APPA 2020 Spring Meeting Resident Poster Presentation

Abstract 20-1-01

Title: Distal Trisomy 15q

Authors: Nicholas Rivers, Dusty Trotman, Janaki Nimmagadda M.D., Clinton Martin M.D.

Case:
An 8yo male with disruptive mood dysregulation disorder, intellectual disability, attention deficit disorder, insomnia, dysmorphic features and scoliosis presented to the psychiatrist for management of his behavior. His caretaker described a pattern of defiant and asocial behavior, including intentional defecation around the home, scratching and threatening to harm his family, cursing outbursts, and destructive tendencies. In addition, he has a history of head banging and household chemical ingestions. All labs were within normal limits. Pt is in the first percentile for height and weight, has prominent thoracic scoliosis, as well as craniofacial abnormalities including broad nasal bridge, small triangular mouth, and micrognathia. Previous genetic analysis revealed a duplication on the long arm of chromosome 15.

Discussion:
Trisomy 15q is a rare chromosomal disorder defined by partial duplication of the distal long arm of chromosome 15. Since originally described in 1974 by A. Fujimoto, less than 50 cases of trisomy 15q have been reported[1,2]. Complete Trisomy 15 is a lethal condition, while mosaic and partial trisomies can be viable[3]. In most cases, distal trisomy 15q is passed down from a balanced translocation in one of the parents[1]. However, there have been reports of de novo duplication[4].

The common clinical features of Trisomy 15q are variable but include[1]:

- Intellectual disability
- Distinctive head and facial abnormalities
    - Microcephaly from craniosynostosis
    - Abnormal frontal and occipital prominence with a sloping forehead
    - Micrognathia
    - Downward slant of palpebral fissures
    - Strabismus
    - Broad nasal bridge with bulbous nose
- Growth abnormalities
- Severe scoliosis
- Heart defects
- Predisposition to hematologic malignancy[5]

The location and size of the duplication determines the resulting phenotype. The gene for IGF1R is located at 15q26.3[6]. Duplication can lead to overgrowth, or in the case of our patient, interruption can lead to undergrowth[6]. The long arm of chromosome 15 is also implicated in other conditions. Imprinting abnormalities of 15q11-q13 lead to the intellectual and behavioral phenotypes of Prader-Willi and Angelman's syndrome[7]. While our patient has never undergone IQ testing, his comorbid behavioral issues from trisomy 15q may be due to more than intellectual disability alone.

References:
1. Chromosome 15, Distal Trisomy 15q. Available at: https://rarediseases.org/rare-diseases/chromosome-15-distal-trisomy-15q/. Accessed February 13 2020.

2. Fujimoto A, Towner JW, Ebbin AJ, Kahlstrom EJ, Wilson MG. Inherited partial duplication of chromosome No. 15. J Med Genet 1974; 11:287-291.
3. Philipp T, Terry J, Feichtinger M, Grillenberger S, Hartmann B, Jirecek S. Morphology of early intrauterine deaths with full trisomy 15. Prenat Diagn 2018; 38:267-272.
4. Kim EY, Kim YK, Kim MKet al. A case of de novo duplication of 15q24-q26.3. Korean J Pediatr 2011; 54:267-271.
5. Smith SR, Rowe D. Trisomy 15 in hematological malignancies: six cases and review of the literature. Cancer Genet Cytogenet 1996; 89:27-30.
6. Cannarella R, Mattina T, Condorelli RAet al. Chromosome 15 structural abnormalities: effect on IGF1R gene expression and function. Endocr Connect 2017; 6:528-539.
7. Kalsner L, Chamberlain SJ. Prader-Willi, Angelman, and 15q11-q13 Duplication Syndromes. Pediatr Clin North Am 2015; 62:587-606.

Abstract 20-1-02

Title: 49,XXXXY Klinefelter Syndrome

Authors: Jennifer Lamar MS3, Janaki Nimmagadda M.D., Clinton Martin M.D., Kathrine Moody M.D.

Learning Objectives:

1. Understand how 49 XXXXY Klinefelter Syndrome occurs
2. Recognize the challenges faced with 49 XXXXY Klinefelter Syndrome
3. Management and treatment options available
4. Importance of early intervention

Case Presentation: 26-year-old short, thin, Caucasian male with 49, XXXXY Klinefelter Syndrome presented with functionally impairing hypersexuality and impulsivity. The patient underwent genetic testing at birth after presenting with low set ears and microphallus. He had marked physical and mental delays during childhood with an IQ of 45. He had limited verbal skills with an inability to read, write, and perform basic mathematics. His medical history included brittle bones treated with biweekly Testosterone injections, olecranon abnormalities, dysmorphic facial features, recurrent upper respiratory and urinary tract infections, anxiety, and repetitive behaviors, obsessions, and compulsions.

At 16 years, he sought treatment for hypersexuality and impulsivity, which was unresponsive to Paxil, but well controlled with Zoloft. His current dose of Zoloft was 50 mg but his parents stated an increase in inappropriate sexual behaviors and gestures which was impairing his sleep, public decency, and volunteer employment. During the interview he was quiet and timid, shielding his face with his hands.

Discussion: 49, XXXXY Klinefelter Syndrome occurs from random double maternal nondisjunction. It is the rarest and most severe form of Klinefelter Syndrome. Although there are common features among all Klinefelter variants, each additional X chromosome alters the phenotype. In additional to dysmorphic facial features, short stature, musculoskeletal abnormalities, these individuals have a significant decline in cognitive function, marked impairment in communication and social skills, and fluctuating behavioral issues. Care for these individuals requires a multi-disciplinary approach. Albeit they have cognitive impairments, these individuals have normal non-verbal skills and visual perception leaving these individuals with a discordance between receptive and expressive communication skills. Issues such as impulsivity, irritability, hyperactivity, anxiety, temper outbursts, and obsessive-compulsive behaviors are commonly reported. With delayed speech and motor development, early interventions are imperative to promote healthy alternative ways of learning and interacting with society. Using sign language as a means of communication and implementing mostly visual and constructive tasks as a learning style may lessen the behavioral patterns experienced by these individuals. Pharmacological therapies are effective

for moderate to severe behavioral problems, but early intervention with consistent behavior management based on a reward system is the most effective therapy.

Abstract 20-1-03

Title: Autonomic nervous system dysfunction in autism spectrum disorder

Authors: Shyla Hossain, MA; Tina Jackson, MD; Sandra Parker, MD.

Summary: Autism spectrum disorder is associated with autonomic nervous system dysfunction. Patients with autism may not physiologically respond in the expected manner during times of stress or illness. Therefore, acute clinicians must be alert to the subtle (and sometimes not so subtle) differences seen in diagnostic indicators and clinical signs while assessing these patients.

We present a case of a 17-year-old African-American male with autism who presented to the PICU with septic shock. He was treated for his illness and, despite showing overall improvement, continued to have tachycardia. The primary team consulted with psychiatry to request consideration of discontinuation of his antipsychotics in order to normalize his heart rate.

References:

1. Bharath R, Moodithaya SS, Bhat SU, Mirajkar AM, Shetty SB. (2019). Comparison of physiological and biochemical autonomic indices in children with and without autism spectrum disorders. *Medicina,* 55:346-357.
2. Kushki A, Drumm E, Mobarak MP, Tanel N, Dupuis A, Chau T, et al. (2013) Investigating the autonomic nervous system response to anxiety in children with autism spectrum disorders. *PLoS One,* 8(4):e59730.
3. Ming X, Patel R, Kang V, Chokroverty S, Julu PO. (2016). Respiratory and autonomic dysfunction in children with autism spectrum disorders. *Brain & Development,* 38:225-232.
4. Quadt L, Critchley HD, Garfinkel SN. (2018). The neurobiology of interoception in health and disease. *Ann NY Acad Sci,* 1428:112-128.
5. Rees CA. (2014). Lost among the trees? The autonomic nervous system and paediatrics. *Arch Dis Child,* 99:552-562.

Abstract 20-1-04

Title: A Case of Tourette's Disorder

Authors: Meghana Gaini, Janaki Nimmagadda M.D., Clinton Martin M.D.

Summary: The patient is a 6-year-old boy with a past medical history of ADHD who is seen for the management of severe tics and behavior problems. His symptoms began at 3 years of age and at age 5, he had to be pulled out of public school due to worsening behavior problems. He currently suffers from both motor and vocal tics, characterized by neck jerking and humming. The tics are worsened by stress. He was first prescribed methylphenidate for his ADHD symptoms. At a follow up visit, his parents noted that since starting the medication, his tics had worsened. He was then prescribed guanfacine as an adjunct to the methylphenidate, which improved the severity of the tics. At his fourth clinical visit, the patient and his father unfortunately noted that his tics had returned to full severity and the neck jerks were causing the child pain, which prompted a referral for HRT.

The optimal management of tic disorders should address a patient's hierarchy of impairments. A physician's focus should be on treating the most socially and occupationally debilitating condition, which

is usually the comorbid condition, such as ADHD or OCD, and then continuing to monitor the child's tics. Medications approved for the management of tics include 1st and 2nd generation antipsychotics. Alpha-2 adrenergic agonists, such as clonidine and guanfacine are indicated for the treatment of tics and comorbid ADHD. Some patients have described worsening of their tics due to stimulant treatment for ADHD, but this association has not been proven in the literature. In addition to medication, it is very important to educate the family and teachers about the course and the prognosis of tic disorders. Treatment planning should include accommodations in the classroom such as an Individualized Education Plan/504 Plan. Children with moderate to severe tics and/or have comorbid conditions that respond to behavioral therapy should be considered for Comprehensive Behavioral Intervention for Tics (Habit Reversal Training), which entails awareness training, developing a competing response to the urge to tic, and social support. This service has been shown to significantly reduce tic severity and improve function.

References:

1. Murphy, Tanya K. et al. Practice Parameter for the Assessment and Treatment of Children and Adolescents With Tic Disorders. Journal of the American Academy of Child & Adolescent Psychiatry, Volume 52, Issue 12, 1341 – 1359.

2. American Psychiatric Association. The diagnostic and statistical manual of mental disorders (5th ed.; DSM-V). Arlington, VA: American psychiatric Association: 2013.

3. Leckman JF, Zhang H, Vitale A, et al. Course of tic severity in Tourette syndrome: the first two decades. Pediatrics. 1998;102(1 Pt 1):14–19. doi:10.1542/peds.102.1.14.

4. Yael D, Vinner E, Bar-Gad I. Pathophysiology of Tic Disorders. Volume 30. Issue 9. 1171-1178. doi: 10.1002/mds.26304.

Abstract 20-1-05

Title: In search of 'Pixie Dust': Neuropsychiatric sequelae of Williams syndrome and its treatment

Authors: Darshana Pai, M.D.; Lori Lowthert, M.D.

Introduction:

Amongst genetic disorders, Williams syndrome (WS) is rare with a prevalence of 1 in 20000[1]. Williams syndrome is a sporadically occurring rare developmental disorder characterized by hemizygous deletion of approximately 25-28 genes on chromosome 7q11.23[2]. Individuals with WS have distinct phenotype of dysmorphic facies also known as elfin or pixie facies, cardiovascular disease, mental retardation, a characteristic cognitive profile and idiopathic hypercalcemia. The deleted portion of the chromosome includes ELN gene which codes for structural protein elastin, an important component of elastic fibers found in many connective tissue organs. In WS individuals tend to be hyper sociable, very friendly and empathetic[3]. Anxiety is one of the most dominant features of WS with prevalence ranging from 16.5-82.2 %[4,5], with considerable variability in prevalence estimates, possibly due to methodological differences[6]. There is limited literature on the pharmacologic treatment of anxiety in WS.

We describe the case of a middle-aged male with WS who presented with suicidal ideation, Attention Deficit Hyperactivity disorder (ADHD) and marked anxiety which was successfully treated with Bupropion.

Case presentation:

A 41-year-old male presented was referred from ER to our hospital for evaluation of suicidal ideation. He had depressed mood, anhedonia, decreased appetite, insomnia, marked anxiety, lack of concentration,

feelings of worthlessness, low self esteem and suicidal ideation. He had an ongoing stressor of strained interpersonal relation with his wife who filed for divorce. There were some concerns raised by the patient that his wife could be cheating on him which appeared like obsessional jealousy but did not appear delusional in intensity. He had dysmorphic facial features such as stellate iris, up slanting palpebral fissures, depressed nasal bridge, broad nose, smooth philtrum, poor dentition, retrognathia, prominent ear lobes which raised concern for William syndrome (WS). He was diagnosed with ADHD in childhood, heart murmur, hypertension, anxiety symptoms and cognitive deficits in adult life which go in favor of WS. He would hear music and different songs all the time in his brain, which were distinct from hallucinations. These helped him cope with stress which is consistent with the pattern seen in WS. He was diagnosed initially with unspecified mood disorder which was later changed to major depressive disorder with anxious distress, ADHD, combined type, Neuropsychiatric sequelae of WS, other specified obsessive-compulsive and related disorder (obsessional jealousy type). The laboratory findings at the time of admission were significant for positive cannabinoids on urine drug screen and rest of the lab values were within normal limits including serum calcium. As per the past records, EEG was normal and cardiac ECHO showed mild tricuspid insufficiency with normal ejection fraction. Psychological testing revealed significantly impaired attention and concentration, slow processing speed and intelligence in the lower normal range. During hospitalization, he did not display any overt psychotic symptoms. He was initiated on trazodone 100 mg at bedtime for insomnia and bupropion XL 150 mg daily for depression, anxiety and to help with ADHD. He also received individual and group therapy. He reported improvement in insomnia, anxiety and depression.

Discussion:

The rarity of WS precludes its diagnosis in routine psychiatric presentations. This patient had morphology and behavioral patterns seen in WS such as hypersociability, dysmorphic facies, anxiety, cognitive deficits, learning disorder and ADHD in childhood. However, our diagnosis of WS was mainly clinical based. This diagnosis can be confirmed by fluorescent in-situ hybridization (FISH) studies. WS has autosomal dominant inheritance, thus, has genetic implications. The patient was informed about the heritability and advised to seek genetic counselling services. This patient had predominant mood, anxiety and ADHD symptoms for which we decided to choose bupropion as patient had a good response to it in the past and it would target some ADHD symptoms. There is evidence on effectiveness of Bupropion in the treatment of ADHD in adults[7]. In our literature review, there are not many studies on treatment of anxiety and depression in WS.

Conclusion:

We described a rare case of WS with anxiety and co-morbid ADHD which was treated with Bupropion. There are cases of Generalized Anxiety Disorder that have been treated with Buspirone[8]. However, in our literature search we did not come across Bupropion as a treatment of anxiety in WS. This finding may need further validation by systematic studies. This case emphasizes the importance to look for the presence of congenital syndromes that could manifest with psychiatric symptoms.

References:

1. Korenberg, J. R., Bellugi, U., Salandanan, L. S., Mills, D. L., & Reiss, A. L. (2003). Williams syndrome: A neurogenetic model of human behaviour. Nature Encyclopaedia of the Human Genome. https://dx.doi.org/10.1038/npg.els.0005148.
2. Hillier, L. W., Fulton, R. S., Fulton, L. A., Graves, T. A., Pepin, K. H., Wagner-McPherson, C., … Wilson, R. K. (2003). The DNA sequence of human chromosome 7. Nature, 424(6945), 157–164.
3. Jones, W., Bellugi, U., Lai, Z., Chiles, M., Reilly, J., Lincoln, A., & Adolphs, R. (2000). Hypersociability in Williams syndrome. Journal of Cognitive Neuroscience, 12, 30–46.
4. Stinton, C., Elison, S., & Howlin, P. (2010). Mental health problems in adults with Williams syndrome. Ajidd-American Journal on Intellectual and Developmental Disabilities, 115(1), 3–18. doi:10.1352/1944-7558-115.1.3

5. Woodruff-Borden, J., Kistler, D. J., Henderson, D. R., Crawford, N. A., & Mervis, C. B. (2010). Longitudinal course of anxiety in children and adolescents with Williams syndrome. American Journal of Medical Genetics, Part C-Seminars in Medical Genetics, 154C(2), 277–290. doi:10.1002/ajmg.c.30259.
6. Green, T., Avda, S., Dotan, I., Zarchi, O., Basel-Vanagaite, L., Zalsman, G., et al. (2012). Phenotypic psychiatric characterization of children with Williams syndrome and response of those with ADHD to methylphenidate treatment. American Journal of Medical Genetics, Part B-Neuropsychiatric Genetics, 159B(1), 13–20
7. Verbeeck W, Bekkering GE, Van den Noortgate W, Kramers C. Bupropion for attention deficit hyperactivity disorder (ADHD) in adults. *Cochrane Database Syst Rev*. 2017;2017(10). doi:10.1002/14651858.CD009504.pub2

Abstract 20-1-06

Title: Antidepressant choice in hepatic dysfunction

Authors: Morgan Read, Clinton Martin, M.D. FAPA, UAB Huntsville Department of Psychiatry

Learning Objective: To investigate the literature for dosing adjustment and best therapy for treatment of depression in patients with chronic liver disease.

Case Presentation: This review was inspired by a 62-year-old patient with stage IV liver cirrhosis, uncontrolled diabetes mellitus and additional comorbidities. The patient was experiencing an increase in depressive symptoms: low energy, trouble sleeping, irritable mood, anhedonia, and helplessness. Currently taking Duloxetine 30 mg three times per day.

Discussion: Liver function has dynamic effects on pharmacokinetics: biotransformation, plasma protein binding, liver blood flow, and biliary excretion. CYP450 is the major player in antidepressant metabolism and is variably affected in CLD patients. Mirtazapine is a preferred antidepressant for liver failure patients. It demonstrates ~33% reduction in clearance and increase in half life. Start with 50% normal dose and mindful titration. SSRI with appropriate dose adjustments are also preferred. Liver transplant patients require different considerations, focusing more on drug interactions and analyzing drug-specific effects on the P450. Escitalopram is widely recognized for its safety in respect to drug interactions and can be useful in OLT patients with extensive medication lists. Suggest dosing in normal loading dose with a 50% decrease in maintenance dose vs hepatic healthy patients.

References:

1. Mullish, BH, Kabir, MS, Thursz, MR, & Dhar, A. Review article: Depression and the use of antidepressants in patients with chronic liver disease or liver transplantation. Alimentary Pharmacology & Therapeutics. 2014;40(8):880-892. doi:10.1111/apt.12925
2. Verbeeck, RK. Pharmacokinetics and dosage adjustments in patients with hepatic dysfunction. Eur J Clin Pharmacol. (2008)64:1147-1161. doi:10.1007/s00228-008-0553-z
3. Huet PM , Villeneuve JP , Fenyves D. Drug elimination in chronic liver diseases. *J Hepatol*. 1997;26 Suppl 2:63–72. PubMed PMID: 9204411

Abstract 20-1-07

Title: The Mind-Body Connection: Managing Depression in Patients with Crohn's Disease

Authors: Amy Hudson, MS3; Clinton Martin, MD

Introduction:
Recent research indicates depression and Crohn's disease may share a similar pathophysiology.[1] Patients with Crohn's disease tend to have higher rates of anxiety and depression compared to the general population.[2] Pediatric patients are especially prone to developing depression and anxiety after diagnosis.[3] Higher rates of anxiety and depression are associated with patients in active disease rather than patients in remission.[4,5] Antidepressant use is associated with fewer relapses in disease activity.[4,6] However, screening for psychiatric illness is not a part of standard IBD treatment. Adequate management of both Crohn's disease and depression/anxiety may lead to a more benign disease course and relief of psychiatric symptoms

Case Presentation:
A 17-year-old Caucasian female with a history of Crohn's disease (CD) presented for an initial psychiatric evaluation with worsening depression over the past few months. No specific trigger for depression was identified. Depressed mood was associated with increased irritability causing interpersonal conflicts, increased weight gain, sedation, decreased concentration, and low self-esteem.
Patient was diagnosed with Crohn's disease at 13 years old. She was given prednisone to induce CD remission. While taking prednisone, she had a psychotic episode requiring hospitalization. Complicating her case, she had a family history of bipolar disorder. Due to concerns of distinguishing between steroid-induced psychosis and the first presentation of bipolar disorder, she was previously treated with antipsychotics. Additionally, previous trials of SSRIs to treat depression seemed to worsen her mood symptoms.
Over the course of 6 months since her initial evaluation, the patient has been treated with bupropion and lamotrigine and has experienced fewer depressive symptoms. She reports her mood has improved, along with her social anxiety, resulting in enhanced work performance. Her most recent PHQ-A assessment scaled her depression as a 6 out of a 27-point scoring system, indicating mild depression.

Discussion:
The etiology of Crohn's disease is theorized as an abnormal inflammatory response to commensal bacteria in the gastrointestinal tract. TNF-alpha is a pro-inflammatory cytokine elevated with depression and Crohn's disease.[7] Bupropion increases monoaminergic and dopaminergic tone by increasing intracellular cAMP and is hypothesized to lower TNF-alpha levels through this mechanism. Bupropion has been associated with inducing remission in CD and other autoimmune diseases.[7-8] Clinical studies reveal that antidepressant usage in patients with CD improved remission rates.[6] These findings suggest patients should be screened for depression and anxiety at the time of initial diagnosis and during active flare-ups. Clinicians should consider bupropion as a possible first line treatment for managing depression in patients with CD.[8]

References:
1. Martin-Subero M, Anderson G, Kanchanatawan B, Berk M, Maes M. Comorbidity between depression and inflammatory bowel disease explained by immune-inflammatory, oxidative, and nitrosative stress; tryptophan catabolite; and gut-brain pathways. *CNS Spectr*. 2016;21(2):184–198. doi:10.1017/S1092852915000449

2. Lesley A. Graff, PhD, John R. Walker, PhD, Charles N. Bernstein, MD, Depression and Anxiety in Inflammatory Bowel Disease: A Review of Comorbidity and Management, *Inflammatory Bowel Diseases*, Volume 15, Issue 7, 1 July 2009, Pages 1105–1118, https://doi-org.ezproxy3.lhl.uab.edu/10.1002/ibd.20873

3. Mikocka-Walus A, Knowles SR, Keefer L, Graff L. Controversies Revisited: A Systematic Review of the Comorbidity of Depression and Anxiety with Inflammatory Bowel Diseases. *Inflamm Bowel Dis*. 2016;22(3):752–762. doi:10.1097/MIB.0000000000000620

4. Bi-directionality of Brain–Gut Interactions in Patients With Inflammatory Bowel Disease, Gracie, David J. et al., Gastroenterology, Volume 154, Issue 6, 1635 - 1646.e3

5. Neuendorf R, Harding A, Stello N, Hanes D, Wahbeh H. Depression and anxiety in patients with Inflammatory Bowel Disease: A systematic review. *J Psychosom Res*. 2016;87:70–80. doi:10.1016/j.jpsychores.2016.06.001

6. Kristensen MS, Kjærulff TM, Ersbøll AK, Green A, Hallas J, Thygesen LC. The Influence of Antidepressants on the Disease Course Among Patients With Crohn's Disease and Ulcerative Colitis-A Danish Nationwide Register-Based Cohort Study [published correction appears in Inflamm Bowel Dis. 2019 Oct 18;25(11):e152]. *Inflamm Bowel Dis*. 2019;25(5):886–893. doi:10.1093/ibd/izy367

7. Kast RE. Anti- and pro-inflammatory considerations in antidepressant use during medical illness: bupropion lowers and mirtazapine increases circulating tumor necrosis factor-alpha levels. *Gen Hosp Psychiatry*. 2003;25(6):495–496. doi:10.1016/s0163-8343(03)00093-8

8. Kane S, Altschuler EL, Kast RE. Crohn's disease remission on bupropion. *Gastroenterology*. 2003;125(4):1290. doi:10.1016/j.gastro.2003.02.004

Abstract 20-1-08

Title: The Ketogenic Diet as a Treatment for Schizophrenia?

Authors: Anthony Picco, OMS3, Lori Lowthert, MD

Summary: Schizophrenia affects 1% of the population and is a debilitating disease. The mainstay of treatment focuses on targeting excess dopamine in the brain, but recent research suggests that glucose tolerance and insulin resistance abnormalities may also play a role. The ketogenic diet is a low carbohydrate, medium protein, high fat diet in which the body uses ketone bodies instead of glucose as the primary energy source. Several case studies have been reported over the years where patients have experienced remission of longstanding psychotic symptoms with the diet, some even tapering off all of their antipsychotic medications. The mechanism of action of the diet is still not completely understood. Some hypothesize that it helps increase the GABA:glutamate ratio to help compensate for the GABA depleted schizophrenic brain. Others note the diet has been proven effective for treatment-resistant epilepsy, and anti-epileptic medications are well known to be efficacious in the treatment of psychiatric disorders. Studies using mouse models of schizophrenia have also shown that that the metabolic changes induced by the ketogenic diet may be therapeutic for pathologic behaviors seen in the disease. Although there are no completed randomized clinical trials with humans, one such trial is in the works and is expected to be completed by the end of 2020. This is a review of the available literature on this topic.

References:

1. Ketogenic Diet for Psychotic Disorders - Full Text View. (2019, March 14). Retrieved March 8, 2020, from https://clinicaltrials.gov/ct2/show/study/NCT03873922
2. Kraeuter, A. K., Loxton, H., Lima, B. C., Rudd, D., & Sarnyai, Z. (2015). Ketogenic diet reverses behavioral abnormalities in an acute NMDA receptor hypofunction model of schizophrenia. *Schizophrenia Research*, *169*(1-3), 491–493. doi: 10.1016/j.schres.2015.10.041
3. Palmer, C. M. (2017). Ketogenic diet in the treatment of schizoaffective disorder: Two case studies. *Schizophrenia Research*, *189*, 208–209. doi: 10.1016/j.schres.2017.01.053

4. Palmer, C. M., Gilbert-Jaramillo, J., & Westman, E. C. (2019). The ketogenic diet and remission of psychotic symptoms in schizophrenia: Two case studies. *Schizophrenia Research*, *208*, 439–440. doi: 10.1016/j.schres.2019.03.019
5. Perry, B. (2017). The Association Between First-Episode Psychosis and Abnormal Glycaemic Control: Systematic Review and Meta-analysis of Clinical Studies. *The Lancet Psychiatry*, *3*(11), 1049–1058. doi: 10.26226/morressier.5885d716d462b8028d891aaa
6. Włodarczyk, A., Wiglusz, M. S., & Cubała, W. J. (2018). Ketogenic diet for schizophrenia: Nutritional approach to antipsychotic treatment. *Medical Hypotheses*, *118*, 74–77. doi: 10.1016/j.mehy.2018.06.022

Abstract 20-1-9

Title: Is it time to add sleep studies to mental health workups?

Authors: Brooks Burns, MS; Tina Jackson, MD; William B. Brooks, MD.

Summary: It is well known that psychiatric patients do not receive services for medical comorbidities at the same rate as other patients. It is becoming more evident that patients with severe mental illnesses are at an increased risk for disordered sleep. Research in sleep medicine raises compelling questions about whether sleep evaluations may be warranted as part of the initial work up for some psychiatric patients.

We present a case of a 25 yo AAM who presented to the outpatient psychiatric clinic with his first psychotic episode. He was minimally responsive to treatment with medications and continued to have frequent suicidal gestures and emergency room visits. He self-referred for a sleep study and was diagnosed with central sleep apnea. After initiating CPAP his mental health status stabilized quickly and he has been maintained on a low dose antipsychotic along with continued CPAP.

In addition, an informal survey of psychiatrists was performed on a social media platform asking about current practices in monitoring formal sleep evaluations.

References:

1. Lane, A. R., Foley, S., Siskind, D. (2019) We snooze, they lose: where is the conversation about obstructive sleep apnoea in early psychosis? *Australasian Psychiatry,* 27(3), 314.
2. Laskemoen, J. F., Simonsen, C., Buchmann, C., Barrett, E. A., Bjella, T., Lagerberg, T. V, Vedal, T. J. Andreassen, O. A., Melle, I., Aas, M. (2019) Sleep disturbances in schizophrenia spectrum and bipolar disorders—a trnsdiagnostic perspective. *Comprehensive Psychiatry,* 91: 6-12.
3. Kaskie, R. E., Ferrarelli, F. (2020) Sleep disturbances in schizophrenia: what we know, what still needs to be done. *Current Opinion in Psychology,* 34:68-71.
4. Farrell, P. C., Richards, G. (2017) Recognition and treatment of sleep-disordered breathing: an important component of chronic disease management. *Journal of Translational Med,* 15: 114-126.
5. Knechtle, B., Economou, N., Nikolaidis, P. T., Velentza, L., Kallianos, A., Steiropoulos, P., Koutsompolis, D., Rosemann, T., Trakada, G. (2019) Clinical characteristics of obstructive sleep apnea in psychiatric disease. *Journal of Clinical Med,* (8) 534.

Abstract 20-1-10

Title: Post-Traumatic Growth and Its Association with Delay Discounting

Authors: Kara Kilpatrick, MD; Mai Nguyen, BA; Justin Strickland, PhD; Craig Rush, PhD; Amy Meadows, MD

Summary:
Introduction:
Delay discounting has been shown to be related to substance use, gambling, traumatic events, and increased health care costs. Post-traumatic growth has been studied as a potential indicator of well-being after trauma, but little research exists correlating post-traumatic growth and delay discounting. We hypothesized an increase in post-traumatic growth would be associated with decreased delay discounting.

Methods:
Participants were recruited using Amazon.com Mechanical Turk (mTurk), an online crowdsourcing platform. For eligibility, participants were required to have a 99% prior task approval rate, be living in the United States, age 18+, and have completed at least 500 HITs prior tasks. In 8/2019, 560 participants were recruited and completed all study measures, including the Post-Traumatic Growth Inventory and the Monetary Choice Questionnaire, a validated measure of delay discounting. The University of Kentucky Medical IRB reviewed and approved all study procedures. Post- traumatic growth inventory scores were categorized into 2 groups: =50 and =49. One-way ANOVA determined group differences between delay discounting and post-traumatic growth inventory scores.

Results:
This was a primary data analysis from 560 adults (age 37.09±10.95 years, 45% female). Results demonstrated adults who reported high post- traumatic growth inventory had significantly higher delay discounting ($F(1, 558) = 8.02$, $p=.005$).

Discussion:
Outcomes associated with post-traumatic growth include reductions in psychopathology and increases in well-being and wisdom. In this analysis, however, higher post-traumatic growth was associated with increased delay discounting, which was unexpected. Further work is needed to better understand trauma and post-traumatic growth effects on delay discounting.

Abstract 20-1-11

Title: Pediatric SSRI Induced Urinary Hesitancy

Authors: Raymond Moosavi M3, Shanti Ghatla MD

Summary:
Overview:

- Rates of having been diagnosed with either anxiety or depression among children aged 6-17 years has increased from 5.4% in 2003 to 8% in 2007 and to 8.4% in 2011–2012(Bitsko et al).
- Anxiety and depression have been shown to have a high concordance rate (Axelson, D. A., & Birmaher, B).

- Most all cases of anxiety and depression are treated pharmacologically, with the SSRIs being a popular option.
- The most common side effects of SSRIs include sexual dysfunction, weight gain, and sleep disturbance (Ferguson, J. M.).
- SSRIs are also a frequently unrecognized cause of the potentially distressing symptom of urinary retention.
- One study showed that urinary retention occurred in 10% of patients perscribed SSRIs and the symptom is often severe enough to lead to the discontinuation of the medication (Choong S, Emberton M.)

Case:
Mr.X is a 14-yo Caucasian male diagnosed with anxiety and depression. Mr.X was started on a trial of Escitalopram 10mg PO qd. 5 days later Mr.X reported urinary hesitancy, starting with difficulties urinating and progressing to complete urinary retention. Mr.X was taken off Escitalopram which lead to a complete resolution of symptoms. Another SSRI, Sertraline, was started which led to alleviation of anxiety and depression with no adverse effects of urinary retention.

Discussion:
- Urinary retention is defined as impaired bladder emptying which results in residual urine post-void.
- Urinary retention is subjectively uncomfortable for the patient but can sometimes become so severe that it leads to renal failure.
- Urinary retention is immediately relieved by straight catheterization, but the root cause must always be identified (Choong S, Emberton M).
- Medication induced causes of urinary retention include tricyclic antidepressants, anticholinergic agents, antiparkinsonian agents, antipsychotics, sympathomimetics and muscle relaxants (Trombetta, D et al).
- The SSRI Escitalopram has been theorized to promote urinary retention through several mechanisms, including a small degree of muscarinic/cholinergic antagonism (M1), inhibition of neurotransmitter reuptake like dopamine and norepinephrine, inhibition of the CYP enzymes, and/or a direct effect on serotonergic neurons in Onuf's and Raphe's nuclei (Trombetta, D et al).

References:

1. Bitsko, R. H., Holbrook, J. R., Ghandour, R. M., Blumberg, S. J., Visser, S. N., Perou, R., & Walkup, J. T. (2018). Epidemiology and impact of health care provider–diagnosed anxiety and depression among US children. Journal of developmental and behavioral pediatrics: JDBP, 39(5), 395.
2. Axelson, D. A., & Birmaher, B. (2001). Relation between anxiety and depressive disorders in childhood and adolescence. Depression and anxiety, 14(2), 67-78.
3. Ferguson, J. M. (2001). SSRI antidepressant medications: adverse effects and tolerability. Primary care companion to the Journal of clinical psychiatry, 3(1), 22.
4. Choong S, Emberton M. Acute urinary retention. BJU International. 2000; 85:186-201.
5. Trombetta, D., Garrett, K., & Harrison, M. (2013). Escitalopram-associated acute urinary retention. The Consultant Pharmacist®, 28(10), 661-669.

Abstract 20-1-12

Title: A case presentation of frontal meningioma associated treatment resistant mania

Authors: Richard Trieu, Giann Bhatt, Darshana Pai M.D., Alex Ghilezhan D.O., William J. Billet M.D.

Summary:

Introduction:

Meningiomas are the most common intracranial tumors typically arising from the dura mater and/or pia mater. They comprise 34% of all primary brain tumors and are classified by the WHO into 3 different classifications describing their likelihood of recurrence and metastasis[3,4]. They are one of the most common primary central nervous system tumors with an incidence rate of 2:9000[1]. These tumors are found primarily in women with a median age of diagnosis of 65 years old[2]. Risk factors for these tumors are radiation exposure, NF2 patients, MEN1 patients, SMARCB1 mutations, post puberty females, history of breast cancer, and obesity[4].

Meningiomas are typically asymptomatic with mass effect due to enlargement underlying much of the location dependent symptomatology. Headaches, nausea, vomiting, seizures, confusion, hearing loss, muscle weakness, visual disorders, neurologic deficits, hearing loss, tinnitus are the most common symptoms at presentation. In diagnosing, imaging is useful in generating a differential diagnoses and localization. However, biopsy is necessary for definitive diagnosis. We report a woman presenting with treatment refractory mania in the setting of previously diagnosed Bipolar 1 disorder. This is an uncommon presentation for these types of tumor with less than 30 cases reported to the best of our knowledge[5-11].

Case presentation

A 55-year-old obese unkempt female presented with inappropriate behavior including hypersexuality and acute mania requiring psychiatric evaluation. She as had multiple episodes of mania due to noncompliance with a noted attempt at suicide at the age of 19 via loxapine overdose. Her history is significant for diabetes mellitus, asthma, hypertension, right bundle branch block, and pseudotumor cerebri. Her upbringing was marked by a strained interpersonal relationship amongst her and her parents. Her family history was significant for heart disease and maternal schizophrenia treated with chlorpromazine. Past medication trials included quetiapine, haloperidol, chlorpromazine, divalproex sodium, lithium, oxcarbazepine, diazepam and carbamazepine.

At the time of admission, she had increased psychomotor activity, was internally stimulated, and appeared animated with gesticulations. Her mental status exam was significant for pressured speech, disorganized thoughts with flight of ideas, delusions of persecution via paranoia, and high distractibility. Her mood was elevated and labile. She lacked judgement and insight and was overtly hypersexual towards the opposite sex. On physical exam, she was hemodynamically stable. Her review of system was significant for bilateral hand tremors. Her urine drug screen was positive for benzodiazepines and had a prescription for zolpidem. Delirium was ruled out with routine labs that were within reference range. Prior labs revealed an episode of hyponatremia due to oxcarbazepine.

On admission, she was started on cariprazine 3mg daily for management of mania with psychosis and clonazepam 0.5mg BID for agitation. She improved with clonazepam 0.5mg TID, Trileptal 300mg BID, cariprazine 4.5 mg QD and was transferred to a step-down unit for further stabilization. However, the patient did not improve with optimal dose of cariprazine which was transitioned to quetiapine then to a trial of haloperidol and lorazepam. The patient continued to have manic symptoms with predominant hypersexuality, initiating the need to receive frequent medications for behavioral control throughout each trial.

Treatment resistance prompted a review of neurology record revealing a recently visualized meningioma in the right frontal region via MRI imaging. The meningioma had grown 7 mm over 5 months noted on her last MRI. The presence of this tumor raised clinical suspicion that it could be

potentiating the manic symptoms as well contributing to treatment resistance. Overall, patient improved with the addition of lithium 900mg allowing for the discontinuation of Ativan. However, an abnormal lithium level of 1.3 was noted during standard follow up labs which lead to a decrease in total dose to 750mg. Repeat lithium level paradoxically increased to 1.5 prompting discontinuation. The Patient ultimately remained stable enough to be discharge to a group home.

Discussion:

We present a patient with refractory manic and psychotic symptoms due to a symptomatic frontal meningioma found incidentally. Meningiomas are largely asymptomatic unless it becomes large enough to cause mass effect to local structures. In our case the mass caused symptomatology consistent with treatment resistant bipolar type 1 disorder with psychotic features. This case shows the importance of thorough investigation of a patient's history and the continual need for physicians to be detailed and persistent in their search for an organic cause for psychiatric conditions in the context of symptoms refractory to evidence based treatments. This case highlights It is possible for clinicians to be blindsided by the pre-existence of psychiatric illness. Therefore, it is important to have a sufficient degree of clinical suspicion in cases with exaggerated symptoms or treatment resistance.

References:

1. Ostrom QT, Gittleman H, Truitt G, et al. CBTRUS Statistical Report: Primary Brain and Other Central Nervous System Tumors Diagnosed in the United States in 2011-2015. Neuro Oncol 2018; 20 Suppl 4:iv1.
2. Liu Y, Li F, Zhu S, et al. Clinical features and treatment of meningiomas in children: report of 12 cases and literature review. Pediatr Neurosurg 2008; 44:112.
3. Rogers L, Barani I, Chamberlain M, et al. Meningiomas: knowledge base, treatment outcomes, and uncertainties. A RANO review. *J Neurosurg*. 2015;122(1):4–23. doi:10.3171/2014.7.JNS131644
4. Buerki RA, Horbinski CM, Kruser T, Horowitz PM, James CD, Lukas RV. An overview of meningiomas. *Future Oncol*. 2018;14(21):2161–2177. doi:10.2217/fon-2018-0006
5. Hussin S, Yusoff SSM, Zin FM, Ghani ARI. Frontal meningioma with psychiatric symptoms. *J Family Med Prim Care*. 2018;7(1):252–254. doi:10.4103/jfmpc.jfmpc_157_17
6. Yakhmi S, Sidhu BS, Kaur J, Kaur A. Diagnosis of frontal meningioma presenting with psychiatric symptoms. *Indian J Psychiatry*. 2015;57(1):91–93. doi:10.4103/0019-5545.148534
7. Hunter R, Blackwood W, Bull J. Three cases of frontal meningiomas presenting psychiatrically. *Br Med J*. 1968;3(5609):9–16. doi:10.1136/bmj.3.5609.9
8. Meningioma and psychiatric symptoms: A case report and brief review. Subramoniam Madhusoodanan, Shama Patel, Jonathan Reinharth, Adam Hines, Mark Serper Ann Clin Psychiatry. 2015 May; 27(2): 126–133.
9. Seo EH, Kim SG, Cho YS, Yoon HJ. Tuberculum sellae meningioma with possible tacrolimus neurotoxicity manifesting as manic-like psychosis after kidney transplantation. *Ann Gen Psychiatry*. 2019;18:18. Published 2019 Sep 5. doi:10.1186/s12991-019-0242-6
10. Mahapatra A, Sood M, Khandelwal SK. Multiple Meningioma in a Patient of Bipolar Disorder: The Dilemma of Detecting Structural Brain Lesions in the Backdrop of a Long Standing Psychiatric Illness. *J Clin Diagn Res*. 2016;10(8):VD01–VD02. doi:10.7860/JCDR/2016/18817.8332
11. Wei CP, Wang AD, Tsai MD. Resection of giant olfactory groove meningioma with extradural devascularization. *Skull Base*. 2002;12(1):27–31. doi:10.1055/s-2002-21570-1

Abstract 20-1-13

TRACKING DISTRESS AMONG UNIVERSITY OF ALABAMA AT BIRMINGHAM RESIDENTS AND FACULTY BY THE WELL-BEING INDEX

Austin Luker, MD; Manasa Enja MD; Adriane Meadows MD; William Tennant MD; Boone Rountree DO; Brenda Burnett DO; Taylor Preston, MD; James Meador-Woodruff, MD; Irena Bukelis, MD, The University of Alabama at Birmingham

Introduction: Burnout is a well-established problem among physicians, which can negatively affect doctors themselves, as well as patients, through medical errors and compromised care. The Mayo Clinic Physician Well-Being Index is a measure shown to be useful in evaluating distress among physicians. This study is a follow up study of data from the 2018-19 academic year where found evidence of distress among UAB psychiatry faculty and residents by the Well-Being Index, but there was limited sample size. This study aimed to use same Well-Being Index to measure distress among The University of Alabama at Birmingham (UAB) residents, fellows, and faculty for the 2019-2020 academic year and compare to the 2018-2019 data.

Methods: The Mayo Clinic Physician Well-Being Index was used as part of a university-wide electronic survey to residents and faculty. Higher scores indicate higher distress. A cutoff of >3 was used to quantify "high distress," because this cutoff score has been demonstrated to correlate to increased chances of medical error, poor quality of life, and burnout.

Results: Current results continue to be collected and analyzed, with the goal of increasing faculty and resident participation compared to last year. These results will be compared to national data. Data collection will conclude and be analyzed prior to presentation.

Conclusions: Burnout and distress are problems across medicine, but an often difficult to measure problem, with selection bias and concerns about confidentiality being major barriers to data collection and interpretation.

Abstract 20-1-14

Title: Mirtazapine Induced Nightmares in an Adult Female

Authors: Jeanetta Malone, MS-3; Samantha Lee, MS-3; Bradley Brooks, D.O.; William B. Brooks, M.D.

Abstract:
Mirtazapine is a tetracyclic noradrenergic and serotonergic atypical antidepressant that is useful in the treatment of major depressive disorder. Previous studies have demonstrated that mirtazapine is unique from other antidepressants in its effect on the sleep cycle, which leads to an increase in slow-wave sleep and total sleep time. For this reason, mirtazapine has been one of the first line therapies for patients suffering from depression and insomnia. However, recent cases suggest that mirtazapine may be associated with vivid nightmares. We discuss the case of a 46-year-old female who presented with depressive symptoms consistent with major depressive disorder. The patient was tried on a variety of antidepressants with little success. She was eventually placed on mirtazapine and rapidly began experiencing vivid nightmares of being murdered. The patient was subsequently taken off mirtazapine, and her nightmares resolved. Nightmares are generally isolated to the REM stage of sleep architecture, and it has been reported that patients with depression show disturbances in polysomnographic

recordings demonstrating increased REM sleep. It is hypothesized that mirtazapine's unique effect upon the sleep cycle in addition to the sleep disturbances present in depressive patients may lead to the manifestation of vivid nightmares. Our case presentation demonstrates a close temporal association suggestive of a causal role for Mirtazapine in inducing the nightmares. Clinicians should be aware of this possible side effect of mirtazapine and monitor patients appropriately.

References:

1. Davis R, Wilde MI. Mirtazapine: A Review of its pharmacology and therapeutic potential in the management of major depression. CNS Drugs 1996; 5:389-402
2. Schittecatte M, Dumont F, Machowski R, Cornil C, Lavergne F, Wilmotte J. Effects of mirtazapine on sleep polygraphic variables in major depression. Neuropsychobiology 2002; 46: 197–201
3. Thase, M. E. Depression and sleep: Pathophysiology and treatment. *Clinical Research*, 2006; *8*(2),10.
4. Matthews, M., Basil, B., Evcimen, H., & Adetunji, B. Mirtazapine-Induced Nightmares. *The Primary Care Companion to The Journal of Clinical Psychiatry*, 2006; *08*(05), 310–311.
5. Menon V, Madhavapuri P. Low-dose mirtazapine-induced nightmares necessitating its discontinuation in a young adult female. J Pharmacol Pharmacother. 2017;8(4):182–4.
6. Dang, A, Garg, G, Rataboli, PV. Mirtazapine induced nightmares in an adult male. Br J Clin Pharmacology, 2008; 67: 135–136.