Manifestations of Stroke

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Aphasia refers to a disorder of language processing caused by a dysfunction in specific brain regions. It is common after stroke and associated with relevant disability and higher mortality. Evaluation of language function (spontaneous speech, auditory comprehension, naming, repetition, reading and writing), allows classification of aphasia. Most patients present some degree of recovery. Speech and language therapy is an effective treatment for aphasia following stroke. Other approaches, e.g. pharmacotherapy, transcranial magnetic stimulation, are being investigated. Other language disorders are mentioned. Epidemiology Together with traumatic brain injury and degenerative dementias (e.g. Alzheimer disease), stroke is a major cause of aphasia. The percentage of aphasia in acute stroke patients ranges from 14 to 38% [2, 3] and this difference may be explained by different methods of evaluation (i.e. sophisticated batteries [2] vs. basic bedside evaluation [3]), time of testing after stroke onset (vascular aphasia evolves rapidly during the first hours) and diverse methods for classification. In a large recent study, aphasia was detected in 26% of patients with first-ever stroke [3]. One study that assessed patients within the first week showed that global aphasia was the most common aphasia subtype in the acute phase (32%), followed by Wernicke’s (15%) and Broca’s aphasia (12%) [2]. Aphasia is associated with relevant disability after stroke and higher mortality. Although it was suggested that aphasia was more severe in women than in men, this was not confirmed in recent studies. Moreover, aphasia was not more frequent in older patients than in younger ones in the acute stroke phase. However, the relationship between age and aphasia and its subtypes may depend on time of evaluation after stroke. Studies performed in the non-acute phase showed a predominance of fluent aphasia in older patients.

Epidemiology
Together with traumatic brain injury and degenerative dementias (e.g. Alzheimer disease), stroke is a major cause of aphasia. The percentage of aphasia in acute stroke patients ranges from 14 to 38% [2, 3] and this difference may be explained by different methods of evaluation (i.e. sophisticated batteries [2] vs. basic bedside evaluation [3]), time of testing after stroke onset (vascular aphasia evolves rapidly during the first hours) and diverse methods for classification. In a large recent study, aphasia was detected in 26% of patients with first-ever stroke [3]. One study that assessed patients within the first week showed that global aphasia was the most common aphasia subtype in the acute phase (32%), followed by Wernicke’s (15%) and Broca’s aphasia (12%) [2]. Aphasia is associated with relevant disability after stroke and higher mortality. Although it was suggested that aphasia was more severe in women than in men, this was not confirmed in recent studies. Moreover, aphasia was not more frequent in older patients than in younger ones in the acute stroke phase. However, the relationship between age and aphasia and its subtypes may depend on time of evaluation after stroke. Studies performed in the non-acute phase showed a predominance of fluent aphasia in older patients.

Evaluation
The first goal of the examination is to establish whether a patient has a language disturbance or not, and, if present, try to identify the aphasic disorder and syndrome. Although evident deficits may
de disclosed during history taking, formal assessment is often required. Moreover, non-linguistic items of the neurological examination (e.g. motor, sensitive) may help in the classification of the stroke syndrome (table 1). Knowledge of the patient’s handedness is fundamental. Exceptionally, right-handed individuals develop aphasia as a result of right cerebral lesions (‘crossed aphasia’).

Six items of the language function should be tested, namely spontaneous speech, auditory comprehension, naming, repetition, reading and writing. This can be done by bedside examination, but for detailed evaluation, several batteries (e.g. Boston Diagnostic Aphasia Examination and Montreal-Toulouse battery) are available.

Classifications
The most commonly used classifications in clinical practice are outlined in table 1 [4].

Broca’s Aphasia. It refers to a primary deficit in language output with relative preservation of comprehension. The spontaneous speech is non-fluent: slow and labored with long pauses, reduced phrase length, impaired melody and agrammatism (failure to follow grammatical rules and improper use of conjunctions, prepositions and auxiliary verbs). Patients may present recurring utterance. Although comprehension for conversations and grammatically simple sentences is preserved, patients may have difficulties with more complex sentence structures, such as passive voice. Patients have more troubles to name verbs than nouns. Broca’s aphasia is caused by lesions in the territory of the anterior division of the left middle cerebral artery (MCA) involving the Broca’s area (the inferior frontal gyrus, BA 44 and 45) and the surrounding frontal areas, underlying white matter and subjacent basal ganglia. In patients with lesions restricted to Broca’s area, the only deficit may be impaired motor planning and programming of motor speech. This disorder is called apraxia of speech, ‘Broca area aphasia’, mini-Broca or baby Broca. Even more restricted damage to Broca’s area may produce a closely related syndrome called ‘aphemia’ which is characterized by disturbance of speech without a true language defect [1].

Wernicke’s Aphasia. It is characterized by poor comprehension of words, sentences or
conversation and fluent but meaningless spontaneous speech and repetition. Jargons comprised of either real words or neologisms are common as well as semantic paraphasias (wrong words related by meaning). Patients may be unaware of the disturbance (anosognosia) and develop agitation and paranoid ideation. The condition is usually caused by a lesion in the posterior division of the MCA involving the Wernicke’s area (posterior, superior temporal gyrus, most of BA 22).

**Global Aphasia.** This syndrome is characterized by an almost complete loss of the ability to formulate speech or comprehend language, combining the deficits of Broca’s and Wernicke’s aphasia. Spontaneous speech, naming and repetition may be limited to a single preservative word or non-word utterance. In a significant proportion of patients, comprehension recovers well, changing from global to Broca’s aphasia (*Syndromenwandeln*). The damage usually involves most of MCA territory. More rarely the damage is caused by two lesions, one frontal and other parietotemporal, sparing parts of the sensory and motor cortex. These patients may have transient or no hemiplegia (‘global aphasia without hemiplegia’).

**Conduction Aphasia.** It refers to severely impaired repetition with relatively fluent, accurate spontaneous speech with phonemic paraphasias (responses phonetically related to the target word). Some patients may struggle to approximate the target phonemes (*conduits d’approche*). The vascular lesion often involves a terminal branch of the MCA. It was proposed that conduction aphasia represented one of the disconnection syndromes, due to disruption of the arcuate fasciculus which connects the anterior and posterior perisylvian language areas [5]. However, many reported patients had lesions in the supramarginal gyrus and deep parietal white matter and patients with lesions of the arcuate fasciculus did not consistently present this subtype of aphasia.

**Transcortical Aphasias.** These syndromes are characterized by preserved repetition. The motor variant has many features of the Broca’s aphasia but with normal repetition. There is involvement of the left frontal cortices above and in front of the Broca area caused by occlusion of the anterior cerebral artery (ACA) or by borderzone infarcts between the territory of the MCA and ACA. The sensory variant consists of fluent, semantic jargon, poor comprehension and good repetition. It is attributed to posterior lesions around the Wernicke area caused by infarcts in the territory of the posterior cerebral artery (PCA), posterior branches of the MCA or borderzone infarcts between the MCA and PCA. It has also been described after thalamic lesions. The mixed type, also known as ‘isolation syndrome’, has features of both motor and sensory transcortical aphasias. The lesions tend to surround the MCA territory, often in watershed areas, isolating the language areas.

**Anomic Aphasia.** This is the mildest form of aphasic syndromes, with fluent speech, preserved comprehension but with naming and word finding difficulty. This syndrome is not specific of a cerebral region, but may be present after a wide distribution of infarcts.

**Subcortical Aphasia.** The clinical features of subcortical aphasia are not uniform. Some authors divide this syndrome in two groups, anterior and posterior syndromes. Patients with the anterior syndrome, due to striatocapsular stroke, have a non-fluent, dysarthric, paraphasic speech with varying degrees of comprehension, naming and repetition. Right hemiparesis is often present. In the posterior syndrome, secondary to thalamic stroke, mutism and comprehension deficits are initially present. During recovery, spontaneous speech may be reduced, or, more rarely, fluent and paraphasic with jargons. Reading and writing may be affected or not. Some patients may present transcortical sensory aphasia. However, this categorization of subcortical aphasia may be too simplistic and different features have been reported after specific (head of caudate nucleus, tuberothalamic artery territory) subcortical lesions [6].

Aphasia and Other Language Disorders
Other Disturbances

Pure ‘Word’ Deafness. In this rare syndrome there is an impairment of auditory comprehension and repetition, but non-verbal hearing, spontaneous writing and comprehension of written language are normal. Bilateral damage of the superior temporal gyri is the probable anatomical substratum of the syndrome.

Alexia without Agraphia (Pure ‘Word’ Blindness, Pure Alexia). It is a disconnection syndrome characterized by impairment in reading but with spared writing and recognition of words spelled aloud. It results from occlusion of the left PCA with infarction of the left occipital cortex and splenium of the corpus callosum. Due to right hemianopia, all visual information is processed in the right occipital cortex and cannot be transferred to language areas in the left hemisphere because of the lesion in the corpus callosum. Some patients with this syndrome may be unable to name objects presented visually but can do it from tactile exploration.

Foreign Accent Syndrome. This is a rare disorder characterized by the emergence of new prosodic features that listeners perceive as a foreign accent and is usually due to left hemisphere lesion [7].

Recovery

Early recovery of aphasia is likely to be secondary to restoration of flow, while later stages may be linked to reorganization of structure [4]. In one study, about 40% of the patients with aphasia in the acute stroke phase presented almost complete recovery in 1 year and those who still had language disturbances presented a milder form (fig. 1) [2]. The change is always from non-fluent to fluent, never the reverse. A significant number of patients with global aphasia at admission present Broca’s aphasia later.

Treatment

A recent review supports the effectiveness of speech and language therapy for people with aphasia following stroke [8]. Studies of pharmacotherapy for aphasia have focused on four drugs:
bromocriptine, amphetamines, piracetam and donepezil. Although preliminary evidence with some of these drugs is encouraging, randomized controlled trials are necessary. Transcranial magnetic stimulation is also being investigated as a method of enhancing aphasia recovery.

References


