A case of crossed aphasia with apraxia of speech

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Abstract

Apraxia of speech (AOS) is a rare, but well-defined motor speech disorder. It is characterized by irregular articulatory errors, attempts of self-correction and persistent prosodic abnormalities. Similar to aphasia, AOS is also localized to the dominant cerebral hemisphere. We report a case of Crossed Aphasia with AOS in a 48-year-old right-handed man due to an ischemic infarct in right cerebral hemisphere.

Key Words

Apraxia of speech, crossed aphasia, crossed apraxia of speech, right-handed

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Introduction

The term "Apraxia of speech" (AOS) was coined by Darley in 1969, and he defined it as "a disorder of motor speech processing, manifested primarily by errors of articulation".[1] It is an articulatory disorder, occurs due to impaired ability to programme the positioning and the sequencing of muscle movements for the volitional production of phonemes, without any significant weakness, slowness, or incoordination in reflex and automatic acts. AOS is rarely present in its pure form.[2,3] It commonly coexists and is often indistinguishable from Broca's aphasia and dysarthria. The features suggestive of AOS are irregular articulatory errors, attempts of self-correction, and persistent prosodic abnormalities.

Similar to aphasia, the AOS in right-handed individuals is typically localized to the left cerebral hemisphere.[3-14] The concept of crossed apraxia of speech (CAS) was 1st introduced by Balasubramanian and Max in 2004.[13] We report a case of crossed aphasia with AOS in a 48-year-old right-handed man due to an ischemic infarct in right cerebral hemisphere.

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Case Report

A 48-year-old right-handed man presented with sudden-onset difficulty in speaking and left-sided weakness. He was able to understand what was spoken to him and was communicating using gestures. There was no difficulty in chewing or swallowing. He was a chronic smoker and was diagnosed with chronic obstructive pulmonary disease 6 months back. There was no history of any other medical comorbidity. His general physical examination revealed absent right carotid, brachial and radial pulses; with feeble other peripheral pulses. Blood pressure was not recordable in right upper limb, 100/60 mmHg in left upper limb, 130/100 mmHg in left lower limb, and 134/110 mmHg in right lower limb. He was conscious, alert, and attentive. Neurological examination revealed differential left hemiparesis (upper limb Medical Research Council (MRC) 0/5 and lower limb MRC 2/5) with ipsilateral facial palsy. He was producing only few incomprehensible sounds, with intact verbal and reading comprehension; and was able to communicate using gestures. Despite being right-handed with no motor weakness in the right hand, he could not write. [Figure 4a] He was able to copy simple figures but not able to draw a clock. He was also having ideational (buccofacial and limb) and ideomotor apraxia, but no dressing apraxia, right-left confusion, or finger anomia. Rest of the cranial nerves including bulbar examination and tongue movements were normal. There was no family history of left handedness or ambidexterity. After establishing strong right-hand dominance by Edinburgh Handedness Inventory, we made a provisional diagnosis of the left hemiparesis with Crossed Broca's aphasia with AOS, with ideational and ideomotor apraxia.

His routine investigations including complete blood count, blood sugar, lipid profile, liver, and renal functions were

normal. His abdominal ultrasound was normal; 2d-Echo showed concentric left ventricular hypertrophy with type-I diastolic dysfunction and High Resolution Computed Tomography (HRCT) thorax which showed bilateral emphysematous changes.

His magnetic resonance imaging (MRI) brain showed acute ischemic infarct in right peri-insular, fronto-temporal, and periventricular white matter including centrum semiovale [Figure 1a,b]. Positron emission tomography (PET) scan revealed hypometabolism in the right cerebral hemisphere and opposite cerebellum [Figure 2]. In the magnetic resonance MR arteriogram, the right brachicephalic trunk, vertebral arteries, common and internal carotid arteries could not be visualized; there was focal stenosis in the left proximal subclavian artery; however, the celiac, superior mesenteric and renal arteries were normal in their course and caliber [Figure 3a,b]. The patient was further investigated for the cause of aortic vasculopathy. (Anti nuclear antibody, Anti-neutrophil cytoplasmic antibody, Rheumatoid factor ANA, ANCA, RA factor, Venereal Disease Research Laboratory, Human immunodeficiency virus, Hepatitis B surface antigen VDRL, HIV, HBs Ag, and anti Hepatitis C virus HCV were all negative.

A final diagnosis of left hemiparesis with crossed Broca's aphasia with AOS, with ideational and ideomotor apraxia due to acute ischemic stroke in right hemisphere with Takayasu's arteritis (TA) was made. The patient was discharged on antiplatelet (Aspirin 150 mg/day) and statin (Atorvastatin 20 mg/day) along with physiotherapy and speech therapy. At 2-months follow-up, he was able to walk independently with minimal improvement in upper limb power. His speech was significantly improved and was characterized by start hesitation, reduced fluency, word finding difficulty, and dysprosody. He was able to speak short sentences (3-4 words/sentence) which were meaningful, but with some grammatical errors and phonemic paraphasia [Video]. The articulatory errors in speech, while reading aloud and writing were irregular with attempts of self-correction. [Figure 4b] He was able to name objects, could repeat simple sentences with intact comprehension. Now, he could do calculations, draw a clock with improved ideational and ideomotor apraxia. On further follow-up at 4 and 8 months, his speech showed further improvement in the aspects of start hesitation and fluency; but irregular articulatory errors, attempts of self-correction, grammatical errors, and phonemic paraphasia were still persistent [Figure 4c and d].

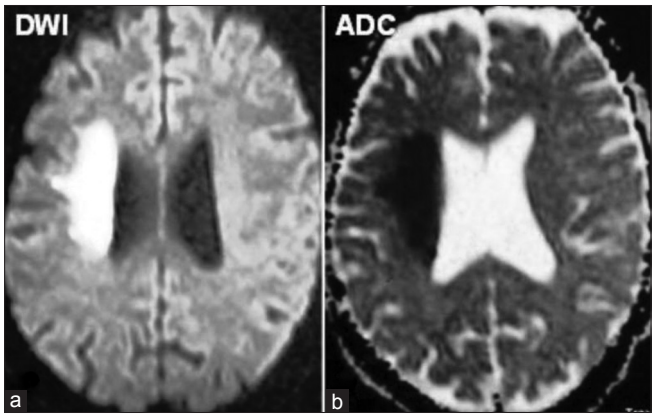

Figure 1: (a, b) Magnetic Resonance Imaging MRI brain Diffusion Weighted Imaging/Apparent Diffusion Coefficient DWI/ADC axial images showing acute infarct in right peri-insular and adjacent white matter

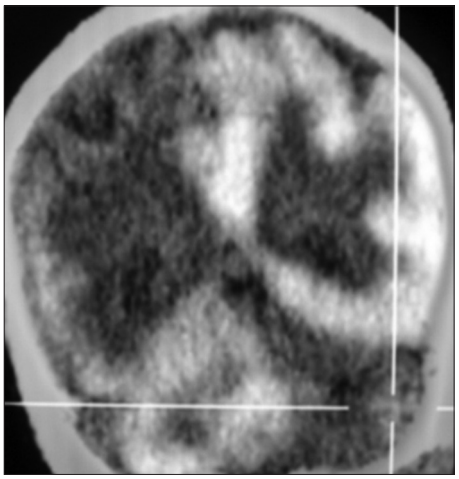

Figure 2: Positron emission tomography images showing hypometabolism in right cerebral hemisphere and opposite cerebellum

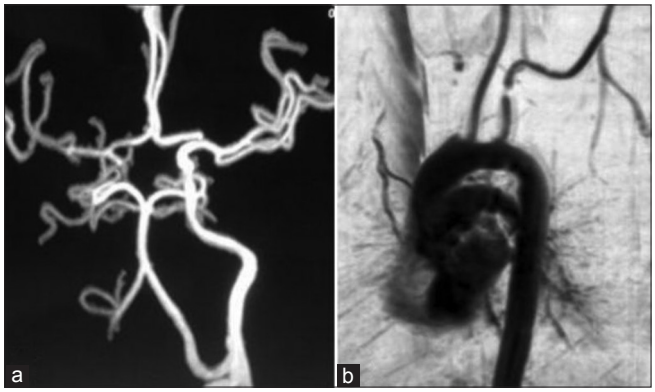

Figure 3: (a, b) Magnetic Resonance Angiography (MRA) of brain and aorta revealed non-visualization of the right brachiocephalic trunk, common carotid artery, and internal carotid artery with focal stenosis in left proximal subclavian artery

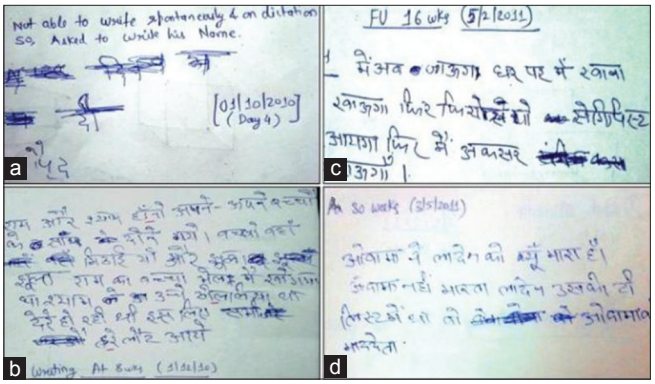

Figure 4: (a-d) Initially he was not able to write any meaningful words with attempts of self-correction. At follow-up at 8, 16, and 30 weeks, writing improved but with persistent grammatical errors, phonemic paraphasia with self-corrective attempts

Discussion

The hallmark features of AOS are irregular articulatory errors, attempts of self-correction, and abnormal prosody. The other features are difficulty in initiating utterances, articulatory inconsistency on repeating same utterance, frequent error in substitution of sounds; phonetically complex sounds are frequently affected; more errors on non-sense than meaningful words; and volitional than automatic speech.[15,16]

At the time of presentation, our patient had total inability to speak and write with intact comprehension (verbal and reading), and prominent apraxia (buccofacial and limb); which made us to keep a possibility of Broca's aphasia with AOS as a language dysfunction. The clinical picture is clearer at 2 months (intermediate phase) follow-up evaluation He fulfills all three characteristic features of AOS; irregular articulatory errors, attempts of self-correction, and abnormal prosody, which further confirmed our diagnosis of AOS as a prominent speech dysfunction. The features that suggest coexistent Broca's aphasia are presence of grammatical errors and phonemic paraphasia. The concept of lateralization of language in dominating left hemisphere in a right-handed person and Crossed Aphasia is well established.[17] Neuroanatomical localization of AOS is controversial, has been described with lesions of left inferior frontal, temporoparietal cortex, superior-anterior regions of insula fronto-subcortical white matter, internal capsule, and/or basal ganglia.[3-14]

The concept of 'crossed AOS' (CAS) was recently proposed by Balasubramanian and Max (2004) in a 69-year-old right-handed woman after a hemorrhagic stroke in the right frontal lobe.[13] More recently, Assala *et al*., (2012) described progressive CAS in a 64-year-old right-handed woman as the 1st manifestation of corticobasal degeneration, in whom MRI showed right perisylvian and insular atrophy.[14]

Our patient is right-handed by Edinburgh Handedness Inventory and clinically, he had AOS with Broca's aphasia, ideational and ideomotor apraxia which suggests the dominance of the right cerebral hemisphere, which was further established by restricted involvement of the right cerebral hemisphere on neuroimaging (MRI and PET scan). In our case, the involvement of right peri-insular and adjacent fronto-temporal subcortical white matter is most likely responsible for both AOS and Broca's aphasia. The involvement of opposite cerebellar hemisphere as evident on PET scan is likely due to the diaschisis phenomenon. In view of above findings, our patient is a possible case of crossed Broca's aphasia with CAS due to acute ischemic stroke in the right hemisphere involving peri-insular and adjacent subcortical white matter.

The natural history of patients with AOS is not well described. We followed-up our patient up to 8 months. He showed some initial improvement in fluency, start hesitation and word finding; but irregular articulatory errors, attempts of self-correction, dysprosody, grammatical errors, and phonemic paraphasia were persistent.

Also, our patient fulfills the diagnostic criteria of TA (American College of Rheumatology ACR 1990). It is a chronic granulomatous vasculitis of medium-large sized arteries, especially aorta and its branches, usually seen in women, in their 2nd-3rd decades. Neurological involvement is seen in 10-20% of cases during the course of TA; but rarely as a first manifestation.[18,19] Ischemic strokes in a middle-aged man is an uncommon first presentation of TA in our patient.

Conclusion

The overall concept of AOS and localization is now well established. A proper assessment of speech and/or language during follow-up assessment may be helpful to establish the type of disorder. Further studies are needed to establish the outcome of AOS and for comparison with prognosis of aphasic patients.

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