ALABAMA PSYCHIATRIC PHYSICIANS ASSOCIATION
A District Branch of the American Psychiatric Association

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Abstract 19-1-01

Title: Reawakening Encephalitis Lethargica

Authors: Griffin F. Gibson II, MD; Marcus W. Lee, MD

Summary: Encephalitis Lethargica (EL) was first described in 1917 by Constantin Von Economo[1]. During EL's global pandemic period of 1919-1930s, an estimated 22,942 deaths were attributed to EL in the US alone[2] However, as abruptly as it presented it resolved with only rare cases reported since then worldwide. EL is difficult to confirm with lack of consensus regarding diagnostic criteria and uncertainty with pathoetiology. Presently, it is diagnosed following encephalitis with resulting parkinsonism and/or neuropsychiatric symptoms. This case is regarding a 20-year-old male with rapid-onset, unspecified encephalopathy, parkinsonism, and catatonia. He had failure of treatment using benzodiazepine and Electroconvulsive Therapy (ECT) but had improvement of all symptoms with addition of dopaminergic medication confirming diagnosis of EL.

References:

1. v. Economo C. 1917 Encephalitis Lethargica, Wien Klin Wschr 30:581-585
2. Linder F Grove R. *Vital statistics rates in the United States: 1900-1940*.Washington: USGPO,1943
3. Crookshank FG. A note on the history of epidemic encephalitis. *Proc R Soc Med*1918;12: 1–21
4. Leslie A Hoffman, Joel A Vilensky; Encephalitis lethargica: 100 years after the epidemic, *Brain*, Volume 140, Issue 8, 1 August 2017, Pages 2246-2251, https://doi.org/10.1093/brain/awx177
5. Ravenholt RT,FoegeWH.1918 Influenza, encephalitis lethargica, parkinsonism. *Lancet* 1982;2:860–4
6. Russell C. Dale, Andrew J. Church, Robert A. H. Surtees, Andrew J. Lees, Jane E. Adcock, Brian Harding, Brian G. R. Neville, Gavin Giovannoni; Encephalitis lethargica syndrome: 20 new cases and evidence of basal ganglia autoimmunity, *Brain*, Volume 127, Issue 1, 1 January 2004, Pages 21–33, https://doi.org/10.1093/brain/awh008
7. Hu SC, Hebb AO. Effect of deep brain stimulation on postencephalitic parkinsonism: a case report. *Mov Disord* 2012; 27: 1071–2

Abstract 19-1-02

Title: A curious case of odd smells: Depression secondary to phantosmia.

Authors: Darshana S. Pai, M.D., Malik J. McMullin MS3, Elisabeth Potts MS3, Kinjal Ghelani, M.D., J.Luke Engeriser, M.D.

Summary: The intersection of the field of psychiatry and neurology has always been intriguing. It is known that psychiatric disorders are more prevalent in patients with epilepsy than the general population. Among the epileptic disorders, temporal lobe epilepsy is known to be more complex in symptomatology. It has been an area of interest to researchers due to its varied presentation such as déjà vu experiences, gastrointestinal upset, lipsmacking movements, pelvic thrusting, language difficulties, etc. Phantosmia (phantom smell), an olfactory hallucination, is a phenomenon of smelling an odor which is not actually there. Olfactory hallucinations can be seen in temporal lobe epilepsy[1]. There are studies which have shown an association between a higher incidence of depression and temporal lobe epilepsy, particularly in patients with left temporal lobe epilepsy[2,3,4,5].

We present a case of a middle-aged male with no past history of psychiatric illness who presented with depressive symptoms with suicidal thoughts and olfactory hallucinations. One year ago, the patient developed an odd sensation of smelling burnt plastic which nobody else experienced. He would have episodes during which he would get odd smells associated with short-term memory loss. In an attempt to alleviate the symptoms, the patient used strong scents such as vapor sticks with no relief. The patient was evaluated by an otorhinolaryngologist, but no abnormalities were detected. He was seen by a neurologist and an EEG was performed but was negative possibly because he had taken diazepam prior to the EEG. An MRI of the brain revealed left hippocampal atrophy. Gradually with time these smells became much more frequent, persistent, and resulted in his social isolating. His symptoms interfered with his work performance, resulting in a demotion at his workplace. The patient became significantly distressed to the point that he became depressed, could no longer experience pleasure, felt hopeless, worthless, and inadequate, lacked concentration and was not able to sleep and eat. He developed recurrent suicidal thoughts and expressed a passive death wish. The patient presented to a private hospital and was then referred to our facility for further management. He was diagnosed with adjustment disorder with depressed mood and anxiety with olfactory hallucinations and a provisional diagnosis of temporal lobe epilepsy and major depressive disorder. He was initiated on venlafaxine 75 mg daily which was increased to 150 mg daily and mirtazapine 15 mg at night. He also received individual psychotherapy and group therapy sessions. During the hospital stay, he had a few episodes of phantosmia with confusion which subsided either spontaneously or with clonazepam 0.5 mg PRN. Once the patient's depressive symptoms resolved and he was no longer suicidal, he was discharged home. The patient then followed up with neurology and received another EEG which showed epileptiform activity in the left temporal/frontotemporal region. He showed good response to valproic acid with symptomatic improvement. This case highlights the importance of having a clinical suspicion for and understanding of underlying neurological problems that could manifest with psychiatric symptoms.

References:

1. Chen C, Shih YH, Yen DJ, Lirng JF, Guo YC, Yu HY, Yiu CH. 2003. Olfactory auras in patients with temporal lobe epilepsy. Epilepsia. 44(2):257–260.
2. Shukla, G.D., Srivastava, O.N., Katiyar, B.C., Joshi, V., Mohan, P.K., 1979. Psychiatric manifestations in temporal lobe epilepsy: a controlled study. Br. J. Psychiatry 135, 411–417.
3. Perini, G., Mendius, R., 1984. Depression and anxiety in complex partial seizures. J. Nerv. Ment. Dis. 172, 287– 290.

4. Robertson, M.M., 1989. The organic contribution to depressive illness in patient with epilepsy. J. Epilepsy 2, 189– 230.
5. Altshuler, L.L., Devinsky, O., Post, R.M., Theodore, W., 1990. Depression, anxiety, and temporal lobe epilepsy. Arch. Neurol. 47, 284–288.

Abstract 19-1-03

Title: Violence in the Emergency Department (ED) and Caring for the Acute Psychiatric Patient.

Authors: Nicholas Quigley, OMS3; Tina Jackson, PGY3; Lori Lowthert, MD

Summary: Violence against ED staff is on the rise. More than 40% of ED physicians polled believe that over half of assaults committed in the ED are by patients with a psychiatric illness. When patients present in acute psychiatric crisis or under the influence of illicit drugs, close interaction with hospital staff is required to provide necessary medical interventions. These encounters place staff at increased risk for injury. As more psychiatric care is being shunted to the ED because of shortages in available psychiatric care facilities, the need to address staff safety in the ED is a more pressing topic of discussion in urgent care settings. A case-control study found that length of stay longer than 24 hours in an emergency care setting was associated with the use of restraints. We present the case of a 28-year-old male who presented acutely psychotic to the ED in the custody of the police. Due to shortages of psychiatric beds, the patient required acute stabilization in the ED until a bed became available. During administration of an injection, one of the staff members was attacked and received a needle injury. Topics explored include the legality of physical restraints, chemical restraints, pressing charges against patients, and psychiatric ED units. This case also highlights the need for implementing and adhering to protocols when dealing with agitated patients in the ED.

References:

1. Violence in Emergency Departments is Increasing, Harming Patients, New Research Finds. Newsroom.acep.org.http://newsroom.acep.org/2018-10-02-Violence-in-Emergency-Departments-Is-Increasing-Harming-Patients-New-Research-Finds. October 2, 2018.
2. Cowan E, Macklin R. Unconsented HIV testing in cases of occupational exposure: ethics, law, and policy. Acad Emerg Med. 2012;19(10):1181-7.
3. Dutton, Audrey. She Went to the Hospital for Mental Illness. She Ended Up Charged with 2 Felonies. Idahostatesman.com.https://www.idahostatesman.com/news/politics-government/state-politics/article195828984.html. January 20, 2018.
4. Workplace Violence in Healthcare. Osha.gov.https://www.osha.gov/Publications/OSHA3826.pdf
5. State Laws that address High-Impact HIV Prevention Efforts. Cdc.gov. https://www.cdc.gov/hiv/policies/law/states/index.html. December 14, 2018.
6. Schyve, Paul. Violence in the Heath Care Setting. Jointcommision.org. htftps://www.jointcommission.org/search/?Keywords=restrain&f=sitename&sitename=Joint+Commission. May 14, 2014.
7. Nordqvist, Christian. Healthcare most dangerous place for workplace injuries. Medicalnewstoday.com. https://www.medicalnewstoday.com/articles/263709.php. July 20, 2013.
8. Thomas J, Moore G. Medical-legal Issues in the Agitated Patient: Cases and Caveats. *West J Emerg Med*. 2013;14(5):559-65.
9. Park JM, Park LT, Siefert CJ, et al. Factors associated with extended length of stay for patients presenting to an urban psychiatric emergency service: a case-control study. J Behav Health Serv Res. 2009;36(3):200-208.

Abstract 19-1-04

Title: A Tale of Two Brains: Personality Change Due to a Medical Condition

Authors: James R. Le Page, D.O., Darshana S. Pai, M.D., Candace Perry, M.D.

Summary: Brain tumors are relatively common, with an annual incidence of 9 per 100,000 patients for primary brain tumors and 8.3 per 100,000 patients for metastatic brain tumors.[1] Brain tumors often present with headache, nausea, projectile vomiting and blurring of vision. However, any acute and drastic change in the patient's behavior should also raise concern for underlying brain pathology.[2,3] Personality change may be the first signs of illness in about 15-20% of patients with brain tumor.[1] Personality change following a frontal lobe insult has been described in the literature as far back as 1835, and was popularized with the case of Phineas Gage in the mid-19th Century.[4] In this case series, we present two cases illustrating the DSM-5 diagnosis of Personality Change Due to a Medical Condition.[5] We first present the case of a 28 year-old female patient who is allegedly responsible for a case of severe neglect involving her child, resulting in her incarceration. This behavior was completely uncharacteristic of the patient based on descriptions of her personality given by friends and family. She later presented to the hospital ED from jail with chief complaint of bitemporal vision loss, and was found to have a giant olfactory groove meningioma measuring 6.0 x 6.5 x 4.7 cm centered in the anterior cranial fossa, exerting significant mass effect on bifrontal lobes and temporal horns. The mass was resected and following the surgery, a consult was performed to assess suicide risk due to a suicidal threat she made while in the ED. She was found to have profound flattening of affect, anhedonia, and apparent indifference to her numerous biopsychosocial stressors. The second case we present is that of a 46 year-old male patient who presented with new onset of cognitive deficits, depression, suicidal ideation, unprovoked aggression, and worsening impulsive behavior at home. The case is unique, in that this patient had a metastasis of primary melanoma to the calvarium, which is known from the literature to be a relatively uncommon phenomenon.[6] There were initially two hemorrhagic lesions found in the temporoparietal area, measuring 4.2 x 3.9 x 3.9 cm and 3.9 x 3.1 x 3.0 cm, respectively. At time of consult, the patient was status post resection of left posterior temporal mass, and status post left parietal and medial right parietal radiotherapy. His behaviors of exorbitant spending, severe aggression towards his wife, new onset of substance use, and leaving his family to live homeless for days were also completely out of character based on collateral obtained from family. This case is an example of the DSM-5 combined type of this disorder, with aggressive and disinhibited features.[5]

References:

1. Madhusoodanan S, Ting MB, Farah T, Ugur U. Psychiatric aspects of brain tumors: A review. *World J Psychiatr.* 2015; 5(3): 273-285.
2. Pranckeviciene A, Deltuva VP, Tamasauskas A, Bunevicius A. Association between psychological distress, subjective cognitive complaints and objective neuropsychological functioning in brain tumor patients. *Clinical Neurology and Neurosurgery.* 2017; 163: 18-23.
3. Richter A, Woernle CM, Krayenbuhl N, Kollias S, Bellut D. Affective symptoms and white matter changes in brain tumor patients. *World Neurosurg.* 2015; 84(4): 927-932.
4. Barrash J, Stuss DT, et al. "Frontal lobe syndrome"? Subtypes of acquired personality disturbances in patients with focal brain damage. *Cortex.* 2018; 106: 65-80.
5. American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.). Arlington, VA: American Psychiatric Publishing.
6. Harrison RA, Nam JY, Weathers SP, DeMonte F. Intracranial dural, calvarial, and skull base metastases. *Handb Clin Neurol.* 2018; 149: 205-225.

Abstract 19-1-05

Title: Human Side of Medicine: Understanding Physician Burnout at UAB Department of Psychiatry

Authors: Austin Luker, MD ; Adefolake Akinsanya, MD; Abhishek Reddy, MD; Laura E Lockwood, DO; Lee I Ascherman, MD; James Meador-Woodruff, MD; Irena Bukelis, MD

Summary:
Objectives: 1) Explore the rate of UAB attending burnout in the psychiatry department. 2) Compare the rate of attending burnout at UAB in the psychiatry department to the overall rate of attending burnout at UAB. 3) Compare the rate of burnout to nationwide burnout rates, both for psychiatry and overall. 4) Discuss possible reasons for the high burnout rate. 5) Discuss the impact of work-life balance on burnout.

Methods: The UAB Faculty Engagement survey was administered to all attending physicians. There were questions regarding “Do you feel burned out?” and “Do you have a good work-life balance?” Thirty-five responses were received within the psychiatry department among attending physicians and were assessed.

Results: Of UAB attending physicians as a whole, 39% answered that they were burned out, and 29% that they were somewhat burned out. Of the UAB psychiatry attending physicians who responded, 47% responded that they were burned out, and 28% that they were somewhat burned out. Seventy percent of UAB psychiatry attending physicians answered that they had a good work life balance. The results of which are pending.

Conclusions: This is compared with national data for burn out. Nationally in a recent Medscape poll 44% of all physicians were burned out. For psychiatrists, the burnout rate was 39%. There are many factors which contribute to burnout. These include promotion and tenure, efficiency, use of electronic medical record (EMR) systems, reputation, sense of well-being at work, as well as sense of work-life balance. Therefore, the high work-life balance among the UAB psychiatry attending physicians does not exclude the higher than national average burnout rate. Psychiatry is traditionally one of the medical professions with a lower burnout rate. However, there are unique factors which can contribute to burnout among psychiatry, include the unique and sometimes emotionally demanding relationship among providers to their patients. There is a dearth of psychiatrists in Alabama, which may contribute to the higher than average rate of burnout among the UAB psychiatry faculty.

Abstract 19-1-06

Title: Clinical Course of a Patient Undergoing Delayed Withdrawal from Alprazolam (Xanax)

Authors: Jazmín Scott, BS; Praveen Narahari, MD

Summary: Benzodiazepines are one of the most widely prescribed classes of medications despite their association with multiple adverse events such as misuse, tolerance, and withdrawal. They are considered most appropriate for short-term use of 2 to 4 weeks with increased risk if used long-term. This case describes a 54 year old female who was admitted to an inpatient psychiatric facility following a suicide attempt in which she overdosed on an opioid and a benzodiazepine. She experienced a delayed withdrawal syndrome nearly two weeks after her last dose of Alprazolam, which she had been taking for the past two years. The goal of this case is to raise awareness of the risks associated with long term benzodiazepine use and delayed withdrawal after completion of clinical institute withdrawal assessment protocol-benzodiazepines. Additionally, this case raises awareness of the danger of benzodiazepine use when coupled with opioid medications.

References:

1. Ait-Daoud, N., Hamby, A. S., Sharma, S., & Blevins, D. (2017). A Review of Alprazolam Use, Misuse, and Withdrawal. Journal of Addiction Medicine, 12(1), 4-10. doi:10.1097/adm.0000000000000350
2. Hood, S. D., Norman, A., Hince, D. A., Melichar, J. K., & Hulse, G. K. (2014). Benzodiazepine dependence and its treatment with low dose flumazenil. British Journal of Clinical Pharmacology,77(2), 285-294. doi:10.1111/bcp.12023
3. Meyer, J. S., & Quenzer, L. F. (2013). Web Box 18.4: Pharmacology in Action: GABA-A Receptor Subunit-selective Drug Development. Retrieved February 27, 2019, from http://sites.sinauer.com/psychopharm2e/webbox18.04.html
4. National Institute on Drug Abuse. (2018, March 15). Benzodiazepines and Opioids. Retrieved February 27, 2019, from https://www.drugabuse.gov/drugsabuse/ opioids/benzodiazepines-opioids
5. September;12(9):55-56, C. P. (2018, April 16). Consider this slow-taper program for benzodiazepines. Retrieved February 27, 2019, from https://www.mdedge.com/psychiatry/article/777248/addiction-medicine/considerslow-taper-program-benzodiazepines
6. Soyka, M., MD. (2017). Treatment of Benzodiazepine Dependence. New England Journal of Medicine,376(24), 2397-2400. doi:10.1056/nejmc1705239

Abstract 19-1-07

Title: Hyperglycemia-Induced Resistance to Antipsychotic Treatment

Authors: Taylor Ousley, MS3, Shanthi Gatla, MD, Praveen Narahari, MD

Summary:
Introduction: The association between diabetes mellitus and psychiatric disorders, such as schizophrenia, is apparent and complex. It has been shown that those with severe mental illness have a higher prevalence of metabolic syndrome, which can be contributed to side effects of antipsychotic medications. The most common of these medications are clozapine and olanzapine, which are both second-generation antipsychotics. Most of the other second-generation antipsychotic medications have been reported to be associated as well. First-generation antipsychotics have a lower risk of metabolic syndrome but have a higher risk of extra-pyramidal side effects. The choice of antipsychotic medication is influenced by its efficacy, side effect profile, and a comparison of the risks and benefits regarding the individual patient. Remarkably, there has even been evidence that diabetes was more common in patients diagnosed with schizophrenia before the use of antipsychotic drugs for treatment. This suggests that there is an unknown association between these diseases independent of medication use. Therefore, it is obvious that the interaction between the disease processes of diabetes mellitus and schizophrenia is multifaceted, complex, and largely indistinct.

Case Report: Here we present two different cases, a 51-year-old African American male and a 38-year-old African American male, with past medical histories of schizophrenia and schizoaffective disorder, bipolar type, respectively. In addition, these patients both had an underlying diagnosis of diabetes mellitus type II and were refusing to take medication or insulin for it. In both cases, these patients were not responding to first-generation antipsychotic medications for the treatment of their psychotic symptoms. They were both evaluated for any causal abnormalities that could explain the resistance to treatment and the patients were found to be hyperglycemic. The hyperglycemia was then corrected using forced insulin therapy in both cases. Subsequently, they showed significant response to the same antipsychotics after the hyperglycemia was corrected. In review of the course of treatment of their psychotic symptoms, it is proposed that their hyperglycemic state was preventing them from responding to the antipsychotic medications.

Discussion: Emerging evidence on the crosstalk between insulin and dopamine (DA) signaling pathways provides a potential explanation to this proposed phenomenon. Insulin has been shown to modulate DA activity via the dopamine transporter (DAT), which is responsible for the reuptake of DA that helps to determine the strength and duration of dopaminergic neurotransmission. Activation of insulin receptors triggers the PI3-K/AKT signaling cascade. Once AKT is activated, DAT cell surface expression is promoted leading to enhanced DA clearance. Conversely, in low levels of insulin, there is a reduction in the phosphorylation of AKT with diminished DAT mRNA expression and delayed DA reuptake kinetics.

This is what could have been happening with our patients in their initial weeks of hospitalization. Their hyperglycemia reflected their body's inability to produce an adequate amount of insulin. Without insulin, DAT could not be properly expressed leaving more DA in the synaptic cleft to compete with DA-blocking haloperidol. In fact, one study investigating hypoinsulinemic diabetic rats (induced by chemical destruction of pancreatic β-cells) observed a 2.2 fold decrease in haloperidol potency, demonstrating that disturbed levels of insulin can directly alter the efficacy of an antipsychotic. Furthermore, treatment with haloperidol has been associated with augmented AKT phosphorylation in mice which is believed to compensate for the depressed phosphorylation of AKT implicated in schizophrenia.

In addition to impaired insulin production, insulin resistance is an important component of diabetes and may represent another pathophysiological mechanism in which uncontrolled diabetes mellitus could have a blunted response to antipsychotic treatment. A set of researchers fed rats a high caloric diet for 28 days inducing a hyperinsulinemic, insulin-resistant, obese state. Interestingly, instead of finding increased phosphorylation of AKT as would be expected at elevated levels of insulin, the researchers observed diminished levels of AKT phosphorylation with corresponding reduction in DAT cell surface expression and clearance of DA from synapses. One thought is that prolonged states of increased insulin leading to insulin resistance ultimately results in downregulation of DATs. The findings demonstrate that positive regulation of DAT expression, a mechanism that facilitates the efficacy of antipsychotics, requires insulin and an intact insulin signaling mechanism, both of which are perturbed in diabetes mellitus.

References:

1. Vincenzi, B., MD, & Henderson, D. C., MD. (2018, April 19). Metabolic Syndrome in patients with severe mental illness: Epidemiology, contributing factors, pathogenesis, and clinical implications. Retrieved March 10, 2019.
2. Jibson, M. D., MD, PhD. (2017, May 15). Second-generation antipsychotic medications: Pharmacology, administration, and side effects. Retrieved March 10, 2019.
3. Jibson, M. D., MD, PhD. (2018, May 24). First-generation antipsychotic medications: Pharmacology, administration, and comparative side effects. Retrieved March 10, 2019.
4. Lean, M. E., MA, MB, BCHIR, MD, FRCP, & Pajonk, F., MD. (2003). Patients on Atypical Antipsychotic Drugs: Another high-risk group for type 2 diabetes. *Diabetes Care, 26*(5), 1597-1605. Retrieved March 10, 2019.
5. Alexander P John MB BS, MD, FRANZCP, Radhakrishnan Koloth MB BS, DPM, Milan Dragovic PhD, Stephen C B Lim PhD, Prevalence of metabolic syndrome among Australians with severe mental illness, Medical journal of Australia, Volume 190, Issue 4, February 2009, Pages 176-179.
6. John W Newcomer, MD1, Dan W Haupt, MD2, The Metabolic Effects of Antipsychotic Medications, Can J Psychiatry, Vol 51, No 8, July 2006.
7. Marc De Hearht, Vincent Schreurs, Arc De Hert, Vincent Schreurs, Davy Vancamfort and Rudd Vancampfort. Metabolic syndrome in people with schizophrenia: a review, World Psychiatry. 2009 Feb; 8(1): 15–22.
8. Nash, A. I. (2016). Crosstalk between insulin and dopamine signaling: A basis for the metabolic effects of antipsychotic drugs. J Chem Neuroanat. doi:10.1016/j.jchemneu.2016.07.010
9. Figlewicz, D. P., Brot, M. D., McCall, A. L., & Szot, P. (1996). Diabetes causes differential changes in CNS noradrenergic and dopaminergic neurons in the rat: a molecular study. Brain Res, 736(1-2), 54-60.
10. Patterson, T. A., Brot, M. D., Zavosh, A., Schenk, J. O., Szot, P., & Figlewicz, D. P. (1998). Food deprivation decreases mRNA and activity of the rat dopamine transporter. Neuroendocrinology, 68(1), 11-20.
11. Sevak, R. J., Koek, W., & France, C. P. (2005). Streptozotocin-induced diabetes differentially modifies haloperidol- and gamma-hydroxybutyric acid (GHB)-induced catalepsy. Eur J Pharmacol, 517(1-2), 64-67. doi:10.1016/j.ejphar.2005.05.043
12. Emamian, E. S., Hall, D., Birnbaum, M. J., Karayiorgou, M., & Gogos, J. A. (2004). Convergent evidence for impaired AKT1-GSK3beta signaling in schizophrenia. Nat Genet, 36(2), 131-137. doi:10.1038/ng1296
13. Roh, M. S., Seo, M. S., Kim, Y., Kim, S. H., Jeon, W. J., Ahn, Y. M., . . . Kim, Y. S. (2007). Haloperidol and clozapine differentially regulate signals upstream of glycogen synthase kinase 3 in the rat frontal cortex. Exp Mol Med, 39(3), 353-360. doi:10.1038/emm.2007.39

Abstract 19-1-08

Title: Acute Psychosis, Alopecia, and Partial Edentulism in a Narcoleptic Patient on Prescription Dextroamphetamine/Amphetamine.

Authors: Bradley Harris, MS3. Shanthi Gatla, MD. Praveen Narahari, MD.

Summary: Mixed dextroamphetamine/amphetamine salts are common treatments for Narcoleptic patients. Despite their prevalence, few case studies have been published regarding the over use of these prescription stimulants in the sleep disorder patient population. In this article, we present a case of prescription stimulant abuse in a narcoleptic patient that manifested as psychosis, alopecia and partial edentulism. Narcolepsy is characterized by excessive daytime sleepiness with irresistible sleep attacks, cataplexy, hypnagogic hallucination, and sleep paralysis. The condition is thought to be caused by the loss of hypothalamic hypocretin neurons. Currently, the preferred treatment for narcolepsy is daily stimulants, such as amphetamine and dextroamphetamine.[1] Chemically, prescription stimulants are extremely similar to the illicit drug methamphetamine. Therefore, abusing prescription stimulants may lead to symptoms similar to that of methamphetamine use. With abuse of stimulants on the rise, understanding of this case presentation is vital.[2]

Methamphetamine has been associated with psychosis for many years.[3] While several symptoms overlap with primary disorders, such as schizophrenia, methamphetamine induced psychosis may be clinically differentiated. In addition to psychosis, methamphetamine can cause physical symptoms, such as alopecia and tooth loss. While the mechanism is not fully understood, Seo JW et al. demonstrate that methamphetamine causes significant vasoconstriction through the endothelin receptor.[4] Likely, local vasoconstriction from stimulants leads to a decreased nutritional supply, manifesting as alopecia and edentulism. Given their chemical similarity, it is reasonable to assume dextroamphetamine/ amphetamine works in a similar mechanism of action. In our case report, the patient experienced somatic symptoms for several months before her acute psychotic episode. She had consulted her providers to address the alopecia and partial edentulism. She had several laboratory work ups and received extensive imaging for these issues, but the possibility of her prescription dextroamphetamine/amphetamine causing these symptoms was never considered.

References

1. Akintomide GS, Rickards H. Narcolepsy: a review. Neuropsychiatr Dis Treat. 2011;7:507-518. doi:10.2147/NDT.S23624
2. Weyandt LL, Oster DR, Marraccini ME, et al. Prescription stimulant medication misuse: Where are we and where do we go from here? Exp Clin Psychopharmacol. 2016;24(5):400-414. doi:10.1037/pha0000093
3. Sato M. A lasting vulnerability to psychosis in patients with previous methamphetamine psychosis. Ann N Y Acad Sci. 1992;654:160-170.
4. Seo J-W, Jones SM, Hostetter TA, Iliff JJ, West GA. Methamphetamine induces the release of endothelin. J Neurosci Res. 2016;94(2):170-178. doi:10.1002/jnr.23697

Abstract 19-1-09

Title: Stiff Man Syndrome: The Journey of Finding the Right Diagnosis

Authors: Adrienne Vickers MS-3; Tina Jackson, MD; W. Bogan Brooks, MD

Summary: Stiff-Person Syndrome a rare neuroimmunological disorder was first described in 1956. Since then, research has made marked clinical progress and improvements in understanding the disease, its diagnostic work up and treatment. Cases are usually very complex and presentation varies along the course of the disease making it difficult to recognize. This disorder is characterized by rigidity and muscles spasm of the trunk and proximal extremities with the hallmark of the disease being its association with glutamic acid decarboxylase antibodies. The patients suffer from falls and have been reported as having a 'wooden-man' appearance. These episodes are often triggered by excitability, stress, emotions, sudden movement or noises. One of the main initial presentations of Stiff-Person Syndrome is severe anxiety, which can result in it being misdiagnosed as a psychiatric condition. Diagnosing this disorder often requires input from multiple disciplinary areas in medicine including psychiatry and neurology. Therefore it is important that psychiatrists learn about Stiff-Person Syndrome and place it on their list of differentials. As psychiatrists move away from using benzodiazepines for treating anxiety long term, psychiatrists need to know that benzodiazepines are currently the best medication for treating Stiff Person's Syndrome. Psychiatrists also need to know that a multidisciplinary approach to care for a patient with Stiff Man syndrome leads to better outcomes.

We present a case of a patient who presented with severe panic attacks. Her medical history was not significant except for past diagnosis of hereditary spastic paraparesis and her social history was non-contributory. The patient was diagnosed with Panic Disorder, and aggressively treated for anxiety. When her panic attacks failed to improve, neurology was consulted and the diagnosis of Stiff-Person Syndrome led to better able to treatment for this patient.

References:

1. Ameli, R., et al. "A Neuropsychological Assessment of Phobias in Patients with Stiff Person Syndrome." Neurology, vol. 64, no. 11, June 2005, pp. 1961–1963., doi:10.1212/01.wnl.0000163984.71993.fe.
2. Balint, Bettina, and Hans-Michael Meinck. "Pragmatic Treatment of Stiff Person Spectrum Disorders." Movement Disorders Clinical Practice, vol. 5, no. 4, July 2018, pp. 394–401., doi:10.1002/mdc3.12629.
3. Cervantes, Carmen Elena, et al. "Why It Is Not Always Anxiety: A Tough Diagnosis of Stiff Person Syndrome." Case Reports in Neurological Medicine, vol. 2017, 14 Aug. 2017, pp. 1–3., doi:10.1155/2017/7431092.
4. Grativvol, Ronnyson Susano, et al. "Updates in the Diagnosis and Treatment of Paraneoplastic Neurologic Syndromes." Current Oncology Reports, vol. 20, no. 11, 10 Nov. 2018, doi:10.1007/s11912-018-0721-y.
5. Zdziarski, Przemyslaw. "A Case of Stiff Person Syndrome." Medicine, vol. 94, no. 23, June 2015, doi:10.1097/md.0000000000000954.

Abstract 19-1-10

Title: Lorazepam injection therapy at home for a patient with catatonia

Authors: Abhilasha Pandey, MD; Olga Belotserkovskaya, MD; Charles Nevels, MD

Summary: Catatonia is a state of unresponsiveness to external stimuli in a person who is apparently awake. It is a presentation of many neuropsychiatric conditions. Lorazepam is one of the treatments of choice for catatonia. The use of injectable lorazepam is usually in clinic or hospital settings. In our case we used injectable lorazepam as a home treatment. This resulted in a prolonged remission from symptoms of catatonia and minimized the relapses. The possible reasons for this improvement are discussed.

Case Summary: Patient is a 65 year old white female with several medical conditions, but had no prior psychiatric treatment. Her first psychiatric unit admission was about 4 years ago when patient was stressed by life events and developed agitation, paranoia, combativeness, and hyperreligiosity. On her first admission to psychiatry, the patient was placed on injectable Lorazepam, mood stabilizers, and antipsychotics. Patient showed improvement and was send home with home with these medications. She did well over the next 15 months with continuing outpatient treatment. Her outpatient medications were injectable Risperdone and lorazepam. A new major stressor in patient's life occurred and the patient began to lose weight and became withdrawn. On this admission pt was placed on lorazepam injections, lithium , armodafinil, Vitamin D2, Folate, and injectable Vitamin B12. The patient was discharged with improvement risperidone 6 mg night time, lorazepam 1 mg three times a day , armodafinil 75 mg every morning and lithium 300 mg night time. Just 4 weeks later, the patient was readmitted with the same symptoms. She responded very well to lorazepam 1 mg intramuscular, three times a day. At that time, possibility of ElectroConvulsive Therapy (ECT) was discussed but declined by the patient and her family. The patient was readmitted within 3 weeks with similar symptoms. Her Mini Mental Status Examination (MMSE) was 15/30. A trial of aripiprazole failed. Lorazepam injections were helpful, however when beginning a switch to oral lorazepam the patient began to regress. At this point the patient and her family agreed for her to have a course of electroconvulsive therapy (ECT). Unfortunately ECT was unsuccessful in relieving her symptoms in a meaningful way. The patient's psychiatric condition continued worsen to a point that she required help with all activities of daily livings (ADLs). Her Mini Mental Status Examination (MMSE) was 10/30. Lorazepam was reinstated and the patient's antipsychotic was switched from risperidone to olanzapine. Patient seemed to respond better on olanzapine. All attempts to change lorazepam injection to oral lorazepam led to a worsening of her catatonic symptoms. Finally family agreed to arrange lorazepam injections at home. The patient has continued to respond well to it. In conclusion, though this treatment seems to be unorthodox, placing the patient on lorazepam injections at home, has allowed her a longer period of remission and independence level of functioning.

References:

1. Medscape- overview of catatonia Retrieved from, https://emedicine.medscape.com/article/1154851-overview on 01/31/2018
2. Tandon R1, Heckers S Bustillo J and Barch DM, et al. Catatonia in DSM-5. Schizophr Res. 2013 Oct;150(1):26-30. doi: 10.1016/j.schres.2013.04.034. Epub 2013 Jun 24.
3. Unal A1, Altindag A, Demir B, Aksoy I. The Use of Lorazepam and Electroconvulsive Therapy in the Treatment of Catatonia: Treatment Characteristics and Outcomes in 60 Patients. J ECT. 2017 Jun 20. doi: 10.1097/YCT.0000000000000433. [Epub ahead of print]

4. Pascal Sienaert,1,2,* et al, A Clinical Review of the Treatment of Catatonia Published online 2014 Dec 9. Prepublished online 2014 Oct 3. doi: 10.3389/fpsyt.2014.00181 Front Psychiatry. 2014; 5: 181.PMCID: PMC4260674
5. Mann SC, Caroff SN, Bleier HR, et al. Lethal catatonia. Am J Psych. 1986;143:1371–1381.[PubMed]
6. Singerman B, Raheja R. Malignant catatonia — a continuing reality. Ann Clin Psych. 1994;6:259–266. [PubMed]
7. Ali SF1, Gowda GS2, Jaisoorya TS3, Math SB3 Resurgence of catatonia following tapering or stoppage of lorazepam - A case series and implications. Asian J Psychiatr. 2017 Aug;28:102-105. doi: 10.1016/j.ajp.2017.04.002. Epub 2017 Apr 4. Benzodiazepines and schizophrenia, a review of the literature].[Article in French]
8. Gaillard R1, Ouanas A, Spadone C, Llorca PM, Lôo H, Baylé FJ. Catatonia: a clinician's guide to diagnosis and treatment M Fink, MA Taylor - 2006 Catatonia: Our current understanding of its diagnosis, treatment and pathophysiology
9. Sean A Rasmussen, Michael F Mazurek, and Patricia I Rosebush J ECT. 2010 Dec;26(4):253-8. doi: 10.1097/YCT.0b013e3181fbf96d. Etiopathogenesis of catatonia: generalizations and working hypotheses. Dhossche DM1, Stoppelbein L, Rout UK.

Abstract 19-1-11

Title: Treating Bipolar I Disorder in a Natural Killer Cell Deficiency Patient: A Cautionary Tale

Authors: Christopher Tidwell, MS3, Tina Jackson, MD, and Bogan Brooks, MD

Summary:
Introduction**:** Bipolar I disorder has an international prevalence of nearly 1% of the world's population. In a disorder that elevates one's chances of committing suicide by 30 times that of the general population, it is clear to see the necessity of implementing successful treatment of persons afflicted by this serious mental illness. Current practice guidelines indicate that first-line maintenance therapy should consist of the treatment which resolved the acute manic or depressive episode. What happens when these therapies are given as maintenance to a patient with a major primary immunodeficiency?

Case Presentation: Here, we present a 48-year-old Caucasian female with classical natural killer (NK) cell deficiency type I and psychiatric diagnoses of bipolar I disorder, post-traumatic stress disorder, and panic disorder presenting with an acute manic episode. NK cell deficiency in this patient resulted in severe infections with HPV, CMV, HSV, and VZV. Viral infection resulted in anal, vulvar, and skin cancers requiring multiple rounds of chemotherapy and surgery. She presented with acute mania despite compliance with medications. A multitude of treatments were attempted without either efficacy or tolerability. Finally, lithium was initiated with a resolution of the patient's mania. The question arose: if lithium therapy was continued as guidelines suggest, what would be the long term effects on her NK cell count and function?

Discussion/Conclusions: Knowledge of the effects of psychiatric drugs on the immune system is essential for the practicing psychiatrist. The initial drugs that come to mind are the antipsychotic drugs, which have been well documented to have suppressive effects on the bone marrow. Lithium, being a first-line therapy and mood stabilizer for patients with bipolar disorder, is an important drug to consider in our patient, as it was the only drug that was both effective and tolerated. This report examines the body of literature that evaluates lithium treatment and NK cells. In real human patients with mood disorders, these effects are all challenging to tease out from the inherent effects of depressive mood disorders on NK cells through an aberrant brain-immune axis. Compiling all findings, it appears lithium is likely suppressive of NK cells as a long term therapy but may boost NK cell precursors as a mitogenic agent acutely. The take home point of this case study is not to suggest that lithium or any other successful treatment for bipolar disorder should not be used, but rather to emphasize the importance of considering the off-effects of drugs that may only have significance in patients with rare disease processes.

References:

1. Moreira ALR, et al. Review and meta-analysis of epidemiologic studies of adult bipolar disorder. Journal of Clinical Psychiatry. 2017;78(9), e1259–e1269. https://doi.org/10.4088/JCP.16r11165.
2. Malhi GS, Gessler D, Outhred T. The use of lithium for the treatment of bipolar disorder: Recommendations from clinical practice guidelines. Journal of Affective Disorders. 2017;217, 266–280. https://doi.org/10.1016/j.jad.2017.03.052.
3. Orange JS. Natural killer cell deficiency. Journal of Allergy and Clinical Immunology. 2013;132(3):515-525.
4. Spits H, Di Santo JP. The expanding family of innate lymphoid cells: regulators and effectors of immunity and tissue remodeling. Nature Immunology. 2010;12, 21. Retrieved from https://doi.org/10.1038/ni.1962.

5. Jones J, Yeralan O, Hines G, Maher M, Roberts D, Benson W. Effects of lithium and rubidium on immune responses of rats. Toxicology Letters. 1990;52.
6. Karpiński P, et al. Further evidence for depletion of peripheral blood natural killer cells in patients with schizophrenia: A computational deconvolution study. Schizophrenia Research. 2018;201, 243–248. https://doi.org/10.1016/j.schres.2018.04.026.
7. Karpiński P, et al. Reduced number of peripheral natural killer cells in schizophrenia but not in bipolar disorder. Brain, Behavior, and Immunity. 2016;54, 194–200. https://doi.org/10.1016/j.bbi.2016.02.005.

Abstract 19-1-12

Title: Mistaken Affect – not a laughing matter: A case report pseudobulbar affect misidentified as worsening depression.

Authors: Brad Brooks DO; Praveen Narahari MD

Summary: Pseudobulbar affect (PBA) is a disorder of emotional expression that manifests as involuntary, and often inappropriate, spells of crying or laughter.[1] These spells are characteristically incongruent or disproportionate to a patient's current mood. PBA is known to occur secondary to neurological conditions or insults, including amyotrophic lateral sclerosis (ALS), multiple sclerosis (MS), stroke, and traumatic brain injury (TBI).[2] PBA has been estimated to affect 1.8-7.1 million people in the United States, however, it remains both under-recognized and undertreated as it can be mistaken for depression or other mood disorders.[3] Prevalence of PBA in patients with preexisting mood disorders, as well as the pediatric population in general, remains ill-defined and thus often presents a diagnostic quandary. Here, we report a case of a 14-year-old Caucasian female with a past psychiatric history significant for well-treated depression who presented with acute new onset of symptoms of PBA following a TBI. Her symptoms were initially mistaken for an exacerbation of her major depressive disorder. The patient's principal manifestations were inappropriate and uncontrollable crying spells that were incongruent with her emotional state. The duration of these spells were often less than 60 seconds and occurred at a frequency of one to ten times per day. Occasionally, she would laugh in a manner disproportionate to stimuli in conversation. A Center for Neurologic Study-Lability Scale (CNS-LS) was administered with the patient subsequently scoring an 18; consistent with the clinical suspicion and suggestive of the diagnosis of PBA.[4] Our goal is to remind physicians to be vigilant to other etiologies of crying that potentially have overlapping symptoms.[5] This case highlights the challenges of diagnosing PBA in patients with preexisting depression and displays the importance of doing a comprehensive psychiatric evaluation to ensure both timely identification and treatment.

References:

1. Ariel Miller, Hillel Pratt & Randolph B Schiffer (2011) Pseudobulbar affect: the spectrum of clinical presentations, etiologies and treatments, Expert Review of Neurotherapeutics, 11:7, 1077-1088, DOI: 10.1586/ern.11.68
2. Schiffer R, Pope LE. Review of pseudobulbar affect including a novel and potential therapy. J. Neuropsychiatry Clin. Neurosci. 17(4), 447–454 (20005)
3. Work SS, Colamonico JA, Bradley WG, Kaye RE. Pseudobulbar affect: an under-recognized and under-treated neurological disorder. Adv Ther. 2011;28(7):586–601.
4. Brooks BR, Crumpacker D, Fellus J, Kantor D, Kaye RE. PRISM: a novel research tool to assess the prevalence of pseudobulbar affect symptoms across neurological conditions. *PLoS One*. 2013;8(8):e72232. Published 2013 Aug 21. doi:10.1371/journal.pone.0072232
5. Engelman W, Hammond FM, Malec JF. Diagnosing pseudobulbar affect in traumatic brain injury. *Neuropsychiatr Dis Treat*. 2014;10:1903-10. Published 2014 Oct 7. doi:10.2147/NDT.S63304i:10.2147/NDT.S63304