Variability in aphasia following subcortical hemorrhagic lesion

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KEY WORDS

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ABSTRACT

Background: Vascular lesion of the subcortical structures leads to aphasia. Cortical hypoperfusion has been proposed to be the etiological mechanism in aphasia following subcortical vascular lesion. Subcortical aphasia shows considerable variability in its clinical profile. Such variability has been attributed to the variable sites of cortical hypoperfusion following ischemic lesion of the subcortical structures. **Purpose:** This study investigated the variability in clinical aphasic profile following subcortical hemorrhagic lesion. **Methods:** We retrospectively investigated the clinical aphasic profiles of twelve patients who reported to our hospital during a period of one year with subcortical hemorrhagic lesions. All patients underwent routine neurological examination, neuroimaging (CT/MRI) investigations and linguistic assessment. **Results:** Eight patients exhibited lesion to the basal ganglia and four showed thalamic lesion. All of them showed considerable variability in their aphasic profile. **Conclusion:** Subcortical hemorrhagic lesion leads to variability in aphasia. Variability in aphasia may be considered as an important consequence in subcortical vascular lesion. Observations from this study were suggestive of better preservation of, and when affected, faster recovery of comprehension skills.

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Introduction

The association between left hemispheric cortical structures and language functions has been recorded for more than a century.[1] However, the subcortical structures were not implicated with linguistic functions until the X-ray-based imaging techniques were applied in neuroscience around the second half of 20th century.[2] Although aphasia has, ever since then, been reported in patients with subcortical damage,[2] its clinical manifestation shows considerable variability.[3]

In the past, several hypotheses have been framed to explain the role of subcortical structures in the manifestation of aphasia. In an extensive review, Nadeau and Crosson[4] compiled these hypotheses that included: i) *diaschisis* – the physiological dysfunction of structurally intact areas remote from the site of lesion; ii) damage or infarction of basal ganglia structures directly involved in language processes; iii) disconnection of cortical structures involved in language processing; iv) impaired release of cortically formulated language segments into the output; and v) stenosis of large cerebral vessels that independently cause subcortical stroke and hypoperfusion of the cortex. In their study, although Nadeau and Crosson[4] defended the role of thalamus in linguistic processing, basal ganglia were projected to have no direct role in language processing, which, in turn, invited criticisms.[5]

In the literature, cortical hypoperfusion (i.e., reduced hemodynamic flow) has increasingly been proposed as the etiological mechanism in aphasias resulting from vascular lesion of the subcortical structures.[3] For instance Vallar and colleagues[6] attributed aphasia and neglect following subcortical lesion to the cortical hypoperfusion. Participants in their study exhibited both ischemic and hemorrhagic lesions. Recently, Hillis and colleagues[7] provided experimental evidence for aphasia and neglect associated with cortical hypoperfusion subsequent to subcortical damage. Further, these authors demonstrated the recovery of aphasia and neglect as the cortical blood flow was restored. Thus, it is apparent from their study that the vascular lesion of the subcortical structures could lead to cortical hypoperfusion and subsequent impairment of those functions subserved by the hypoperfused areas.

Although aphasia subsequent to subcortical lesions has been abundantly reported in the literature, variability in its profile has been investigated only in ischemic lesions.[3] In this context, we aimed to investigate the variability in aphasic profiles following subcortical hemorrhagic lesions.

Methods

Subjects

Twelve right-handed, first-ever hemorrhagic stroke-aphasic subjects (11 males and 1 female) with mean age of 58.3 (SD = 13.5) years were retrospectively selected for a period of one year from the medical case records of our hospital. All subjects had undergone neurological examination. Those subjects with non-hemorrhagic lesions as well as those who had not undergone a formal language evaluation owing to the poor general medical condition or CT/MRI study during the period of hospitalization were excluded from this study. The participants' (or the close relatives, wherever, applicable) consent was obtained.

Linguistic examination

During the course of the medical treatment in the hospital, the linguistic functions were evaluated by an experienced speech-language pathologist with the Kannada version[8] of Western Aphasia Battery.[9] All subjects underwent the initial linguistic examination 3-6 days post-stroke (See Table 1 for the demographics). Those who exhibited conspicuous changes in their communication skills underwent the follow-up examination at the time of discharge (results in parenthesis).

Results

As evident from Table 1, eight of 12 subjects experienced lesion in the basal ganglia and remaining four experienced in the thalamus. Among the eight subjects with basal ganglia lesion,

Table 1: The demographic and clinical profile of participants in the study

Patient	Gender	Age (years)	Lesion site	Type of lesion	Post-stroke evaluation day	Aphasia type
BG1	M	47	Left Globus Pallidus, Putamen, Internal Capsule	Hemorrhage	6	Broca's
BG2	M	62	Left caudate nucleus	Hemorrhage	3 (12)	Global (Broca's)
BG3	M	65	Left putamen	Hemorrhage	6	Anomic
BG4	M	43	Left putamen	Hemorrhage	5	Broca's
BG5	M	33	Left putamen	Hemorrhage	3 (9)	Global (Broca's)
BG6	M	67	Left putamen	Hemorrhage	4 (13)	Global (Broca's)
BG7	M	74	Left corona radiata, lentiform nucleus	Hemorrhage	5	Transcortical motor
BG8	M	53	Left putamen	Hemorrhage	5 (12)	Global (Mixed transcortical)
T1	M	58	Left thalamus	Hemorrhage	3	Transcortical sensory
T2	M	63	Left thalamus	Hemorrhage	4	Anomic
T3	F	58	Left thalamus	Hemorrhage	6 (11)	Mixed transcortical (Anomic)
T4	M	76	Left thalamus	Hemorrhage	5	Anomic

BG – Basal ganglia; T – Thalamus; (parenthesis) – re-evaluation data

four exhibited global aphasia at the time of first evaluation (mean 4.17 days post onset). None of these subjects remained *status quo* as the subsequent re-examination (mean 11.4 days post onset) revealed recovery to Broca's aphasia in three subjects and transcortical mixed aphasia in one. Remaining four subjects in the basal ganglia group showed apparently variable aphasic profiles (Broca's – two; transcortical motor – one; and anomic – one) at the time of first evaluation. The thalamic group also showed apparent variability in its aphasic profile (see Table 1).

Discussion

Aphasia has frequently been reported following lesion to the subcortical structures.[1] Further, a few group of authors have reported that the hypoperfusion of the cortex as the causative mechanism behind aphasia (and neglect) following ischemic[3] and hemorrhagic lesion[6] of subcortical structures. In this context, the manifestation of aphasia by our subjects with subcortical hemorrhage could be attributed to the cortical hypoperfusion.

Hillis and colleagues[7] reported of variability in aphasia following ischemic lesion of the subcortical structures. They argued that the variability in aphasia resulted from variable sites of cortical hypoperfusion following large vessel stenosis and subsequent ischemic infarcts in the subcortical structures. In the present study, the participants with hemorrhagic lesion in the subcortical structures showed extreme variability in their aphasic manifestation (see Table 1). For instance, four of eight subjects with lesion in the basal ganglia initially exhibited global aphasia and later recovered to either Broca's or transcortical mixed aphasia. Remaining four showed Broca's, transcortical mixed or anomic aphasias. Similarly, heterogeneity was also observed in subjects with thalamic lesion (see Table 1). From these observations, it is apparent that lesion to the basal ganglia and thalamus resulted in variability in clinical aphasic profile. In the light of our observations as well as the previous reports on variable aphasic manifestation, it may be argued that the variability is the rule in subcortical aphasia, resulting from the variable sites of cortical hypoperfusion.[7]

Finally, our study showed an additional finding. That is, all participants with non-global aphasia showed preserved comprehension skills. Further, those with initial global aphasia exhibited recovery of comprehension skills, thus evolving to Broca's aphasia at the time of second evaluation. These observations call for further research on the recovery pattern in subcortical aphasias as it may have important implications while planning the rehabilitation programs for people with subcortical aphasia.

Conclusion

The present study revealed three major outcomes. First, it extended the previous reports on variability in aphasia following ischemic lesion of subcortical structures to the hemorrhagic lesion. Second, by supporting previous investigations, it confirmed that the heterogeneity is the rule in subcortical aphasia. Finally, this study showed that the comprehension skills are relatively spared, and when affected, showed faster recovery, thus providing insights into the planning of rehabilitative programs for individuals with subcortical aphasia.

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