

Clinical Communications: Adults

A CASE OF FOREIGN ACCENT SYNDROME

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Abstract—Background: Foreign accent syndrome is a rare but potentially devastating clinical condition associated with altered speech rhythm and prosody, often occurring after a cerebral vascular accident. Missing this diagnosis can lead to delayed stroke work-up and treatment. **Objective:** We report a case of foreign accent syndrome in a 60-year-old woman who presented to the Emergency Department (ED) with 3 weeks of altered speech pattern, widened gait, bilateral leg heaviness, and mild headache. **Case Report:** The patient had a history of Type 2 diabetes, malignant hypertension, toxic nodular goiter, and hyperlipidemia. She initially presented to the ED at the onset of symptoms, was thought to have had speech change secondary to a goiter impinging on the recurrent laryngeal nerve, and was discharged. She returned to the ED 3 weeks later when outpatient imaging revealed subacute infarction of the left hemispheres and absent flow within the left vertebral artery. On examination, the patient was alert and conversational. She spoke fluently with an accent that had components of Jamaican and the accent of an Italian speaking English. Neurology was consulted and the patient was admitted. The patient was treated medically, and was discharged home with primary care follow-up. She developed a second, more significant stroke 1 month later, with unilateral weakness and slurred speech in the middle cerebral artery distribution. **Conclusion:** Clinicians should be aware that some stroke patients present with various atypical symptoms, and should suspect stroke in any patient with acute-onset neurological symptoms, including speech change. © 2013 Elsevier Inc.

Keywords—foreign accent syndrome; pseudo-foreign accent; cerebrovascular accident; stroke; speech change

INTRODUCTION

Foreign accent syndrome (FAS) is a rare, acquired syndrome that occurs most commonly after stroke. It involves altered speech rhythm and prosody, which listeners perceive as foreign, without the patient having previous personal or family exposure to that accent. Since the first described case by Pierre Marie in 1907, the mechanism remains unclear, and < 20 cases have been reported worldwide, with accompanying details of lesion localization (1,2). Monrad-Krohn reported one of the more famous cases in which a Norwegian woman was ostracized from her community after developing a foreign German accent from a traumatic Broca aphasia amidst the Nazi occupation of Norway during World War II (3). A common single location of infarct has not yet been attributed to this syndrome. Reported locations include the middle cerebral artery (MCA), pre-central and post-central gyri, parietal lobe, inferior frontal gyrus, internal capsule, and basal ganglia – almost always in the left hemisphere. Etiologies other than stroke include traumatic brain injury, early multiple sclerosis, and psychogenic (4,5).

CASE PRESENTATION

A 60-year-old African-American singer with a history of type 2 diabetes, malignant hypertension, toxic nodular goiter, and hyperlipidemia noticed a change in her speech pattern, a widened gait, bilateral leg heaviness, and mild

4/10 headache. The patient described her change in speech as developing a Jamaican accent. Her travel history was unremarkable.

She presented to the Emergency Department (ED), where records indicated normal vital signs and cranial nerves II–XII intact. Gait was not commented on, and no other neurologic testing was done. Computed tomography was unremarkable. The speech change was thought to be secondary to a goiter impinging on the recurrent laryngeal nerve, and the patient was discharged.

The patient followed-up with her primary care physician 3 weeks after the onset of symptoms. A magnetic resonance imaging study demonstrated subacute infarction of the left hemi-pons (Figure 1) and absent flow within the left vertebral artery concerning for occlusion.

The patient then presented again to the ED for further evaluation. She complained of ongoing altered speech pattern, a dull 4/10 right frontal and posterior headache, and widened gait. She denied diplopia, unilateral facial or body weakness, sensory deficits, coordination problems, fevers, chills, or other symptoms.

Her past medical history included the above stated issues as well as obstructive sleep apnea, left ventricular hypertrophy, and asthma. Her medications included aspirin, atenolol, glyburide, metformin, hydrochlorothiazide, lisinopril, methimazole, and cetirizine. She had no history of alcohol or tobacco use. The review of systems was otherwise negative.

On our examination, the patient was in no acute distress, and was alert and conversational. Mental status was fully intact: she was oriented to self, place, and time. Fund of knowledge, concentration, repetition, and three-step tasks were all normal. She spoke fluently with an accent that we felt had components of a Jamaican accent and the accent of an Italian speaking English, ending most words with “-ah.” Cranial nerves II–XII were intact. Motor strength was 5/5 in all groups, except right knee flexion, which was 4+. All deep tendon reflexes were 2+, with plantar response downward bilaterally. Sensations were equal and symmetric in upper and lower extremities to light touch, pinprick, vibration, and temperature. Finger to nose, heel to shin, and rapid alternating movements were intact without action tremor or dysmetria. General gait was notable for a wide base, and tandem gait was unsteady.

Pertinent imaging included magnetic resonance angiography (MRA) of the head and neck, which re-demonstrated the left pontine infarct without hemorrhage. Additionally, multiple areas of irregularity and narrowing were noted in the anterior circulation, as well as diminished flow in the posterior circulation most pronounced in the basilar artery. Computed tomography angiography was not performed due to contrast allergy. Electrocardiogram showed normal sinus rhythm



Figure 1. Unenhanced axial T2 magnetic resonance imaging scan of the head showing left pontine restricted diffusion.

with no ST-segment abnormalities. Laboratory assessments, including complete blood count, basic metabolic panel, international normalized ratio, and activated partial thromboplastin time, were within normal limits.

The patient was admitted to the Neurology service. Neurology agreed with our assessment of the accent sounding Jamaican. The patient was not a tissue plasminogen activator candidate given that she was last at baseline many days prior. Further stroke work-up included carotid artery duplex studies, which demonstrated no significant stenosis. Transthoracic echocardiogram showed an ejection fraction of 65–70% and no vegetations or thrombus. MRA with contrast revealed high-grade stenosis of the basilar artery, fetal origin of the left posterior cerebral artery, and multiple areas of narrowing in both MCAs. The patient scored a 50 on the Boston Naming Test for aphasia (mean is 53.3, with an SD of 4.6); this score was within normal limits. Speech pathology noted epenthesis, adding “-uh” to the end of words and adding “schwa” to the middle or end of words. This was found to be consistent with FAS. Given the patient’s minimal deficits, the patient was discharged home with close follow-up and management of her risk factors with her primary care physician. She subsequently developed a second cerebral vascular accident (CVA) 1 month later, a more significant one with unilateral weakness and slurred speech in the MCA distribution.

DISCUSSION

The pathophysiology behind FAS remains unclear. Among case reports, the general consensus currently is that rather than the patient acquiring a specific regional accent, he or she damages a component of the motor speech network that includes cortical and subcortical components as well as the connections between them. Kwon and Kim reported a case of a 71-year-old Korean woman who, after a left MCA stroke, acquired a changed dialect from that of the Cholla-buk province to that of the Kangwon province (6). The Korean version of the Western Aphasia Battery revealed fluent and spontaneous speech, but with significant reading, writing, and naming deficits. Acoustic analysis comparing speech frequencies of the patient and control patients from the Cholla-buk area revealed that the patient's speech was reflective of the Kangwon province. However, importantly, the patient occasionally mixed words and suffixes from several other dialects. Three of five listeners wavered between identifying the accent as Kangwon vs. North Korean. This case, along with others, supports the hypothesis that rather than acquiring a new accent, patients suffer damage to their segmental and prosodic circuits, which listeners then attribute to accents they are familiar with.

This perspective explains why, in many case studies, the accent comes close to, but does not exactly match, that of the true accent listeners perceive it to be. Naidoo et al. reported a case of a 50-year-old monolingual woman with an acute infarction to the left internal capsule, basal ganglia, and frontal corona radiata, resulting in a switch from a Canadian east coast accent to a Southern Ontario accent (2). Linguistic analysis of her speech was conducted on three interviews beginning at 4 weeks post-stroke. Phonological analysis of these recordings revealed that many elements of segmental (consonant and vowel changes) and prosodic (rhythm, intonation) speech were consistent with that of a Southern Ontario accent. However, the changes were not consistent. "Th" segments were pronounced as "d," but occasionally "th" sounds broke through. Additionally, there were inconsistent features of the patient's accent, such as the insertion of vowels into consonant clusters ("duh-ream" for "dream"). The segmental distortions that occurred with complicated sequences suggest a deficit in motor speech planning.

A key question connecting FAS with other speech disorders is how it relates to aphasias and dysarthrias after stroke. Some researchers have speculated that FAS represents a mild case along the spectrum of aphasias and apraxias of speech; however, more recent literature argues against this (1,2,5,7). Ryalls and Whiteside presented a 57-year-old woman from New York City who experienced a left internal capsule lacunar infarct

and developed a British/American accent (1). Extensive aphasic and cognitive-linguistic tests were performed, which showed good command of prosody, and no difficulty with reading comprehension. She performed exceptionally on the Boston Diagnostic Aphasia Examination, which assessed subclausal, single-clause, and multi-clause utterances when naming a series of pictures. Overall, her performance distinguishes FAS from aphasias. Chanson et al. report a 39-year-old French woman who presented with subacute mild right facial palsy and a German accent, and was found to have a large left perrolandic white matter lesion with multiple smaller lesions of the deep white matter on T2-weighted images consistent with multiple sclerosis (5). She had no evidence of aphasia; her speech was fluent with normal semantic and phonemic patterns, and showed normal comprehension, naming, word-finding, and reading abilities. Therefore, the speech patterns of FAS and Broca aphasia are likely not differences in severity along the same continuum.

Treatment for FAS involves the same interventions for CVA if that is indeed the cause. Incidentally, Cohen et al. report a case of "paradoxical facilitation" in which a patient's FAS secondary to a left frontoparietal infarct resolved after subsequently developing a right inferior cerebellar hemorrhage from over-anticoagulation (8). The authors submit that the right cerebellar stroke paradoxically extinguished the FAS. They postulate that normal cerebellar controls involve competitive interactions between the two hemispheres, and that lesions of one hemisphere may lead to disinhibition of the other. Persistent maladaptive stimulation of the right cerebellum may have prevented the left cerebellum from processing temporal information, specifically controlling the rhythmic and prosodic patterns of speech. This idea offers intriguing implications for research and therapeutic modalities in the future.

CONCLUSION

Clinicians should be aware that some stroke patients present initially with various uncommon and atypical symptoms. Importantly, physicians should suspect stroke in patients presenting with an abrupt onset of any neurological symptoms, including speech and gait difficulties, and perform a complete and systematic neurological examination.

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