Research article

Aphasia in patients with subcortical ischemic stroke

Huynh Quoc Huy[1], Nguyen Hung Sang[2], Cao Phi Phong[3]
[1]People's Hospital 115
[2]Pham Ngoc Thach University of Medicine, Ho Chi Minh City
[3]University of Medicine and Pharmacy at Ho Chi Minh City

Abstract

Objectives: To investigate the characteristics of aphasia in patients with subcortical infarction and evaluate the relationship between clinical language impairment and lesion location.

Methods: This prospective descriptive study was conducted on patients with subcortical infarction admitted to the Cerebrovascular Disease Department, 115 People's Hospital, from November 2017 to May 2018. Inclusion criteria consisted of first-ever ischemic stroke, symptom onset within seven days before enrollment, subcortical lesions confirmed by CT/MRI, and the ability to undergo standardized language assessment. Aphasia was evaluated using the Western Aphasia Battery (WAB), and lesion locations were analyzed based on neuroimaging. Data were processed using SPSS 16.0, with descriptive statistics and Chi-square or t-tests applied where appropriate (significance level: $p < 0.05$).

Results: Among 263 patients with subcortical infarction, 68 (25.9%) exhibited aphasia. The ratio of aphasia showed no significant difference between males (51.5%) and females (48.5%) (p = 0.86). The mean age of aphasic patients was 61.57 ± 12.81 years, with females being significantly older than males (p = 0.001).

All aphasic patients had dominant hemisphere involvement (100%), with a statistically significant difference compared to the non-aphasic group (p = 0.001). The most commonly affected subcortical structures associated with aphasia were the putamen, internal capsule, and caudate nucleus. Broca's aphasia was the most frequent subtype (22.1%), followed by Wernicke's aphasia (16.2%) and global aphasia (1.5%).

Clinically, 66.2% of aphasic patients had facial palsy, and 94.1% were right-handed; however, no statistically significant difference was found between aphasic and non-aphasic groups (p > 0.05). Lesions in the putamen-anterior limb of the internal capsule were significantly associated with Broca's aphasia, whereas thalamic lesions were more frequently linked to global aphasia.

These findings suggest a strong correlation between language impairment in subcortical infarction and lesions in the dominant hemisphere, particularly within the basal ganglia and internal capsule.

Conclusions: Aphasia was observed in 25.9% of patients with subcortical infarction, predominantly involving the dominant hemisphere, particularly the putamen, internal capsule, and caudate nucleus**.** Broca's aphasia was the most common subtype, followed by global and Wernicke's aphasia**.** Lesions in the putamen-anterior limb of the internal capsule were associated with Broca's aphasia, while thalamic infarcts correlated with global aphasia. These findings enhance the understanding of the role of subcortical structures in language processing, aiding in the diagnosis and management of post-stroke aphasia.

Keywords: aphasia, subcortical ischemic stroke

1. INTRODUCTION

Stroke is the third leading cause of death worldwide, with ischemic stroke accounting for the majority of cases and often resulting in severe and lasting sequelae [1]. Aphasia is one of the common complications, significantly impairing communication ability, quality of life, and recovery prognosis in affected patients [1], [2].

Previous studies have primarily focused on cortical aphasia, while the role of subcortical structures in language remains controversial [3], [5]. Some hypotheses suggest that subcortical damage may directly impact language networks or disrupt connections between cortical regions. However, the relationship between specific subcortical lesion sites and aphasia subtypes has yet to be fully elucidated.

In Vietnam, research on aphasia due to subcortical infarction remains limited [4]. Therefore, this study was conducted to describe the characteristics of aphasia in patients with subcortical ischemic stroke and evaluate the correlation between clinical symptoms and lesion location, thereby contributing to improved diagnosis and treatment strategies.

2. METHODS

2.1. Study Population

This prospective descriptive study was conducted at the Department of Cerebrovascular Diseases, 115 People's Hospital, from November 2017 to May 2018. The study included all eligible patients admitted with a diagnosis of subcortical ischemic stroke during the study period.

2.2. Inclusion Criteria

• First-ever ischemic stroke confirmed by clinical assessment and brain imaging (CT or MRI).

• Time from symptom onset to enrollment was no more than 7 days.

• Presence of subcortical lesions on CT or MRI imaging.

• Sufficient cognitive and physical ability to undergo standardized language testing.

2.3. Exclusion Criteria

• Cortical or posterior fossa infarctions.

• History of prior stroke or other known brain pathologies.

• Patients or their legal representatives who did not provide informed consent to participate.

2.4. Study Design and Sampling

This was a prospective, observational study with a convenience sampling method. All patients meeting the inclusion criteria during the study period were enrolled.

The dependent variable was aphasia (presence and subtype), while independent variables included lesion location, clinical signs, and demographic characteristics.

2.5. Language Assessment

Aphasia was evaluated using the Western Aphasia Battery (WAB), a standardized and validated tool for classifying aphasia syndromes based on fluency, comprehension, repetition, and naming. The Western Aphasia Battery (WAB) was selected due to its structured approach and widespread use in aphasia classification. In our study, a Vietnamese-translated version adapted

from the original English tool was used, which has been applied in clinical settings, although not yet formally validated.

2.6. Neuroimaging Analysis

Lesion locations were determined through review of CT or MRI scans. Findings were correlated with radiology reports from the Department of Imaging. Lesions were mapped to specific subcortical regions including the putamen, internal capsule, caudate nucleus, thalamus, and periventricular white matter.

2.7. Statistical Analysis

Data were analyzed using SPSS version 16.0. Descriptive statistics were used to characterize the sample. Chi-square tests were applied for categorical variables, and t-tests were used for continuous variables. A p-value of less than 0.05 was considered statistically significant.

2.8. Ethical Considerations

This study was observational and non-interventional, adhering to standard treatment protocols of the hospital. Ethical approval was obtained from the Ethics Committee of 115 People's Hospital. All participants or their legal guardians provided informed consent before participation.

3. RESULTS

From November 2017 to May 2018, a total of 263 patients with subcortical ischemic stroke were enrolled in the study. Among these, 68 patients (25.9%) exhibited aphasia and 195 (74.1%) did not. Demographic characteristics and clinical profiles of the study population are presented below.

3.1. Characteristics of Aphasia in Patients with Subcortical Ischemic Stroke

Table 1. Gender Distribution

Gender	Number (n = 68)	Percentage (%)
Male	35	51.5%
Female	33	48.5%

Comment: The proportion of males and females in the aphasia group was nearly equal (51.5% vs. 48.5%). There was no statistically significant difference in gender between aphasic and non-aphasic groups (p = 0.86). The nearly equal gender distribution suggests that gender is not a major determinant of aphasia occurrence in subcortical stroke.

Table 2. Age group distribution in aphasic patients

Age group (years)	Number	Percentage (%)
< 45	6	8.8%
45–65	41	60.3%
> 65	21	30.9%
Total	**68**	**100%**

Comment: The majority of aphasia cases occurred in the 45–65 age group, which is consistent with the peak incidence of small vessel disease-related stroke, the most common etiology of subcortical infarcts. A notable proportion of patients over 65 also developed aphasia, highlighting the additive effect of age-related vascular risk and reduced compensatory mechanisms on language outcomes.

Table 3. Mean Age by Gender

Gender	Mean Age ± SD	p-value
Male	56.66 ± 11.16 years	
Female	66.79 ± 12.52 years	0.001

Comment: The average age of the aphasic group was 61.57 ± 12.81 years. Female patients were significantly older than male patients (p = 0.001). This finding may reflect age-related

vulnerabilities such as microvascular changes, reduced neuroplasticity, or delayed access to acute stroke care, which may contribute to higher susceptibility to language impairment in older women following subcortical infarction.

Table 4. Association between Aphasia and Dominant Hemisphere

Hemisphere	Aphasia Group (n = 68)	Non-Aphasia Group (n = 195)	p-value
Dominant hemisphere	68 (100%)	94 (48.2%)	0.001
Non-dominant hemisphere	0 (0%)	101 (51.8%)	

Comment: All patients with aphasia had dominant hemisphere lesions. The difference was statistically significant (p = 0.001), indicating a strong association between aphasia and dominant hemisphere infarction.

Table 5. Relationship between handedness and aphasia

Handedness	Aphasia (n=68)	No aphasia (n=195)	p-value
Right	64 (94.1%)	176 (90.3%)	0.33
Left	4 (5.9%)	19 (9.7%)	

Comment: No statistically significant difference was observed in the distribution of handedness between groups. However, the high proportion of right-handed individuals among aphasic patients is consistent with the anatomical principle that left hemisphere dominance for language is more prevalent in right-handed individuals. These results support the clinical utility of handedness as a surrogate for language-dominant hemisphere identification, particularly in resource-limited settings.

Table 6. Aphasia Subtypes

Aphasia Type	Frequency (n = 68)	Percentage (%)
Broca's Aphasia	15	22.1%
Wernicke's Aphasia	11	16.2%
Global Aphasia	1	1.5%
Transcortical Motor Aphasia	7	10.3%
Transcortical Sensory Aphasia	9	13.3%
Mixed Transcortical Aphasia	7	10.3%
Conduction Aphasia	5	7.4%
Anomic Aphasia	13	19.4%

Comment: Broca's aphasia (22.1%) and anomic aphasia (19.4%) were the most common subtypes. Global aphasia had the lowest prevalence (1.5%). Transcortical aphasias accounted for over 30%, highlighting the significant role of subcortical damage in language networks. The diversity of aphasia types observed supports a distributed language network model, where subcortical lesions can mimic cortical aphasia patterns depending on tract-specific disruption.

3.2. Association Between Aphasia and Clinical Symptoms

Table 7. Aphasia and Facial Palsy

Facial Palsy	Aphasic Group (n = 68)	Non-Aphasic Group (n = 195)	p-value
Present	45 (66.2%)	122 (62.6%)	0.81
Absent	23 (33.8%)	73 (37.4%)	

Comment: There was no statistically significant difference in the rate of facial weakness between patients with and without aphasia. This suggests that facial paresis is not a reliable clinical marker for the presence of aphasia in subcortical stroke. Although both facial and language functions may be affected by lesions in the internal capsule or adjacent white matter, they can also occur independently, reflecting distinct but overlapping neural pathways.

Table 8. Aphasia and Dysarthria

Dysarthria	Aphasic Group	Non-Aphasic Group	p-value
Present	58 (85.3%)	156 (80.0%)	0.30
Absent	10 (14.7%)	39 (20.0%)	

Comment: Dysarthria was common in both groups and showed no significant association with aphasia. This finding supports the distinction between motor speech disorders (dysarthria) and language impairment (aphasia). In subcortical stroke, dysarthria may result from corticobulbar tract involvement, whereas aphasia reflects damage to language-processing networks, primarily in the dominant hemisphere.

Table 9. Aphasia and Sensory Disorders

Group	Sensory Disorder	No Sensory Disorder	Total
Aphasic	15 (22.05%)	53 (77.95%)	68
Non-Aphasic	39 (20%)	156 (80%)	195

Comment: There was a slightly higher proportion of sensory deficits in the aphasic group, but the difference was not significant ($p = 0.7$). The comparable frequency of sensory disturbances in both groups suggests that somatosensory deficits are not strongly associated with the presence or absence of aphasia in subcortical stroke. This supports the view that sensory and language functions are mediated by partially distinct subcortical circuits, despite occasional anatomical overlap—especially in lesions involving the thalamus or posterior periventricular white matter.

Table 10. Aphasia and Hemiparesis (Muscle Strength)

Muscle Strength	Aphasic Group (n, %)	Non-Aphasic Group (n, %)
0/5	1 (1.5%)	5 (2.6%)
1/5	8 (11.8%)	12 (6.2%)
2/5	5 (7.4%)	20 (10.3%)
3/5	37 (54.4%)	34 (17.4%)
4/5	15 (21.1%)	97 (49.7%)
5/5	2 (2.9%)	27 (13.8%)

Comment: Muscle strength grades were significantly lower in aphasic patients, with over half exhibiting moderate paresis (MRC 3/5) and only 2.9% reaching normal strength (5/5). In contrast, nearly two-thirds of non-aphasic patients had good to normal strength (MRC 4–5). These findings confirm a clear association between greater motor impairment and the presence of aphasia, likely due to shared subcortical lesion territories affecting both corticospinal and language-related fiber tracts.

Table 11. Aphasia and NIHSS Score

Group	Mean NIHSS Score ± SD
Aphasic	7.9 ± 2.08
Non-Aphasic	4.8 ± 1.67

Comment: Patients with aphasia had significantly higher NIHSS scores, indicating more severe neurological impairment. Since the NIHSS includes a language component, higher scores may partially reflect the presence of aphasia itself. However, the overall increase also supports the notion that aphasia in subcortical stroke is associated with larger or more functionally critical lesions, possibly involving both motor and associative fiber tracts.

3.3. Association Between Aphasia and Lesion Location on Imaging

Table 12. Subcortical Lesions and Aphasia

Lesion Site	Aphasic Group (n = 68)	Non-Aphasic Group (n = 195)	p-value
Putamen	59 (86.8%)	146 (74.9%)	0.04
Internal Capsule	55 (80.9%)	146 (74.9%)	0.32
Caudate Nucleus	15 (22.1%)	40 (20.5%)	0.79
Thalamus	10 (14.7%)	22 (11.3%)	0.46
Periventricular White Matter	12 (17.6%)	29 (14.9%)	0.68

Comment:
- The putamen was significantly associated with aphasia (p = 0.04), highlighting its key role in subcortical language networks.
- Although the internal capsule had a high frequency of lesions in the aphasia group (80.9%), the difference was not statistically significant (p = 0.32).
- The caudate nucleus, thalamus, and periventricular white matter did not show significant differences between groups (p > 0.05), suggesting a less decisive role in aphasia development.

Table 13. Correlation Between Aphasia Subtypes and Subcortical Lesion Combinations

Aphasia Type	Putamen-ALIC	Putamen-Caudate-ALIC	PVWM	Putamen-PVWM	Putamen-PLIC	Putamen-PVWM-PLIC	Thalamus	Total Cases
Broca's	55.5% (5)	40% (6)	20% (3)	6.6% (1)	–	–	–	100% (15)
Wernicke's	–	–	–	18.2% (2)	63.6% (7)	18.2% (2)	–	100% (11)
Global	–	–	–	100% (1)	–	–	–	100% (1)
Transcortical Motor	33.3% (3)	14.2% (1)	28.5% (2)	14.2% (1)	–	–	–	100% (7)
Transcortical Sensory	–	–	11.1% (1)	–	44.1% (4)	–	44.1% (4)	100% (9)
Mixed Transcortical	–	–	57.1% (4)	14.3% (1)	–	–	28.6% (2)	100% (7)
Conduction	–	–	20% (1)	80% (4)	–	–	–	100% (5)
Anomic	11.1% (1)	–	30.7% (4)	38.5% (5)	–	–	23.1% (3)	100% (13)

Abbreviations:
- ALIC – Anterior Limb of the Internal Capsule
- PLIC - Posterior Limb of the Internal Capsule
- PVWM - Periventricular White Matter

Lesion combinations explained:
- Putamen–ALIC: Lesions involving both the putamen and anterior limb of internal capsule.

- Putamen–Caudate–ALIC: Combined lesions in the putamen, caudate nucleus, and anterior limb.
- Putamen–PVWM: Lesions involving both the putamen and periventricular white matter.
- Putamen–PLIC: Lesions involving both the putamen and posterior limb of the internal capsule.
- Putamen–PVWM–PLIC: Extensive lesions involving all three regions.
- Thalamus: Isolated thalamic lesions.

Comment:

- Anterior subcortical lesions (especially Putamen–ALIC) were strongly associated with Broca's aphasia.
- Posterior lesions (Putamen–PLIC, PVWM) were commonly found in Wernicke's and transcortical sensory aphasia.
- Mixed lesions involving PVWM and internal capsule components appeared in anomic and mixed transcortical aphasias.
- Thalamic lesions were observed primarily in anomic and transcortical sensory aphasia, consistent with the thalamus's semantic role.

4. DISCUSSION

4.1. Characteristics of Aphasia in Patients with Subcortical Ischemic Stroke

In this study, 25.9% of patients with subcortical ischemic stroke exhibited aphasia, which is slightly lower than previous findings in general ischemic stroke populations, such as those by Nguyen Thanh Hong (37.7%) and Bohra V. (27.9%) [1], [3]. However, this prevalence remains consistent with the general estimation that 15–40% of ischemic stroke patients experience aphasia. These results reinforce the notion that subcortical lesions, although not directly affecting cortical language areas, can still induce language dysfunction through mechanisms such as disruption of corticocortical and corticothalamic pathways, or perfusion deficits in language-related cortical territories due to large-vessel occlusion [5].

All patients with aphasia in our study had lesions located in the dominant hemisphere, highlighting its crucial role in language processing even in the presence of deep brain lesions. This aligns with previous literature suggesting that subcortical structures like the internal capsule, putamen, caudate nucleus, and adjacent white matter play an integral role in maintaining the functional connectivity of the language network [6].

In our study, all aphasic patients had lesions in the dominant hemisphere, which is consistent with the anatomical dominance of the left hemisphere for language in most right-handed individuals. Aphasia resulting from non-dominant hemisphere infarction is rare and typically occurs in individuals with atypical or bilateral language representation, a pattern not observed in our sample.

The mean age of patients with aphasia was 61.57 ± 12.81 years, significantly higher in females than males (p = 0.001), indicating a possible age- and sex-related vulnerability. While the underlying reasons for this gender difference remain unclear, it may reflect differences in cerebrovascular architecture, hormonal

factors, or compensatory mechanisms of neuroplasticity.

Regarding the types of aphasia, Broca's aphasia was the most frequently observed subtype, followed by global and transcortical forms. This distribution supports the hypothesis that anterior internal capsule and putaminal lesions are more likely to interrupt the frontostriatal language pathways, leading to non-fluent expressive aphasia. These findings correspond to the "disconnection model," where the breakdown in connectivity between the Broca's area and supplementary motor regions results in a clinical phenotype indistinguishable from cortical lesions [7].

Although facial weakness and dysarthria were more common in the aphasia group, these differences were not statistically significant. This may indicate that subcortical aphasia can occur independently of overt motor symptoms, reinforcing the concept that language deficits may be the only or primary clinical manifestation of deep infarcts depending on the location and extent of fiber tract involvement [5].

4.2. Association Between Aphasia and Clinical Symptoms

The clinical presentation of subcortical stroke is highly variable and often overlaps across functional domains. In this study, we investigated the relationship between aphasia and a range of neurological signs, including motor and sensory deficits, dysarthria, and overall stroke severity.

Our findings revealed no statistically significant association between aphasia and the presence of facial weakness, dysarthria, or sensory disturbance. These results suggest that aphasia, particularly in the context of subcortical lesions, may develop independently of primary corticobulbar or thalamocortical sensory involvement. While these symptoms are common in subcortical strokes, their presence alone does not appear to predict language impairment. This underscores the idea that aphasia in subcortical stroke is more related to fiber tract disconnection within language-specific networks rather than to generalized neurological dysfunction [5].

In contrast, a significant association was observed between aphasia and limb weakness, as well as with lower overall muscle strength (MRC scale) and higher NIHSS scores. Specifically, patients with aphasia had a higher prevalence of moderate-to-severe hemiparesis and were less likely to retain normal muscle strength. This co-occurrence likely reflects the anatomical convergence of corticospinal and language pathways within the internal capsule, corona radiata, and periventricular white matter—regions often implicated in subcortical aphasia [6]. Furthermore, the markedly elevated NIHSS scores among aphasic patients emphasize the role of aphasia as a marker of greater stroke severity. Although the NIHSS includes a language component, the overall increase in score suggests broader dysfunction, potentially due to larger lesion volumes or multi-tract involvement affecting both motor and language functions [6].

Collectively, these findings support the concept of "strategic subcortical infarction", wherein relatively small but

eloquent lesions can disrupt critical integrative pathways, leading to prominent deficits in both speech and motor domains. Recognizing these patterns has practical implications for prognosis, early intervention, and rehabilitation targeting in post-stroke aphasia [2].

4.3. Lesion location in subcortical stroke plays a key role in shaping aphasic symptoms.

Our data demonstrated statistically significant associations between aphasia and lesions located in the internal capsule, lentiform nucleus, periventricular white matter (PVWM), and thalamus. Notably, lesions in the anterior limb of the internal capsule and putamen–anterior capsule complex were most frequently associated with Broca's aphasia, emphasizing the critical role of these regions in expressive language processing [6].

This is consistent with the models proposed by Damasio, Naeser, and Alexander, which suggest that subcortical aphasia arises not from isolated deep lesions per se, but from disruption of language-related white matter tracts, including the frontostriatal circuits and arcuate fasciculus. Lesions in these pathways may hinder information transfer between the Broca's area and prefrontal/motor areas, leading to decreased verbal fluency, effortful speech, and impaired repetition [7].

Furthermore, lesions involving the posterior internal capsule, corona radiata, and PVWM posteriorly were associated with global aphasia and mixed transcortical aphasia, indicating a broader disruption of the entire language network.

These findings align with the view that extensive white matter involvement, particularly when both anterior and posterior segments are affected, can result in more severe and widespread language dysfunction [6].

Interestingly, transcortical sensory aphasia and thalamic aphasia were associated with lesions in the thalamus and posterior PVWM, highlighting the thalamus's role in semantic processing and integration of auditory-verbal stimuli. The thalamus, traditionally viewed as a relay center, is increasingly recognized as a functional hub modulating language comprehension, lexical access, and attention [6].

It is noteworthy that not all patients with similar lesion locations exhibited the same type of aphasia. This heterogeneity suggests a degree of individual variation in neural plasticity and network architecture. It also supports the theory that subcortical aphasia reflects functional disconnection in a distributed language network, rather than focal structural damage alone [7].

4.4. Strengths and Limitations of the Study

Strengths:

• This is one of the few prospective observational studies focusing on aphasia secondary to subcortical infarction in a Vietnamese population.

• A relatively large sample size (n = 263), with 68 patients presenting aphasia, provided adequate statistical power for clinical-radiological correlation analysis.

• The use of the Western Aphasia Battery (WAB) enabled standardized and internationally comparable classification of aphasia subtypes.

- MRI imaging was available for a subgroup of patients, enhancing the anatomical precision of lesion mapping.

Limitations:

- Although prospectively conducted, the study did not include longitudinal follow-up to evaluate the recovery trajectory of language function.

- Not all patients underwent MRI, which may have reduced the sensitivity in identifying small or strategically located infarcts. While cortical lesions were excluded based on clinical evaluation and imaging reports, we acknowledge that the absence of universal MRI use may have limited the precision in excluding cortical aphasia. This represents a methodological limitation of the study.

- The dominant hemisphere was inferred based on handedness alone, which may not be reliable in all cases, particularly in left-handed individuals.

- The analysis did not account for common stroke risk factors, such as hypertension**,** diabetes mellitus, smoking, and hyperlipidemia. These comorbidities may influence both the extent of subcortical infarction and the potential for language recovery.

- Other relevant variables such as lesion volume, edema, cerebral perfusion, and functional imaging (e.g. fMRI, DTI) were not evaluated.

Despite these limitations, the study provides valuable insight into the patterns and mechanisms of subcortical aphasia, highlighting the importance of lesion location in predicting language deficits and suggesting the need for further longitudinal and functional imaging studies.

5. CONCLUSION

Overall, this study provides further evidence on the relationship between aphasia and subcortical lesion location, contributing to a clearer understanding of the role of subcortical structures in language function. These findings are not only meaningful for the diagnosis and classification of aphasia, but also have implications for guiding language rehabilitation strategies in post-stroke patients.

REFERENCES

1. **Bohra Vikram, Gajurel B.P., Gupta M. et al.** (2015). Clinicoanatomical correlation in stroke related aphasia. *Annals of Indian Academy of Neurology*, 18(4), 424–429.
2. **Bùi Kim Mỹ** (2004). Rối loạn ngôn ngữ. *Thần kinh học lâm sàng*, Nhà xuất bản Y học TP. Hồ Chí Minh, tr. 71–74.
3. **Nguyễn Thanh Hồng** (2007). *Nghiên cứu mất ngôn ngữ và hình ảnh học ở bệnh nhân nhồi máu não trên lều*. Luận văn thạc sĩ y học, Đại học Y Dược TP. Hồ Chí Minh.
4. **Nguyễn Thi Hùng** (1999). *Góp phần nghiên cứu đặc điểm hình thái học và tiên lượng của nhồi máu não qua kỹ thuật chụp cắt lớp vi tính*. Luận án tiến sĩ y học, Đại học Y Dược TP. Hồ Chí Minh.
5. **Vũ Tuấn Hùng** (2000). *Nhận xét sơ bộ về liên quan giữa mất ngôn ngữ với vị trí tổn thương trên hình ảnh CT Scans trong nhồi máu não*. Luận văn chuyên khoa II, Đại học Y Dược TP. Hồ Chí Minh.
6. **Vũ Anh Nhị** (2002). Lời nói và ngôn ngữ. *Thần kinh học lâm sàng*, Nhà xuất bản Đại học Quốc Gia TP. Hồ Chí Minh, tr. 249–266.
7. **Kuljic-Obradovic D.C.** (2003). Subcortical aphasia: three different language disorder syndromes? *European Journal of Neurology*, 10(4), 445–448.