

## CORRELATION OF NEUROLOGIC DYSFUNCTION WITH CT-SCAN BRAIN FINDINGS AND CAROTID DOPPLER STUDY IN ACUTE ISCHAEMIC STROKE

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**ABSTRACT: BACKGROUND AND OBJECTIVES:** stroke is one of the most common debilitating diseases with a huge burden related to both mortality and morbidity, ischemic stroke is far common compared to haemorrhagic stroke and also associated with significant carotid stenosis. Hence this present study is carried out to evaluate all the aspects of this disease. **OBJECTIVES:** 1. To correlate the nature of lesion, site of lesion, and severity of lesion on clinical grounds with CT-Scan findings. 2. To find the prevalence of Carotid Artery Stenosis in Acute Ischaemic Stroke patients. 3. To find if there is any association between Carotid Artery Stenosis and risk factors such as Diabetes mellitus, Hypertension, Hyperlipidemia, Smoking and Age. **DESIGN:** Cross sectional single center study. **MAIN OUTCOME MEASURES:** CT-scan and Carotid Doppler studies on patients admitted into our institution with signs and symptoms of stroke. A detailed and thorough history, physical examination and investigations were performed, studied and noted. **RESULTS:** Out of 100 patients clinically diagnosed as acute stroke 84 patients showed infarcts in the CT-Scan and in 16 patients the CT-scan was normal. These, 16 patients maybe having small lacunar infarcts which are not picked up by CT or early CT scan taken within 24 hours maybe normal. 6 patients out of 100 patients diagnosed clinically as stroke probably due to haemorrhage (ICH) showed massive infarcts with midline shift in the CT-Scan. The severity of lesion clinically does not correlate with size and extent of lesion in CT-Scan. 75 patients out of 100 in the study underwent Doppler Ultrasonography of the Carotid Arteries. The other 25 patients refused carotid Doppler due to affordability or it was not feasible. The prevalence of the carotid stenosis in this study is 58% (44 out of 75 had stenosed carotids). **CONCLUSION:** CT Scan was positive in 84% of the clinically diagnosed Stroke and Negative (i.e. Normal) in 16% of the patients. Clinical localization correlated well in majority (70%) of cases with CT Scan brain. 58% of ischaemic stroke patients had carotid stenosis in our study. The prevalence of carotid stenosis increases with increase in age, male gender, smoking, diabetes mellitus, Hypertension & Hyperlipidemia.

**KEYWORDS:** Ischaemic stroke, carotid stenosis.

**INTRODUCTION:** Stroke remains the second leading cause of death worldwide,<sup>1</sup> after Ischaemic Heart Disease. 85% of stroke cases are due to infarction and 15% are due to haemorrhage. Carotid athero sclerosis remains an important cause of ischaemic stroke.

Early diagnosis and treatment is necessary to prevent mortality and morbidity.<sup>2</sup> Stroke mimics are to be differentiated from stroke to avoid inappropriate treatment. Though there are many investigations, CT and MRI<sup>3</sup> play major role in the diagnosis of Stroke.

Though MRI is superior to CT scan, the higher cost of the MRI,<sup>4</sup> and the easy availability of CT scan brain makes CT the commonest investigation in diagnosis and treatment of stroke.

CT<sup>5</sup> plays a major role in Stroke to assess site, size and nature of the lesion.

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Carotid atherosclerosis occurs in patients with atherosclerotic risk factors like diabetes mellitus, hypertension, smoking and hyperlipidemia. The internal carotid artery is the commonest site of atherosclerosis next to abdominal aorta, followed by the common carotid artery. The extra cranial part of internal carotid artery is the commonest site of atherosclerosis than the intracranial part of internal carotid artery. Carotid atherosclerosis leads to plaque formation and these plaques gradually increase in size and cause stenosis. Atherosclerotic plaques interrupt the endothelium and then ulcerate. As the endothelium is breached, platelets adhere to the wall and a hemostatic plug is formed. This platelet nidus initiates coagulation cascade and an occlusive thrombus is formed.

Thrombus formation on an atherosclerotic plaque leads to distal embolization and causes occlusion of blood vessels (or) a severe stenosis may cause hypo perfusion and infarct of the brain tissue.

Atherosclerotic plaques and stenosis can be detected by non- invasive ultrasound imaging of the carotid arteries which has high sensitivity and specificity in detecting carotid artery stenosis. Patients with carotid artery stenosis are at higher risk of development of stroke and recurrence of stroke after a stroke/TIA.

### **MATERIALS AND METHODS:**

#### **STUDY SUBJECTS:**

- Number of subjects included under study:100 patients

#### **SOURCE OF DATA:**

- All the patients admitted to Basaveshwar Teaching and General Hospital, Gulbarga with Neurological Deficit due to suspected cerebrovascular accident during the period of two years (2011-2013).

#### **METHODS OF COLLECTION OF DATA:**

- Informed and written consent was obtained from all the patients or their relatives after explaining the procedure.
- The study protocol was approved by the institute's Ethics Committee.

#### **INCLUSION CRITERIA:**

- All the patients admitted to Basaveshwar Teaching and General Hospital, Gulbarga with Neurological Deficit due to suspected cerebrovascular accidents less than one week duration during the period of two years (2011-2013).

#### **EXCLUSION CRITERIA:**

- Duration of Stroke more than one week.
- Patients with haemorrhagic stroke.
- Patients with history of head injury.
- Systemic illness such as:
  - Haemodynamically unstable patients.
  - Malignancy.
  - Unconscious patients.

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- Stroke like syndromes.
- Infections such as TB.
- Metabolic Emergencies.
- Poor General Condition.

**METHODOLOGY:** All the patients post a complete history recording underwent a thorough and detailed general and neurological examination. All other systems examination was done in all the patients.

- Neurological examination was done with reference to motor, sensory, autonomic and higher functional disturbances.
- Power is graded according to MRC grading system from 0 to 5.
- Sensory system examinations were done for somatic, special and cortical sensation.
- Higher functions testing were done with mini mental scale.
- Lobar functions were done in all these patients.

### **CLINICAL ASSESSMENT OF NATURE OF LESION:**

- Haemorrhagic lesions in CT Scan brain was suspected clinically by the presence of intense throbbing headache, projectile vomiting, seizures, altered sensorium, Neck stiffness, dissociated eye movements, Papilloedema in a known hypertensive patients.
- Absence of these clinical findings with pure motor hemiplegia, intermittent progression of signs, relative preservation of consciousness and some degree of recovery were suspected to be Infarcts in CT Scan brain.
- Autonomic functions for sympathetic and parasympathetic system were done for all patients.

After careful clinical examination of the patients all the patients were submitted to the following investigations.

### **BASIC LAB INVESTIGATIONS:**

- Urine analysis.
- Blood-Sugar, Urea, Creatinine.
- Serum electrolytes.
- Lipid profile.
- ECG.
- Echo.
- CT Scan Brain.

**CAROTID DOPPLER ULTRA SOUND EXAMINATION:** Post stabilization of the patient, they are made to lie in supine position for examining the carotid arteries, out of 100 patients 75 underwent the Carotid Doppler examination.

**STATISTICAL ANALYSIS:** Statistical analysis was carried out for 100 subjects. Age, Presence of diabetes, hypertension, Smoking, Alcoholism and hyperlipidemia were analyzed in patients with and without carotid stenosis admitted for acute ischaemic stroke. The statistical significance was calculated using Chi-square test. Statistical significance was taken when P value was <0.05. Statistical

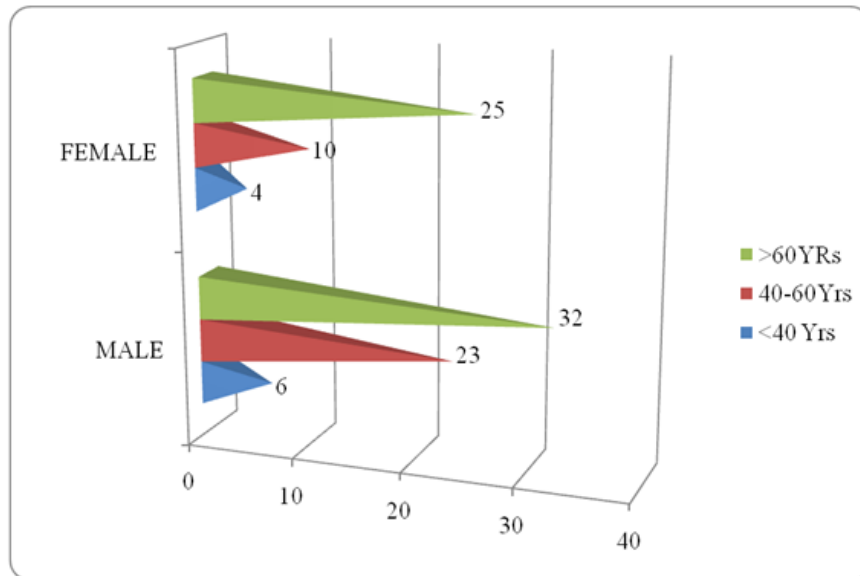
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analysis was carried using standard formulae. Microsoft Excel 2007 and SPSS (Statistical Package for Social Sciences).

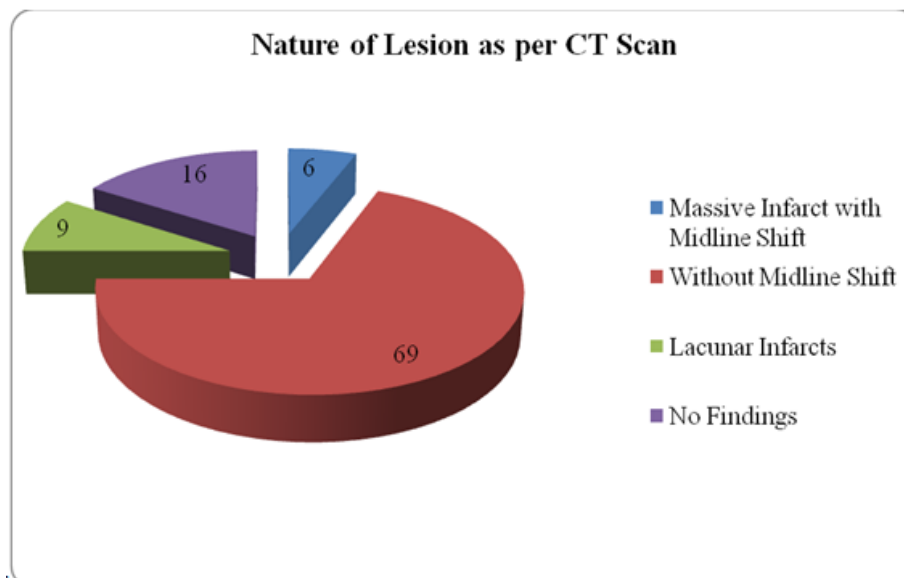
Version 13.0 soft wares were used for data entry and analysis.

### RESULTS AND OBSERVATIONS:

**Clinical Correlations with CT-Scan Brain Finding:** Out of 100 patients, 61 were male and 39 were female.



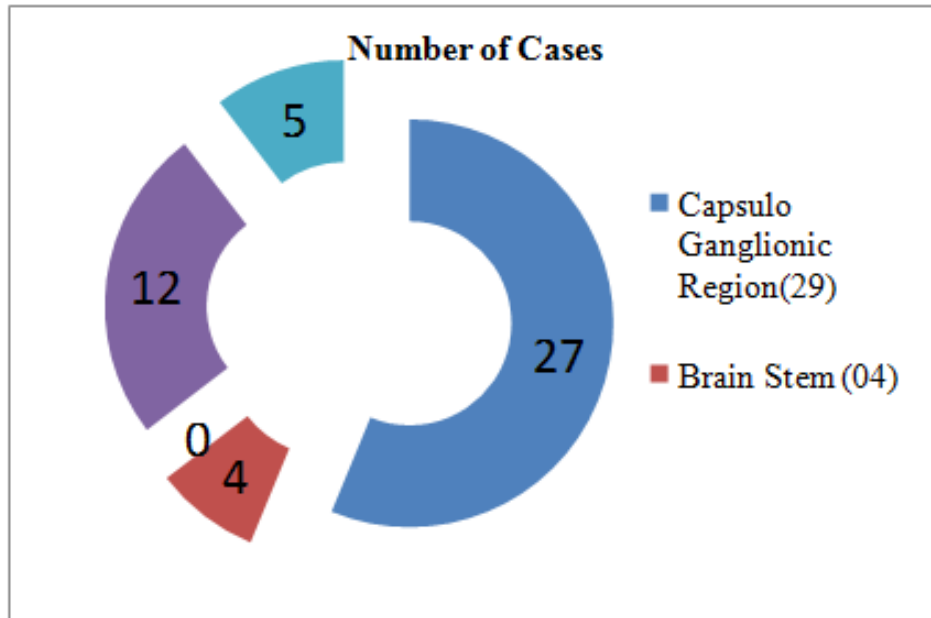
**Graph 1: Age and Sex Distribution**



**Graph 2: Nature of lesion as per CTs**

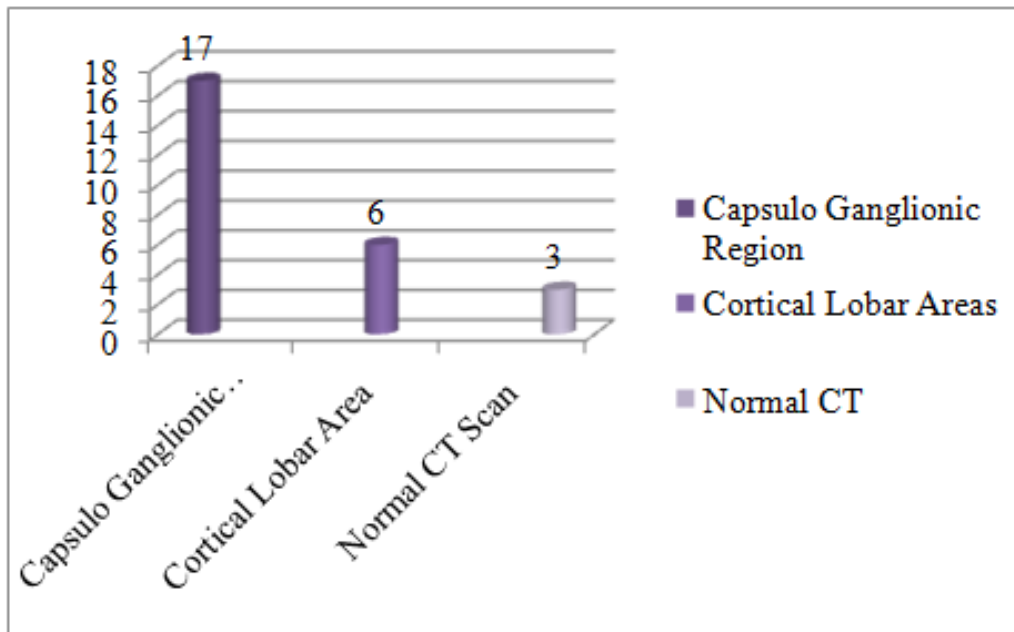
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## A. PURE MOTOR HEMIPLEGIA:



**Graph 3: Site of Lesion in 48 Patients with Pure Motor Hemiplegia**

## MOTOR HEMIPLEGIA WITH CORTICAL DEFECTS:

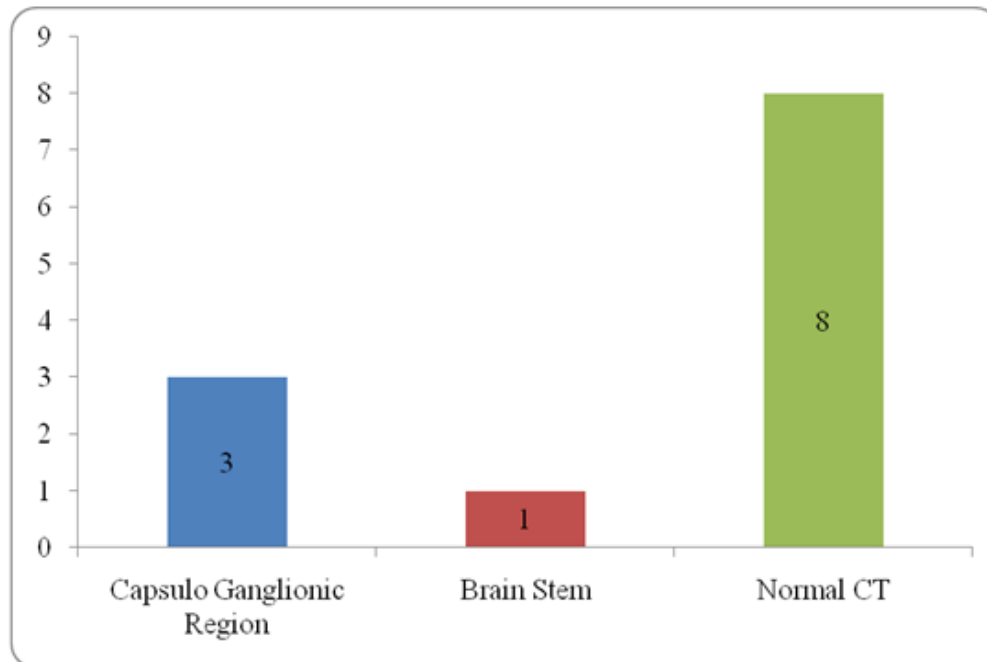


**Graph 4: Site of Lesion in 26 Patients with Motor Hemiplegia with Cortical defects**

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**PURE SENSORY DEFECTS:** No patients presented clinically with pure sensory deficits.

**BRAIN STEM DEFICITS:**



**Graph 5: Site of Lesion in 12 Patients with brain stem deficits**

**CEREBELLAR STROKE:** Only two patients showed clinically cerebellar signs, both the patients had lesion in cerebellar hemispheres.

Capsulo Ganglionic Region (Sub Cortical Region)	Brain Stem	Cerebellum	Cortical Lobar Areas	Normal Ct Scan
4	0	0	8	0

**Table 1: Site of Lesion in 12 Patients with Motor Hemiplegia with Higher Cerebral Dysfunction**

Total 12 patients showed motor hemiplegia with higher cerebral dysfunction in the study, out of which 4 patients had presented clinically with frontal lobe dysfunctions. Of the 4 with frontal lobe showed lesion in the subcortical region (capsule ganglionic region) in CT. the rest in Fronto Parietal region. This correlated well. 6 Patients presented clinically with parietal lobe dysfunction, showed lesion in the non-dominant lobe, which correlated well with the clinical features. Out of two patients who showed occipital lobar lesions clinically 1 showed lesion in the occipital cortex and the other in the capsuloganglionic region.

From these results it was clear that most of the cases showed lesion in the subcortical white matter. i.e., capsuloganglionic area in the study.

It is also found that the Hypertensive and Diabetic patients also showed lesion predominantly in subcortical white matter (capsuloganglionic area).

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Since capsuloganglionic area is the mostly involved site, it is clear that 'MCA territory' is the artery most commonly involved in hemiplegia patients in the study. Also clinical features correlated well with the site of lesion in CT scan except few cases (16 patients) whose CT was Negative.

These patients may be investigated by MRI scans for better correlations and occult lesions missed by CT scan.

**ANALYSIS OF SEVERITY OF LESION:** 5 patients out of 100 patients presented clinically with pure dense hemiplegia of power 0/5 and were disoriented and drowsy on examination. These patients who were expected to have large lesions in CT Scan showed only tiny lesions (<5mm) in CT Scan brain.

From this it was clear that severity of lesion clinically does not correlate with size and extent of lesion in CT Scan.

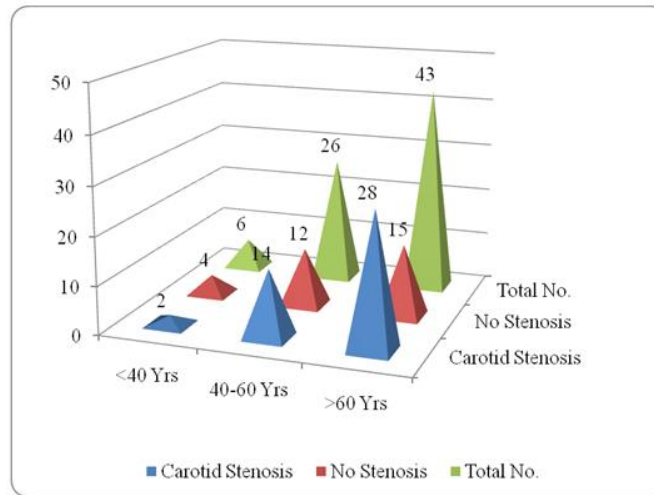
### RISK FACTOR ANALYSIS:

Risk Factor	No. of Patients	Nature of Lesion in Ct-Infarct	Nature of Lesion in Ct-Normal	Most common site of Lesion in CT Scan
Hypertension	42	35	7	Capsulo Ganglionic Region (30 Patients) 71.42%
Diabetes mellitus	28	24	4	Capsulo Ganglionic Region (19 Patients) 67.85%
Ischaemic heart disease	18	16	2	Capsulo Ganglionic Region (10 Patients) 55.55%
Rheumatic heart disease (atrial fibrillation)	04	04	0	Capsulo Ganglionic Region (4 Patients) 100%
No Risk Factors	08	05	03	Capsulo Ganglionic Region (4 Patients) 50%

Table 2: Risk factor analysis

**CAROTID DOPPLER OBSERVATIONS:** Out of 100 ischaemic stroke patients, carotid Doppler was done in 75 patients. In the other 25 patients carotid Doppler could not be performed because of issues related to affordability, in few cases they went against medical advice even before the procedure could be performed. A very few were lost to mortality before procedure.

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**Graph 6: Age distribution of carotid stenosis in stroke patients**

Sex	Total	Stenosis	No. Stenosis	Percentage Stenosis
Male	51	35	16	69%
Female	24	9	15	37%
<b>Total</b>	<b>75</b>	<b>44</b>	<b>31</b>	

**Table 3: Sex distribution of Carotid Stenosis**

It was found that more male patients had Carotid Stenosis than female patients and it was statistically significant ( $P < 0.001$ ).

Characteristic	Present No.	Absent No.
Diabetes	27	48
Hypertension	39	36
Smoking	37	38
Increased total Cholesterol	41	34

**Table 4: Patient Characteristics – Risk Factors**

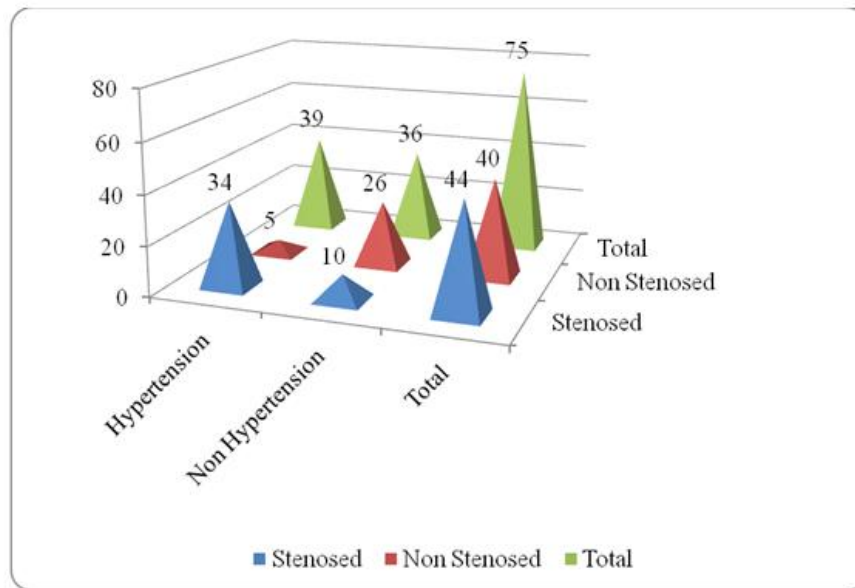
	Total	Stenosed	Non-Stenosed
Diabetes Mellitus	27	24	3
Non-Diabetes	48	20	28
<b>Total</b>	<b>75</b>	<b>44</b>	<b>31</b>

**Table 5: Correlation between diabetes mellitus and Carotid Stenosis**

There was a correlation between Diabetes mellitus and Carotid Stenosis. More diabetes mellitus patients had carotid stenosis than non-diabetic patients and it was statistically significant ( $P$  value  $< 0.001$ ).

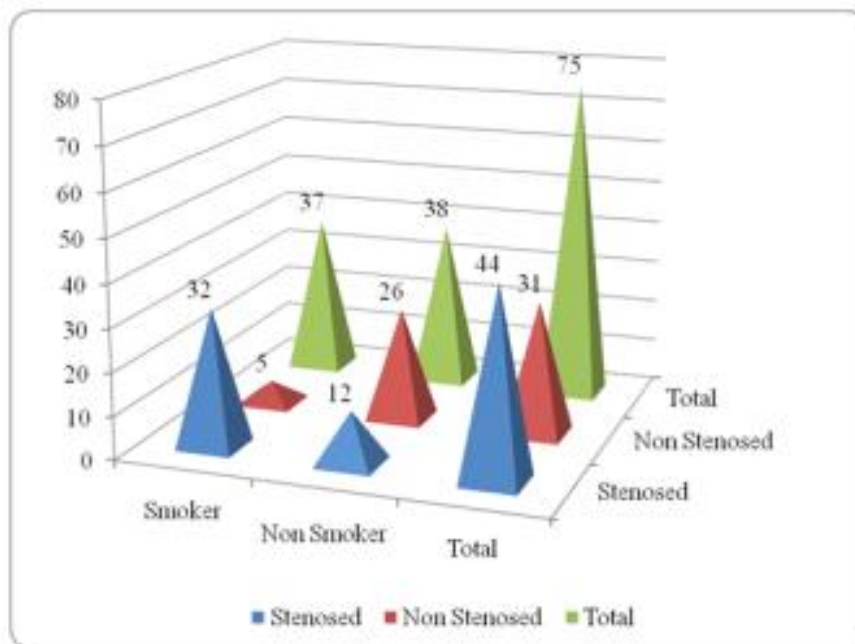


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**Graph 7: Correlation between Hypertension and Carotid Stenosis**

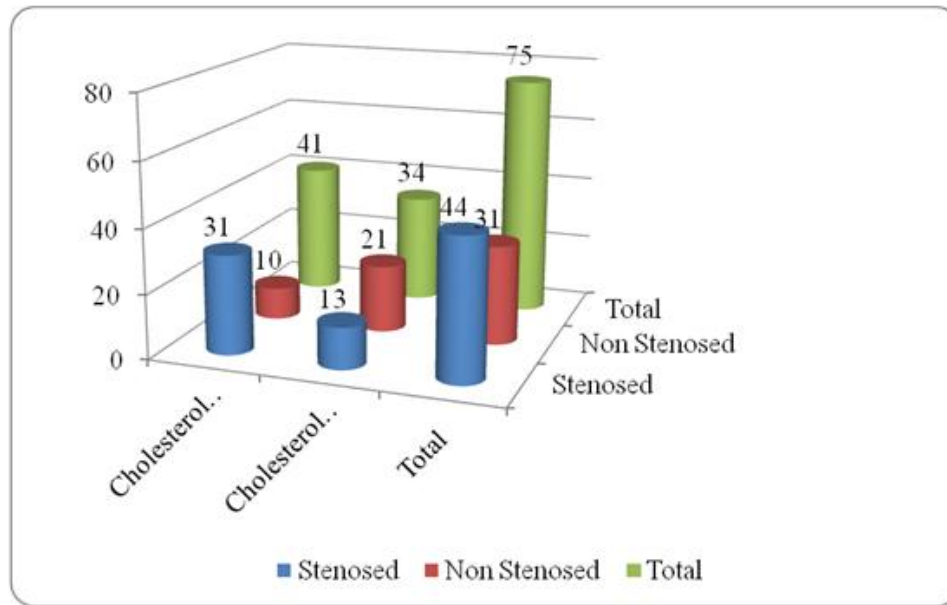
Prevalence of carotid stenosis was more in hypertensives than in non hypertensives and it was statistically significant ( $P < 0.001$ )



**Graph 8: Correlation between Smoking and Carotid Stenosis**

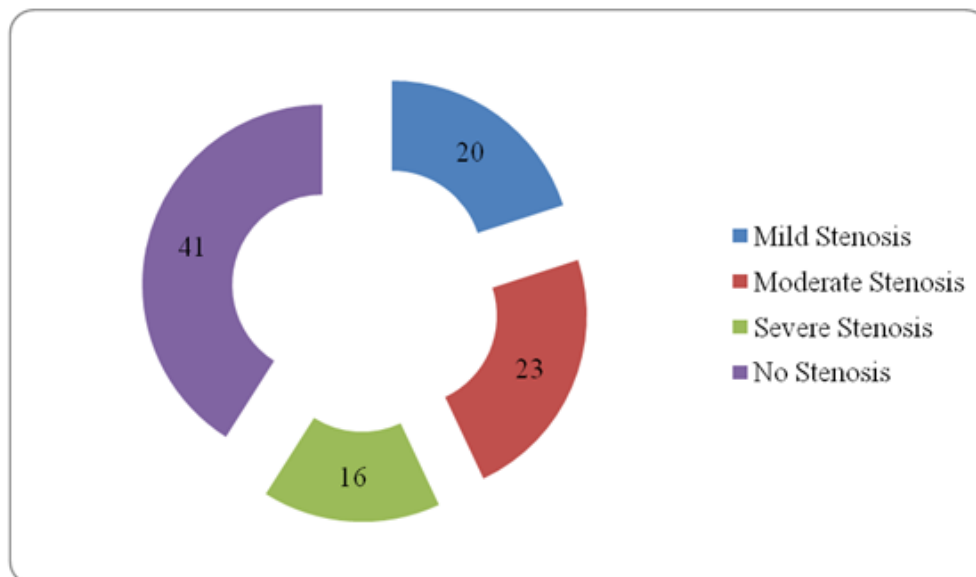
Prevalence of carotid stenosis was more in smokers than in non-smokers and it was statistically significant ( $P \text{ value} < 0.001$ ).

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**Graph 9: Correlation between patients with increased cholesterol and carotid stenosis**

Prevalence of carotid stenosis was more in patients with increased cholesterol than in patients with normal values and it was statistically significant (P value < 0.001)



**Graph 10: Degree of Carotid Stenosis in Ischaemic Stroke Patients**

The prevalence of mild stenosis and moderate stenosis were more than severe stenosis.

## DISCUSSION:

- I. CLINICAL CORRELATION WITH CT-SCAN BRAIN FINDINGS:** It has been established well in this study that there are some variations in the clinical and CT -Scan findings and they do not correlate exactly in all.

**Diagnosis of Stroke-Clinical Vs. CT:** Out of the 100 patients clinically diagnosed as stroke 84 patients showed lesion (i.e. positive CT) in the CT-Scan and in 16 patients CT-Scan showed a normal Brain study.

Out of 16 patients who showed normal CT, 8 patients belonged to Posterior Circulation Stroke (Brain Stem Stroke) clinically (50% of normal CT).

**Nature of Lesion – Clinical Vs. CT:** 6 patients out of 100 patients diagnosed clinically as stroke probably due to haemorrhage (ICH) showed massive infarct with midline shift in CT-Scan Brain.

Thus it was clear that clinical signs and symptoms alone cannot establish an exact diagnosis of nature of lesion like haemorrhage or infarction and hence CT is mandatory<sup>6</sup> before starting anti-platelets.

## SITE OF LESION – CLINICAL VS. CT:

**a. Predominant Site of Lesion in Pure Motor Hemiplegia:**

- b.** Out of 48 patients who presented clinically with pure motor hemiplegia alone without any cortical lobar dysfunction or brainstem or cerebellar signs, 27 revealed lesion in capsuloganglionic area in CT-Scan.

It was clear that the site of lesion correlated well in patients with pure motor hemiplegia in this study.

The rest of the lesions were in fronto parietal and brain stem regions. This was explained by the “Phenomenon of cortical subcortical diaschisis”<sup>7</sup>. This refers to functional deactivation of morphologically intact subcortical brain region remote from but connected to an area of cortical primary structural damage and also due to interruption of neural connections between cortical and subcortical structures according to Von Monakow study in 1914.

**Site of lesion in motor hemiplegia with cortical deficits:** 26 patients out of 102 presented clinically as motor hemiplegia with cortical deficits like aphasia, neglect, visual field deficits, visuo spatial abnormalities, apraxia, and release reflexes. Most of these patients about 19 showed lesion in capsuloganglionic area, this was explained by “Phenomenon of sub-cortical cortical diaschisis”<sup>8</sup>

- c. Site of Lesion in Brain Stem Deficits:** Most of the patients (about 8) with brainstem stroke (posterior Circulation Stroke) showed normal CT possibly due to bony artifact and lack of sensitivity of CT in detecting posterior fossa lesions<sup>9</sup>.
- d. Site of Lesion in Cerebellar Stroke:** Two patients presented clinically with cerebellar signs. Both had lesions in cerebellar hemispheres which correlated well.
- e. Site of Lesion in Motor Hemiplegia with Higher Cerebral Dysfunction:** 6 patients who presented clinically with parietal lobe dysfunctions like sensory neglect, hemianopia, anosognosia, dressing and construction apraxia, visuospatial disorientation showed lesion exactly in non-dominant parietal lobe which correlated well.(100%).

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Out of the 2 patients who presented, clinically with occipital lobe features like hemianopia, cortical blindness, one showed lesion exactly in occipital lobe which correlated well (50%). Patients presented with frontal lobe features showed lesion in capsuloganglionic area predominantly which doesn't correlate which could be explained by the phenomenon of "subcortical cortical diaschisis".

**Severity of Lesion – Clinical Vs CT:** Seven patients presented clinically with dense pure motor hemiplegia of power 0/5. These patients were also drowsy and were also disoriented on clinical examination. These patients were suspected to have larger lesions in CT scan brain. But they showed only tiny lesions (<5mm), but on vulnerable regions i.e., Internal capsule.

From this it was clear that severity of lesion clinically does not correlate with the size and extent of lesion in CT scan.<sup>10</sup>

**II. CAROTID DOPPLER STUDY:** In this study we have found that the prevalence of carotid stenosis in acute ischaemic stroke is about 58%, consistent with studies done by Oliviero et al <sup>11</sup> In their study the prevalence of carotid stenosis was about 43% in ischaemic stroke patients. The percentage of patients with The prevalence of significant stenosis in a study conducted by M.M.Singh et al was about 32%<sup>12</sup>

**AGE AND CAROTID STENOSIS:** We found in our study the percentage of patients who had carotid stenosis, increased with increase in age <sup>13</sup>The prevalence in patients <40 years, 40-60 years, >60 years was about 33%, 54%, and 65% respectively. In a study conducted by K.Rajamani et al <sup>13</sup> showed increasing incidence of carotid stenosis with increase in age in African American men.

**SEX AND CAROTID STENOSIS:** Prevalence of carotid stenosis was more in males (69%) than females (37%) which was consistent with studies conducted by Jacob et al.<sup>14</sup>

**RISK FACTORS FOR CAROTID STENOSIS:**

**DIABETES MELLITUS AND CAROTID STENOSIS:** Carotid artery stenosis was more common in diabetics (24 out of 27 patients) than in non-diabetics (20 out of 48 patients) and it was statistically significant. K.Rajamani et al<sup>13</sup> have shown in their study that carotid stenosis was more common in diabetics (22%).

**HYPERTENSION AND CAROTID STENOSIS:** In our study we found that hypertension was one of the risk factors for carotid stenosis and the prevalence of carotid stenosis was more in hypertensives (34 out of 39 patients) than in normotensives (10 out of 36 patients) consistent with studies done by Duncan et al, Sutton et al.<sup>15</sup>

**SMOKING AND CAROTID STENOSIS:** More smokers (32 out of 37 patients) had carotid stenosis than non-smokers (12 out of 38 patients), which is also shown by I-LR. Muller et al.<sup>16</sup>

**HYPERLIPIDAEMIA AND CAROTID STENOSIS:** In the study the prevalence of carotid stenosis was more common in patients with cholesterol more than 200 (31 out of 41 patients) than in patients with normal cholesterol (13 out of 34 patients).

**CONCLUSION:**

1. Regarding the role of CT Scan Brain in the diagnosis of Stroke it has been found that in this study, CT Scan was positive in 84% of the clinically diagnosed Stroke and Negative (i.e. Normal) in 16% of the patients.
2. Regarding the nature of lesion in Stroke it has been found that in some cases Massive infarcts in CT Scan mimicked Haemorrhagic stroke clinically:
  - A. Regarding the site of lesion in CVA Clinical localization correlated well in majority (70%) of cases with CT Scan brain.
  - B. It has been found that, the most common site of lesion in CT Scan was found to be capsuloganglionic region indicating MCA territory involvement.
3. Size and extent of lesion in CT scan does not correlate with severity of lesion clinically.
4. Finally to conclude over all, the various clinical aspects in CVA do not correlate exactly with CT Scan findings in all cases. Hence both thorough clinical examination and CT Scan Brain are mandatory.
5. Carotid stenosis is one of the common causes of ischaemic stroke.
6. 58% of ischaemic stroke patients had carotid stenosis in our study.
7. The prevalence of carotid stenosis increases with increase in age, male gender, smoking, diabetes mellitus, Hyper tension& Hyper lipidemia.
8. A simple, non-invasive screening procedure like Doppler sonography of the carotid arteries in high risk individuals could therefore have profound diagnostic and therapeutic implications in predicting and preventing a potentially fatal and devastating stroke

**BIBLIOGRAPHY:**

1. Longo DL, Fauci AS, Kasper DL et al, editors. Harrison's principles of internal medicine, 18<sup>th</sup> edition. New York: Mc GrawHill; 2011.
2. Murray CJ, Lopez AD, et al. Mortality by cause for eight regions of world. Global burden of disease study: LANCET 1997, May 3: 349; 1269-76.
3. Murray CJ, Lopez AD, et al. Mortality by cause for eight regions of world. Global burden of disease study: LANCET 1997, May 3: 349; 1439-42.
4. Mohr JP, Biller J, Hilal SK, et al: Magnetic resonance versus computed tomographic imaging in acute stroke. Stroke 16: 807- 812, 1995.
5. Barber PA, Demchuk AM, Zhang J, Buchan AM: Validity and reliability of a quantitative computed tomography score in predicting outcome of hyperacute stroke before thrombolytic therapy. Lancet 355 (9216): 1670-1674, 2000.
6. Mazzocchi F, Vignolo LA: Localization of lesions in CVA: Clinical-CT scan correlations in stroke patients. Cortex 15: 627, 1979.
7. Feeney DM, Baron JC: Diaschisis, Stroke 17: 817-830, 1986.
8. Demeurisse G, Capon A, Verhas M, et al: Pathogenesis of aphasia in deep-seated lesions: Likely role of cortical diaschisis. Eur Neurol 30: 67-74, 1990.
9. Dirisadale HB: Lesions in posterior fossa. A study of primary pontine, cerebellar lesions with observations on pathogenesis.
10. Stroke - pathophysiology, diagnosis and management - 5th ed.

## ORIGINAL ARTICLE

11. Oliviero U, Orefice G, Coppola G, Scherillo G, AscioncS, Casaburi C, Barbieri F, Saccà L. Carotid atherosclerosis in ischaemic stroke patients. *Int. Journal of Angiology*. 2002 Jun; 21 (2): 117-22.
12. Singh M.M, Gupta S. et al. Carotid stenosis in Stroke, *JAPI-1996*; 44 (12): 954-956.
13. Rajamani K. et al. Carotid stenosis in African-American men. *Journal of Vascular Surgery*.43; 1162-1165.
14. Seihub J et al. Association between Homocysteine& Carotid stenosis. *NEJM*, 1995, Vol.333. Page 325 Suttan, Tyrell et al: Predictors of Carotid Stenosis in Older Adults with and without Isolated Systolic Hypertension: *Stroke*. 1993: 24:355-36 1.
15. Suttan, Tyrell et al: Predictors of Carotid Stenosis in Older Adults with and without Isolated Systolic Hypertension: *Stroke*. 1993: 24; 355-36 1.
16. Muller H.R et al: Smoking and carotid stenosis: *Journal of Neurology*: 1990: 97-102.

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