A 7-year-old Boy with ADHD
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A 7-year-old white boy had been diagnosed with attention-deficit/hyperactivity disorder (ADHD), inattentive type, 1 year before. He also had a reading disorder with verbal comprehension impairment. He was brought into the emergency room for agitation and self-injurious behavior while at a day camp. He had barricaded himself with stacked chairs and was banging his head repeatedly into the file cabinet after an altercation with his friends.

After unsuccessful attempts to calm him, and out of fear for the safety of the other children, the day camp counselor called the mother and police. The patient continued to entrap himself, make animal sounds, sing, and repeat single words while the police brought him into the emergency room for psychiatric evaluation.

There was no history of depressive or manic symptoms, no changes in sleep, appetite, or weight, and no history of suicidal or homicidal ideation. The patient had a history of a regulatory sensory processing disorder, which was diagnosed a few months before. He has been enrolled in an individualized education program (IEP) for a few months.

The mother reported a sudden dramatic change in her son’s behavior 1 month after the IEP was initiated. There was sequential development of symptoms not previously seen in the patient, starting with increased aggression and irritability, and he became physically assaultive toward his brother by trying to choke him. He had showed impulsive behavior and was nearly hit by a dump truck after running into the street when his mother refused to buy him ice cream. A few days after that, he exhibited symptoms of social withdrawal and refused to go to school. When in school, he would hide under desks or in closets.

His mother said there were newly developed food preferences, and he began to refuse certain foods based on their texture, such as gravy or mashed potatoes. During a visit with a child neurologist, the patient started rocking back and forth and was banging his head repeatedly against the wall. He displayed rapid, non-rhythmic movements of hands, repeated vocalizations of single words, and choreiform head movements. He had no eye contact. His behavior was temporarily controlled by verbal intervention by the neurologist.

A recommendation was made for psychiatric evaluation and an appointment was made. However, his symptoms then escalated to the point that he had to be brought to the ER because of extreme agitation and aggressive behavior during the day camp visit.

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He was born prematurely at 34 weeks by cesarean section, and...
his birth weight was 5 lb, 14 oz. Postnatally, he was in the neonatal intensive care unit for 1 week and required ventilation for 1 day. Developmental milestones were reached on time, except for speech and language. Between 18 months to 3 years, there was a phase of inflexible and rigid behavior. He also received speech therapy between 3 and 4 years. Between 4 and 6 years, he showed poor eye contact, even with familiar people.

His mother reported that he was getting above-average grades in all subjects, except reading and comprehension, before the worsening of his symptoms. Family history includes a mother with anxiety disorder; a maternal aunt with bipolar I disorder; a paternal grandmother with obsessive-compulsive disorder (OCD); and a maternal cousin with Tourette’s syndrome.

On examination in the emergency room, he was 50.5 inches tall and weighed 65 lb. He was clean and neat in appearance, with appropriate posture, decreased motor activity, slowed speech, poor eye contact, anxious mood, and constricted affect. The patient did not respond to questions regarding any perceptual disturbances; suicidal or homicidal ideations; delusions; obsessions; phobias; or themes. Insight was impaired, as he was blaming his siblings for his behavior, and his judgment was poor, as evidenced by his abnormal behavior at the vacation camp.

The patient was discharged home with his mother, and a crisis appointment was scheduled for 2 days later. At that point, he was evaluated in the outpatient clinic by a child psychiatrist for the crisis appointment. During this visit, he was hiding under a chair in the therapist’s office and required coaxing to be guided out. His eye gaze was persistently diverted to the left, and he had gait problems. On two separate occasions, he nearly ran into a wall to his left in the short distance between the offices. He focused on circumscribed behavior (tossing transformer balls), repeated single words, and had periodic verbal outbursts.

His mother also reported recent deterioration of the patient’s handwriting. Because he suddenly developed worsening of OCD, ADHD, gait ataxia, and the appearance of what looked like symptoms of pervasive developmental disorder (PDD), the psychiatrist asked his mother if he ever had sore throats. His mother reported that he had recurrent upper respiratory tract infections (Streptococcus) between 1 and 3 years, and he also had five episodes of scarlet fever.

The last episode of scarlet fever was 5 weeks earlier, and he was treated with a 10-day course of amoxicillin. (Note: At this crisis visit, he had tonsillar enlargement. An ENT consultation was made, and tonsillectomy was recommended.)

With the possible diagnosis of pediatric autoimmune neuropsychiatric disorder associated with streptococcal infection (PANDAS) and PDD, recommendations to monitor antibody ASO titers on a regular basis were made. The patient was started on risperidone 0.125 mg oral two times a day for aggression. Antibiotics were restarted and continued. The ASO antibody titers obtained were elevated to levels more than 600 IU/mL (normal: 150 IU/mL). This level decreased to 263 IU/mL after the regimen of amoxicillin. The anti-DNAse antibody level was 960 IU/mL (normal range for school-age group: <170 IU/mL).

During the next visit, his father reported a marked improvement in behavior. Risperidone, which had been titrated to 0.25 mg oral two times a day, was then tapered to 0.125 mg oral in the morning and 0.25 mg oral at night, as he was much improved. Risperidone was then soon after discontinued with the plan to follow-up the ASO titers. The ASO titers declined, and he became more interactive and playful with peers and family members.

The patient currently is following up with a child psychiatrist and a specialist in PANDAS. The ASO titers last taken were 194 IU/mL. The anti-DNAse B antibody level reduced to 471 IU/mL. Throughout the duration of care, the patient received approximately four to five courses of antibiotics due to high ASO titers, even in the absence of physical manifestations of streptococcal infection. The patient was prescribed azithromycin 125 mg tablet as a prophylaxis to prevent further infections. The child responded well to the occupational therapy, individualized education, and the psychiatric interventions.
DISCUSSION

PDD refers to a group of neurodevelopmental conditions that involve delayed or impaired communication and social skills, behaviors, and cognitive skills (learning). It is also known as autism spectrum disorder (ASD). Many unpublished reports were gathered from parents stating that their children with PDD-not otherwise specified (NOS) or atypical PDD have had chronic recurring strep throat and/or strep-related ear infections. The parents also noted an increase in stereotypic movements or tics, OCD, aggression, withdrawal, and other autistic-like symptoms during these infections.1 Prophylactic use of antibiotics to prevent recurrence of PANDAS causes Candida infections, which has itself been implicated as a potential etiology in some cases of ASD.2

Swedo et al have identified bacteria-triggered autoimmune response as the cause in some cases of childhood neuropsychiatric illness.3 Kerbeshian et al4 proposed the neuropsychiatric-developmental model, which explains that neuropsychiatric developmental disorder (NPDD) is a group of conditions with a childhood onset and a long-term course, with symptom expression varying over the course of maturation. These symptom variations are reflective of biogenetic elements transacting over time with developmental and epigenetic influences.3 It is proposed that a vulnerable basal ganglia substrate via autoimmune processes and resulting dopaminergic dysfunction could play a central role in the co-occurrence of Tourette’s syndrome and ASD.4

According to the clinical recommendations offered by American Academy of Child and Adolescent Psychiatry (AACAP), the management recommendations of PANDAS include laboratory testing, use of antibiotics, symptomatic management of neuropsychiatric symptoms, and immunomodulatory therapies.5

There is considerable research associating PANDAS with the symptoms of childhood-onset OCD, tic disorder, and ADHD. However, there are limited studies showing an association between the symptoms of PDD (particularly the subtypes of autism and Asperger’s syndrome) and PANDAS.6 The objective of this case report is to highlight the above-mentioned association so that the level of awareness is increased and appropriate measures are taken early during the course of symptoms.

CONCLUSION

Children who suffer from streptococcal infection may present with behavioral changes consistent with PDD disorder related to PANDAS beginning 4 to 6 weeks after the resolution of the acute physical symptoms. Careful monitoring and screening by pediatricians is required and immediate assistance of a psychiatrist should be sought if there are any behavioral changes.

REFERENCES

2. Waltz M. Pervasive Developmental Disorders: Diagnosis, Options, and Answers. Arlington, TX; Future Horizons: 2003;90.