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CASE REPORT



PATIENT WITH FOREIGN ACCENT SYNDROME IN POST INFARCT THROMBOTIC STROKE: A CASE REPORT

Firstiafina Tiffany*, Ira Ristinawati, Shafira Nur Hanifa

*Correspondence: firstiafina.tiffany@gmail.com Department of Neurology, Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Indonesia

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ABSTRACT

Background: Stroke is the second leading cause of death in the world and is the leading cause of disability worldwide. One of the disabilities is language disorder. Foreign Accent Syndrome (FAS) is a clinical condition associated with a noticeable change of accent, that could affect verbal communication and social interaction skills. By identifying this case earlier, we hope that the patient's activity daily living is less disrupted.

Case: A 54-year-old woman presented with right limb weakness and tingling, along with a change in speech accent from Javanese to Madurese, despite no prior exposure to Madurese. Neurobehavioral examination revealed language and memory disturbances, and MRI showed cerebral infarction in the bilateral hippocampi, small vessel ischemia in the bilateral frontal lobes, and corona radiata.

Discussion: The accent change, known as FAS that was experienced by the patients are thought to be due to neurological causes, which was stoke. In a study using a lesion network mapping approach concluded that the lesion causing accent disorder was located within a single network in the bilateral frontal lobe. In line with the existing theory, the MRI results in this patient showed ischemia in the blood vessels of the bilateral frontal lobes.

Conclusion: Clinical symtoms related to stroke depending on the location of the lesion. The accent change in this patient, known as Foreign Accent Syndrome (FAS), is believed to be caused by stroke affecting the bilateral frontal lobes. Early recognition of FAS can help reduce disruptions in communication and social interactions.

Keywords: foreign accent syndrome, neurobehavior, stroke infarction



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Introduction

A stroke is a neurological deficit caused by acute focal injury to the central nervous system due to vascular causes, especially cerebral infarction. There are two types of stroke: hemorrhagic stroke and ischemic stroke.¹ The Trial Organization 10172 in Acute Stroke Treatment (TOAST) categorizes ischemic strokes into some number of subgroups, including cardioembolism, small vessel occlusion, major artery atherosclerosis, and strokes with unknown causes. In 2019, stroke being the second-leading cause of death globally, count for 11.3% of all cause of deaths, and the third-leading cause of morbidity and mortality. Disability-adjusted life years (DALYs) increased by 32% and stroke mortality by 43% between 1990 and 2019.² One of the disabilities that was caused by the stroke is language disorder.³ Language disorder can manifest as fluently disorder, language comprehension disorder, repetition, item naming, reading, or writing.⁴ 25% to 40% of stroke survivors experience language disorder.³ Language disorder associated with more severe stroke, which makes post-stroke language disorder a significant disability for patients and a risk factor for high mortality. Additionally, people with language disorder are more likely to have depression and lower chance of going back to work, all of which have a detrimental impact on their quality of life.⁵

Good sentence structure and understaneable accent are crucial elements of language, as these two factors could affect the sentence's comprehension that would accepted by the interlocutors.⁶ Variations in pitch, pressure, and rhythm in accented speech; articulation; slow production of speech can give words emotional meaning, in which this components may be disrupted in patient with Foreign Accent Syndrom (FAS).⁷ FAS is a clinical condition associated with a noticable change of accent. Foreign accent impression in patient with neurogenic FAS was resulted from a misinterpretation by the listener.⁸ This condition being

Case Report

A 54-year-old woman complained of tingling and weakness in her right limbs as well as a change in her language accent when she visited Dr. Moewardi at the RSUD Neurology Clinic. Before the stroke attack, the patient was still able to move, talk, and carry out daily activities. The patient's right limb began to weaken 4 years after the stroke attack. The patient had a Javanese accent when speaking before having a stroke. One year later, the patient, who had never lived in Madurese or learned the language before the stroke, began speaking with a Madurese accent. The patient has reported experiencing tingling for a year following their stroke, although these concerns have started to go better. The patient had a history of hypertension and consistently took medication before the stroke attack. The patient denied having undergone surgery or experienced any prior trauma.

a major concern for the patient, because it could affect their verbal communication and social interaction skill.⁹ In fact, FAS in patient with the history of stroke could seriously impair their verbal communication and social interaction skill.¹⁰ While studies on FAS after stroke are relatively scarce, those that are accessible focus more on language disorders in post-stroke patients.

On the physical examination, the consciousness level was E4V5M6 and the blood pressure was 152/106 mmHg. On the physical examination, right-limb weakness was found. The patient speaks fluently; there is no repetitiveness, they can name objects, and he understands phrases well. However, patients may struggle to communicate what they want to say. Additionally, a motor assessment revealed no paresis, paralysis, or apraxia. The patient was determined to have issues in the language and memory domain during the neurobehavioral evaluation. A brain infarction in the hippocampal region and small vessel ischemia in the frontal lobe and corona radiata were both seen on magnetic resonance imaging (MRI) (Figure 1). Examination of electrolytes, electrocardiography, and blood sugar within normal bounds. Candesartan 1x8 mg, cilostazol 1x100 mg, piracetam 1x800 mg, and citicolin 1x500 mg were given to patients as part of their treatment.

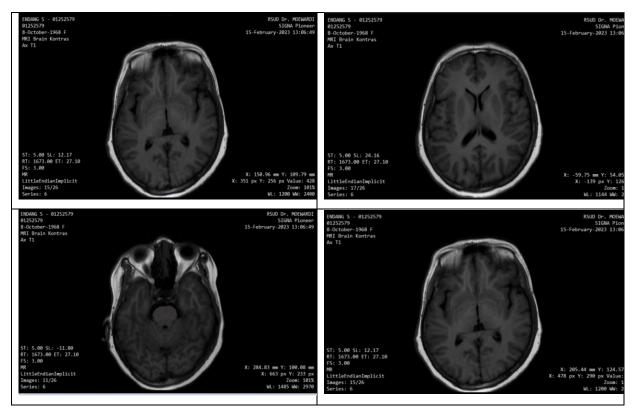


Figure 1. MRI on T1 slice of the patient

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Figure 2. MRI on T2 slice of the patient

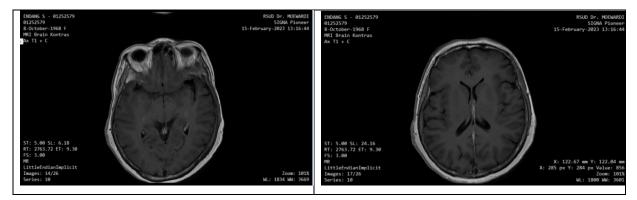


Figure 3. MRI on T1+C slice of the patient

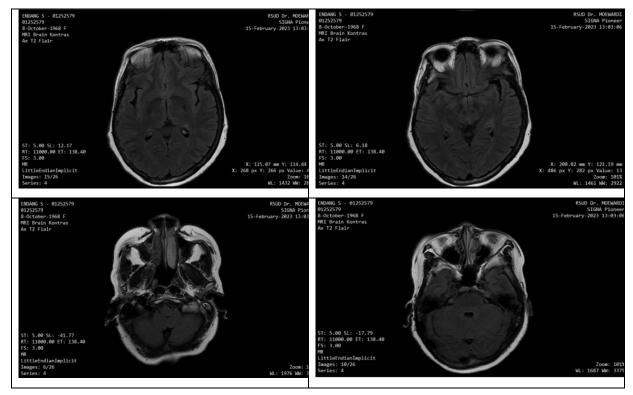


Figure 4. MRI on T2 FLAIR slice of the patient

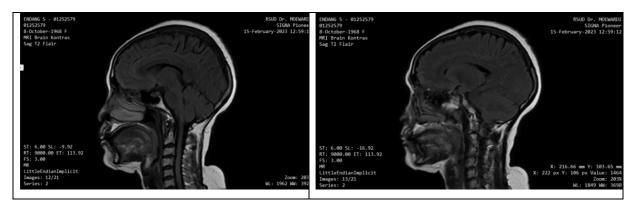


Figure 5. MRI on T2 FLAIR slice of the patient

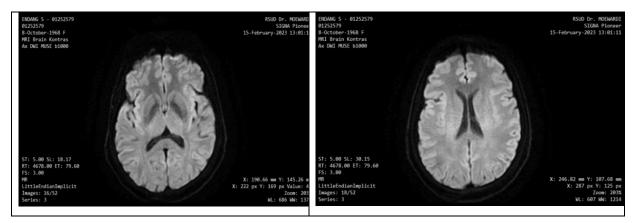


Figure 6. MRI on DWI slice of the patient



Neurobehaviour Clinic

RSUD Dr. Moewardi

KSM/Neuroscience Laboratory

Jl. Kolonel Sutarto 132 Solo

Telp 0271 634 634

Neurobehaviour Examination Test Result

Name	Ny. E	Examination Date	11 May 2023
Age	55 y.o.	No RM	01252xxx
Address	Sragen		
Occupation	Housewife		

Anamnesis Patients complain that they occasionally still forget to put things away, but they claim that this is better than it was two years ago. According to the patient, accent language disorders like the Madurese continue to alter. History of strokes in 2019.

No	Test	Score	Normal	Description
1	Global Cognition Function 1. MoCA-Ina	24	≥ 25.8	less
2	Atention 2.1. Forward Digit Span	8	5	normal
3	Language 3.1. Boston Naming Test 3.2. Verbal Fluency Test	11 16	13.84±1.23 19.84±5.98	less normal
4	Memory 4.1. Word List Memory Task 4.2. Word List Memory Recall 4.3 Word List Memory Recognition 4.4 Recall of Constructional Praxis	18 6 10 9	19.84±5.98 8.05±1.66 9.88±0.36 10.61±0.97	normal less normal less
5	Executive Function 5.1. Trail Making Test B 5.2. Verbal Fluency	145 16	113.48±58.48 19.84±5.98	normal normal
6	Visuokonstruction 6.1. Constructional Praxis	11	10.61±0.97	normal
7	Language Comprehension Test 7.1. Token Test	30	>29	normal
8	Lawton IADL Score	8	8	normal

Conclusion
The current neurobehavior examination found disturbances in the global cognition function domain, language subdomain and memory subdomain. Impressions towards Vascular Cognitive Impairment.

Figure 7. Neurobehavior Examination Test

Discussion

There are modifiable and non-modifiable risk factors for ischemic stroke. History of diabetes coronary heart disease, history mellitus, of hypertension, socioeconomic status, lifestyle, and history of alcohol use are lists of factors that could be modified. While age, gender, and race are risk factors that cannot be modified.¹¹ The most frequent risk factor affecting the incidence of stroke is hypertension, both in young-onset (age group less than 50 years) and in old-onset.¹ When the patient experiences hypertension, the increase in intraluminal pressure will eventually damage the endothelium. Blood and endothelium interact due to endothelial injury, resulting in systemic lesions and localized thrombus development.⁷ The patient has hypertension, and according to the anamnesis, the patient consumes antihypertension that was given by the doctor regularly. However, when the patient went to the doctor and the blood pressure was measured, it was found that the patient's blood pressure was 152/105 mmHg, which was classified as type I hypertension. Clinical symptoms that were found in stroke patients differ according to the location of the lesion.

In this case, the patient experienced right limb weakness following a stroke 4 years prior, along with speech difficulties. One year after the incident, patient was able to talk clearly. The patient, who had previously spoken with a Javanese accent, suddenly switched to a Madurese accent, which the patient had never learnt before.

FAS is a relatively rare motor speech disorder characterized by speech errors which are perceived as a foreign accent by members of the same language community as the patient.¹² FAS disorders or accent change are typically caused by neurological or biological factors, although it can also be caused by psychological factors (such as functional or psychogenic disorders).⁸ The most common etiology of FAS is stroke, followed by head trauma, metastatic brain tumor, multiple sclerosis, progressive degenerative brain disease, including primary aphasia, learning progressive disorders and psychogenic disorders, in the intermediate period ranging from about three weeks to three months postonset (Figure 2).¹² More than 70% of cases are caused by neurological factor, resulting from stroke, and the majority of these conditions are related to supratentorial lesions in the right hemisphere (Figure 3).⁹

Neurogenic FAS commonly related to combination of segmental and suprasegmental pronounciation characteristics which can be deviate from what is expected on the basis of the speech community to which the speaker belongs. At segmental level, the problem related to the phonetic distortions and phonemic paraphasia.¹³ At the suprasegmental level, neurogenic FAS often noted to have change in speech rhythm which is described as slow or problem with stress placement, different, isosyllabic, staccato, scanning or syllable-timed (Figure 4).¹⁴ The morphology of the lesion remaining the same, neurobehavioral symptoms in the earlier period (the acute phase; until approximately 3 weeks post-stroke) are often more severe than the lesion would suggest (due to the additional effect of diaschisis affecting ipsi- or contralateral brain areas, mass effect and peri-lesional damage (penumbra) (Figure 5). The effects of damage also depend on the affected brain area and they may become less severe in the later period (the chronic phase; after approximately 3 months post-stroke) due to the opposite effect of functional compensation resulting from spontaneous recovery or therapy. Neurocognitive and neurolinguistic disorders presumably mirror the effect of the lesion most faithfully in an intermediate period called the lesion phase of the stroke (Figure 6).¹²

Preservation of speech intelligibility could be the pathognomonic feature to differentiate the organic type from the functional type. It was shown from the case report presenting two types of foreign accent syndrome, organic and functional type, the patient with the organic type has a score of 95.5 from speech intelligibility rating compared to the functional type with a score of 55.3.8 In a study that used "lesion network mapping", it was discovered that the lesion responsible for accent disorder share common functional network located in the bilateral posterior region of the frontal lobe.⁷ Several areas in the frontal lobe that predominantly contribute to the emergence of accent disorders: bilateral middle portion of the precentral gyrus, the bilateral lower portion of the precentral gyrus extending to the insular cortex, and the supplementary motor area (SMA).^{7,12} Medial part of the pre-central gyrus is a motor cortex area, which has a specific role in controlling larynx intrinsic muscle.7

Larynx was the phonation organ, which consist of two groups of muscle, namely intrinsic and extrinsic larynx muscle, that regulate the vocal folds. The intrinsic muscles control 2 dimensions of vocal-fold movement to modify the positioning and tension of the vocal folds. One dimension involves the opening (abduction) and closing (adduction) of the glottal space, while the other involves tensing and relaxing of the vocal folds for the purpose of altering vocal pitch (F0 variation) and vocal intensity. Control of vocal pitch, corresponding to the vocal fold eigenfrequencies, is achieved mainly by varying the stiffness and tension of the vocal folds through the activation of the intrinsic laryngeal muscles, especially the cricothyroid muscle. The intrinsic muscles also increase vocal intensity, either elevating the subglottal pressure, which increases vibration amplitude, or increasing vocal fold adduction.^{7,13} Other than that, this region play a role in vowel phonation and glottal stop (i.e., forced closure of the glottis in the absence of vocalizing.¹³

According to these characteristics, FAS characterized by altered normal pitch and intensity may be attributed to intrinsic larvngeal muscle dysfunction controlled by the medial pre-central gyrus.¹⁵ The Rolandic operculum, which consists of the inferior precentral gyrus and the post-central gyrus, is involved in lip and tongue movement.¹⁶ The Rolandic operculum is an important area for intra- and inter-syllable coordination of complex articulation movements.¹⁷ Supplementary Motor Area (SMA) located in the medial part of the frontal lobe anterior. Accent disorder could result from SMA lesions. Additionally, it is known that damage to the SMA, particularly the left portion, can result in SMA aphasia, a peculiar aphasia that is characterized by a lack of spontaneous speech initiation and well-maintained articulation when speech is began. SMA also regulates behavioral functions, internal driven activities, sequential activities, the process of learning new tasks, and cognitive processes in addition to language functions.^{17,18} The patient's accent changes, from Javanese to Madurese, were made possible by interference in the bilateral frontal lobes, in accordance with the MRI findings, which showed the presence of ischemia of the small vessels in the patient's bilateral frontal lobes. The patient's MRI findings are consistent with those of the neurobehavioral test, which revealed an impairment on language domain.

Patient with a history of stroke could also have a cognitive impairment. In a study stated that the prevalence of cognitive defect in patient with a history of stroke between 11.6% and 56.3%. 38% of them were assessed to have a cognitive defect three monts post stroke using MMSE scoring. Dominantly, the patients which have cognitive impairment had lesions in frontotemporal areas. Some of patients with cognitive impairment post stroke had a problem witth arithmetics. Arithmetics requires intact short- and long-term memory functions, attention, and mental flexibility and is subserved by mainly parietal and pre-frontal cortex. Basal ganglia lesions, attested for, give rise to similar cognitive impairments as frontal lesions due to a network of fronto-subcortical loops. The basal ganglia are implicated in a wide variety of cognitive functions, including executive functions, attention, visual perception, sequential processing and learning-which were variably disrupted in-but also speech. These behavioural effects are demonstrated in vascular FAS cases with basal ganglia damage.¹³

FAS to disruption of the cortico-striatopallidalthalamic pathway, consisting of many regions that cooperate in an integrated fashion to regulate many different cognitive functions.¹⁹ Although many issues must be resolved, this circuit is clearly involved in various speech and language processes. Lesion in hippocampus presents as an anterograde amnesia, but is not always associated with retrograde amnesia, and old memories are often retained. Widespread damage to the medial aspect of the temporal lobe, such as the hippocampus and parahippocampal gyrus, impairs retrieval of old memories and results in severe amnesia.18,20 backward According to the neurobehavioural test result, the patient having any disturbance in the global cognition function domain, especially the memory subdomain. In line with the MRI result, stated that the patient has an infarct in the hippocampal region and small vessel ischemia in the frontal lobe, which these areas responsible for memory function (Figure 7).

Conclusion

Clinical symptoms related to stroke depending on the location of the lesion. In this case, the patient had speaking difficulties after a stroke. One year later, the patient started to talk clearly, but her speaking accent had changed. The existence of ischemia in the blood vessel of the frontal lobes bilateral, where these findings are congruent with the patient's MRI results, allowed for the shift in accent in this case. FAS are still infrequently recognized. In fact, FAS in post-stroke patients can seriously hinder verbal communication and social interaction.

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