adolescent patients ($N = 5$) on an inpatient psychiatric unit. Additionally, a cross-sectional self-reported questionnaire examined the awareness and attitudes toward this practice among unit staff ($N = 41$). The patients' attitudes toward clothing restrictions was predominantly negative, noting a lack of self-expression, feeling like a mental health patient, desires to wear ones' personal clothing, and feelings of shame and punishment. Among the staff there was a modest correlation between age, number of years practicing as a health professional, and years practicing in a pediatric setting with feelings of a need for a change in the clothing policy to allow patients to wear their own clothing on admission. Staff age and number of years working at the institution demonstrated a modest correlation between awareness of legal statutes regarding patients' rights to their own clothing. This research found a readiness among staff to adopt a clothing policy that would permit patients to wear their own clothing on admission, which would improve the negative experiences described among the patients in the sample.

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Careful, Women! Is Orgasm Worth the Cost of Your Cerebellum? Flibanserin-Induced Cerebellar Dysfunction

D. Birwatkar[1], S. Kalita[1] and A.R Hirsch[2]

[1]Spartan Health Sciences University, Saint Lucia and [2]Smell and Taste Treatment & Research Foundation, Chicago, IL, USA

Abstract

Introduction. Flibanserin, a serotonin antagonist currently indicated for treatment of female sexual dysfunction disorder, has not heretofore been described to worsen cerebellar function. Such a case is presented.

Methods. A 60-year-old woman, 8 months prior to presentation, had an acute onset of fainting and hitting her head into a wall without loss of consciousness. She could not stand up, had left-sided weakness, and vomiting, with garbled, slow speech and severe headache. Findings in the emergency room showed a left cerebellar parenchymal hemorrhage of 3.2 x 3.1 x 2 cm with the epicenter at the dentate nucleus, extending medially towards midline into the cerebellar vermis, with surrounding perilesional edema extending into the middle cerebellar peduncle. Also, 5.2 cm of the hemorrhage extended from the petrous of the tentorium to the cerebellar vermis. Moreover, a ventral left thalamic hemorrhage with subependymal clot at the foramen of Monroe extended into the dependent portion of lateral ventricles without midline shift. Post one month of physical therapy, speech, walking, and coordination improved but she continued to have delayed speech and trouble getting up, with a wide stance.

Results. Neurologic Examination: Cranial Nerve (CN) Examination: CN XI: Sternocleidomastoid hypertrophy, horizontal titubation. Motor examination: Drift test: L pronator drift with L abductor digiti mini sign. Gait examination: heel walking, dystonic posture of L hand. Tandem gait: unstable, wide based. Cerebellar examination: Both (B) finger-to-nose dysmetria, Left > Right. Slow rapid alternating movements (RAM) L Upper Extremity (UE). Due to absent sexual desire she started 100 mg of flibanserin nightly. Maintaining this for 5 weeks, her coordination markedly worsened with poor balance and a need for a cane to ambulate. She would stumble, with a wider gait, and found climbing stairs challenging. Physical examination displayed worse cerebellar function: prominent horizontal titubation. Finger-to-nose—dysmetria L>R. Decreased RAM, L UE. Markedly positive Holmes Rebound phenomenon, Bilateral UE. Tandem gait: unstable. A week post stopping flibanserin, gait and cerebellar examination returned to baseline.

Discussion. The temporal correlation between the use of flibanserin and transient worsening of cerebellar function strongly suggests that this is the causative agent. Since serotonin is essential in cerebellar function, including its action on the cerebellar cortex and deep cerebellar nuclei, it strongly suggests that its action as a serotonin antagonist is the mechanism whereby flibanserin is causing cerebellar symptoms. In those on flibanserin, investigation to detect the presence of cerebellar dysfunction is warranted. Assessment for the presence of cerebellar dysfunction in those who are on anti-serotonin drugs, such as cyproheptadine and methysergide, may be worthwhile.

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Too Sweet to Eat: Delusional Hypergeusia

S. Kalita[1], D. Birwatkar[1], A. Sundar[2] and A.R Hirsch[3]

[1]Spartan Health Sciences University, Saint Lucia, [2]Aureus University School of Medicine, Aruba and [3]Smell and Taste Treatment and Research Foundation, Chicago, IL, USA

Abstract

Introduction. Delusional hypergeusia has not heretofore been reported.

Methods: Case report. A 62-year-old right-handed woman described a plethora of complaints after exposure to a solvent aroma, including headaches, diffuse weakness, fatigue, hallucinated smells and tastes, burning mouth syndrome, and panic attacks. The apogee of her symptoms was that salty taste was 800% of normal, making food taste disgustingly salty. She was unable to tolerate potato chips, pizza, spaghetti sauce, Coca Cola, root beer, Sprite, 7 Up, and even bottled water. Sugar was also too sweet, 600% of normal. Foods which were unbearably sweet included cookies, sugar, and breakfast cereals. Sour and bitter were normal.

Results. Abnormalities in Neurological Examination: Mental Status Examination: hyperverbal, loud, overly inclusive, irritable with pressured speech; disheveled, racing thoughts, and tangential. Motor Examination: Drift Test: right pronator drift with right abductor digiti mini sign. Gait Examination: heel walking with bilateral decreased arm swing. Reflexes: bilateral quadriceps

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femoris 3+, positive left (L) Hoffman's reflex. Chemosensory Testing: Olfaction: Brief Smell Identification Test: 12 (normosmia). Retronasal Olfactory Testing: Retronasal Smell Index: 1 (Anosmia). Gustatory Testing: Propylthiouracil Disc Taste Test: 10 (normogeusia). Waterless Empirical Taste Test: sweet: 4, sour: 3, salty: 7, bitter: 5, brothy: 0, total: 30 (ageusia to umami, otherwise normogeusia). Neuropsychiatric Testing: Go-No-Go Test: 2/6 (abnormal).

Discussion. Perhaps hypergeusia may not have been true hypergeusia but a misperception of retronasal smell associated hyperosmia with physiologic synesthesia manifested as taste. Peradventure, the perceived hypergeusia, is just one component of a generalized delusional paradigm, where many sensory perceptions are intensified. The perceived delusional hyperosmia may be intensification of the sensory misperception due to an underlying dysgeusia. This may represent a variant of the two-factor hypothesis of delusions whereby a distorted sensory perception is then misrepresented in a delusion. Dysfunction of the right hemisphere, which normally acts to censor the left, allows the delusion to manifest. While two different anatomical abnormalities (one left and one right hemisphere) have been postulated to be the foundation of such delusions, it is distinctly possible that a single lesion of the inferior parietal lobule may be sufficient for both sensory distortions to be produced as well as loss of inhibition of delusional interpretation of distorted sensation of the frontal lobe by the right parietal lobe, yclept the sensorialist hypothesis. In those who present with hypergeusia, search for delusional origin is warranted and in those who present with delusions, query as to perceived hypergeusia may be revealing.

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Number Needed to Treat and Number Needed to Harm From Two Phase 3 Studies of Sublingual Dexmedetomidine for Treating Acute Agitation in Patients With Schizophrenia and Bipolar Disorder

Citrome L[1], Risinger R[2], Rajachandran L[2], Patton E[2] and Robison H[2]

[1]Department of Psychiatry and Behavioral Sciences, New York Medical College, Valhalla, NY, USA and [2]BioXcel Pharmaceuticals, New Haven, CT, USA

Abstract

Background. Episodes of acute agitation can occur in individuals who suffer from schizophrenia or bipolar disorder and these can be a significant challenge for patients and for those who provide care to them. Sublingual dexmedetomidine is a selective alpha2-adrenergic receptor agonist that was recently approved by the US Food and Drug Administration for the treatment of agitation in adults with schizophrenia or bipolar disorder. The sublingual form of dexmedetomidine does not undergo first-pass hepatic metabolism, thus resulting in greater absorption than when ingested. In two Phase 3 studies of adults with schizophrenia or bipolar disorder, sublingual dexmedetomidine significantly reduced acute agitation at 2 hours, as measured by the five-item Positive and Negative Syndrome Scale-Excited Component (PEC). When initially appraising the potential utility of a new medication, number needed to treat (NNT) and needed to harm (NNH) can be helpful to assess the size of the treatment effect and, hence, clinical relevance.

Objective. Calculation of NNT and NNH through post hoc analysis of Phase 3 data.

Methods. Post hoc analysis of data were performed on data from two double-blind, randomized, placebo-controlled studies of sublingual dexmedetomidine in adults with schizophrenia or bipolar disorder experiencing acute agitation. Patients were randomized to a single dose of sublingual dexmedetomidine 180 µg, 120 µg, or placebo. The primary endpoint was mean change from baseline in the PEC total score. A therapeutic response was defined as a ≥40% reduction from baseline in PEC total score at 2 hours. NNT was calculated for PEC response rate for sublingual dexmedetomidine versus placebo. NNH was calculated using the incidence of adverse events for sublingual dexmedetomidine versus placebo. Likelihood to be helped or harmed (LHH) was calculated as the ratio of NNH to NNT.

Results. NNT (95% CI) was 3 (2, 3) for 180 mcg and 3 (3, 4) for 120 ug in patients with schizophrenia and 3 (2, 3) for 180 mcg and 4 (3, 6) for 120 ug in patients with bipolar disorder. NNH was greater than 10 for all AEs except somnolence, where NNH was 7 (5, 10) for all doses pooled from both studies. LLH values were greater than 1 for efficacy versus applicable tolerability outcomes in all cases.

Conclusions. This post hoc analysis demonstrated favorable NNT and NNH values for sublingual dexmedetomidine. In all instances therapeutic response was encountered more frequently than any adverse event. These values compare favorably to similar analyses for other approved agents for the treatment of agitation associated with schizophrenia or bipolar disorder, including intramuscular and inhaled formulations.

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Clinical Determinants, Patterns, and Outcomes of Antipsychotic Medication Prescribing in the Treatment of Schizophrenia and Schizoaffective Disorder: A Naturalistic Cohort Study

Emily Groenendaal, MD[1], Sean Lynch, MD[2], Rhea Dornbush, PhD, MPH[1], Lidia Klepacz, MD[1] and Stephen Ferrando, MD[1]

[1]New York Medical College, Valhalla, NY, USA and [2]Mount Sinai Beth Israel, New York, NY, USA

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