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Tic Disorder and High Mycoplasma Levels in Children: A Case Study

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ABSTRACT

J.F. is a six-year-old male child with a new onset of transient tic disorder after having a mycoplasma infection. The child was evaluated by a pediatric neurologist, who confirmed the tic disorder but did not recommend treatment or follow-up. The primary health care team obtained lab work for Complete blood count (CBC), Comprehensive metabolic panel (CMP), Mycoplasma IgG/IgM, ASO, Anti-DNAse B antibody, sedimentation rate, Lyme ELISA and western blot, serum copper, iron, vitamin D 25 hydroxy, zinc and heavy metals. Labs were unremarkable except positive Mycoplasma IgG and IgM. Azithromycin was prescribed for five days and blood work was repeated for Mycoplasma antibodies 8 weeks after treatment.

Keywords

Transient tic disorder, Pediatric tic disorder, Pediatric neurology, Azithromycin therapy.

Introduction

Tic disorders are common childhood neuropsychiatric conditions. These conditions are characterized by sudden, repetitive motor movements or vocalizations that can be briefly suppressed and are often preceded by an urge [1]. Children can experience symptoms such as clearing of the throat, blinking, head jerking, or grunting. Often, tic disorders are a comorbidity with obsessive-compulsive behavior, attention deficit hyperactivity disorder (ADHD), depression, anxiety, and other behavioral disorders [2].

Tics are believed to result from a multifactorial etiology involving genetic, immunologic, psychological, and environmental factors. Both streptococcal and mycoplasma infections have been associated with the development of tic disorders in children. Growing evidence indicates that childhood infections, including Mycoplasma pneumoniae, are associated with an increased risk of developing major mental disorders.

Mycoplasma pneumoniae infections in individuals with Tourette syndrome have reported that macrolide antibiotics effectively alleviated symptoms, suggesting that mycoplasma infection may serve as an aggravating factor in the manifestation or severity of the disorder [2]. A study published in China discovered that children treated with a macrolide after testing positive for mycoplasma pneumonia IgM improved with symptoms and once antibiotics were discontinued there was an associated increased risk of chronic tic disorder. Although the exact mechanism is not fully understood, a positive IgM response may indicate immune system activation that could contribute to the development or exacerbation of tics. Vocal tics have a significant higher recurrence risk compared to motor tics [3].

Mycoplasma pneumonia may be a critical infectious activation of Pediatric Acute-onset Neuropsychiatric Syndrome (PANS), and should be considered for any child presenting with motor of vocal tics. According to a study published by Schnell et al., [4], Mycoplasma IgG positivity was associated with higher tic severity in children and adolescents with established chronic tic disorders.

Case

This case study examines a 6-year-old Caucasian male with no prior psychiatric issues presenting with sudden onset of motor tic. Two weeks prior to the tic the child presented with fever, cough, and sore throat and was diagnosed with viral upper respiratory

infection (URI). The child's medical history includes a diagnosis of mild intermittent asthma, which is controlled by fluticasone and albuterol as needed. The mother denies a family history of tic disorder or neuro-psychiatric disorders.

Upon evaluation of the child post infection, the mother presented video of the child's tic behavior. This was evident by clearing of throat and jerking movement of the neck. The child demonstrated recurrent symptom episodes occurring multiple times per day. Lab work was obtained and mycoplasma IgM antibody was 1,376 U/mL and IgG was 2.84 U/mL. The child was treated with a five-day course of azithromycin and symptoms resolved over time.

Eight weeks later the child presented with cough and similar motor tic. Mycoplasma antibodies were repeated and elevated with an IgM titer of 1,885 U/mL and an IgG titer of 3.25 U/mL only two months post initial infection. The child was treated with clarithromycin for three weeks and symptoms again resolved. Mom states the child develops similar motor tic with any URI symptoms or cough but they do not las as long as the initial infection.

The child's symptoms subsequently improved, and no further pediatric reevaluation was sought for this concern. More recently, he exhibited declining academic performance and impaired attention, leading to a diagnosis of attention deficit hyperactivity disorder (ADHD) inattentive type.

Conclusion

This case highlights a possible association between *Mycoplasma* infection and transient tic disorder in children. Some infectious triggers are treatable, and addressing them may reduce symptom severity or prevent recurrence. Recognition of this potential relationship may encourage clinicians to consider infectious etiologies in pediatric tic presentations and guide management decisions.

References

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