Type of Research: Case Study exceeds word count limit

Title: A Tale of Two Lithium Toxicities

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Introduction/Background: Lithium is one of the oldest treatments in psychiatry and is still the mainstay of treatment in bipolar disorder. The exact mechanism of lithium has yet to be elucidated but it is well understood that lithium has a narrow therapeutic index and is highly associated with overdose and toxicities [1]. The common presenting symptoms of lithium toxicity are confusion, cerebellar dysfunction, extrapyramidal symptoms, nausea, vomiting, diarrhea, nephrogenic diabetes insipidus, arrhythmias, shock, and respiratory distress [2]. Due to the pharmacokinetic properties of lithium, it has been demonstrated that these toxic effects tend to be more common and severe in patients that are chronically treated with lithium [3]. Here we report 2 cases, both with lithium toxicity, however the manifestation of symptoms in the cases were paradoxical.

Description:
Patient presentation 1

69-year-old female with complaints of progressively worsening lower extremity weakness and mental status changes.

Past medical history of schizoaffective disorder, hypertension, hyperlipidemia, spinal stenosis of the lumbar foramina, and type 2 diabetes mellitus.

Home medications included lithium 300 mg twice per day, aspirin 81 mg once per day, benztropine 1 mg once per day, chlorpromazine 50 mg once per day, clonazepam 1 mg twice per day, pravastatin 40 mg once per day, meloxicam 15 mg every 6 hours, lisinopril 40 mg once per day, hydrochlorothiazide 25 mg once per day, metformin 500 mg twice per day, fluoxetine 20 mg once per day, sliding scale regular insulin, lansoprazole 40 mg once per day, levetiracetam 1500 mg/Sodium Chloride 115 mL @ 400 mL/hr every 12 hours.

Patient presentation 2

40 year old female with one week history of 6-8 watery non bloody bowel movements per day and nausea/vomiting.

Past medical history significant for schizoaffective disorder and polycystic ovarian syndrome

Home medications include cariprazine 3 mg once a day, Lithium 300 mg twice a day, Hydrochlorothiazide 25 mg once a day, and Trizepatide 15 mg once a month

Discussion:
• Lithium remains the treatment of choice for bipolar patients despite its narrow therapeutic index and high propensity to cause toxic effects [7].

• Lithium toxicity is divided into three distinct categories: acute, acute on chronic, and chronic [7].

• Acute toxicity occurs in lithium overdose in patients that do not regularly take lithium, which typically presents with gastrointestinal upset, confusion, and slurred speech [7].

• Acute on chronic toxicity is an acute overdose in patients that currently take lithium therapy [7].

• Chronic toxicity refers to the gradual accumulation of lithium levels in patients currently on lithium therapy and is often due to secondary causes that alter the renal clearance of lithium [8].

• Neurotoxicity associated with lithium more commonly presents in acute on chronic or chronic situations due to lithium's slow distribution into the central nervous system [7].

• In a study of patients with lithium toxicity, the duration of exposure to elevated lithium levels correlated with the severity of the observed neurotoxicity and the absolute serum level of lithium did not correlate with the severity of toxicity, indicating that a gradual increase in lithium levels may present more severely than an acute overdose event [3].

• It has been demonstrated that in patients experiencing lithium toxicity, the patients that were chronically exposed to lithium, had a significantly decreased renal clearance and an increased elimination half life of lithium [10].

• Lithium variably enters different tissues, entering the kidneys most rapidly (minutes) and the brain most slowly (hours), which further demonstrates why chronic lithium intake is more likely to result in neurotoxic effects [12].

• Patient 2 had severely high levels of lithium in their blood (5.7) yet demonstrated much milder symptoms (paresthesias and prolonged QT interval) than Patient 1. This is likely an artifact of this being a case of acute on chronic toxicity in which this patient had been chronically treated with lithium but acutely had a Clostridium difficile infection also continued to overdose by ingesting lithium tablets despite the medication being stopped by the medical team.

• Patient 1, this was likely a chronic lithium toxicity case in which the patient had been treated with lithium over a long period of time but did not have an acute event that precipitated symptoms. Instead, over the course of time the patient experienced worsening lower extremity weakness (out of proportion for lumbar stenosis level), altered mental status, and diarrhea that could not be explained by an acute infection.

• Both patients were highly susceptible to lithium toxicity given their chronic treatment with lithium, potential medication interaction with SSRIs and antipsychotics, and diuretic use. Specifically, hydrochlorothiazide which has been shown to increase lithium levels greater than other diuretics by decreasing lithium excretion [13].

Conclusion: Despite its narrow therapeutic index, lithium is still the mainstay of treatment for bipolar disorder. Toxicity can manifest with a wide variety of symptoms that can range from GI upset to several neurological disturbances like confusion and polyneuropathy. With its high propensity to reach dangerous blood levels in patients, especially those that are chronically treated, the use of lithium in patients must be carefully considered. Patients must undergo routine monitoring of blood levels, receive education of symptoms to look out for, and drug interactions need to be considered before prescribing additional therapies. Here, we report two differing cases of lithium toxicity in two chronically treated patients with widely different presentations of symptoms.

References:

[1] Malhi GS, Tanious M, Das P, Coulston CM, Berk M. Potential mechanisms of action of lithium in bipolar disorder. Current understanding. CNS Drugs. 2013 Feb;27(2):135-53. doi: 10.1007/s40263-013-0039-0. PMID: 23371914.

[2] Won E, Kim YK. An Oldie but Goodie: Lithium in the Treatment of Bipolar Disorder through Neuroprotective and Neurotrophic Mechanisms. Int J Mol Sci. 2017 Dec 11;18(12):2679. doi: 10.3390/ijms18122679. PMID: 29232923; PMCID: PMC5751281.

[3] W.S. Waring, W.J. Laing, A.M. Good, D.N. Bateman. Pattern of lithium exposure predicts poisoning severity: evaluation of referrals to a regional poisons unit. QJM, 100 (5) (2007), pp. 271-276

[4] Shorter E. The history of lithium therapy. Bipolar Disord. 2009 Jun;11 Suppl 2(Suppl 2):4-9. doi: 10.1111/j.1399-5618.2009.00706.x. PMID: 19538681; PMCID: PMC3712976.

[5] Geddes JR, Miklowitz DJ. Treatment of bipolar disorder. Lancet. 2013 May 11;381(9878):1672-82. doi: 10.1016/S0140-6736(13)60857-0. PMID: 23663953; PMCID: PMC3876031.

[6] Carter CJ. Multiple genes and factors associated with bipolar disorder converge on growth factor and stress activated kinase pathways controlling translation initiation: implications for oligodendrocyte viability. Neurochem Int. 2007 Feb;50(3):461-90. doi: 10.1016/j.neuint.2006.11.009. Epub 2007 Jan 18. PMID: 17239488.

[7] Ivkovic A, Stern TA. Lithium-induced neurotoxicity: clinical presentations, pathophysiology, and treatment. Psychosomatics. 2014 May-Jun;55(3):296-302. doi: 10.1016/j.psym.2013.11.007. Epub 2013 Nov 28. PMID: 24388123.

[8] R.F. McKnight, M. Adida, K. Budge, et al. Lithium toxicity profile: a systematic review and meta-analysis. Lancet, 379 (9817) (2012), pp. 721-728

[9] P.W. Oakley, I.M. Whyte, G.L. Carter. Lithium toxicity: an iatrogenic problem in susceptible individuals. Aust N Z J Psychiatry, 35 (6) (2001), pp. 833-840

[10] G. Ferron, M. Debray, F. Buneaux, F.J.Baud, J.M. Scherrmann. Pharmacokinetics of lithium in plasma and red blood cells in acute and chronic intoxicated patients. Int J Clin Pharmacol Ther, 33 (6)(1995), pp. 351-355

[11] H. Klemfuss, T.T. Bauer, K.E. Greene, D.F. Kripke. Dietary calcium blocks lithium toxicity in hamsters without affecting circadian rhythms. Biol Psychiatry, 31 (3) (1992), pp. 315-321

[12] Peces, R., Fernández, E. J., Regidor, D., Peces, C., & Sánchez, R. (2006). Treatment of acute lithium intoxication with high-flux hemodialysis membranes. Nefrología (English Edition),26(3), 372-378.

[13] Crabtree BL, Mack JE, Johnson CD, Amyx BC. Comparison of the effects of hydrochlorothiazide and furosemide on lithium disposition. Am J Psychiatry. 1991;148(8):1060-1063. doi:10.1176/ajp.148.8.1060

Type of Research: Case Study

Title: Are There Risks of Dextroamphetamine-Amphetamine Use in Patients with Cerebral Aneurysms?

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Introduction/Background: Dextroamphetamine-amphetamine, a stimulant medication prescribed for attention-deficit/hyperactivity disorder (ADHD), is known to affect cardiovascular function by increasing blood pressure and heart rate.1 Cerebral aneurysms, characterized by abnormal dilations of blood vessels in the brain, pose significant risks if they rupture, including hemorrhagic stroke, brain damage, coma, and even death.2 Concerns arise regarding the safety of this medication in patients with pre-existing vascular conditions, such as cerebral aneurysms, particularly considering the potential for stimulant abuse.

Description: We present a 32-year-old woman with a history of cerebral aneurysm, ADHD, benign essential hypertension, bipolar II disorder, and generalized anxiety disorder with panic attacks. She has been on long-term therapy, having used the medication inconsistently for two years before her aneurysm diagnosis. Recent evaluations focus on assessing the impact of continued mixed salt amphetamine drug use and potential abuse on her aneurysm stability and cardiovascular health.

Discussion and Conclusion: Current literature provides limited direct evidence linking mixed salt amphetamine drug use to increased risk of complications in patients with cerebral aneurysms. A case report by Harrington et al. identified intracerebral hemorrhage associated with oral amphetamine use, noting abnormal cerebral vessels and suggesting mechanisms such as acute blood pressure increases and vascular toxicity.3 A recent report described rapid cerebral aneurysm growth associated with methamphetamine abuse.4 Rumbaugh et al. documented severe cerebrovascular damage in Rhesus monkeys from methamphetamine, including decreased caliber and slow flow in small cerebral arteries, and significant brain damage such as ischemia and infarction.5 Despite these concerns, Zhang et al. found no overall increase in cardiovascular disease with ADHD medication use but recommended further research into risks like cardiac arrest and tachyarrhythmias, particularly in those with pre-existing conditions.6 An editorial emphasized caution when prescribing ADHD medications, particularly for elderly patients, women, and those with cerebrovascular conditions, underscoring the need for careful prescribing practices and enhanced monitoring.7

Although definitive evidence linking mixed salt amphetamine drug abuse to cerebral aneurysm complications is lacking, the potential for stimulant-induced vascular stress and damage necessitates cautious prescribing practices. Further research is essential to clarify the relationship between stimulant abuse and cerebral aneurysm complications and to establish safer prescribing guidelines.

References:

1. Wilens, T. E., Hammerness, P. G., Biederman, J., Kwon, A., Spencer, T. J., Clark, S., Scott, M., Podolski, A., Ditterline, J. W., Morris, M. C., & Moore, H. (2005). Blood pressure changes associated with medication treatment of adults with attention-deficit/hyperactivity disorder. The Journal of clinical psychiatry, 66(2), 253-259. https://doi.org/10.4088/jcp.v66n0215

2. Nissen, S. E., & Cardiovascular and Renal Drugs Advisory Committee (2005). Report from the Cardiovascular and Renal Drugs Advisory Committee: US Food and Drug Administration; June 15-16, 2005; Gaithersburg, Md. Circulation, 112(13), 2043-2046. https://doi.org/10.1161/CIRCULATIONAHA.105.57310

3. Harrington, H., Heller, H. A., Dawson, D., Caplan, L., & Rumbaugh, C. (1983). Intracerebral hemorrhage and oral amphetamine. Archives of neurology, 40(8), 503-507. https://doi.org/10.1001/archneur.1983.04210070043012

4. Fowler, J., Fiani, B., Quadri, S. A., Cortez, V., Frooqui, M., Zafar, A., Ahmed, F. S., Ikram, A., Ramachandran, A., & Siddiqi, J. (2018). Impact of Methamphetamine Abuse: A Rare Case of Rapid Cerebral Aneurysm Growth with Review of Literature. Case reports in neurological medicine, 2018, 1879329. https://doi.org/10.1155/2018/1879329

5. Rumbaugh, C. L., Bergeron, R. T., Scanlan, R. L., Teal, J. S., Segall, H. D., Fang, H. C., & McCormick, R. (1971). Cerebral vascular changes secondary to amphetamine abuse in the experimental animal. Radiology, 101(2), 345-351. https://doi.org/10.1148/101.2.345

6. Zhang L, Yao H, Li L, et al. Risk of Cardiovascular Diseases Associated With Medications Used in Attention-Deficit/Hyperactivity Disorder: A Systematic Review and Meta-analysis. JAMA Netw Open.2022;5(11):e2243597. doi:10.1001/jamanetworkopen.2022.43597

7. Ziegelstein RC. Paying Attention to Attention-Deficit/Hyperactivity Disorder Medications and Cardiovascular Risk. JAMA Netw Open. 2022;5(11):e2243606. doi:10.1001/jamanetworkopen.2022.43606

Type of Research: Case Study

Title: A Trauma Informed Classroom: An Educational Intervention to Address Behavioral Needs for Elementary Students in Rural Alabama

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Introduction/Background: Half of all mental disorders start by 14 years, are often preceded by non-specific psychosocial disturbances, and can evolve into major mental disorders accounting for 45% of the global disease burden across the 0-25 age span. During this critical period, mental health needs are largely unmet, making school-based services crucial for addressing children's emotional and social needs. However, studies on these interventions' impact on behavioral regulation and internalizing and externalizing factors are limited. This study assesses the effectiveness of a trauma-informed classroom (TIC) in rural Alabama for students with behavioral regulation issues, aiming to transition them back to traditional classrooms with individualized education plans (IEPs). This overview focuses on four children receiving the highest level of TIC services.

Description:
Case 1: A 9 y/o male with childhood trauma, exposure to substances in-utero, ADHD, and anxiety who demonstrated physical and verbal aggression at school. Prior to entering the TIC, he was expelled from two schools and was academically and developmentally behind. After 2.5 years in the TIC, he no longer exhibited aggression and was transitioned back to a traditional classroom with academic improvement.

Case 2: A 10 y/o male with childhood trauma, ADHD, anxiety, and impairment in reading who demonstrated physical aggression towards peers. Prior to the TIC he repeated kindergarten. After the TIC, he performed at grade level and reduced behavioral disturbance by 80%.

Case 3: An 11 y/o male with separation anxiety and disruptive mood dysregulation disorder, who struggled with school refusal and behavioral aggression when directed in the classroom. The student's family was non-compliant with TIC recommendations. The student continued to have behavioral and academic problems.

Case 4: An 11 y/o female with ADHD, anxiety, and autism who was physical aggressive with staff and peer and was mostly non-verbal prior to the TIC. After 2 years, the student performed at a 1.5 grade level in reading.

Discussion and Conclusion: The findings suggest that TICs could be an effective school-based strategy for improving behavioral regulation skills, with potential benefits for students' success in a traditional classroom setting. However, further work needs to be done.

References:
Colizzi, M., Lasalvia, A., & Ruggeri, M. (2020). Prevention and early intervention in youth mental health: is it time for a multidisciplinary and trans-diagnostic model for care? International Journal of Mental Health Systems, 14(1). https://doi.org/10.1186/s13033-020-00356-9

Compas, B. E. (2006). Psychobiologial process of stress and coping: Implications for Resilience in Children and Adolescents—Comments on the Papers of Romeo & McEwen and Fisher et al. Annals of The New York Academy of Science, 1094(1), 226-234. https://doi.org/10.1196/annals.1376.024

Cree, R. A., Bitsko, R. H., Robinson, L. R., Holbrook, J. R., Danielson, M. L., Smith, C.,…Peacock, G. (2018). Health Care, Family and Community Factors Associated with Mental, Behavioral, and Developmental Disorders and Poverty Among Children Aged 2-8 Years-United States, 2016. Morbidity and Mortality Weekly Report, 67(50), 1377-1383.

Durlak, J. A., Weissberg, R. P., Dymnicki, A. B., Taylor, R. D., & Schellinger, K. B. (2011). The Impact of Enhancing Students' Social and Emotional Learning: A Meta Analysis of School-Based Universal Interventions. Child Development, 82(1), 405-432. https://doi.org/10.1111/j.1467-8624.2010.01564.x

Farmer, E. M. Z., Burns, B. J., Phillips, S. D., Angold, A., & Costello, E. J. (2003). Pathways Into and Through Mental Health Services for Children and Adolescents. Psychiatric Services, 54(1), 60-66. https://doi.org/10.1176/appi.ps.54.1.60

Tkacz, J., & Brady, B. L. (2021). Increasing rate of diagnosed childhood mental illness in the United States: Incidence, prevalence and costs. Public Health in Practice, 2, 1-7. https://doi.org/10.1016/j.puhip.2021.100204

Type of Research: Case Study

Title: Diazepam-associated periorbital and facial edema: A rare allergic reaction

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Introduction/Background: Benzodiazepines (BZDs) are frequently administered for agitation on inpatient psychiatry units. Allergic reactions to BZDs are rare and typically present as cutaneous symptoms such as urticaria, erythema, and angioedema. A few case reports describe more severe reactions to BZDs, however causality was not definitively established. The frequent and repeated use of benzodiazepines necessitates further characterization of BZD-induced allergic reactions. Here we describe a 34-yr-old male with chronic schizophrenia (SCZ), who most likely developed a rare allergic reaction to oral diazepam (DZM).

Description: A 34-year-old male with chronic SCZ, no significant medical history, and no known drug allergies (NKDA) was admitted to the inpatient psychiatric unit in a florid psychotic state. Oral haloperidol was immediately initiated. On hospital day 2, he received 2mg of oral lorazepam for agitation, which he tolerated well. Despite demonstrating a partial treatment response from up-titrated haloperidol, the patient's agitation increased with new onset homicidal ideation. On hospital day 12, oral DZM 5 mg three times daily was initiated. On day 15, after five doses of oral DZM, the patient developed significant bilateral periorbital and facial edema, without respiratory, hemodynamic or visual compromise. DZM was discontinued and oral diphenhydramine was initiated. The patient's allergic symptoms rapidly improved, and resolved completely by day 18. Oral lorazepam 1mg twice daily was initiated on hospital day 19, which was tolerated well without recurrence of symptoms.

Discussion and Conclusion: The patient's symptoms were consistent with existing reports of BZD-induced angioedema, and the ADR Probability Scale (Naranjo) of this adverse event was 6. The precise mechanism of BZD allergy is unknown, however the lack of cross-reactivity with lorazepam in this case suggests that the allergy is most likely specific to DZM and may be due to its unique metabolism or an excipient in its formulation. Clinicians should remain vigilant for allergic symptoms when prescribing a BZD, even in patients with NKDA. Additionally, it is possible to trial an alternative BZD agent with simpler metabolism, such as lorazepam, when a specific BZD is poorly tolerated.

References:

1. Adverse Drug Reaction Probability Scale (Naranjo) in Drug Induced Liver Injury. In: LiverTox: Clinical and Research Information on Drug-Induced Liver Injury. edn. Bethesda (MD); 2012.

2. Edinoff AN, Nix CA, Hollier J, Sagrera CE, Delacroix BM, Abubakar T, Cornett EM, Kaye AM, Kaye AD: Benzodiazepines: Uses, Dangers, and Clinical Considerations. Neurol Int 2021, 13(4):594-607.

3. Haybarger E, Young AS, Giovannitti JA, Jr.: Benzodiazepine Allergy With Anesthesia Administration: A Review of Current Literature. Anesth Prog 2016, 63(3):160-167.

4. Martinez-Tadeo JA, Perez-Rodriguez E, Hernandez-Santana G, Garcia-Robaina JC, de la Torre-Morin F: Anaphylaxis caused by tetrazepam without cross-reactivity with other benzodiazepines. Ann Allergy Asthma Immunol 2012, 108(4):284-285.

5. Reid Finlayson AJ, Macoubrie J, Huff C, Foster DE, Martin PR: Experiences with benzodiazepine use, tapering, and discontinuation: an Internet survey. Ther Adv Psychopharmacol 2022, 12:20451253221082386.

Type of Research: Case Study exceeds word count limit

Title: Thyroid Abnormalities Presenting as Psychosis

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Introduction/Background: Thyroid disease of all etiology can present with a wide range of symptoms affecting all organ systems in the body. Hypothyroidism typically manifests as fatigue, weight gain, dry skin, hair loss, temperature sensitivity, hoarseness, muscle weakness, and constipation. In extreme cases, patients can present comatose requiring immediate medical intervention. On the other end of the spectrum, hyperthyroidism classically presents with palpitations, heat intolerance, weight loss, anxiety, or gastrointestinal hypermobility. Thyroid storm, a severe and life-threatening manifestation of hyperthyroidism, presents with seizures, delirium, and hyperreflexia. Interestingly, both hyper- and hypothyroidism can present with psychotic symptoms. While it is more common for patients with hypothyroidism to present with depressive symptoms, an estimated 3% of patients present with manic and psychotic symptoms. This key presentation of mania and psychosis helps differentiate the possibility of depression with psychotic features, a manifestation that could be expected in within the classical hypothyroid symptoms. Hyperthyroidism can classically present as mania, with or without psychotic features. Patient presenting with hyperthyroidism also reported significantly worse memory, attention, planning, and productivity prior to treatment, with some patients experiencing lingering deficits after treatment. Since both hyperthyroidism and hypothyroidism can present with similar psychiatric symptoms, thyroid testing is of utmost importance for patient presenting to medical care with psychosis. The purpose of this case study is to explore two presentations of thyroid disease with psychosis and highlight the importance of a complete workup of patients presenting with such symptoms.

Description: A 59-year-old female with a past medical history of type 2 diabetes, COPD, cirrhosis and "some thyroid problem" presents to the hospital due to altered mental status. Patient had one psychiatric admission for a depressive episode more than a decade ago. She does not currently take any medication for any medical or psychiatric conditions. The patient endorsed feelings of paranoia and religious delusions. Labs were notable for TSH-383, undetectable T3 /T4, and elevated CCK suggesting hypothyroidism. Patient has a family history of maternal thyroid problems. Patient first refused treatment, but eventually was amenable to intravenous levothyroxine. After three days, the patient was alert, oriented, and had better insight allowing for discharge. One week later, the patient returned to the emergency room with similar symptoms secondary to medication noncompliance. Patient was paranoid and distrustful of medical care. She denied hallucinations, though did report seeing children in the hallway. The patient was initially resistant to medications but eventually agreed to paliperidone and levothyroxine. After two days of treatment, the patient improved and was discharged home.

A 21-year-old female presenting to the hospital for an involuntary admission by family. She was reportedly throwing knives at family and resisted law enforcement. The patient has a history of polysubstance use and one psychiatric admission. On arrival, the patient was pacing and constantly mumbling. She could not provide history but denied taking medications for any psychiatric or medical problems. The patient had a blood pressure of 146/60 and a heart rate of 116-bpm. The patient had notable exophthalmos but initially declined physical exam. The patient had undetectable TSH and free T4-6.95ng/dL. The patient denied chest pain, palpitations, nausea or vomiting but endorsed mild menorrhagia. Besides exophthalmos, physical exam noted an enlarged thyroid gland. An ultrasound showed an enlarged, vascular gland without any nodules. The patient was paranoid and guarded, though

she did share her mother had a thyroid problem. Patient was started on olanzapine, methimazole, and propranolol which decreased her T4 to 5.86 after one day. After three days of treatment, the patient improved and was discharged home on medications started at the hospital.

Discussion and Conclusion: For both of our patients with thyroid disease and psychosis, their psychotic symptoms resolved upon treatment of their underlying thyroid disease. Interestingly, both patients had similar presentations of paranoia. Both patients had a family history of thyroid disease and neither patient had a family or personal history of autoimmune diseases. Thyroid disease overall is more common in women, while the link between gender and thyroid disease associated psychosis is yet to be studied. While only the hyperthyroidism patient presented with non-psychiatric symptoms of her disease, both patients benefitted immensely from the treatment of their thyroid disease. For long-term management of these patients, we plan on maintaining the patients on antipsychotic medication in addition to thyroid medication until thyroid lab values are within normal limits. At that time, there can be a discussion of tapering antipsychotics depending on patient comfort. Overall, these two distinct cases show the importance of medical workup for patients presenting with acute psychosis.

Abbreviations:
COPD: chronic obstructive pulmonary disease
TSH: thyroid stimulating hormone
T3: triiodide
T4: thyroxine
CCK: cholecystokinin
BPM: beats per minute

References:

1. Kaplan, J. L., & Castro-Revoredo, I. (2020). Severe Hypothyroidism Manifested as Acute Mania with Psychotic Features: A Case Report and Review of the Literature. Journal of psychiatric practice, 26(5), 417-422.

2. Mohamed, M. F. H., Danjuma, M., Mohammed, M., Mohamed, S., Siepmann, M., Barlinn, K., Suwileh, S., Abdalla, L., Al-Mohanadi, D., Silva Godínez, J. C., Elzouki, A. N., & Siepmann, T. (2021). Myxedema Psychosis: Systematic Review and Pooled Analysis. Neuropsychiatric disease and treatment, 17, 2713-2728.

3. Stern, R. A., Robinson, B., Thorner, A. R., Arruda, J. E., Prohaska, M. L., & Prange, A. J., Jr (1996). A survey study of neuropsychiatric complaints in patients with Graves' disease. The Journal of neuropsychiatry and clinical neurosciences, 8(2), 181-185.

Type of Research: Case Study

Title: Serotonin Withdrawal Syndrome induced by THC vape

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Introduction/Background: Serotonin Withdrawal Syndrome is usually the effect of abrupt cessation of antidepressant therapy (SSRIs, SNRIs), which would decrease neurotransmitter levels in the synaptic cleft. Symptoms of serotonin withdrawal can be summarized by the mnemonic "FINISH" which includes flu-like symptoms, insomnia, nausea, imbalance, sensory disturbances (electric shock and tingling feelings), and hyper-arousal, such as anxiety and agitation. Symptoms can occur within 2-4 days of drug cessation and usually last 1-2 weeks, with some persisting up to a year. Sociodemographic and clinical factors have not been identified for Serotonin withdrawal syndrome. Symptoms of serotonin withdrawal may vary in severity, duration, series, and trajectories. However, in the case that compliance is met with antidepressant medications, could THC withdrawal be a potential cause?

Description: The patient is a 15-year-old male with PMH of GAD, major depression, sleep disturbance, and vaping engagement who has been taking sertraline 75 mg, clonidine 0.1mg, and hydroxyzine 25 mg for several months. Recently he presented to his primary care clinic with complaints of twitching and worsening anxiety that have been occurring for two weeks. After evaluation from PCP, an increase in blood pressure was noted and patient was referred to psychiatry for a potential serotonin syndrome case. Upon further evaluation, we discovered his twitching was actually "electric shock" and "pins and needles" like sensations in his extremities. Along with increased anxiety, he reports difficulty sleeping over these 2 weeks. Patient reports to have been using a THC vape for several months but recently stopped 2-3 weeks ago. Sertraline dose had been increased from 50mg to 75mg three months ago. There had been no other medication changes recently and he reports compliance to all medications.

Discussion and Conclusion: Tetrahydrocannabinol (THC) predominately attaches to cannabinoid receptors (CB1 and CB2) on neurons, activates them and releases neurotransmitters, or inhibits them and decreases reuptake. Although CB1 and CB2 are the most widely acknowledged cannabinoid receptors, several other receptors ranging from G-protein coupled receptors to ion channel and nuclear receptors have been reported to interact with cannabinoids (4). In addition, animal studies have demonstrated that potent cannabinoid receptor agonists, such as THC, may activate the serotonin receptors (5-hydroxytryptamine1A and 5-hydroxytryptamine2A), as well as inhibit serotonin re-uptake. Therefore, THC abuse in high concentrations can mimic serotonin syndrome (2). However, in addition to autonomic and mental status changes, symptoms of Serotonin Syndrome include hyper-reflexia, myoclonus, and hyperthermia, which our patient did not present with. Due to the ability for THC to cause Serotonin Syndrome and our patient's presentation, we have concluded that a withdrawal of THC could potentially cause Serotonin Withdrawal Syndrome. With the increased use of vapes and other substances, additional research is needed in this area to further educate and inform patients as well as providers on the effects of THC toxicity and withdrawal. This information will help prevent recurrent relapse in addition to tolerance to THC.

References:

1. Gabriel M, Sharma V. Antidepressant discontinuation syndrome. CMAJ. 2017 May 29;189(21):E747. doi:10.1503/cmaj.160991. PMID: 28554948; PMCID: PMC5449237.

2. Hill MN, Sun JC, Tse MT, Gorzalka BB. Altered responsiveness of serotonin receptor ubtypes following long-term cannabinoid treatment. Int J Neuropsychopharmacol. 2006 Jun;9(3):277-86.doi: 10.1017/S1461145705005651. Epub 2005 Jun 21. PMID: 15967059.

3. M. Fornaro, C.I. Cattaneo, D. De Berardis, F.V. Ressico, G. Martinotti, E. Vieta, Antidepressant discontinuation syndrome: A state-of-the-art clinical review, European Neuropsychopharmacology, Volume 66,2023,Pg1-10,ISSN0924-977X, https://doi.org/10.1016/j.euroneuro.2022.10.005.

4. Zou S, Kumar U. Cannabinoid Receptors and the Endocannabinoid System: Signaling and Function in the Central Nervous System. Int J Mol Sci. 2018 Mar 13;19(3):833. doi: 10.3390/ijms19030833. PMID: 29533978; PMCID: PMC5877694.

5. Galaj, Ewa & Xi, Zheng-Xiong. (2020). Possible Receptor Mechanisms Underlying Cannabidiol Effects on Addictive-like Behaviors in Experimental Animals. International Journal of Molecular Sciences. 22. 134. 10.3390/ijms22010134.

6. Haddad, Peter & Anderson, Ian. (2007). Recognising and managing antidepressant discontinuation symptoms. Advances in Psychiatric Treatment. 13. 447-457. 10.1192/apt.bp.105.001966.

7. Atakan, Zerrin. (2012). Cannabis, a complex plant: different compounds and different effects on individuals. Therapeutic advances in psychopharmacology. 2. 241-54. 10.1177/2045125312457586.

Type of Research: Case Study exceeds word count limit

Title: Sneaky Serotonin Syndrome

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Introduction/Background: Serotonin syndrome is an iatrogenic syndrome typically caused by an excessive amount of serotonin at the postsynaptic receptors. This can be due to therapeutic use of a single serotonergic drug or as an interaction between multiple serotonergic medications. The typical presenting features are autonomic instability diarrhea, restlessness, agitation, hyperreflexia/myoclonus, rigidity, and delirium.1 Serotonin increasing medications can be a slippery slope because the medications impact every patient differently. Sometimes a small medication increase can result in onset of the symptoms mentioned above. It is imperative when dealing with a possible serotonin syndrome presentation to get a detailed history and timeline of symptoms, and to have a strong clinical suspicion.

Description: We present a 30 y/o female with a history of GAD, nicotine use disorder, and MDD presents to the clinic with concerns for serotonin syndrome. Her Prozac was increased to 80 mg and she was started on Trazodone 100 mg 10 days ago at the same visit due to worsening anxiety and insomnia. Today, patient presents with, N/V, diarrhea, HA, and restlessness that all started 2 days after her medication change and has worsened over time. She presented to her PCP 4 days ago with the same symptoms that she presents with today. At that time she was normotensive with no autonomic dysfunction. She was instructed to discontinue her trazodone, and her Prozac was decreased back to 40 mg. The physician reached out to her psychiatrist, and an appointment was scheduled for today. At this visit she has a BP of 142/82. She is currently on amlodipine for BP regulation, and her BP is last visit was 118/80. Patient reports she is very fatigued. She reports a decrease in appetite to less than 1 meal/ day and SOB. Her physical exam was notable for rigidity and hyperreflexia.

All serotonergic medications were discontinued. A tough clinical decision was made to allow this patient to go home with cyproheptadine 4mg BID. The patient was counseled to drink plenty of water and agreed to go to the emergency room or call 911 if there is acute worsening of symptoms. She made a full recovery within a few days.

Discussion and Conclusion:
Serotonin Syndrome Diagnostic Criteria:
Autonomic dysfunction: Diaphoresis, tachycardia, HTN, Mydriasis
Neuromuscular excitability : Hyperreflexia, myoclonus, rigidity
Altered Mental Status: Delirium, psychomotor agitation/restlessness, coma

Treatment
STOP THE OFFENDING MEDICATION
Cyproheptadine
Hospitalization to monitor depending on severity

Cyproheptadine
serotonin receptor antagonist. By blocking these receptors, the symptoms of serotonin syndrome can resolve as soon as 24 hours of the initial dose.2 Common side effects include thickening of bronchial

secretions, drowsiness, constipation, and dry mouth. Seizures have been documented, but they are extremely rare. Drowsiness is the most common AE.2

Clinical Learning Point
trazadone 100 mg and doubling the dose of her SSRI might not have been the right decision. However, when these medications were added the patient had functionally disabling insomnia and worsening anxiety/depression. If we were to do anything different, we might have increased the Prozac from 40 mg to 60 mg instead of 80 mg. Either way, the eventual target dose for treating her anxiety and depression would be 80 mg. At the time of prescribing these medicines our patient required an aggressive medication intervention. There have been multiple patients double their dose of Prozac in our clinic that have had no interactions. Different patients have different reactions to serotonergic medications making it almost impossible to predict who is more at risk for developing serotonin syndrome.1

This case exemplifies the importance for a clinical suspicion of serotonin syndrome needed for all patients taking serotonergic medications. Patient's do not always have to meet the exact book description for serotonin syndrome before you need to take action. Early recognition and treatment is crucial to prevent significant morbidity and mortality.

References:
1) Mikkelsen, Nicolaj, et al. Serotonin Syndrome - A Focused Review, 17 Mar. 2023, https://doi.org/10.22541/au.167905813.34118618/v1.

2) Poian, Leila R, and Silvana Chiavegatto. "Serotonin syndrome: The role of pharmacology in understanding its occurrence." Cureus, 11 May 2023, https://doi.org/10.7759/cureus.38897.

Type of Research: Case Study exceeds word count limit

Title: Lewy Body Dementia

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Introduction/Background: Dementia with Lewy Bodies is the second most common dementia behind Alzheimer's disease. Lewy Body Dementia is an umbrella term that encompasses DLB and Parkinson's disease dementia.

Distinguishing PDD from DLB is challenging, and a detailed history is critical for the diagnosis.

Although this disease is currently viewed as somatic, there are recent studies suggesting possible genetic influence that are associated with the onset of DLB.

DLB's prevalence is 7.5% of all dementia cases. However, in a retrospective study looking at patient's with dementia via autopsy after death, 25% of the brains from the people enrolled had Lewy body features.

median survival of a patient with DLB is 4.7 years from diagnosis

Due to this rapid decline, this diagnosis should be heavily considered, and the news of this diagnosis should be delivered to the patient with compassion and empathy.

Description: A 71-year-old male with a history of Bipolar Disorder Type I with rapid cycling, parkinsonism, Lewy body dementia, and normal pressure hydrocephalus (NPH), anxiety, multiple prior CVAs and a prior DVT in the right thigh presents with worsening visual and auditory hallucinations and insomnia.

History of Present Illness: The patient has experienced intermittent visual and auditory hallucinations during the day for several years, but these symptoms have recently worsened and now predominantly occur at night. He describes REM sleep disturbances and hallucinations that begin around 9-10 pm and continue intermittently throughout the night. The hallucinations are vivid and distressing; for example, the patient recently believed he was being kidnapped by pirates and screamed out in fear. Although he eventually recognized this as a hallucination, he felt compelled to jump out of a window to escape which demonstrates the patient and his family's fear of self harm. These symptoms have notably worsened following a fall that resulted in a subdural hematoma one week ago. He was hospitalized for the subdural hematoma, but no surgical intervention was required. He is being followed by neurosurgery on an outpatient basis at this time. Since the fall, he has been averaging only 2 hours of sleep per night, although he naps frequently during the day, accumulating several hours of sleep.

Psychiatric History: The patient has a long-standing history of psychiatric care. Recently, his dose of Wellbutrin was increased to 150mg every morning and 300mg nightly due to worsening depressive symptoms. His most recent lithium level was 0.72 which was measured at 9:30 am while he was hospitalized for the subdural hematoma. His initial depressive symptoms were treated with multiple SSRIs, which precipitated mania and led to significant personal and professional consequences, including the loss of his job as a regional director of sales for a medical supply company. Over the years, he has trialed numerous medications, including Keppra and Depakote, which provided some benefit. Geodon and Abilify were discontinued due to tardive dyskinesia and dystonia. SSRIs have since been avoided due to their propensity to induce mania, although he recently trialed Vilazodone, which was subsequently

discontinued. He is followed by a neurologist who recently diagnosed the patient with diffuse Lewy body dementia.

Discussion and Conclusion:

Diagnostic Criteria:
Parkinsonism's: Can be a resting tremor, rigidity, bradykinesia, or postural instability (i.e. shuffling gait, decreased arm swing, stooped posture).
dementia documented: Montreal cognitive assessment (MoCA): 24/30 onset 1-2 years before or after PD diagnosis
difficult to distinguish DLB from PDD because the average timeline for PDD is typically 2 years after diagnosis. While DLB is one
Pathology
DLB starts with alpha synuclein in the cortex, while PDD originates in the basal ganglia.
2. DLB and PDD alike are both accumulation of alpha-synuclein

Currently there is no treatment available

Key distinguishing factors:
LBD vs. Alzheimer's
Hallucinations had 83% PPV for LBD over AD
LBD vs PDD
Within 1 year of PD diagnosis = LBD
>1 year = PDD
Both have same pathology and treated the same clinically

Clinical Learning Point:
This patient's story of diagnosis was very discouraging from a medical student's perspective. The patient reported that physician wrote the diagnosis on a piece of paper, handed it to the patient, and told them to have a good day as he went to see the next patient. This case was a reminder that patients are people. The job of a doctor is not just being a good diagnostician. We have a duty to be there for our patients through it all, and we MUST show empathy and compassion in the good news and, especially, the bad.

References:

1) Sanford, Angela M. â€œLewy body dementia.â€ Clinics in Geriatric Medicine, vol. 34, no. 4, Nov. 2018, pp. 603-615, https://doi.org/10.1016/j.cger.2018.06.007.

2) Galasko D.: Lewy body disorders. Neurol Clin 2017; 35: pp. 325-338.

3) Walker, Zuzana, et al. "Comparison of cognitive decline between dementia with Lewy bodies and Alzheimer's disease: A cohort study." BMJ Open, vol. 2, no. 1, 2012, https://doi.org/10.1136/bmjopen-2011-000380

Type of Research: Case Study

Title: The Neuropsychiatric Manifestations of Pai Syndrome

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Introduction/Background: Pai syndrome is a rare condition characterized by a triad of developmental anomalies: a complete median cleft lip, cutaneous polyps, and midline lipomas surrounding the corpus callosum.1 Believed to result from disruptions in embryonic fusion process, Pai syndrome is among the most under-studied disorders, with only 67 cases reported globally.2 Pericallosal lipomas, though rare, have been linked to neurological issues, including epilepsy and psychiatric manifestations, due to their impact on the corpus callosum. Severe cognitive impairment has been documented in two cases, highlighting the potential for significant neuropsychiatric outcomes despite the syndrome's rarity.2 This report explores the neuropsychiatric manifestations associated with Pai syndrome to enhance understanding of its clinical spectrum and impact on affected individuals.

Description: The patient is a 10-year-old male with ADHD, Autism Spectrum Disorder (ASD), and Pai Syndrome, presenting with severe impulsivity, skin-picking, and irritability despite treatment with clonidine and Quillivant XR. He experiences daily "explosive" episodes triggered by minor events. His Pai Syndrome manifests through distinctive features, including a submucosal cleft palate, a fatty tumor on the corpus callosum, facial skin tags, heart murmur, sacral dimple, hypercholesterolemia, nasal cyst, and a missing right eyelid. His developmental history includes delays due to maternal exposure to synthetic marijuana, alcohol, tobacco, Vicodin, and physical trauma during the first trimester. He has required speech and occupational therapy since early childhood. Psychological testing reveals a Full-Scale IQ of 70 and a specific learning disorder. Previous medications, including Concerta and risperidone, were either ineffective or caused adverse effects. Current treatment involves discontinuing Quillivant XR due to worsening symptoms, starting methylphenidate immediate release, and adjusting clonidine dosing.

Discussion and Conclusion: This case underscores Pai Syndrome's multifaceted nature and its considerable impact on neuropsychiatric functioning. The patient's irritability and impulsivity seem compounded by both physical and developmental challenges. Prenatal substance exposures and stressors, particularly during midline structure formation, may have contributed to his condition.3 A comprehensive treatment strategy with tailored medication adjustments and close follow-up will be crucial in managing his behavioral needs. There is need for further research to elucidate the relationship between Pai Syndrome's physical and neuropsychiatric features so that more effective treatments are developed.

References:

Olivero, Francesca, et al. "Pai Syndrome: A Review." Child's Nervous System: ChNS: Official Journal of the International Society for Pediatric Neurosurgery, U.S. National Library of Medicine, Nov. 2020, www.ncbi.nlm.nih.gov/pmc/articles/PMC7575485/.

Carlos F. Ugas Charcape a 1, et al. "PAI Syndrome Associated with Cerebral Arteriovenous Malformation." Journal of Oral and Maxillofacial Surgery, Medicine, and Pathology, Elsevier, 20 Sept. 2023, www.sciencedirect.com/science/article/pii/S2212555823002090.

"Knowledge on Rare Diseases and Orphan Drugs." Orphanet: Clinical Signs and Symptoms, www.orpha.net/en/disease/sign/1993. Accessed 5 Sept. 2024.

Type of Research: Case Study

Title: Treatment Resistant Depression in Spinocerebellar Ataxia

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Introduction/Background: Spinocerebellar ataxia (SCA) is an autosomal dominant inherited progressive neurodegenerative disease with symptoms including cerebellar ataxia, pyramidal signs, and eye movement disorders.1 It has been linked to psychiatric disorders and symptoms such as depression, anxiety, and issues with impulse control.2 The association with depression in particular has been well studied, with many studies estimating the prevalence of depression in SCA being between 40-60%.3 One study found that of the 300 SCA patients enrolled, 52% had suicidal ideation.1 Other neurodegenerative disorders are known to cause depression through degeneration of brain circuits that are involved in depression, such as Huntington's disease and Parkinson's disease.1 It is unknown whether SCA's association with depression is caused by a similar mechanism, but it is theorized that the cerebellar degeneration could cause depression through degeneration of connections with the frontal lobe as well as other areas of the brain.1 While there is plentiful research on the association with depression, there is a paucity of information on treatment considerations, particularly with treatment resistant depression.

Description: The patient is a 64-year-old male who presents for continuous depression and anxiety for the past 4 years. Symptoms began 4 years prior with a loss of motor skills, tremors, and increasing psychiatric symptoms including depression, anxiety, and persistent suicidal ideation. He has had a 60-pound weight loss in the previous year due to lack of appetite and increased sleeping, and he has consistently had daily suicidal ideation. The patient has no history of psychiatric disorders prior to age 60. Prior to symptom onset he was successful in his career. In the prior 4 years he has had 6 inpatient admissions for depression and suicidal ideation including a two-week admission for inpatient Electroconvulsive Therapy (ECT) which was unsuccessful. He has previously trialed ECT, transcranial magnetic stimulation therapy, bupropion, risperidone, duloxetine, clonazepam, olanzapine, quetiapine, and cariprazine for depression and anxiety unsuccessfully. When he first presented to our clinic, he was on lamotrigine, fluoxetine, and lorazepam, with only lorazepam alleviating anxiety symptoms and seemingly no effect of the other medications on depression symptoms. He was then started on aripiprazole and on follow up showed no improvement.

Discussion and Conclusion: There is limited information on the treatment of depression in SCA. Some studies have mentioned the use of selective serotonin reuptake inhibitors (SSRIs) and cognitive behavioral therapy, but the use of these in these studies is low with less than 35% of patients utilizing these in some studies leading to the possibility of depression being undertreated in SCA patients.3 When considering treatment for treatment resistant depression in SCA patients, there are additional concerns as drug classes such as atypical antipsychotics have the potential of worsening motor symptoms through interactions with the nigrostriatal dopamine pathway. Some case studies have documented treatment of psychotic features in SCA patients with antipsychotics successfully, but there have been no studies on the safety of these medications in these patients.3 While unsuccessful in this patient, transcranial magnetic stimulation could potentially prove useful as a noninvasive treatment option as it is known to have less

systemic side effects than medication.4 Overall, future research needs to be conducted on the safety of these treatments in SCA patients.

References:

1. Lo RY, Figueroa KP, Pulst SM, et al. Depression and clinical progression in spinocerebellar ataxias. Parkinsonism Relat Disord. 2016;22:87-92. doi:10.1016/j.parkreldis.2015.11.021

2. Lin CR, Kuo SH, Opal P. Cognitive, Emotional, and Other Non-motor Symptoms of Spinocerebellar Ataxias. Curr Neurol Neurosci Rep. 2024;24(3):47-54. doi:10.1007/s11910-024-01331-4

3. Karamazovova, S., Matuskova, V., Ismail, Z., & Vyhnalek, M. (2023). Neuropsychiatric symptoms in spinocerebellar ataxias and Friedreich ataxia. Neuroscience and biobehavioral reviews, 150, 105205. https://doi.org/10.1016/j.neubiorev.2023.105205

4. Cosmo, C., Zandvakili, A., Petrosino, N. J., Berlow, Y. A., & Philip, N. S. (2021). Repetitive Transcranial Magnetic Stimulation for Treatment-Resistant Depression: Recent Critical Advances in Patient Care. Current treatment options in psychiatry, 8(2), 47â€“63. https://doi.org/10.1007/s40501-021-00238-y

Type of Research: Case Study exceeds word count limit

Title: Paraneoplastic Anti-NMDA Receptor Encephalitis Causing Psychosis

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Introduction/Background: Anti-N-methyl-D-aspartate receptor (NMDAR) encephalitis is an autoimmune condition caused by autoantibodies against a glutamate subunit (GluN1) of the NMDA receptor found in the central nervous system5. These antibodies can be produced in the presence or absence of an associated neoplasm, infection, or other inciting factor3. While anti-NMDAR encephalitis was first described in 2007, hundreds of cases have allowed researchers to outline a typical demographic and clinical course. It affects primarily children and young adults, with a predominance of cases seen in females. Up to 58% of these affected young female patients have an associated ovarian teratoma1. In young adults, neuropsychiatric symptoms seen in anti-NMDAR encephalitis are classically preceded by a prodromal phase that resembles a viral illness: fever, coryza, and myalgia. This is followed by the acute onset of neuropsychiatric symptoms: seizures, agitation, hallucinations, aggression, and cognitive impairment. Lastly, dysautonomia and coma can develop without treatment7. A recent retrospective study found that anti-NMDAR encephalitis accounted for 1% of all young adult admissions to an intensive care unit1. The purpose of this case study is to discuss a presentation of psychosis due to paraneoplastic anti-NMDA receptor encephalitis, and to recognize its presentation in the pediatric patient in order to promote prompt treatment.

Description: A 15-year-old female with past medical history of anxiety was brought to the hospital with four-day history of increasingly erratic behavior, now presenting with altered mental status and worsening aggression. According to her guardian, the patient has been “speaking gibberish” and mumbling to herself overnight. Upon arrival, the patient was extremely agitated and belligerent, necessitating sedation with intravenous haloperidol and ketamine. She was unable to form complete sentences and repeatedly shouted “stay straight”. Labs were significant for leukocytosis [14.47] with a predominance of neutrophils, and her urine drug screen was positive for tetrahydrocannabinol (THC). Approximately eight hours post-admission, the patient was observed to have an isolated tonic-clonic seizure, which was managed with Ativan. Subsequent electroencephalography (EEG) showed diffuse background slowing. Imaging studies, including head CT, MRI (limited by orthodontic hardware), and chest x-ray were unremarkable. A lumbar puncture revealed increased white blood cell count [44] with normal cerebrospinal fluid (CSF) protein and glucose levels. CSF meningitis panel and cultures were negative. Due to continued agitation and aggressive behavior, the patient was placed in soft restraints and administered continuous intravenous dexmedetomidine for sedation. She developed persistent hypertension, necessitating the initiation of a nicardipine drip. Given the persistence of symptoms for six days, a chest/abdomen/pelvis CT was performed, which revealed a left ovarian teratoma. Subsequently, a left salpingo-oophorectomy was performed on the second day of hospital admission. Given the high suspicion for anti-NMDA encephalitis, pulse-dose methylprednisolone and intravenous immunoglobulin (IVIG) were initiated. The confirmatory autoimmune encephalitis panel returned strongly positive for anti-NMDAR antibodies. Due to minimal response to steroid and IVIG treatment, the patient underwent therapeutic plasma exchange and rituximab therapy. Three weeks after her initial presentation, the patient required intubation and mechanical ventilation for acute respiratory failure. She was administered Ativan for episodes of agitation. Over the course of five days, she was successfully weaned off mechanical ventilation and required fewer doses of Ativan. The patient began to follow commands, engage in conversation with the

staff, and gradually return to her baseline. Five weeks after her initial presentation, she was transferred to a stepdown unit for extensive inpatient rehabilitation.

Discussion and Conclusion: The broadly accepted diagnostic criteria for probable anti-NMDAR encephalitis consists of the following: rapid onset of neuropsychiatric symptoms, an abnormal EEG or CSF pleocytosis, and reasonable exclusion of other disorders. In the case of a positive CSF anti-NMDAR antibody titer, a diagnosis of definite anti-NMDAR encephalitis can be made if the above conditions are also met4. The MRI is often normal or shows nonspecific inflammatory changes in most autoimmune encephalopathies1. In this case presentation, clinical improvement was seen shortly after removal of the ovarian teratoma. It is common to see clinical improvement after removal of a neoplasm and immunotherapy in cases of anti-NMDAR encephalitis with associated paraneoplastic syndrome6. If autoimmune encephalitis is suspected, the mainstay of treatment is to first begin intravenous immunoglobulin and methylprednisolone. If there is no clinical improvement within two weeks, plasmapheresis and/or immunomodulation is typically initiated4. This case shows the importance of recognizing the clinical syndrome and appropriate workup for autoimmune encephalitis, and the prompt initiation of treatment to ensure the best clinical outcomes.

Abbreviations:
CT – computed tomography
MRI – magnetic resonance imaging

References:

1. Dalmau, J., Graus, F. (2018). Antibody-mediated encephalitis. New England Journal of Medicine, 378(9), 840-51. doi: 10.1056/NEJMra1708712.

2. Dalmau, J., Tüzün, E., et. al. (2007). Paraneoplastic anti-N-methyl-D-aspartate receptor encephalitis associated with ovarian teratoma. Annals of Neurology, 61(1), 25-36. doi: 10.1002/ana.21050.

3. Endres, D., Rauer, S., et. al. (2019). Psychiatric presentation of anti-NMDA receptor encephalitis. Frontiers in Neurology, 10(1086). doi: 10.3389/fneur.2019.01086.

4. Graus, F., Titulaer, M., et. al. (2016). A clinical approach to diagnosis of autoimmune encephalitis. Lancet Neurology, 15(1), 391-404. http://dx.doi.org/10.1016/.

5. Huang, Y., Xiong, H. (2021). Anti-NMDA receptor encephalitis: a review of mechanistic studies. International Journal of Physiology, Pathophysiology, and Pharmacology, 13(1), 1-11. ISSN:1944-8171/IJPPP0127015.

6. Sameshima, A., Hidaka, T., et. al. (2011). Anti-N-methyl-D-aspartate receptor encephalitis associated with ovarian immature teratoma. Journal of Obstetrics and Gynecology Research, 37(12), 1883-86. doi: 10.1111/j.1447-0756.2011.01671.x.

7. Varley, J. A., Strippel, C., et. al. (2023). Autoimmune encephalitis: recent clinical and biological advances. Journal of Neurology, 270:4118-4131. https://doi.org/10.1007/s00415-023-11685-3.

Type of Research: Case Study

Title: Unspoken Pain: Navigating Self-Injurious Behavior in a Non-Verbal Patient with MELAS

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Introduction/Background: Self-injurious behavior (SIB) involves deliberate self-harm without suicidal intent and is common in individuals with severe to profound intellectual disability (ID). SIB often manifests as repetitive hitting, biting or scratching. It becomes especially challenging to treat when patients cannot communicate. This case report discusses the treatment of a 29-year-old non-verbal female with profound ID associated with mitochondrial encephalopathy, lactic acidosis, stroke-like episodes (MELAS), who presented with escalating SIB to an inpatient psychiatric hospital in Alabama.

Description: Upon admission, a detailed review of the patient's history through past records and consultation with her mother and group home staff, prompted a reevaluation of her treatment plan. Polypharmacy, sensory overstimulation, mood dysphoria and chronic pain were suspected to be contributing to her presentation. Her home medication regimen included: Benztropine 0.5 mg BID, Tranxene 3.75 mg BID, Depakote 250 mg BID, Haldol 5 mg BID, Zyprexa 10 mg TID, Zoloft 100 mg QD, Zonisamide 100 mg BID, and Trazodone 150 mg QHS. The patient's medications were gradually tapered. Her Tranxene, Trazodone, Benztropine, Zoloft, and Depakote were eventually discontinued. Zyprexa was reduced to 10 mg BID. Zonisamide was maintained for seizure control and chronic pain management. Haldol was dosed at 2.5mg PRN for breakthrough SIB. Gabapentin and Cymbalta were introduced to further address her presumed dysphoric mood and chronic pain. Environmental modifications included education of behavioral staffing, involving family to participate in assessment and care through videoconferencing, and reducing exposure to overstimulation. Over the hospital course these interventions contributed to a marked reduction in non-distractible SIB and the need for PRN medications. Follow-up with the patient's mother confirmed sustained improvement in her daughter's condition post-discharge.

Discussion and Conclusion: Treating SIB in a non-verbal patient with intellectual disability requires a multidisciplinary, patient-centered approach. This approach enables careful management of behavioral disorders with minimal medication, as individuals with ID are highly sensitive to the side effects of psychotropic medications. This case highlights how targeted treatment, including optimizing medication and incorporating environmental and behavioral strategies, can improve outcomes. Although knowledge gaps remain in managing MELAS, this case offers an example of effectively addressing its associated behavioral symptoms.

References:

1. Niyazov DM, Kahler SG, Frye RE. Primary Mitochondrial Disease and Secondary Mitochondrial Dysfunction: Importance of Distinction for Diagnosis and Treatment. Mol Syndromol. 2016;7(3):122-137. doi:10.1159/000446586

2. Hirano M, Ricci E, Richard Koenigsberger M, et al. MELAS: An original case and clinical criteria for diagnosis. Neuromuscul Disord. 1992;2(2):125-135. doi:10.1016/0960-8966(92)90045-8

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

4. How Do Researchers Define Self-Injurious Behavior? Matson JL, Turygin NC. Research in Developmental Disabilities. 2012 Jul-Aug;33(4):1021-6. doi:10.1016/j.ridd.2012.01.009.

5. Self-Injury. Nock MK. Annual Review of Clinical Psychology. 2010;6:339-63. doi:10.1146/annurev.clinpsy.121208.131258. Leading Journal

6. Epidemiology of Self-Injurious Behaviour in Adults With Learning Disabilities. Collacott RA, Cooper SA, Branford D, McGrother C. The British Journal of Psychiatry: The Journal of Mental Science. 1998;173:428-32. doi:10.1192/bjp.173.5.428.

Type of Research: Case Study

Title: False Positive Oxycodone Results in Patients Receiving Opioid Antagonists

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Introduction/Background: Urine drug testing plays a significant role in healthcare, including monitoring adherence to certain medications and detecting illicit drug use. Enzyme-mediated immunoassays (EIAs) are commonly employed as UDS tests for cost-effectiveness, feasibility, and sensitivity [1]. However, EIAs have limitations, such as decreased likelihood of binding to synthetic drugs, cross-reactivity with certain medications, and potential false positives [1]. Since false positives can negatively impact individuals' lives and overall treatment, confirmatory testing, such as chromatographic methods, warrants consideration. We present two cases involving false positives for oxycodone in patients treated with Naltrexone and Olanzapine/Samidorphan (Lybalvi®).

Description: The first case involves a 25-year-old female in addiction treatment for problematic alcohol and cannabis use. Initial UDS was positive for THC. One month after starting Naltrexone 50 mg daily, her UDS was positive for THC and oxycodone. She denied intentional oxycodone use and was concerned about continuing Naltrexone, given the results. Next, a 52-year-old female with polysubstance use and bipolar disorder sought treatment while trying to regain custody of her children. Medications included Olanzapine/Samidorphan 15 mg/10 mg QHS, Duloxetine 30 mg QD, and Oxcarbazepine 600 mg BID. Despite reported abstinence, her UDS was positive for oxycodone, and social services were concerned about this positive result. Four weeks after discontinuing Olanzapine/Samidorphan due to weight gain, her UDS was negative. In both cases, initial EIA urine screens were presumptively positive for oxycodone, but LC-MS/MS confirmatory tests were negative for Noroxycodone, Oxycodone, and Oxymorphone. False positives were suspected due to Naltrexone and Samidorphan, respectively.

Discussion and Conclusion: Naltrexone and Samidorphan act as mu-opioid receptor antagonists, and their similar chemical structures likely led to cross-reactivity with the oxycodone immunoassay [2,3]. While Naltrexone has previously been implicated as the causal agent of false positive results for oxycodone on immunoassay, this is the first case in literature to our knowledge that discusses Samidorphan in combination with Olanzapine [4,5]. The prescribing information for Olanzapine/Samidorphan (Lybalvi®) was recently updated to warn of possible false positives for opioids and recommends alternative testing methods for confirmation [2]. These cases highlight the importance of urine drug testing literacy and the need for continued education on the topic to reduce stigma and enhance patient care.

References:

1. Keary CJ, Wang Y, Moran JR, Zayas LV, Stern TA. Toxicologic testing for opiates: understanding false-positive and false-negative test results. Prim Care Companion CNS Disord. 2012;14(4):PCC.12f01371. doi:10.4088/PCC.12f01371

2. Lybalvi. Prescribing information. Alkermes; 2024. Accessed September 2, 2024. https://www.lybalvi.com/lybalvi-prescribing-information.pdf

3. Elena R. Beauregard, Elizabeth G. Maguire; Opioid-positive urine drug screen during treatment with oral naltrexone and the clinical implications. Mental Health Clinician 1 April 2024; 14 (2): 102-106. doi: https://doi.org/10.9740/mhc.2024.04.102

4. Singh D, Saadabadi A. Naltrexone. [Updated 2023 May 30]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK534811/

5. Cornel N. Stanciu & Samantha Gnanasegaram (2020) Naltrexone and Its Noroxymorphone Minor Metabolite – A Case Report, Journal of Psychoactive Drugs, 52:2, 169-171, DOI: 10.1080/02791072.2019.1649507

Type of Research: Case Study

Title: Vagus Nerve Stimulation Therapy: An Unforeseen Option for Treatment Resistant Depression

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Introduction/Background: Vagus nerve stimulation (VNS) therapy was originally developed for the management of epilepsy, and is now FDA approved for use in treatment resistant depression (TRD). It is hypothesized that VNS imposes serotonergic effects on the brain. In this case report we discuss a patient participating in a double-blind randomized controlled trial with VNS therapy.

Description: This patient is a 56-year-old female with a history of Bipolar 1 disorder, Generalized Anxiety Disorder (GAD), and Post Traumatic Stress Disorder (PTSD) that presents for evaluation of persistent depressive episodes with infrequent mania. She underwent Transcranial magnetic stimulation (TMS), Electroconvulsive therapy (ECT), and multiple medications regimens with minimal effect. The patient had VNS device implantation in June of 2023. At 6 months post-implantation, she reported no improvement. At 8 months post-implantation, the patient reported improvement in the severity and frequency of depressive episodes. At 11 months post-implantation, she reported a decrease in frequency of depressive episodes and increased interest in her usual hobbies.

Discussion and Conclusion: Depression is becoming increasingly prevalent within the U.S. In 2021, it was estimated that 30.9% of cases will be treatment resistant. TRD is often classified as failure of improvement after two separate antidepressant trials. Treatment for TRD is complex and can involve medication augmentation, psychotherapy, ECT, TMS, deep brain stimulation (DBS), and VNS. ECT is commonly used in TRD patients, but long-term use is associated with memory complications and continuous treatment to maintain improvement. Studies analyzing patient outcomes in those treated with VNS for epilepsy found reductions in depression ratings. Studies have found that VNS therapy shows improvement in long-term depressive symptoms. Although this patient is participating in a double-blind RCT, her improvement is encouraging, if she is in the experimental group. With her previous alternative treatments, VNS therapy could be a favorable option for her to minimize both depressive symptoms as well as repeated procedures.

This case highlights the need for providers to remain cognizant of alternative treatment options for TRD. Specifically, VNS therapy shows promising data and should continue to be studied and considered in the treatment of TRD.

References:

Nemeroff CB, Mayberg HS, Krahl SE, et al. VNS therapy in treatment-resistant depression: clinical evidence and putative neurobiological mechanisms [published correction appears in Neuropsychopharmacology. 2006 Oct;31(10):2329]. Neuropsychopharmacology. 2006;31(7):1345-1355. doi:10.1038/sj.npp.1301082

Zhdanava 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. Published 2021 Mar 16. doi:10.4088/JCP.20m13699

Voineskos D, Daskalakis ZJ, Blumberger DM. Management of Treatment-Resistant Depression: Challenges and Strategies. Neuropsychiatr Dis Treat. 2020;16:221-234. Published 2020 Jan 21. doi:10.2147/NDT.S198774

Type of Research: Case Study

Title: Case Analysis: Comorbidities Confounding the Diagnosis of Autism Spectrum Disorder

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Introduction/Background: Autism Spectrum Disorder (ASD) is a complex neurodevelopmental condition characterized by deficits in social communication and interaction, alongside restricted, repetitive patterns of behavior, interests, or activities.1 However, schizophrenia and other psychotic disorders, particularly when accompanied by substance abuse, can obscure the core symptoms of ASD, leading to delayed or missed diagnosis.2,3

Patients with schizophrenia often present with symptoms that mimic some social deficits seen in ASD.4 This interaction can be further complicated by substance use disorders, which may exacerbate psychiatric symptoms and hinder assessment.5

Description: We present the case of a 37-year-old female with a history of sexual abuse and a family history of Fragile-X Syndrome. She was admitted to the inpatient facility with persecutory delusions, disorganized behavior, and comorbid substance use, leading to a diagnosis of Other Specified Schizophrenia Spectrum Disorder. However, throughout her stay, the patient exhibited signs of ASD, including poor eye contact, lack of facial expression, abnormal social approach, abnormal posture, phrase repetition, and difficulties maintaining and understanding social relationships.1 These suggest an underlying ASD that, while not diagnosed, may have been masked by schizophrenia and substance use.3,4,5

Discussion and Conclusion: ASD is a complex condition with broad symptom presentation, often making it challenging to diagnose in the presence of comorbidities.2,3,4,5 In our case, the primary diagnosis of Other Specified Schizophrenia combined with history of substance abuse presented barriers to identifying possible ASD due to overlapping symptoms. Conversely, ASD-like symptoms contributed to an atypical presentation of psychosis, thus obscuring the overall clinical picture and preventing specific diagnosis.

This case underscores the importance of a careful approach to assessing complex psychiatric presentations. Clinicians should remain vigilant of the possibility of underlying ASD, as early recognition can enable targeted interventions, potentially improving long-term outcomes for these patients.1,3,4

The significant overlap of genetic mechanisms between ASD and schizophrenia provides a promising explanation of comorbidity.6 Moreover, the strong genetic disposition of ASD enables the application of GWAS findings to clinical practice. Ex ante diagnostic metrics principally derived from family history (similar to established methods like Alzheimer's-disease-by-proxy scores) could provide early evidence of ASD in cases where clinical diagnosis is unavailable and/or difficult to make.7

References:

1. Guerrera S, Pontillo M, Chieppa F, et al. Autism spectrum disorder and early psychosis: A narrative review from a neurodevelopmental perspective. Frontiers in Psychiatry. 2024;15:1362511. https://doi.org/10.3389/fpsyt.2024.1362511

2. Doshi-Velez F, Ge Y, Kohane I. Comorbidity clusters in autism spectrum disorders: an electronic health record time-series analysis. Pediatrics. 2014;133(1). https://doi.org/10.1542/peds.2013-0819

3. Kiyono T, Morita M, Morishima R, et al. The prevalence of psychotic experiences in autism spectrum disorder and autistic traits: A systematic review and meta-analysis. Schizophrenia Bulletin Open. 2020;1(1). https://doi.org/10.1093/schizbullopen/sgaa046

4. Ribolsi M, Fiori Nastro F, Pelle M, et al. Recognizing psychosis in autism spectrum disorder. Frontiers in Psychiatry. 2022;13:768586. https://doi.org/10.3389/fpsyt.2022.768586

5. Haasbroek, H., Morojele, N. A Systematic Literature Review on the Relationship Between Autism Spectrum Disorder and Substance Use Among Adults and Adolescents. Rev J Autism Dev Disord 9, 1â€“20 (2022). https://doi.org/10.1007/s40489-021-00242-1

6. Grove J, Ripke S, Als TD, et al. Identification of common genetic risk variants for autism spectrum disorder. Nature Genetics. 2019;51(3):431-444. https://doi.org/10.1038/s41588-019-0344-8

7. Marioni, R.E., Harris, S.E., Zhang, Q. et al. GWAS on family history of Alzheimer's disease.Transl Psychiatry 8, 99 (2018). https://doi.org/10.1038/s41398-018-0150-6

Type of Research: Case Study

Title: Navigating Anorexia Nervosa in an 11-Year-Old Male: A Case Study

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Introduction/Background: Anorexia nervosa (AN) is a severe eating disorder characterized by self-starvation and an intense fear of gaining weight, leading to significant weight loss and malnutrition.1 It is predominantly observed in adolescent females, with a one-year prevalence of 0.16% for females and 0.09% for males in the United States.2 The disorder typically manifests during adolescence, presenting with weight loss, distorted body image, and an obsessive focus on weight and shape. AN is classified into two subtypes: the restricting type, where weight loss is achieved through dieting, fasting, or excessive exercise, and the binge-eating/purging type, where the individual eats large amounts of food in a short time followed by vomiting or using laxatives or diuretics.1

Description: An 11-year-old Latino male was sent from his pediatrician's office to be admitted to the children's hospital due to concerns for malnutrition and bradycardia. The pediatrician found that in the 3 months since the patient was last seen his weight decreased from 100lbs to 73lbs. The admitting team determined that the patient had been engaging in a pattern of restrictive eating and frequent exercise. Psychiatry was consulted and made a diagnosis of AN but could not rule out mood disorders. The patient was started on mirtazapine to target mood symptoms and stimulate appetite. When his medical condition improved, he was discharged to follow up with an outpatient therapist, psychiatrist, and pediatrician through an integrated care model.

Discussion and Conclusion: This case describes a presentation of AN in a patient of unusual age, gender, and cultural background.3,4 Despite its atypical presentation, this case highlights the difficulty in treating AN and the importance of a multidisciplinary approach to treatment. There remains debate on pharmacologic treatment of AN so flexibility and responsiveness from the care team are key for successful treatment.5 We discuss pharmacologic, as well as psychodynamic and cultural considerations for those treating this complex disorder in clinical practice.

References:

1. Parpia R, Spettigue W, Norris ML. Approach to anorexia nervosa and atypical anorexia nervosa in adolescents. Can Fam Physician. 2023 Jun;69(6):387-391. doi: 10.46747/cfp.6906387. PMID: 37315981; PMCID: PMC10266399.

2. Deloitte Access Economics. The Social and Economic Cost of Eating Disorders in the United States of America: A Report for the Strategic Training Initiative for the Prevention of Eating Disorders and the Academy for Eating Disorders. June 2020.

3. Nuñez A, González P, Talavera GA, Sanchez-Johnsen L, Roesch SC, Davis SM, Arguelles W, Womack VY, Ostrovsky NW, Ojeda L, Penedo FJ, Gallo LC. Machismo, Marianismo, and Negative Cognitive-Emotional Factors: Findings From the Hispanic Community Health Study/Study of Latinos Sociocultural Ancillary Study. J Lat Psychol. 2016 Nov;4(4):202-217. doi: 10.1037/lat0000050. Epub 2015 Oct 19. PMID: 27840779; PMCID: PMC5102330.

4. Caballero AE. Understanding the Hispanic/Latino patient. Am J Med. 2011 Oct;124(10 Suppl):S10-5. doi: 10.1016/j.amjmed.2011.07.018. PMID: 21939793.

5. Frank GKW. Pharmacotherapeutic strategies for the treatment of anorexia nervosa - too much for one drug? Expert Opin Pharmacother. 2020 Jun;21(9):1045-1058. doi: 10.1080/14656566.2020.1748600. Epub 2020 Apr 13. PMID: 32281881; PMCID: PMC7491209.

Type of Research: Case Study

Title: The Argument for Education: A Case Report of MDD Following Screening for Terminal Illness.

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Introduction/Background: The Beta Amyloid 42/40 Serum Ratio is a predictive test for beta-amyloid plaques in the brain for patients with MCI or Dementia. A positive test is associated with a positive amyloid PET and aids in the diagnosis of Alzheimer's dementia. We present a patient who presented to the Behavior Health Unit with MDD and suicidal ideations who recently received a positive Beta-Amyloid 42/40 Serum Ratio test.

Description: This patient is a 73-year-old female with a past medical history of glaucoma, OSA, and insomnia who presented to the ED due to suicidal ideations. The patient recently saw a neurologist who ordered a Beta Amyloid 42/40 ratio test and received a positive result. After receiving this news, she has had increased anxiety and fear of developing dementia, along with the suspicion that it was due to her chronic use of a sleeping aid medication. On admission, a SLUMS screening was performed that resulted in a 22/30, indicating mild cognitive impairment.

Discussion and Conclusion: Depression is becoming increasingly more common, with an estimated lifetime prevalence of 12%, and studies have shown an upward trend in recent years. Receiving a life-altering medical diagnosis can impact patients both emotionally and physically, and providers must remain aware of the possible detriment that can result when providing education on diagnostic results. This patient was educated on the positivity of a Beta Amyloid 42/40 ratio test, indicating that she is at increased risk of developing Alzheimer's dementia. Given her age, she is at risk of developing memory deficits associated with normal aging, but difficulty is encountered when delineating between normal aging and dementia. Studies have shown that patients receiving diagnostic results for specific medical conditions (cancer, chronic lung disease, heart disease, arthritis) predispose them to concurrent depression as a result. There is a scarcity in the literature that describes the impact of receiving positive predictive tests indicative of terminal illnesses. This leads us to ask, as screening for terminal illnesses becomes more and more prevalent, how do we, as physicians, help guide and educate our patients through testing, results, and life after diagnosis?

References:

Weber DM, Taylor SW, Lagier RJ, Kim JC, Goldman SM, Clarke NJ, Vaillancourt DE, Duara R, McFarland KN, Wang WE, Golde TE, Racke MK. Clinical utility of plasma Aβ42/40 ratio by LC-MS/MS in Alzheimer's disease assessment. medRxiv [Preprint]. 2023 Dec 14:2023.12.12.23299878. doi: 10.1101/2023.12.12.23299878. Update in: Front Neurol. 2024 Mar 25;15:1364658. doi: 10.3389/fneur.2024.1364658. PMID: 38168329; PMCID:

Bains N, Abdijadid S. Major Depressive Disorder. [Updated 2023 Apr 10]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK559078/

Goodwin RD, Dierker LC, Wu M, Galea S, Hoven CW, Weinberger AH. Trends in U.S. Depression Prevalence From 2015 to 2020: The Widening Treatment Gap. Am J Prev Med. 2022;63(5):726-733. doi:10.1016/j.amepre.2022.05.014

Polsky D, Doshi JA, Marcus S, et al. Long-term risk for depressive symptoms after a medical diagnosis. Arch Intern Med. 2005;165(11):1260-1266. doi:10.1001/archinte.165.11.1260

Type of Research: Original Research exceeds word count limit

Title: Racial Disparities in Perinatal Insomnia, and Treatment Engagement and Outcomes: A Literature Review

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Introduction/Background: Historically, African American (AA) women have had little to no access to health care and treatment, especially in obstetrics and gynecology, and this inequity is rooted in slavery. Slave owners forced black women to procreate with limited access to health care. As a result of this accumulation of disadvantages across generations, AA women face a public health crisis. Black women are disproportionally burdened with higher rates of maternal mortality, anemia, cardiovascular disease, and diabetes. The Center for Diseae Control and Prevention (CDC) reported that AA women were three times more likely to die from pregnancy-related complications compared to White women. Underlying conditions such as hypertension, cardiovascular disease, diabetes, and obesity are all associated with maternal morbidity, and in all these conditions, racial disparities persist. There is reason to believe that perinatal insomnia can exacerbate these elevated risk factors already persistent among AA women. These conditions increase the risk of complications in perinatal health and infant mortality.

Methods: An electronic literature search was performed of Medline, Directory of Open Access Journals, Google Scholar, and PubMed from 1989 to 2024. This literature review includes results from double-blind, placebo-controlled studies, population-based studies and review articles.

Results: A key finding in an epidemiology study found that black women during pregnancy exhibited a heightened risk of inflammation associated with poor sleep quality. In this observational study, 79 black women followed the PSQI subscale to measure sleep quality, and it was found that black women experienced 10.2 times greater likelihood of preterm birth and poor sleep quality, and heightened circulating interleukin (IL)-8 levels compared to white women. Poor sleep quality, shorter sleep duration, and greater sleep latency were associated with elevated serum IL-8 in this study. This study suggests that poor sleep quality is associated with shorter gestation and a greater risk of preterm births, and the mediating role of this relationship is increased inflammation associated with shortened sleep duration. Elevated IL-8 is particularly consequential in that studies have seen elevated serum IL-8 plays a role in the development of atherosclerosis, and endothelial dysfunction, which can impede placental blood flow and is implicated in an increased risk of preterm delivery.

Insomnia and depression are strongly correlated, a study examined the prevalence of insomnia and if it is a predictor of postpartum depression. This longitudinal, population-based study presented data from (>2,000 women) a hospital in Norway and found that among women with depression before pregnancy, insomnia during pregnancy may be a marker of postpartum depression. Knowledge about this may warn clinicians to increased risk of postpartum depression in women experiencing insomnia during pregnancy. This study does present with limitations, in that women with depressive symptoms were less likely to complete the study. The postpartum period is already associated with a heightened risk of depression, but this study suggests that insomnia is a helpful marker to help clinicians conduct an early intervention.

Discussion and Conclusion: Digital Cognitive Behavioral Therapy (CBT) has an added benefit that is more accessible and convenient for expecting mothers. Studies also show that it is an effective intervention for improving insomnia symptoms during pregnancy. Recent studies have addressed the

disparity of black women disproportionately affected with prenatal insomnia. CBT has been effective in treating insomnia and prenatal insomnia, but racial minorities have exhibited poor outlines from prenatal care compared to white patients, and this trend persists in black women with prenatal insomnia. In another study, results found that CBT treatment tailored to black women was more effective and exhibited high rates of treatment engagement, and improved symptoms in this study were attributed to the culturally tailored intervention.

There is more literature to suggest that access to digital health tools that are culturally tailored can increase engagement and outcomes in black and Latinx women; however, very few of these tools are tailored for racial/ethnic minorities, despite them being the population with the greatest need for it.

References:

Zhou ES, Ritterband LM, Bethea TN, Robles YP, Heeren TC, Rosenberg L. Effect of Culturally Tailored, Internet-Delivered Cognitive Behavioral Therapy for Insomnia in Black Women: A Randomized Clinical Trial. JAMA Psychiatry. 2022;79(6):538–549. doi:10.1001/jamapsychiatry.2022.0653

Chandler R, Guillaume D, Parker A, Wells J, Hernandez ND. Developing culturally tailored mhealth tools to address sexual and reproductive health outcomes among Black and Latina women: a systematic review. Health Promot Pract. 2022;23 (4):619–630. https://doi.org/10.1177/15248399211002831

Singh, G. K., & Yu, S. M. (2018). Infant Mortality in the United States, 1915-2017: Large Social Inequalities have Persisted for Over a Century. International Journal of Maternal and Child Health and AIDS, 8(1), 19-31. https://doi.org/10.21106/ijma.271

Singh, G. K., & Yu, S. M. (2018). Infant Mortality in the United States, 1915-2017: Large Social Inequalities have Persisted for Over a Century. International Journal of Maternal and Child Health and AIDS, 8(1), 19-31. https://doi.org/10.21106/ijma.271

Type of Research: Original Research

Title: Exploring the Interplay of Sleep and Nicotine Vaping in Adolescents: An Evidence- Based Narrative Review

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Introduction/Background: Although combustible tobacco cigarettes (CC) use has decreased amongst adolescents, the rapid emergence of popular vaping products (e-cigs, e-cigars, e-hookahs, JUUL) has transformed the landscape of adolescent substance use in the US, becoming a critical public health issue. Nicotine is the most commonly vaped substance, enticing users through appealing packaging and flavors. Though the long-term sequelae of vaping nicotine are, at present, poorly understood, the short-term effects include tachycardia, coughing, and wheezing. While inhaling nicotine from CC is known to induce sleep disruption, the implications from vaping nicotine are less clear, especially amongst adolescents. Here, we describe an evidence-based narrative review exploring the interplay of sleep and nicotine vaping in adolescents.

Methods: PubMed search from 2006 to 2024 using the keywords “Nicotine sleep adolescents,” “vaping sleep adolescents,” and “e-cigarette sleep adolescents” yielded 159 articles. Activating the filters “Humans,” “English,” and “Age-Birth-18 years” reduced number to 124 articles. Independent screening of abstracts by three authors (SB, BGB, and AR) for cross-sectional studies that described sleep disturbances yielded 9 articles for inclusion. The outcome measures of sleep duration and insufficient sleep were assessed categorically in self-reported hours. Sleep latency and daytime drowsiness were assessed using the Pittsburgh Sleep Quality Index (PSQI). The data from various cross-sectional surveys were pooled together.

Results: A combined sample size of 106,628 adolescents (aged 12-18 years, males=females) was analyzed. E-cigarettes were the most common vaping devices used, and e-cig users and dual (e-cigs + CC) users had increased odds of reporting <7-8 hours of sleep on school nights when compared to non- users. E-cigs are more likely to have insufficient sleep than those who only used CC. In adolescent males, dual use was associated with increased sleep latency, as measured by the PSQI.

Discussion and Conclusion: Survey studies indicate vaping nicotine and dual product users may be associated with sleep disturbances in adolescents. Further investigation through longitudinal studies are needed to determine factors such as the causal relationship, dose-response and product-specific effects. Clinicians should educate children, adolescents, and parents about potential detrimental effects of the interplay between vaping and sleep during healthcare visits or school seminars.

References:

1) Association between use of electronic vaping products and insufficient sleep among adolescents: Findings from the 2017 and 2019 YRBS. Philip Baiden, Samantha P Spoor, Julia K Nicholas, Fawn A Brown, Catherine A LaBrenz, Christine Spadola

2) Vaping and Sleep as Predictors of Adolescent Suicidality. Cody W Welty, Lynn B Gerald, Uma S Nair, Patricia L Haynes

3) Sleep deprivation and adolescent susceptibility to vaping in the United States. Kristen D Holtz, Andrew A Simkus, Eric C Twombly, Morgan L Fleming, Nicole I Wanty

4) Assessing the potential impact of age and inhalant use on sleep in adolescents. Clare Kamini Malhotra, Deepti Gunge, Ira Advani, Shreyes Boddu, Sedtavut Nilaad, Laura E Crotty Alexander

5) Risk Factors and Medical Symptoms Associated With Electronic Vapor Product Use Among Adolescents and Young Adults. Sarah E Benyo, Tyler J Bruinsma, Elizabeth Drda, Jodi Brady-Olympia, Steven D Hicks, Sue Boehmer, Robert P Olympia