Case report

Pronounced isolated severe apraxia of speech, a rare presentation of an acute ischaemic stroke

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A 39-year-old previously healthy woman presented with sudden onset loss of speech. Except for subtle right facial asymmetry, no other neurological deficits were found. A non-contrast CT scan was unremarkable. She was thrombolised with iv alteplase (0.9mg/kg standard dose) after 2 hours from the onset.

The facial asymmetry improved over 24 hours, post thrombolysis. Her motor, sensory, coordination and cranial nerve examination were normal. She was having severe difficulty in production of speech but was able to comprehend and write normally. She could cough and produce simple monosyllable sounds and repeat the monosyllable sounds. Mild buccopharyngeal apraxia was noted in blowing. However, when she attempted to speak, it was very effortful.

Her MRI brain, which was performed after 10 days revealed T2W, FLAIR, DWI hyperintensity in left inferior frontal gyrus. However, no restricted diffusion in ADC or contrast enhancement was noted. This T2 shine through in the left inferior frontal gyrus confirmed a sub-acute infarction (Figure: MRI Brain T2W, FLAIR, DWI, ADC). Magnetic resonance angiogram of neck and brain vessels did not show any occlusions.

She was normotensive, serum cholesterol was elevated and had a normal HbA1c. ESR, CRP, complete blood counts and blood picture were normal and ANA, HIV antibodies, and VDRL were negative. Her ECG, 24-hour Holter and transthoracic echocardiogram were unremarkable. No right to left shunts were detected in transoesophageal echocardiogram. Genetic thrombophilia screening tests were negative for factor V Leiden mutation, Prothrombin 20210 gene mutation and Methylenetetrahydrofolate reductase (MTHFR) gene mutation.

Discussion

This woman presented with subtle right lower facial weakness and a speech problem as an acute ischemic stroke, and she was thrombolised in 2 hours. Her MRI brain revealed a subacute infarction in the left inferior frontal gyrus.

She had normal bulbar muscle function with normal swallowing and had normal cerebellar functions. She was not abulic since her activities were normal except for speech. Her speech disorder was considered as apraxia of speech (AOS) as her verbal output was impaired with intact repetition, and mild orobuccal apraxia.

Apraxia of speech (AOS) has emerged as the term to describe a motor speech disorder characterized by an impaired ability to coordinate the sequential, articulatory movements necessary to produce speech sounds (Wertz et al., 1984). Confusion in the literature around AOS stems from the fact that terminology associated with this disorder has varied greatly. Also, symptoms associated with AOS often co-occur or overlap with those caused by neuromuscular deficits indicative of the dysarthrias and the linguistic errors associated with aphasia. AOS is, however, a distinct motor speech disorder. Apraxia of speech is a relatively a new terminology, which is characterised by inability to motor programming and coordinating the sequence of articulatory movements[1]. The general term “apraxia” was introduced by Liepmann in 1908 and defined it as the inability perform motor tasks without impairment of muscle strength[2]. Darley in 1969 coined the term apraxia of speech for the Liepmann’s notion of “apraxia of the glosso-labio-pharyngeal structures”[3].

A similar speech disorder with presence of preserved language skills and unimpaired muscular function was described by Paul Broca in 1861. He named it as aphemia, and he localised it to a cerebral lesion.

It is important to differentiate AOS from a language disorder (aphasia) and dysarthria due to muscle weakness or incoordination. Although our patient had mild facial weakness, it fully recovered in 24 hours but her speech impairment persisted. Her comprehension and written language were preserved, so as other orobulbar muscle functions.

Neuroanatomical localization of the AOS is difficult but many studies have demonstrated a lesion in the left frontal cortex. The commonest cause of this rare phenomenon is considered to be vascular lesions.

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Tumours, trauma, and neurodegenerative disorders such as primary progressive aphasia and corticobasal degeneration are other causes[5,6].

Functional speech disorders also could mimic AOS, and this could be extremely challenging especially when in therapeutic decision making in neurological emergencies like hyperacute ischemic strokes like in our case[7].

In mildly apraxic patients, poor prosody may be the primary speech deficit and, therefore, goals designed to improve intonation and stress may be the most appropriate. Treatment of moderate and severe AOS is mainly considered to be relearning oral postures for individual speech sounds[5]. We now understand AOS to be a unique speech disorder that is distinct from other speech and language deficits such as dysarthria, aphasia or stuttering.

Our patient's stroke work up was negative for cardiac and large vessel studies and genetic thrombophilia screening. She was started on antiplatelets and moderate intensity lipid lowering treatment. She made a marked improvement in post stroke one month following speech and language therapy.

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