

A Longitudinal Study of Aphasia Due to Pure Sub-Cortical Strokes

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Abstract

Introduction: Contemporary neuro-imaging techniques have significantly advanced our understanding of the brain organization of language and the involvement of subcortical areas in aphasia. However, articles on sub-cortical aphasia, particularly in non-western languages, remain to be few and far between. We set out to explore aphasia typology in sub-cortical strokes among Bengali-speaking population with a focus towards providing a longitudinal view over a period of 3 months post-stroke. **Methods:** Bengali version of Western Aphasia Battery (BWAB) was used to assess and classify language dysfunction in our study participants. Conventional brain imaging techniques (CT scan & MRI) were used to detect and localize strokes. Uni-variate analysis for categorical variable (location versus aphasia typology) was performed using Chi square and Fischer's exact test (as applicable). Directional measures were calculated using lambda and Goodman-Kruskal tau (Range of -1 to + 1). Boot strapping was applied while calculating the directional measures because of inadequate numbers in some sub-sections the sample. **Results:** Frequency of sub-cortical aphasia was observed to be 29.80% (62/208) in the index study. Four location of strokes were associated with language dysfunction, of which putamen (53.23%) was the commonest followed by striato-capsular region (33.87%). Thalamus and peri-ventricular white matter (PVWM) strokes (6.45% each) were infrequent in our sample of sub-cortical aphasia. Global aphasia (30/62, 48.38%) was the most frequent type observed in acute phase while Broca's aphasia (23/53, 43.39%) dominated among the follow up cases. Aphasia recovery (with follow up AQ of 90.0 or more) was observed in 12 (22.64%) patients of whom majority (8/12) had striato-capsular strokes. **Conclusion:** The present paper illustrates the epidemiological aspects as well as longitudinal course aphasia following pure sub-cortical strokes.

Keywords: Aphasia, longitudinal, sub-cortical

Guest editor's notes: Clinico-anatomical correlational studies of aphasia have been performed since late 1970s and will remain worthwhile for different ethnic groups and languages, with newer imaging techniques and ingenious research questions, as exemplified by this paper. Apart from fMRI and DTI, conventional imaging aided by new software for mapping and tagging of regions (in normalised Brain Atlases) and measurements of lesion volumes (volumetry) are also shedding new light

INTRODUCTION

During the late 19th century it was usually assumed that aphasia can be due to a subcortical pathology. Wernicke^[1] in his classical aphasia classification introduced the subcortical aphasia subtype. At the beginning of the 20th century, however, Marie^[2] rejected this idea and proposed that subcortical damage involving the basal ganglia (an area further known as "Marie's quadrilateral space") would result in dysarthria, not really in aphasia. Sometime later, Dejerine^[3] described the so-called brain's "language zone" corresponding to the perisylvian area of the left hemisphere, without any specific mention to subcortical structures. During the following decades, the idea of "subcortical aphasia" was somehow forgotten. Only during the late 20th century with the introduction of the computed tomography (CT) scan it was observed that aphasia was frequently associated with subcortical pathology, and the discussion and interpretation of subcortical aphasia re-emerged.

Contemporary neuroimaging techniques have significantly advanced our understanding of the brain organization of language and the involvement of subcortical areas in aphasia.^[4,5] Nonetheless, whether true aphasia results from isolated subcortical brain damage, or whether it is due to a cortical extension or cortical deactivation, remains unanswered.^[6,7] For example, Nadeau and Crosson^[8] suggested that linguistic

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impairments associated with striatocapsular pathology are predominantly related to sustained cortical hypoperfusion and infarction not visible on structural imaging studies.

Subcortical pathology usually includes dysarthria, frequently beginning with total mutism followed by hypophonic, slow, sparse output, and poorly differentiated, amelodic speech. In addition to the evident speech disorders, sometimes language impairments (aphasia) are also found.^[9,10]

Two neuroanatomical areas are most frequently discussed in subcortical aphasias: the striatocapsular region and the thalamus. In a pioneer article Alexander, Naeser, and Palumbo^[11] analyzed the aphasia profiles of 19 cases with subcortical pathology. Several components of speech and language, including sentence length, grammatical form, ease of speech initiation, articulation, voice volume, and auditory comprehension, were individually correlated with CT lesion site. In a further analysis including 61 subcortical cases in the neurological literature the authors proposed six subtypes of verbal output impairment. These subtypes are dependent on the specific neuroanatomical locus of striatocapsular damage, demonstrating that considerable variation in speech and language defects can follow this type of pathology. Nonetheless, frequently, extension that involves the cortex was present in these cases.

It is usually assumed that extensive subcortical damage is required to produce a pure striatocapsular aphasia.^[12] Otherwise, only speech defects will be found. In an influential paper, Mega and Alexander^[13] evaluated 14 cases of striatocapsular aphasia. The clinical profiles of the patients were quite similar, varying in severity in rough proportion to lesion size and varying in quality in proportion to anterior paraventricular extent. Larger lesions were associated with impaired “executive” and “generative” language functions. Similar aphasia profiles in patients with deep frontal and paraventricular white matter (PVWM) lesions suggested that damage to a frontal-caudate functional system underlies a “core” aphasia profile in these patients. Recently, Bouvier *et al.*^[14] reviewed the literature describing oral language disturbances following subcortical non-thalamic stroke affecting the basal ganglia and the surrounding white matter. They selected 22 articles including 114 participants. The results suggested a predominance of deficits in more complex and demanding language levels (ex. discourse, syntax) and in language production (versus comprehension). Rapid recovery was expected, particularly for lexical-semantic and receptive deficits.

It is important to note that speech and language disturbances associated with basal ganglia damage are heterogeneous. Hillis *et al.*^[15] selected 24 patients with left caudate infarcts. Specific regions in perisylvian cortex were rated for the percentage of the region that was hypoperfused. Results demonstrated that in patients with acute left caudate infarct, the presence and type of aphasia reflected regions of hypoperfusion, and generally followed predictions based on chronic lesion studies, regarding anatomical lesions associated with classic aphasia types. Radanovic and Mansur^[7] conducted a comprehensive review

of language disturbances after vascular basal ganglia lesions. They selected 57 papers including 303 patients. Results showed that aphasias caused by basal ganglia lesions are heterogeneous with weak clinicoanatomical correlations. Data suggested that subcortical aphasia involves hypoperfusion in the cortical territories of the middle cerebral/internal carotid arteries and their branches. Consequently, the pathology is subcortical but the aphasia anyhow is cortical.

The second major subtype of subcortical aphasia is the thalamic aphasia. Thalamic damage usually produces an acute, catastrophic clinical disorder with hemiplegia, hemisensory loss, and alterations in the level of consciousness.^[16] The initial language abnormality is mutism, which typically improves to a verbose, paraphasic, but hypophonic jargon output. Anomia is observed and often it is severe. Patients with thalamic aphasia present a significant decreased comprehension. A similarity to transcortical (extrasilvian) sensory aphasia has been suggested, even though some syntactic difficulties have also been reported.^[17] Usually, thalamic aphasia is observed in cases of left pulvinar nucleus pathology; noteworthy, the pulvinar nucleus projects to an extensive occipital-temporal and parietal cortical area, including Brodmann areas 18, 19, 37, 39, and 40. The damage in these areas have been frequently related with extrasylvian sensory aphasia. It has been further suggested that thalamic nuclei and systems are involved in multiple processes that directly or indirectly support cortical language functions: lexical-semantic functions, working memory, visual processing in reading, and category-specific naming.^[18] It has been also proposed that the left thalamus seems to bring online the cortical network involved in language processing.^[19] Matsuzono *et al.*^[20] suggested that subcortical diaschisis due to left thalamotuberal artery area infarction results in a transcortical sensory aphasia. Infarcted thalamic nuclei result in disrupted integrative thalamus/cortex function.

Fritsch *et al.*^[21] conducted a retrospective analysis of 1064 consecutive stroke patients. It was observed that 52 (4.9%) had an isolated lesion in the thalamus. In patients with isolated lesion in the thalamus, 6/52 had aphasic symptoms. Aphasic symptoms after isolated lesion in the thalamus were only present in patients with left anterior lesion location. It was concluded that aphasia in thalamic stroke is strongly associated with left anterior lesion location. In thalamo-cortical language networks, specifically the nuclei in the left anterior thalamus could play an important role in integration of left cortical information with disconnection leading to aphasic symptoms.

Some of the authors have distinguished more than two different subcortical aphasia syndromes. Kuljic-Obradovic^[22] selected 32 stroke patients with subcortical aphasia. Patients were divided into three subgroups based on CT and magnetic resonance (MR) images: striato-capsular aphasia, aphasia associated with white matter paraventricular lesions, and thalamic aphasia. Clinical presentation of subcortical aphasias was observed to characterize with preserved repetition, but the three subgroups also presented specific features of language impairment. Striato-capsular

aphasia and aphasia associated with white matter paraventricular lesions were characterized by lack of speech fluency, occurrence of literal paraphasias, mainly preserved comprehension and naming. Thalamic aphasia was characterized with fluent output, impaired comprehension and naming with predominant verbal paraphasias. The author suggested that the most prominent feature in striato-capsular aphasia was phonetic impairment of language, opposite to thalamic aphasia where lexical-semantic processing seemed to be most affected. Copland and Angwin^[23] argue that the thalamus influences lexical-semantic processing through attentional engagement, while striatal-thalamic-cortical circuits most likely influence lexical-semantic functions, bilingual language processing, and sentence comprehension through domain-general mechanisms, including controlled selection and suppression.

The present study attempts to advance our understanding of subcortical aphasia by exploring location-wise distribution of aphasia typology, both in acute as well as sub-acute phases, following pure sub-cortical strokes. We selected, from our existing database of post-stroke aphasia, 62 patients with pure subcortical lesions and distinguished four different pathology areas: putamen, thalamus, periventricular white matter (PVWM), and striato-capsular region. Out of these, 53 were followed up; change in aphasia type across a period of 3 months post-stroke was analyzed.

Methods

With prior approval from the Institutional Ethics Committee, we conducted an observational study in the stroke unit of our center that included subjects with first ever stroke over a period of two years (2016-2018). The results of this “Kolkata aphasia study” have been presented in several papers so far.^[24-26] The current paper, which is part of this larger epidemiological study, attempts to explore sub-cortical aphasia in particular.

Consecutive patients with first ever acute stroke presenting to our stroke unit were recruited for the “Kolkata aphasia study”. The inclusion criteria were, (1) adult (>18 years) literate participants with aphasia due to first ever stroke; (2) conscious and alert at the time of language assessment; (3) Bengali speakers. The exclusion criteria were as follows: (1) aphasia caused by vascular catastrophe inside intra-cranial space-occupying lesion; (2) pre-morbid psychiatric illness affecting communication (such as personality disorder); (3) pre-morbid dementia (documented or, suspected); (4) alcohol or drug abuse; (5) significant non-linguistic cognitive disturbance. Table 1 presents the general characteristics of the sample.

The Bengali version of Western Aphasia Battery (BWAB), a validated tool for assessing the language function in adults, was used to measure the presence, severity, and type of aphasia.^[27] Language tests were independently administered and interpreted by 2 experienced behavioral neurologists (DL and SD) across different time points in the study period. Any dispute was solved by the senior neurologist in the team (BKR) who opined without the previous knowledge of the findings by

Table 1: Population characteristics of the patients presenting sub-cortical aphasia

Parameters	Values
<i>n</i>	62
Age in years (mean/SD)	54.8/7.74
Gender (M/F)	50/12
Handedness (R/L)	62/0
Side of stroke (L/R) ¹	56/6
Education years (mean/SD)	7.0/4.07
Bilingualism (No/Yes)	48/14
NIHSS (mean/SD) ²	13.8/5.28
Stroke type (Ischemic/Hemorrhagic)	27/35
Lesion volume (mean/SD) ³	8.4/3.84
Aphasia severity (AQ) (median/IQR) ⁴	5.1 (2.8-79.2)
Lesion location (<i>n</i> , %)	Putamen (33, 53.23%) Striato-capsular (21, 33.87%) Thalamus (4, 6.45%) PVWM (4, 6.45%)

¹ Right-sided lesions were distributed as follows—Putamen (1); Striato-capsular (3); Thalamus (1) & PVWM (1); ² The National Institutes of Health Stroke Scale (NIHSS) is a systematic assessment tool that provides a quantitative measure of stroke-related neurologic deficit. Its score ranges from 0 (normal) to 42 (severely abnormal); ³ Volume is measured in mL (cm³); ⁴ IQR refers to Inter-quartile Range between 25 and 50%

DL or SD. The parameters of BWAB included Spontaneous Speech, Comprehension, Repetition, Naming, and Aphasia Quotient (AQ). The AQ is a measure of severity of language impairment and is a composite score based on the oral-auditory language subscales of Fluency, Comprehension, Repetition, and Naming. Aphasia quotient below 93.8 is considered the quantitative cut-off for diagnosis of aphasia. Language disorders were classified into conventional types as per WAB. The severity scale as specified in WAB-R was utilized to classify the language dysfunction into mild (AQ 76-93.8), moderate (AQ 51-75), severe (AQ 26-50) and very severe (AQ 25 or less) categories. Mild and moderate categories (with AQ > 50) were designated as non-severe while severe and very severe categories, taken together, were designated as severe aphasia. Language examination in the study subjects were carried out between 7 and 14 days following onset of stroke symptoms and was repeated between 90 and 100 days post-stroke. The change in aphasia severity and typology was documented. All the recruited patients received conventional speech language therapy for 2 hours every week up to a period of 8–10 weeks. Aphasia recovery following stroke was categorized as no change, change to a milder type/lower severity score across severity scales or complete recovery.^[28] AQ score of 90.0 or more in follow up assessment was decided as complete recovery cut-off. The cut-off score assigned to complete recovery was data-driven. The lower and upper bounds of AQ (in first visit) were 2.0 and 90.0 respectively in our study population. It needs to be mentioned that patients designated as recovered (during the follow up assessment) in this paper based on the cut-off AQ score of 90.0 may not have full clinical recovery. WAB indeed does not provide a cut off AQ score for recovery.

Edinburgh Handedness Inventory^[29] was used to determine each participant's handedness and laterality index. Right-handed participants (n = 509) represented the majority (98.8%) of our sample.

Brain imaging for ischemic strokes in the present study was performed by Siemens 3T MRI machine (MagnetomVerio DOT, 16 channels) using a standard quadrature head coil. Computed tomography (CT) scan (Philips, 16 slice) was used for initial brain imaging of subjects with hemorrhagic stroke. Brain images for the recruited patients were obtained both during acute and sub-acute post-stroke phases. The images were read by neuro-radiologists to determine the location of lesion. Infarcted tissue was defined as tissue having abnormal high signal on T2 weighted and/or FLAIR (Fluid attenuated inversion recovery sequences) images. Diffusion weighted imaging was employed for detection and localization of acute infarct. Location and extent of the lesion were determined in respect to the sulcal anatomy. Region of interest (ROI) software was employed to specify lesion site as well as calculate the volume. Lesion volume was determined by using standard ABC/2 method in CT images for intra-cerebral hemorrhage^[30] and in MRI images (DWI or FLAIR) for ischemic stroke.^[31] For subjects (3) with ischemic stroke and contraindication for MRI, infarct volume was determined by ASPECT scoring and CT perfusion score.^[32] Lesions which did not reveal any cortical involvement and were limited to sub-cortex only were designated as pure sub-cortical strokes. Pure sub-cortical lesions were divided in 4 categories-basal ganglia; thalamus; para-ventricular white matter, and striato-capsular. Table 2 shows the aphasia severity according to the lesion locations.

Statistical analyses were performed using SPSS latest version. Uni-variate analysis for categorical variable (location versus aphasia typology) was performed using Chi square and Fischer's exact test (as applicable). Directional measures were calculated using lambda and Goodman-Kruskal tau (Range of -1 to + 1). Boot strapping was applied while calculating the directional measures because of inadequate numbers in some sub-sections the sample.

RESULTS

Among the 515 consecutive patients with first ever acute stroke recruited in our larger epidemiological study, 175 were found to have pure sub-cortical strokes, which constitute 33.98% of the study population. Sub-cortical aphasia was diagnosed in 62 patients among the total tally of 208 who presented post-stroke aphasia in our dataset. Therefore, the incidence of sub-cortical aphasia in our study was 29.80%. Four sub-cortical stroke locations were observed to have been associated with aphasia—putamen, striato-capsular, PVWM, and thalamus. The population characteristic of sub-cortical aphasia in the present study has been depicted in Table 1. The distribution of aphasia severity according to location of stroke has been elaborated in Table 2. Maximum severity was noted with putaminal strokes as all the cases belonged to the severe aphasia class

while PVWM strokes presented minimal severity. For the striato-capsular and thalamic strokes, the majority (61.9% and 75%, respectively) belonged to non-severe class.

Table 3 illustrates the outcome of language dysfunction after 3 months post-stroke in terms of no change, change to a milder type or severity class or complete recovery. Table 4 represents location-wise distribution of aphasia typology both during acute phase and at follow up visit. During acute phase, it was observed that global aphasia is the commonest type (48.38%) of language dysfunction in sub-cortical strokes. Putamen was the most frequent location (53.22%) associated with any form of language dysfunction, of which global aphasia was most common followed by Broca's aphasia. Striato-capsular region was the 2nd most frequent location (33.87%) associated with aphasia and the aphasia types for this location were as follows—Broca's; TCM and anomic aphasia. The distribution of typology was most heterogeneous for thalamic strokes. Global, isolation, TCM, and TCS aphasia one of each were found in the total of 4 thalamic aphasias. PVWM strokes were associated with TCM and anomic aphasia. Although the association between location of sub-cortical stroke and aphasia typology was found to be statistically significant ($X^2 = 84.06$, $P < 0.001$), the analysis suffers due to inadequate numbers in some sub-sections. The lambda and Goodman-Kruskal tau (directional measures) for location-dependent association of aphasia typology are 0.655 and 0.597, respectively.

In the follow up visit, 53 patients were available for repeat language assessment which was performed between 90 and 100 days post-stroke. Broca's aphasia was the most frequent form of language dysfunction, constituting 43.39% of follow up observations. Putaminal aphasias were mostly (16/29; 55.17%) found to have transformed into Broca's aphasia. Complete recovery was observed in 12 (22.64%) patients of whom majority (8/12) had striato-capsular strokes. The association between location of sub-cortical stroke and aphasia typology (including complete recovery) in follow up visit was found to be statistically significant ($X^2 = 65.70$, $P < 0.001$), the

Table 2: Location wise distribution of aphasia severity

Location	Non-severe (AQ>50)	Severe (AQ < 50)
Putamen	0	33
Thalamus	3	1
PVWM	4	0
Striato-capsular	13	8

Table 3: Location wise distribution of aphasia outcome in sub-cortical strokes

Location	No change	Change	Recovery
Putamen	14	15	0
Thalamus	1	0	1
PVWM	1	0	3
Striato-capsular	9	1	8

Table 4: Pattern of change in aphasia typology during early post-stroke phase in patients with pure sub-cortical stroke

Location	Global	Broca's	Isolation	TCM	Wernicke's	TCS	Conduction	Anomia	Recovery*
Putamen	29/13	4/16	0/0	0/0	0/0	0/0	0/0	0/0	0
Thalamus	1/0	0/0	1/1	1/0	0/0	1/0	0/0	0/0	1
PVWM	0/0	0/0	0/0	2/0	0/0	0/0	0/0	2/1	3
Striato-capsular	0/0	8/7	0/0	8/2	0/0	0/0	0/0	5/1	8

/Represents numbers in follow up visit. *Represents the numbers only in follow-up visit

analysis suffers due to inadequate numbers in some sub-sections. The lambda and Goodman-Kruskal tau (directional measures) for location dependant association of aphasia typology are 0.500 and 0.459, respectively. Table 4 shows the pattern of change in aphasia typology during early post-stroke phase.

DISCUSSION

The participation of sub-cortical structures in language function has been a subject of interest in aphasiology since the 19th century. To explore the stroke model is the most practicable way of approaching this question from a scientific perspective mostly because degenerative models are not only particularly concerned with cortical language functions but also usually involve extensive and diffuse pathologies. Body of published research on this topic so far is relatively small but growing. Nevertheless, studies dealing with sub-cortical aphasia in non-western languages remain to be few. The present series, to the best of our knowledge, is the largest available series of aphasia after sub-cortical strokes. Moreover, this is the first account of sub-cortical aphasia in Bengali-speaking population. The importance of the present study also lies in the fact that the participants were followed up over a duration of approximately 3 months which gives us the opportunity to have a longitudinal view of the aphasia typology as well as outcome in respect to sub-cortical location of lesion.

In the present dataset, 175 patients presented pure sub-cortical strokes which constitute approximately 1/3rd of the study population. As has already been discussed in an earlier paper^[4] the incidence of sub-cortical aphasia is definitely on the higher side (29.80%) in our study population—an observation possibly linked to higher incidence of sub-cortical strokes in eastern India.^[33] If the participants' vernacular language had any contribution to this relatively higher incidence of sub-cortical aphasia is a matter of speculation as of now and can only be clarified with future studies comparing sub-cortical aphasia across different languages. As far as similar studies on Indian languages are concerned, the one by Bohra *et al.*^[34] reports a much lower (16.6%) incidence of sub-cortical aphasia in Hindi-speaking participants. Hindi has some linguistic similarity to Bengali, particularly if one considers their common origin from Devnagari which has high phonetic transparency. Functional studies as well as tractography based analysis are required to shed some light on the cross-linguistic diversity of sub-cortical white matter participation in language tasks.

There were four sub-cortical locations associated with aphasia as observed in our data. The commonest location was the putamen (53.23%) followed closely by striato-capsular (33.87%) region. Aphasia due to periventricular white matter (6.45%) and thalamic lesions (6.45%) were relatively uncommon. The most striking observation, however, is the pattern of language impairment in respect to location of stroke. A strong predominance towards non-fluent forms of aphasia was seen in study participants across a follow up period of 3 months. Global aphasia was predominant in the initial assessment while Broca's aphasia was frequent among the follow up cases. Wernicke's aphasia was not observed in our sample of sub-cortical strokes, neither in acute nor in sub-acute phase. This observation goes on to show that comprehension tasks were rarely found impaired in isolation in sub-cortical aphasia irrespective of the lesion site. However, one needs to consider the level of complexity of the comprehension tasks in such situation. It is possible that comprehension tests in WAB were not demanding enough to expose the minor deficits.

Aphasia following basal ganglia strokes is known to be mostly of non-fluent type. In our results, as well, basal ganglia lesions comprising of the sub-groups putamen and striato-capsular region were observed to have global aphasia and Broca's aphasia respectively; both of which are non-fluent forms of aphasia. Interestingly, with the emergence of Broca's aphasia in the follow up assessment, the pattern persisted to be non-fluent throughout the course of early post-stroke phase up to a period of around three months. Thus, the initial pattern of fluency impairment can be assumed to be fairly stable in basal ganglia strokes. The comprehension scores, however, improved with time in patients having putaminal strokes, mostly hemorrhagic type. Our observation is fairly in agreement with the report by Krishnan *et al.*^[35] who also found 50% of the basal ganglia related aphasia was global aphasia- a form of non-fluent aphasia. However, their study was retrospective and language assessments were carried out only in acute post-stroke phase. The present analysis, on the other hand, provides a longitudinal view of the aphasia symptomatology in basal ganglia strokes.

The pattern of thalamic aphasias is known to be variable, particularly after subsidence of the acute phase, when global aphasia may dominate the clinical presentation. Although the characteristic presentation is fluent output, impaired comprehension and naming along with abundant verbal paraphasias,^[22] fluency impairment has also been reported.^[36] Thalamic aphasia is thought to result from the disconnection between cortical language centers and thalamic nuclei.

Left thalamus involvement is mostly implicated in causing aphasia^[18,37] while infrequent cases of crossed thalamic aphasia have also been noted. In our study, on the contrary, fluent aphasia was not observed in thalamic strokes neither in acute nor in sub-acute phases. We documented a case of TCS aphasia following thalamic stroke that subsequently went on to recover. The other three cases presented global, Broca's, and TCM aphasia each. Therefore, an obvious trend towards fluency impairment was observed in thalamic aphasia. Although we had only four cases, this indeed is a noteworthy difference from the existing knowledge and needs to be explored further with larger samples of thalamic aphasia.

Another notable observation in the present series is the number of crossed sub-cortical aphasia. We had previously reported the relatively high incidence of crossed aphasia in Bengali-speaking population.^[38] Our sample of crossed aphasia (n = 14) included of 6 cases of pure sub-cortical strokes which are also part of the present analysis. Therefore, the proportion of sub-cortical language dysfunction in both crossed (6/14; 42.85%) as well as uncrossed aphasia (56/194, 28.86%) remains fairly comparable although statistical conclusions cannot be drawn due to small sample size of crossed aphasia.

The idea of cross-linguistic variation in brain representation of language has gained considerable interest in contemporary literature over last two decades.^[39] Idiosyncrasies of different languages are thought to affect their respective organization in brain. It is evident that the general principles of cerebral language representation remain similar across languages. Smaller variations, however, have been observed to exist so far. If sub-cortical contribution to such variations holds importance needs to be explored further. The knowledge of sub-cortical aphasia typology in different languages may lend help in this matter. In our previous papers,^[24-26,38] we have attempted to explore this front by elaborating various aspects of Bengali aphasia including uncrossed aphasia, crossed aphasia, and lesion-discordant aphasias. The index paper demonstrates various aspects of sub-cortical aphasia in Bengali-speakers. Interestingly, non-fluent pattern has so far been seen to dominate the picture of Bengali aphasia when viewed from different angles. The observation, however, should be subjected to further experimental effort in order to get a more transparent view of the subject.

Finally, some important limitations regarding the sample's demographic characteristics, the assessment instruments including radiological tools that were used, and the idiosyncrasies of the participants' language should be recognized. Extended studies with diverse populations are required to pinpoint the generalizability of our results.

CONCLUSION

The present paper illustrates the epidemiological aspects as well as longitudinal course of aphasia following pure sub-cortical strokes. It not only adds to the small but growing body of knowledge on this topic but also is among the handful

of papers that discuss sub-cortical aphasia in non-western languages. We observed a relatively high incidence of sub-cortical aphasia in our study population as compared to previous similar papers. Location-wise distribution of aphasia severity and typology was presented in this article although the statistical analysis suffers at some points due to small sample size. Yet, to our knowledge, this is the largest series of sub-cortical aphasia reported so far. We also observed a trend towards non-fluent aphasias among the participants, both in acute and sub-acute phases. More elaborated studies involving larger sample of sub-cortical aphasia and employing advanced neuro-imaging techniques, such as DTI, will be required to further our understanding of the subject.

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Conflicts of interest

There are no conflicts of interest.

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