APPA 2019 Fall Meeting Resident Poster Presentation

Abstract 19-2-01

Title: Inflammation and Depression: A Perfect Storm

Authors: Skyler G. Jones; Clinton Martin M.D.

Summary: A major breakthrough in understanding depression is the observation that exposure to pro-inflammatory cytokines produces a sickness syndrome that mimic depression and can be treated with antidepressants. Recent research has centered around potential novel therapeutics in relation to chronic inflammation's alteration of normal CNS neurotransmission (*monoamine hypothesis*) and of neuroprotective factors (*neurotrophic hypothesis*). While the exact etiology is unknown, leading hypotheses suggest epigenetic alterations in acute stress response, alterations in CNS transport mechanisms, and inhibition of anti-inflammatory T-cell properties all underlie this association. Accumulation of interleukin-6 receptors is the strongest inflammatory marker regarding depression incidence and recurrence[1], though TNF-alpha and C-reactive protein have also been implicated as predictive markers.[2] Uncontrolled comorbid inflammatory illnesses in patients with depression result in a reduced quality of life and daily functioning, and is associated with poorer psychiatric prognosis and response.[3]

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Abstract 19-2-02

Title: Worsening Depression in Neurosarcoidosis

Authors: Ashley Ford, MS-3; Clinton Martin, MD

Summary: Studies now indicate that sarcoid-related fatigue is different than fatigue experienced by the general population. Interestingly, younger patients with sarcoid had a much higher prevalence, with female patients more affected than males. The best tool for evaluating fatigue is with the Fatigue Assessment Scale. [4] Sarcoid patients without neurological involvement had no decline in executive function compared to healthy controls.[5] Deficits in cognitive function can manifest as problems with memory, attention, concentration, and self-management. However, there is no data on the extent of cognitive under-performance.[7] While prevalence of depressive symptoms is 27 – 66%, it is not known if sarcoid causes depression. It is proposed that fatigue and decreased overall quality of life in addition to the CNS effects of the disease itself play some role.[3] Pharmacologic therapies for neurosarcoid focus on symptom relief, and are not curative at this time. The latter is why appropriate assessment is crucial at managing psychological symptoms of this disease. Clinical and research infrastructures that measure depression in sarcoid patients have not been specific enough for these patients.

The SHQ (Sarcoidosis specific health questionnaire) was designed to allow patients with sarcoid to rate their overall life satisfaction in areas the patients deemed most important. The SHQ requires no supervision, is easily scored, time sensitive, and has been proven superior to other assessment models.[6] To date, there is no clear association that any of the first line antidepressant regimens are superior for treatment of depression related sarcoid versus depression secondary to other causes.

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Abstract 19-2-03

Title: Fragile X

Authors: Chodaba, Martha, MS4; Martin, Clinton, MD

Summary:

Overview:

- Fragile X syndrome is caused by a CGG triplet repeat in the fragile X mental retardation 1 gene (FMR1).
- X-linked dominant – 1 X chromosome is enough to pass the gene to both males and females.
- FMR1 gene premutation in females can expand to more than 200 CGG repeats during oogenesis. This expansion does not occur in males, so males are only able to pass a premutation but not the mutation.
- 1:4000 males and 1:8000 females.
- It is the most common genetic cause of autism spectrum disorder (ASD) and accounts for 5% of ASD.
- There is a 15-60% prevalence of ASD in males with Fragile X.
- It is associated with multiple comorbidities that include ASD, anxiety disorder, sleep issues, and ADHD.
- ADHD is the most common behavior abnormality in patients with the syndrome.

Case:

29-year-old Caucasian male who initially began showing difficulties with communication, learning and inattention / impulsivity symptoms which was later diagnosed as Fragile X syndrome at age 7. At age 9, he began taking methylphenidate for his ADHD. He had been taking Aripiprazole but developed tardive dyskinesia, so it was discontinued. He tried several other antipsychotic trials medications for 2 years but had difficulty with increased irritability and subsequently was stabilized on Brexiprazole.

Discussion:

- 84% of boys and 67% of girls with FXS display clinical symptoms of inattention and impulsivity
- 66% of boys and 30% of girls display abnormal amounts of hyperactivity
- 54-59% prevalence of ADHD in FXS – these are higher rates than in other neurodevelopmental disorders (Grefer et al.)
- If ADHD is found early, there are lower rates of hyperactivity and improved cognitive outcomes and reduced autism symptoms
- The FMR gene is associated with lower levels of melatonin

- Patients with ASD tend to have sleep disorder and 77% of patient with FXS and ASD are affected with sleep disturbance. (Won et al.)
- Sleep disturbances in individuals with FXS is associated with: impaired vigilance, deficits in learning and memory, and autistic behavior with abnormal and anxiety response. (Won et al.)
- Studies of melatonin supplementation in these individuals have shown better sleep, enhances mechanisms associated with learning, and increased neuroplasticity (Won et al.)

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Abstract 19-2-04

Title: 22q11.2 Deletion Syndrome and its relation to Schizophrenia

Authors: Chodaba, Martha, MS4; Martin, Clinton, MD

Summary:

Overview:

- 22q11.2 deletion syndrome is the most common chromosomal microdeletion disorder occurring in 1 in 2,000-4,000 live births.
- This syndrome is characterized by its highly variability in its phenotypic expression; this case report will focus on psychiatric abnormalities- particularly schizophrenia. (McDonald-McGinn)
- 25% of patients affected with 22q11.2DS have schizophrenia and they account for 1-2% of all schizophrenia cases.

Case:

Pt is 32 yo male with a past medical history of schizophrenia secondary to DiGeorge Syndrome, depression, and anxiety coming in for a 3 month follow up and increasing psychotic features over the past month. He has auditory hallucinations at baseline, but they have been louder and speaking to him more frequently. The hallucinations began originally at age 18 and presented with visual hallucinations in addition to auditory. Since the initial presentation, he has not had visual hallucination. The only medication the patient can tolerate is Zyprexa, which cannot be generic. Other medications have been

tried in the past, but he had adverse reactions or no affect from them: risperidone, apriprazole, paroxetine, and first-generation antipsychotics. The patient was diagnosed with DiGeorge syndrome when he was 12 years old. The patient was adopted, and there is suspicion that he inherited the syndrome from his mother. When the patient was about 9 years old, he began falling behind in math and reading- which is where his reading and arithmetic level remain.

- The 22q11.2 deletion syndrome is de novo in 90% from a non-homologous meiotic recombination event.
- The deletion affects 90 genes.
- The COMT enzyme sequence is found within the region and is important in metabolizing catecholamines. The activity of the enzyme is important in regions of the brain with low presynaptic dopamine transporter. (McDonald—McGinn et al.)
- COMT enzyme polymorphisms has been explored in respect to schizophrenia, but there has been no association found. (Monks et al.)

Discussion:

- Research studying adolescents and young adults with 22q11.2 DS, before they develop psychotic or schizophrenic symptoms, is being done. The research hopes to find behavioral and cognitive markers that will help identify schizophrenia before the onset of their first psychotic episode (RE Gur et al.)
- Mild IQ decline with increasing age is common with 22q11.2 DS. The verbal domain is most commonly affected. There is a sharper decline in IQ status at a younger age in patients that develop psychotic illness similar to observations seen in schizophrenia in the general population. (RE Gur et al.)
- Both schizophrenia and psychosis secondary to 22q11.2 have a similar response to antipsychotics (RE Gur et al.)

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Abstract 19-2-05

Title: Management of ADHD in Fragile X Syndrome

Authors: Alex Kearns; Janaki Nimmagadda MD; Clinton Martin MD

Summary:

Overview:

Fragile X Syndrome(FXS) is the most common inherited cause of intellectual disability. Common psychiatric comorbidities of FXS include cognitive dysfunction, Autism Spectrum Disorder(ASD), and Attention Deficit Hyperactivity Disorder (ADHD). ADHD is the most common comorbidity occurring with an estimated 84% of males and 67% of females with FXS. ADHD diagnosis in individuals with FXS may further increase risk of other psychiatric comorbidities, and negatively impact quality of life.

Case:

42 year-old Caucaisan male with developmental delays initially presented with difficulty with impulse control, intellectual disability, poor concentration, and tremor. He was unable to sit still for extended periods of time, and during initial office visits would run around and climb objects impulsively. These symptoms are associated with social anxiety, inability to live independently and requiring a caregiver for daily needs.

Discussion:

Early identification and treatment of ADHD symptoms, in children with FXS, demonstrates improved long-term outcomes, improved prosocial behaviors, and lower rates of secondary comorbidities like Conduct Disorder, and Oppositional Defiant Disorder. Due to the challenges with early diagnoses, temperament model can aid in early identification of ADHD symptoms in children with FXS, when used from ages 3-6. Increased Surgency traits include Impulsivity and Activity level. Negative Affectivity traits include shyness and decreased smiling/laughter. Among these, two traits impulsivity serves as the strongest temperamental predictor of ADHD, in children age 3-6. Pharmacologic intervention is effective in 70% of patients, associating with improved socialization skills and attention span.

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Abstract 19-2-06

Title: Pseudobulbar Affect after Opiate Overdose

Authors: Alex Kearns; Anupama Yedla MD; Clinton Martin MD

Summary:

Overview:

Pseudobulbar Affect (PBA) is a disorder of affective regulation, resulting from neurologic diseases. Common causes include Multiple Sclerosis, Alzheimer's disease, and stroke. Patients experience uncontrolled, exaggerated laughing or crying. This is often a disproportionate response to an emotional stimulus, but it can also be an inverted response (i.e. laughing at a funeral), or entirely spontaneous. The physical pathway of the disease is poorly understood, but is proposed to be due to disruption of structure or neurotransmission within the corticopontocerebellar network. In this network, impaired inhibitory control at the cortical or cerebellar levels may lead to increased activation and involuntary motor expression.

Case:

29yo female who was initially diagnosed with anxiety at age 18 and treated with SSRI medications. At the age of 19 she developed opiate use disorder after initially having pills offered to her, by a friend, to relieve migraine headaches. She had an accidental overdose on opiates at age 21, leading to encephalitis with seizure. During her hospitalization she was diagnosed as having PBA which was not sufficiently controlled by SSRI medications. The patient had spontaneous, inappropriate laughing and crying episodes. Neurology was consulted and she was started on Dextromethorphan, but could not tolerate it

due to GI side effects. Her medications were subsequently switched to Oxcarbazepine, which has stabilized her affect and improved social functioning. However, she now has blunted emotions.

Discussion:

PBA can manifest in any etiology of neurologic injury, including opiate overdose. PBA has distressing psychiatric complications, of both the disease and the treatment. Pharmacologic intervention is aimed at improving regulation of affective expression. Management primarily involves Dextromethorphan, an

NMDA receptor antagonist. “Nuedexta” is the Dextromethorphan-Quinidine combination approved by the FDA for use in PBA. However, as many as 13% of patients may experience the drug’s primary side effects of GI upset and diarrhea. Alternatively, uncontrolled trials have demonstrated significant efficacy of SSRI and TCA. Management should also include counseling of patients and families, to educate about the condition. It is important to mitigate feelings of guilt or blame.

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Abstract 19-2-07

Title: Anxiolytic Use Disorder after a Panic Attack

Authors: Alex Kearns; Anupama Yedla MD; Clinton Martin MD

Summary:

Overview:

1% of the US population are at risk to develop anxiolytic use disorder in their lifetime. Alabama Ranks 4th highest prescribing state among the Nation. Benzodiazepines have been used as treatment in Generalized Anxiety Disorder and panic disorders. However, their use declined since the 1990s, with the advent of serotonergic antidepressants. Benzodiazepine dependence involves reduced GABA receptor responsivity, leading to reduced neuroinhibition. Patients experience tolerance, with returning anxiety symptoms, requiring increasing doses. Benzodiazepine withdrawal can cause increased anxiety, tremor, palpitations, pain, vomiting, seizures, and acute psychosis. Benzodiazepine toxicity involves cognitive and motor impairment and, in severe cases, coma and respiratory depression.

Case:

31 year old male with chronic anxiety and depression with somatic complaints was initially treated with SNRI beginning at age 18. Despite treatment he continued to experience panic attacks and, during one of the episodes, went to the emergency room. He was prescribed Alprazolam at that time and subsequently developed dependence. The patient was weaned off Alprazolam but worsening anxiety symptoms prompted adjunctive treatment with Aripiprazole. The patient developed akathisia, which was misinterpreted as increased agitation, and Aripiprazole dose was further increased. After the increase in Aripiprazole, patient had a seizure. Subsequently, the patient developed mistrust of

medications, refusing any antidepressant. He has been unable to cope with his anxiety, unable to work, and unable to maintain relationships. He continues to be resistant to treatment with any pharmacotherapy.

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Abstract 19-2-08

Title: Psychosis in a 6 year old?

Authors: Sawyer Mullen, MS4, Milza Howard, MS4, Janaki Nimmagadda, MD

Summary: This is a case report of a 6-year-old female with command auditory hallucinations. The voices give her instructions, which include things such as telling her to wake up, go to the bathroom, eat food, and pick up toys. Her history is notable of gross motor and speech delay. She started speaking at age 4 and that's when she informed her family about the voices. She had intrauterine exposure to methamphetamine until second trimester of the pregnancy. She had disruptions in the home environment, which includes mother losing custody and she witnessed domestic violence between parents on multiple occasions until age 3. At the age of 4, the patient was started on Sertraline 20mg daily. The patient also had some aggressive, hyperactive type behaviors and defiance problems. PCP then tapered the Sertraline and started on Aripiprazole 2.5mg daily 3 weeks prior to her presentation in the clinic. Family reported that her aggressive symptoms are better after starting Aripiprazole but she continues to hear voices. With such a varied presentation and associated symptomatology, a clinical decision on AH at age 6 can be difficult. However, it is important to note that majority of children who exhibit psychotic-like symptoms do not have a true psychotic disorder. Current literature suggests the need to rule out comorbid psychiatric diseases and maintains a need for caution when instituting antipsychotic medications for AH in young children. Several clinical questionnaires have been developed to assist clinicians in determining the significance of AH in pediatric populations. Until further research merges to aid clinical judgment, it would seem that the best policy for caring for a pediatric patient with AH would be to thoroughly examine the patient's symptoms and compare them against normal development and to rule out or treat any underlying psychiatric pathology to ensure the highest level of

care and the best outcome.

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Abstract 19-2-9

Title: A case of anti-NMDA-receptor Encephalitis causing Psychosis

Authors: Kenneth Holt MS4; Clinton Martin M.D.

Summary: Anti-NMDA-receptor encephalitis has been growing in recognition as a major cause of encephalitis. One study in 2012 found Anti-NMDA-receptor encephalitis to be more prevalent of a cause of encephalitis in young persons than any individual viral cause (Gabel, 2012). Recently, increased awareness of anti-NMDA-receptor encephalitis has caused the disease to rise to front of differentials for patients previously thought to have encephalitis of viral or unknown etiology. In this case report we discuss a patient who presented with seizures and episodic confusion who was diagnosed with Anti-NMDA-receptor encephalitis.

Discussion: Anti-N-methyl-D-aspartate Receptor (NMDAR) encephalitis is an autoimmune condition that can occur in the presence or absence of neoplasms. Clinical syndrome manifests with memory and behavioral disturbances, catatonia, agitation, psychosis, seizures and dyskinesias.

Probable anti-NMDA receptor encephalitis:

Diagnosis can be made if 3 of the following criteria have been met

1. Rapid onset (less than 3 months) of at least 4 of the 6 following major groups of symptoms:

    - Abnormal (psychiatric) behavior or cognitive dysfunction

- Speech dysfunction (pressured speech, verbal reduction, mutism)
- Seizures
- Movement disorder, dyskinesias, or rigidity\abnormal postures
- Decreased level of consciousness
- Autonomic dysfunction or central hypoventilation

2. At least 1 of the following laboratory study results:

- Abnormal EEG (focal or diffuse low or disorganized activity, epileptic activity, or extreme delta brush)
- CSF with pleocytosis or oligoclonal bands

3. Reasonable exclusion of other disorders

Diagnoses can also be made in the presence of 3 of the above group symptoms accompanied by her systemic teratoma.

Definite anti-NMDA receptor encephalitis:

Diagnosis can be made in the presence of 1 or more of the 6 major groups of symptoms and IgG anti-GluN1 antibodies, after reasonable exclusion of other disorders.

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Abstract 19-2-10

Title: Psychogenic Non-Epileptic Seizures in a 15 Year Old Female.

Authors: Taylor Blaine Jordan, MS-4; Clinton Martin, MD.

Summary: A previously healthy 15 year old Caucasian female presents to the office following multiple ED visits as well as a hospital admission for a wide variety of symptoms ranging from loss of consciousness, to 5- 6 episodes of seizure like activity, and multiple somatic complaints. All of which began one month after she reported to her local ED and was diagnosed with a panic attack. The seizures have been witnessed by her grandmother and are described as "drawing up" her arms and legs and laying unresponsive for 5-10 minutes. The patient describes them as beginning with a warm feeling in her face before blacking out and awakening several minutes later. There have been several incidents of loss of bladder control during these episodes and witnesses report that she is confused and difficult to arouse for up to 30 minutes after the episodes. All of these episodes she describes have a wide variety of symptoms; including chest pain, vomiting, sharp abdominal pain, and difficulty breathing. At her outpatient visit she was aware that her episodes are not related to epilepsy and was aware of the term "psychogenic seizures".

Upon further questioning it became apparent that she had been suffering from symptoms of severe anxiety for several years that had interfered with her education, interpersonal relationships, as well as employment. It was discovered that her mother had passed away when the patient was 4 years old, the man she believed to be her father was actually a step-father who had abruptly stopped having contact with the patient in the past one year. Her biological father signed away all parental rights during her early childhood, has attempted to reconnect with her in the past year., coinciding with her stepfathers absence. Additional stressors include her close friend was recently killed in a MVA which has directly contributed to her anxiety with driving and being in cars.

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Abstract 19-2-11

Title: Long-standing Traumatic Brain Injury with superimposed cognitive decline and sleep-wake disorders

Authors: Heidi Lai, OMS3; Lori Lowthert, MD

Summary: Traumatic brain injury (TBI) is defined as an interference in brain function that is caused by a physical harm to the head. Whether or not the TBI is mild, moderate, or severe, there are studies that show that it carries an increased risk of developing dementia later in life. The neurodegeneration and progressive brain atrophy following the TBI plays a role in the pathology of this risk. Furthermore, sleep wake disturbances are a potential sequelae of TBI. The damage to orexin neurons may contribute to this outcome, leading to clinical features like excessive daytime sleepiness, increased sleep need, insomnia, and circadian rhythm disturbances.

Our case is a 72 year old female admitted for court ordered evaluation for major neurocognitive disorder with behavioral disturbances. Upon evaluation, she was found to have a past medical history of traumatic head injury with cervical spine injury due to a car accident in her twenties. Furthermore, she reports being diagnosed with obstructive sleep apnea and narcolepsy by a polysomnography after this accident. A MoCA was performed and she received a score of 17/30, which puts her in the range of having cognitive impairment. This case explores the associations between traumatic brain injuries, cognitive decline, and sleep wake disorders.

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Abstract 19-2-12

Title: Quality Improvement Project at Mobile, AL Buprenorphine Clinic – Teaching Model Using a Real-World Example

Authors: Michael E. Heisler, BS; Tina Jackson, MD; Lindsey Stewart, MD; Marianne Saitz, DO.

Summary: Institutions are constantly seeking ways to improve the quality of patient care. The most effective and sustainable approach to address this concern by organizations requires educating future physicians on implementing patient-centered changes to existing healthcare delivery. The Accreditation Counsel for Graduate Medical Education (ACGME) guidelines for quality improvement curricula state that "*residents must receive training and experience in quality improvement processes*", which are essential in *"developing the ability to identify and institute sustainable systems-based changes to improve patient care.*" Despite ACGME recommendations, medical schools and residency programs continue to struggle implementing and sustaining quality improvement curricula. Some evidence suggests that programs tend to favor traditional bioscientific research over quality improvement research because the former serves to boost the respective program's reputation. As opposed to clinical trials, quality improvement initiatives assess the impact of interventions on the quality of patient care at an individual and/or population level in practice. Plan-Do-Study-Act (PDSA) cycles are an example of this method. The Plan-Do-Study-Act (PDSA) model uses an iterative process to test small-scale changes in routine practice with aims to improve quality of healthcare delivery. These cycles are repeated with alterations until the desired improvement to the system is achieved. The PDSA method was used when the department of psychiatry at University of South Alabama recognized that patients were failing to self-administer buprenorphine at an appropriate dose because they struggled to interpret instructions on a brochure provided by a buprenorphine clinic in Mobile, AL. After the brochure underwent modification via the PDSA model, patients found it easier to follow, which led to an enhanced response. Quality improvement practices help address the disconnect between actual and ideal patient care by improving our ability to provide favorable outcomes that are beneficial, efficacious, and safe.

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Abstract 19-2-13

Title: Lennox-Gastaut Syndrome

Authors: Alex Kearns; Janaki Nimmagadda MD; Clinton Martin MD

Summary:
Overview:
Lennox-Gastaut Syndrome (LGS) is a neuropsychiatric disturbance of childhood, which involves recurrent seizures and cognitive decline. Up to 59% of patients are developmentally normal until onset of LGS. Patients also frequently experience other psychiatric symptoms, with features of psychosis, ADHD, Autism, dyslexia, and tics.
30-50% of children with infantile spasms will develop LGS. 70% with LGS will show cognitive impairment at diagnosis and more than 50% suffer from behavioral symptoms including rage attacks, aggression, hyperactivity, autistic features and sleep disturbances. Up to 60% of patients graduate high school, given appropriate assistance like special education classroom or home tutoring. Traditional pharmacotherapy typically involves antiepileptic drugs: Valproate, Lamotrigine, or Topiramate.
Case:
13 yo male has a history of persistent seizures since age 5 following a severe streptococcal infection. He previously had normal developmental milestones but has progressive cognitive decline since the onset of seizures. He has been through several anticonvulsant trials, with little result, until he began using Clobazam, which provided moderate seizure reduction. Now he has near-total seizure reduction on combination therapy with Clobazam and Cannabidiol. His comorbid symptoms include poor communication, inattention, hyperactivity, aggression, and impulsivity. His aggressive and impulsive behaviors have been worsening with his cognitive decline, necessitating trials of multiple SSRIs and antipsychotics. He is currently on two antipsychotics and 2 SSRIs. Attempts have been made to reduce his number of medications, but his behaviors become unmanageable, each time.

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Abstract 19-2-14

Title: Inflammatory Markers in Adolescents with Major Depressive Disorder

Authors: Abhishek Reddy, MD; Mounica R. Thootkur, MBBS; Li Li, MD

Summary:

Objectives: Studies in adults have shown that patients with MDD show increased circulating levels of inflammatory markers such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNFα). However, there is limited or no data on the relationship between inflammatory markers and adolescent MDD. The objective of our study was to explore the role of inflammatory markers in adolescents with MDD.

Methods: Eighty-five female and male, black and white, adolescents aged 15–18 years completed the study. Adolescents were diagnosed with MDD according to the *DSM-5* as confirmed by the Mini International Neuropsychiatric Interview. The severity of depression was assessed using the Quick Inventory of Depressive Symptomatology by self-report. Blood samples were collected from each participant for measuring the inflammatory factors.

Results: Analysis of covariance adjusting for race, gender, BMI, and early life stress status indicated that the adolescent group with MDD had significantly elevated TNFα levels compared with the control group. However, other measured inflammatory factors such as IL-6, IL-8, IL-12, and C-reactive protein did not differ between the 2 groups. The levels of TNFα, an inflammatory marker, were greatly elevated in the group with MDD (1.5 ± 0.3 vs. 0.5 ± 0.1 pg/ml) compared with the control subjects.

Conclusions: Very few studies in the past have explored the role of inflammatory markers such as TNFα in adolescent depression. In our study, adolescents with MDD had significantly elevated TNFα levels compared with the control group, which is consistent with findings in adult studies. Our findings add to the literature the role of inflammatory markers in adolescent MDD.

Abstract 19-2-15

Title: Wounded Healers: Distress Among UAB Residents and Faculty as assessed by the Well-Being Index

Authors: Austin Luker, MD; Abhishek Reddy, MD; Laura Lockwood, DO; Taylor Preston, MD; James Meador-Woodruff, MD, PhD; Lee Ascherman, MD, MPH; Irena Bukelis, MD

Summary:

Background: Burnout is a well-established problem among physicians, which can negatively impact 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 aimed to use the established Well-being Index to measure distress among UAB residents, fellows, and faculty.

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," as this cutoff score has been demonstrated to correlate to increased chances of medical error, poor quality of life, and burnout.

Results: There were 573 faculty responders, 15 of whom were psychiatry faculty. The mean distress score among UAB psychiatry faculty was 0.93, compared with 2.14 for UAB faculty overall, and 1.73 for physicians nationally. Using a cut-off of >3 for "high distress," 20% of UAB psychiatry faculty were found to be in high distress, compared with 45% for UAB faculty as a whole, and 39% for physicians nationally. The mean distress score among UAB psychiatry residents (n=10) was 2.90, compared with 2.84 for UAB residents overall. Twenty percent of UAB psychiatry residents were found to be in high distress, compared with 23% for UAB residents as a whole.

Conclusion: Burnout and distress is a problem across medicine, with UAB being no exception. Though high distress among UAB psychiatry was lower than that of the national average, the study was limited by small response rate and there is the possibility of selection bias, as, given the voluntary nature of the survey, those with significant distress and burnout may not have been willing or able to complete the survey.

Abstract 19-2-16

Title: Supplement Induced Mania in a Patient with Bipolar Disorder

Authors: Evan Chavers, MS, MS2; Griffin Gibson II, MD; Praveen Narahari, MD

Summary: Energy/workout supplements and over the counter (OTC) herbal medications have become commonly used substances over the past several decades. Unfortunately, there is limited scientific evidence to establish the safety and efficacy of most energy enhancing products. We present this case of supplement induced mania to draw attention to the importance of obtaining more thorough histories based on recent trends of increasing OTC supplement(s) use. A 42 year-old Caucasian male with bipolar one disorder presented with recurrent mania with psychotic features in context of a negative urine drug screen (UDS) and medication compliance with mood stabilizer and long-acting injectable (LAI). After multiple hospitalizations for mania, the consumer revealed he was using body-building supplements between episodes. With continued prescription medication use and abstinence from OTC body building supplements, the consumer has avoided hospitalization for the past several years. We recommend considering use of commercially available supplements when discussing treatment plans for consumers with mood disorders.

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Abstract 19-2-17

Title: A case of the creepy crawlies: A unique presentation of acute psychosis in chronic schizophrenia

Authors: Taylor Ousley, MS4; Lori Lowthert, MD

Summary:

Introduction: Schizophrenia is diagnosed by the presence of a variety of different types of psychosis, as well as negative symptoms. In order to have this diagnosis, a patient must have two or more symptoms which include delusions, hallucinations, disorganized speech or behavior, and negative symptoms such as avolition or decreased emotional expression. The patient must also experience these symptoms for more than 6 months to have the official diagnosis. Tactile hallucinations are not a common presentation of psychosis in this illness. The tactile hallucination of bugs crawling is usually more indicative of substance use, such as cocaine or alcohol, or of a diagnosis of delusional parasitosis.

Case Description: Here we present a patient that is a 61-year-old male with a prior diagnosis of schizophrenia, paranoid type. He also had a history of medication noncompliance for approximately 6 months prior to his presentation. Our patient was experiencing an exacerbation of his illness that included a unique symptom of psychosis, which he had not typically dealt with in the past. He had chronic paranoid delusions, but he was also experiencing tactile hallucinations of feeling bugs crawling

all over him. This was a very unusual hallucination for a patient that was not withdrawing from an illicit substance. Our patient had a negative drug screen and he denied any drug use. Throughout his hospital stay, he was exhibiting signs of anxiety and depression in addition to his usual paranoid delusions. He also had several traits that pointed to a possible diagnosis of obsessive-compulsive disorder (OCD). This made his differential diagnosis for his current presentation difficult to pinpoint due to the possible overlap of unique presentations of a variety of psychiatric illnesses. In this case, we highlight the common comorbidity of OCD in patients with a previous diagnosis of schizophrenia.

Discussion: It has been shown that there is a correlation between OCD and schizophrenia due to the strong propensity for patients with either diagnosis to also develop the other disorder. It is common for both OCD to occur after the onset of schizophrenia and vice versa, as well as for both to present at the same time. Because of the common connection between traits and symptoms of OCD in patients with schizophrenia, it has even been suggested that there is evidence for a schizo-obsessive subtype of schizophrenia. This association has led to various studies that are examining the possibility of a similar mechanism of psychopathology between the two disorders.

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Abstract 19-2-18

Title: Ischemic stroke with oxymetazoline nasal spray use: A Case Report

Authors: Darshana S. Pai, M.D.; Nikky Bardia, M.D.; Severin Grenoble, M.D.

Summary:

Introduction: The nasal decongestants are mostly readily available over-the-counter and widely used by people for allergies. The systemic effects of these medications would include tachycardia, hypertension and in rare circumstances damage to end organs[1]. There have been case reports which show an association of oxymetazoline and stroke[2]. We would like to describe a case of a middle-aged female with chronic oxymetazoline nasal spray use presenting with abnormal choreiform movements secondary to an ischemic stroke.

Case report: The patient is a 58-year-old female with no past history of psychiatric illness was admitted to the USA Hospital with chief complaints of involuntary movements of the left hand and twitching movements of the left side of the face. She gave a history of receiving a steroid injection locally in her right hand for chronic pain secondary to trauma around 10 days ago and after that she developed these involuntary movements and also noted some funny speech at that time. She was evaluated by the internal medicine and neurology team. Her lab workup did not reveal any significant findings and her urine drug screen was positive for benzodiazepine. The patient's CT scan of the brain revealed hypodensity in the left frontal matter region just lateral to the caudate nucleus. The neurology team opined that these movements are consistent with sudden onset chorea likely secondary to a vascular etiology of ischemic stroke in the peri-caudate fibers. The neurology team recommended to start Risperdal 1 mg at bedtime for 1 week and then gradually increase it to 1 mg twice a day.

The patient endorsed using nasal spray containing oxymetazoline for around 3-4 times per day for the last 2 years for allergies. She stated that if she would not use a spray she would get clogged up and feel stuffy. She gave history of smoking cannabis around 3-4 times per week and also consuming Xanax and Percocet but denied any habitual use. She endorsed history of consuming alcohol about twice a week and smoking nicotine cigarettes around 1 pack of cigarettes which would last for around 3-4 days. The patient denied symptoms of depression, mania, anxiety, psychosis or suicidal ideation. The patient's abnormal movements intensified during periods of stress and were noted to disappear upon distraction raising a concern for unspecified somatic symptom and related disorder. The patient was provided with reassurance, asked to abstain from drug use including oxymetazoline and follow up with neurology and psychiatry outpatient clinic.

Discussion and conclusion:
Oxymetazoline is an alpha 2 adrenergic agonist, a component of nasal decongestant spray. There is a case report of patient having cerebral vasoconstriction with use of oxymetazoline[3]. This patient developed sudden involuntary choreiform movements of the left hand and left side of the face which could correlate with the ischemic lesion found near the right caudate nucleus. This case was unusual in that the abnormal movements had a psychological component and would increase in intensity with stress and disappear on distraction. As nasal decongestants are meant for local use, short term use is considered relatively safe. However, chronic habitual use may give rise to systemic symptoms due to catecholamine surge including a potential to cause stroke.

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Abstract 19-2-19

Title: Agitation, the barrier for discharge in a patient with Familial Encephalopathy with Neuroserpin Inclusion Bodies (FENIB)

Authors: Katherine E. Glosemeyer, M.D.; Tarak Vasavada, M.D.

Summary: Mr. S., a 56-year-old Caucasian male with Familial encephalopathy with neuroserpin inclusion bodies (FENIB) with associated dementia and cognitive deficit in communication, presents to the psychiatric consult service for agitation. FENIB is an autosomal dominant adult-onset dementia due to neuroserpin, which is a protease inhibitors, depositing in the cortex. Mutations in the SERPIN1 gene have been isolated as the cause of this disorder as a mutation causes the neuroserpins to fold abnormally. The patient was admitted to the internal medicine service for acute encephalopathy from a skilled nursing facility. The patient sustained a fall at the outside facility and was noted to have an elevated temperature of 100 degrees Fahrenheit. CT head was negative for acute intracranial hemorrhage. Infectious etiologies were ruled out with negative urinalysis, normal urine culture, normal complete blood count, and negative blood culture. Psychiatric consult service was asked to assist in the treatment management of agitation given the patient's FENIB. Quetiapine and Clonazepam were added initially in addition to the patient's home doses of divalproex and carbidopa-levodopa. Despite medication changes, the patient still displayed agitation and violent behavior temporally related to dressing changes; therefore, medication doses were increased. Patient was put in soft restraints episodically throughout the hospital course. Literature review of FENIB was scarce thus impeding improvement in treating agitation in this FENIB patient. Ultimately the patient's agitation was controlled, and he was successfully placed at a skilled nursing facility. In this poster, we discuss an overview of FENIB diagnosis and the shortcomings and challenges of providing recommendations for the treatment of agitation in a patient with the neuropsychiatric disorder of FENIB.

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Abstract 19-2-20

Title: A case report of priapism secondary to low dose trazodone use.

Authors: Alexandru Ghilezan, DO; Praveen Narahari, MD

Summary: Priapism is a urological emergency which presents as a persistent erection in the absence of sexual arousal and is a well-known, albeit fairly uncommon, side effect of trazodone.

Here we present the case report of priapism in a 21 year old patient with a history of borderline personality disorder, major depressive disorder, and unspecified anxiety disorder who was taking 50mg of trazodone as needed for occasional insomnia. Patient experienced an erection which awoke him from sleep in the night and persisted until noon, lasting roughly 12 hours, before he reported it to the staff's attention. Patient required transfer to local emergency department for appropriate treatment after which his condition resolved. On interview the following day, patient stated this had been the second time he had experienced such symptoms, reporting that he had had a similar erection which lasted 8 hours during his current hospitalization but had been too embarrassed to report it.

This case illustrates the need to properly educate patients regarding the side effects of trazodone as well as fostering an environment of trust that would allow them to report such events. Although priapism is a well-known side effect to medical professionals it is still a rare occurrence and patients may disregard the symptoms, or be too embarrassed to report it, which could lead to serious consequences. Recognition by patients' of this side effect is critical to receiving timely care.

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Abstract 19-2-21

Title: Synthetic Cannabinoid (Spice) induced Pancreatitis: Acute Synthetic Cannabinoid Induced Pancreatitis and Psychosis.

Authors: Praveen Narahari, M.D., Shanthi Gatla, M.D., Kelsey Templeton, M4.

Abstract: Synthetic Cannabinoids (Spice) is created via an herbal mixture sprayed with active, synthetic cannabinoids that activate similar receptors to THC.[1] These chemicals create the sensations of euphoria and relaxation, but also disinhibition. Synthetic cannabinoids were previously legally available to the general public, and have continued to remain on the market while sold as incense, or with constantly varying ingredients (including pesticides and insecticides) that help the product evade legal prohibition. Due to the lack of regulation, the exact dosages of the THC-like compounds are varying and often unknown contributing to the varying clinical reactions to the products.[2] synthetic cannabinoid consumption has led to serious physical consequences, such as, seizures, myocardial infarction and renal damage but has not been associated with pancreatitis.[3]

Cannabis-induced pancreatitis has been previously reported several case reports.[4,5] Studies have demonstrated the existence of both CB1 and CB2 receptors in the endocrine pancreas which regulates Ca+2 signals and insulin secretin.[6] There is no clear existing data available on spice induced pancreatitis in our literature review.

We report a case of a 28-year-old female admitted to an inpatient psychiatry hospital for psychosis and bizarre behavior after consumption of spice who complained of nausea and epigastric pain found to have acute pancreatitis requiring admission to the ICU for stabilization and prior to transferring back to the psychiatric hospital for stabilization of psychosis. No other risk factors for acute pancreatitis were present except the use of spice.

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Abstract 19-2-22

Title: Hypothyroidism induced psychosis or primary psychotic disorder?

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Summary: Hypothyroidism affects up to 5% of the world population, with a further estimated 5% being undiagnosed. The most common cause of hypothyroidism worldwide is iodine deficiency, while in iodine replete areas; the most common cause is the autoimmune condition, Hashimoto's disease. Hypothyroidism is diagnosed biochemically with overt primary hypothyroidism being defined as serum thyroid-stimulating hormone (TSH) concentration above and thyroxine concentrations below the normal reference range. The clinical presentation of thyroid deficiency is varied and non-specific, including fatigue, lethargy, cold intolerance, weight gain, constipation, change in voice, and dry skin, but clinical presentation can differ with age, sex, and other factors. Dysthyroidism is involved in various psychiatric pathologies, especially mood disorders. The association of thyroid dysfunction with alterations in mood and cognition has been recognized since some of the earliest descriptions of thyroid disease. However, there are few reported cases of hypothyroidism induced psychosis. A retrospective analysis of 15 patients with hypothyroidism induced mental disorder, after primary overactive thyroid surgery or ^{131}I therapy, documented clinical symptoms including fatigue and mood swings accompanied by hallucinations, delusions, mental retardation, and behavioral disorders.

We report two patients admitted to an involuntary psychiatric hospital with similar presentations of acute psychosis in the context of sever hypothyroidism. The first patient is a 65 year old female diagnosed with hypothyroidism at the age of 40 after Radioiodine (I-131) therapy for hyperthyroidism. She was later diagnosed with psychotic disorder at age 61 after wandering off in the streets, which was her first episode. She became homeless for 2 years and was not compliant with treatment and then presented to inpatient psychiatry with acute psychosis in a different city. She was diagnosed with schizophrenia upon admission and comorbid hypothyroidism with TSH of 75.6 mU/L a week later. Similarly, the second patient presented with untreated hypothyroidism post-thyroidectomy but had no known psychiatric illness. The patient had a good social history on admission and had worked as an FBI agent for 20 years and is now retired. He was diagnosed with psychotic disorder due to general medical condition. These similar presentations of acute psychosis in the presence of hypothyroidism illustrate thyroid deficiency as a probable cause of psychotic manifestations. This highlights the need to eliminate the possibility of psychosis due to general medical condition before diagnosing the patient with a primary psychotic disorder which may require lifelong treatment.

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Abstract 19-2-23

Title: Anxiety as the presenting symptom for a neurologic disorder: A case of Stiff Person Syndrome

Authors: Loftin, Cade MS; Jackson, Tina MD; Brooks, William MD.

Summary: Anxiety is a common presenting symptom for many patients in different clinical settings. For the vast majority, the basis of their discomfort will be found in the mental health aspect of their workup. However, many disease processes have anxiety as an associated symptom. It is imperative that we do not forget to be vigilant to keep our diagnostic possibilities open. The literature is replete with examples of psychiatric cases that had medical diagnoses that were overlooked when physicians declined to consider options.

We present a case of a 39 yo AAF who initially presented to the psychiatrist after 5 years of anxiety that progressively worsened in the context of later emerging neurologic symptoms. She was not diagnosed with Stiff Person Syndrome until more than 8 years of symptom onset. During that time she was treated with many psychotropic drugs and at one point was on as many as 4 anxiolytic medications.

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