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Functional Neurological Symptom Disorder (FNSD)

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Abstract **Case Report**

Functional Neurological Symptom Disorder (FNSD) is characterized by neurological, sensory, or motor symptoms without identifiable neurological pathology. In children and adolescents, FNSD presents additional complexities due to frequent comorbidities such as anxiety, depression, and functional pain, complicating diagnosis and treatment. This study explores the clinical presentations, diagnostic challenges, and the organo-psychiatric interplay in FNSD through three pediatric cases. Case 1 involved a 12-year-old boy with epileptiform seizures, behavioral disinhibition, and cognitive deterioration, eventually diagnosed with bilateral temporal focus and hippocampal atrophy. Case 2 describes a 16-year-old boy with treatment-resistant headaches, paraplegia, and post-traumatic stress disorder (PTSD), where no structural abnormalities were found. Case 3 details a 12-year-old girl with motor tics and tremors, later diagnosed with Wilson's disease. The study highlights the need for a multidisciplinary approach to diagnose and manage FNSD, as physical and psychological stressors significantly contribute to its manifestations.

Keywords: Functional Neurological Symptom Disorder (FNSD), Pediatric FNSD, Diagnostic Challenges, Organo-psychiatric Interplay, Multidisciplinary Approach.

INTRODUCTION

Functional Neurological Symptom Disorder (FNSD) manifests through neurological, sensory, or motor symptoms without an explanation from an identifiable neurological pathology. Patients with FNSD may exhibit a variety of symptoms, such as psychogenic non-epileptic seizures (PNES), abnormal movements (tremors, dystonia, gait abnormalities), loss of motor function (limb paresis), or sensory losses (blindness, deafness, loss of sensation).

In children and adolescents, these clinical manifestations are often complex due to the frequent presence of comorbidities such as anxiety, depression, functional pain, and non-specific somatic symptoms [1]. These factors complicate the diagnosis and treatment of this condition, emphasizing the necessity of an integrated multidisciplinary approach.

Modern classification systems, such as the International Classification of Diseases (ICD) and the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), conceptualize FNSD as a functional disorder, that is, a dysfunction of the nervous system in the absence of structural lesions [2]. This framework is reinforced by the negative results from tests such as computed tomography (CT) and magnetic resonance imaging (MRI) in patients with FNSD.

Study Objective

This study aims to describe the different clinical presentations of FNSD, explore the diagnostic and therapeutic challenges, and highlight the organo-psychiatric interplay in the etiopathogenesis of this disorder, based on three clinical cases.

CLINICAL CASES

Case 1: M., 12-year-old boy

M. is a 12-year-old boy referred to the child psychiatry department for the management of epileptiform seizures. His medical history begins at birth with neonatal distress, followed by a diagnosis of epilepsy at the age of 2. His antiepileptic treatment failed to control his seizures, which persisted until the age of 6. Complementary examinations (EEG, brain MRI) performed at that age returned normal results, ruling out any structural abnormalities.

At the age of 9, M.'s clinical picture worsened with the onset of complex psychiatric symptoms. He presented logorrhea, characterized by rapid associations of ideas and incoherent speech. His hyperactive behaviors, such as fleeing the house and wandering

aimlessly, were also concerning. Behavioral disinhibition was noted, with inappropriate sexual remarks, as well as excessive familiarity with strangers.

In school, M. regularly disrupted classes and often displayed grandiosity, with an overestimation of his abilities and unrealistic anticipation of his future. His seizures became more complex, alternating between convulsions and erratic behaviors.

At the age of 12, his condition deteriorated further, with postictal confusion and an intensification of hypomanic symptoms. Faced with this worsening, additional examinations revealed a bilateral temporal focus on EEG and left hippocampal atrophy on MRI. His treatment was adjusted with the introduction of carbamazepine and aripiprazole, leading to a significant improvement in epileptic seizures and behavioral disorders.

Case 2: A., 16-year-old boy

A., a 16-year-old adolescent with no significant medical history, began to experience treatment-resistant headaches at the age of 11. The initial treatment with Laroxyl was ineffective. A few days later, paresthesias in the lower limbs appeared, followed by paraplegia and sphincter incontinence. The adolescent also suffered from retrograde amnesia. Given the severity of the symptoms, he was hospitalized in the pediatric department.

Complementary examinations (MRI, EEG) showed no neurological abnormalities. He was then transferred to the child psychiatry department for evaluation. The initial interview revealed a major depressive syndrome with psychotic features, including hallucinations. Treatment with risperidone and fluoxetine was initiated, but no notable improvement was observed.

At the age of 12, A. developed diabetic ketoacidosis due to newly diagnosed diabetes, requiring a new hospitalization. During his stay in pediatrics, suspicious sexual behaviors between him and his 25-year-old brother were observed. A more in-depth re-evaluation of his history led to the diagnosis of post-traumatic stress disorder (PTSD) associated with functional neurological symptom disorder.

The treatment with aripiprazole and fluoxetine was adjusted, resulting in a slight improvement in mood but persistence of the neurological symptoms.

Case 3: K., 12-year-old girl

K. is a 12-year-old girl with no significant medical history, referred to the child psychiatry department for the management of motor tics. She presented with eye blinking and a brief head tilt to the right. After initial management with alpha-agonists, a moderate improvement in symptoms was noted.

A few months later, she developed tremors in her extremities. Functional neurological symptom disorder was suspected and confirmed by a detailed clinical evaluation. Shortly after, she developed dysphagia associated with intermittent aphonia, prompting further testing that revealed copper overload, consistent with Wilson's disease.

The specific treatment for Wilson's disease was initiated, and close monitoring was implemented to track the evolution of the functional neurological symptoms.

DISCUSSION

A detailed medical history and careful observation of the patient, along with a formal neurological examination, are essential for identifying inconsistencies that either support or contradict functional neurological presentations. This is illustrated by the first clinical case, where a thorough analysis revealed postictal confusion, along with signs of fatigue, a general decline in health, and behavioral deterioration proportional to the intensification of seizures. These symptoms served as warning signs, leading to additional clinical and paraclinical investigations.

The rate of misdiagnosis of an organic disease in patients with conversion disorder appears to be around 5% [2]. This was confirmed in the short-term follow-up of our patient. There is abundant literature on clinical indicators for distinguishing conversion disorder in children with neurological symptoms. Table 1 describes some of the classic signs useful for this distinction [3].

The second patient in our study had presented headaches before the onset of motor symptoms in a context of psychological stress. Clinicians in the 19th century had already recognized that functional neurological symptoms appeared in contexts of stress, pain, fatigue, injuries, or psychological trauma, in response to sudden, intense, and overwhelming emotions [4, 5]. In the 20th century, the diagnostic classification systems for functional neurological disorders (FND) focused exclusively on psychological stress factors, neglecting physical events. However, this view evolved in the 21st century. In 2013, the DSM-5 abandoned the requirement for psychological stressors to make a diagnosis of FND [6].

A study by Pareés *et al*., (2014) showed that in 50 adult patients with functional motor symptoms, approximately 80% of presentations were triggered by physical events. Consistent with earlier clinical observations, 70% of patients also experienced panic symptoms, such as nausea, numbness, and tremors, at the onset of FND symptoms. Additionally, several studies have suggested that adult patients with FNSD are in a state of chronic hyperarousal [7]. Neuroimaging studies have revealed hyperactivity in the brain regions involved in emotional arousal and processing, which appear to

overconnect with motor regions, disrupting their function (see Blakemore *et al*., [8] for a review).

Similar results have been observed in children and adolescents in three different research cohorts. Physical events preceded the onset of functional neurological symptoms in 49% to 60% of patients [9-11]. Minor physical injuries (in over one-fifth of the patients) and viral illnesses (in over one-tenth of the patients) were the most common triggers. Family reports indicate that physical events often act in combination with other relational or psychological stressors. Furthermore, high rates of non-specific somatic symptoms (60% to 88%) and comorbid pain (61% to 84%) have been observed. These somatic symptoms are often linked to increased autonomic arousal and motor activation [1]. Using biological markers, it has been confirmed that these patients present in a state of heightened brain-body arousal and motor readiness (see Kozlowska *et al*., [11] for a review).

Neurological symptom	**Neurological signs used by the neurologist to support a diagnosis of TNF (Tumor Necrosis Factor)**
Crossed symptoms	The symptoms are more pronounced when the child is focused on them and less pronounced when the child's attention is directed elsewhere. The symptoms vary depending on the context (for example, a child with vision loss may use their phone but cannot see the text they need to read in class; a child's difficulty walking is present when walking forward but not when turning around; tremors in a limb are less noticeable when the child is distracted by the neurologist).
Difficulty walking	A wavering gait or apparent loss of balance with a narrow-based walk. Each foot is lifted off the ground as if requiring a great effort and placed back as if requiring a great effort. The child walks with bent knees (which requires more strength than normal walking)
Weakness (generalized or partial)	Inconsistency between the strength or functional capacity of the affected body part of the child during a formal examination and during routine tasks (e.g., moving across the hospital bed). Weakness in the limbs that does not conform to an anatomical distribution (e.g., weakness in the arms and legs on opposite sides of the body)
Tremor	Variable distribution or frequency of the child's tremors when examined at different times. The child's tremors change with contralateral body movements (training).
Sensory symptoms (pain excluded)	Sensory symptoms that do not conform to a dermatomal distribution. Hemisensory loss with a clear median distribution.
Loss of vision"	Tunnel vision. Preserved response to a 'threat reflex' (the rapid approach of an object)

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