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Case report

Post-stroke pure apraxia of speech – A rare experience

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ABSTRACT

Apraxia of speech (AOS) is a motor speech disorder, most typically caused by stroke, which in its ''pure'' form (without other speech-language deficits) is very rare in clinical practice. Because some observable characteristics of AOS overlap with more common verbal communication neurologic syndromes (i.e. aphasia, dysarthria) distinguishing them may be difficult. The present study describes AOS in a 49-year-old right-handed male after left-hemispheric stroke. Analysis of his articulatory and prosodic abnormalities in the context of intact communicative abilities as well as description of symptoms dynamics over time provides valuable information for clinical diagnosis of this specific disorder and prognosis for its recovery. This in turn is the basis for the selection of appropriate rehabilitative interventions.

1. Introduction

In 1969, Fred Darley argued for distinguishing the term ''apraxia'' from other verbal communication neurologic disorders (aphasia, dysarthria) due to the observation of brain-damaged patients with impairment restricted to specific speech subsystems not crossing other communication modalities [1]. This impairment, called acquired apraxia of speech (AOS), is usually defined as a ''disorder of learned volitional actions associated with breakdown in planning or programming movements needed for speech'' [2]. It is assumed that problems in sequencing the spatiotemporal and force aspects of movement (muscle tone, resistance, and absolute force, direction, speed, range, and rate of motion) distort the positioning of the speech musculature in a coordinated and well-timed manner for volitional speech production [2,3]. Therefore AOS is predominantly a disorder of articulation and prosody (tune and rhythm), manifested especially in actions requiring volitional control of speech [2–4].

AOS most frequently emerges as a consequence of a stroke (93%) [5], although it is increasingly recognized in neurodegenerative diseases (e.g. motor neuron disease), and may be also a result of tumors or a trauma [6]. Pinpointing a singular structural pathology underlying AOS remains controversial. The majority of focal lesions were reported within the vasculature of left middle cerebral artery with the most common damage to the left posterior inferior frontal gyrus (inter alia to posterior Broca's area and adjacent cortex), precentral gyrus of the anterior insula, other frontal and

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temporoparietal cortex, as well as to left subcortical structures, particularly the basal ganglia [6,7].

There is limited information about the prevalence of AOS. This is partly due to problems in definition and delineation of AOS from more common speech and language disorders. In Duffy's study [8], AOS was the main cause of communication problems in 7.6% of patients with acquired neurologic motor speech disorders. However, AOS frequently co-occurs with non-fluent aphasia (linguistic disorder with agrammatism and naming difficulties which in a severe form is almost always accompanied by AOS) or overlaps with conduction aphasia (linguistic deficits with phonemic paraphasias and repetition difficulties) or dysarthria (neuromuscular disorders affecting execution of oromotor and speech functions).

In addition to accompanying communication disorders, post-stroke AOS may co-occur with contralesional hemiparesis, sensory deficits and other forms of apraxia. Among the latter, oral apraxia (sometimes called nonverbal apraxia) happens quite frequently. It affects voluntary but non-verbal movements of the vocal tract (larynx, lips, tongue, palate) either on command or during imitation, despite the appropriate strength and range of motion. Examples of tasks are: to protrude tongue, the tongue raised to the top teeth, the lips round [1].

AOS very rarely occurs in a ''pure'' form (without coexisting aphasia or dysarthria), which provides an understanding of its symptoms. Isolated AOS is characterized by slow rate of speech with a tendency to break words into individual syllables [4]. The most common abnormalities are: (1) trial-and-error groping of articulatory movement with attempts to self-correct, (2) persistent dysprosody in which there are no extended periods of normal rhythm, stress and intonation, (3) articulatory inconsistency on repeated productions of the same words, (4) difficulty initiating utterances [9], and (5) typical self-awareness of speech problems [1–3].

The present study aims at describing AOS symptoms and their dynamics in a patient with no other difficulties in speech and language abilities. Prospective characteristics of AOS provide valuable information for clinical diagnosis of this rare disorders and prognosis for recovery from it.

2. Case report

2.1. *Patient's demographic and clinical characteristics*

A 49-year-old right-handed man, native Polish speaker with secondary education (he worked as a professional driver), was hospitalized after having experienced sudden inability to speak and right-sided facial asymmetry. His medical history revealed a hypertension, hyperthyroidism (Graves-Basedow disease), transient ischemic attack with temporary paresis of the right upper limb five years earlier, inferior wall myocardial infarction seven years earlier, and chronic nicotine addiction. He had negative histories of psychological disorders and neurological conditions other than stroke. No prior speech or language disturbance was reported.

On admission, the patient was conscious, could not speak and even emit voice but his non-verbal responses on the yes–no questions (e.g. relating to taken medications and their doses) were quick and correct. He had minor right central facial paresis (the National Institutes of Health Stroke Scale – NIHSS for facial nerve item was scored 1) and mild right arm weakness (NIHSS motor arm item was scored 0). ECG examination revealed supraventricular tachycardia, which passed in atrial flutter after antiarrhythmic treatment and 24 h later stabilized as sinus rhythm. A CT revealed no fresh brain pathology (hypodensity or hemorrhage), apart from a small old lesion in the right occipital lobe.

The patient was treated with intravenous thrombolysis with no significant change in the clinical picture.

CT re-examination of the brain, performed on the second day of hospitalization, showed an area of vascular ischemic damage localized within the left frontal lobe, near fronto-parietal junction (Fig. 1). NMR performed a week from onset indicated a mild hemorrhagic transformation of the left hemispheric infarct located on the boundary of the left frontal and parietal lobes (Fig. 2).

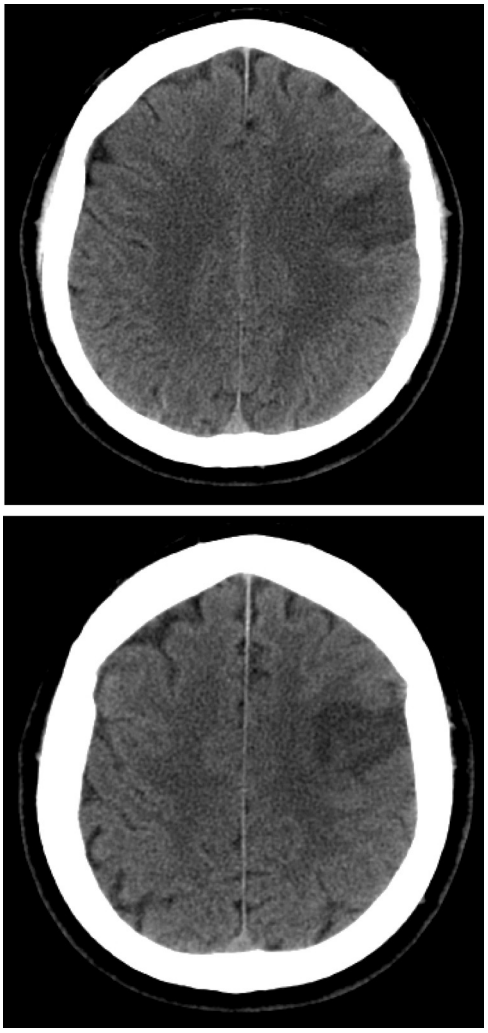

Fig. 1 – CT shows left hemispheric damage located within the frontal lobe, near fronto-parietal junction.

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