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Misdiagnosis of Neurobehavioral Disorders Associated with Fetal Alcohol Exposure in Adults

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Abstract

This case report addresses the misdiagnosis of Neurobehavioral Disorder Associated with Prenatal Alcohol Exposure (ND-PAE) in adults. The American Psychiatric Association's (APA's) Diagnostic and Statistical Manual - Fifth Edition (DSM-5) has proposed Fetal Alcohol Spectrum Disorders (FASD) be designated as ND-PAE. ND-PAE is prevalent in some contexts and populations, and is the cause of significant morbidity and mortality.

The authors present a case history and use this common presentation to highlight several important issues. First, although both are caused by fetal alcohol exposure, some argue whether FASD and ND-PAE capture the same clinical disorder. It's the authors understanding that the APA's fetal alcohol work group (that proposed the diagnostic criteria) intended for ND-PAE to encompass FASD. Second, the report illustrates that without a thorough neonatal history (frequently not explored with adults); a developmental, educational, and employment history, ND-PAE will be missed. Such patients often have psychotic symptoms that get misdiagnosed as Schizophrenia; symptoms of sadness and demoralization that get misdiagnosed as Major Depressive Disorder; and symptoms of affect dysregulation that get misdiagnosed as ADHD, Impulse Control, Bipolar, or Anxiety/Panic Disorder. The outcome of such a misdiagnosis ultimately leads to a misunderstanding of the psychopathology, which is clearly organic in nature and not strictly psychological. As a result, the patient is often given inappropriate psychosocial treatment and polypharmacy often occurs. In addition, poor social judgment and intellectual disability may possibly put them at risk for suicide, drug use, victimization, homelessness, unemployment, and incarceration because of perpetration of violence due to poor affect regulation. Due to the lack of awareness of this problem, there has been little research in how to treat such patients, and, to date; an efficacious pharmacologic treatment has not been explicated.

Keywords

Case history, Misdiagnosis, FASD, ND-PAE

Introduction

ND-PAE is a proposed diagnostic category used to describe the sequelae of developmental, behavioral, intellectual, and functional problems seen in people who had prenatal alcohol exposure [1]. ND-PAE is often missed in children as it can be disguised as mood disorders, conduct disorders, ADHD [2], or as any psychiatric condition that presents concurrently. It may even be a passed off as a bad attitude or unwillingness to learn. FASD [3] is reported to be the most common identifiable cause of ADHD [4], and Intellectual Disability in children [5], which makes proper diagnosis critical to the lifelong health and mental health of these patients. Because ND-PAE is a neurodevelopmental disorder that begins in childhood, it is not well known how the disorder evolves and persists into adulthood. Adults with ND-PAE can present with what seems to be Bipolar Disorder, Major Depression, Anxiety Disorder, Panic Disorder, Schizophrenia, and many other disorders. In order properly to identify an adult patient with ND-PAE, a thorough neonatal, developmental, educational, and employment history needs to be obtained and familiarity with the proposed criteria of ND-PAE are essential. Otherwise, the patient with ND-PAE will be treated with various medications that are ill-suited to ameliorate the patient's psychiatric symptoms.

Case History

JP (not the patient's real initials) is a 46-year-old African-American male who presented to the Jackson Park Hospital Family Medicine Center for a psychiatric evaluation following the retirement of his previous psychiatrist. The patient brought in a handwritten list of several medications for various psychiatric illnesses. He reports being previously diagnosed with Bipolar Disorder, Major Depression, and Schizophrenia, for which he was taking alprazolam 2 mg TID, trazodone 100 mg at HS, sertraline 50 mg at AM, benzotropine in 1 mg BID, bupropion 150 mg BID, and risperdone 3 mg at AM. JP's history reveals he has symptoms of short episodes where he sweats, has dyspnea, gets jittery, feels scared, has a racing heartbeat, and is nauseated with such attacks occurring 2-3/week, lasting for 15 minutes to an hour, and dependent on situations he is in that are frustrating to him and which he does not understand. He states that he sometimes hears voices (which began after he reached 30 years old) telling him to "go with them" referring to his childhood companions who passed away traumatically (one hung himself and one died of cancer). He also states that he thinks that people are trying to hurt him and does not like to go outside for this reason - thus he reports that he is "paranoid". JP also reports of past instances where

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he gets down and depressed, especially from feeling ill-equipped to deal with life, and has thoughts of wanting to kill himself. Such feelings have never lasted for two weeks, and, accordingly he did not fit criteria for a Major Depressive Disorder. He reports a couple of hospitalizations for his depression and suicidal thoughts, which is a common happenstance for patients with neurodevelopmental disorders. He thinks he was diagnosed with Bipolar Disorder because of his "mood swings", which last for a couple of hours to a day, ergo this length of time does not meet the criteria for Bipolar Disorder. His diagnosis of Schizophrenia was based on his hearing voices and not being able to adapt to the usual demands of life. This was not a true deterioration in psychosocial functioning as JP's history reveals he was never fully functioning up to par. For these reasons, JP does not meet the criteria for Schizophrenia. Without a history of neonatal status, development, education, or employment, JP's history might be mistaken for a diagnosis of Panic Disorder, Schizophrenia, Major Depressive Disorder, and Bipolar Disorder. Accordingly, further history was required to understand JP's plethora of problems.

Digging deeper into JP's history revealed several other areas of dysfunction. The patient reports being in and out of jail or prison most of his life. He states he is not quite sure how to stay out of prison as being incarcerated gives him certainty he will be taken care of and he will be safe. He also says he has a sense of belonging when he is locked up. He states that adapting to life has not become easier with age and is continually getting harder for him. Now that he is out of prison, he requires the help of a family friend to complete certain daily living tasks such as buying clothes and giving him a place to live. JP does not drink alcohol, but smokes cigarettes. JP states that these areas of dysfunction make him feel inadequate, like "something is wrong with me".

JP also reports having a difficult childhood. His mother was absent for most of JPs life. He shared that shortly after he was born, a White male raped JP's mother who was picking cotton down South and she cut her attacker's throat, killing him. She went to prison as a result, and JP says he missed having a relationship with her. He says he used to feel abandoned by her but now knows the true reason she was not there for him and he accepts it. When asked if his mother drank alcohol while pregnant he said he did not know, but confirmed that she drank socially. He does not know if he was premature or full term, however, JP reported that he has always had a bad memory since childhood and had difficulties learning in school. He says he only made it to the 8th grade before dropping out. JP said he had trouble with spelling and reading, with longer words being exceptionally challenging for him. He spelled world "WRLD" and backwards he spelled "DLRW". When asked if he could subtract 7 from 100 he said, "I will need a pencil and paper for that". Several minutes later, after writing the equation down, he was able to produce the correct answer - 93 (this is 4th grade math - [6]). JP's difficult time growing up was both situational as well as attributed to significant neurodevelopmental and learning problems.

JP appears to have some vestiges of Fetal Alcohol Syndrome (FAS), i.e. epicanthal folds, flat midface/nasal bridge and a thin upper lip. JP's philtrum is not completely flat; however, it is expected for the facial features of FAS to fade over time. The DSM-5 states that prenatal alcohol related dysmorphology can be used as evidence of significant levels of prenatal alcohol exposure [1]. JP's history and physical features of FAS fulfills the proposed criteria in the DSM-5 for Neurobehavioral Disorder Associated with Prenatal Alcohol Exposure (ND-PAE) [1]. He has impaired neurocognitive functioning in several areas such as intellectual impairment, history of learning problems, and poor memory since childhood [1]. He shows impaired self-regulation such as difficulty maintaining attention and mental effort during the interview. JP also has poor impulse control, as evidenced by repeated incarcerations for stealing on impulse. JP shows impairment in adaptive functioning, as evidenced by his difficulty in understanding the world around him and requiring extra help with everyday living. He also has poor affect regulation and does not understand the consequences of his impulsive actions. JP's history shows that his impairment started in childhood, and has caused him clinically significant distress and difficulties in several areas of functioning throughout his life. Confirmation of gestational exposure to alcohol and diagnoses of FAS is not possible, e.g. low birth weight, prematurity, heart murmur, etc., as JP's mother has passed away and he knows little about his birth. Despite this, JP's facial signs, history, and lifelong impairment in functioning beginning in childhood, are consistent with the diagnostic criteria of ND-PAE.

Discussion

The DSM-5 reports that 90% of individuals with histories of prenatal alcohol exposure (ND-PAE) will have co-morbid psychiatric conditions [1]. JP's case shows a 46-year-old male with the clinical signs and symptoms of ND-PAE that was missed in childhood (when neurodevelopmental disorders are usually diagnosed). JP is a classic picture of an adult with ND-PAE who has developed concurrent psychiatric co-morbidities including Panic Disorder/Anxiety, Psychosis not otherwise specified, and Depression not otherwise specified.

It is clear JP was misdiagnosed with Bipolar Disorder, Depression, and Schizophrenia. His "mood swings" are a manifestation of poor affect regulation, which is commonly seen with ND-PAE. However, further questioning revealed the time JP was "manic" was not of sufficient duration to meet the criteria for a manic episode, i.e. it only lasted a few hours and not a full week. JP is also not schizophrenic, as his thought process does not show any signs of loose associations, tangentially, or a formal thought disorder. JP's fears of "someone hurting him" are not a paranoid delusion, but are completely valid for someone who is developmentally disabled and having difficulties understanding the world around him. JP's auditory hallucinations are likely secondary to the impaired brain chemistry from fetal alcohol exposure or perhaps JP cannot distinguish between his inner dialogue and "hearing voices" because of corpus callosum difficulties seen in the brains of patients exposed to fetal alcohol [7]. Considering the corpus callosum is the last brain structure to complete myelination early the third decade, this may also explain the observation that patients with ND-PAE frequently do not develop their psychotic symptoms until around this time of their lives [8]; as well as explaining why it may be misdiagnosed as Bipolar Disorder [9]. His inability to adapt to life was apparently taken as evidence of his having psychosocial deterioration instead of realizing JP has never been at a higher level of functioning, e.g. he has never worked except for a week or two. JP's history and symptoms show that people with prenatal alcohol exposure can have a plethora of problems and are often misdiagnosed. Proper diagnoses can only be accomplished via a thorough psychiatric and childhood history. As ND-PAE is not an official diagnosis in DSM-5, JP is currently and correctly diagnosed with Mixed Developmental Disorder (secondary to a high-suspicion of prenatal alcohol exposure) with Anxiety Disorder Not-Otherwise-Specified, Psychosis Not-Otherwise-Specified, and Depression Not-Otherwise-Specified due to being developmentally- delayed from prenatal alcohol exposure. JP's treatment was fraught with high doses of multiple different medications. It is the author's experience that patients with ND-PAE often find themselves in psychiatric treatment facilities [10,11].

As the diagnosis of Neurobehavioral Disorder Associated with Prenatal Alcohol Exposure is a newly proposed term, it requires further study to bring increased awareness of the disorder. It is likely the prevalence rate of ND-PAE is much higher than FASD, due to a presumably large number of adults like JP, who have had a missed diagnosis in childhood and now have completely incorrect diagnoses. Recognizing ND-PAE requires a thorough history that addresses birth, childhood, education and social histories. It is crucial that clinicians are aware of the signs and symptoms of ND-PAE and be open to reassessing and screening patients for it, especially those with multiple psychiatric diagnoses. Now that there is a proposed criterion for a diagnosis that encompasses the spectrum of disorders caused by fetal alcohol exposure, more cases like JP's can be identified and demystified.

In certain demographic areas such as remote Western Australian communities [12], South Africa [13], and the South Side of Chicago [8], the prevalence of ND-PAE is alarmingly high. In a remote Western Australian community, the rate was found to be 120/1,000 [12]. In South Africa - 135.1-207.5 per 1,000 [13]. At Jackson Park Hospital Family Medicine Clinic, the prevalence of psychiatric patients who meet the diagnostic criteria for ND-PAE was 388/1,000 [8]. Accordingly, JP's case is not alone and various communities [14] may be more plagued than other communities [15]. Bell [16] reassessed research done in 1979 which evaluated children in special education on Chicago's South Side [17] and concluded at least 55% of the 274 children studied probably had ND-PAE. In a 2011 chart audit of the children being served by four University of Illinois school-based clinics run by nurses for children with special needs, [16] estimated 39% had fetal alcohol exposure. From consultation work at Cook County Juvenile Detention Center, it was estimated that two-thirds to three-quarters of the youth had speech and language problems, ADHD, intellectual disabilities, and specific learning disorders [16], and the leading cause of these disorders is FAE [4,5].

Conclusions

The authors present a case history of ND-PAE and highlight that the sequelae of prenatal alcohol exposure is a serious problem that can be seen in adults. The senior author has seen more than 500 patients like JP, and none of them were thought to have neurodevelopmental disorders that developed in childhood, but were all diagnosed in adulthood with adult psychiatric disorders despite not meeting diagnostic criteria. Accordingly, it is the author's experience this common problem is frequently misdiagnosed and mistreated. A careful history of a person with several intellectual and functional impairments with a plethora of comorbid psychiatric conditions can unmask ND-PAE. ND-PAE is still a newly proposed diagnostic term. There is much to be studied and discovered to develop a better treatment plan and support for patients like JP. This case report also illustrates overlap between psychiatry, neurology, pediatrics, and obstetrics (there is some evidence that ND-PAE may be preventable using choline supplements prenatally [18] and postnatally [19]).

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