Neurobehavioral Disorder Associated with Prenatal Alcohol Exposure and Comorbid Psychiatric Illness

Gurtej S. Pannu, MD, PGY-1
Oakland Physicians Medical Center Psychiatry Residency Program

Background
Prenatal alcohol exposure (PAE) is a common cause of intellectual disability worldwide and is the most common preventable cause of mental retardation in the US. Alongside characteristics facial features, there is impairment in various domains, including neurocognitive functioning, self-regulation, and adaptive functioning, as outlined in Neurobehavioral Disorders Associated with Prenatal Alcohol Exposure (ND-PAE) (2). Due to the deleterious effects of alcohol on the fetal brain and subsequently reduced volume, specific deficits in cognition, judgment, impulse control and emotional regulation can be seen. The use of psychopharmacology and various psychosocial interventions can result in improved outcomes in patients who suffer from ND-PAE. (1)

Objective
To discuss the use of specific psychopharmacology and psychotherapy in targeting various clusters of symptomology present in ND-PAE.

Method: Case Report
A 46-year-old African American male with behavioral disturbances and suspected FASD/ND-PAE, intermittent explosive disorder, and unspecified psychosis was admitted to an inpatient psychiatric unit at Pontiac General Hospital after getting into an altercation at his adult foster care home. The Patient presented with poor frustration tolerance and impulsive behavior. Cognitive inflexibility and subsequent poor adaptive functioning lead to increased aggressive tendencies, impaired reality testing and delusional thought content. Emotional dysregulation was also a prominent observation, including frequent outbursts out of proportion to the situation, irritability and mood instability. The patient’s length of stay was 17 days, after which he was discharged to an interim AFC home.

Results
The patient was treated with a second generation antipsychotic (quetiapine), a mood stabilizer (sodium valproate), and an anxiolytic (hydroxyzine). Early in the course of hospitalization, the patient’s impulsive behavior, poor frustration tolerance remained largely unchanged. He continued to present with severe emotional dysregulation and cognitive inflexibility with frequent, unexpected outbursts. Although medication up titration yielded modest improvement, it was limited due to adverse effects such as orthostatic hypotension.

Nonpharmacological interventions were then intensified, leading to further symptom amelioration. The patient responded well to daily group therapy sessions and talk therapy. Supporting staff were recruited to introduce environmental modifications and utilize behavioral management strategies to good effect.

Discussion
Pharmacological Treatment of ND-PAE can be approached by considering distinct symptom clusters, which include hyperarousal, emotional dysregulation, hyperactivity and cognitive inflexibility. The most problematic clusters in this patient were cognitive inflexibility and emotional dysregulation, and as such, they were addressed first.

Cognitive inflexibility and associated poor adaptive functioning arises due to alcohol’s deleterious effects on dorsolateral and orbitofrontal lobe function. (1) Atypical antipsychotics such as quetiapine are a first line treatment modality. Emotional dysregulation, secondary to prenatal exposure of alcohol to the hippocampus, prefrontal cortex, and HPA axis can be treated with mood stabilizers such as sodium valproate. (1)

Nonpharmacological interventions were emphasized to complement pharmacotherapy in order to reach treatment goals. Psychosocial interventions such as functional behavior analysis, environmental modification, and behavioral management led to decreased stress levels and increased adaptive functioning resulting in more meaningful group therapy and psychotherapy sessions. Ultimately, this contributed to quicker achievement of acute inpatient treatment goals.

References