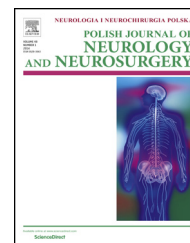


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Case report

Pure apraxia of speech due to infarct in premotor cortex

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ABSTRACT

Apraxia of speech (AOS) is now recognized as an articulation disorder distinct from dysarthria and aphasia. Various lesions have been associated with AOS in studies that are limited in precise localization due to variability in size and type of pathology. We present a case of pure AOS in setting of an acute stroke to localize more precisely than ever before the brain area responsible for AOS, dorsal premotor cortex (dPMC). The dPMC is in unique position to plan and coordinate speech production by virtue of its connection with nearby motor cortex harboring corticobulbar tract, supplementary motor area, inferior frontal operculum, and temporo-parietal area via the dorsal stream of dual-stream model of speech processing. The role of dPMC is further supported as part of dorsal stream in the dual-stream model of speech processing as well as controller in the hierarchical state feedback control model.

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1. Introduction

Apraxia of speech is a disorder of motor speech planning characterized by slow speech rate, segmentation of syllables, sound distortions, distorted substitutions, trial-and-error articulatory movements, and increased difficulty with increased length and complexity of utterances. It is termed Aphemia in its most severe form. It is distinct from Broca's

aphasia given intact repetition, grammar, syntax and writing. It is also distinct from transcortical motor aphasia (TCMA) since articulation is normal on repetition in TCMA. Aphasia may co-occur with aphasia so AOS has been divided into two types – pure AOS or AOS with aphasia. The earlier neuroanatomic correlations in patients with AOS had an infarction in multiple structures around the left inferior frontal gyrus including the adjacent motor cortex, deep white matter, and insula; however these studies had a limited sample size and imaging resolution on CT scans [1]. Further studies on AOS

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36 involving a large number of patients and improved imaging
 37 technique like MRI, emphasized the role of the insular region
 38 [2]. However, this “lesion overlap” approach was criticized and
 39 failed to explain the absence of AOS in all insular strokes.
 40 Attempts to explore such neuroanatomical correlation be-
 41 tween insula and AOS found no significant association,
 42 instead demonstrated either structural damage or hypoperfu-
 43 sion in left posterior inferior frontal gyrus on functional
 44 imaging; and few cases had lesions only in precentral and
 45 postcentral gyrus [3]. A larger study involving stroke patients
 46 with pure AOS demonstrated isolated infarcts in the premotor
 47 cortex (PMC) and adjacent motor cortex [4]. This line of
 48 evidence reconciled with studies of neurodegenerative AOS
 49 where PMC and supplementary motor area (SMA) have been
 50 implicated [5]. We present a case of pure AOS with a lesion in
 51 dorsal premotor cortex (dPMC) and adjacent motor cortex; and

discuss lesion evidence in support of dPMC in speech planning
 and coordination.

2. Case presentation

A 60-year-old right-handed man with past medical history of
 ischemic cardiomyopathy with systolic heart failure, hyper-
 tension, hyperlipidemia, and gout presented with sudden
 onset of slurred speech and right arm weakness. Exam showed
 left upper motor neuron facial palsy and left arm weakness
 mainly in forearm extensors and wrist extensors. MRI of brain
 showed an acute infarct in left dorsal premotor cortex and left
 motor cortex. The infarcts in other areas included the left
 prefrontal cortex (Fig. 1) and left cerebellum (not shown) were
 chronic with no restricted diffusion. The etiology of infarct was

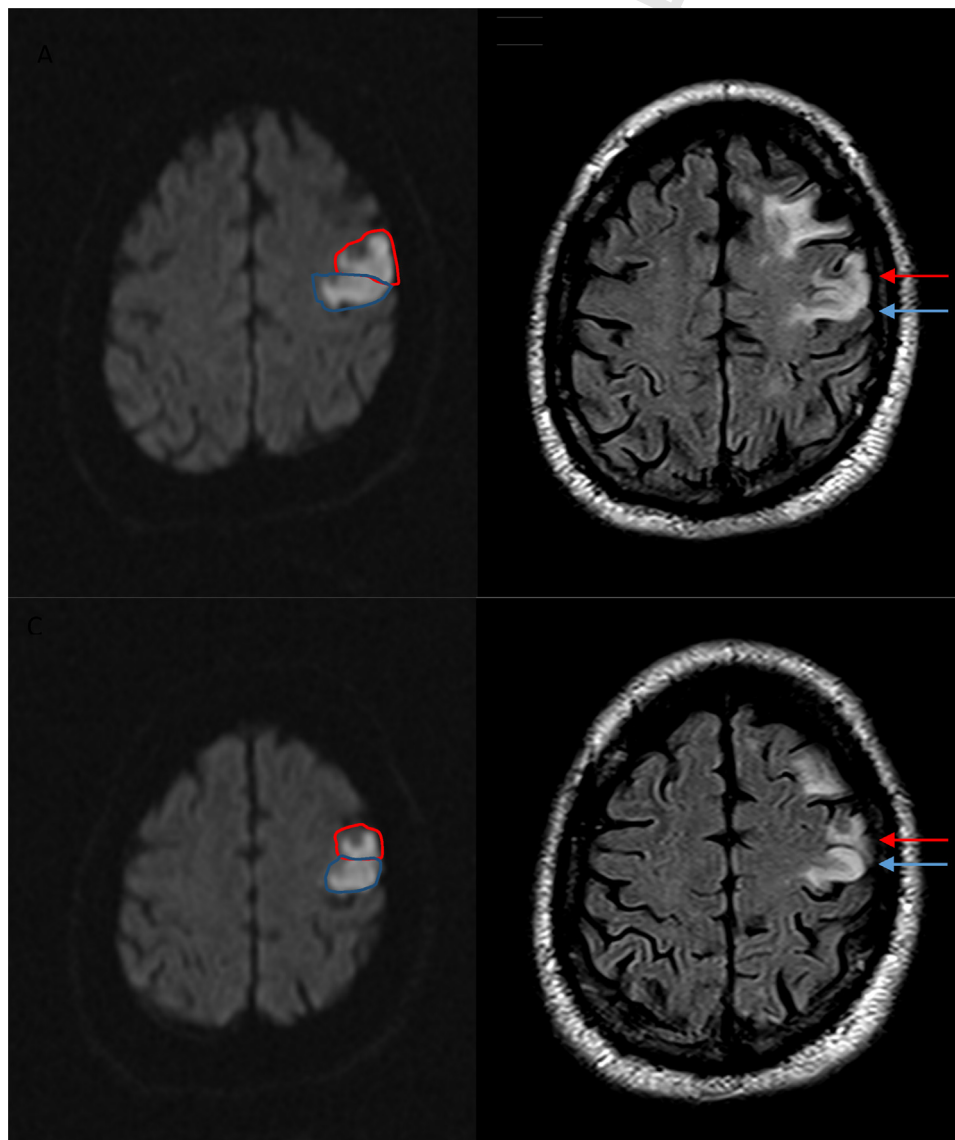


Fig. 1 – MRI Brain. (a) and (c) DWI sequence showing restricted diffusion in left dorsal premotor cortex (outlined in red) and left cortical motor cortex (outlined in blue). (b) and (d) FLAIR sequence showing infarcts in left prefrontal cortex (arrow head), left dorsal premotor (red arrow) and left motor cortex (blue arrow). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

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