Variability in aphasia following subcortical hemorrhagic lesion

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KEY WORDS

Subcortical aphasia
Hypoperfusion
Hemorrhage
Variability

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 Crosson⁴ compiled these hypotheses that included: i) diaschisis – the physiological dysfunc-

tion 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 Crosson⁴ 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 colleagues⁶ 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 vascu-
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. Further, a few group of authors have reported that the hypoperfusion of the cortex as the causative mechanism behind aphasia (and neglect) following ischemic and hemorrhagic lesion 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 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.

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 rehabilitative 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.

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

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