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A Review Based on the Pandas /Pans

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ABSTRACT:
Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal infections (PANDAS) is a proposed clinical entity first described in 1998, following observations that some children with Sydenham's chorea A condition that arises as a result of rheumatic fever.exhibited obsessive-compulsive symptoms, tics, emotional instability, and anxiety. Researchers identified a group of children with similar presentations and proposed five diagnostic criteria: presence of OCD or a tic disorder, prepubertal symptom onset, an abrupt and episodic course, a temporal relationship with group A beta-hemolytic streptococcal (GABHS) infections, and associated neurological signs such as choreiform movements or motor hyperactivity. PANDAS typically presents in children aged 3 to puberty and is marked by sudden onset of neuropsychiatric symptoms, often accompanied by personality changes, cognitive dysfunction, sensory sensitivities, and behavioral regression. The proposed pathophysiology involves molecular mimicry, where antibodies produced in response to GABHS infection mistakenly target neuronal tissues, particularly within the basal ganglia, leading to inflammation and symptom manifestation. This mechanism parallels that of Sydenham's chorea, suggesting a shared autoimmune basis. Diagnosis remains clinical, as there are currently no definitive biomarkers to confirm PANDAS, and symptoms can significantly overlap with other pediatric psychiatric or neurological disorders. Treatment strategies typically include antibiotics to address underlying streptococcal infections, cognitive-behavioral therapy (CBT), selective serotonin reuptake inhibitors (SSRIs), and, in severe or refractory cases, immunomodulatory therapies such as intravenous immunoglobulin (IVIG) or plasmapheresis. Despite ongoing clinical use, the PANDAS diagnosis remains controversial due to inconsistent immunologic and epidemiologic findings. Further research is needed to clarify its etiology, validate diagnostic tools, and develop evidence-based treatment protocols.

Keywords: PANDAS, GABHS, molecular mimicry, basal ganglia, Neuroimaging , pediatric autoimmune disorders, Sydenham's chorea, PANS , MRI scan, treatment , etc.

I. INTRODUCTION

The concept of PANDAS emerged after noticing that some individuals with Sydenham's chorea (from acute rheumatic fever) showed anxiety, emotional instability, OCD symptoms, and tics. In 1998, researchers from the National Institute of Mental Health described 50 such patients and proposed PANDAS as a distinct condition with five specific diagnostic criteria.

1. The occurrence of either obsessive-compulsive disorder (OCD) or a
2. Onset of symptoms during the prepubertal period (from around age 3 until the start of puberty).[2]
3. An episodic pattern, marked by sudden, severe onset and noticeable symptom flare-ups.
4. A time-related connection between group A beta-hemolytic streptococcal (GABHS) infections and the onset or worsening of symptoms.Association with neurological signs, such as choreiform movements, increased motor activity, or tics.[3]

Personality shifts, cognitive issues, motor problems, heightened sensory responses, behavioral regression, and sometimes psychotic symptoms are commonly reported . PANDAS syndrome is thought to be linked to group A beta-hemolytic streptococcal (GABHS) infections through molecular mimicry, where antibodies against streptococcal proteins mistakenly attack brain proteins, especially in the basal ganglia. PANDAS shares immune- related characteristics with Sydenham's chorea (SC), an established autoimmune condition triggered by infection and

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closely associated with OCD. However, unlike SC, the diagnosis of PANDAS remains controversial due to inconsistent immunologic and epidemiologic evidence and the absence of distinct clinical features or biomarkers that clearly differentiate it from childhood-onset OCD or tic disorders[5].

II. EPIDEMIOLOGY OF PANDAS:

Accurate epidemiological data on PANDAS are limited. The difficulty in understanding and diagnosing the condition stems mainly from its clinical variability and challenges in proving a causal link. Both GAS infections and OCD/tic disorders are common in children, but while PANDAS is generally considered low-risk, its exact prevalence and incidence remain unclear[6,7,8,9] Early reports suggest the condition is more common in males, with a male-to-female ratio ranging from 2.6 to 4.7 to 1. The median age of symptom onset is about 6.3 years for children with tics and around 7.4 years for those with OCD. Recent studies also support a male predominance[10].

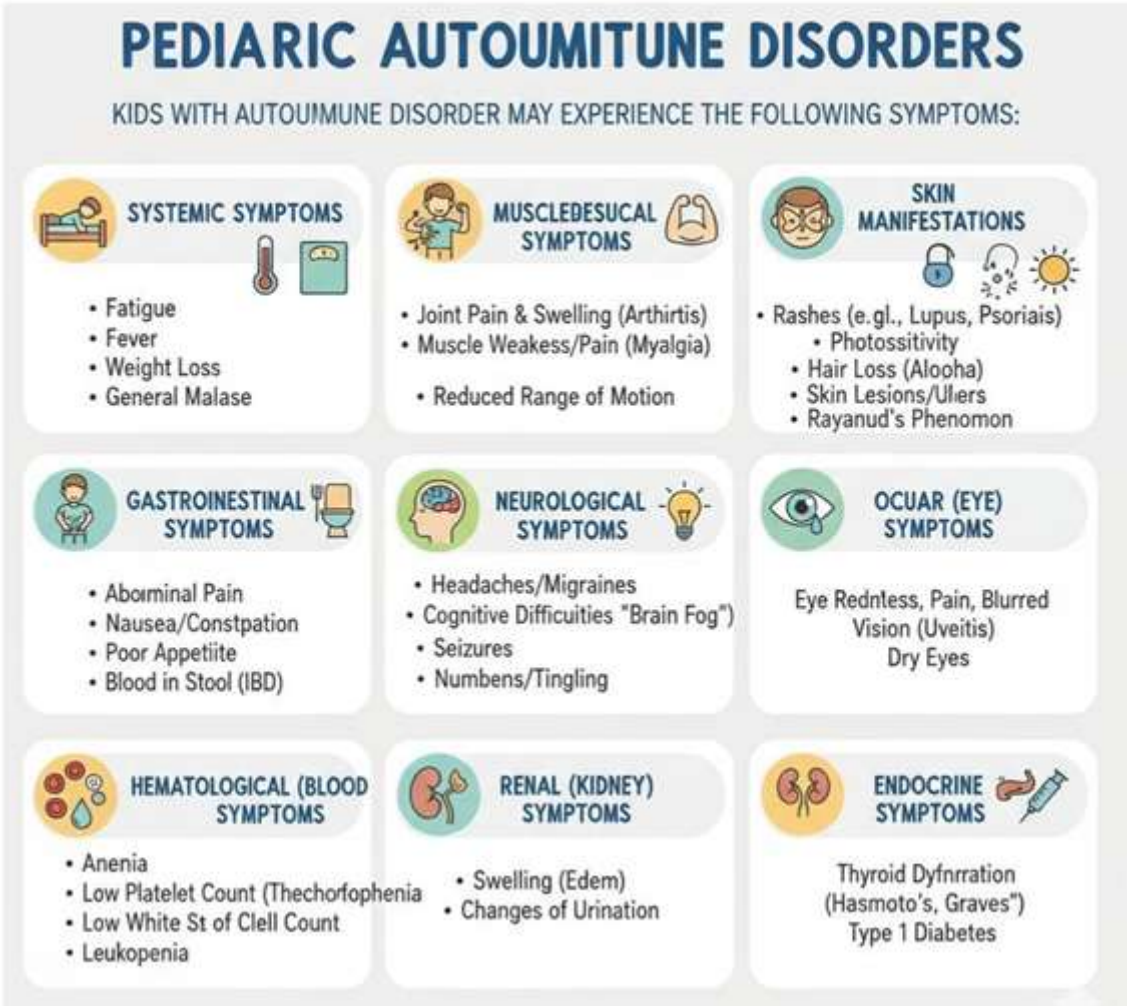

Figure 1

III. PATHOPHYSIOLOGY

PANDAS is classified as an autoimmune disorder because its link to streptococcal infections can be demonstrated. The primary pathophysiological mechanism involved Molecular mimicry is when a foreign antigen resembles the body's own proteins, causing the immune system to mistakenly target itself resembles the body's own antigens in sequence or structure[6]. Streptococcal bacteria can evade the immune system by imitating host cells, resulting in the generation of antibodies that mistakenly target the

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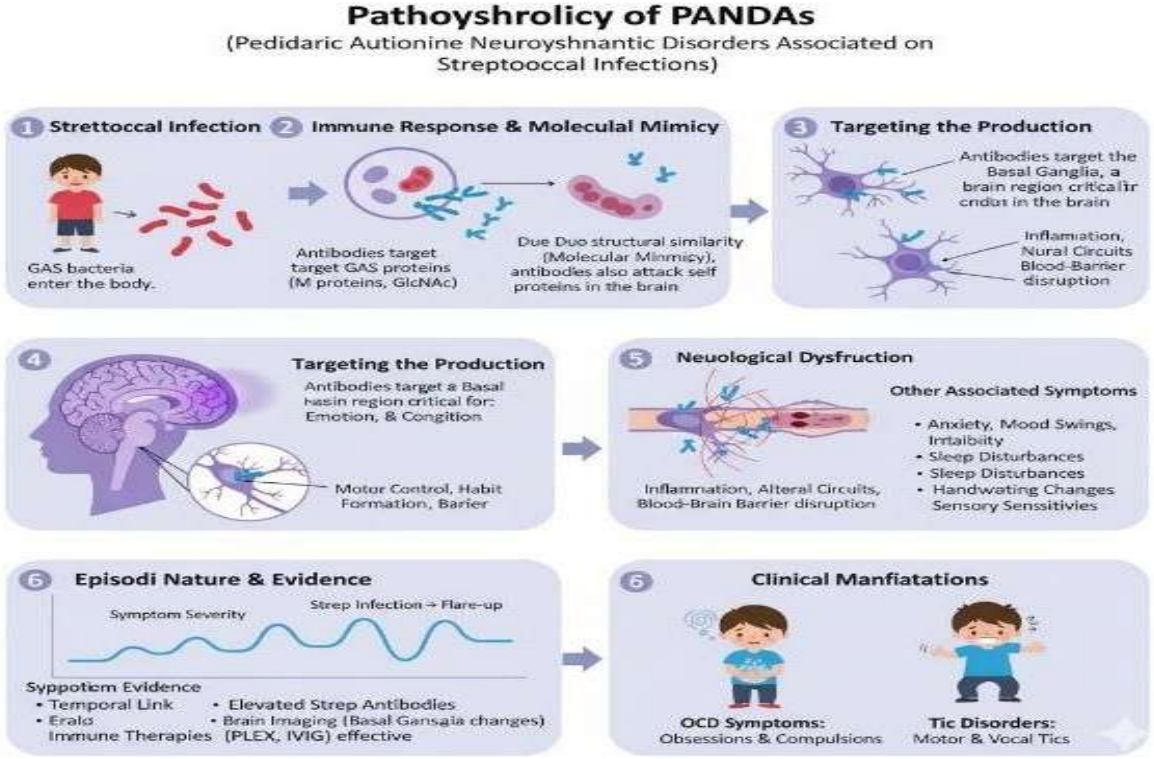
body's own tissues. These cross-reactive antibodies can cross the blood-brain barrier, interact with antigens in the basal ganglia, and trigger neuropsychiatric symptoms. [19] Anti-neuronal autoantibodies interact with specific brain antigens such as lysoganglioside, dopamine receptors, tubulin, and also activate calcium-calmodulin–dependent protein kinase II (CaMKII) in human neuronal cells.

Experimental studies in mice have shown that anti-brain autoantibodies produced in response to streptococcal infection target the dopamine D2 receptor (D2R) and activate CaMKII. These mice exhibited obsessive behaviors resembling those observed in human PANDAS cases. [20]. Several studies have investigated specific antibodies in the brains of PANDAS patients, suggesting that autoimmune responses to streptococcal infections may target neuronal tissues. Identifying these antibodies could improve diagnostic accuracy and guide the development of targeted therapies for affected children. However, the findings so far have been inconclusive, failing to establish a consistent link between symptom recurrence and the detection of specific autoantibodies indicates an autoimmune reaction targeting neuronal tissues antibodies, and in some cases raising concerns about the research methods used.

Autoantibodies originally associated with Sydenham's chorea—such as those against dopamine receptors D1 and D2, β-tubulin, lysoganglioside-GM1 (lyso-GM1), and CaMKII activity—have been suggested as potential biomarkers for PANDAS. In both chorea and PANDAS patients, CaMKII activity has been observe to increase in response to monoclonal antibodies. These antibodies bind not only to epitopes in the GABHS cell wall but also lysoganglioside, supporting the theory of molecular mimicry as a mechanism underlying the autoimmune response.[21]



IV. DIAGNOSIS OF PANDAS

The diagnosis of autoimmune hepatitis (AIH) relies on a combination of clinical presentation, biochemical markers, immunological findings, and histological examination, along with the exclusion of other liver diseases that may show overlapping serological or histological characteristics, such as hepatitis B, C, and E, Wilson disease, nonalcoholic steatohepatitis, or drug-induced liver injury. Liver biopsy remains essential to confirm the diagnosis and assess the

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extent of hepatic damage.[11,12] Since no single test can definitively establish AIH, the International Autoimmune Hepatitis Group (IAIHG) developed a diagnostic scoring system for research and comparison, which uses both positive and negative criteria to generate a score indicating probable or definite AIH. A later simplified version of the IAIHG[13,14]scoring system is considered more practical for clinical use.[15] However, both systems have limitations in diagnosing juvenile AIH, particularly in cases of fulminant hepatic failure[16]. Additionally, autoantibodies in pediatric patients are often present at lower titers than the thresholds defined for adults, making diagnosis more challenging[17]. Neither IAIHG system can distinguish AIH from autoimmune sclerosing cholangitis (ASC), which requires cholangiographic evaluation at the time of diagnosis[18]

V. LABORATORY TEST

A U.S.-based research group introduced the first standardized panel of laboratory tests, known as the Cunningham Panel, which was developed and marketed to aid in diagnosing and monitoring the severity of PANS/PANDAS. While some research has shown a moderate link between CaMKII levels and symptom severity in patients with PANS or PANDAS, other studies have not found consistent evidence to support the Cunningham Panel as a reliable diagnostic tool for these conditions[22]. The Cunningham Panel measures CaMKII activity and identifies

autoantibodies against D1/D2 dopamine receptors, β-tubulin, and lyso-GM1. The measurement of serum CamKII activity, as outlined by Kirvan et al. (2003) and Moleculera (2016), involves sending the serum collected for a Cunningham Panel in Sweden to Wieslab, where it is frozen[23].Antistreptolysin O and anti-DNase B antibody levels were positive in all individuals diagnosed with PANDAS, whereas these antibodies were not detected in any PANS patients. In contrast, 11 (42.3%) PANS patients tested positive for anti-*Mycoplasma pneumoniae* antibodies, while 5 (19.2%) had anti–Epstein–Barr virus nuclear antigen antibodies. Among those diagnosed with PANDAS, only 74 cases (21.4%) showed clear clinical evidence of a streptococcal infection at the time their neurological symptoms began[24].

5.1 Neuroimaging

The selected studies included various brain imaging methods and metrics. On the structural side, they involved magnetic resonance imaging (MRI) morphometric measures such as brain volume, surface area, and cortical thickness (both region-specific and across the whole brain), along with structural connectivity assessed through diffusion tensor imaging (DTI)[25].

5.2 MRI SCAN

Brain imaging studies suggest that PANS involves an organic cause, where antibodies crossing a weakened blood- brain barrier damage brain areas like the basal ganglia and amygdala. This leads to changes in brain circuits similar to those seen in Sydenham's Chorea, including increased striatal volume and inflammation detected by PET scans[22].

VI. TREATMENT APPROACHES

6.1 Treatment of GAS in children with PANS

An early course of antimicrobial therapy for acute streptococcal infection is advised, even if group A streptococcus (GAS) is not confirmed at diagnosis, in line with the initial management guidelines for rheumatic fever (Gerber et al., 2009). While controlled clinical trial evidence is limited, prompt detection and treatment of GAS infection are considered a reasonable approach to reduce the potential risk of neuronal damage [26]. Literature suggests that beta- lactam antibiotics, particularly cephalosporins, along with azithromycin and clindamycin, may be beneficial for treating affected patients. In contrast, some experts advise against using amoxicillin in PANDAS, as it lacks improved intracellular penetration and is effective only in the course of bacterial cell division [30].

The review panels included experts in diagnosing and treating PANS/PANDAS, along with professionals from various disciplines such as child psychiatry, pediatrics, infectious diseases, microbiology, neurology, neuroimmunology, immunology, and rheumatology. [27]

Immunomodulatory treatment is generally recommended in PANS patients with substantial to severe symptoms. Oral corticosteroids are often effective, especially when started soon after symptom onset, with earlier use linked to faster recovery (Brown et al., 2017a). In cases of persistent or more severe symptoms, extended courses with tapering or steroid pulses may be necessary. High-dose intravenous methylprednisolone (15–30 mg/kg, up to 1 g daily

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for 3–5 days) is frequently chosen as first-line therapy due to its ability to produce rapid and significant improvement, similar to its use in autoimmune encephalitides such as NMDAR encephalitis (Dalmau et al., 2011) [28].

Several studies have reported the use of selective serotonin reuptake inhibitors (such as fluoxetine, fluvoxamine, sertraline, and paroxetine), cognitive-behavioral therapy, and exposure–response prevention to address obsessive- compulsive disorder (OCD) and other severe psychiatric symptoms in PANDAS patients. However, the findings were inconclusive, as these treatments did not show clear superiority over placebo[29] .

6.2 Steroids

The initial major study on steroid treatment in PANS, a retrospective case review, found that patients receiving corticosteroids experienced significantly greater overall improvement and shorter flare durations compared to those who did not receive steroids. The treatment regimen involved 1–2 mg/kg for 5 days, with a maximum dose of 60 mg twice daily[22]

6.3 Treatment of Pandas With The help of Probiotics :

Probiotics are beneficial microorganisms that support digestive health. They are available as dietary supplements and are also found in fermented foods like yogurt, sauerkraut, and kimchi.

In the context of PANDAS, probiotics may offer support by helping to prevent digestive issues that can arise from antibiotic use, which is a common treatment for the condition. Some research suggests that probiotics may be effective in this role

.Another potential benefit of probiotics relates to the gut-brain axis — the communication link between the digestive system and the brain. The balance of microorganisms in the gut may influence both brain function and immune responses.

A 2018 study found that children diagnosed with PANDAS exhibited distinct gut microbiome profiles compared to those without the disorder. The researchers proposed that streptococcal infections might disrupt gut bacteria, which could, in turn, affect the brain and immune system. However, further research is needed to explore and validate this theory.

Currently, it is unclear whether probiotics can meaningfully alter the gut microbiome in children with PANDAS or lead to significant symptom improvement. Clinical trials will be necessary to determine their effectiveness in this regard.

Omega-3 fatty acids are commonly found in various foods, especially in seafood, and are also available as dietary supplements.

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Since the body cannot produce omega-3s on its own, they are considered essential nutrients and must be obtained through diet or supplementation.

These fatty acids play a crucial role in maintaining the health of many body tissues, including the brain. They support brain structure and function and are known to have anti-inflammatory properties.

Due to these benefits, some people include omega-3 supplements in their approach to managing PANDAS. However, there is currently limited scientific evidence on how effective omega-3s are in relieving symptoms of the condition. [33]

6.4 Prophylaxis Of Pandas :

Commonly used antibiotics for prophylactic purposes include Augmentin (around 400 mg per day), azithromycin (about 250 mg per day), or penicillin (250 mg taken orally twice daily). The exact dosage may vary based on the patient's body weight and tolerance [34].

Penicillins are typically the first-line treatment for GABHS due to their narrow spectrum, but daily dosing can hinder outpatient compliance. Azithromycin, which also covers GABHS, has a long half-life and can be dosed three times weekly—making adherence more likely.[35]

The patient tolerated this regimen well with no side effects and showed good response, supporting its effectiveness. Azithromycin was planned as indefinite prophylaxis, though current literature offers little guidance on the appropriate duration of such treatment [36]

Many herbs like turmeric, ginger, and holy basil have anti-inflammatory effects. To find the most suitable option for your child, consult a certified herbalist. It's important to seek professional guidance or complete proper training before using herbs at home.[37]

For those exploring Eastern Medicine for PANDAS/PANS, work with a certified pediatric acupuncturist. Located at Light & Dark Acupuncture, treatments are personalized based on each child's unique symptoms and constitution, ensuring a tailored, effective approach rather than a generic one.[38]

VII. CHALLENGES AND CONTROVERSIES

7.1 Clinical Trials

Clinical trials provide additional evidence linking S. pyogenes to neuropsychiatric symptoms. Drawing from observations in acute rheumatic fever (ARF)—where penicillin prophylaxis helped prevent recurrences of ARF or Sydenham chorea by blocking S. pyogenes infections—similar studies have been carried out in patients with PANDAS[31].

7.2 Clinical Significance :

Until more research identifies effective behavioral and psychiatric treatments for children with PANS and PANDAS, clinicians must rely on existing literature and clinical experience. This article bridges the gap between empirical practice and available evidence by presenting consensus guidelines for the symptomatic management of these conditions[32]

VIII. CONCLUSION

Pediatric Autoimmune Neuropsychiatric Disorders, such as PANDAS and PANS, involve a complex interaction between infectious triggers, immune system abnormalities, and neuropsychiatric symptoms. Epidemiological data indicate that these disorders predominantly affect school-aged children, often presenting with sudden-onset obsessive-compulsive symptoms, tics, or behavioral changes. The pathophysiology involves autoimmune responses targeting basal ganglia structures following infections, most commonly Streptococcus pyogenes, although other triggers have been implicated. Diagnosis remains clinical, guided by established criteria, detailed history, and exclusion of other neurological or psychiatric conditions. Investigations may include laboratory testing for infection and inflammatory markers, as well as neuroimaging when indicated. Management is multifaceted, combining immunomodulatory therapy, antibiotics when infection is suspected, and psychiatric interventions such as cognitive- behavioral therapy or SSRIs. Early recognition and timely treatment are critical to improving outcomes, reducing symptom severity, and preventing long-term neuropsychiatric complications.

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