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PROJECT REPORT

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MONTH & YEAR OF SUBMISSION : **NOVEMBER-2004**

PROJECT REPORT

TITLE OF PROJECT: APHASIA IN ACUTE STROKE- A PROSPECTIVE STUDY OF CLINICORADIOLOGICAL CORRELATION, PATTERNS AND PREDICTORS OF RECOVERY

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CERTIFICATE

I, *DR RUCHIR DIVATIA* Hereby declare that I have actually carried out the project, under report.

Place: Thiruvananthapuram
3rd November 2004

Signature: _____

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Forwarded: He has carried out the project, under report.

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INTRODUCTION

Localization of aphasia has been a center of controversy from the times of Paul Broca (1824-1880) and Carl Wernicke (1848-1905). Many post-mortem and radiological studies have determined the patterns of association between circumscribed brain lesions and aphasia syndrome. Most classical clinical-anatomic correlations have been established in small series of patients using post-mortem analysis. Several studies conducted in large groups of patients have systematically examined lesion location according to aphasia type. They have shown that an unexpectedly large proportion of aphasias deviate from classic clinical-anatomic correlation. Several studies have formally examined the association between aphasic syndrome and lesions localization using a systematic neuroradiologic analysis. They failed to demonstrate any consistent association, especially in subcortical lesions and questioned the existence of anatomy of aphasia. (1) However, Kreisler et al (2) have conclusively shown that the lesion location is the main determinant of aphasia in the acute stage. In his study, clinicoradiologic correlation supported the classic anatomy of aphasia. Stroke is one of the ideal situation to study the function of specific brain areas, especially in situations in which it is localized to specific functional areas. The study of brain behavior

assumes that abnormality of imaging signal is associated with major dysfunction or death of previously intact nervous system.

Recovery patterns following aphasia in stroke has been studied by several investigators since early 1940s. However, published reports from India have been few in these areas.

This study aims to establish the types of aphasia in patients with stroke and also proposes to study clinicoradiologic correlation, recovery patterns and predictors of recovery for aphasia.

REVIEW OF LITERATURE

(1) Definition

The most acceptable definition of aphasia would be *"acquired loss of language due to cerebral damage characterized by errors in speech (par aphasia), impaired comprehension and word finding difficulty"* (3).

Though alexia, agraphia, acalculia and apraxia are argued to be an integral part of deficit but aphasia is sufficiently distinct from these abnormalities.

(2) Historical perspective

References to speechlessness have been found even in ancient medical writings, which include the Edwin Smith Surgical Papyrus an Egyptian manuscript that dates up to 1700 B.C.

The first major study of an aphasic disorder was by Johann Gesner entitled 'speech amnesia' in 1770. Jean Baptiste Bouillaud (1796-1818) classified aphasic disorders for the first time into two basic types, one being articulatory and the other amnesic in nature. This was the precursor of 'fluent' and 'non fluent' types. Franz Joseph Gall (1758-1828) first proposed the theory that human brain is an 'assembly of organs' with specific cognitive abilites and that there

are two 'organs' of language, one for the speech articulation and the other for memory. This provided Paul Broca (1824-1880) impetus to examine brains of two aphasia patients in whom he showed that lesion responsible for nonfluent aphasic disorder was situated in both cases in left frontal lobe. Though this was probably discussed by Marc Dax in 1836 prior to Broca's description which resulted in one of the most debated controversies in Neurology. Subsequently German neurologist Carl Wernicke (1848-1905) demonstrated the occurrence of 'amnesic'type of aphasia related to left temporal lobe disease.

By early 20th century there were two schools of thought prevailing:

1. 'Associationist' school which conceptualized discrete cortical and subcortical centers and their connections as the neurological basis of language.

2. 'Cognitive' school that viewed aphasia as a single disorder that necessarily incorporated the component of intellectual defect.

The path breaking and influential paper by Norman Geshwind in 1965, in which agnosic, apraxic and aphasic disorders are interpreted as product of neural disconnection provide the current approach to the anatomical studyof aphasic syndromes.

(3) Classification

The issue of classification has been a topic of controversy in aphasic syndromes as whether aphasia is a unitary disturbance or there are several types of aphasia. It is recognized that there are distinct clinical syndromes of aphasia that recur regularly. The topographical distribution of language function in brain and topography of arterial occlusions in stroke are combined to provide a recognizable and reproducible taxonomy of disorders. Cluster analytic studies have supported the distinctiveness and significant clustering of most individuals in a stroke population of aphasics rather than even distribution (Kertesz 1979).

However, there are several limitations to this, such as,

(1) Numerous transitional forms

(2) Evolution of aphasic types

(3) Disagreement among observers

Also the purpose and methodology (linguistic v/s clinicoanatomical) of classification differs widely. Following table shows how classification of aphasia has evolved over time.

The Classifications of Aphasia

Principal author	*Year*	*Broca's-Motor*		*Wernicke's- sensory*		*Conduction*	*Anomic*	*Global*	*Trans cortical*
Broca	1861	Aphemia					Verbal amnesia		
Wernicke& Lichtheim	1874 1886	Subcortical Motor	Motor	Sub Cortical Sensory	Sensory	Conduction	Amnesic	Global	TM, TS
Head	1926	Verbal		Syntactic		-	Nominal	-	Semantic
Goldstein	1948	Peripheral motor	Central Motor	Peripheral sensory	Central sensory	Central	Amnesic	Global	TM, TS, Isolation
Benson & Geschwind	1971	Aphemia	Broca's	Pure word deafness	Wernicke's	Conduction	Anomic	Global	TM, TS, Isolation
Goolglass & Kaplan	1972	Broca's		Wernicke's			Anomic	Global	TM, TS, Isolation
Kertesz	1979	Pure motor	Broca's	Wernicke's		Efferent - afferent Conduction	Anomic	Global	TM, TS, Isolation
				Pure Word deafness	Neologistic jargon				

Characteristics of aphasia syndromes

Type of aphasia	Spontaneous speech	Naming	Comprehension	Repetition
Global	Mute or nonfluent	Impaired	Impaired	Impaired
Broca's	Nonfluent, mute or telegraphic	Impaired	Intact	Impaired
Wernicke's	Fluent	Impaired	Impaired	Impaired
Transcortical motor	Nonfluent	Impaired	Intact	Intact
Transcortical sensory	Fluent	Impaired	Impaired	Intact
Isolation	Nonfluent, echolalic	Impaired	Impaired	Intact
Conduction	Fluent	Moderately impaired	Intact	Severely impaired
Anomia	Fluent	Impaired	Intact	Intact

(4) Assessment of aphasia

Broca himself tested his second patient named Lelong with conversational questions, writing, arithmetic and also described his gestures and tongue movements.

Formal aphasia testing is a relatively recent method of measurement. Several batteries of tests have been described

- Boston diagnostic aphasia examination (BDAE) by Goodglass and Kaplan (1972) assesses conversational speech, auditory comprehension and oral expression. Reading, writing and supplementary language tests explore various psycholinguistic factors.

- Western aphasia battery by Kertesz (1982) mainly aims to classify various aphasic syndromes and to evaluate the severity of aphasic impairment. This has four language subtests - spontaneous speech, comprehension, repetition and naming with three performance subtests writing, praxis and construction.

The summary score is the Aphasia Quotient (AQ) and it has been used extensively to follow the recovery of stroke patients. It is the only test that provides score based criteria to classify each patient. Validation studies have shown interest reliability.

(5) Language processes and cerebral localization

Hughling Johnson's warning that 'to localize the damage which destroys speech and to localize speech are two different things' is still valid.

Wernicke's "Diagram" and Lichtheim's "House" were the earliest hypothesis regarding the localization of aphasia but these were refuted by Henry Head (1926) who described them as "diagram makers". Neuroanatomical correlations also lost credibility because of work of Von Monakow (1914) who showed the extensive distance effect of tumors, diaschisis due to large sudden lesions and emphasized the variability of behavior produced by these factors.

The various methods employed in clinico-anatomical correlation of aphasias include autopsy studies, radioisotope studies, CT scan, MRI and now MRI and PET studies.

(A) Radioisotope scans

It has been used to provide localization in aphasias with brain tumors or stroke. This showed lesions for various aphasia in a study (4) as follows.

Radioisotope localization of aphasia in stroke

Number	Type of aphasia	Localization
1	Global aphasia	Inferior frontal, superior temporal and sylvian fissure, basal ganglia and large area of subcortical white matter
2	Broca's aphasia	Smaller more anteriorly located lesions invariably involving 3rd frontal convolution
3	Wernicke's aphasia	Parietal and temporal invariably involving superior temporal gyrus
4	Conduction aphasia	Superior lip of sylvian fissure consisting of inferior ends of pre central, post central, inferior parietal and supramarginal gyri
5	Transcortical motor	Perirolandic portion,ACA territory in one patient
6	Transcortical sensory	Posterior parietal predominantly sub cortical
7	Isolation aphasia	Watershed areas of cerebral circulation
8	Amnesia	Posterior parietal, temporal lesions usually sparing superior temporal gyrus.

The limitations of this technique are:

- Variability of technique
- Difficulty in transferring the areas of isotope uptake to anatomical structures.

(B) CT scan

A substantial number of CT scan studies have contributed to localization of aphasia. A review of various studies with CT scan showing localization of aphasia follows:

(i) Global aphasia

Naeser et al (5) (1977)	Large portion of frontal, temporal and parietal lobes - cortical and sub cortical
Hayward et al (6) (1977)	Large lesions of frontal, parietal and temporal lobes
Hojo et al (7) (1984)	70% had extensive lesions involving both Broca's and Wernicke's areas. Some patients had small and confined lesions
Poeck et al (1) (1993)	Large anterior and posterior lesions. Some patients had small lesions.

As seen above some of the studies have shown unexpected findings with small lesions even with persisting global aphasia.

(ii) Broca's aphasia

Naeser et al (5) (1977)	Lateral aspect of left anterior horn of lateral ventricle out to cortical presentation of Broca's area Inferior portion of motor strip, caudate, putamen, globus pallidus, anterior limb of internal capsule, insula
Hayward et al (6)(1977)	Area lateral to inferior portion of frontal horn of lateral ventricle, fronto parietal operculum,insula. Extension to motor strip
Hojo et al (7) (1984)	Deep structures of lower part of precentral gyrus, insula, lentiform nucleus.
Alexander et al (8) (1990)	Frontal operculum, lower motor cortex, subcortical white matter, limbic frontal pathway of medial subcallosal fasciculus.

Clinical radiological correlation for Broca's aphasia is weaker compared to other aphasic syndrome. In the study by Hojo et al. only 60% of patients with persistent Broca's aphasia had lesions in the characteristic areas. Similar finding has been reported by others. (1)

(iii) Wernicke's aphasia

Naeser et al (5) (1977)	Wernicke's cortical area, parietal lobe - supramarginal gyrus, deeper white matter including optic radiations.
Hayward et al (6)(1977)	Wernicke's cortical area.
Hojo et al (7) (1984)	Wernicke's cortical area, subcortical lesions of superior and middle temporal gyri.

Overall, studies have shown a good clinicoradiologic correlation for Wernicke's aphasia. (1,7)

Transcortical motor aphasia is usually seen with lesions involving Broca's area and structures deep to it, small lesions scattered anteriorly and superiorly to Broca's area in frontal region. (5) This is the only aphasic syndrome seen with ACA territory infarcts.

Transcortical sensory aphasia is usually seen with posterior parieto occipital locations, which can be separated into 2 groups,

(1) Medial, inferior and posterior lesions in PCA territory.

(2) Lateral, superior and anterior in watershed between MCA and PCA territories. (9)

Mixed Transcortical aphasia has been described with watershed area infracts which fall between ACA and MCA territories. (3) Conduction aphasia is usually localized to posterior part of superior temporal lobe and inferior part of supramarginal gyrus. However,some studies have shown that Wernicke's area is usually spared and lesions are usually deep to it involving the arcuate fasciculus. (5)

Anomia is the least localizable of all types of aphasia. It has been described with widely scattered lesions including parietal, temporal or even Broca's area, striatum, insula, and thalamus. (3)

Aphasia with subcortical lesions

The role of subcortical structures is less well-known in language than their motor function.

Naeser et al (10) have described three subcortical aphasia syndromes based on CT findings

Subcortical Aphasia – Lesion locations & Language abnormality

	Lesion location	*Language abnormality*
1	Capsular/Putaminal with anterior superior white matter extension	Good comprehension but slow, dysarthric speech
2	Capsular/Putaminal with posterior extension	Poor comprehension, fluent Wernicke's type speech
3	Capsular/Putamanial with anterior-superior and posterior extension	Globally aphasic

Thus, these three types of subcortical aphasia resemble the more classic types of aphasia seen with cortical involvement.

Thalamic lesions have been reported to cause a peculiar aphasia syndrome with anomia,persevertion and at times neologisms with intact comprehension and repetition which is similar to transcortical sensory aphasia. (Mohr et al,1975)

However, current views on subcortical aphasia are different as reviewed by Nadeau et al (11). Absence of aphasia in 17 reported cases of dominant hemisphere striatocapsular infarction and finding of nearly every conceivable pattern of language impairment in 33 different reported cases of striatocapsular infarction provides evidence against a major direct role of basal ganglia in language. Detailed consideration of vascular events leading to striatocapsular infarction suggests that associated linguistic deficits are predominantly related to sustained cortical hypoperfusion and infarction not visible on structural imaging studies. Thalamic disconnection, as may occur with striatocapsular infarcts with extension to temporal stem and putaminal hemorrhages may contribute to language deficits in some patients. Also head of the caudate nucleus may have an important role in language.

(C) Localization with MRI

In a recent study, by Kreisler et al (2) 107 patients were examined with a standardized aphasia battery and MRI.

The lesion locations for various aphasic syndromes can be summarized as follows:

Lesion localization in Aphasia – MRI study (Kreisler et al)

Aphasic syndrome	No.	Main lesions (left hemisphere)
No aphasia	23	
Global	17	Large anterior posterior (n=16) Posterior region, striatum and insula (n=1)
Broca's	10	Insula-external capsule region (n=7) Rolandic operculum (n=6) Inferior frontal gyrus (n=6) Central region (n=6) Anterior part of temporal gyri (n=5)
Wernicke's	10	Insula-external capsule region (n=8) Posterior part of temporal gyri (n=7) Anterior part of temporal gyri (n=6) Inferior parietal lobulus (n=4)
Transcortical motor	3	Thalamus (n=1) Thalamus, striatum, and insula (n=1) Insula, striatum, and central and parietal cortices (n=1)
Transcortical sensory	2	Large perisylvian and deep areas (n=1) Fronto-parietal cortices, centrum semiovale (n=1)
Subcortical	3	Thalamus (n=1) Thalamus, internal capsule, centrum semiovale, striatum (n=2)
Anomia	3	Thalamus (n=1) Medical temporal (n=1) Frontal cortex, insula, anterior part of temporal gyri (n=1)
Nonclassified	21	Various
Word finding difficulty	7	Various

In addition, specific language deficits were also correlated with various locations.

Language deficits and lesion locations – MRI study (Kresler et al)

	Language deficit	Lesions location
1	Mutism	Fronto-putaminal lesion
2	Low fluency	Inferior frontal gyrus Putamen Anterior centrum semiovale extending to putamen or inferior parietal lobule
3	Repetition disorder	External capsule or posterior area of internal capsule
4	Oral comprehension	Posterior part of temporal gyri Inferior frontal gyrus
5	Impairment of picture naming & word finding difficulty	Large variety of lesions involving anterior and posterior cortex Thalamus
6	Verbal paraphasia	Temporal or caudate lesion
7	Phonemic paraphasia	External capsular lesion extending to posterior part of temporal lobe or interior capsule
8	Perseveration	Head of caudate

(6) Recovery following aphasia

Aphasic syndromes are not stable as a rule but show a variable degree of recovery especially in the first 3 months following a stroke. Following the initial period of 2-3 weeks when edema subsides there is a second stage of recovery which goes on for months or years.

Sudden lesion as occurs in a large stroke causes much more damage then one would observe from a slowly growing lesion such as a tumor of same size. This has been attributed to shock effect on surrounding tissue, called diaschisis.

(A) Spontaneous recovery

There is a general agreement that some natural recovery takes place in majority of patients with or without any intervention. However, there is a lack of consensus regarding duration of spontaneous recovery period. Most investigators have found that maximum recovery occurs in first 2-3 months following ictus but this continues even beyond 6 months. Though some authors believe that spontaneous recovery does not occur after 1 year. (12)

(B) Age and recovery

Age is considered as an important variable in outcome but various studies have failed to show a consistent effect of age on outcome.

Kertesz et al. (12) reported an inverse correlation, in other words younger the patient higher the initial recovery rate. However other investigators have found it to be a weak factor (13).

(C) Gender and recovery

Observations on gender and recovery from aphasia in stroke are also variable. However some studies have shown females to have a better recovery which has been further explained on the basis of more bilateral presenation of language in females. (13)

(D) Type and severity of aphasia and recovery

Kertesz and McCabe noted that Broca's aphasias had the highest rate of recovery and lowest rate of recovery occurred in untreated global aphasic and anomic groups.

Wernicke's aphasia had a bimodal pattern of recovery, those with initially low scores generally did poorly, those with higher scores had better prognosis (12).

(E) Neuroradiologic correlates of recovery

Yarnell et al. studied 14 aphasic patients 8 months post stroke and concluded that patients with large dominant hemisphere lesion, either one large and many small ones found poorly, whereas those with lesser lesions did better. Bilateral lesions at times unrecognized clinically helped to account for significant aphasia residuals. (14)

(F) Psychosocial factors

Occupational status before illness were not found to be related to recovery. However, several other investigators have found depression, counciety, and paranomia as factors that have a negative effect on the outcome. Effect of education on recovery has not shown consistent results (12,15,36,37).

(7) Therapeutic approaches to aphasia

The role of therapeutic strategies in aphasia has been considered controversial. However, research in last 10 years has changed that concept. In a meta analysis by Robey et al, effect of therapy beginning at initial stage of recovery was nearly twice as large as effect of spontaneous recovery alone, while treatment received after the acute period achieved a smaller but appreciable effect (15).

There are several approaches to treatment of aphasia, which include output-focused therapy, which includes melodic intonation therapy (MIT), treating linguistic deficits by psycholiguistic approach and related neurological deficits by cognitive neurorehabilitation (!6).

Computer aided therapy uses a computerized visual system for patients with severe aphasia where they can use alternative symbol system for communication.

Several drugs have been used for pharmacotherapy of aphasia, which include sodium amytal, Meprobamate, Methylphenidate, Propranolol, Bromocriptine. However, currently there is no proven role of any of these in treatment of aphasia (17).

AIMS OF STUDY

(1) To study various types of aphasic syndromes in relation to stroke.

(2) To study the clinicoradiologic correlation of various aphasic syndromes.

(3) To study the course and predictors of recovery from aphasia in stroke patients.

MATERIALS AND METHODS

This is a prospective study of 95 patients with acute hemorrhagic or nonhemorrhagic strokes seen between January 2002 to august 2004.

Inclusion criteria

(1) Subject should have developed aphasia as a consequence of stroke.

(2) Referral to our center should be within three months of ictus.

(3) Subject should not have a past history of stroke.

Patients who developed another stroke during the follow up, cognitive decline or expired were excluded.

All patients were seen by a clinical neurologist and a speech pathologist at baseline and were subsequently followed up at 3,6 and 12 months interval.

The initial assessment by clinical neurologist consisted of types of aphasia, presence of dysarthria, motor, sensory or cerebellar deficits, cognitive deficits, any past history of stroke, presence of risk factors-Hypertension, Diabetes Mellitus, Hyperlipidemia, Coronary artery disease, Smoking, Alcohol intake and presence of atrial

fibrillation. Patients were also investigated with echocardiogram and neck vessel Doppler. Cerebral angiogram was done whenever indicated.

Stroke sub typing was done using TOAST classification.

All patients had neuroimaging in the form of either CT scan or MRI which were assessed by a neuroradiologist who was blinded to clinical data. Only CT scans were taken for analysis of clinicoradiologic correlation as few patients had MRI.

Language was assessed by speech therapist using Malayalam adaptation of Western Aphasia Battery (WAB). The WAB subcomponent scores were used to classify aphasia type. Based on subcomponent score aphasia quotient (AQ) was calculated as described in original manual. 12 patients had received speech therapy initially and were prescribed home therapy.

OBSERVATIONS

(A) Demography

The mean age of study population was 54.89±14.9 years (Range 22-87). Mean education was 9.4±4.614 years.

There were 68 (70.8%) male and 28 (29.2%) female patients. All were right handed individuals. There was one patient with crossed aphasia.

(B) *Time of assessment*

Table 1-Time of assessment

	No.	Min.	Max.	Mean	Standard deviation
Baseline assessment (clinical)	95	0	154	22.56	31.1
Baseline assessment (speech therapist)	49	1	378	22.22	55.0
WAB assessment - baseline	26	4	74	18.92	14.2
WAB assessment- 1st	54	43	136	104.44	17.9
WAB assessment- 2nd	30	137	225	185.17	24.5
WAB assessment- 3rd	28	274	460	374.04	43.0

All patients were assessed clinically at baseline. In addition 49 patients were assessed at bedside and 26 with WAB at baseline by speech therapist.

(C) Stroke subtype

There were 87 patients with infarcts and 8 patients with hemorrhage.

Table 2-Frequency of stroke subtypes

Subtype	Frequency	Percent
Large vessel disease	23	26.4
Cardioembolic	26	29.9
Other etiologies	5	5.7
Undetermined	26	29.9
Data insufficient	7	8.0
Total	**87**	**100.0**

Cardioambolic stroke was the commonest subtype , which is due to large number of patients with cardiological disorders being treated at our center. However, in a significant number of patients subtype was undetermined even after full evaluation.

26.4% had large vessel disease and 5 patients had other etiologies for stroke (carotid dissection 2,venous infarct 2, Takayasu's arteritis 1).

(D) Types of aphasia

84 patients were purely aphasic, 9 having aphasia with dysarthria and 2 having anarthria at initial assessment. The type of aphasia was determined by WAB assessment whenever available followed by bedside assessment and by speech therapist and clinical assessment if none of the above were available.

Table 3-Frequency of types of aphasia

Type of aphasia	Frequency (%)	Mean age	Standard deviation
Global	41 (42.7%)	54.00	16.901
Broca's	21 (21.9%)	53.00	12.538
Wernicke's	15 (15.6%)	58.60	14.681
Transcortical motor	13 (13.5%)	54.08	12.958
Transcortical sensory	2 (2.1%)	51.50	4.950
Mixed Transcortical	1 (1.0%)	86.00	
Conduction			
Anomia	3 (3.1%)	57.00	12.767
Total	**95 (100%)**	**54.89**	**14.967**

The commonest type of aphasia at baseline was global. There were no patients with conduction aphasia and only one patient with

mixed Transcortical aphasia. Mean age was maximum for Wernicke's aphasia (58.6) followed by anomia while lowest for Transcortical sensory (51.5) followed by Broca's (53.0).

Broca's aphasia were more cardioambolic in etiology (52.6%, p=0.146) and Wernicke's aphasia were more atherothrombotic (42.9%, p=0.122). However, this was not statistically significant. Etiology was undetermined in 54.6% of Transcortical motor aphasics.

Table 4-Types of aphasia and stroke subtype

Types of aphasia	Subtype (%)					Total
	Large vessel disease	Cardioembolic	Other etiology	Undetermined	Data insufficient	
Global	11 (28.9%)	8 (21.1%)	4 (10.5%)	11 (28.9%)	4 (10.5%)	38 100.0%
Broca's	3 (15.8%)	10 (52.6%)		4 (21.1%)	2 (10.5%)	19 100.0%
Wernicke's	6 (42.9%)	3 (21.4%)	1 (7.1%)	3 (21.4%)	1 (7.15)	14 100.0%
Transcortical motor	2 (18.2%)	3 (27.3%)		6 (54.5%)		11 100.0%
Transcortical sensory		1 (50.0%)		1 (50.0%)		2 100.0%
Mixed Transcortical		1 (100.0%)				1 100.0%
Anomia	1 (50.0%)			1 (50.0%)		2 100.0%
Total	23 (26.4%)	26 (29.9%)	5 (5.7%)	26 (29.9%)	7 (8.0%)	87 100.0%

(E) Clinicoradiological correlation

Neuroimaging was available in 80 (84.2%) patients, 7 patients had MRI and 70 had a CT scan. Mean duration of CT scan was 9.5 days after ictus, 74% of patients has CT done within one week of ictus, 18.2 had CT scan done on the same day of ictus.

Table 5-Frequency of type of stroke

Radiological abnormality	Frequency	Percent
Infarcts	54	74.0
Hemorrhage	7	9.6
Hemorrhagic infarcts	12	16.4
Total	**73**	**100.0**

Types of aphasia and radiological areas involved

Table 6– Types of aphasia and lesion location

Areas involved	Global aphasia (N=31)		Broca's aphasia (N=14)		Wernicke's aphasia (N=11)		Transcortical motor aphasia (N=8)	
	Frequency	%	Frequency	%	Frequency	%	Frequency	%
Caudate nucleus	6	19.4	3	18.8	1	9	4	50.0
Putamen	11	35.5	4	20.0	-	-	4	50.0
Globus pallidus	8	25.8	2	12.5	1	9	3	37.5
Anterior limb of internal capsule	7	22.6	3	18.8	1	9	3	37.5
Genu of internal capsule	7	22.6	2	13.3	1	9	2	25.0
Post limb of internal capsule	7	22.6	1	6.7	-	-	2	25.0
Insula	21	67.7	3	18.8	2	18.2	1	12.5

Areas involved	Global aphasia (N=31)		Broca's aphasia (N=14)		Wernicke's aphasia (N=11)		Transcortical motor aphasia (N=8)	
	Frequency	%	Frequency	%	Frequency	%	Frequency	%
Cortical frontal operculum	13	41.9	1	6.3	-	-	1	12.5
Sub cortical frontal operculum	13	41.9	6	37.5	-	-	1	12.5
Cortical temporoparietal operculum	22	71.0	4	25.0	5	45.5	2	25.0
Sub cortical temporoparietal operculum	21	70.0	5	31.3	5	45.5	3	37.5
Middle temporal gyrus	15	50.0	2	12.5	6	54.5	1	12.5
Superior temporal gyrus	20	64.0	3	18.8	5	45.5	2	25.0
Angular gyrus	18	60.0	3	18.8	7	63.6	1	12.5
Thalamus	1	3.2	-		-	-	-	-
Lateral occipital	8	25.8	-		4	36.4	-	-
Dorsolateral frontal cortical	10	32.3	1	6.3	-	-	2	25.0
Dorsolateral frontal sub cortical	11	35.5	4	25.0	-	-	3	37.5
Parietal cortical	12	48.0	3	21.4	6	60.0	-	-
Parietal sub cortical	3	21.4	5	35.7	7	70.0	-	-

Globus aphasics mainly had involvement of insula, cortical and subcortical frontal and tempororparietal operculum, temporal and parietal areas. Subcortical structures including caudate, putamen and globus pallidus were involved less frequently.

Subcortical frontal operculum appeared to be the most commonly involved area in Broca's aphasics. Cortical frontal operculum was involved in only one patient. Surprisingly,

temporoparietal operculum, parts of temporal gyrus and parietal areas were also involved.

Distribution of lesions in Wernicke's aphasia was concentrated in temporal and parietal regions. None of them had frontal operculum involvement. Insula was involved in 2 patients.

Subcortical structures (caudate, putamen, globus pallidus, anterior limb of internal capsule), Dorsolateral frontal cortical and subcortical areas were involved most commonly in Transcortical motor aphasia. CT scan did not show any abnormality in one patient, which was done the day of ictus.

There were only 2 patients with Transcortical sensory aphasia. One had involvement of lateral occipital area with angular gyrus and parietal cortical and subcortical involvement. Other had involvement of lateral occipital area with dorsolateral frontal involvement.

Among 3 patients with anomia CT scan of one patient was normal while one patient had involvement of angular gyrus alone. Third patient had MRI, which showed involvement of middle temporal gyrus.

Only one patient had mixed Transcortical aphasia whose MRI showed multiple infarcts involving ACA and MCA territory in medial

and dorsolateral frontal, insula with frontal and tempo parietal operculum and basal ganglia involvement.

There were 8 patients with lesions restricted to striatocapsular and thalamic areas. Radiological and clinical characteristics of these patients are described in following table.

Number	Abnormality	Areas involved	Types of aphasia	Dysarthria
1	Infarct	Caudate,Putamen, globus pallidus and anterior limb of internal capsule	Transcortical motor	Yes
2	Infarct	Thalamus	Broca's	No
3	Infarct	Genu and posterior limb of internal capsule	Broca's	Yes
4	Infarct	Caudate nucleus and corona radiata	Broca's	No
5	Hemorrhage	Caudate, thalamus, anterior limb and genu of internal capsule.	Broca's	Yes
6	Infarct	Caudate, Putamen, globus pallidus, anterior limb and genu of internal capsule, subcortical temporo parietal operculum	Transcortical motor aphasia	Yes
7	Infarct	Bilateral centrum semiovale right anterior limb of internal capsule, Pons	Broca's	Yes
8	Hemorrhage	Putamen, posterior limb of internal capsule, parietal subcortical	Broca's	Yes

Majority of patients with subcortical lesion location had infarcts, most had associated dysarthria. Caudate nucleus followed by putamen and anterior limb of internal capsule were most commonly affected structures. There was one patient with thalamic infarct and another one had a thalamic hemorrhage which also had involved striatocapsular region.

(F) Recovery patterns

Recovery patterns of various types of aphasia are summarized in the figures. The time from ictus is plotted against severity of aphasia measured as aphasia quotient (AQ). The type of aphasia is mentioned in bracket at each time point.

Global aphasia patients showed bimodal pattern of recovery with 7 patients either becoming anomic or normal and 6 remaining global at the end of one year.

Table 8-Recovered global aphasics

Number	Age	Gender	Subtype	Areas involved
1	27	Female	Undetermined	Basal ganglia, temporoparietal operculum, superior temporal & angular gyrus
2	23	Male	Other etiology (venous infarct)	Temporoparietal operculum, angular gyrus parietal cortical
3	22	Male	Cardioembolic	Basal ganglia, insula, frontal & temporoparietal operculum, Dorsolateral frontal operculum
4	30	Female	Other etiology (ICA dissection)	Striatocapsular, insula, frontal & temporoparietal operculum, superior temporal gyrus, parietal, Dorsolateral frontal.
5	45	Male	Undetermined	Striatocapsular, insula, frontal & temporoparietal operculum, superior temporal gyrus, Dorsolateral frontal subcortical
6	48	Male	Other etiology (ICA dissection)	Imaging not available
7	48	Male		Imaging not available

Table 9-Global aphasics with poor recovery

No.	Age	Gender	Subtype	Areas involved
1	78	Male	Undetermined	Insula, temporoparietal operculum, superior temporal, middle temporal & angule gyri, parietal
2	62	Male	Cardio embolic	Insula, frontal & temporoparietal operculum, superior temporal & angular gyri, Dorsolateral frontal cortical
3	51	Male	Undetermined	Basal ganglia, Insula, temporoparietal operculum, superior temporal, middle temporal & angular gyri, lateral occipital, parietal
4	35	Female	Undetermined	Insula, frontal & temporoparietal operculum, superior temporal, middle temporal & angular gyri, dorsolateral frontal cortical
5	55	Male	Undetermined	Basal ganglia, Insula, frontal & temporoparietal operculum, superior & middle temporal gyri, thalamus, Dorsolateral frontal subcortical
6	65	Male	Data insufficient	Imaging not available

Such a pattern is also seen for Broca's and Wernicke's aphasia but further analysis is difficult in view of small number.

Overall, anomia was commonest end stage of recovery. Some patients with global aphasia tend to become Broca's or Wernicke's at the end stage. One patient with Wernicke's aphasia became conduction and one transcortical sensory at the end of one year.

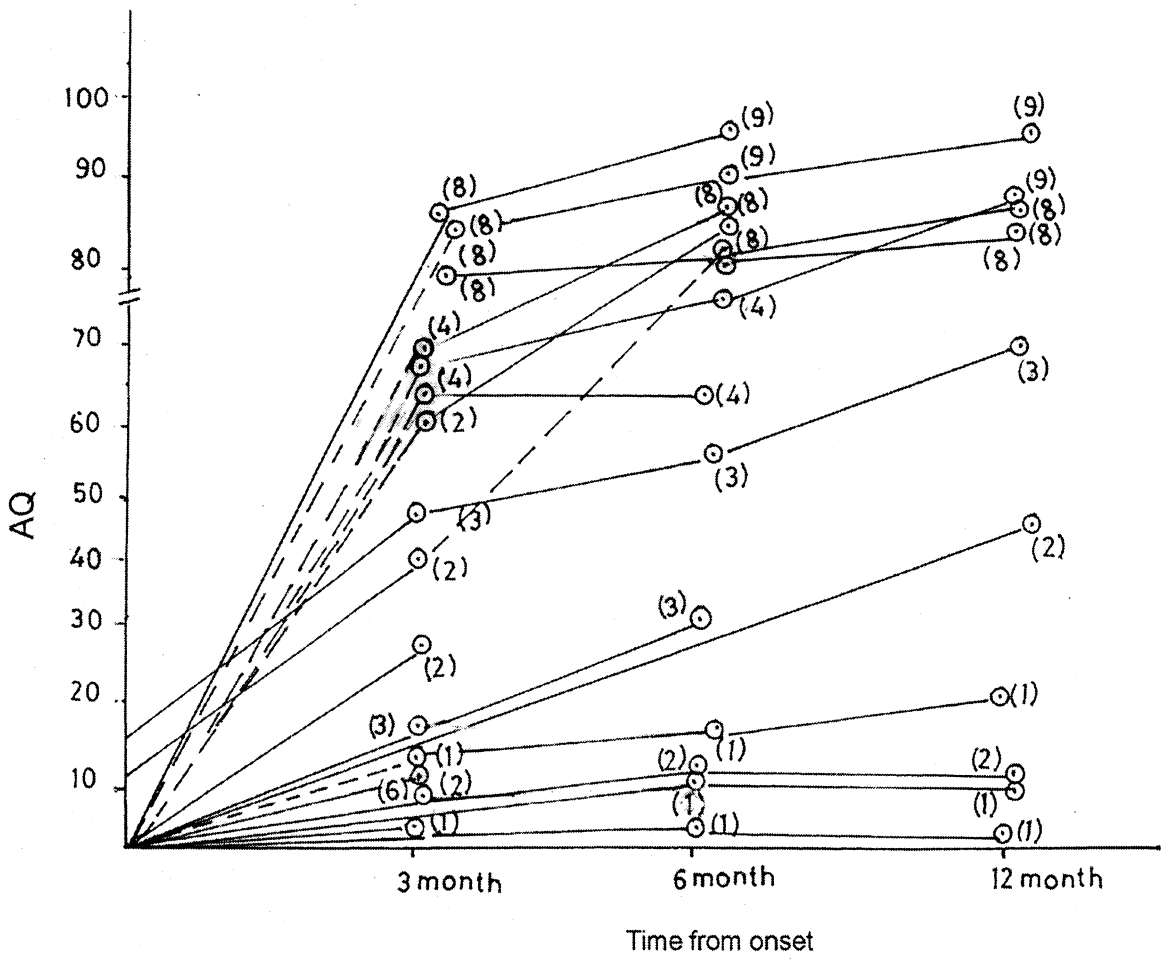

Fig 1. Global aphasia - recovery pattern

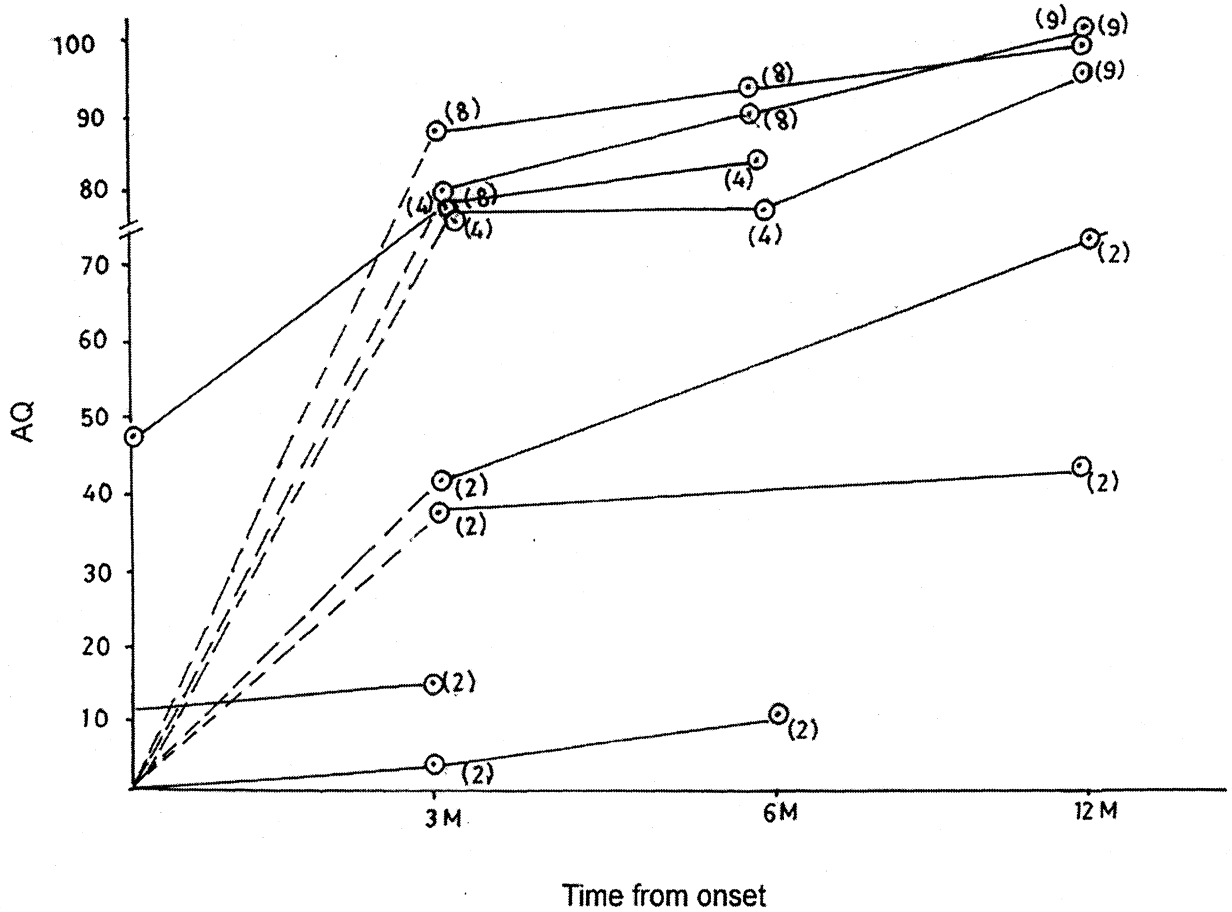

Fig 2. Broca's aphasia - recovery pattern

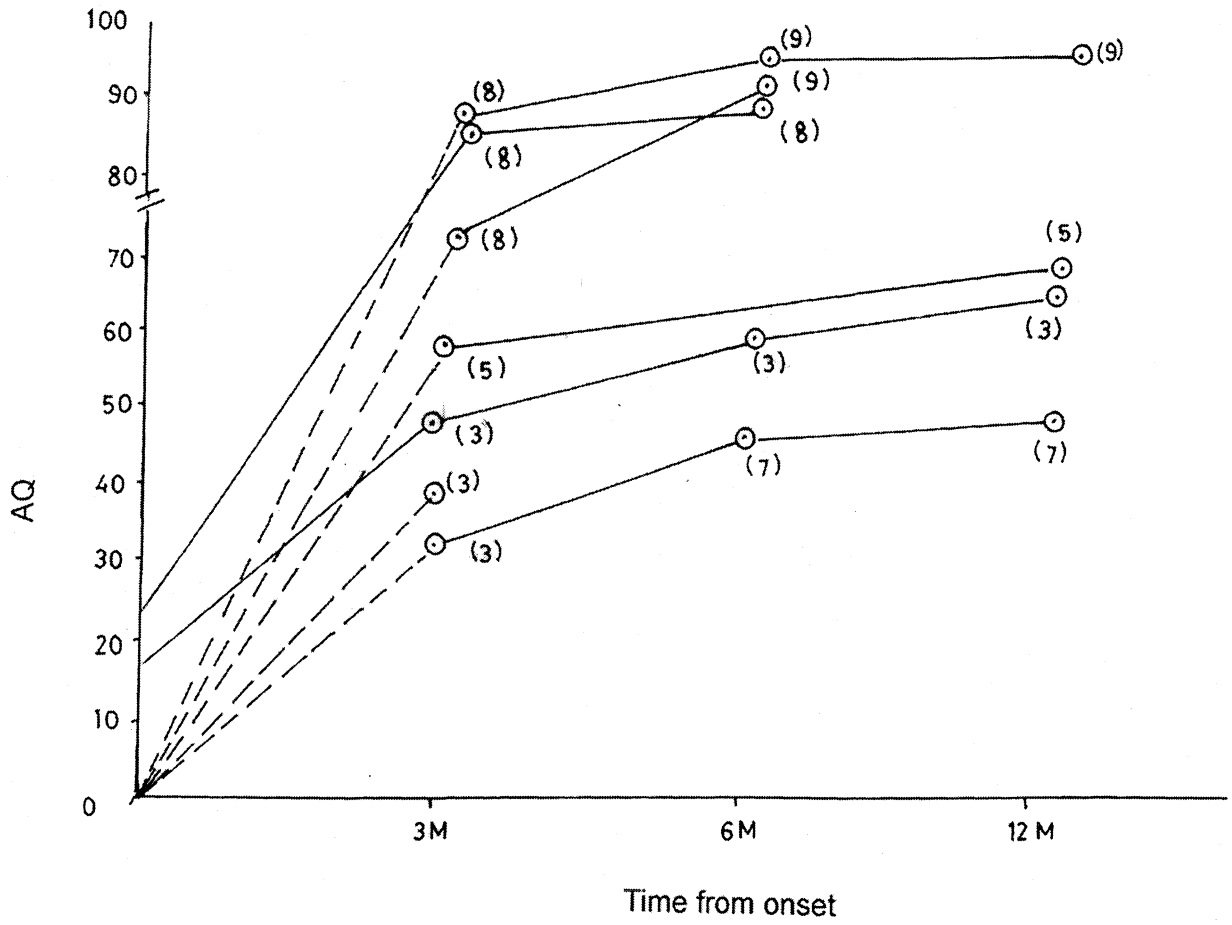

Fig 3. Wernicke's aphasia - recovery pattern

(G) ***Rate of recovery***

Rate of recovery was measured as mean change in aphasia quotient (AQ) between 0 to 3,3 to 6, 6 to 12 months post stroke.

Table 10- Rate of recovery

Period of follow up	Mean change in AQ	Standard deviation	Sig. (2-tailed)
0-3 months	20.925	9.2	0.000
3-6 months	6.088	6.0	0.000
6-12 months	4.237	4.6	0.000

Maximum rate of recovery was seen in first 3 months, which was still significant between 3-6 months and 6-12 months though to a lesser extent.

(H).Predictors of outcome

There were 39 patients with either one-year follow up or achieving good recovery before one year (anomic and normal). Univariate analysis was done for predictors like age, education, gender, AQ at baseline, stroke subtype, risk factors, infarct v/s hemorrhage & subcortical location.

Table 11 – Type of aphasia and outcome

Aphasia type	Poor outcome (AQ<75)	Good outcome (AQ≥75)	Total
Global	8 50.0%	8 50.0%	16 100.0%
Broca's	2 28.6%	5 71.4%	7 100.0%
Wernicke's	2 40.0%	3 60.0%	5 100.0%
Transcortical Motor		7 100.0%	7 100.0%
Transcortical Sensory		2 100.0%	2 100.0%
Anomia		2 100.0%	2 100.0%
Total	12 30.8%	27 69.2%	39 100.0%

Out of 12 patients with poor outcome 8 were global aphasics. 71.4%of Broca's aphasics and all patients with transcortical motor, transcortical sensory and anomia showed good recovery.

Table 12-Stroke subtype and outcome

Subtype	Poor outcome (AQ<75)	Good outcome (AQ≥75)	Total
Large vessel disease	3 37.5%	5 62.5%	8 100.0%
Cardioembolic disease	2 25.0%	6 75.0%	8 100.0%
Other etiology		2 100.0%	4 100.0%
Undetermined	4 28.6%	10 71.4%	14 100.0%
Data insufficient	2 100.0%		2 100.0%
Total	**11** **32.4%**	**23** **67.6%**	**34** **100.0%**

There was no significant difference in outcome between large vessel disease and cardioembolic stroke.

Table 13-Predictors of outcome

Predictors	Poor outcome (AQ<75) (Mean/%)	Good outcome (AQ≥75) (Mean/%)	P value
Age	59.0 ± 13.7	50.4 ± 14	0.090
Education	6.7 ± 4.9	11.4 ± 3.5	0.003
Severity at baseline (AQ)	12.4 ±18.2	64.4 ± 22.8	0.008
Subcortical aphasia	20%	80%	0.506
≥2 Risk factors	34.8%	65.2%	0.386
Types of stroke • Infarcts • Hemorrhage • Hemorrhagic infarcts	 43.5% - -	 56.5% 100.0% 100.0%	0.102

Though patients with poor outcome were older than those with good outcome difference was not statistically significant (P 0.090). However, low education and low AQ at baseline were significantly correlated with poor outcome (P value 0.003 & 0.008 respectively).

Patients with ≥ 2 stroke risk factors (HT, DH, hyperlipidemia, CAD, smoking, alcohol intake) had a trend towards poor outcome but difference was not statistically significant (P 0.386).

All patients with hemorrhage or hemorrhagic infarcts had a good outcome compared to those with infarcts. However the difference was not assuming statistically significance due to small number (P 0.102)

Out of 8 patients with subcortical aphasia follow up was available for 5; 4 of whom showed good recovery. One patient with subcortical aphasia expired during follow up.

Of the 12 patients who received speech therapy initially 36.8 % had poor outcome and 63.2 % had good outcome. However, in the group without speech therapy also outcome good in 73.7 % of patients. There was no statistical significance in outcome in these two groups. (P 0.364)

DISCUSSION

The present study describes several important aspects of aphasic syndromes in stroke patients, which are important in context of comprehensive stroke care.

(A).Types of aphasia

The most common type of aphasia encountered in acute stroke patients was global aphasia. This was seen in 42.7% of patients which correlates with several other studies (18, 19, 34).

(B).Types of aphasia, age and stroke subtype

In current study there was trend showing Broca's aphasia being more common in cardioembolic strokes and Wernicke's in large vessel disease. There are only few studies correlating stroke subtype with type of aphasia. In some of the previous studies Wernicke's aphasia has been seen more commonly in cardioembolic strokes (20,21,22). However, not all patients were investigated in these studies with echocardiography and neck vessel doppler. So subtyping may not have been accurate.

Wernicke's aphasia has been described in older patients compared to Broca's (23,24). In our study, there was no statistical significance but patients with Wernicke's aphasia tended to be older

than Broca's (58.6 Vs 53.0). This has been explained by various reasons.

(1) Change in speech organization with age such that Wernicke's aphasia could result in an older patient with a lesion in various areas in the language zone.

(2) Older patients may have a tendency for more posterior infarcts, as proximal MCA becomes more horizontal and may cause inferiorly resulting in more posterior infarcts as age advances.

(C).Clinicoradiological correlation

Clinicoradiological correlation in aphasia syndromes has several inherent limitations.

(1) Recovery is observed spontaneously in aphasia to a certain extent. This may be due to multiple presentation of language in brain. Thus correlating specific imaging abnormality to a language dysfunction may be difficult.

(2) There is a change in language deficit over a time in some patients while some remain stable. Thus timing of neuroimaging becomes very important.

In addition, in present study CT scans have been used as imaging modality which has a limitation of underestimating the lesion size. Some CT scans done on the same day of ictus were normal. MRI was available only for 7 patients, so it could not be analyzed.

With these limitations in background it is difficult to arrive at specific conclusions regarding clinicoradiological correlation. However, some observations can be discussed.

Global aphasia patients had extensive lesions including frontal operculum, temporoparietal operculum, insula, temporal and parietal regions with involvement of basal ganglia in some cases. However there were three cases where lesions were restricted to temporal gyri, angular gyrus and parietal cortex with a stable global aphasia. Thus it is possible that in patients with global aphasia and restricted lesions MRI could have picked up more lesions.

This has been seen previously in a CT scan based study when stable global aphasia has been demonstrated in patients with restricted lesions (8). However, subsequently a study based in MRI showed that 16 out of 17 cases with global aphasia had large anterior posterior lesions (2). Thus it is possible that in patients with global aphasia and restricted lesions MRI could have picked up more lesions.

Broca's aphasia group showed maximum variability in areas involved. Though frontal operculum was involved 7 out of 14 cases but several patients had temporoparietal operculum, temporal gyri and parietal involvement. Poorest correlation for lesion localization has been seen in Broca's aphasia where only 35.4% of patients showed involvement classical anterior involvement (1). However, other studies have shown consistent radiological pattern in all Broca's aphasia cases but lesions are not restricted to classical Broca's area alone (5). In current study, also no lesion restricted to frontal operculum producing a lasting Broca's aphasia was seen. This correlates with Mohr's observations that Broca's aphasia is seen with lesions much extensive than Broca's area alone. Lesions of anterior part of temporal gyri were seen with Broca's aphasia in MRI based study. (2). Even lesions of supramarginal gyrus have been seen in Broca's aphasia (8). However, it is difficult to comment on contribution of these other areas to the language deficit in Broca's aphasia.

Wernicke's aphasia patients had fairly consistent pattern of involvement of temporoparietal operculum, superior temporal gyrus, middle temporal gyrus and angular gyrus with parietal cortex. Similar pattern has been reported in other studies (2,5). The correlation for

lesion localization is better for Wernicke's aphasia than Broca's (1) as can be seen in this study also.

Transcortical motor aphasia mainly involved striatocapsular region and dorsolateral frontal subcortical regions, which also is consistent with other studies (2,5).

Subcortical aphasia

6 out of 8 of our cases with subcortical aphasia had dysarthria also which has been described as common accompaniment of subcortical aphasia (25)

Most common type of aphasia seen in patients with subcortical lesions was Broca's followed by transcortical motor aphasia and none had global aphasia.

The combination of lesion involving caudate, putamen and anterior limb of lateral capsule has been described as having good association with aphasia among subcortical locations. (25)

This particular combination was seen in 2 of our patients. Further 2 patients had involvement of at least 2 of above-mentioned areas. None of our cases had lesions restricted to striatum alone which usually produces only mild word finding difficulty or no aphasia. In

subcortical aphasia white matter pathways are considered critical structures and not basal ganglia (26).

Damasio et al. (25) highlighted the importance of anterior limb of internal capsule in subcortical aphasia. This particular structure carries several important fiber systems including – projections from auditory cortex to the head of caudate nucleus, frontal cortex to pons, motor thalamus to frontal premotor cortex, caudate nucleus to pallidum and from mediodorsal thalamus to prefrontal cortex. Damage to these subsystem is likely to cause dysarthria, aural comprehension defects and paraphasia. However, the current concept on subcortical aphasia suggests that language deficits are mainly related to associated cortical hypoperfusion which can not be studied be CT scan or MRI. (11)

(D) Patterns of recovery from aphasia

From the graphs shown above a bimodal type of recovery pattern can be seen particularly for global aphasia with two extremes, patients either remaining global only or showing excellent recovery to anomia or becoming normal.

Most of the studies have shown that global aphasia finally has a poor recovery as a rule(12,27,28,29). Though in a study by Pashek et al (30) when patients were examined at frequent interval following

stroke two patterns of recovery were noticed one being early rapid change and other a slower recovery. Findings in present study support this observation. The reasons behind initial recovery seen in global aphasics could be recovery of ischemic penumbra or recanalization of middle cerebral artery. In a recent study based on perfusion-CT images and diffusion weighted MRI it was shown that recovery of comprehension, repetition and fluency has a good correlation with rescue of penumbra involving the particular structure responsible for causing the specific language modality. They could not show any correlation between global aphasia score change and left MCA recanalization. (34)

The most common end stage of recovery of anomia which has been described previously (12). The maximum recovery was usually within first 3 months as described in other studies (12,27,28,29).

(E) Predictors of outcome

In the analysis of predictors of outcome of aphasic patients, education and severity as measured by AQ at baseline were significant.

Patients with more education tend to improve more and this trend is most remarkable in anomic aphasia (28). However, some other studies have not shown any correlation between education and

recovery. (13). In the only Indian study on recovery in aphasia (31) only bilinguality was tested. Damasio et al. suggested that the neurological structures whose lesions determine aphasia are the same for literate and illiterate patients and development and final arrangement of these structures does not depend on acquisition of reading and writing skills. (35) However, the mechanisms behind poor recovery in patients with low education are not known.

Severity at baseline has correlation with poor outcome (12,27,28,29) and this finding is further supported by this study.

Thus patients with a lower education and severe aphasia at the onset with aphasia quotient < 75 may require more intensive therapeutic intervention.

Recovery in Broca's aphasia has been documented to be better compared to Wernicke's or global (32). There was a trend for better recovery in Broca's (71.4%) vs global (50%) and Wernicke's. (60%) This has been attributed to preserved comprehension in patients with Broca's aphasia compared to Wernicke's.

Hemorrhagic strokes overall have a better recovery than ischemic which can be extrapolated to patients with stroke having aphasia also as all patients in this group had a good recovery.

Subcortical aphasics have good recovery (25) and this study shows similar trend.

Younger age is a predictor for good outcome (12). However some studies are at variance with this (13,33). This study shows a trend towards patients with good recovery being younger but this did not reach statistical significance. However, in global aphasics, the patients who had good recovery were younger compared to those remaining global at 1 year (mean age 34.7 vs. 57.6).

CONCLUSIONS

(1). The most common type of aphasia seen in acute stroke patients is global aphasia.

(2). Global aphasia patients usually have large lesions involving the majority of MCA territory though there can be some exceptions to this with smaller restricted lesions. Wernicke's aphasia has consistent pattern of temporoparietal location of infarcts.

(3). Patients with subcortical location of infarcts and aphasia commonly have involvement of caudate, putamen and anterior limb of internal capsule. They tend to have a good recovery.

(4). Maximum recovery following aphasia is seen in first 3 months.

(5). Anomia is commonest end stage of recovery.

(6). Global aphasics have a bimodal recovery pattern with younger patients having good recovery in initial 3 months. Recovery for transcortical motor, transcortical sensory and anomia is usually good.

(7). Predictors of poor outcome is aphasia following stroke are low education and initial severity of aphasia.

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