treatment of IGD which currently does not have any official treatment recommendations.
Funding. No Funding

Resolution of COVID-19 Chemosensory Loss Upon COVID-19 Reinfection

Yesung Chi[1] and Alan R Hirsch, MD[2]

[1]Aureus University School of Medicine, Oranjestad, Aruba and [2]Smell & Taste Treatment and Research Foundation, Chicago, IL

Introduction. The pandemic of coronavirus disease 2019 (COVID-19) has been associated with widespread myriad chemosensory dysfunction with smell loss as high as 99% and taste loss in 89% of those studied (Kim 2021, Paderno 2020, Renaud 2020). Other chemosensory problems, occur often in combination, including dysgeusia and hyposmia (3.2%), dysgeusia and anosmia (3.4%), ageusia and hyposmia (3.4%) and, ageusia and anosmia (8.5%) (Giacomelli, 2020). Hyperosmia has also been reported to be precipitated by COVID-19 infection (Di Stadio 2022, Kamali 2021). COVID-19 induced persistent hyposmia with resolution of smell and taste dysfunction with COVID-19 reinfection has not heretofore been described. Such a case is presented.
Methods. Two years prior to the presentation, a 26-year-old right-handed single male developed COVID-19 at which time his sense of smell and taste were totally eliminated. His smell gradually returned to 40-50% of normal whereby he was able to smell everything, but less intensively than he normally did. Furthermore, some odors he experienced in a hyperosmic fashion, such as smoke which was 200% more intense than normal. Prior to COVID-19 exposure, he experienced frequent flavorful eructations which were eliminated after COVID-19 exposure. His taste was initially absent after the COVID-19 exposure, but gradually returned to 60-70% of normal whereby he would taste everything but less intensively. Two years and one month after the initial COVID-19 infection despite being fully vaccinated, he contracted a recurrent COVID-19 infection. Prior to the infection, his smell and taste were 60% of normal. Acutely with the reinfection, his smell and taste ability abruptly dropped to 0% for 16 hours, after which his smell returned to 90% of normal and his taste returned to 100% of normal. During this time period, his other chemosensory problems including dysosmia, dysgeusia, cacogeusia, hyperosmia, and phantosmia all resolved, and his flavorful eructations returned. His sense of smell has remained normal for five months, but his sense of taste has gradually dropped down to 50% of normal.
Results. Abnormalities in Physical Examination: General: Scalloped tongue. Neurological Examination: Cranial Nerve (CN) Examination: CN II: Visual acuity 20/25 OU. CN III, IV, VI: left ptosis. CN V: decreased light touch left V_1 and V_2. CN IX and X: Uvula deviates to the right. Reflexes: Absent bilateral triceps. Neuropsychiatric Testing: Clock Drawing Test: 10/10 (normal). Animal Fluency Test: 26 (normal). Go-No-Go Test: 6/6 (normal). Center for Neurologic Study Lability Scale: 8/10 (normal). Chemosensory Testing: Olfaction: After the first infection prior to the second infection: Alcohol Sniff Test: 3 (Anosmia). Chemosensory Testing: 1 month after the recurrent COVID-19: Alcohol Sniff Test: 9 (hyposmia). Brief Smell Identification Test: 9 (normosmia). Retronasal Olfaction Test: Retronasal Index: 8 (normosmia). Gustation: Phenylthiocarbamide Taste Test: 9 (normogeusia). Waterless Empirical Taste Test: sweet: 6 (normogeusia) sour: 8 (normogeusia) salty: 6 (normogeusia) bitter: 4 (hypogeusia) brothy: 0 (ageusia) total: 37 (normogeusia).
Conclusions. COVID-19 induced smell loss has been observed to worsen after reinfection, improvement has not been described (Jain, *Ear, Nose & Throat Journal*, 2021, Lechien, *Journal of Internal Medicine*, 2021). Possibly the recurrent infection induced hyperosmia which superimposed upon the underlying COVID-19 induced anosmia caused an additive effect, combining together to induce normosmia as opposed to COVID-19 induced hyposmia. This may be due to persistent inflammation of the olfactory bulbs and frontal lobes, inducing excessive neuronal sprouting and associated hyperosmia (Di Stadio, *European Review for Medical and Pharmacological Sciences*, 2022). Alternatively, COVID-19 may have acted on not only the olfactory nerves but rather on a central basis, enhancing neuronal firings in the anterior insula and hippocampus, areas involved with the olfactory integration and which have enhanced gray matter volume in states of hyperosmia (Wabnegger, 2019). Even though COVID-19 vaccination has been noted to worsen the chemosensory function (Konstantinidis, *International Forum of Allergy & Rhinology*, 2021), COVID-19 immunization induced improvement in smell has also been reported (Plaza, *Annals of Neurology*, 2021). It is possible that such an improvement is through infection induced activation of inflammatory immune responses which then acts on the infected olfactory bulbs to reduce pathology. The current case of COVID-19 infection enhancing smell and taste strengthens such an autoimmune explanation. In those with recurrent COVID-19 infection, query and investigation as to presence of improvement of chemosensory dysfunction is warranted.
Funding. No Funding

Power-Up: Dissecting Neurobiological Mechanisms Underlying Internet Gaming Disorder

Chaden Noureddine, MD[1], Yucera Salman, MD[1], William Fletes, MD[1] and James Sherer, MD[2]

[1]Icahn School of Medicine at Mount Sinai, Beth Israel, New York, NY and [2]Jersey Shore University Medical Center, Neptune, NJ

Background. The DSM V-TR places video game addiction, also known as Internet Gaming Disorder (IGD) within the section that suggests the need for additional research. Simultaneously, there has been a remarkable surge in the consumption of video games,

https://doi.org/10.1017/S1092852924002013 Published online by Cambridge University Press

with this medium attracting more funding than ever before. Additionally, as this disorder gains increased attention from the psychiatric world, there is a lack of any formal guidelines on the treatment of IGD, or for the superiority of any specific pharmaceutical treatment. The aim of this project focuses on reviewing the neurobiological mechanisms involved in IGD in order to garner a more robust understanding of the neural pathways involved.

Methods. Google Scholar and PubMed were explored using search terms including "Internet gaming disorder," "neurology," "imaging," "mechanisms," and "comorbid" in various permutations. Thirty-seven articles were included from 100 search results that addressed IGD neural and biological mechanisms, and their potential comorbidity with other mental disorders.

Results. The literature suggests that some of the neural findings in IGD are similar to those found in other addiction disorders, which include the following mechanisms: (i) Activation of brain regions associated with reward, as observed in cue exposure and craving studies. Neurotransmitter system studies further suggest the involvement of dopamine-mediated reward mechanisms. (ii) Decreased activity in areas responsible for impulse control and impaired decision-making. (iii) Reduced functional connectivity in brain networks related to cognitive control, executive function, motivation, and reward. Another study suggested that the severity of IGD and depression symptoms predict each other reciprocally. Neurologically, individuals with IGD exhibited enhanced rsFC between the left amygdala and the right dorsolateral prefrontal cortex, inferior frontal gyrus, and precentral gyrus compared to control participants. The baseline amygdala-frontoparietal connectivity negatively predicted the reduction in depression symptoms following a psychotherapy intervention. Other studies suggest that altered executive control mechanisms in attention deficit hyperactivity disorder (ADHD) would be a predisposition for developing IGD. Furthermore, according to the literature, it was indicated that engaging in Internet game playing was linked to reduced white matter density in brain areas responsible for decision-making, inhibiting behavior, and regulating emotions.

Conclusions. The literature findings regarding IGD's neural and biological pathways, as well as the association of these findings with other disorders such as depressive disorders and ADHD reflect behavioral patterns in individuals with IGD. These mechanisms can be utilized to maximize behavioral and pharmaceutical interventions.

Funding. No Funding

Diagnostic Challenges of Late-Onset Mania versus Dementia with Behavioral Disturbances: A Case Report

Yuxi Zhang, MD[1] and Nina Ballone, MD[2]

[1]Saint Elizabeths Hospital DC DBH, Washington, DC and [2]Sibley Memorial Hospital/Johns Hopkins Medicine, Washington, DC

Introduction. Late-onset mania presents as a diagnostic challenge given the interplay between psychiatry and neurology. Neurocognitive disorders, especially the behavioral subtype of Frontotemporal Dementia (FTD), are among one of the differential diagnoses in patients presenting later in life with manic-like symptoms. Here we discuss a case of new-onset manic symptoms in an elderly patient with no prior history of bipolar disorder and conduct a comprehensive literature review to assist with the diagnostic challenges of late-onset mania and dementia with behavioral disturbances.

Case. Mrs. X is a 67-year-old female with bipolar disorder diagnosed 2 months prior to admission who presented to the hospital after a mechanical fall and witnessed seizure-like activity thought to be due to benzodiazepine withdrawal. She was found to have new atrial fibrillation that was stabilized, and clonazepam was resumed. Psychiatry was consulted to assist with manic behaviors. On initial evaluation, Mrs. X presented as anxious, distractible, tangential with pressured speech, increased psychomotor activity, and paranoid towards her husband. She was treated for a urinary tract infection but otherwise workup was unremarkable. Head imaging demonstrated gray matter volume loss that appeared more prominent in the temporal and frontal lobes. Once transferred to the inpatient psychiatry unit, she presented with elevated mood, excessive jocularity, disinhibition, and poor insight. SLUMS of 18 noted poor attention, processing, and short-term recall. She was started on divalproex sodium titrated to 1500 mg daily (VPA level 108 mcg/mL) and olanzapine 25 mg daily. Observations throughout this time included minimal change in affect, elevated mood, memory deficits and social disinhibition. She did have less aggression, minimal paranoia towards her husband and circumstantial thought process although noted with confabulation.

Methods. We completed a comprehensive literature review utilizing PubMed and Google Scholar. Search terms included combinations of "late-onset", "bipolar disorder", "mania", "neurocognitive disorder", "dementia", "frontotemporal", "behavioral disturbances".

Discussion. Once Mrs. X's initial delirium resolved, she remained with manic-like symptoms. Collateral, brain imaging of reduced gray matter volume in the frontal and temporal lobes, and lack of response to high doses of a mood stabilizer and antipsychotic favor a probable neurocognitive disorder with behavioral disturbances. Literature reviewed helped to narrow down the differential diagnosis for Mrs. X and allowed for a more comprehensive treatment plan.

Conclusion. Diagnostic elucidation of late-onset mania relies on a comprehensive investigation into psychiatric and non-psychiatric etiologies, a detailed collateral history, and a neuropsychiatric lens. Neurocognitive disorders remain an important differential diagnosis.

Funding. No Funding

https://doi.org/10.1017/S1092852924002013 Published online by Cambridge University Press