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Acquired stuttering in a 16-year-old boy

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ABSTRACT

This case study describes a 16-year-old boy who started to stutter after a rotavirus infection followed by signs suggestive of a cerebellar encephalitis. It illustrates the fact that acquired stuttering can be observed in younger children and that it may be difficult to distinguish neurogenic from psychogenic forms of acquired stuttering in some cases. This is especially true following a disease that is not commonly associated with acquired stuttering. Speech, language and medical results of our patient are reported in detail. Similarities with previously reported cases and comparisons to (speech) characteristics of psychogenic and neurogenic stuttering will be discussed.

1. Introduction

Acquired stuttering is a disorder characterized by stuttering-like disfluencies which appear gradually or suddenly in mostly adult patients who have no previous history of stuttering. It rarely occurs in children (e.g. Nass, Schreter, & Heier, 1994; Yeoh, Lind, & Law, 2006) and contrasts with developmental stuttering which normally has its onset between the age of 2 and 6 years (Bloodstein, 1995). Acquired stuttering can be differentiated in a neurogenic and a psychogenic form. Neurogenic stuttering typically occurs in adults following stroke, traumatic brain injury or neurodegenerative disease (De Nil, Rochon, & Jokel, 2008; Theys, van Wieringen, & De Nil, 2008). It has also been described following epilepsy (Michel et al., 2004), encephalitis (Chen & Peng, 1993), use of medication (Movsessian, 2005), anorexia (Byrne, Byrne, & Zibin, 1993) and other disorders that might affect brain function. In contrast,

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psychogenic stuttering most likely appears as a consequence of an emotional or psychological trauma (De Nil et al., 2008) and has been characterized as a conversion reaction (Mahr & Leith, 1992). It often co-occurs with nonorganic somatic complaints, which may raise a suspicion of neurologic disease and thus complicate the differential diagnosis between neurogenic and psychogenic stuttering (Roth, Aronson, & Davis, 1989).

In this paper we present the case of a 16-year-old boy who started to stutter following a rotavirus infection, accompanied by signs suggestive of a cerebellar encephalitis. This case illustrates the fact that acquired stuttering can be observed in younger children and that it may be difficult to distinguish neurogenic from psychogenic forms of acquired stuttering in some cases, especially following a disease that is not commonly associated with acquired stuttering.

2. Case description

At the age of 16, D. had a throat infection followed by episodes of vomiting, diarrhea and loss of consciousness. He was admitted to hospital where he was diagnosed as having a rotavirus infection. While he was admitted, he was short of breath and experienced elevated blood pressure. These symptoms were diagnosed as fear-triggered hyperventilation and D. was discharged from hospital after one week. Seventeen days later, he started experiencing neurological problems, including difficulties with normal daily activities (such as writing and getting dressed), reduced talkativity, episodes of crying, and mood changes. His body temperature dropped below 36 °C. In addition, he reported dizziness and headache. These signs lasted for a few days and an EEG and CT-scan were performed. Both were found to be within normal limits. Four days after the first occurrence of the neurological symptoms, D. additionally experienced loss of hand control, loss of consciousness (lasting for a few seconds) and naming difficulties. It was at that time that stuttering was first observed. According to D. and his parents the stuttering started suddenly and was not associated with an emotional or stressful event. On the contrary, D. expressed regret about the occurrence of his stuttering and of the other neurological symptoms because they interfered with the many activities he had planned for that period. The case history did not reveal evidence of past emotional or behavioral problems. According to D. and his mother, there was no history of stuttering in childhood, nor a family history of stuttering. However, he did receive one year of therapy for speech and language delay at the age of four. Additionally, he underwent an adenotonsillectomy at the age of three and a pharyngoplasty due to hypernasality at the age of five.

Over the next days the neurological symptoms worsened. There was further deterioration of speech and walking, and dizziness, headache and disturbance of equilibrium were reported, which led to admission to the emergency department of the hospital. At admission, he was focused into himself, did not speak and only showed minimal reactions to his environment. The EEG, ECG, NMR and lumbar puncture performed in the hospital were within normal limits, but since the symptoms were suggestive of cerebellar encephalitis, a treatment with Zovirax was started. Following the initiation of this drug treatment, the symptoms gradually improved and D. was discharged from the hospital 10 days later. When he returned home, the symptoms worsened again and he was referred to another hospital for further investigation. This time the attending neurologist concluded that there was an inconsistency in the clinical-neurological symptoms since she observed that D. did not always stutter and that his loss of strength and gait disturbance disappeared when he was not aware of the examinations. Consequently, D. was referred for a psychiatric evaluation. This assessment led to insufficient indications to support a psychiatric diagnosis following the DSM-IV criteria (American Psychiatric Association, 2000). However, the psychiatrist concluded that it was not impossible that D.'s disease could have been intensified by some psychological factors since he was characterized as a sensitive boy in his puberty, and had some difficulties with the language courses in school. Because a straightforward diagnosis of either a neurological or psychiatric cause could not be made, the diagnosis of a somatoform disorder was given.

During his psychiatric evaluation, D. was assessed for the first time by a speech-language pathologist. During the assessment, his speech was reported to be continuously disfluent. D. remarked that his speech was more disfluent in the morning and evening when he was tired. He still dared to speak, even to strangers, but he found the stuttering very annoying and did not understand why the words