michael BD, Nicholson TR, Pollak TA: COVID-19 and psychosis risk: real or delusional concern?. *Neurosci Lett.* 2021, 741:135491. 10.1016/j.neulet.2020.135491

Anglin DM, Galea S, Bachman P: Going upstream to advance psychosis prevention and improve public health. *JAMA Psychiatry.* 2020, 77:665-6. 10.1001/jamapsychiatry.2020.0142

Brauner JM, Mindermann S, Sharma M, et al.: Inferring the effectiveness of government interventions against COVID-19. *Science.* 2021, 371:1-10. 10.1126/science.abd9338

Plagg B, Engl A, Piccoliori G, Eisendle K: Prolonged social isolation of the elderly during COVID-19: Between benefit and damage. *Arch Gerontol Geriatr.* 2020, 89:104086. 10.1016/j.archger.2020.104086

Li YC, Bai WZ, Hashikawa T: The neuroinvasive potential of SARS-CoV2 may play a role in the respiratory failure of COVID-19 patients. *J Med Virol.* 2020, 92:552-5. 10.1002/jmv.25728

Atkins JL, Masoli JA, Delgado J, Pilling LC, Kuo CL, Kuchel GA, Melzer D: Preexisting comorbidities predicting COVID-19 and mortality in the UK Biobank community cohort. *J Gerontol A Biol Sci Med Sci.* 2020, 75:2224-30. 10.1093/gerona/glaa183

Bianchetti A, Rozzini R, Guerini F, et al.: Clinical presentation of COVID19 in dementia patients. *J Nutr Health Aging.* 2020, 24:560-2. 10.1007/s12603-020-1389-1

Taquet M, Luciano S, Geddes JR, Harrison PJ: Bidirectional associations between COVID-19 and psychiatric disorder: retrospective cohort studies of 62 354 COVID-19 cases in the USA. *Lancet Psychiatry.* 2021, 8:130-40. 10.1016/S2215-0366(20)30462-4