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CASE REPORT

Foreign accent syndrome and psychosis: a case report

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This is a case report on a 57-year-old lady who presented to the Psychiatry Department of Tallaght Hospital after being referred by Neurology Department. She was initially attending Neurology Department for idiopathic Parkinson's disease which was later refuted as there was no objective evidence of response to treatment. She attended the A&E department with altered consciousness and headache, but her computed tomography of the brain was normal, and was diagnosed with migraine. But it was noted that she was reporting paranoia and auditory hallucinations, and her family had noticed marked change in her accent. On assessment in psychiatric outpatient she was noted to have psychotic symptoms. She reported these symptoms to be ongoing for the last 4 years, but have worsened to an extent that she was extremely distressed by them. She had acquired a Scottish accent. The change in accent coincided with worsening of her psychotic symptoms. Based on her assessment she was diagnosed with schizophrenia and was commenced on olanzapine. Not only her psychotic symptoms resolved but also her accent reverted to native form.

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Introduction

Foreign accent syndrome (FAS) is a speech disorder characterized by a sudden change of speech so that a native speaker is perceived to speak with a foreign accent. FAS was first described by Pierre Marie in 1907 and Pick in 1919. More than 100 case reports have been published. FAS is most often caused by damage to the brain due to stroke or traumatic brain injury. Other reported causes include multiple sclerosis, psychotic and conversion disorders. In some cases no clear cause is identified. Different explanations of the functional origin of FAS have been suggested, one of the more frequent being impaired access to verbal–motor patterns or a mild form of apraxia of speech. Clinical manifestations can vary but FAS usually involves segmental (changes in vowel length and tenses) and prosodic (inappropriate word and sentence stress) deficits.

Case history

Background and medical history

AH, a 57-year-old mother of two was referred to the Neurology Department of Tallaght Hospital in April 2005. She reported a 12-month history of left hand tremor and left arm weakness. She also reported headaches for 2 years, a long-standing lisp and intermittent choking sensations on solid food. On assessment she had a mild left postural tremor, there was no bradykinesia, no cranial nerve deficits, no cerebellar signs and her peripheral nervous system examination was normal. She had a dopamine transporter (DAT) scan that showed decreased uptake in left basal ganglia. Her full blood count, ceruloplasmin, liver function tests, renal profile, serum protein electrophoresis, magnetic resonance imaging of the brain and barium swallow were normal. Based on the DAT scan, mild early idiopathic Parkinson's disease was diagnosed and she was commenced on selegiline 5 mg daily. Her symptoms persisted with no improvement (or deterioration) at follow-up.

Course

During the course of follow-up, her social circumstances changed. She moved from her home of 20 years due to antisocial behavior by neighbors. Her sleep became disturbed with nightmares and she presented to the Emergency Department (ED) in July 2011. She reported palpitations and chest tightness. Cardiac examination and investigations were normal and her symptoms were attributed to anxiety. In September 2011, she re-attended the ED with headaches and drowsiness. Her clinical examination did not reveal any neurological signs, computed tomography (CT) of the brain was normal and there was no evidence of stroke. She was diagnosed with migraine. In July 2012, she re-presented to the ED in similar circumstances with headaches and drowsiness. She was admitted to hospital and was found to be dehydrated. Further investigations did not reveal a cerebrovascular event. However, 12 hours after presentation, her speech had

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taken on an unusual characteristic. She was born and raised in inner city Dublin but now had a Scottish accent for the first time in her life. Her family confirmed that this was a new change in her accent. She has been a housewife for most part of her life, and has never been to Scotland before.

In March 2013, AH was assessed by a Neuropsychologist for an opinion on whether her change of accent was functional in origin or secondary to an organic cause such as a cortical ischemic infarction. Her Neuropsychologist did not conduct psychometric testing and concluded that AH had started to like her new accent as it helped her to distance herself from life events. She was found to be comfortable with her new accent and was fluent and forthcoming during the assessment. Her Neuropsychologist concluded that it was plausible that her symptoms and new accent represented a psychologically mediated phenomenon. No intervention was recommended at the time but AH was offered the opportunity to re-engage if she had further concerns in relation to her speech or communication.

Follow-up

In 2015, at a follow-up neurology appointment, she was assessed by a different Consultant Neurologist who concluded that her presentation was not consistent with Parkinson's disease. It was noted that she did not show classic signs of the condition and that anti-Parkinsonian medication was ineffective. The diagnosis was refuted and she was subsequently discharged from the neurology service. No follow-up or speech and language therapy for her change of accent was arranged.

Psychiatric history

In 2015, AH was referred to the Tallaght Mental Health Service with a first presentation of psychotic symptoms. She had moved house at this stage and described paranoid delusions about her neighbors trying to hurt her. She also reported auditory hallucinations – hearing her new neighbors threatening her. Her paranoia and hallucinations started in 2011 and was worsening, which led to her disclosing them now for the first time. She described associated social withdrawal, reduced self care, poor motivation and low energy.

Collateral history

AH's husband reported changes in her behavior. She was noted to be anxious and increasingly suspicious of her neighbors. He first attributed this to antisocial behavior in her neighborhood in 2011. However, her symptoms persisted in spite of moving to a new neighborhood where antisocial behavior was not a problem. He confirmed that her accent had changed in 2012. He also endorsed reduced interests, social withdrawal and poor motivation.

Treatment

AH was commenced on olanzapine 10 mg daily and followed up in the Acute Day Hospital. She improved on antipsychotic treatment to the point where her psychotic symptoms abated; she started functioning at a better level and started socializing again with her husband. Further, her accent returned to native form. At follow-up in the outpatient department, her improvement was sustained, she was not psychotic and denied any trouble with her neighbors. Her accent remained in native form. A diagnosis of schizophrenia was made on the basis of overt psychotic symptoms, negative symptoms and functional impairment lasting for 4 years before treatment.

Discussion

The etiology of FAS in the case of AH merits exploration. Differential diagnoses to schizophrenia were also considered, including psychosis due to medical condition. She first reported a change in accent around the time she first developed psychotic symptoms with social withdrawal. FAS is rare in psychosis and schizophrenia with deficits of speech (reduced comprehension, semantic deficits, deficits in discourse planning, reduced use of expansive language) and formal thought disorder (positive and negative) more commonly described (DeLisi, 2001). However, the possibility of an association between FAS and psychosis in AH's case must be considered given the temporal relation between the onset of her psychotic symptoms and the change in her accent. Further, within a few months of treatment with antipsychotic medication her accent returned to its native form, supporting the association between psychosis and FAS in this case.

In terms of other explanations, the possibility of a conversion disorder needs to be considered. Gurd *et al*. (2001) reported a case of FAS with an unclear etiology in a 47-year-old female who presented with FAS 1 month following the sudden onset of aphasia and headache. Although the patient had several small lesions on imaging, the authors 'doubted whether all of the patient's symptoms could be accounted for organically.' Verhoeven *et al*. (2005) described a case of FAS in an instance of probable conversion disorder. A case report (Haley *et al*. 2009) described a case of FAS associated with conversion disorder in a 36-year-old female with intermittent episodes of neurological symptoms such as gait disturbances, sensory changes and weakness. A recent case report (Keulen *et al*. 2016) hypothesized that FAS is a mild subtype of apraxia of speech. AH reported distress due to her living situation with much antisocial behavior by her

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neighbors. She attended the ED with symptoms of panic and anxiety in 2011. However, this was after she had moved house and was out of her stressful situation. According to ICD-10 conversion disorders typically occur in the context of traumatic events and tend to remit after a few months. AH's FAS only remitted after introduction of antipsychotic medication and her move of house away from her antisocial neighbors had no effect on her accent. This weakens the argument that AH's FAS was related to an underlying anxiety disorder. Her first referral to a mental health service occurred in 2015 and she reported psychiatric symptoms for previous 4 years. From reviewing her case notes and discussions with AH and her family, her early psychiatric symptoms seemed more consistent with prodromal symptoms with attenuated paranoia and a reduced level of functioning than a primary anxiety disorder.

The possibility of a neurological cause to AH's FAS should also be considered. In the majority of cases published between 1907 and 2014, FAS was thought to have a neurogenic etiology. A recent case report (Keulen *et al*. 2016) hypothesized that FAS is a mild subtype of apraxia of speech. In a review by Baumgartner (1999), the importance of the patient's medical history and other symptoms was highlighted. If medical history, onset of symptoms, symptom characteristics and their evolution, neurological examinations, neuroimaging and cognitive work-up do not unambiguously point toward a neurological disorder, an alternative interpretation should be considered. In AH's case, she presented to the ED with headaches and drowsiness shortly before she developed FAS. Clinical examination and investigations including CT of the brain could not find a conclusive neurological explanation for her symptoms and her presentation was attributed to dehydration. No focal neurological signs were observed and her headaches and drowsiness resolved. Although it is not beyond the range of possibility that she had a subtle neurological event, we believe there is insufficient evidence to attribute her FAS to an underlying neurological cause. The improvement in AH's psychiatric symptoms and FAS on antipsychotic medication points toward a relationship between FAS and psychosis more than other explanations.

The association between FAS and psychosis is not as well established as the association between FAS and neurological disorders. However, a number of case reports exist. Reeves *et al*. (2007) and Reeves & Norton (2001) described a total of three patients with psychosis who exhibited FAS which correlated in severity to the severity of their psychotic signs and whose speech gradually returned to baseline with improvement of their psychosis. The first case reported was of a 30-year-old man with history of schizophrenia. He was born in Alabama but had acquired Jamaican accent during exacerbation of his illness; 6 days after recommencing olanzapine, his accent resolved. The second case reported was of a 53-year-old American lady with history of bipolar disorder who was admitted to hospital with manic psychosis. She was perceived to have a European accent. After being treated with sodium valproate, olanzapine and lithium for 15 days, her psychosis improved and her European accent became less prominent. The third case they reported was of a 66-year-old male with diagnosis of schizophrenia. There were three occasions where the patient had a psychotic episode associated with change of accent. He had lived his entire life in the United States and spent 2 years abroad in Korea. When psychotic he adopted a British accent. On each occasion, treating his psychosis resulted in a resolution of his accent followed by reduction in his psychotic symptoms. Reeves & Norton (2001) reported the case of FAS in a 65-year-old man with a long history of schizophrenia, which resolved after taking the antipsychotic risperidone.

Conclusion

This case adds to the literature of the association between psychosis, in this case schizophrenia, and FAS. Schizophrenia is often characterized by speech and language problems and this case report suggests that FAS could be a rare but associated symptom of the disorder. From this case and the current literature, the association seems to be stronger with advancing age. Although FAS most often occurs in association with an underlying neurological disorder (e.g. stroke), in the absence of a clear neurological disorder or focal neurological signs screening for psychotic symptoms should be pursued.

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Conflicts of Interest

None.

Ethical Standards

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committee on human experimentation with the Helsinki Declaration of 1975, as revised in 2008. The authors assert that ethical approval for publication of this audit has been provided by their local REC.

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