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A Study Of Recovery Pattern Of Aphasia In Stroke Patients.

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ABSTRACT

Stroke is defined as a focal or at times global neurological impairment of sudden onset, lasting more than 24 hrs. and of vascular origin. Cerebrovascular accidents rank among the leading causes of death and physical disability in the general population. Of the stroke population 20 – 30 % suffer from communication deficits and aphasia due to brain tissue damage. This interferes adversely with the patient's physical, mental, and occupational rehabilitation. So thorough knowledge of aphasia is essential for stroke rehabilitation. To study the time course of spontaneous recovery of aphasia in stroke patients. To compare the recovery patterns between different types of aphasia. A detailed history was taken and a complete clinical examination was done. Blood biochemical analysis, hemogram, ECG, and Echocardiogram were done. CT scan brain was taken. The bedside analysis of aphasic disorders entails the systematic testing of six aspects of language function. On a 10-point scale, overall language function improved by a mean of 1.07 points per week, confidence interval [0.46, 1.71], with 30 of 21 patients showing positive changes. The trajectory of recovery was approximately linear over this period. There was significant variability across patients, and patients with more impaired language function at Day 2 poststroke experienced greater improvements over the subsequent 2 weeks. Patterns of recovery differed across language domains, with consistent improvements in word finding, grammatical construction, repetition, and reading, but less consistent improvements in word comprehension and sentence comprehension. Overall language function typically improves substantially and steadily during the first 2 weeks after stroke, driven mostly by recovery of expressive language. Information on the trajectory of early recovery will increase the accuracy of prognoses and establish baseline expectations against which to evaluate the efficacy of interventions. Our findings have an immediate clinical application in that they will enable clinicians to estimate the likely course of recovery for individual patients, as well as the uncertainty of these predictions, based on acutely observable neurological factors.
Keywords: Cerebrovascular Accident, Ischemic Stroke, Aphasia, Languages, Speech

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INTRODUCTION

Aphasia is one of the most common and debilitating consequences of stroke. Fortunately, most stroke patients experience some degree of recovery of speech and language function over time [1]. Recovery has a decelerating time course, with the greatest gains taking place early, and the slope of change then decreasing [2]. Several decades of work have established that the primary predictors of the extent of recovery, or outcome, are lesion location and lesion extent, and especially lesion extent in key posterior perisylvian regions [3]. In contrast, demographic factors such as age, sex, handedness, and education have minimal predictive value. Despite much productive research on recovery from aphasia after stroke, it remains challenging to translate what has been learned into clinical practice, specifically for accurate prognostication, which is important for educating patients and caregivers and planning rehabilitation services [4]. The overarching goal of the present study was to provide a clear and comprehensive description of patterns of recovery that will allow clinicians to answer a pressing question that arises every day in stroke units all over the world: for a given pattern of brain damage, to what extent can speech and language deficits be expected to resolve over time [5]. Some studies have investigated recovery in cohorts of individuals with aphasia that were defined in whole or in part by lesion location. These studies have been very informative, but all have had relatively small sample sizes, and most have investigated patients with just one or two different lesion sites, with no studies providing a comprehensive and comparative description of a range of different patterns of damage [6]. Other studies have investigated the predictive value of damage to specific brain regions, especially posterior perisylvian regions. While these studies have firmly established the critical role of the posterior perisylvian cortex in determining aphasia outcome, which concords with other sources of information, the data have not been presented in such a way as to allow clinicians to determine likely outcomes for patients with particular lesion locations [7,8].

MATERIALS AND METHODS

This Cross-sectional prospective study. Patients who developed aphasia with or without other neurologic deficits due to acute stroke were admitted to the Department of General Medicine, Government Medical College, and Hospital, Namakkal, Tamil Nadu, India in the year between 2021-2022.

Inclusion Criteria

- Right-handed persons fulfilling the criteria for handedness.
- Patients with lesions in the left hemisphere as confirmed by CT scan.
- Patients with Tamil as their mother tongue.
- Patients with normal hearing threshold
- Patients coming for regular follow-up

Exclusion Criteria

- Pre-existing language or speech disorder.
- Psychiatric disease or previous stroke.
- Aphasia was secondary to head trauma and tumor.
- Equivocal handedness.
- Evidence of right hemisphere lesion.

A detailed history was taken and a complete clinical examination was done. Blood biochemical analysis, hemogram, ECG, and Echocardiogram were done. CT scan brain was taken. we extracted anonymized IPD on demography (age, sex, handedness, language of assessment, education level, socioeconomic status), time from stroke onset to inclusion in the primary research study, type of stroke, study design, language outcome data (overall language ability, auditory comprehension, naming, other spoken language, reading comprehension, writing, and functional-communication), and timing of language assessment. Assessment instruments were categorized by the language domain measured; categorizations were reviewed, discussed, and accepted by the Rehabilitation and Recovery of People With Aphasia After Stroke Collaborators a priori. We retained complete IPD on language domain assessments where available at both baseline and first follow-up. The bedside Language function was assessed by a Tamil version of the modified western aphasia battery system at the end of the 4th week (T1) and repeated at the 8th week (T2) 16th week (T3) and at the 24th week (T4) with a variation of plus or minus one week.

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RESULTS

Out of 30 patients,10 had diabetes. Out of 10,3 developed global aphasia,2 Broca,3 Wernicke which showed poor recovery and 2 patients developed conduction aphasia and showed good recovery. Out of 30 patients, 14 patients had high blood pressure as a risk factor. Out of 14,5 patients developed global aphasia,3 patients developed Broca,3 patients developed Wernicke, 2 had conduction aphasia and 1 patient developed transcortical motor aphasia. Out of 30 patients, 22 patients were smokers. 8 developed global aphasia, 9 developed Broca's aphasia, 2 developed Wernicke's aphasia, 2 had conduction aphasia and 1 developed transcortical motor aphasia. Out of 30 patients, 6 patients were alcoholics.4 patients developed Broca's aphasia,1 developed Wernicke's aphasia and 1 patient developed global aphasia.

Table 1: Risk Factors Of Stroke And Types Of Aphasia.

Risk factor	N	Global	Broca	Wernicke	Conduction	TCM
HT	14	5	3	3	2	1
DM	10	3	2	3	2	0
Smoking	22	8	9	2	1	2
Alcoholic	6	1	4	1	0	0

Table 2: Global Aphasia And Test Scores

Case No.	T1(4wks)(%)	T2(8wks)(%)	T3(16wks) (%)	T4 (24wks) (%)	The initial type of aphasia	Final evolution
1	2.8	3.6	4.2	4.2	Global	Global
2	2.2	2.4	2.6	2.6	Global	Global
3	2.2	3.6	4.0	4.0	Global	Global
4	3.0	8.0	16.0	18.0	Global	Global
5	4.8	5.6	8.6	10.0	Global	Global
6	2.6	3.8	6.2	9.8	Global	Global
7	8.8	36.4	48.0	62.0	Global	Broca
8	13.4	38.6	54.0	60.0	Global	Broca
9	13.6	38.8	56.0	64.0	Global	Broca
10	4.8	5.8	8.8	12.0	Global	Global
11	4.8	5.6	8.6	12.0	Global	Global

Table 3: Broca's Aphasia And Test Scores

Case No.	T1(4 Weeks) %	T2 (8 Weeks) %	T3 (16 Weeks) %	T4 (24 Weeks) %	Initial type of aphasia	Final Evolution
12	60.2	70.4	70.8	95.2	Broca	TCM
13	36.0	48.0	49.0	52.0	Broca	Broca
14	26.0	33.2	42.2	50	Broca	Broca
15	21.0	23.0	25.0	28.0	Broca	Broca
16	56.4	65.2	70.0	90.4	Broca	TCM
17	22.0	31.0	40.0	50.0	Broca	Broca
18	21.0	23.0	25.0	28.0	Broca	Broca
19	22.0	31.0	42.0	52.2	Broca	Broca
20	22.0	33.0	46.0	54.0	Broca	Broca

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Table 4: Wernicke's Aphasia And Test Scores

Case No.	T1(4 Weeks) %	T2(8 Weeks) %	T3(16 Weeks) %	T4(24 Weeks) %	Initial type of aphasia	Final evolution
24	24.6	35.0	36.0	36.0	Wernicke	Wernicke
25	20.0	26.0	26.8	34.0	Wernicke	Wernicke
26	20.0	26.2	26.8	36.0	Wernicke	Wernicke
27	22.0	26.0	26.0	28.0	Wernicke	Wernicke
28	20.0	28.0	32.0	36.0	Wernicke	Wernicke

Table 5: Conduction Aphasia And Test Scores

Case No.	T1(4 Weeks) %	T2 (8 Weeks) %	T3 (16 Weeks) %	T4 (24 Weeks) %	Initial Type of Aphasia	Final Evolution
21	81.7	88.0	92.8	94.0	Conduction	Conduction
22	58.4	72.0	92.0	92.0	Conduction	Anomic
23	53.4	68.0	86.0	92.0	Conduction	Anomic

Table 6: Transcortical Motor Aphasia And Test Scores

Case No.	T1(4 Weeks) %	T2 (8 Weeks) %	T3 (16 Weeks) %	T4 (24 Weeks) %	Initial Type of Aphasia	Final Evolution
29	50.0	64.0	78.0	92.0	TCM	Normal
30	50.0	66.0	80.0	94.0	TCM	Normal

TABLE 7: PROGNOSIS OF VARIOUS APHASIAS

Aphasia type initially	N	Poor 0-25	Fair 26-50	Good 51-75	Excellent 76-100
Global	11	8	0	3	0
Broca	9	2	0	5	2
Wernicke	5	1	4	0	0
Conduction	3	1	0	0	2
Transcortical Motor	2	0	0	0	2

Table 8: Initial Scores And Outcome Of Various Aphasias

Type of Aphasia	N	Initial AQ (%) T1 (4 Weeks)		Final AQ (%) T4 (24 Weeks)		Outcome
		Range	Mean	Range	Mean	
Global	11	2.2 – 13.6	5.72	2.6 – 64.0	23.5	Poor
Broca	9	21.0 – 60.2	31.0	25.0 – 95.2	58.0	Good
Wernicke	5	26.0 – 24.6	21.0	28.0 – 36.0	33.0	Fair
Conduction	3	58.4 – 81.7	70.0	92.0 – 94.0	93.0	Excellent
TCM	2	50.0 – 52.0	51.0	92.0 – 94.0	93.0	Excellent

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Table 9: Evolution Of Aphasia

Initial Aphasia	N	End Stage	N
Global	11	Global Broca	12
Broca	9	Broca TCM	8
Wernicke	5	Wernicke	5
Conduction	3	Conduction Anomic	4
TCM	2	Normal	1

DISCUSSION

The nature and severity of acute aphasia severity was strongly determined by lesion location and extent. Much of the variance in subsequent trajectories of recovery could be explained by initial scores and the effect of time, but lesion location and extent also made a major contribution to predicting the extent of recovery. In certain cases, knowledge of lesion location and extent can lead to very different predictions regarding outcome. For example, for a hypothetical individual with an initial QAB overall score of 2.0, a model without lesion factors provides a point estimate of 6.5 (moderate aphasia) for the QAB overall score at the 1-year time point [8]. However, the model incorporating lesion information yields different estimates depending on lesion location and extent. Our model predicts, for a patient with an F– lesion of average extent: 8.4 (a mild aphasia); for a patient with a TP+ lesion of average extent: 5.8 (a moderate aphasia); and for a patient with an FTP lesion of average extent: 3.6 (a severe aphasia). With some training in neuroanatomy, it should be feasible for clinicians to determine what group a patient belongs to based on examining their clinical MRI or CT images, and we do not envisage that the time-consuming lesion delineation and normalization procedures that we carried out would be necessary in clinical practice [9]. Compared to lesion location and extent, other potential explanatory factors made minimal contributions to explaining recovery. Older age and hemorrhagic stroke were modestly associated with lower initial scores, while none of the factors examined (age, sex, handedness, education, and stroke type) were predictive of the extent of recovery [10]. The amount of speech-language therapy received was also not predictive of recovery, though this finding should be interpreted cautiously since this was an observational study, provision of therapy and compliance with therapy may have been related to other factors predictive of recovery, and the nature and quality of therapy varied widely and was not quantified [11]. Many studies have focused on the recovery pattern of aphasia in stroke patients but there is no uniformity. The patients were not segregated based on the etiology of the stroke. In our study, we have focused only on ischemic and hemorrhagic stroke, excluding trauma, infections, degeneration, tumors, and vasculitis. This study focused on the recovery pattern of aphasias in 30 stroke patients with a Western aphasia battery system [12]. There was a good correlation between the anatomical location of the lesion and the CT brain. Separate lesion sites for Broca's, Wernicke's, global, and transcortical motor aphasias were demonstrated on the CT brain. The lesion sites were consistent with Geschwind's concept of aphasia. Patients whose computerized tomography scans show large dominant hemisphere lesions, many small lesions, or bilateral lesions are less likely to recover than those with smaller or fewer lesions Haemorrhages compress cerebral tissue without destroying it, so recovery from aphasia is better in hemorrhagic strokes than in ischemic strokes although hemorrhages are more fatal [13]. Aphasias due to vascular lesions may be due to ischemic strokes – embolic and thrombotic lesions. Deficit due to embolism may be sudden and maximal at the onset. Deficit due to thrombosis gradually increases and may be waxing and waning. Wernicke's aphasia is mostly due to an embolic lesion in the inferior division of the left middle cerebral artery [14]. Global aphasia is due to embolus to MCA, thrombosis of ICA, and may also be due to hemorrhage into deep basal ganglia due to hypertension. Broca's aphasia is mostly due to a lesion in the superior division of the left middle cerebral artery. Transcortical aphasia is mostly due to water-shed zone infarct between anterior and middle cerebral arteries and at times may be due to occlusion of the anterior cerebral artery. Pure alexia without agraphia may be due to occlusion of the posterior cerebral artery [15]. This suggests that cerebral hypoperfusion is an accurate indicator of aphasia severity in early stroke. The increased perfusion adjacent to the lesion may be crucial for early recovery in aphasia. Critically hypo perfused tissue within the central ischemic region may have a more rapid evolution to irreversible damage and a shorter therapeutic time window for tissue salvage than tissue bordering this zone, in which additional secondary and delayed cellular mechanisms may underlie progression to cell death [16]. This model of penumbral evolution supports early reperfusion but indicates that the location of the penumbra and its distribution in gray and white matter compartments may influence the choice of adjunct therapeutic strategies such as neuroprotection and the time window for their effectiveness. Subsequent language

recovery and the long-term recovery in aphasia may be related to slow and gradual compensatory functions in the contralateral hemisphere, specifically in the homotopic frontal and thalamic areas [17]. Reorganization of structure and function through the expression of neural plasticity plays a crucial role in the recovery of language at least during the subacute phase of weeks to months after the occurrence of an injury [18-20]

CONCLUSION

The recovery pattern of thirty patients with aphasia was studied over six months by measuring language performance at the 4th week,8th week, 16th week, and 24th week using a Tamil version of a modified western aphasia battery in consultation with speech therapists and specialists. Maximum recovery was noted in patients with transcortical motor aphasia and conduction aphasia. Out of three patients with conduction aphasia, two transformed into anomia. Patients with transcortical motor aphasia became normal. Patients with Broca's aphasia showed fair recovery. Two patients out of nine evolved into transcortical motor aphasia. Even though, global aphasia showed poor recovery, auditory word recognition was noted to improve. Three evolved into Broca's aphasia. Among showing recovery groups, significant improvement was noted from the 8th week of stroke. Initial severity and outcome correlated significantly. Patients with initial high scores indicating mild involvement improved better. There was a good correlation between the clinical–anatomical location of the lesion and the CT scan brain. The neurolinguistic aspect of speech recovery in stroke patients and the psychosocial aspects of stroke patients are further continued as an ongoing study in our hospital and are still under follow-up as a continuation of the present study.

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