Chapter

Imaging of Vascular Aphasia

Loïc Duron, Augustin Lecler, Dragos Catalin Jianu, Raphaël Sadik and Julien Savatovsky

Abstract

Brain imaging is essential for the diagnosis of acute stroke and vascular aphasia. Magnetic resonance imaging (MRI) is the modality of choice for the etiological diagnosis of aphasia, the assessment of its severity, and the prediction of recovery. Diffusion weighted imaging is used to detect, localize, and quantify the extension of the irreversibly injured brain tissue called ischemic core. Perfusion weighted imaging (from MRI or CT) is useful to assess the extension of hypoperfused but salvageable tissue called penumbra. Functional imaging (positron emission tomography (PET), functional MRI (fMRI)) may help predicting recovery and is useful for the understanding of language networks and individual variability. This chapter is meant to review the state of the art of morphological and functional imaging of vascular aphasia and to illustrate the MRI profiles of different aphasic syndromes.

Keywords: aphasia, stroke, imaging, MRI, recovery

1. Introduction

Aphasia is an acquired language disorder caused by damage to language regions of the brain that can affect the ability of a person to understand and/or produce language. It is often accompanied by impairment in reading (alexia) and writing (agraphia). Aphasia is one of the most common and debilitating consequences of stroke and is associated with a higher risk of mortality, a poor functional prognosis, and an augmented risk of vascular dementia. Fortunately, some degree of recovery of language function occurs for about 70% of patients with post-stroke aphasia thanks to neural plasticity and speech and language therapy [1–3].

Brain imaging is essential for the initial diagnosis of stroke and the assessment of stroke severity and prognosis. Stroke location and extension are associated with different patterns of aphasia with diverse functional outcomes [2]. Functional imaging, such as positron emission tomography (PET), functional magnetic resonance imaging (fMRI), and magnetoencephalography (MEG), has also been extensively evaluated for its ability to predict recovery from aphasia [4].

The purpose of this chapter is to review the state of the art of morphological and functional imaging of vascular aphasia and to illustrate the MRI profiles of different aphasic syndromes.

2. Brain language areas

Aphasia is caused by a brain damage localized in one or several language areas of the left hemisphere for 95% of right-handed people and 75% of left-handed

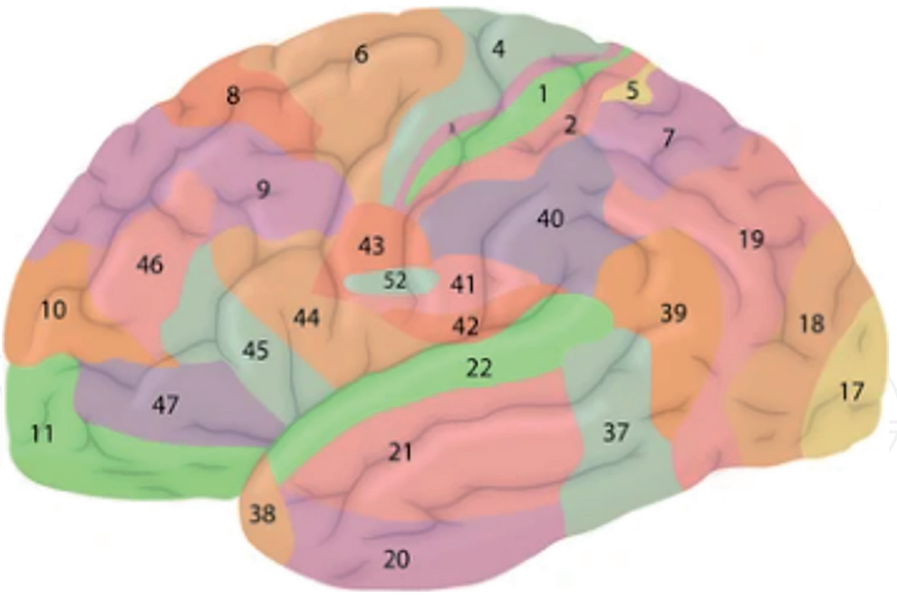

Figure 1.
Lateral view of Brodmann areas.

people. Functional neuroimaging techniques have highlighted Brodman Areas (BA, **Figure 1**) associated with language functions. They are clustered into two main language networks: (1) a language reception/understanding system, including a "core Wernicke's area" involved in word recognition (BA21, BA22, BA41, and BA42), and a peripheral area ("extended Wernicke's area:" BA20, BA37, BA38, BA39, and BA40) involved in language associations; (2) a language production system ("Broca's complex:" BA44, BA45, and also BA46, BA47, partially BA6—mainly its mesial supplementary motor area—and extending toward the basal ganglia and the thalamus. The insula (BA13) may also play a coordinating role in interconnecting these two brain language networks [5–7].

A given language impairment can result from damage or dysfunction of several different brain areas due to the impact of the lesion not only on the function of the affected region but also on the many regions connected to it within the language networks.

3. Imaging of aphasia

Brain imaging is critical to the initial management of ischemic stroke. Magnetic Resonance (MR) diffusion and perfusion imaging as well as computed tomography (CT) perfusion imaging are commonly used in clinical routine for estimation of ischemic core, penumbra, outcome prediction, and treatment decision-making in the acute stroke setting. Functional MRI is used after the initial phase to assess the severity of aphasia and predict recovery.

3.1 Ischemic core

The ischemic core corresponds to brain tissue that has been irreversibly injured and has already turned or will inevitably turn into infarction regardless of treatment [8]. It can be assessed on non-contrast CT as hypo-attenuating areas and on Diffusion Weighted Imaging (DWI) of MRI exams as hyperintense areas with decreased Apparent Diffusion Coefficient (ADC) values on ADC map. The ischemic core can also be assessed using CT or MR perfusion imaging and is currently defined as areas with

relative Cerebral Blood Flow (CBF) < 30% compared with the baseline level. However, hyperintense lesions on DWI, hypodense areas on CT, and areas with CBF < 30% on perfusion CT or MRI can be partially reversible, particularly if reperfusion is performed within 30 min, so that there is no gold standard for ischemic core imaging.

3.2 Penumbra

The ischemic penumbra is defined as an area of nonfunctioning but viable brain tissue that may recover its function if blood flow is restored. Therefore, it is the main target of reperfusion treatments. The ischemic penumbra has been widely investigated because of its potential to personalize therapeutic opportunities. It can be assessed using CT or MR perfusion and is defined as the area outside the ischemic core with a time-to-maximum (Tmax) > 6 s (**Figure 2**). A large area of penumbra associated with a small ischemic core represents a good candidate for reperfusion therapeutics such as intravenous thrombolysis and mechanical thrombectomy. This reperfusion is highly correlated with improvements on specific language tasks [1].

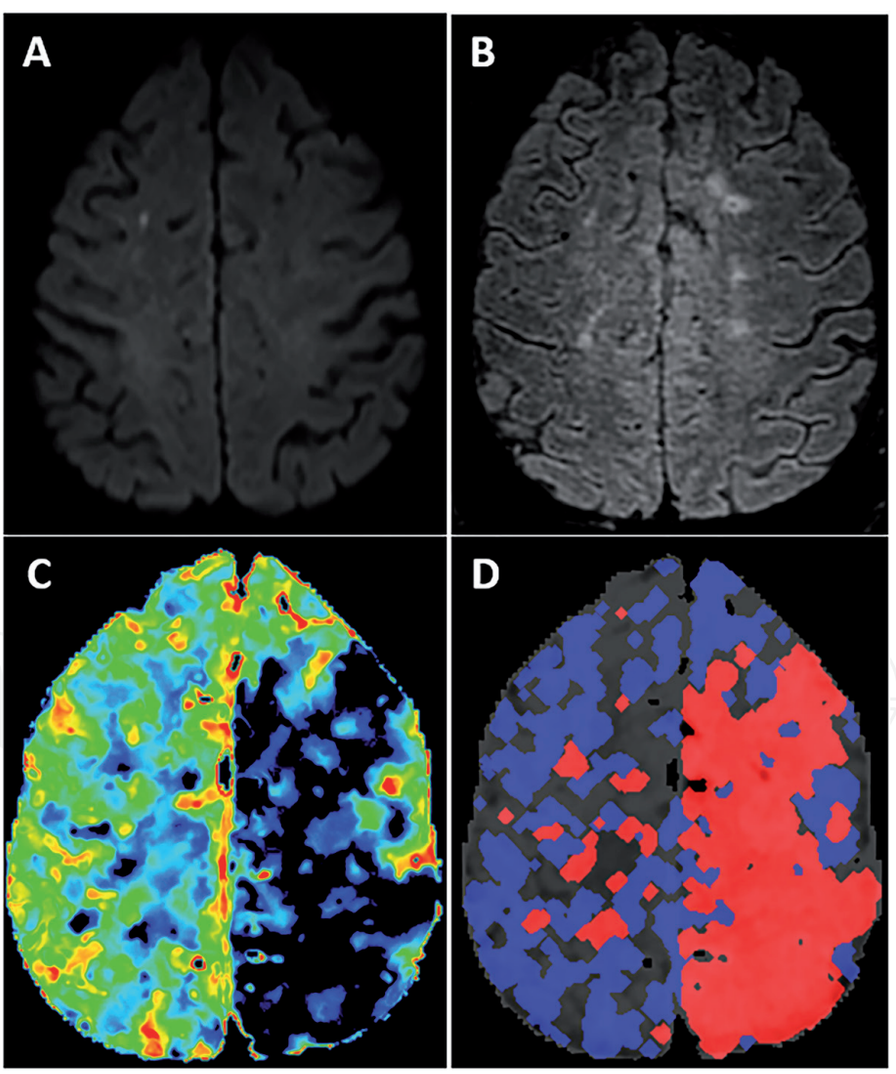

Figure 2.
Left sylvian acute ischemic stroke in a 44-year-old man with global aphasia. DWI (A) and FLAIR (B) are normal in the left sylvian area and show an isolated ischemic spot in the right sylvian area. Perfusion maps show a decrease in cerebral blood flow (C, dark blue) in the left sylvian area, with a consistent increase in Tmax > 6 s (D, red). This corresponds to ischemic penumbra, i.e., salvageable tissue that may benefit from reperfusion treatments.

3.3 Recovery prediction

Depending on the location and extent of the left hemisphere lesion, different mechanisms may concur to recovery from aphasia: (1) right hemisphere reorganization, (2) implication of residual left hemisphere language areas, (3) recruitment of left hemisphere regions not previously involved in language function, and (4) reorientation of domain-general networks not specifically dedicated to language [1, 9–11]. Intensive and targeted language therapy may interact with brain plasticity to favor recovery from aphasia.

Several neuroimaging findings were associated with aphasia severity and poor recovery, such as a large volume of ischemia, a cortical involvement, a non-fluent profile of aphasia, and a high National Institutes of Health Stroke Scale (NIHSS) score at 2 weeks [12].

Functional imaging has also been extensively investigated for its potential to predict recovery from aphasia. However, the generalizability, variability, and interpretability of group-based approaches that most imaging studies use have been criticized because of the variability in mapping function onto macro-anatomy across neurologically healthy individuals, which hinders the interpretation of results at the individual level. Therefore, the methods used in studies must be carefully validated to safely generalize the findings [13].

4. Illustration of aphasic syndromes

The location and extension of the stroke lesions are the main determinants of the aphasic profile. Eight patterns of aphasia will be illustrated below: Broca's aphasia, Wernicke's aphasia, conduction, transcortical, global, anomic, crossed, and subcortical aphasia.

4.1 Broca's aphasia

Broca's aphasia is generally produced by infarcts or severe hypoperfusion of the superior division of the left middle cerebral artery [2, 14, 15]. Brain areas involved in Broca's aphasia are classically:

1. Broca's area: the posterior part of the third frontal gyrus—BA44 and BA45. Lesions in this area determine transitory apraxia of speech. Larger lesions, extending to the subjacent white matter, produce transitory mutism, which is replaced by a rapidly improving syndrome with prominent arthric deformations and deficits in action naming that are more severe than deficits in object naming (**Figure 3**).

2. Rolandic operculum: lower part of motor area: Fa (**Figure 4**).

3. Lesions can extend or separately affect insular cortex (**Figure 5**) and subjacent white matter, centrum semi-ovale, capsule-striatum (caudate nucleus head and putamen), and periventricular areas. Infarctions involving together these structures and Broca's area may also produce the complete syndrome of Broca's aphasia.

4.2 Wernicke's aphasia

Wernicke aphasia is generally produced by infarcts or severe hypoperfusion of the inferior division of the left middle cerebral artery, which supplies the posterior

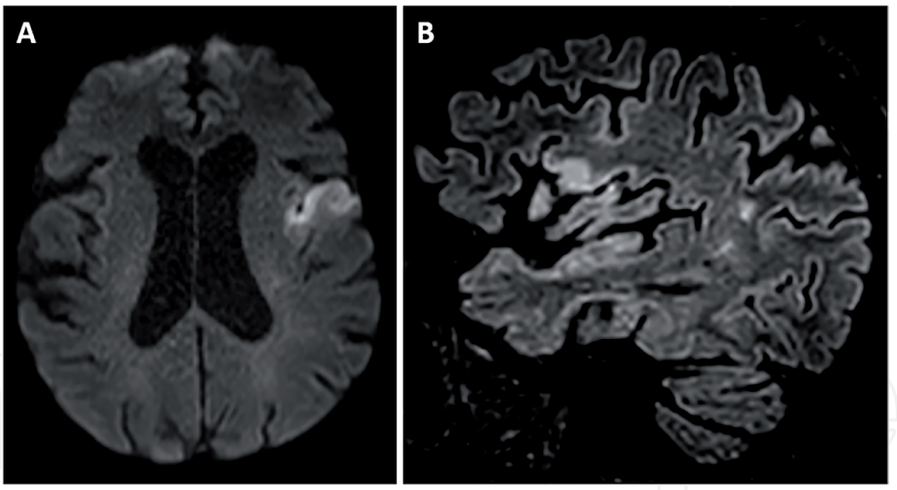

Figure 3.
Broca and left Rolandic operculum acute ischemic stroke in a 65-year-old woman presenting with Broca's aphasia, hyperintense on DWI (A) and FLAIR (B).

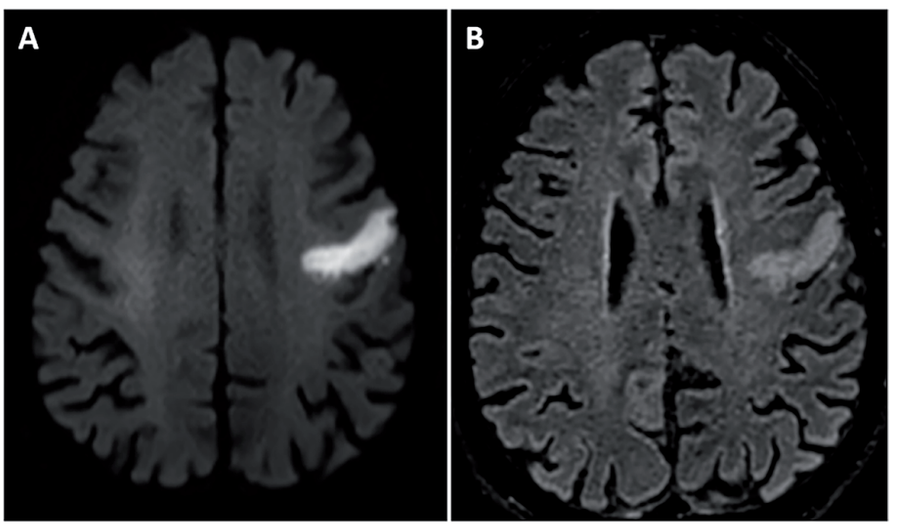

Figure 4.
Left inferior frontal gyrus acute ischemic stroke in a 82-year-old man presenting with Broca's aphasia, hyperintense on DWI (A) and FLAIR (B).

part of the temporal lobe and inferior parietal lobule [2, 14, 15]. Brain areas involved in Wernicke's aphasia are classically:

1. Wernicke's area: posterior part of the first two temporal gyri-T1/T2 (BA22) (**Figure 6**).

2. Inferior parietal lobes: angular gyrus (BA39) and supramarginal gyrus (BA40).

3. Lesions can extend to the insular-external capsule region and anterior part of temporal gyri (BA22). Besides the cortical destructions from these areas, subjacent white matter can be also affected.

4.3 Conduction aphasia

The lesions affect the inferior parietal lobes, especially the supramarginal gyrus and/or the external capsule; they classically disrupt the arcuate fasciculus, although its role remains debated for the repetition impairments: probably

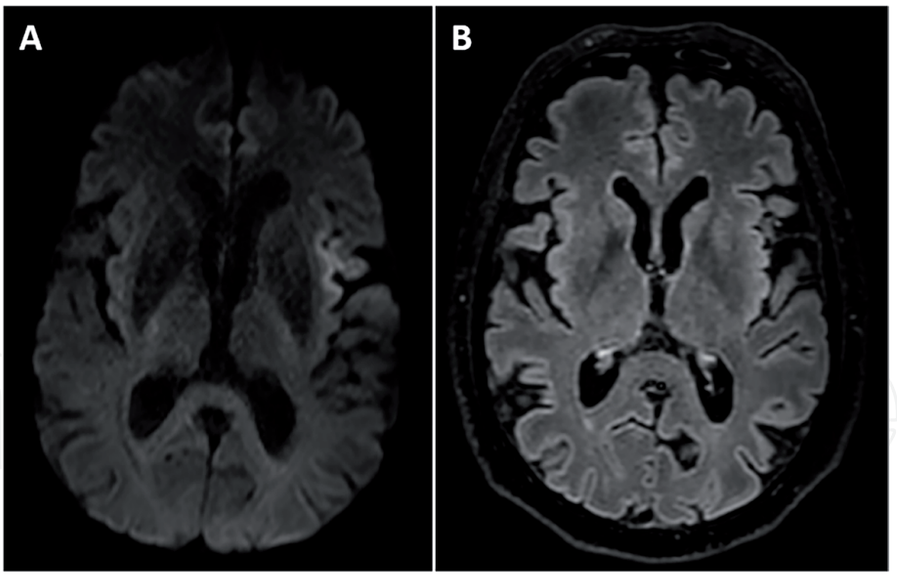

Figure 5.
Acute ischemic stroke of the left insula in a 62-year-old man with hyperacute Broca's aphasia, hyperintense on DWI (A) but still normal on FLAIR (B).

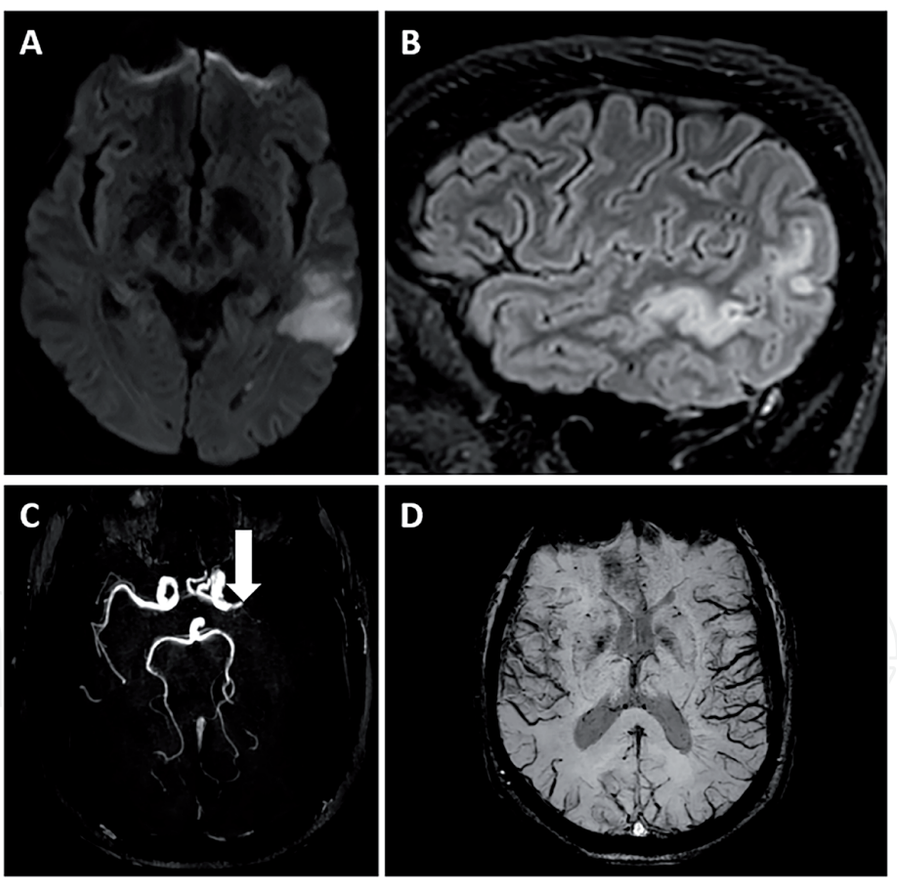

Figure 6.
Acute ischemic stroke of the left temporal gyri in a 63-year-old man presenting with acute Wernicke's aphasia, hyperintense on DWI (A) and FLAIR (B). The left middle cerebral artery is occluded (C, arrow). Dilated cortical veins are visible in the larger hypoperfused area (D), known as "cortical brush sign."

disconnection between the superior temporal cortex and the inferior frontal gyri, respectively [2, 14, 15].

Other explanations for the repetition impairments have been noted, such as short-term memory syndrome (the repetition impairment due to limited working

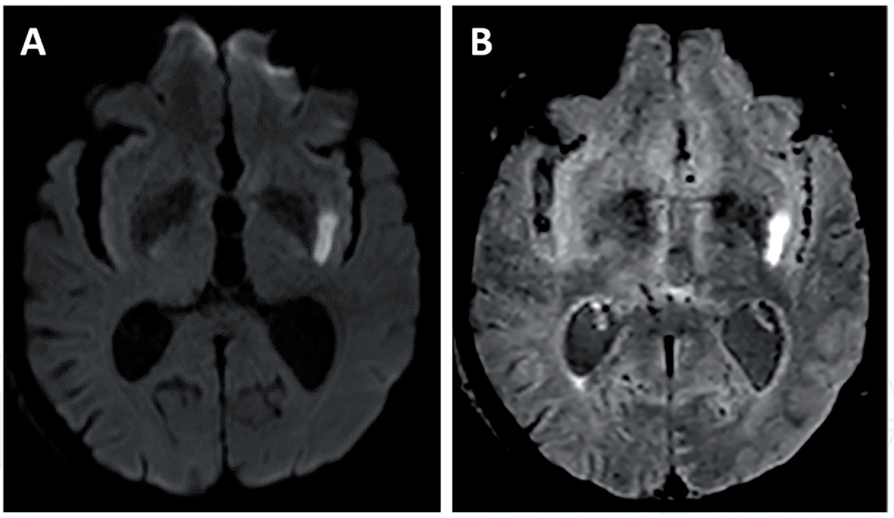

Figure 7.
Acute ischemic stroke of the left external capsule in a 88-year-old man presenting with conduction aphasia, hyperintense on DWI (A) and FLAIR (B).

memory)—so, the associated lesions are situated in areas critical for working memory: inferior parietal lobule (supramarginal and angular gyri), inferior frontal cortex, posterior temporal lobe, and/or their white matter connections (the external capsule (**Figure 7**).

Conduction aphasia is the result of an embolic infarct of the inferior division (posterior temporal or parietal) of the left middle cerebral artery. It is rarely observed at the acute stage of stroke and more frequently affects younger patients [16].

4.4 Transcortical aphasias

Cortical lesions isolating the spared peri-sylvian language areas (watershed territory between the left anterior cerebral artery and middle cerebral artery in addition to the watershed territory between the left middle cerebral artery and posterior cerebral artery).

Subcortical lesions: large thalamic hemorrhage interrupting the temporal isthmus; infarcts in the left thalamus, putamen, and periventricular white matter [17].

4.5 Global aphasia

Extended lesions (including left peri-sylvian anterior and posterior language areas), which are the result of a left middle cerebral artery or carotid artery occlusion (with a total left middle cerebral artery infarct), produce global aphasia with hemiplegia, hemisensory deficits, and hemianopia [2, 14, 15].

Broca's and Wernicke's areas may be simultaneously hypoperfused in the acute period. Thus, global aphasia can be the initial aphasic syndrome (**Figure 8**).

Early involution into Broca's aphasia (with early recovery of comprehension) may result from reperfusion of Wernicke's area. In this case, the patient presents only left frontal lobe, left basal ganglia, and left insula ischemic lesions (diffusion-weighted image shows infarct in superior division of left middle cerebral artery territory, which includes Broca's area), sparing in the same time the left temporo-parietal region (global aphasia with hemiplegia and early improvement of comprehension). Later recovery of comprehension may appear from the reorganization of the language network:

- Frontal and temporoparietal lesions (two lesions) produce global aphasia without hemiplegia. When sensory-motor deficit is missing, we should search for mixed transcortical aphasia.

- Subcortical infarct extended into basal ganglia.

4.6 Anomic aphasia

Acute anomic aphasia may be noted after stroke in many locations. It also represents a stage of all aphasic syndromes when they improve.

4.7 Crossed aphasias

This is a very rare condition (1% of all acute ischemic stroke aphasias), defined by an aphasic syndrome in a right-handed patient (free from developmental disorders and previous brain lesions, fully lateralized, which is demonstrated using a questionnaire such as Edinburgh Inventory), caused by a right hemisphere lesion (non-dominant hemisphere) [2, 14, 15].

The anatomical determinants are similar to those observed in left hemisphere lesion, although a higher proportion of deviant cases are observed, particularly with mild aphasia contrasting with the large lesion. This fact is usually reported as evidence

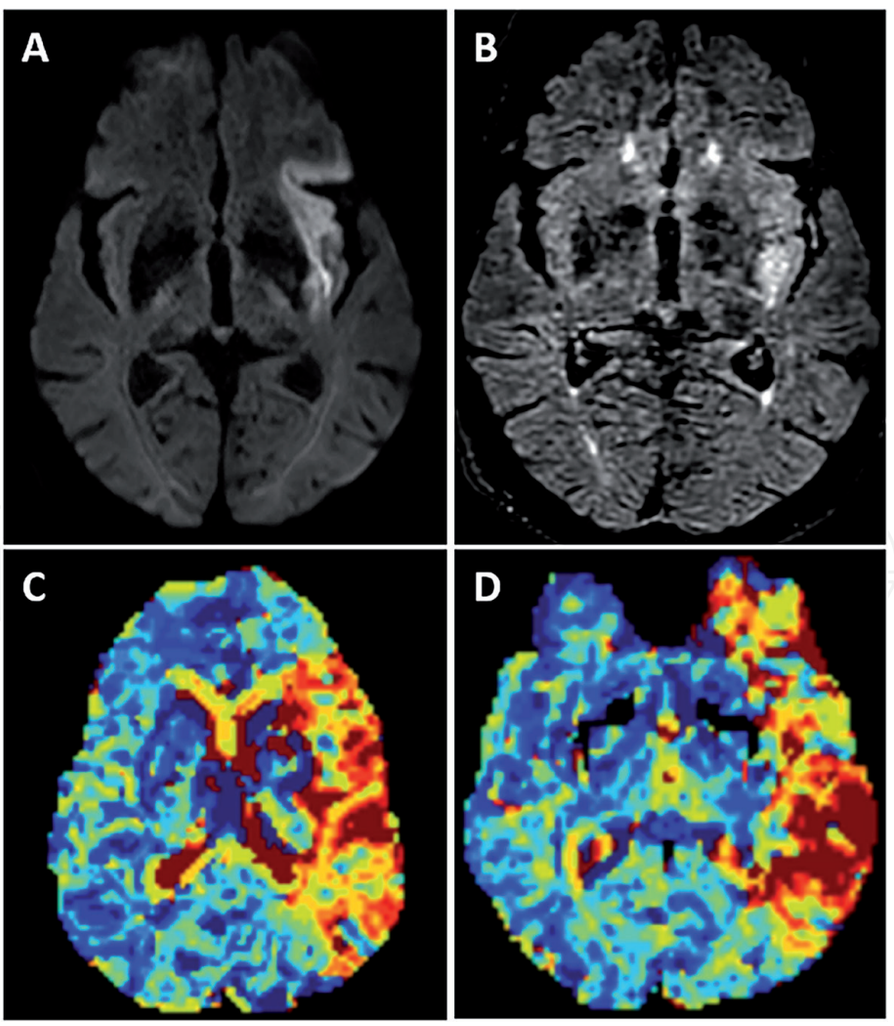

Figure 8.
Acute ischemic stroke of the left sylvian territory in a 76-year-old man with ischemic core involving the Broca's area on DWI (A) and FLAIR (B) and penumbra involving the Wernicke's area (C and D). Global aphasia improved after recanalization, resulting in a chronic non-fluent Broca's aphasia.

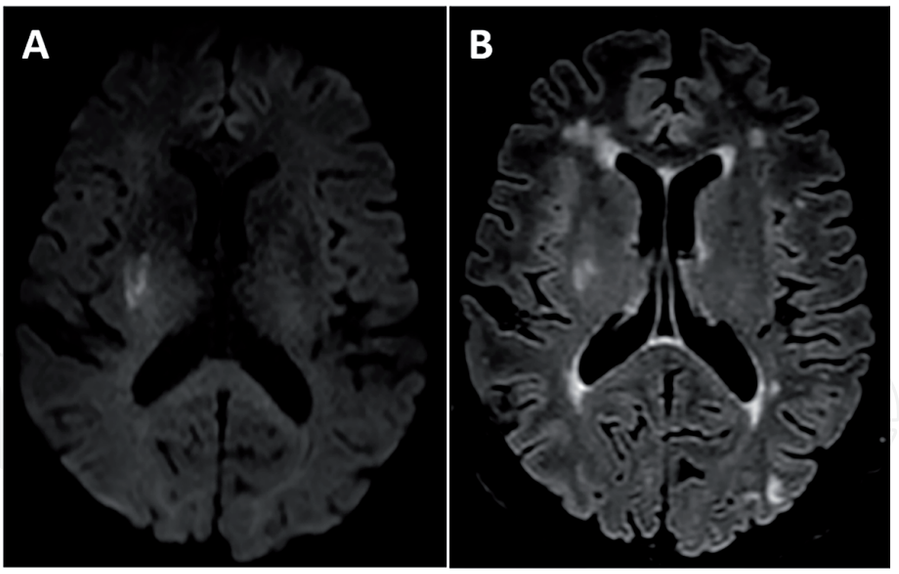

Figure 9.
Acute ischemic stroke of the right external capsule in a 76-year-old man presenting with global aphasia, hyperintense on DWI (A) and FLAIR (B).

for bilateral representation of the language. In the past, crossed aphasia was considered to be non-fluent, although today is reported that all aphasic syndromes can be registered (some cases of crossed Wernicke's aphasia in right-handed patients with lesions in the homologous area of the right cerebral hemisphere are noted) (**Figure 9**).

4.8 Subcortical aphasias

Pure left striato-capsular infarcts (left deep middle cerebral artery infarcts) can produce different types of aphasias (mainly non-fluent, especially motor transcortical aphasia and Broca's aphasia) (**Figure 10**). Frequently, hypophonia (poor speech volume) can be noted [2, 14, 15].

Fluent and non-fluent aphasias have been reported in thalamic lesions. Usually, a thalamic aphasia presents a significant impairment of spontaneous speech, with verbal paraphasias, but with oral comprehension and repetition relatively spared [1, 2, 5]. Patients with subcortical aphasias are older, because the main mechanism of ischemic stroke is small vascular disease.

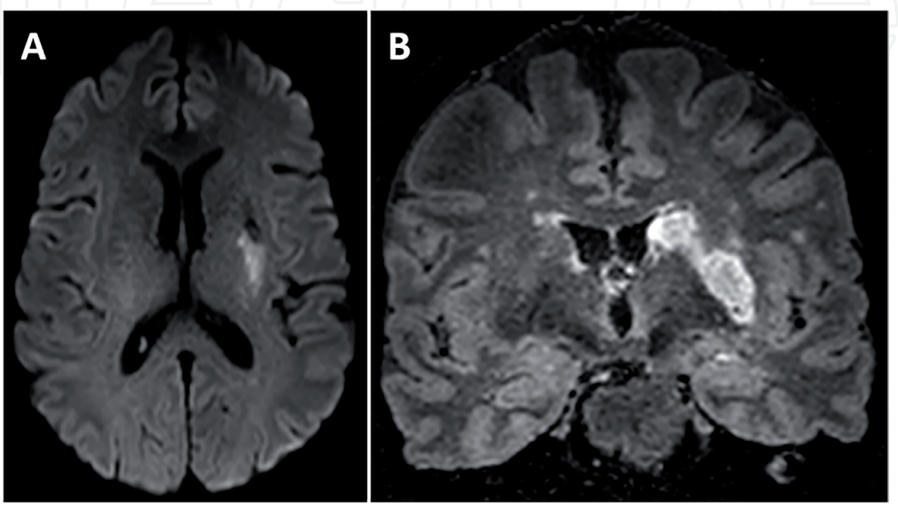

Figure 10.
Acute ischemic stroke of the left striato-capsular area in a 66-year-old woman with subcortical aphasia, hyperintense on DWI (A) and FLAIR (B).

There are two distinct mechanisms concerning subcortical vascular aphasias: (a) a possible sustained cortical hypoperfusion and infarction not visible on structural imaging studies and (b) a possible thalamic disconnection, due to striato-capsular infarcts.

5. Conclusions

Brain imaging, especially MRI, is the cornerstone of the etiological diagnosis and prognostic evaluation of vascular aphasia. Location and extent of the ischemic core are valuable information to assess the severity of aphasia and predict recovery. Despite overlap in MRI patterns between aphasic syndromes, two main networks are known to induce specific language deficit: the anterior network centered on the Broca's area and the posterior network centered on the Wernicke's area. Perfusion imaging is helpful to determine the mismatch between irreversibly injured tissue in the ischemic core and salvageable tissue in the ischemic penumbra that may benefit from reperfusion treatment and result in symptoms recovery.

Future work may focus on the discovery of new imaging biomarkers to help predict aphasia recovery with better accuracy and orient specific treatments.

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Conflict of interest

The authors declare no conflict of interest.

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