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Neurological Evaluation in a Child Presenting with Tic-like Symptoms: A Case Report

Tik Benzeri Belirtilerle Başvuran Bir Çocuğun Nörolojik Değerlendirmesi: Olgu Sunumu

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Abstract: This case report highlights the importance of excluding neurological etiologies and employing a multidisciplinary approach in children presenting with tic-like symptoms. It describes the clinical and diagnostic process of a seven-year-old male initially referred to the child psychiatry clinic with a presumptive diagnosis of tic disorder, who was later redirected to pediatric neurology due to concerns about an underlying neurological condition. The patient exhibited intermittent, involuntary muscle twitches in the right eye and right hand. Electroencephalography (EEG) revealed sharp wave activity in the right frontotemporal region, while brain magnetic resonance imaging (MRI) demonstrated a nonspecific T2/FLAIR hyperintensity in the left frontoparietal area. Cerebrospinal fluid (CSF) analysis and infection panels were unremarkable. Clinical improvement was observed following treatment with intravenous immunoglobulin (IVIG) and pulse corticosteroids. This case underscores that tic-like symptoms are not always indicative of primary tic disorders and should prompt thorough evaluation for alternative etiologies, including epileptic activity, structural brain abnormalities such as focal cortical dysplasia, and post-inflammatory neurological processes..

Keywords: Tic, epilepsy, child, focal cortical dysplasia, IVIG, EEG

Özet: Bu olgu sunumu, tik benzeri semptomlarla başvuran çocuklarda nörolojik etiyolojilerin dışlanmasının ve multidisipliner bir yaklaşım benimsenmesinin önemini vurgulamaktadır. Sunumda, başlangıçta tik bozukluğu ön tanısıyla çocuk ve ergen psikiyatrisi polikliniğine yönlendirilen, ancak altta yatan nörolojik bir durumdan şüphelenilmesi üzerine pediatrik nörolojiye sevk edilen yedi yaşındaki erkek bir hastanın klinik ve tanısal süreci anlatılmaktadır. Hastada sağ gözde ve sağ elde aralıklı, istemsiz kas seyirmeleri gözlenmiştir. Elektroensefalografi (EEG) incelemesinde sağ frontotemporal bölgede keskin dalga aktivitesi saptanmış, beyin manyetik rezonans görüntülemesinde ise sol frontoparyetal bölgede nonspesifik T2/FLAIR hiperintensitesi izlenmiştir. Beyin omurilik sıvısı (BOS) analizi ve enfeksiyon panelleri normal olarak değerlendirilmiştir. Hastada intravenöz immünoglobulin (IVIG) ve pulse kortikosteroid tedavisi sonrası klinik düzelme gözlenmiştir. Bu olgu, tik benzeri semptomların her zaman primer tik bozukluklarına işaret etmeyebileceğini ve epileptik aktivite, fokal kortikal displazi gibi yapısal beyin anormallikleri ile postinflamatuar nörolojik süreçler dahil olmak üzere alternatif etiyolojiler açısından ayrıntılı bir değerlendirme yapılması gerektiğini ortaya koymaktadır.

Anahtar Kelimeler: Tik, Epilepsi, Çocuk, Fokal Kortikal Displazi, IVIG, EEG

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1. Introduction

Tic disorders are common neuropsychiatric conditions in childhood, with a prevalence estimated between 1% and 3% in school aged children. They are typically characterized by sudden, rapid, nonrhythmic motor movements or vocalizations, and most cases follow a benign and self-limited course[1]. However, clinical features such as persistent asymmetry, focality, or resistance to standard psychiatric therapies may suggest an underlying neurological disorder[2].

Differential diagnosis is crucial, as certain neurological conditions including epilepsy, autoimmune encephalitis, and focal cortical dysplasia (FCD) may present with tic-like or paroxysmal movements. FCD, in particular, is a malformation of cortical development that frequently manifests with seizures and, less commonly, abnormal movements. Similarly, post-infectious or immune mediated neurological syndromes may mimic tic disorders, especially in the pediatric population[3]. In the literature, the absence of neurological evaluation in the differential diagnosis of tic-like has been reported to lead to delayed diagnosis and treatment failure[4,5].

This case report demonstrates through a concrete example that not every patient presenting to child and adolescent psychiatry outpatient clinics with tic complaints necessarily has a primary tic disorder. The principal contribution of this article to the literature is its demonstration that structural abnormalities, such as focal cortical dysplasia or post-inflammatory processes, may underlie atypical motor phenomena that outwardly resemble typical tics. In this context, our case reveals that a diagnosis based solely on clinical observation may be misleading and highlights how vital advanced investigations such as EEG and MRI are in cases with an atypical course. In particular, the rapid response to immunotherapeutic options such as IVIG and pulse steroids clearly documents the decisive role of immune-mediated mechanisms in both the diagnosis and management of such secondary tic presentations. In conclusion, this study occupies a distinctive place in the literature by emphasizing the critical importance of multidisciplinary collaboration between psychiatry and pediatric neurology in achieving accurate diagnosis and successful treatment.

2. Case Presentation

A seven-year-old male patient was admitted to the child psychiatry outpatient clinic due to an involuntary and sudden twitching movement resembling a spasm in the right eye and right hand. He says that it appeared involuntarily without an obvious trigger and that he could not control it. During the examination, tic-like eye movements were observed localized to the right side, which were repeated many times. Four years ago, it was learned that similar complaints were seen for a short period of time and resolved spontaneously. It was also stated that the patient also had an excessive blink episode at the age of 1.5 months and improved with topical treatment given considering possible ocular infection. In the family history, a paternal uncle had epilepsy. He has recently been treated for frequent upper respiratory tract infections and acute gastroenteritis. In the process of evaluation for adenoidal hypertrophy due to oral inhalation during sleep.

He was referred to pediatric neurology due to his atypical scenario in the patient's anamnesis. Cranial computed tomography (CT) and diffusion-weighted magnetic resonance imaging (MRI) findings were normal. Electroencephalography (EEG) results revealed sharp wave activity in the right frontotemporal region along with significant muscle artifacts. Clinically, choreiform and myoclonic movements were observed in the right hand, as well as clonic twitches in the right eye and thumb. No neurocutaneous findings were detected, his reflexes were not abnormal, and the cerebellar examination was within normal limits. Brain MRI revealed a 7 mm T2/FLAIR hyperintense focal signal change at the left frontoparietal cortico-subcortical junction. This lesion was considered important for FCD. Magnetic resonance angiography and CSF analyses, viral polymerase chain reaction (PCR) tests (for herpes simplex virus and enterovirus), oligoclonal bands, measles IgG, anti-NMO, anti-MOG, paraneoplastic panel and limbic/autoimmune encephalitis antibodies were within normal limits or gave negative results.

The patient was treated with methylprednisolone (30 mg / kg for three days) and intravenous immunoglobulin (IVIG, 2 g / kg for five days) due to suspected neuroinflammation. There was a significant decrease in involuntary eye and hand

movements and an improvement in general clinical condition. In outpatient treatment, clonazepam, initially administered 0.5 mg + 1 mg, was gradually reduced to 0.5 mg, valproic acid continued as 500 mg, due to the risk of seizures. Oral steroids were discontinued, IVIG was continued for maintenance with six total doses for six months. At the six-month follow-up, both the control EEG and neurological examination were unremarkable. Follow-up brain MRI performed at the same time demonstrated complete resolution of the previously identified lesion. The patient was subsequently monitored for one year with evaluations at six-month intervals. Throughout this period, no seizures or additional neurological events were reported, and his symptoms resolved completely.

3. Discussion

Tic-like movements in pediatric patients can be challenging to diagnose, as they may reflect a wide spectrum of etiologies, ranging from benign primary tic disorders to more serious neurological or neuroimmunological conditions. While primary tic disorders such as Tourette syndrome often present during early childhood and follow a waxing and waning course, atypical features, such as asymmetry, persistence during sleep, or the presence of other neurological signs, should prompt consideration of alternative diagnoses[4,5].

The patient was initially presented with focal intermittent motor movements involving the right eye and hand, which were assumed to be simple motor tics. Further examinations were requested because the examination was performed and neurological reasons that may come with tic-like movements were suspected in his history. The presence of epileptiform discharges on the Eeg and an MRI finding suggestive of fcd shifted the diagnostic focus to a neurological origin. FCD is a common cause of medically refractory epilepsy in children and can occur not only with seizures, but also with movement disorders, including tic-like or myoclonic activity, especially when the motor cortex is involved[6,7]. The lesion in the left frontoparietal cortex likely corresponded to contralateral motor symptoms, supporting this hypothesis.

FCD classification is important for both prognosis and management, particularly in the context of epilepsy. According to the Palmini classification, FCD is divided based on histopathological features into Type I (characterized by abnormal cortical layering or dyslamination) and Type II (marked by abnormal layering with dysmorphic neurons, with or without balloon cells). Notably, FCD Type II is more frequently associated with drug-resistant epilepsy and displays characteristic MRI findings, such as cortical thickening and T2/FLAIR hyperintensities at the cortical–subcortical junction[8]. While histopathological confirmation was not possible in our patient, the imaging and clinical findings were consistent with FCD, likely Type II, supporting the diagnosis and influencing subsequent management decisions.

Moreover, the significant clinical improvement following immunomodulatory therapy with IVIG and corticosteroids suggested a possible overlap with autoimmune or post-infectious inflammatory encephalitis. Neuroinflammation is increasingly recognized as a modifiable contributor to pediatric epileptic and neuropsychiatric presentations[9,10]. In anti-NMDA receptor encephalitis and other autoimmune encephalitis, movement disorders such as chorea, dystonia and stereotypes can be imitated[11]. Importantly, response to IVIG or steroids, even in the absence of detectable antibodies, may support the diagnosis of an immune-mediated encephalopathy[12].

A critical point to focus on in this case is that tic-like symptoms may actually be caused by non-psychiatric neurological mechanisms and may mimic primary tic disorders. Cortical overstimulation, paroxysmal dyskinesias, or mild focal seizures in which consciousness is not affected may appear as simple motor tics. It is vital to carefully distinguish these conditions from each other, because the treatment process may require antiepileptic or immune system regulatory therapies rather than or in addition to psychiatric interventions[13].

In addition, this case shows that transient post-infectious neurological syndromes should definitely be considered in the differential diagnosis. Gastroenteritis and frequent recurrent upper respiratory tract infections in the recent history of the patient strengthen the possibility that an immune system response may have caused the neurological picture. Although rare, it is known that such syndromes can cause reversible movement disorders and changes in brain imaging. Moreover, this condition can sometimes be experienced even without serological or CSF abnormalities[14]. Both the improvement of MRI findings and the disappearance of symptoms after treatment support this assumption.

As a result, the cooperation between child psychiatry and neurology has enabled the deconstruction of this

complex picture and the patients recovery. Especially when unusual tic-like symptoms are observed in children with a family history of epilepsy or a recent history of infection, it is of great importance to take a comprehensive and multimensional approach.

4. Conclusion

Clinicians working in child and adolescent psychiatry should keep in mind not only the primary tic disorder in children presenting with tic-like symptoms, but also the neurological or immunological processes that may underlie it. Especially unilateral involvement, sudden onset, atypical motor patterns or concomitant neurological signs are noteworthy. A family history of epilepsy or the presence of a recent infection should also be stimulating. In these cases, further evaluation with EEG and neuroimaging should be considered. As can be seen in this case, epilepsy and immune-mediated mechanisms may initially appear with typical looking tic-like findings. If not carefully evaluated, this picture may lead to diagnostic delay. Therefore, early detection of warning signs and timely neurological evaluation are of great importance.

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