Original Article

Clinicoanatomical correlation in stroke related aphasia

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Abstract

Context: With advances in neuroimaging, traditional views regarding the clinicoanatomic correlation in stroke patients with aphasia are being challenged and it has been observed that lesions at a given cortical or subcortical site may manifest with different aphasia profiles. **Aims:** To study as to whether there is a strict clinicoanatomical correlation between the type of aphasia and lesion site in patients with first ever stroke. **Settings and Design:** Observational study, based in a tertiary care center. **Materials and Methods:** Stroke patient's ≥18 years of age were screened and those with first ever stroke and aphasia were subjected to a detailed stroke workup and language assessment using the Hindi version of Western Aphasia Battery (WAB). Statistical analysis was done with χ^2 test with Yates correction and Kruskal-Wallis test. The level of significance was set at $P < 0.05$. **Results:** Overall aphasia was detected in 27.9% of the 260 screened cases with stroke. Amongst 60 cases with first ever stroke and aphasia, the aphasia type was: Global (33.33%), Broca's (28.3%), transcortical motor (13.33%), transcortical sensory (10%), Wernicke's (8.33%), anomic (5%), and conduction (1.67%) aphasia. A definite correlation between the lesion site and the type of aphasia as per the traditional classification was observed in 35% cases only. **Conclusions:** No absolute correlation exists between the lesion site and the type of clinical aphasia syndrome in majority of the patients with cortical and subcortical stroke.

Key Words

Aphasia, clinico-anatomic correlation, clinico-topographic correlation, stroke

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Introduction

Stroke is among the leading causes of the disability worldwide and aphasia is one of the common presentations of stroke. The frequency of aphasics among stroke patients ranges from 21 to 38% (Pedersen *et al*., 1995; Wade *et al*., 1986; Kauhanen *et al*., 2000; Brust *et al*., 1976; Siirtola *et al*, 1977).[1] Aphasia is defined as a disorder of language that is acquired secondary to brain damage (Alexander and Benson, 1997).[2] It was one of the first higher cortical functions to be used for localization of brain lesions. Various studies have been done to validate if there is strict clinicotopographic correlation seen between the aphasia type and location of the brain lesion in stroke patients. While some studies have concluded that lesion location is the main determinant of the aphasic syndrome encountered in a stroke patient (Hayward *et al*., Kreisler *et al*., Yang *et al*.), other studies have opined against such a definite localization concept (Peychinska *et al*., Joseph *et al*.).[18] For subcortical aphasias also, most of the studies have opined against any definite association between classical aphasic syndromes and subcortical lesions (Colombo *et al*., D'Esposito and Alexander). This study was undertaken with the aim of studying the validity of correlating the clinical aphasia profile with lesion site and comparing aphasia profiles in cortical versus subcortical insults. As the aphasia profile changes over time, we also planned to study the clinical aphasia profile in acute, subacute, and chronic insults with regards to a given lesion site.

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Materials and Methods

The study was carried out at a tertiary healthcare center in Central India (Govind Ballabh Pant Hospital, New Delhi). Stroke patients reporting to the hospital over a 1-year period from March 2012 to March 2013 were screened for the presence of aphasia. Patients with mental obtundation, dementia, or recent head injury were excluded. Aphasia was detected in 72 (27.69%) out of 260 screened stroke patients. Aphasics with recurrent stroke were excluded and 60 aphasic patients ≥18 years of age with first ever stroke (ischemic/hemorrhagic) were included in the study. Ischemic stroke cases were classified as per the trial of ORG 10172 in acute stroke treatment (TOAST) classification. Detailed aphasia assessment was done with the help of the Hindi version of Western Aphasia Battery (WAB) and classified

into different types of fluent (Wernicke's, transcortical sensory, conduction, and anomic aphasia) or nonfluent (Broca's, global, transcortical motor, transcortical mixed, or isolation) aphasia based on their scores for fluency, comprehension, repetition, and naming subtests. All patients were also investigated for stroke-related risk factors and subjected to neuroimaging (computed tomography (CT) or magnetic resonance imaging (MRI) brain) for localization of the lesion. Data was fed on excel sheet and analyzed using SPSS software version 20.χ^2 test with Yates correction and Kruskal-Wallis test, when appropriate, were used. The level of significance was set at $P < 0.05$.

Results

Out of 260 stroke patients screened for aphasia, there were 160 (61.5 %) males and 100 (38.5 %) females. Ischemic stroke accounted for 85.36% and hemorrhagic stroke for 14.62% of the cases. Aphasia was detected in 27.69% of the cases. There was no statistically significant difference in the occurrence of aphasia in ischemic (27.9% cases) vs hemorrhagic stroke (26.3% cases) and gender had no impact on the occurrence of aphasia. Aphasia was observed in 48.4% cases with left and 8.8% cases with right hemispheric involvement, confirming the statistically significant association between left hemispheric lesions and aphasia ($P < 0.001$).

Overall, 60 stroke patients with aphasia were included in the study [Table 1]. Age of the patients ranged from 21 to 80 years (median age-60 years). Around 43.3% (26/60) of these cases had young onset (<50 years) stroke. Majority (95%) of the patients were right-handed and literate (66.7%) and crossed aphasia was seen in four (6.67%) cases among the study group. Around 68.33% of the cases hailed from an urban, while 31.67% belonged to a rural background. The most common risk factor for stroke was hypertension (40%), followed by smoking (35%), alcoholism (25%), coronary artery diseases (11.67%), atrial fibrillation (AF; 11.7%%), diabetes mellitus (10%), valvular heart diseases (10%), and dilated cardiomyopathy (3.37%).

Echocardiographic abnormalities were detected in 45% cases and included left ventricular hypertrophy with diastolic dysfunction (18.3%); valvular heart disease (6.67%); left auricular/left ventricular (LA/LV) thrombus (6.67%); dilated cardiomyopathy (3.3%); and regional wall motion abnormalities and systolic dysfunction due to coronary artery disease (10%). On carotid Doppler study, internal or common carotid artery stenosis was detected in 26 (43.3%) patients.

Lesion localization was based on MRI brain in 46 (76.67%) and CT head in 14 (23.3%) patients. Majority of the patients (n = 53, 88.3%) had either a single infarct or hemorrhage, but seven (11.67%) patients, despite no history of stroke in the past, revealed multiple infarcts on imaging. Overall, 47 (78.33%) patients had ischemic infarcts; 11 (18.33%) had hemorrhagic infarcts, while two (3.33%) had intracerebral hemorrhage. Location of the infarct/hemorrhage was purely cortical in two (3.33%), subcortical in 10 (16.67%), and corticosubcortical in 48 (80%) cases.

Etiology or cause of ischemic stroke was: Atherothrombotic large vessel disease in 24 (40%); cryptogenic in 19 (31.67%); cardioembolic in 11 (18.3%); artery to artery embolism in three (5 %), and lacunar stroke/small vessel disease in one (1.67%).

Table 1: Master sheet showing the lesion location and aphasia type among the study group

Age (years)/ sex	Cortical/ subcortical	Lesion location	Aphasia type
50/F	Both	Parietal	Transcortical Sensory
67/M	Both	Fronto-parietal with caudate and lentiform nucleus	Global
62/F	Both	Fronto-parietal	Transcortical Motor
28/F	Both	Parietal	Broca's
60/M	Both	Fronto-parieto-temporal	Global
65/M	Both	Parietal	Anomic
65/F	Subcortical	Fronto-parietal with caudate nucleus	Global
50/M	Both	Fronto-parietal	Broca's
47/M	Both	Parietal	Broca's
80/M	Subcortical	Parietal	Wernicke's
50/M	Both	Frontal	Transcortical Sensory
25/F	Subcortical	Fronto-parieto-temporal	Broca's
46/M	Both	Fronto-parietal	Broca's
37/M	Cortical	Frontal	Anomic
45/F	Subcortical	Temporal lobe and lentiform nucleus	Broca's
54/M	Both	Fronto-parieto-temporal	Transcortical Motor
48/M	Both	Fronto-parieto-temporal with lentiform nucleus	Global
60/M	Both	Parieto-temporal	Wernicke's
40/M	Both	Parietal	Conduction
65/M	Both	Frontal with lentiform nucleus	Global
60/F	Both	Fronto-parietal	Transcortical Sensory
78/F	Both	Frontal	Transcortical Motor
60/M	Both	Fronto-parietal with lentiform nucleus	Broca's
55/F	Both	Fronto-parieto-occipital	Broca's
32/M	Both	Parieto-temporal	Transcortical Motor
24/F	Both	Parieto-occipital with caudate nucleus	Global
63/M	Both	Parieto-temporal	Wernicke's
70/F	Both	Temporal lobe and lentiform nucleus	Global
60/F	Both	Fronto-parietal	Transcortical Sensory
70/M	Subcortical	Lentiform nucleus	Broca's
45/M	Both	Fronto-parietal	Global
42/F	Both	Parieto-temporal	Global
45/M	Both	Parietal	Transcortical Motor
63/M	Both	Parietal	Transcortical Motor
55/M	Subcortical	Lentiform nucleus	Global
52/M	Subcortical	Caudate and lentiform nucleus	Broca's
50/F	Both	Fronto-parietal	Global
65/M	Both	Fronto-parieto-temporal	Global
40/M	Subcortical	Caudate and lentiform nucleus	Wernicke's
27/M	Both	Fronto-parietal	Global
55/M	Both	Fronto-parieto-temporal with caudate and lentiform nucleus	Global

Table 1: (Continued)

Age (years)/ sex	Cortical/ subcortical	Lesion location	Aphasia type
50/M	Both	Fronto-parietal	Broca's
27/M	Subcortical	Fronto-parietal with caudate and lentiform nucleus	Broca's
42/M	Both	Fronto-parietal	Broca's
35/M	Both	Parieto-temporal with caudate and lentiform nucleus	Global
21/M	Cortical	Fronto-parietal	Global
80/M	Both	Fronto-parietal	Broca's
46/M	Both	Frontal with lentiform nucleus	Global
54/M	Both	Fronto-parietal	Transcortical Sensory
61/M	Both	Frontal	Transcortical Motor
46/M	Both	Fronto-parietal with lentiform nucleus	Broca's
49/M	Both	Fronto-parieto-occipital	Broca's
57/M	Both	Parieto-temporal	Transcortical Motor
41/M	Both	Parieto-occipital with caudate nucleus	Global
50/M	Both	Parieto-temporal	Wernicke's
72/M	Both	Temporal lobe and lentiform nucleus	Global
74/M	Both	Frotno-parietal	Transcortical Sensory
39/M	Both	Parietal	Anomic
47/M	Subcortical	Fronto-parietal with caudate nucleus	Global
43/M	Both	Fronto-parietal	Broca's

F = Female, M = male

Aphasia assessment was done in the acute stroke phase (<2 weeks post stroke) in 22 (36.67%), in the subacute stroke phase (2 weeks-3 months) in 23 (38.33%), and in the chronic stroke phase (>3 months) in 15 (25%) cases. Post stroke duration for assessment of aphasia varied from 3 days to 1 year. Nonfluent aphasia was seen in 77.3% cases in the acute stroke phase, 56.2% cases in the subacute stroke phase, and all the cases in the chronic stroke phase.

The most common type of aphasia in our study group was global aphasia (33.33%) followed by Broca's (28.3%), transcortical motor (13.33%), transcortical sensory (10%), Wernicke's (8.33%), and anomic (5%) aphasia. Conduction aphasia was seen in one case only, while there was no case of isolation or transcortical mixed aphasia. Behavioral change at stroke onset was observed in 66.67% cases with aphasia. Behavior was agitated in 33.33%; apathetic in 25%, and depressed in 8.33% cases. Aphasia was accompanied by hemiplegia in 73.33% and faciobrachial paresis in 16.67%, but 10% cases had only aphasia. Among patients with only aphasia, three had transcortical motor, while one each had Wernicke's, transcortical sensory, and anomic aphasia. Aphasia severity, as determined by the aphasia quotient (AQ) derived from the WAB, was mild (AQ 75-93.8) in five (8.33%), moderate (AQ 50-74) in 13 (21.67%), and severe (AQ < 50) in 40 (70%) cases.

Based on the history, the pattern of aphasia at stroke onset and at the time of assessment remained unchanged in 44 (73.33%) cases. However in 16 (26.67 %) cases with a global aphasia profile at stroke onset, the pattern of aphasia changed over a period ranging from 3 days to 6 months. Majority (68.7%) of these cases evolved into other nonfluent types of aphasia (Broca's - 43.75% and transcortical motor - 25%). Evolution to Wernicke's aphasia was seen in 12.5%, while one case each (6.25%) evolved into transcortical sensory, conduction, and anomic aphasia.

In two cases with pure cortical lesions, global aphasia was seen in one and anomic aphasia in the other. In 10 cases with pure subcortical lesion, 50% had Broca's aphasia, 30% had global aphasia, and 20% had Wernicke's aphasia. In 48 cases with corticosubcortical lesions, 33.3% had global aphasia, 25% had Broca's aphasia, 16.7% had transcortical motor aphasia, 12.5% had transcortical sensory aphasia, 6.2% had Wernicke's aphasia, 6.2% had anomic aphasia, and 2.1% had conduction aphasia.

Conformity between lesion location and the type of aphasia as per the traditional localizationist model was observed in 21 (35 %) patients only. Majority (65%) of the patients failed to show any conformity between the location of the brain lesion and the type of aphasia. Frontal lesions were accompanied by more severe impairment of fluency ($P = 0.189$) and naming ($P = 0.26$); parietal by comprehension ($P = 0.42$) and naming ($P = 0.28$); temporal by naming ($P = 0.17$), comprehension ($P = 0.18$), and repetition ($P = 0.28$); while no significant effect was observed with occipital lesions. In the setting of subcortical lesions, lentiform nucleus lesions were accompanied by significant impairment in comprehension ($P = 0.05$) and repetition ($P = 0.03$). Lesions of the caudate nucleus caused less severe impairment of fluency ($P = 0.196$), comprehension ($P = 0.127$), repetition ($P = 0.116$), and naming ($P = 0.869$) [Figures 1-9].

Discussion

Ischemic stroke occurred in 85.36% and hemorrhagic stroke in 14.62% of our cases and this matches the stroke incidence mentioned in other Indian studies[1] as well as the Harvard Stroke Registry.[2] Young stroke (<50 year) constituted 43.3% of cases. Various studies in past have documented aphasia in 22.7-67.6% of the stroke cases.[2-5] In our study, aphasia was present in 27.69% of the cases and stroke type or gender had no impact on the occurrence of aphasia. In previous studies, crossed aphasia has been reported in 1-4% of the stroke cases.[6,7] In our study, 95% of patients were right-handed and crossed aphasia was seen in 6.67% of the cases.

The three most common risk factors for stroke, as documented in earlier studies, include hypertension, diabetes, and AF.[7,8] In our study also hypertension was the most common risk factor (40%), but other modifiable stroke risk factors like smoking (35%), alcoholism (25%), and cardiac disorders (25%) were more common as compared to AF (11.7%) and diabetes (10%). Cardioembolic stroke has been reported as one of the most common cause of stroke in previous studies (48-62%), but was seen in 18.3% of our cases only.[7,8] Atherothrombotic large vessel disease (40%) emerged as the most common cause, while the etiology remained cryptogenic in 31.67% of the cases.

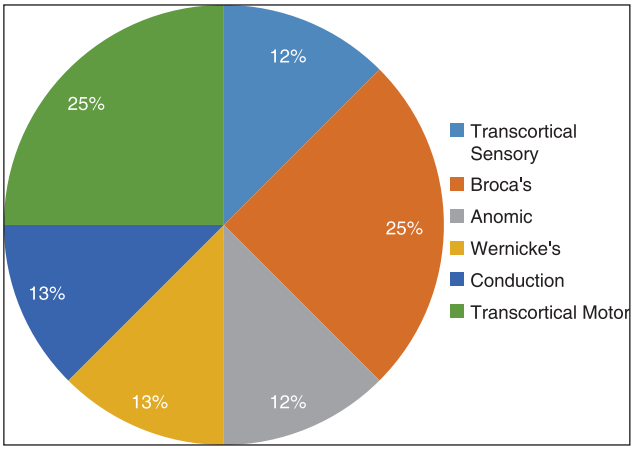

Figure 1: Distribution of aphasia types in patients with parietal lobe lesions

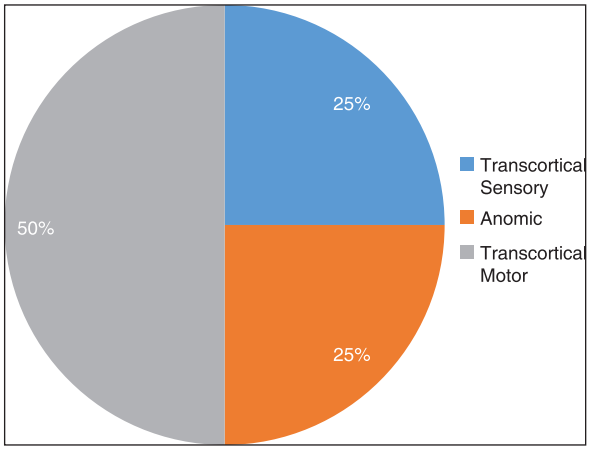

Figure 2: Distribution of aphasia types in patients with frontal lobe lesions

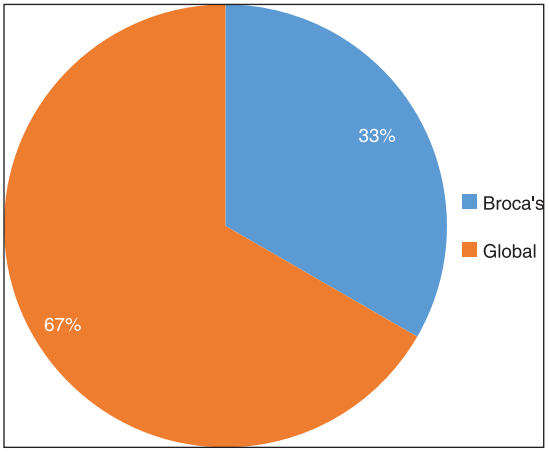

Figure 3: Distribution of aphasia types with temporal lobe lesion *All patients had associated lentiform nucleus involvement

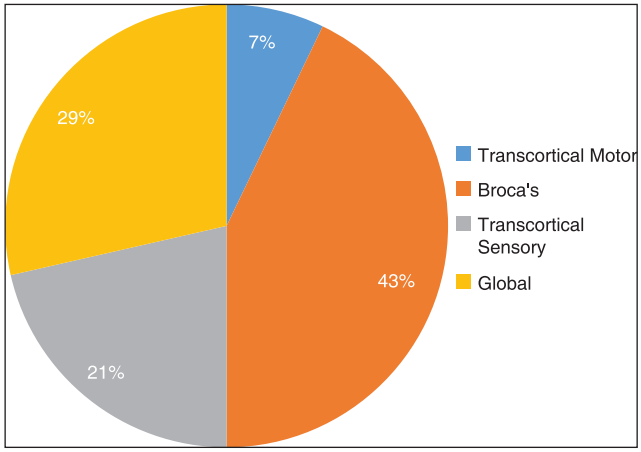

Figure 4: Distribution of aphasia types with frontoparietal lesions

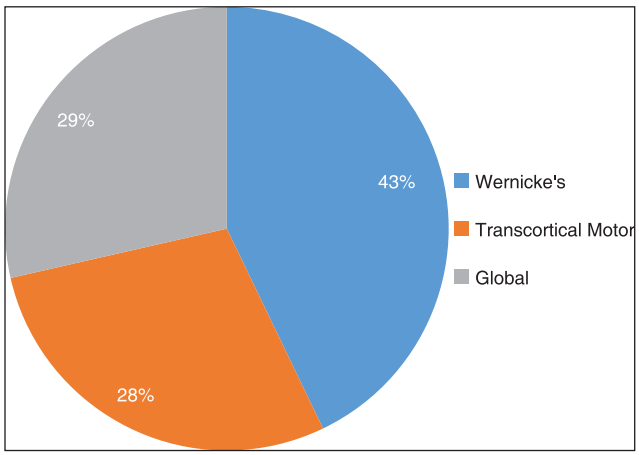

Figure 5: Distribution of aphasia types with parietotemporal lesions

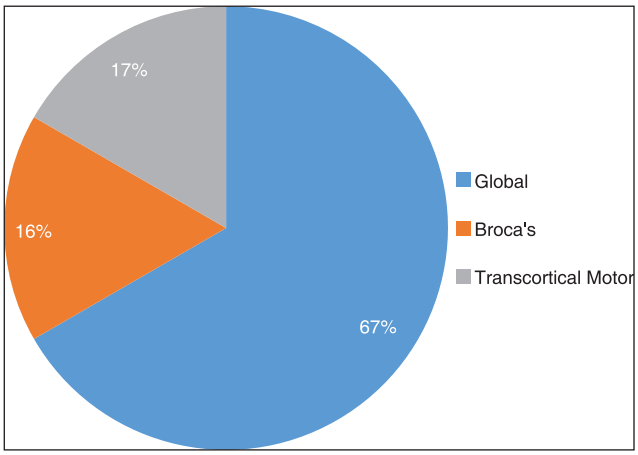

Figure 6: Distribution of aphasia types with frontoparietotemporal lesions

A preponderance of nonfluent aphasia was seen in our study group, the most common type being global (33.33%) followed by Broca's (28.3%), transcortical motor (13.33%), transcortical sensory (10%), Wernicke's (8.33%), anomic (5%), and conduction aphasia (1.7%). Overall, nonfluent aphasia was seen in 77.3% of the cases with acute, 56.2% cases with subacute, and all the cases with chronic post stroke aphasia. Earlier studies[3,5,8-10] have also reported a higher incidence of nonfluent aphasia with global and Broca's aphasia being the major subtypes. However, anomic aphasia which has been reported with a frequency of 16-25%

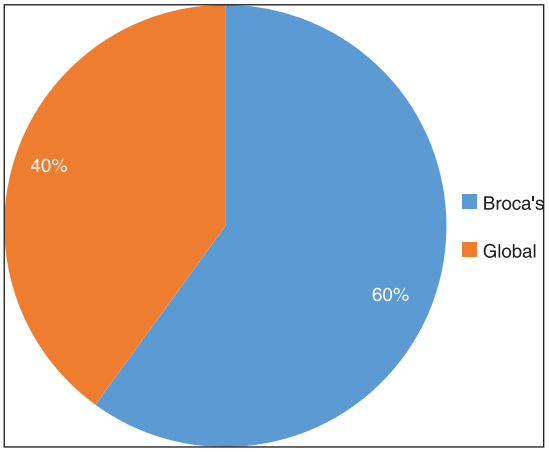

Figure 7: Distribution of aphasia types with frontoparietal with basal ganglia (caudate and/or lentiform) lesions

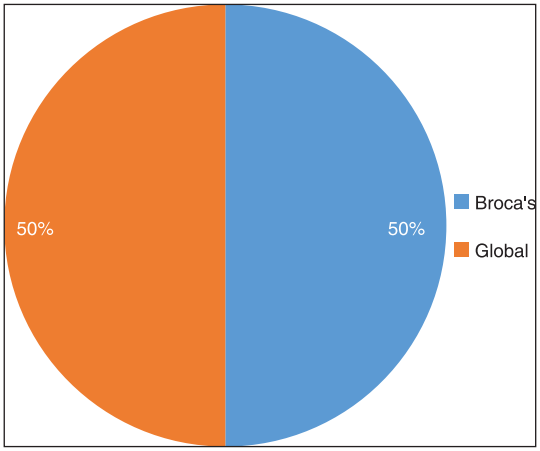

Figure 8: Distribution of aphasia types in patients with parietooccipital lesion. Those with Broca's aphasia had additional involvement of frontal lobe, while those with global aphasia had additional involvement of caudate nucleus

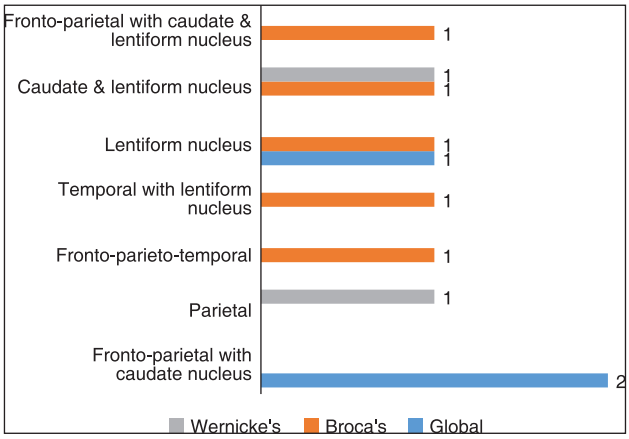

Figure 9: Distribution of aphasia types among those with different subcortical lesion sites

in previous studies[3,9,10] was seen in 5% of our cases only. Pure subcortical lesions were seen in 16.67% of our cases. In one study by Colombo *et al*., the incidence of fluent and nonfluent aphasia was similar in patients with subcortical lesions.[11] In contrast, 80% of our cases with subcortical lesions had nonfluent aphasia.

In a study by Pedersen *et al*.,[9] it was observed that stroke related aphasia usually evolves to a less severe form over time. In our study, a change in aphasia profile over time was observed in 16 (26.67%) cases only. Majority (68.75%) of these cases with global aphasia evolved into other nonfluent aphasia types, while the rest evolved into fluent aphasia (e. g., global to Wernicke's and Broca's to anomic). The overall aphasia pattern remained unchanged in cases presenting with a fluent aphasia at the outset. Aphasia was accompanied by hemiplegia or faciobrachial paresis in 90% of our cases. Aphasia as the only manifestation of stroke has been reported in around 5.1% cases in a study by Fennis *et al*.,[12] but was seen in 10% of our cases.

While some previous studies favor[10,13-15] the concept of a definite correlation between the aphasia type and lesion location in patients with stroke, other studies negate it;[11,16-18] and the debate continues. Majority (65%) of our patients failed to show any conformity between the lesion site and the type of aphasia as per the traditional localizationist aphasia model. A definite correlation between the lesion site and the type of aphasia was observed in 35% cases only. Frontal lesions were more frequently associated with impairment of fluency and naming; parietal with impairment of naming and comprehension and temporal with impairment of comprehension, naming, and repetition. Occipital and caudate lesions did not have any significant effect on fluency, comprehension, repetition, or naming. Lesions of the lentiform nucleus showed severe impairment of comprehension and repetition. In conclusion, clinicotopographical correlation is not seen in the majority of stroke patients with aphasia and lesions at different sites may produce a similar clinical aphasic profile.

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