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Multidisciplinary Management of Adolescent Presenting with Severe Pediatric Acute-Onset Neuropsychiatric Syndrome (PANS)

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Abstract

Purpose: Pediatric Acute-onset Neuropsychiatric Syndrome (PANS) is characterized by a sudden onset of obsessive-compulsive behaviors and/or restricted food intake, accompanied by other neuropsychiatric symptoms. The condition remains underrecognized due to its broad presentation and lack of standardized diagnostic tools. We present a severe case of PANS in an adolescent male following a suspected post-infectious immune response. **Observations:** A 16-year-old previously healthy Hispanic male developed abrupt-onset neuropsychiatric symptoms, including catatonia, obsessive-compulsive behaviors, cognitive slowing, expressive aphasia-like symptoms, regressive behavior, and severe food restriction, after a flu-like illness. Initial infectious testing was positive for Influenza B and Mycoplasma pneumoniae. Neurologic workup, including MRI, EEG, and CSF analysis, was unremarkable. Despite extensive testing, no alternative diagnosis was confirmed. Clinical presentation met diagnostic criteria for PANS. Multidisciplinary care included psychiatric treatment, gastrostomy tube placement for nutritional support, and escalating immunomodulatory therapies, including high-dose IVIG, corticosteroids, and plasmapheresis. Improvement in motor function, OCD symptoms, and nutritional status followed plasmapheresis therapy. **Conclusions and Importance:** This case underscores the diagnostic complexity and therapeutic challenges of PANS, particularly in severe and functionally impairing presentations. Early recognition, exclusion of mimicking conditions, and coordinated interdisciplinary management are essential. Immunomodulatory therapy may offer benefit in refractory cases, though evidence remains limited.

This report highlights the urgent need for further research to define diagnostic criteria, biomarkers, and standardized treatment protocols for PANS.

Keywords

Pediatric Acute-Onset Neuropsychiatric Syndrome (PANS), Obsessive-Compulsive Disorder (OCD), Restricted Food Intake, Post-Infectious Neuroinflammation, Neuropsychiatric Symptoms, Plasmapheresis

1. Introduction

Pediatric acute-onset neuropsychiatric syndrome, or PANS, is a clinical syndrome defined by an abrupt onset of obsessive-compulsive disorder (OCD) and/or severely restricted food intake. These symptoms are accompanied by at least two additional severe neuropsychiatric symptoms: anxiety, depression, liability, irritability, aggression, oppositional behaviors, developmental regression, sensory (hallucinations) or motor (tics or dysphagia) abnormalities, and/or somatic symptoms (sleep disturbances or enuresis). These symptoms are not better explained by a known neurological or psychiatric disorder [1]. Due to PANS's rarity and the lack of large-scale studies, there are no estimated prevalences and incidences for PANS. In 2025, the American Academy of Pediatrics (AAP) explicitly stated that prevalence/incidence remains undetermined [2].

This syndrome has varied and misunderstood causes. It seems to involve a mix of factors including the immune system, infections, and genetics. PANS often appears after an infection, but these may vary. While strep throat (group A strep), other bacteria, and viruses have all been linked to PANS, many children develop symptoms without any obvious illness beforehand. One of the main theories is that PANS is a post-infectious immune reaction dysregulation. Through molecular mimicry, parts of the brain, especially the basal ganglia, are targeted. This can cause neuroinflammation and lead to sudden psychiatric symptoms. Some recent research supports this, showing that certain antibodies in children with PANS bind to specific brain cells, suggesting an immune-related cause, at least for some children. Neuroimaging and animal studies have demonstrated basal ganglia inflammation in its pathophysiology. Serum IgG from PANS patients has been shown to selectively bind to striatal cholinergic interneurons, supporting a neuroimmune pathogenesis. Other factors such as genetic susceptibility and non-infectious triggers (psychological stress and metabolic disturbances) may also play a role in its development [3]-[8].

PANS is diagnosed clinically, based on the abrupt onset of symptoms previously described. Currently, there are no laboratory, imaging, or disease-specific biomarkers. Diagnostic workup may include supportive tests, such as testing for group A strep, but it is mainly diagnosed through its clinical pattern (sudden onset and symptom combination). PANS remains an exclusionary diagnosis [3]-[6].

There are several important differential diagnoses that have to be ruled out before considering PANS. The first group of conditions is primary psychiatric disorders. We have to consider in our differential: Obsessive-compulsive Disorder (OCD), Tourette Syndrome or Chronic Tic Syndrome, Anorexia Nervosa (or other eating disorders), and Major Depressive Disorder with behavioral changes. The second group are neuropsychiatric conditions such as: Sydenham Chorea, functional (psychogenic) disorders, Anti-NMDA Receptor Encephalitis, other viral encephalitis, and Wilson Disease [9]-[14].

The outlook for children with Pediatric Acute-onset Neuropsychiatric Syndrome (PANS) varies widely. Some recover fully, while others experience persistent or relapsing symptoms. According to the American Academy of Pediatrics, many children show some improvement over time, but a significant number, especially those with other medical or developmental conditions, or those who are diagnosed late, go on to develop chronic or recurring neuropsychiatric issues. Possible complications include ongoing obsessive-compulsive behaviors, extreme food restrictions that can lead to malnutrition, chronic anxiety, mood swings, tics, cognitive decline, and difficulties at school. Long term, children may face social isolation, falling behind academically, and increased strain on family life. In the most severe or treatment-resistant cases, symptoms may progress to debilitating conditions such as treatment-resistant OCD, depression, or even suicidality. Relapses are common and are often brought on by infections or stressful life events. These episodes tend to worsen the persistence of symptoms and lead to more significant functional impairment. Children with immune system deficiencies, frequent infections, or neurodevelopmental disorders are at higher risk for more severe and long-lasting illness [8] [15]-[17].

There are currently no established guidelines for treating PANS. The American Academy of Pediatrics, in 2025 published a summary of current expert opinion on managing this syndrome. The primary treatment is focused on its psychiatric manifestations, especially for OCD symptoms and anxiety, which are the most common. SSRIs are the preferred medication, fluoxetine and sertraline usually being the preferred ones for children. Cognitive behavioral therapy (CBT) is also part of the primary response. Combination therapy is considered most effective for OCD and anxiety. If a specific infection is found, such as strep, a course of amoxicillin is recommended. Using antibiotics routinely without a confirmed infection is not recommended. Long-term or prophylactic antibiotic use is also not supported. Anti-inflammatory and immune-based treatments are also used in more moderate to severe cases of PANS. Literature is more limited for this subject. Mild to moderate PANS flares can be treated with NSAIDS or a short course of oral steroids. For more severe or resistant cases, IV steroids and intravenous immunoglobulin have been used. These treatments are not routinely recommended due to insufficient evidence of efficacy and potential for significant adverse effects. Restricted food intake is another crucial aspect of the illness that must be swiftly addressed. It often presents abruptly, characterized by avoidant/restrictive food

intake features. In PANS, food restriction is most commonly driven by obsessive-compulsive fears (contamination, choking, or vomiting). It requires urgent evaluation of vital signs, weight, height, and growth trajectory, as well as laboratory assessment of electrolytes, and an electrocardiogram (EKG) to screen for cardiac complications of malnutrition. In cases of severe restriction, medical stabilization may require nasogastric feeding tube placement and intravenous fluids to address acute nutritional and hydration needs, as well as correction of electrolyte abnormalities [1] [18]-[26]. The role of a multi-disciplinary approach for these cases cannot be overstated. Best practice recommendations advise a team that includes a pediatrician, psychiatry, infectious diseases, neurology, rheumatology, and immunology to advise and decide on such a broad differential diagnosis and multifactorial pathophysiology of PANS.

2. Case Report

2.1. Patient Demographics and Medical History

The patient is a 16-year-old Hispanic male residing in Ponce, Puerto Rico, living with his parents and siblings in a home environment with multiple pets. Past medical history was notable only for ADHD and a childhood episode of scarlet fever. He was previously healthy, with normal developmental milestones, no prior severe psychiatric or systemic illnesses, and was homeschooled with good academic progress. Family history was notable for bipolar disorder in a brother, autoimmune conditions in maternal relatives, and Alzheimer's Disease on the paternal side, but no known hereditary neurological or autoimmune disorders were identified.

2.2. Clinical Presentation

Initially, the patient developed flu-like symptoms including fever, nasal congestion, myalgias, and malaise while on vacations in Europe. Soon after, his parents noted a gradual onset of motor slowing, manifesting as bradykinesia, affecting fine and gross motor skills. He required assistance with tasks such as tying his shoes, dressing, ambulating, and feeding. Speech fluency decreased, with reduced volume and low tone of voice, though articulation remained intact. He demonstrated dysmetria and tremors, with fluctuating motor retardation affecting gait and coordination, sometimes requiring assistance for ambulation and feeding.

The patient experienced intermittent prolonged episodes characterized by a fixed, vacant gaze and apparent unresponsiveness. During these trance-like states, his upper extremities were internally rotated at the elbows and wrists with clenched fists which persisted for hours multiple times a day. These episodes resembled features of catatonia, with psychomotor retardation and rigidity, though no mutism was observed. Parents described these as "daydreaming" where the patient was bombarded with intrusive thoughts and fantasies that detached him from reality. He presented with compulsions including fixing his gaze on specific floor tiles for extended durations, driven by intense fear that failure to do so would

result in harm. He performed ritualistic repetitive movements such as finger and foot tapping, hand wringing, and other stereotypies. Intrusive, distressing obsessive thoughts created a "loop" of persistent ruminations. The patient was aware of these thoughts, describing them with insight but felt unable to control them. Anxiety was prominent, with frequent panic attacks manifesting as chest pressure, tearfulness, and overwhelming fear. He reported somatic symptoms including dizziness and an intense sensation of coldness when attempting to sleep, accompanied by visual disturbances (flashing colored lights), and auditory phenomena perceived as multiple "voices", which exacerbated his distress and led to fear of sleep. Despite these symptoms, he denied true auditory or visual hallucinations. He also exhibited regressive behaviors such as clinginess, insistence on remaining in his parents' laps, and a need for continuous reassurance.

Cognitive symptoms included decreased concentration and slowed processing, though he remained oriented to person, place, and time. There were notable expressive aphasia-like symptoms characterized by decreased fluency and low volume, while comprehension and articulation were preserved. The patient also experienced intermittent urinary incontinence. Significant weight loss (approximately 30 pounds over a short period of time) was attributed to decreased oral intake secondary to motor and psychiatric symptoms. His BMI measurement was 13.2, indicative of severe malnutrition. Despite poor oral intake, appetite was reportedly preserved. Intermittent low-grade fevers (38.1 - 38.7˚C) and persistent headaches refractory to over-the-counter analgesics were reported. No seizures or focal neurological deficits were observed.

2.3. Hospital Course and Diagnostic Workup

The patient was initially admitted to Hospital La Concepcion, San German, where he tested positive for Influenza B and Mycoplasma pneumoniae and was treated with Decadron. Due to suspected encephalitis, he was transferred to Hospital Pediátrico Universitario (HOPU) for further evaluation. A comprehensive workup included:

Neuroimaging: Brain MRI with and without contrast showed no acute abnormalities, no evidence of encephalitis, ischemia, or structural lesions. Electroencephalogram (EEG): Prolonged awake and drowsy EEG revealed normal background activity without epileptiform discharges. Cerebrospinal Fluid (CSF) Analysis: Mild pleocytosis was noted but was likely secondary to IVIG treatment rather than active infection. Laboratory Tests: Complete blood count and metabolic panel were unremarkable except for mild anemia and electrolyte abnormalities (hypokalemia, hypophosphatemia, and hypocalcemia). Infectious workup including influenza and COVID-19 PCR was negative during admission. Autoimmune Panels: Pending anti-NMDA receptor antibodies and oligoclonal bands. Psychiatric Assessment: Confirmed symptoms of OCD, anxiety, catatonia, and cognitive disturbances without frank psychosis or hallucinations. He scored a 54 out of 100 in the Pediatric Acute Neuropsychiatric Symptom Scale, denoting a categorization

of "extreme" symptomatology (Table 1).

On assessment, the patient was alert and oriented but displayed a flat affect and psychomotor slowing. Speech was soft and hypophonic with preserved coherence. Insight was limited; he recognized the irrational nature of his compulsions but felt compelled to perform them. Mood was anxious and at times dysphoric. Thought processes were circumstantial with notable perseveration of obsessive themes. No delusions or formal thought disorder were evident. No suicidal or homicidal ideation was present. The patient exhibited significant distress related to his symptoms and fear of losing control.

Table 1. Pediatric Acute-onset Neuropsychiatric Symptom Scale (PANSS) score reflecting symptom severity during peak presentation. The total score of 54/100 indicates an extreme severity classification, with prominent obsessive-compulsive (18/25) and anxiety symptoms (16/75). This clinician-rated scale is used to quantify the burden of neuropsychiatric symptoms in PANS, supporting diagnostic clarity and monitoring response to treatment.

Pediatric Acute Neuropsychiatric Symptom Scale Total Score (O-100)	
Obsessive-compulsive Symptoms (O-25)	18
Associated Neuropsychiatric Symptoms (0-75):	36
1. Anxiety Symptoms	16
2. Mood and/or depression	3
3. Irritability or aggressive behavior	0
4. Cognitive/Learning, confusion	5
5. Behavioral regression	4
6. Sensory symptoms	0
7. Hallucinations	0
8. Motor Symptoms	7
9. Urinary symptoms	0
10. Sleep disturbance, fatigue	1
11. Dilated pupils	0
Total	54
Severity	Extreme

2.4. Interdisciplinary Management

Given the complex clinical picture, an interdisciplinary team was involved. Pediatrics: Coordinated consults, ordered and interpreted tests, helped manage anti-inflammatory/immunomodulatory therapies, monitored nutritional and hydration status, provided psychosocial support, educated patient/family, and conducted discharge planning. Neurology: Initiated immunomodulatory therapy with high-dose intravenous immunoglobulin (IVIG) for 5 days followed by monthly maintenance infusions. Steroid pulse therapy was administered with a planned taper. Although some stabilization was achieved, several debilitating neuropsychiatric symptoms remained refractory. These included persistent catatonia-like episodes, severe obsessive-compulsive behaviors, and ongoing anxiety. Additionally, feeding difficulties persisted and his malnutrition status kept deteriorating. Given this constellation of treatment-resistant symptoms and the severity of functional impairment, escalation to plasmapheresis therapy was employed. Psy-

chiatry: Management included Sertraline (increased from 50 mg to 75 mg daily) for OCD and affective symptoms, Olanzapine (2.5 mg five times daily) for agitation and possible psychotic symptoms, Trazodone for sleep, and antihistamines (Vistaril) as adjunctive therapy for anxiety. Benzodiazepines such as lorazepam and clonazepam were trialed but discontinued due to worsening psychiatric symptoms (possible rebound effect). Gastroenterology and Nutrition: Due to severe weight loss and BMI at the 5th percentile (BMI 13.2), nutritional assessment led to percutaneous endoscopic gastrostomy (PEG) placement for enteral feeding. Physical Medicine and Rehabilitation: Consulted for functional rehabilitation recommendations focusing on motor retardation and dysmetria. Palliative Care: Provided family support and coping strategies for chronic illness management.

2.5. Hospital Course and Response to Treatment

Following plasmapheresis, the patient demonstrated gradual improvement in motor function, especially motor retardation, oral intake, anxiety, and OCD symptoms. The patient tolerated bolus and continuous enteral feeding well and gained weight post-PEG placement. Despite persistent anxiety and OCD, his ability to perform ADLs improved. Repeat neurological exams showed no focal deficits. Psychiatric symptoms were managed with optimized pharmacotherapy and outpatient cognitive behavioral therapy (CBT) referral. The patient remained afebrile during most of the hospitalization, with no evidence of active infection. Hemodynamic and respiratory status remained stable throughout. Lab abnormalities including hypofibrinogenemia and leukocytosis were managed conservatively.

2.6. Discharge Plan and Follow-Up

The patient was discharged home after a 9-day hospitalization. He was prescribed a Prednisone taper down therapy, Sertraline 75 mg daily, Olanzapine 2.5 mg five times daily, and Trazodone 50 mg nightly. Monthly IVIG infusions were scheduled. He would continue PEG feeding with supplemental oral intake with close outpatient follow-up with neurology, psychiatry, nutrition, primary pediatric care, and physical therapy/occupational therapy. Parents were educated on symptom monitoring and indications for emergency care, including exacerbation of neuropsychiatric symptoms or signs of infection. Lastly, a genetic evaluation with chromosomal microarray was planned to explore possible underlying genetic susceptibilities.

3. Discussion

Pediatric Acute-onset Neuropsychiatric Syndrome remains a challenging and evolving diagnosis, marked by its sudden onset, broad symptom spectrum, and the absence of disease-specific biomarkers. In this case, the abrupt emergence of obsessive-compulsive behaviors and severe eating restrictions paired with a range of neurological, behavioral, and somatic symptoms highlighted the diagnostic complexity that defines PANS. The variability in symptomatology and the overlap

with other medical and psychiatric conditions make early recognition particularly difficult and demand a comprehensive, multidisciplinary approach. For this patient, collaboration between general pediatricians, pediatric neurologists, child psychiatrists, gastroenterologists, and nutrition specialists were essential in guiding both the diagnostic workup and treatment plan. Given the lack of standardized diagnostic criteria and the continuing absence of reliable laboratory tests, the diagnosis of PANS remains clinical and exclusionary. Ruling out other potential causes such as autoimmune encephalitis, Sydenham Chorea, and primary psychiatric disorders was a critical first step, requiring careful coordination across specialties.

Despite increasing clinical recognition, PANS remains poorly understood. The current literature is limited by a scarcity of randomized controlled trials, and there is still significant uncertainty regarding optimal treatment protocols. As such, management is often symptom-based and must be individualized according to the patient's presentation and disease trajectory. In this case, ongoing clinical flexibility and close monitoring over time were critical to adjust interventions based on evolving needs. Initial treatment aligned with consensus recommendations, prioritizing psychiatric care including cognitive behavioral therapy (CBT) and the cautious use of psychotropic medications. As no active infectious trigger was identified, antibiotics were not administered. However, due to the severity and refractory nature of the patient's symptoms, a more aggressive immunomodulatory approach was eventually undertaken. The use of corticosteroids, intravenous immunoglobulin (IVIG), and plasmapheresis was decided within the context of a multidisciplinary team, weighing the potential risks and benefits in real time. Many unanswered questions remain particularly regarding the role of infection in PANS pathogenesis, the ideal timing and duration of immunomodulatory therapies, the development of validated diagnostic tools, and its genetics. The patient's family history of autoimmune conditions, particularly on the maternal side, suggests a possible inherited tendency toward immune system dysregulation. Although no known genetic disorders were identified, this background raises the question of whether genetic factors may have contributed to the patient's vulnerability. For this reason, genetic testing with a chromosomal microarray was planned to look for any underlying genetic changes that might help explain his presentation. As research on PANS continues to evolve, it is becoming more evident that genetics may play a role in how some children develop these symptoms following infections or immune system activation.

Additionally, the impact of PANS extends beyond the clinical realm. This patient's discharge and follow-up care were significantly complicated by challenges in accessing specialized treatment and secure housing. As is often the case with complex medical conditions, the illness placed a profound strain on the family, who were forced to navigate a multitude of challenges across several domains. Logistically, they had to coordinate care among multiple specialists, each bringing their own perspective, medical terminology, and clinical priorities. Financially,

the prolonged hospitalization, ongoing therapies, and necessary lifestyle adaptations, such as dietary modifications and home safety adjustments, created a significant burden. Emotionally, the family endured the distress of witnessing their son experience severe neuropsychiatric symptoms and undergo intensive interventions, including gastrostomy tube placement and plasmapheresis. The treatment journey was not only medically demanding but also emotionally and practically overwhelming.

This case reinforces the importance of early multidisciplinary involvement, flexible and patient-specific care plans, and the recognition of PANS as not only a medical challenge but a psychosocial one as well. Moving forward, efforts must prioritize improved access to care, clinician education, and the development of research infrastructures capable of supporting high-quality clinical trials. Until clearer answers emerge, clinicians must continue to work across specialties, listen closely to families, and tailor treatments to the unique and often fluctuating needs of each child affected by this complex syndrome.

4. Conclusion

We present a case of a 16-year-old male with acute-onset neuropsychiatric symptoms including catatonia, severe obsessive-compulsive behaviors, and significant functional decline ultimately diagnosed as Pediatric Acute-onset Neuropsychiatric Syndrome (PANS) following a recent infectious illness. Despite extensive workup, including negative autoimmune and infectious panels, the patient's condition necessitated a multidisciplinary approach and escalated immunomodulatory treatment with corticosteroids, IVIG, and plasmapheresis. His partial clinical response and symptom improvement, especially following plasmapheresis, underscore the potential role of immune-based therapies in severe, refractory PANS presentations. This case highlights the importance of early recognition of PANS, comprehensive diagnostic exclusion of mimicking conditions such as autoimmune encephalitis, and the need for individualized treatment strategies guided by a multidisciplinary team. Given the current gaps in standardized diagnostics and evidence-based treatment protocols, clinicians must remain vigilant and flexible in managing such cases. As awareness grows, there is a critical need for further research, particularly randomized controlled trials, to better understand the pathophysiology of PANS and to optimize care for affected children and adolescents.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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