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Case Report

Neurobiology of Epilepsy: Neurosurgical Management Therapy

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Abstract

The co-occurrence of Borderline Personality Disorder (BPD) and Intellectual Disability (ID) is a sparsely covered area in the literature. This case series looks to describe the common presentations of these two disorders, both commonly presenting with self-harm, impulsivity, and intense anger. Additionally, three treatment courses of individuals with co-occurring ID and BPD will be described, illustrating the commonalities as well as the modifications of BPD treatment for individuals and in adapting ID supports for those with BPD.

Of the 3,028 children, 16% of those without autism or a learning disability had been diagnosed with a psychotic disorder. And, for children who had autism or a learning disability, only 7% of those given antipsychotics had a psychotic disorder.

Looking further at these records, we found that the children with an intellectual disability or autism were more likely to be given an antipsychotic drug. In fact, 2.8% of the children with an intellectual disability had been prescribed antipsychotics, and 75% of these had autism. By contrast, 0.15% of those without an intellectual disability had been prescribed the medication.

Keywords

Borderline personality disorder; Intellectual disability; Co-occurring BPD and ID

Introduction

There is little information concerning the prevalence of individuals with intellectual disability (ID) and co-occurring Borderline Personality Disorder (BPD). BPD is the ‒pervasive pattern of instability of interpersonal relationships, self-images, and affects‖ that can affect 6% of the population in the US. While the prevalence of ID is estimated to be about 1% worldwide, the prevalence of co-occurrence of BPD and ID is not well understood the similarities of some of the presenting symptoms of each of these disorders can cause diagnostic confusion. BPD can present with self-injury as deliberate self-harm, and individuals with ID have higher rates of self-injury than the general population. Symptoms of BPD may be attributed to the individual‘s disability rather that to the separate entity of BPD in what is described as _diagnostic overshadowing‘. In addition to the diagnostic difficulty, some authors advise not diagnosing patients with a stigmatized disorder, i.e., BPD, when they have already been diagnosed with ID.

Cognitive impairment in Parkinson’s disease: Multidisciplinary Symposium Clinical Management of Parkinson’s disease

Abstract

People with Parkinson‘s disease (PD) and their care partners frequently report cognitive decline as one of their greatest concerns. Mild cognitive impairment affects approximately 20–50% of people with PD, and longitudinal studies reveal dementia in up to 80% of PD. Through the Parkinson‘s Disease Foundation Community Choice Research Award Program, the PD community identified maintaining cognitive function as one of their major unmet needs. In response, a working group of experts across multiple disciplines was organized to evaluate the unmet needs, current challenges, and future opportunities related to cognitive impairment in PD.

Specific conference goals included defining the current state in the field and gaps regarding cognitive issues in PD from patient, care partner, and healthcare professional viewpoints; discussing non-pharmacological interventions to help maintain cognitive function; forming recommendations for what people with PD can do at all disease stages to maintain cognitive health; and proposing ideas for how healthcare professionals can approach cognitive changes in PD. This paper summarizes the discussions of the conference, first by addressing what is currently known about cognitive dysfunction in PD and discussing several non-pharmacological interventions that are often suggested to people with PD. Second, based on the conference discussions, we provide considerations for people with PD for maintaining cognitive health and for healthcare professionals and care partners when working with people with PD experiencing cognitive impairment. Furthermore, we highlight key issues and knowledge gaps that need to be addressed in order to advance research in cognition in PD and improve clinical care.

Introduction

Neuropathological studies of patients who died with PDD demonstrate widespread cortical and limbic involvement with neurodegeneration, neuronal loss, and deposition of Lewy bodies and Lewy neurites.Clinical correlations, however, yield conflicting results as to which neuroanatomical areas and neuropathologies are most important in the clinical expression of PD cognitive impairment.Basal ganglia pathology, particularly in its associative (cognitive) areas, may also contribute to cognitive deficits.While PD is an α-synuclein-mediated disease, autopsy studies and cerebrospinal fluid biomarker studies suggest that amyloid pathology contributes to cognitive impairment in PD in some cases.Co-existing synuclein and amyloid pathology may invoke synergistic processes.Cerebrovascular disease contributes to some cases of PD cognitive impairment, with evidence of microvascular ischemia on pathology and white matter hyperintensities on neuroimaging.

PD cognitive impairment also reflects dopaminergic, cholinergic, serotonergic, and noradrenergic neurotransmitter deficiencies. Functional neuroimaging and neuropathological studies measuring neurotransmitters support the roles of dopaminergic and cholinergic deficits in PD cognitive impairment.

The prevalence of dementia in PD increases with age, disease duration, motor severity, postural instability/gait disorder phenotype, baseline cognitive impairment, and presence of other non-motor and neuropsychiatric issues.REM sleep behavior disorder is closely related to PD cognitive impairment,and greater daytime sleepiness has been associated with worse cognition in PD.Social isolation, depression, and medical illness may worsen cognition in general and in PD. Even after accounting for these factors, however, cognitive function varies among individuals. This variable expression implies potential genetic or environmental modifiers. Some genetic causes or risk factors for PD (e.g., LRRK2) are generally not associated with prominent cognitive dysfunction, whereas α-synuclein duplication and triplications, GBA, and MAPT mutations have been linked to cognitive deficits and dementia.The *ApoE4* allele has been associated with memory and semantic fluency in PD and may increase the risk of PDD, though studies are conflicting.Study results have been conflicting regarding the role of polymorphisms in BDNF and COMT genes in PD cognitive impairment.In non-PD populations, co-morbidities such as obesity, diabetes, and hypertension may be associated with cognitive decline. This is also the case for diets high in saturated fat, trans-fat, and refined carbohydrates, or low in berries, green leafy vegetables, nuts, vitamin B12, and folate.[3]At present, data are limited regarding the role of co-morbidities and diet in PD cognitive impairment.Preliminary reports suggest that elevated levels of homocysteine and plasma phospholipids and lower levels of serum uric acid may be associated with worse cognition in PD.Environmental risk factors present compelling opportunities for intervention. However, whether modifying these risk factors would change the progression of PD cognitive impairment is unknown.

Cognitive Change

Many people attribute cognitive changes to ―aging,‖ and a major concern expressed by people with PD and their care partners is whether cognitive deficits are related to aging or to PD. Cognitive changes in people with PD need to be benchmarked against normative data and age-related changes. Cognitive decline without dementia can occur in aging, perhaps because neuropathological processes such as neuronal loss, deposition of amyloid, tau, and α-synuclein, and vascular changes, often found post-mortem, are common as we age.The progression of cognitive decline is a key element in attributing changes to underlying disease-related processes. In general, cognitive changes in ―normal‖ aging should not interfere significantly with everyday activities that require cognitive abilities. If they do, however, this may suggest an abnormal process and signal an increased risk of developing MCI or dementia. Changes in functional abilities and everyday activities due to cognitive decline can be difficult to identify if they are mild. Distinguishing whether problems in everyday activities are due to cognitive or motor problems in PD, or a combination of both, can be challenging, and appropriate measures for determining this are needed.

In ―normal‖ aging, cognitive problems typically involve difficulty with recalling and generating words or names (tip of the tongue phenomenon). Deficits in word or name recall, however, are also common in PD.When objective evidence accompanies subjective cognitive changes without a substantial impact on function, this is defined as MCI, a concept also applied in PD.MCI is a risk factor for dementia in both aging and in PD populations. Community-based studies demonstrate that aging is associated with changes in several cognitive domains, notably speeded measures and recall (aspects of the so-called fluid intelligence), but with relative preservation of others such as vocabulary (crystallized intelligence).Normative ranges for cognitive performance have been defined for older adults and throughout the life span.

The Diagnostic Manual for Intellectual Disabilities-2 (DM-ID-2) describes several limitations in diagnosing individuals with ID and personality disorders including ―[taking] into account personal characteristics in the context of a normal cultural framework‖ . This could mean a boisterous airing of grievances needing to be interpreted in the context of the patient‘s culture. A helpful question could be ―Does the patient‘s family of origin see the noted behaviors as aberrant or unusual?‖ DM-ID-2 also suggests that IDD itself may have features in common with personality disorders including impulsivity and difficulty regulating frustration and emotions, and because many people with ID have a protected upbringing, they may have limited experience with social norms and community skills. The DM-ID-2 also suggests the adaptation of moving the age of diagnosis to 22 rather than the DSM-5‘s 18 years of age.

The criteria, otherwise, should be met with a ―pervasive pattern of instability of interpersonal relationships, self-image, and affects, and marked impulsivity‖ and also including five of the nine diagnostic criteria including fears of abandonment, chaotic relationships, unstable self-image, potentially harmful impulsivity, suicidal threats or self-injury, affective instability, persisting feelings of emptiness, anger dysregulation, and stress-induced paranoia . Interpersonal hypersensitivity, while not explicitly one of the diagnostic criteria, is considered an intrinsic component of BPD. Because this disorder can also be considered significantly heritable, with around 50% of variance explained by genetic factors, families can benefit from knowing the disorder is not an individual‘s ―fault..

Case 1- Too many feelings

Ms. A is a 25-year-old black woman with a history of Mild ID and asthma presented for evaluation to the outpatient psychiatric clinic for self-injury. She had previously been administered the Wechsler Adult Intelligence Scale (WAIS)-IV with a verbal score of 66 and full scale score of 64. Ms. A reports, ―When I get too many feelings, I go crazy. When I get too upset, it‘s the only thing that makes me feel better is hurting myself. I mean, I know it‘s not good for me, and I try to stop, but it just happens.‖

Her team brings her in for increased self-harming. Her mother reports, ―She‘s always seemed so sensitive—like her feelings get hurt even when people don‘t mean it‖. Ms. A reports the last significant episode of self-injury occurred when her boyfriend broke up with her at workshop. She reports, ―He did it on purpose just to make me mad to try to get me fired‖. This demonstrates transient stress-related paranoia and anger dysregulation. When asked if she were more sensitive to interactions with others, she agreed that it felt like it was easy to hurt her feelings. She reports that when her habilitation specialist did not say hi to her first thing in the morning, she ―knew‖ that her habilitation specialist was mad at her. As previously noted, individuals with BPD are sensitive to interpersonal rejection which can precipitate dysphoria and suicidality. When screened for idealization and devaluation, she reported that she tends to love her boyfriends and friends when they first meet until she becomes angry at them for some small or large infraction. She reports that they are then ―dead to her.‖ She reports that she feels that her mood is overly reactive to her environment, the criterion for affective instability, and that she worries about people leaving her despite having had a stable upbringing and reliable home providers. She reports sometimes she ―acts up‖ just because she knows they‘ll leave, which is a common manifestation of the fear of abandonment. When screened for impulsive behaviors, her team reports that while she has not had risky sexual behaviors, she has received reprimands at workshop for ―making out under the stairs‖ with two of her last three boyfriends. The team also notes that she will eat anything that is left out, even to the point of making herself sick. The criterion for identity was not able to be elicited as the concept was likely more abstract than could successfully be explained. She did not report dissociative episodes.

Ms. A was diagnosed with Borderline Personality Disorder in accordance with Good Psychiatric Management of Borderline Personality Disorder, by going through each criterion with the team and with Ms. A She and her team were offered psychoeducation about the diagnosis, typical course of the disease including remission in 85% in 10 years, and that the symptoms are significantly heritable . They were relieved and voiced appreciation for the diagnosis.

Treatment

She began individual sessions 2-4 times a month with her mental health counselor. She was started on low dose lamotrigine, which was titrated slowly to an effective dose of 100 mg/day. At 6 months, Ms. A reported a significant decrease in self-injury and was better able to implement the copings skills that she and her therapist had devised together. At 3 year followup, Ms. A reports affective instability, anger dysregulation, and overeating but reports that she feels much better. Her self-injury was reduced to 2-3 times a year and under unusually stressful circumstances.

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