COMPARISON OF EXTRA CRANIAL WITH INTRA CRANIAL CAROTID ARTERY DISEASE IN ISCHEMIC STROKE

Dissertation Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERISTY

CHENNAI

In partial fulfillment of the regulations

For the Award of the Degree of

D.M (NEUROLOGY)

BRANCH -1

INSTITUTE OF NEUROLOGY

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RAJIV GANDHI GOVERNMENT GENERAL HOSPITAL

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AUGUST 2013

CERTIFICATE

This is to certify that the dissertation entitled**"Comparison of extra cranial with intra cranial carotid artery disease in ischemic stroke"**is a bonafide original work of **DR.A.VENI**, in partial fulfillment of the requirements for D.M. Branch– I (NEUROLOGY) Examination of the Tamil Nadu Dr. M.G.R Medical University to be held in AUGUST 2013, under our guidance and supervision.

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I hereby solemnly declare that this dissertation titled **"Comparison of extra cranial with intra cranial carotid artery disease in ischemic stroke"** was done by me in Institute of Neurology, Madras Medical college and Rajiv Gandhi Government General Hospital, Chennai -3, under the guidance and supervision of Prof. **K. BHANU, D.N.B., D.M,** Professor of Neurology, Institute of Neurology, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai. This dissertation is submitted to the Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfillment of requirement for the award of D.M Degree Branch I (NEUROLOGY).

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ACKNOWLEDGEMENT

It gives me great pleasure to acknowledge all those who guided, encouraged and supported me in the successful completion of my dissertation.

Firstand foremost, I express my gratitude to, the **Dean Dr.V.Kanagasabai M.D.**for having permitted me to carry out this dissertation work at Rajiv Gandhi Government General Hospital, Madras Medical College, Chennai.

I am extremely thankful to **Prof. Dr. K.Deiveegan M.ch.,**Professor of Neurosurgery, Head of the department, Institute of Neurology,Rajiv Gandhi Government General Hospital Chennai for his constant encouragement,valuable guidance and support.

I express my deep sense of gratitude and sincere thanks to our respected and beloved **Prof.Dr.K.Bhanu.D.N.B, D.M., Prof.R.M. Bhoopathy, M.D., D.M., Professorof Neurology,** Institute of Neurology,Rajiv Gandhi Government General Hospital, Chennai for his valuable suggestions, constant motivation, kind guidance and moral support without which this study would not have been possible.

I express my sincere thanks and gratitude to our Professors **Dr.C.Mutharasu.D.M., Dr.R.Lakshmi Narasimhan.D.M., Dr.S.Balasubramanian. D.M.,** and **Dr.V.Kamaraj.D.M.,** for their valuable suggestions and support.

I am extremely thankful to our Assistant Professors **Dr.S.Arunan., D.M., Dr.Ramakrishnan.D.M., Dr.S. Shanmuga sundaram.DM., Dr.P.Muthukumar.D.M.** and**Dr. V.Kannan.D.M.,**for their valuable guidance and support.

I owe my sincere thanks to all the patients and the technical staff who participated in the study for their cooperation which made this study possible.

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INTRODUCTION

Stroke is defined as developing symptoms or and signs of global or focal, loss of cerebral function with symptoms lasting for more than 24 hours or leading to death with no apparent cause other than vascular origin.[1]

Globally, the rate of occurrence of stroke is estimated as 400 - 800 per lakh of population. Around 57 lakh deaths occur due to stroke in a year. Approximately, 1.6 crore new acute strokes are reported yearly.[3]

Worldwide, stroke mortality is high in Eastern Europe and Asia. In India, the rate of occurrence of stroke is estimated as 90 - 222 per lakh of population. Approximately, 14 – 16 lakhs new acute strokes are reported yearly. 12% of them occur in the age group of less than 40 years.In fact, it is estimated that the incidence of stroke will reach one million annually by the year 2050.[6]

Stroke is a leading cause of long term disability in our society. As the longevity of the population increase, the incidence is on the rise. Adoption of western diet may further increase the prevalence and incidence of cardiovascular disease and stroke. Of the hundreds and thousands of stroke survivors each year, approximately 30% need

support for day- to-day activities 20% need assistance with ambulation and 16% require institutional care. Hence the need for identifying stroke risk factors to the reduce morbidity and mortality due to stroke.[4]

Atherosclerotic intracranial large artery stenosis accounts for 20% of ischemic stroke. Embolism from major arteries is a common source of stroke. The incidence and prevalence of the condition vary accordingly to the population studied. It is less common in Americans of African descent, Northern Europeans and more common in Asians of China, Japan and Korea.

In the last two decades, theadvances in diagnostic tools have helped us to identify the responsible mechanism in patients with ischemic stroke and has crucial implication in relation to targeted treatment and prevention. At the same time, extraordinary imaging technology has been introduced that allows the physician to make physiologic distinctions between normal, ischemic, and infarcted brain tissue so as to institute appropriate therapy.

Asian studies show, intracranial stenosis is estimated to be 30 to 54%of all strokes in Chinese patients, 46% in Japanese, 48% in Singapore patients, 10-25% in Korean patients, but only 6-10% in whites. [8,9]As Hispanics, Asians and blacks constitute the majority of

the world's population, it may be inferred that intracranial stenosis is the most common stroke mechanism worldwide.[5]

Studies regarding combined intracranial & extracranial stenoocclusive disease is comparitively less. Most of the studies which compared the intracranial and extracranial atherosclerotic disease are invasive. This study compares the concordance and discordance of extracranial and intracranial carotid artery disease in patients with anterior circulation ischemic stroke or carotid transient ischemic attack by using doppler ultra sound study which is a noninvasive method

AIM OF THE STUDY

To assess the following in patients with anterior circulation ischemic stroke:

1. Risk factors involved in carotid artery stroke.
2. Clinical profile, pattern of vascular involvement in carotid artery stroke
3. Extra cranial internal carotid artery disease by carotid doppler.
4. Intracranial carotid artery disease by transcranial doppler in patients who showed extracranial internal carotid artery disease.
5. Comparing the concordance and discordance of carotid artery disease in extracranial and intracranial part of internal carotid artery.

Review of literature

Stroke is a major cause of mortality, disability, and depression in the world. Stroke is no longer a disease of the developed world. Low and middle-income countries account for 85.5% of total stroke deaths worldwide and the number of disability adjusted life years in these countries was approximately seven times that in high income countries

Global Stroke estimates[6,7]

• 400-800 strokes per 100,000

• 5.7 million Deaths

• 16 million new acute strokes every year

• 28-30 day case fatality ranges from 17%-35%

Stroke Morbidity and Mortality in India

• Prevalence 90-222 per 100,000

• 102, 620 million deaths

• 1.44-1.64 million cases of new acute strokes every year

• 12% of strokes occur in the population aged <40 years

Carotid atherosclerosis is recognized as an important cause of stroke and a modifiable factor for the risk reduction of subsequent stroke. Recently, it has been proposed that cognitive impairment may

result due to carotid stenosis.[2] In clinical trials, carotid endarterectomy can now have been replaced with carotid stenting since it is less invasive. Remarkable successes in stroke risk reduction have been achieved by continuous advances in medical therapy.

Risk Factors for Stroke (39-42)

It can be divided into modifiable and non modifiable factors

Non modifiable	Modifiable
Age Gender Race/ethnicity Family history Genetics	Arterial hypertension Transient ischemic attacks Prior stroke Asymptomatic carotid stenosis Cardiac disease Aortic arch atheromatosis Diabetes mellitus Dyslipidemia Cigarette smoking Alcohol consumption Increased fibrinogen Elevated homocysteine Low serum folate Oral contraceptives Obesity

Non Modifiable Risk factors

1) Heredity and Risk of Stroke[19-38]

Genetic factors have been linked with the pathogenesis of ischemic stroke, but specific genetic variants remain largely unknown, and some purported genetic associations have not been replicated (Chinnery et al, 2010). One of the complex polyfactorial diseases of large and medium sized arteries, which also have interaction with multiple genetic and environmental factors, is atherosclerosis. Endothelial dysfunction, accumulation of lipids, calcium, cellular debris and cholesterol within the intima of the vessel is characteristic of atherosclerosis. This results in plaque formation on inner walls of the arteries and narrowing of blood vessels which results in ischemia and tissue hypoxia. [22-23]

Many genetic disorders significantly influence susceptibility of atherosclerotic cardio vascular risk. Genetics also influences the arterial wall property.[24-28].

A number of inherited diseases are associated with non atherosclerotic vasculopathies, including type IV Ehlers-Danlos syndrome, Marfan syndrome, Rendu-Osler-Weber disease, and Sturge-Weber syndrome. Familial atrial myxomas, hereditary

cardiomyopathies, and hereditary cardiac conduction disorders etc, Protein C deficiency and Protein S deficiency or antithrombin (AT), and homocystinuria are associated with stroke. The presence of the apolipoprotein epsilon-2 allele in elderly individuals and deletion of the gene for the angiotensin-converting enzyme may increase the risk for stroke, but the association with stroke subtype is unclear [17, 18]

2) Racial difference and Atherosclerotic disease involving cerebral arteries [9-18]

In Caucasian, carotid bifurcation the neck is known to be among the favored sites for atherosclerosis. On the contrary, primary intracranial atherosclerotic thrombosis was believed to be a rare cause of stroke [8]. Nonetheless, in the past decade, it was established that intracranial atherosclerosis accounted for a large percentage of ischemic stroke, especially among the Asian and the African American population. [9–12]

A pathological study of the Chinese population revealed a high prevalence of intracranial disease, particularly in the middle cerebral arterial territory [13]. Racial differences might, thus, condition the development of atherosclerosis at various sites. However, extracranial atherosclerosis is increasingly observed among Asian populations.

Some investigators propose that the changed lifestyle, particularly the eating habit, might determine the modification of the site of vascular pathology. [*14-16*]

3) Age.

The incidence of stroke doubles each decade after 55 years of age. Half of all strokes occur in people older than 70 to 75 years. Overall, stroke incidence rate is 1.25 times greater in men than women. Men develop ischemic strokes at higher rates than women up to the age of 75 years.[43]

The prevalence of intracranial atherosclerosis was demonstrated to increase with each decade of age. It was found in 23% of those 50-59 years of age, 43% of those 60-69 years of age, 65% of those 70-79 years of age and 80% of those > 80 years of age. A study conducted in the South Korean population observed that for every 10 years of age, the odds of intracranial disease increased by 1.2 [44,45].

4) Sex:

Risk of stroke 25 to 30% greater in males. Intracranial stenosis is more common among men, particularly in younger age groups and in particular locations, such as the basilar artery.

Modifiable risk factors.

1) Hypertension:

It is the major independent modifiable risk factor for intracranial stenosis. Based on epidemiological studies, hypertension is associated with increased odds for the development of intracranial stenosis that ranged from 5 to 9.7 [46, 47]. In fact, the impact of hypertension as a risk factor for intracranial atherosclerosis was already established from autopsy studies [46]. Moreover, the risk further increased when hypertension was associated with other risk factors.[48].

2) Disorders of Lipid Metabolism.

Intracranial stenosis has been associated with dyslipidemia, especially elevated total cholesterol but also its various components. High lipoprotein is an independent marker for a greater extent of disease. There is actually a synergic effect between lipoprotein "a" and diabetes mellitus and resultant intracranial occlusive disease [40,49]. Elevated Low Density Lipoprotein and high total cholesterol has been shown to be a risk factor for atherosclerosis and the development of intracranial stenosis.[49] Lipid lowering agents definitely slow the progression of atherosclerosis and the development of large artery stenosis.

3) Diabetes Mellitus

Diabetes is an independent risk factor for intracranial stenosis. It promotes the accelerated formation of atherosclerotic stenosis through a decrease in fibrinolytic activity [49, 50]. Based on epidemiological studies, the odds ratio associated with diabetes ranges from 4 to 5.9 [47, 49, 50]. In fact, data from the Northern Manhattan Stroke study revealed that patients with intracranial atherosclerosis had a higher prevalence of diabetes (67%) when compared to those with extracranial atherosclerosis (60%).[51] The impact of diabetes was also established in autopsy study done in Hong Kong [50]. These studies, suggest Diabetes Mellitusas the strongest risk factor for intracranial stenosis.

4)Metabolic Syndrome

Results from the Northern Manhattan Stroke Study [51] also demonstrated a higher prevalence of metabolic syndrome in patient with intracranial atherosclerosis when compared to those with extra cranial atherosclerosis, nonatherosclerotic stroke, and controls (62%, 40%, and 35% respectively). Other potential though less well-studied factors include sickle cell disease, meningitis, cranial radiation

therapy, tobacco exposure, family history, and presence of extra cranial carotid atherosclerosis and aortic plaques [52].

Carotid disease and stroke

Anatomy of carotid system:

The common carotid arteries differ in length and in their mode of origin. The left arises from arch of the aorta and has thoracic and cervical portion. The right begins just behind the sterno clavicular joint at bifurcation of the innominate artery and has only cervical portion.

Two carotid arteries arises from common carotid artery are,

E**xternal carotid artery,** supplying the face, and the greater part of the neck and cranial vault

I**nternal carotid artery,** supplying brain and orbital cavities. It has extra cranial and intra cranial part. No branches for extra cranial portion of internal carotid artery.(53,54)

Intracranial portion has several important branches

a. Opthalmic artery
b. Posterior communicating artery
c. Anterior choroidal artery

d. Anterior cerebral artery

e. Middle cerebral artery

The ophthalmic artery is the first major branch arises from supra clinoid portion of the internal carotid artery (ICA). Occlusion can lead to visual symptoms, including visual loss called amaurosis fugax. Next branch is posterior communicating artery, supplying the anterior and medial portions of the thalamus. The anterior choroidal artery supplies portions of the basal ganglia, hippocampus, and posterior limb of the internal capsule. The ICA then divides into anterior cerebral and middle cerebral arteries. These arteries supplies frontal, parietal, temporal cortical and subcortical regions. [55-60] The carotid sinus, or carotid bulb, is a widening of a carotid artery at its main branch point. The carotid sinus contains sensors that help regulate blood pressure. The carotid artery pulse can normally be felt in the neck by pressing the fingertips against the side of the trachea against C4 spinous process.

Clinical presentation:

Transient Ischemic attack:

Defined as brief episode of neurological dysfunction caused by a focal brain or retinal ischemia, with clinical symptoms typically

lasting less than 1 hour and without evidence of infarction. Transient Ischemic attack is an indicator of future stroke risk with 25% to 30% of patients having a stroke over the ensuing 5 years. The risk of stroke is maximum during the first week nearly 11%.

Visual symptoms:

Signs of carotid occlusion include transient monocular blindness or visual loss or dimness of vision with exercise, after exposure to bright light, or on assuming an upright position. The cardinal clinical signs of stenosis, ulcerations, and dissections of the internal carotid artery are TIAs. [(80)]It is a subject of debate whether these are the result of fibrin platelet emboli or a reduction in blood flow.

In carotid stroke the duration of vision loss is usually 1 to 5 minutes and rarely lasts more than 30 minutes. After an episode of amaurosis fugax, the vision is usually fully restored, although some patients may have permanent vision loss caused by a retinal infarction.[61].The internal carotid artery nourishes the optic nerve and retina as well as the brain. For this reason, transient monocular blindness occurs prior to the onset of stroke in 10 to 25 percent of cases of symptomatic carotid occlusion. Yet central retinal artery

ischemia is a relatively rare manifestation of carotid artery occlusion, presumably because of efficient collateral supply in the globe.

Symptoms of Stroke:

- Unilateral paralysis *(opposite side)*
- Numbness *(opposite side)*
- Language disturbance
  - Aphasia
  - Dysarthria
- Visual disturbance *(opposite side)*
- Monocular blindness *(same side)*

In case of occlusion distal perfusion is highly dependent on the configuration of the circle of Willis. For example, when the anterior communicating artery is very small, the ipsilateral anterior cerebral territory is affected as well. In extreme instances where the circle of Willis provides no communication to the side of an occluded carotid artery, thus isolating the hemisphere from other blood flow, massive infarction involving the anterior two-thirds or all of the cerebral hemisphere results. If the two anterior cerebral arteries arise from a common stem on one side, infarction may occur in the territories of both vessels. The territory supplied by the posterior cerebral artery

will also be included if this vessel is supplied by the internal carotid rather than the basilar artery.[66-72] When the circulation of one carotid artery has been incompletely compromised, reducing blood flow in both the middle and anterior cerebral territories on that side, the zone of maximal ischemia lies between the two vascular territories ("cortical or external watershed") or, alternatively, in the deep portions of the hemisphere between the territories of the lenticulo striate branches and the penetrating vessels from the convexity ("internal" or "deep watershed").

The infarction in the first instance occupies a region in the high parietal and frontal cortex and the adjacent subcortical white matter. Its size depends upon the adequacy of collateral vessels. Weakness tends to involve the shoulder and hip more than the hand and face. With long-standing carotid stenosis, the cortical watershed zone shifts downward toward the perisylvian portions of the middle cerebral artery territory, even to the extent that a stroke may weaken facial movement or cause a nonfluent aphasia. With impaired perfusion of the deep watershed, infarctions of varying size are situated in the subfrontal and subparietal portions of the centrum semiovale[73-76]

Clinical and Radiological presentation:[62-67]

The radiological presentation of carotid strokes include

1. Cortically based territorial infarctions,
2. Watershed zone infarct (internal or external)
3. Subcortical infarct,
4. Cortical and subcortical infarcts
5. Multiple fragmented infarct

The mechanism of stroke due to intracranial stenosis is often studied by the pattern of infarction on brain imaging in magnetic resonance imaging. Deep infarcts suggest local thrombosis at the origin of perforators. Complete occlusion distal to the site of stenosis suggests complete segmental occlusion. Single or multiple distal cortical infarct patterns suggest embolism from the site of proximal stenosis. Lastly, a border zone pattern with impaired washout is appears like “rosary bead” or “string of pearls” in the deep white matter territory of the middle cerebral artery. Often, a combined pattern is noted which makes a definitive determination of stroke mechanism a challenge. In the Asian, African and Hispanic populations, White matter lesions (WML), micro bleeds, and lacunes which are observed in magnetic resonance imaging (MRI) are

assumed to be symptoms of cerebral small vessel disease (SVD). Initially these lesions were considered as “silent,” but recent longitudinal studies show that presence of these lesions significantly increases future risks of dementia, stroke, and mortality. Hence it was recommended that preventive trials should target SVD, preferably in its “subclinical” stage. However, conducting a MRI to screen for “subclinical” lesions on recruited subjects in trials is not cost-effective. Till date, there are no simple screening methods for SVD. Hence the pulsatility index (PI), derived from Transcranial Doppler ultrasound, has been proposed to reflect vascular resistance of small vessel for a long time.

A recent case study emphasized how these may interact and provided in vivo evidence of the impaired washout hypothesis [60-65].

Mechanism of Carotid disease:

Carotid arteries like other arteries are made up of three layers of tissue:

- Intima, the smooth innermost layer
- Media, the muscular middle layer
- Adventitia, the outer layer

Intracranial arteries are composed of endothelium, smooth muscle cells and an extracellular matrix consisting of collagen and elastin fibers.(39) The luminal tunica intima includes a single layer of endothelial cells overlying delicate connective tissue which is supported by a dense elastic band, the internal elastic lamina. The middle layer, or intima media, consists of smooth muscle cells. The outermost tunica adventitia is mainly composed of collagen and is predominantly surrounded by only cerebrospinal fluid. A cascade of events including macrophage recruitment and low-density lipoprotein accumulation results in atherosclerotic plaque formation.(41-46)

The several mechanisms of ischemic stroke related to intracranial atherosclerosis include hemodynamic failure, in situ thrombosis from plaque disruption, distal thromboembolism, and perforator artery occlusion by plaque within the parent artery. Perforator artery occlusion seems to be the least frequent mechanism as most subsequent strokes. (91%) [53]. An impaired washout concept has also been a proposed mechanism that results from a combination of hypoperfusion and distal thromboembolism [54]. Thus, progressive arterial narrowing, plaque instability and thromboembolism, and/or exhausted collateral flow with impaired vasomotor reactivity are inter-woven mechanisms that may contribute to ischemic stroke due to

intracranial stenosis. The carotid stenosis causes local reduction in cerebral blood flow. Then the alteration of cellular chemistry that are caused by ischemia and which lead necrosis of neuron, glia and other brain cells

There is growing evidence for these specific individual mechanisms and their potential synergism [55-59]. Thromboembolic mechanisms include in-situ thrombosis with resultant large artery occlusion, perforator (small artery) occlusion, and/or distal embolism. In one study, among 63 patients with middle cerebral artery stenosis and acute ischemic stroke, 32 showed multiple lesions in the MCA territory [57]. The majority had perforating artery infarcts, alone or in combination with distal territory infarcts while a minority had border zone infarcts suggesting flow failure. Yet another study noted that 60% of patients with multiple-infarction on diffusion-weighted imaging had micro embolic signals on TCD monitoring of the middle cerebral artery compared to only 6% in patients with a single perforator or single infarction pattern [60]. Hemodynamic impairment is likely in patients with intracranial occlusive disease. With progressive stenosis, the tissue distal to the lesion may depend on collateral blood flow. In one study, 30% of patients with middle cerebral artery occlusion had impaired cerebral hemodynamics on vasomotor

reactivity testing [58]. In a small positron emission tomography study, 25% of those with symptomatic middle cerebral artery stenosis had abnormal hemodynamics [55]. Using quantitative Magnetic Resonance Angiography to estimate vessel-specific flow among patients with symptomatic vertebra basilar stenosis, 16 of 50 patients had impaired flow [56].

Fisher suggested that atherosclerosis was the likely pathological cause of occlusive carotid artery thrombosis, and he confirmed Chari's earlier observation that thrombosis at this site is mostly preceded by plaque ulceration. This concept has subsequently been confirmed by other studies and carotid sinus plaque rupture has been associated with sites at which a thin, fibrous cap formation, as in the coronary circulation.(68-72)

Diagnosis of Carotid disease :(77-80)

Imaging modalities used to diagnose carotid disease are,

i. Magnetic resonance angiography (MRA),
ii. Computerized tomographic angiography (CTA)
iii. Digital Subtraction Angiography (DSA)
iv. Carotid doppler
v. Trans Cranial Doppler (TCD)

The diagnosis of intracranial stenosis has upgraded with the advent and availability of non-invasive vascular imaging tests.

Magnetic resonance angiography (MRA) is a non-invasive imaging modality which can identifies vessel anatomy, intracranial stenosis, and also plaque character. Contrast MRA can provide anatomy in a better way.

MRA measures flow signal intensity as a function of proton spin, as utilized in time-of flight (TOF) MRA to visualize blood flow changes [101]. The advantages of MRA are radiation exposure or radio contrast risks. MRA has sensitivity of 70% in detecting intracranial stenosis of >50% and 81% sensitivity in detecting intracranial complete occlusion. [101].

Major disadvantages are

1) Vulnerability to motion artifacts,

2) Inability to distinguish between high-grade stenosis and occlusion

3) It has limited role in obese, claustrophobic, or have implanted foreign metallic objects (i.e. pacemakers).

Expanding the role of MRA in detecting intracranial stenosis is Contrast-enhanced MRA which uses gadolinium dye for better visualization of changing blood flow directions.

Quantitative MRA, utilizing phase-contrast techniques, has been used as a diagnostic tool of intravascular blood flow to diagnosis hemodynamic failure in posterior circulation stenosis [102, 103] and screen for post-procedural stenosis at sites of stents or coils. QMRA can also assess regional blood flow which can indicate the overall cerebral hemodynamic and collateral flow status. Another emerging MR technique is high resonance imaging (HR-MRI), capable of visualizing and characterizing plaque morphology and behavior, and discriminating from other non-atherosclerotic etiologies. Further study using these novel modalities may increase the diagnostic utility of MRA and provide valuable prognostic data.

CT angiography (CTA):

It gives better anatomical delineation of vessels. CTA focuses on relative penetrance of contrast material within the vessels. It overcomes the slow flow artifact produced by MRI. It has 98% sensitivity and 99% specificity. Major disadvantage is risk of radio contrast and anaphylaxis.(101)

Digital Subtraction Angiography (DSA)

Conventional catheter-based angiography remains the gold standard but has possibly serious risks and limited availability. Each has its own attributes and limitations. Besides their usefulness in the anatomic diagnosis of intracranial stenosis, non-invasive imaging can also offer critical physiologic information (such as blood flow characteristics) which can support in prognostication and recurrent stroke risk stratification [81]. In comparing the efficacy of non-invasive imaging techniques, Digital Subtraction Angiography (DSA) is considered the reference standard. It provides tremendous overall visualization of vessel contour, anatomic localization, measurement of degree and length of stenosis, and assessment of collateral circulation. Modern DSA also includes three-dimensional reconstructed images from the raw data to provide even greater detail. Due to its invasive nature, there is a small but real risk (approximately 0.7%) of peri-procedural neurologic injury associated with DSA [82]. The most serious complications like ischemic and hemorrhagic stroke, are noted within 24-72 hours of the procedure. Further DSA carries the nephrotoxic risk associated with iodinated contrast injection and the potential for access-related injury (i.e. groin hematoma and limb

ischemia). Even now, for analyzing the cerebro vascular system and its branches, the gold standard is Cerebral angiography.

Carotid Doppler:

Noninvasive ultra sound device used to evaluate blood flow velocity in major cerebral vessels on beat to beat basis and is know as stethoscope of brain. Extra cranial carotid artery studied with carotid Doppler by using 8 MHz probe. It acquire images by transverse and longitudinal plane.[98-100)]

Components of ultra sound examination includes,

Continuous wave doppler

B Mode to study the anatomy

Colour flow imaging

Power Doppler Imaging

The colour velocity image

Doppler velocity spectral display

Parameters assessed,

PSV- Peak systolic velocity

EDV- End diastolic velocity

Intima media thickness

Plaque character , size, echogenicity

Carotid Index

Degree of stenosis

Vessels studied:

Internal carotid artery	Sharp systolic peak with gradual slope in late systole and good forward in entire diastole. Diastolic waveform does not touch the baseline. Clear "window" in both systole and diastole. Peak systolic velocity less than 125 cms/sec or 4 K Hz.
External carotid artery	Sharp systolic peak with minimal or absent flow in diastole
Common carotid artery	Spectrum is a combination of internal and external carotid artery waveform.

Duplex Assessment of Carotid Stenosis

The severity of luminal narrowing is determined with color Doppler image through careful measurement of the diameter, or area of the residual lumen, as seen on transverse images. Measurement at peak systole is suggested, because diastolic measurements may overestimate luminal reduction. Accurate measurement also depends on satisfactory image quality, proper instrument settings, and the depiction of a true cross section of the vessel. For measurement of percent diameter reduction, the margins of the original arterial lumen

also must be precisely defined. The identification of the media of the vessel is the best means for defining the original lumen size.[98]

Trans Cranial Doppler ultrasound: (TCD) [83-100]

TCD is noninvasive and is commercially available. TCD ultrasonography instruments use a low-frequency 2-MHz probe to allow insonation through the cranium. These pulsed-wave Doppler instruments have an effective insonation depth range of 3.0 to 12.0 cm or more that can be evaluated by increments of 2 or 5 mm. At an insonation depth of 50 mm, the sample volume is usually 8 to 10 mm axially and 5 mm laterally.

TCD probes also differ from the 4- to 10-MHz transducers used to monitor the progress of intraoperative neurosurgical procedures (Unsgaard et al., 2002).

Four natural acoustic windows used:

1**). Trans temporal bone window:**

Segments that can be recorded in this window

- MCA (Prox, Mid, Distal)
- ACA
- PCA (P1 and P2)
- tICA (Terminal ICA)

Normal blood flow direction is toward the probe in the MCA and away from it in the anterior cerebral artery. Depending on the position of the window, the probe usually has to be tilted frontally to detect these vessels. A posterior (or occipital) tilt of the probe enables insonation of the PCAs.

2).Trans occipital window:

Flow in the distal vertebral artery and proximal to mid-portions of the basilar artery can be detected in this window. Its direction is away from the probe in these arterial segments.

- Vertebral arteries – 40-95 mm
  - Aim right and left for the vertebral arteries
- Basilar artery – 70-115 mm

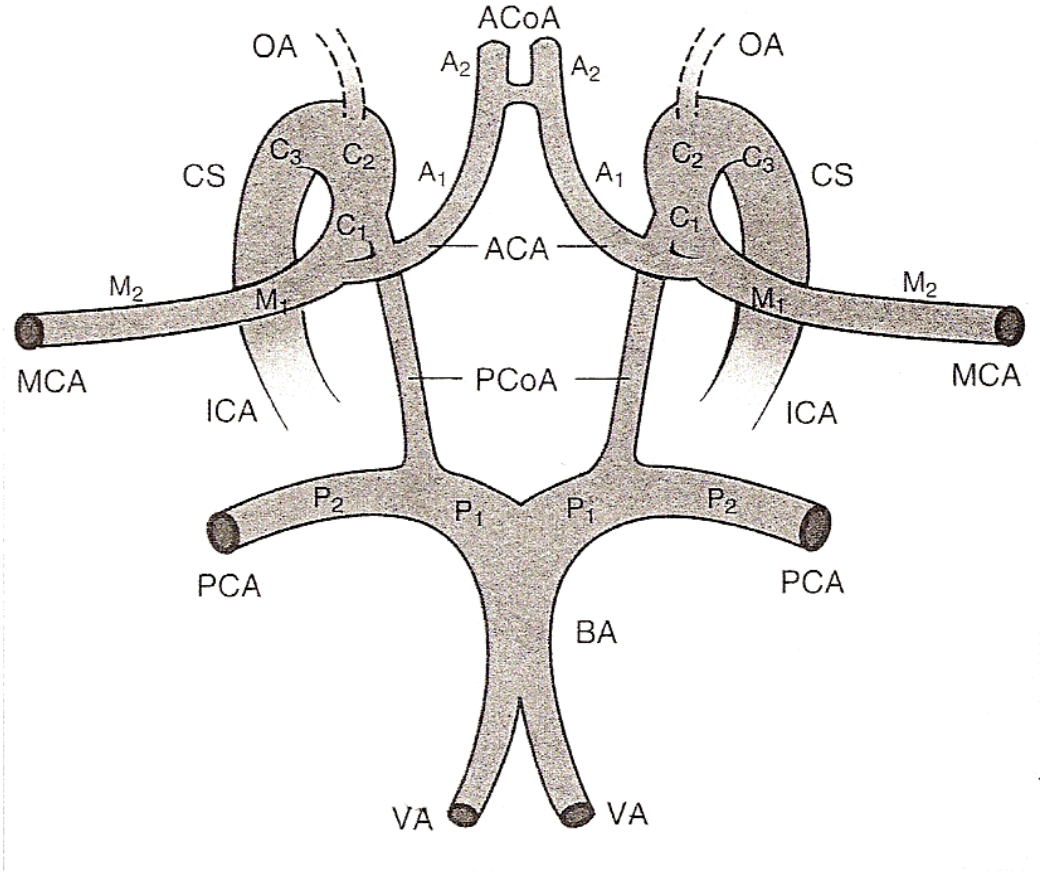
3). Trans orbital Window:

Ophthalmic artery and carotid siphon can be recorded in this window. Flow is towards the signal in these arteries.

- Ophthalmic artery
- Carotid siphon

4).Sub mandibular Window:

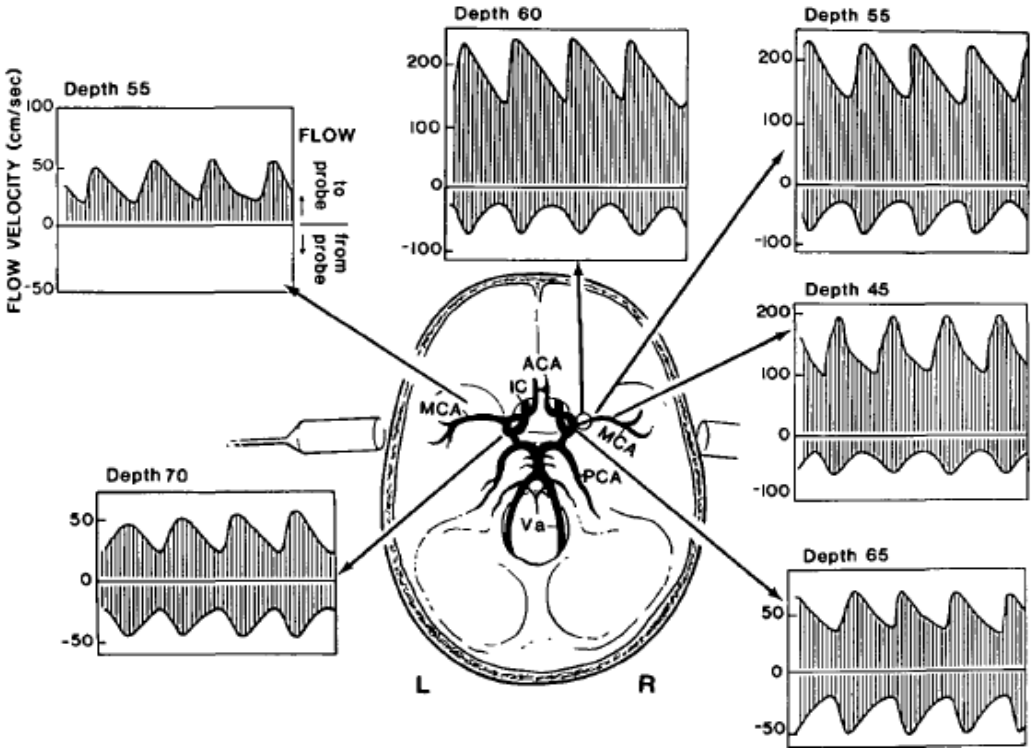
Distal ICA (Internal Carotid Artery) Segment recorded in this window. Useful for the detection of ICA dissection and Chronic ICA occlusion



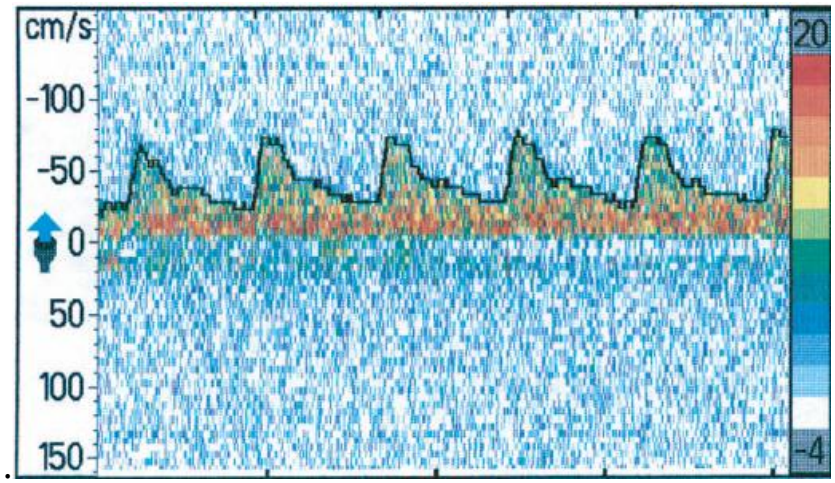
Vessels studied,

- Distal Internal Carotid Artery
- Middle Cerebral Artery
- Anterior Cerebral Artery
- Posterior Cerebral Artery
- Vertebral Artery
- Basilar Artery
- Posterior & Anterior Communicating Arteries

Vessel	Depth-mm	Velocity-cm/s	Direction
MCA	30-60 (50)	43-67 (55)	+
ACA(A1)	60-75 (65)	39-61 (50)	-
PCA(P1)	60-75 (70)	29-49 (39)	+
TICA	55-65	30-48 (39)	+
VA	60-90 (70)	28-48 (38)	-
BA	80-120 (95)	31-51 (41)	-
OA	40-60 (45)	16-26 (21)	+



TCD wave form:



Parameters calculated:

. The pulsatility index (PI):

PI = (Peak systolic velocity - end diastolic velocity)/mean velocity.

PSV- Peak systolic velocity

EDV- End diastolic velocity

MFV- Mean flow velocity

Physical factors affecting cerebral blood flow

- Age
- Gender
- Hematocrit
- Fever
- Hypoglycemia
- Carbon Dioxide

- Heart Rate/Cardiac Output/Blood Pressure
- Brain Activity

Diagnostic Criteria – Stenosis

- Increased flow velocity – generally focal
- Disturbed flow

  Turbulence; spectral broadening
- Covibration phenomena

  Vibration of the vessel wall & surrounding soft tissue
- Drop in post-stenotic velocity
- Changes in post-stenotic waveform morphology

  Prolonged systolic upstroke

  Decreased pulsatility
- Comparison of PSV with contralateral vessel PSV

  Increased PSV >30% - suspicious

  Increased PSV >50% - definite

  - Sensitivity - 100%
  - Specificity - 97.9%
  - Positive Predictive Volume - 88.8%
  - Negative Predictive Volume - 94.9%

Diagnostic Criteria – Occlusion

- Absence of arterial signal at expected depth
- Presence of signals in vessels which communicate with the occluded artery
- Altered flow in communicating vessels, indicating collateralization

Pitfalls & Diagnostic Accuracy

- Lack of flow signal due to an inadequate temporal window
- Misinterpretation of hyper dynamic collateral channels or AVM feeders as stenosis
- Displacement of arteries because of a space-occupying lesion
- Misinterpretation of physiologic variables in the circle of Willis
- Misdiagnosis of vasospasm as stenosis
- Misinterpretation of reactive hyperemia following spontaneous recanalization as stenosis

Applications of Transcranial Color Doppler Imaging[106]

1. Diagnosis of intracranial vascular disease
2. Evaluation of the hemodynamic effects of extra cranial occlusive disease on intracranial blood flow
3. Detection of cerebral emboli
4. Assessment of intracranial collateral pathways
5. Monitoring vasospasm in subarachnoid hemorrhage
6. Intraoperative monitoring
7. Documentation of subclavian steal
8. Monitoring evolution of cerebral circulatory arrest
9. Screening of children with sickle cell disease
10. Evaluation of the vertebrobasilar system
11. Detection of feeders of arteriovenous malformations (AVMs)
12. Monitoring anticoagulation regimens or thrombolytic therapy
13. Monitoring during neuroradiologic interventions
14. Testing of functional reserve
15. Monitoring after head trauma

TCD in the management of Carotid disease [104-106]

- Carotid ultrasound has high sensitivity and specificity in the reliable assessment of the carotid bifurcation, in patients with Transient ischemic attacks and in those with moderate to severe extra- or intracranial carotid stenosis compared to patients with no such findings. Trans cranial Doppler performed within 24 hours of symptoms revealed a threefold greater risk for stroke in the next 90 days in patients with micro embolic signals.
- Micro embolic signals (MES) on Trans cranial Doppler is considered as a marker of risk in patients with emboli of carotid origin. This prompted research into optimal strategies for medical treatment and the timing of endarterectomy in those with an extracranial carotid disease.

Advantages of TCD:

- TCD is relatively in expensive, noninvasive, portable and fairly easy to use.
- It allows frequent repeated measurements and continuous monitoring.
- Immediate, real time detection of changes in cerebrovascular hemodynamics is possible.
- It can be utilized by any medical specialty to evaluate several neurovascular disorders.
- In-expensive
- Portable

Limitation of TCD

- Waveforms can be obtained only in 75-80% of the patient population
- Inter individual variation (observer Bias)
- Needs skill to study all arteries and its flow pattern and anastomosis
- Obesity and thick skull may increase the impedance

Treatment of carotid stroke (106)

Medical treatment:

- Aspirin
- In long term secondary prevention of ischemic strokes, the most effective anti-platelet agents are
- Clopidogrel,
- Combination of aspirin and extended release dipyridamole,
- Ticlopidine and Triflusal.

Surgical Management:

Carotid endarterectomy as the treatment of choice for moderate and severe carotid artery stenosis as a secondary stroke prevention measure.

- Carotid endarterectomy reduced the 5-year absolute risk of any stroke or death in patients with 50–69% stenosis, according to angiographic NASCET, and was highly beneficial in patients with 70–99% stenosis, but showed no benefit in patients with a near occlusion.

2) Carotid stenting: In case of recurrent stenosis and in patients who are unfit for Carotidendarterectomy.

MATERIALS AND METHODS

This cross sectional study was conducted during Jan 2011 to Oct 2012 at Madras Institute of Neurology, Rajiv Gandhi Government General Hospital, and Chennai.

Patients with clinical features suggestive of stroke were enrolled in this study; all were subjected to CT Brain, MRI Brain and Carotid Doppler.

Inclusion Criteria:

1. All the patients with clinical feature suggestive of stroke.
2. Imaging showing ischemic infarct in the anterior circulation.
3. Carotid Doppler showing atherosclerotic carotid artery disease
4. Patients with anterior circulation TIA.

Exclusion Criteria:

1. All hemorrhagic strokes.
2. Posterior circulation stroke
3. Patients without carotid artery disease on carotid doppler
4. Patients with cardiac disease

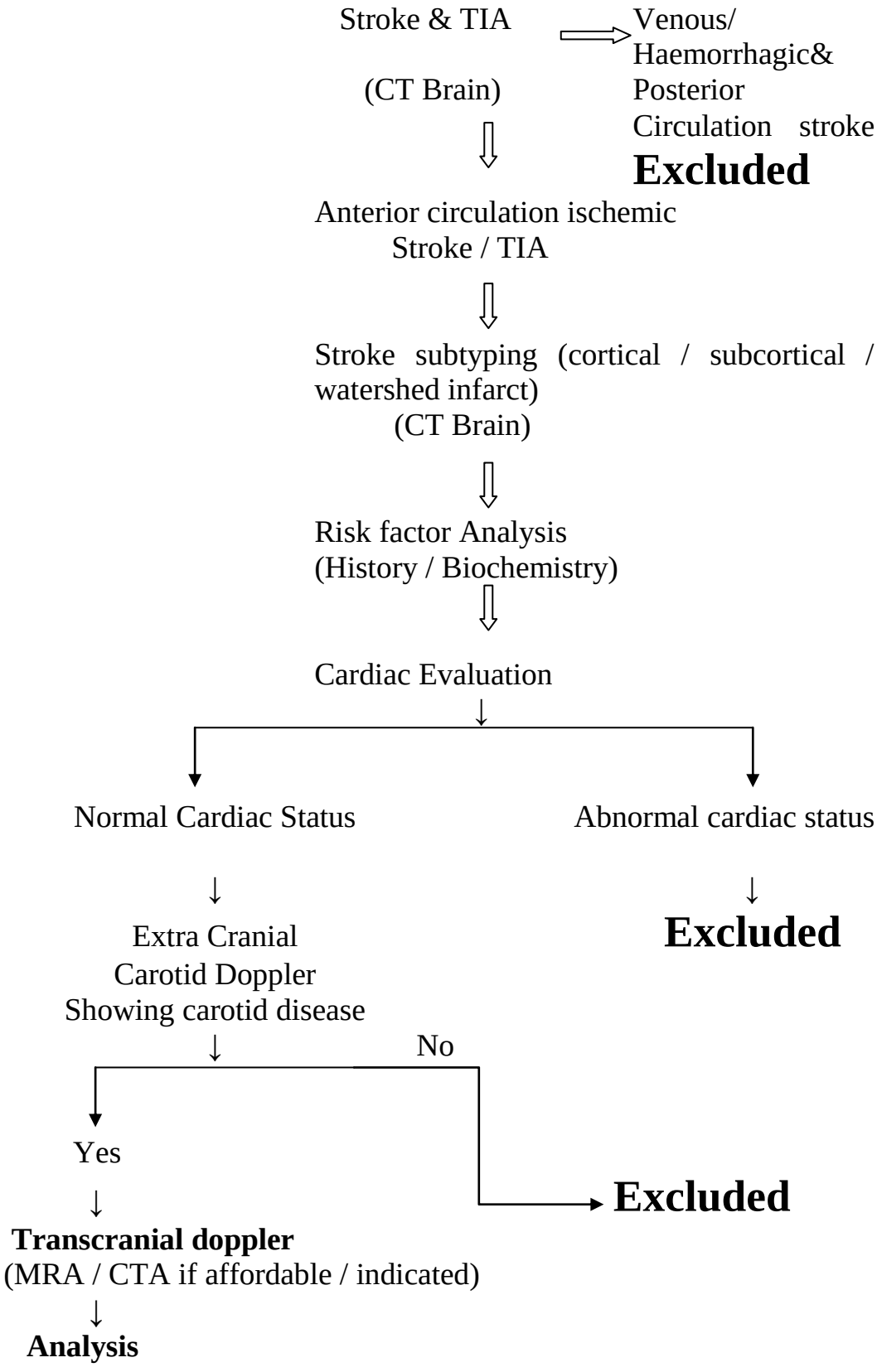
Detailed history regarding age , sex , address, occupation, complaints, evolution of symptoms, previous history of Transient ischemic attacks, amaurosis fugax, Diabetes mellitus, systemic hypertension, coronary artery disease, peripheral vascular disease, drug intake, family history of vascular event, history of vascular risk factors like smoking, alcohol consumption, other form of tobacco use, high risk behavior, other addictions, were obtained.

All the patients were examined according to proforma that included general and systemic examination. All the patients underwent a basic investigation protocol that included

- Complete blood count.
- Renal function test.
- Blood sugar
- Fasting lipid profile
- Electrolytes
- HIV serology(as applicable)
- Urine analysis
- CT Brain- Plain
- Chest X-ray, Electrocardiogram, Echocardiography
- Carotid Doppler

Trans cranial doppler was done through trans orbital window to assess carotid siphon, ophthalmic artery. MRI brain with MRA and DWI, CT angiogram of cerebral vessels had been done wherever indicated. Other laboratory tests such as serum homocysteine, fibrinogen, prothrombin time, partial activated thromboplastintime, anti nuclear antibodies, anticardiolipin antibody, Lupus anticoagulant were done in selected patients wherever needed.

SUMMARY AND STUDY PROCEDURE



OBSERVATION AND RESULTS

Among the total 485 patients, 150 patients (30.9%) with posterior circulation stroke or venous infarcts, intra cerebral hemorrhage were excluded from the study. Remaining 335 patients were subjected to cardiac evaluation, of them 23 patients (6.8%) who showed cardiac abnormalities were excluded from the study. Out of the 312 patients only 70 patients who showed carotid artery disease in carotid doppler were enrolled in this study and subjected to transcranial doppler to study the intra cranial part of internal carotid artery involvement.

1. Sex Distribution: n=70

Male : 58 (83%) Female : 12 (17%)

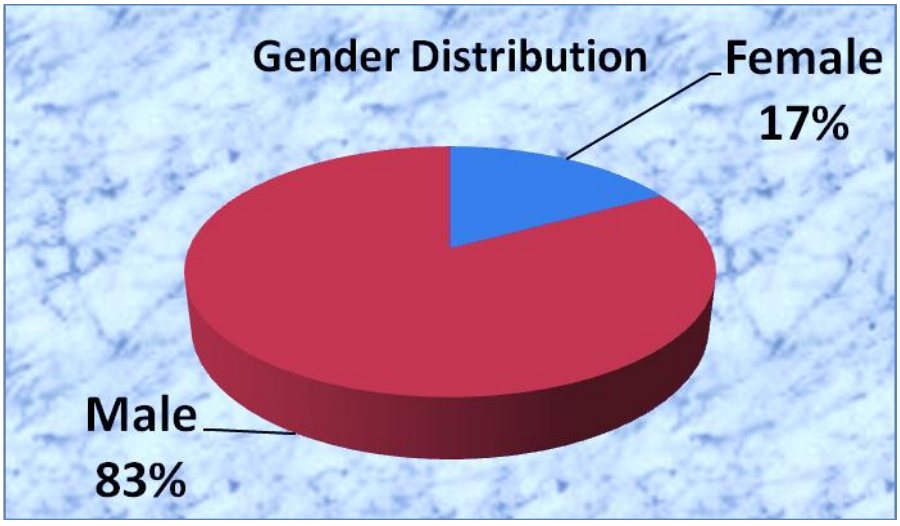


Chart 1. Gender distribution in carotid stroke.

2. Age Distribution:

The study showed mean age of male is 58 years and female is 62 years. The eldest among males is 86years and that in female is 71years. The youngest in male is 35 years and in female is 30 years.

Age (Years)	Percentage (%)
<40	4
41-50	15
51-60	41
61-70	33
>70	7
Total	100

Table 1. Age distribution

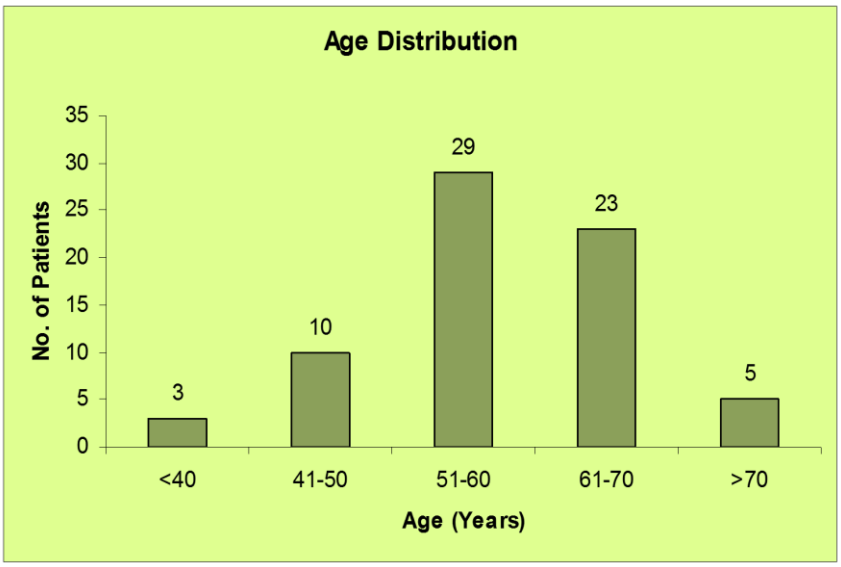


Chart 2. Age Distribution

3. Stroke Risk Factors

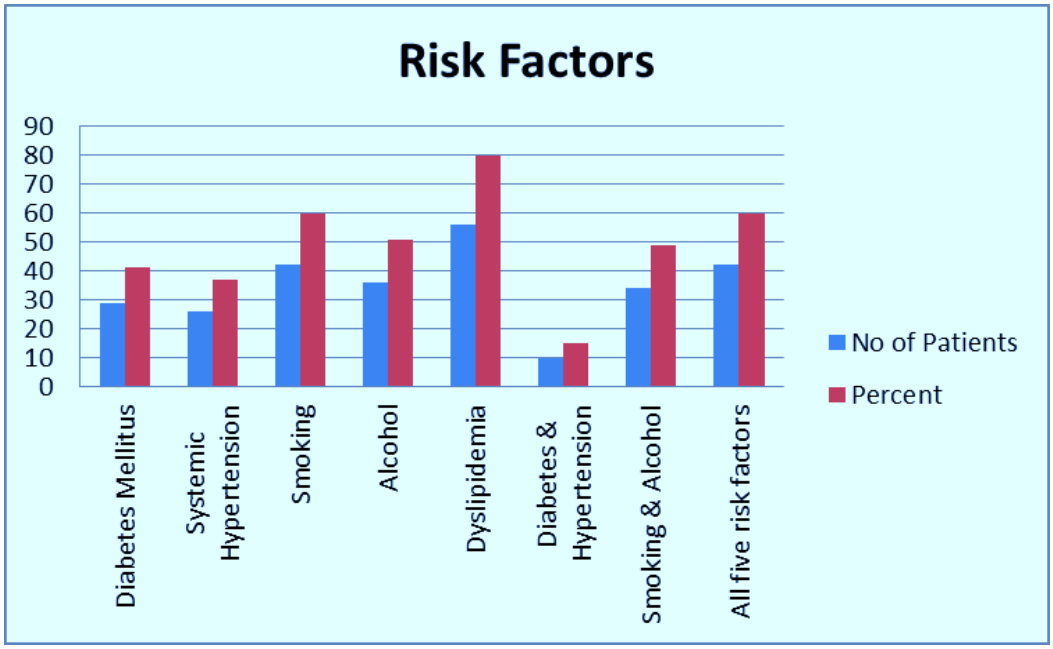


Chart 3. Risk Factors for Carotid stroke.

Most common risk factor seen among 70 patients is dyslipidemia which was present in 56 (80%) patients. 4 patients had only systemic hypertension, 2 patients had only diabetes , and 3 patients had only dyslipidemia. The study also showed that 44patients had double risk factors. Among 44 patients 10 (15%) had Diabetes Mellitus and systemic hypertension, 34 patients (49%) had the habit of both smoking and drinking alcohol. 5 patients(7%) had all the 5 risk factors.

4. Clinical Presentations

Among the Clinical Presentation of 70 patients, ischemic stroke is seen in 63 patients (90%), 6 patients (9%) had transient ischemic attack and one patient (1%) had ocular stroke.

Clinical Presentation	No of patients	Percent
Ischemic Stroke	63	90%
Transient Ischemic Attack	6	9%
Ocular Stroke	1	1%

Table 2. Various Clinical Presentation of carotid disease.

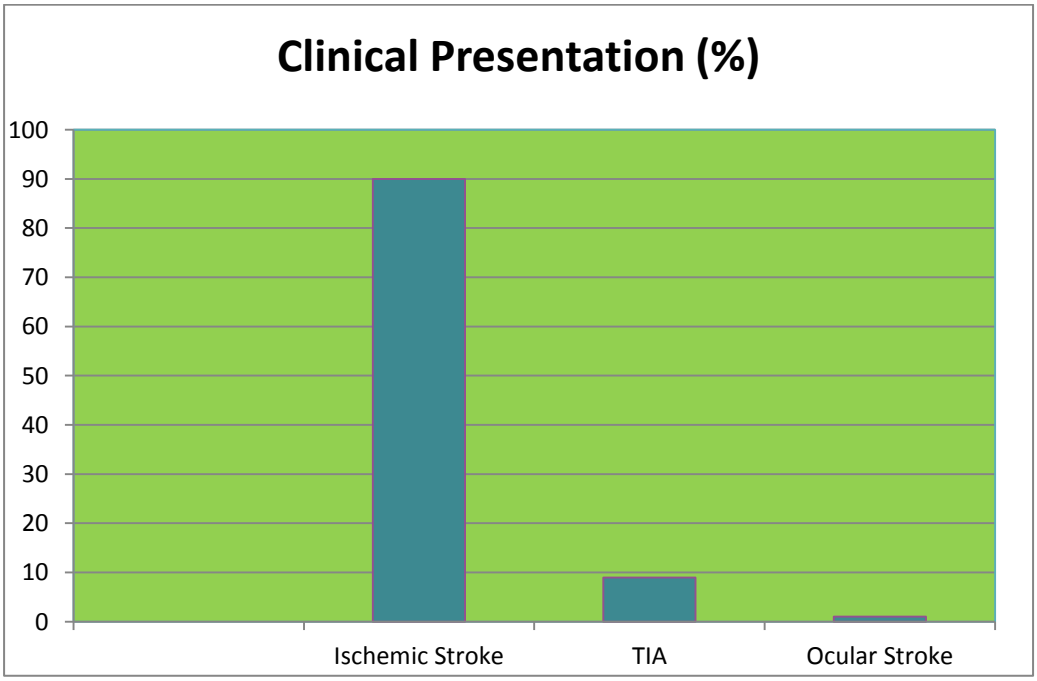


Chart 4. Clinical presentation

5. Incidence of Transient Ischemic attack in Carotid Stenosis:

Study showed total number of 17 patients (24%) with transient ischemic attack, out of which 11 patients later reported with stroke and remaining 6 patients were normal without developing stroke. The remaining 53 out of 70 patients had experienced stroke without preceeding transient ischemic attack.

Transient ischemic attack	**Intracranialstenosis (n-45)**	**Intracranialstenosis (n-15)**
Yes	12	5
No	33	10

Table 3. Incidence of TIA

6. Vascular Territory involved in Carotid Stroke:

In Carotid Stroke, most commonly involved territory is middle cerebral artery territory which accounted for 45patients (64%) and the least affected territory is Ophthalmic Artery which accounted for one patient (1%)

Vascular Territory	Total	Percent
Anterior cerebral artery	6	9%
Middle cerebral artery	45	64%
Normal (TIA)	6	9%
Ophthalmic artery	1	1%
Watershed infarct	12	17%

Table 4. Vascular territory involved in carotid disease

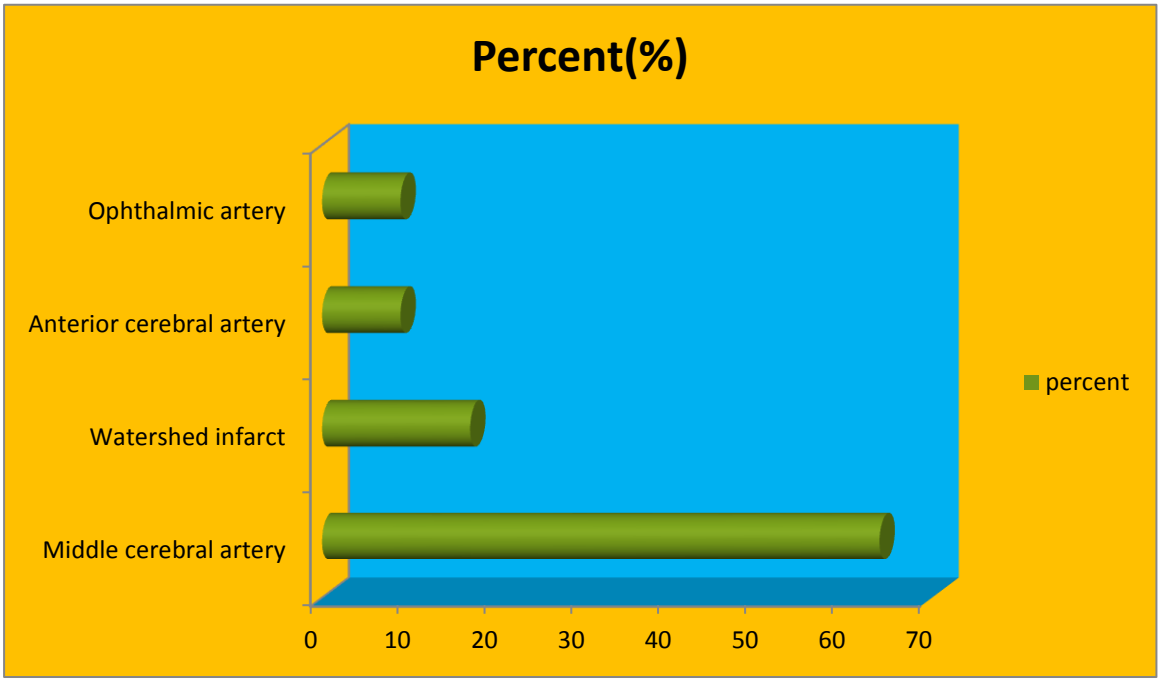


Chart 5. Vascular territories

7. Stroke subtype involved in Carotid Stenosis:

Out of 70 patients, 27 patients (39%) were affected with territorial infarcts, 21 (30%) patients were affected with subcortical infarcts and cortical and subcortical together accounted for 4 patients (6%). Under watershed infarct subtype external and internal ones accounted for 8 patients (11%) and 4 patients (6%) respectively.

Subtype	Total	Percentage
Territorial Infarct	27	39%
Subcortical Infarct	21	30%
Watershed Infract - External	8	11%
No Infarct	6	9%
Cortical + Subcortical Infarct	4	6%
Watershed Infarct - Internal	4	6%

Table 5. Stroke subtypes

8. Extracranial Carotid Stenosis and Stroke:

The study evaluated under percentage of stenosis which showed left unilateral stenosis in 28 patients (39%) closely followed by unilateral right stenosis in 23 patients (34%), and the remaining 27% affected bilaterally.

Percentage of Stenosis	**Unilateral**		**Bilateral**
	Right	**Left**	
No Stenosis (IMT > 1)	2 (3%)	0 (0%)	19 (27%)
< 50%	10 (15%)	8 (11%)	
50 - 69%	4 (6%)	12 (17%)	
70 - 99%	2 (3%)	3 (4%)	
100%	5 (7%)	5 (7%)	

Table 6. Distribution of Extra cranial Stenosis

Less than 50% stenosis were found in 18 patients (26%).Of which 8 (11%) had left sided stenosis and 10 (15%) had right sided stenosis, 50-99% stenosis were found in 21 patients (30%) of which 6 (9%) had left sided stenosis and 15 (21%) had right sided stenosis. 10 patients (14%) had complete (100%) occlusion

9. Comparison of extracranial stenosis with intracranial stenosis in stroke patients.

Among the 70 patients, 19 showed extracranial stenosis. Out of which, 10 had 100% stenosis for whom intracranial stenosis dectection is not possible. In remaining 60 patients, intracranial stenosis was detected in 35. Among them, 5 had bilateral intracranial stenosis. The remaining 25 did not show intracranial stenosis.

Extracranial Stenosis		Intracranial Stenosis	
Percentage	Number of Patients	Yes	No
0 - 99%	60	35	25
100%	10	Not Applicable	

Table 7. Split up of Extra and Intra cranial carotid stenosis

10. Unilateral Stenosis and stroke:

Among the 60 patients, 41 (68%) had unilateral extracranial stenosis of which 19 (31%) showed unilateral intracranial stenosis, one (2%) showed bilateral intracranial stenosis, 21 (35%) did not show stenosis in the intracranial part of internal carotid artery.

11. Bilateral Stenosis and stroke:

Among the 60 patients, 19 (27%) had bilateral extracranial stenosis. Of which, 5 (8%) had bilateral intracranial stenosis, 10 (19%) had unilatral intracranial stenosis of whom, 8 patients showed right sided stenosis and remaining 2 had left sided stenosis.

Patients with Bilateral Stenosis	
Extra Cranial Portion (n - 70)	Intra Cranial Portion (n - 60)
19 (27%)	5 (8%)

Table 8. Bilateral carotid disease

12. Stroke subtype in bilateral Stenosis:

Among the 70 patients, 19 (27%) had bilateral extracranial carotid stenosis of which 6 (8%) had external watershed infarct, 5 (7%) had middle cerebral artery territorial infarcts, 4 (6%) had subcortical infarcts. 4 patients (6%) did not show infarct of whom, 2 presented with monoparesis and 2 had only Transient Ischemic Attack.

13. Stroke subtype in unilateral extra cranial carotid stenosis without Intra cranial stenosis:

Among the 70 patients, 25 (27%) had unilateral extracranial carotid stenosis without intracranial internal carotid artery stenosis of which 11 patients (18%) showed territorial infarct involving middle cerebral artery, 9 (15%) had subcortical infarcts. 5 (8%) had watershed infarcts of whom, 3 (5%) had external watershed infarct and 2 showed Internal watershed infarct.

Discussion

Stroke is the leading cause of mortality and long term disability in adults. Incidence and prevalence of stroke varies according to the racial and ethnic group studied. Various risk factors dyslipidemia, diabetes mellitus, hypertension are responsible for the development of stroke. Carotid artery atherosclerotic disease has been recognized as a major cause of stroke and is responsible for 20 to 50% of ischemic stroke. Several studies have demonstrated that certain subgroups of the general population, such as blacks, Hispanics, and Asians have significantly higher incidence rates of Carotid artery occlusive disease compared to whites. [9,10,12]

Among the 70 patients who were included in the study, 83% of cases were males and remaining 17% were females.This is in line with another study done by Chin Sang Chun et al, from South Korea who had also showed 87.3% of male predominance.

The mean age of males and females are 58 years and 62 years respectively with the age ranging from 35-86 years in males and 30-71 years in females. 74% of patients are in the age group of 50 to 70 years which is similar to that reported by Chin Sang Chun et al with mean age of 65.5 years and age range of 43-81 years. The autopsy

study by Baker et al reported 66% of patients are in the age group of 50 to 70 years[50]

On analysing the risk factors in 70 patients, dyslipidemia has topped the list with 56 patients (80%), followed by smoking in 42 patients (60%), alcohol in 36 patients (51%), Diabetes Mellitus in 29 patients (41%), and finally systemic hypertension in 26 patients (37%). Many of them had more than one risk factors . Single risk factor was present in only 9 patients. Out of which , 2 had diabetes mellitus, 4 had systemic hypertension and 3 had dyslipidemia.

The study also showed that 44 patients had two risk factors. Among 44 patients 10 (15%) had Diabetes Mellitus and systemic hypertension, 34 (49%) had the habit of both smoking and alcohol consumption. 5 (7%) had all the five risk factors.

Our study results are comparable with other studies. Arenillas, et al [49] has reported intracranial internal carotid stenosis has been associated with dyslipidemia especially with increased total cholesterol and it also revealed that elevated low density lipoprotein also a risk factor for internal carotid stenosis.

The North Manhattan stroke study demonstrated that higher prevalence of Diabetes Mellitus in 67% and dyslipidemia in 62% of

patients with intracranial carotid stenosis when compared with extra cranial carotid stenosis in the study group of 714 patients.[51]

Impact of Diabetes Mellitus in the development of intracranial carotid stenosis also established in Wai Hong Chen et al who studied in chinese patients in the study group of 153,which showed 50% association.[13].

The varied clinical presentations are, (90%) 63 patients developed ischemic stroke, followed by transient ischemic attack in 6 patients (9%) and the least being ocular stroke which occured in 1 patient (1%).

On the basis of vascular territory involved, 45 patients (64%) had middle cerebral artery occlusion in which 12 showed multiple lesions in middle cerebral artery territory, which is followed by watershed infarct in 12 patients (17%) and transient ischemic attack in 6 patients (9%). Another 6 patients (9%) had anterior cerebral artery infarct and the least being ophthalmic artery occlusion that was seen in 1 patient (1%). This pattern of involvement comparable with study by Lee et al, lesion patterns and stroke mechanism in atherosclerotic middle cerebral artery disease and early diffusion-weighted imaging study among 63patients (62%) with middle cerebral

artery stenosis,32 patients showed fragmented infarct in the middle cerebral artery territory.[57]

In the overall 70 patients, 17 (24%) experienced transient ischemic attack out of which 11patients (15%) developed stroke and the remaining 6 (9%) did not develop stroke. In this study among 45 patients who had intra cranial stenosis, 12 experienced transient ischemic attack. 5 out of 15 patients without intracranial stenosis experienced transient ischemic attack. This shows that incidence of transient ischemic attack is higher in patients with intracranial stenosis compared to extracranial stenosis.

When categorised as sub types, the study revealed that the territorial infarct was the highest, accounting for 27 patients (39%), and subcortical infarct in 21 (30%), followed by external watershed infarcts in 8 patients (11%), internal watershed infarcts in 4 (6%), Normal imaging (transient ischemic attack) in 6 patients (9%), and finally combined cortical and subcortical involvement in 4 (6%).

Among the 70 patients, 19 (27%) had bilateral extracranial carotid stenosis of which 6 (8%) had external watershed infarct, 5 (7%) had territorial infarcts involving middle cerebral artery, 4 (6%) had subcortical infarcts. 4 patients (6%) did not show any infarct. Of

which, 2 patients presented with monoparesis and 2 patients presented with only transient ischemic attack.

25 patients (27%) had unilateral extracranial carotid stenosis without intracranial internal carotid artery stenosis. Of which 11 (18%) showed territorial infarct involving middle cerebral artery, 9 (15%) showed subcortical infarcts. 5 (8%) showed watershed infarcts of which 3 (5%) had external watershed infarct and 2 had internal watershed infarct.

In this study an attempt was made to analyse the concordance and discordance of carotid artery disease between extracranial and intracranial part of internal carotid artery. 19 patients (27%) had bilateral stenosis as compared to the study conducted in Korea which showed 33%. Less than 50% stenosis were found in 18 patients (26%), 50-99% stenosis were found in 21 patients (30%) of which 6 patients (9%) had left sided stenosis and 15 (21%) had right sided stenosis. 10 patients (14%) had complete occlusion. Only 3% had increased intima media thickness ratio of more than one, without stenosis in the extracranial internal carotid artery.

Out of total 70 patients with extracranial Internal carotid artery disease examined, 10 patients showed complete (100%) occlusion of extra cranial vessels, who cannot be further assessed for intra cranial

stenosis. In the remaining 60 patients, 35 (58%) showed associated intracranial stenosis and in remaining 25 (42%), no intra cranial stenosis was detected.

In a similar but invasive study (CT angiogram) performed by Chin Sang Chun et al at South Korea, discordance was observed in 56.3% of patients with extracranial stenosis. Out of 121 patients studied, concordance in 58 patients (47.9%) also revealed intracranial stenosis.

Among the 60 patients, 41 (68%) had unilateral extracranial stenosis of which 19 (31%) showed unilateral intracranial stenosis, 1(2%) showed bilateral intracranial stenosis and had middle cerebral artery territorial stroke on the side of intracranial internal carotid stenosis . 21 patients (35%) did not show stenosis in the intracranial part of internal carotid artery. Remaining 19 patients (27%) had bilateral extracranial stenosis of which 5 (8%) had bilateral intracranial stenosis, 10 (19%) had unilatral intracranial stenosis. In them, 8 patients showed right sided stenosis and remaining 2 had left sided stenosis.

Studies regarding combined intracranial & extracranial steno-occlusive disease is comparitively less. In a study on Taiwan chinese population by Lui et al, 42.2% of patients had intra cranial and extra

cranial carotid artery disease. But Feldmann et al, reported that only 9% in chinese population.[9]

Solberg et al, studied Cerebral atherosclerosis in Negroes and Caucasians from New Orleans, Jamaica and Norway. They performed 2166 autopsies in blacks and whites which showed the increased occurrence of intracranial stenosisis in blacks 43% compared to 8.5% in whites.[46]

Risk of stroke occurrence increases with increasing degree of stenosis in carotid artery. It is estimated that the annual risk of stroke occurrence increases by less than 1% in patients with <80% stenosis and 4.8% in patients with >90% stenosis. Early intervention in patients with lesser degree of carotid artery stenosis will decrease the risk of future stroke.

Transcranial doppler and carotid doppler are the best noninvasive and widely available screening tool for carotid stenosis. But the disadvantages of trans cranial doppler is that it is operator dependent technique and it is hampered by the 10-15% rate of inadequate temporal windows especially in older individuals. trans cranial doppler can be used as an initial imaging modality to exclude intracranial internal carotid artery stenosis.

Conclusion

1. The common risk factors for carotid artery disease in our study are dyslipidemia, systemic hypertension, diabetes mellitus, smoking and alcohol consumption, in the order of occurrence.
2. Incidence of transient ischemic attack was high in patients with intracranial carotid artery disease indicating the need for intensive management of these patients to prevent morbidity and mortality
3. The most common radiological presentation is the territorial infarct involving the middle cerebral artery territory followed by watershed infarcts.
4. Most of the patients with extracranial internal carotid artery disease also had co-existing intracranial internal carotid artery disease which in turn may further lead to stroke. This emphasizes the need to search for intracranial disease in patients with extra cranial carotid artery disease.
5. Transcranial doppler can be used as a noninvasive initial screening tool for detecting intracranial internal carotid artery stenosis before considering any invasive investigation.

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Comparison of extracranial with intra cranial carotid artery disease in ischemic stroke

Name	:	IP no	:
Age/sex	:	MIN no	:
DOA	:	DOD	:
Phone no	:	Address	:
Occupation	:		

Complaint :

Present history:

a) Time of onset of stroke :

b) What were his preceding symptoms ?

c) How did the associated symptoms appear in chronology ?

i st symptom

ii nd symptom

iii rd symptom

ivth symptom

v th sympom

others

d) Sensorium at onset LOC/FULLY CONSCIOUS/DROWSY

E) Was speech disturbance noted at the time of stroke ?

When ?

What type- Expressive / Sensory

Global

Dysarthria

Mutism

F)PAST HISTORY

H/O TIA No. Of episodes of TIA:

G) RISK FACTORS. SINCE WHEN ON TREATMENT

HTN

DM

CARDIAC DISEASE

Oral Pills

CLAUDICATION

DRUG ABUSE /OTHERS

H) TYPE NUMBER DURATION

CIGARETTES

ALCOHOL

TOBACCO

I) DIET

VEG/NONVEG/EGG+VEG

J) What is the nature of work? Sedentary/moderate/heavy exertion

k) What are the associated diseases and systems involved?

CVS SEIZURES

P/A MOOD DISORDER

R/S MIGRAINE/OTHER HEADACHES

PERIPHERAL VASCULAR DISEASE:

L) TREATMENT/DRUG HISTORY:

What drugs?

Is he regular?

Any side effects?

CLINICAL EXAMINATION

1) Markers of atherosclerosis: Skin xanthomas /arcus/ xanthomata

2) Carotid bruit-

PULSE		Right	Left
	Carotids		
	Brachial		
	Radial		
	Dorsalis pedis		
B.P	SUPINE	mmHg	
	STANDING	mmHg	

3) Waist/hip ratio

4) Ocular fundus- HTN/DM RETINOPATHY

5) Consciousness- NORMAL/ DROWSY/STUPOR/COMA

6) Lobar functions

Frontal : Attention
Apathy
Story recall
Judgement

Parietal : Cortical sensation
Apraxia/Finger body gnosis/Neglect

Occipital : Vision

Temporal : Memory: verbal/ Visual

7)Cranial nerves:

8) Motor system			
		RT	LT
BULK	UL		
	LL		
TONE	UL		
	LL		
POWER	UL PROXIMAL		
	DISTAL		
	LL PROXIMAL		
	DISTAL		

SUPERFICIAL REFLEXES
Plantar
Corneal
Abdominal

DTR	BJ	TJ	SJ	KJ	AJ
R					
L					

GAIT:

9) Sensory

10) Cerebellar Signs

11) EP Signs

12) Bladder /Bowel Symptoms

13) Signs of meningeal irritation: Neck Rigidity/Kernig's sign

14) Other systems

CARDIO VASCULAR SYSTEM:

RESPIRATORY SYSTEM:

ABDOMEN:

15)CLINICAL DIAGNOSIS:

INVESTIGATIONS

Complete haemogram:

TC : Cells/c mm	Urea : mg/dl	URINE R/E
DC : P %L %E %	Creatinine : mg/dl	
HB : gms%	SODIUM: Meq/L	HIV:
PCV : %	potassium: meq/l	
Platelet: lakhs/ mm^3	cholesterol: mg/dl	VDRL:
ESR : mm/hr	HDL: mg/dl	
SUGAR : mgs/dl	TGL: mg/dl	OTHERS

Cardiac evaluation:

ECG
C X R PA VIEW
Echocardiography:
Ejection fraction:
Wall motion abnormality:
Clot:

Other investigations:

CAROTID DOPPLER STUDY

The intima medial thickness in common carotid artery and internal carotid artery RT- mm, LT- mm

ARTERY	RT PSV	RTEDV	LT PSV	LT EDV	PLAQUE
CCA (PROXIMAL)					
CCA(DISTA)					
ICA					
ECA					
CAROTID INDEX					

PLAQUE Location:
Ulceration: Yes/No
Calcification: Yes/No
Measures: mm
% Stenosis:

IMPRESSION:

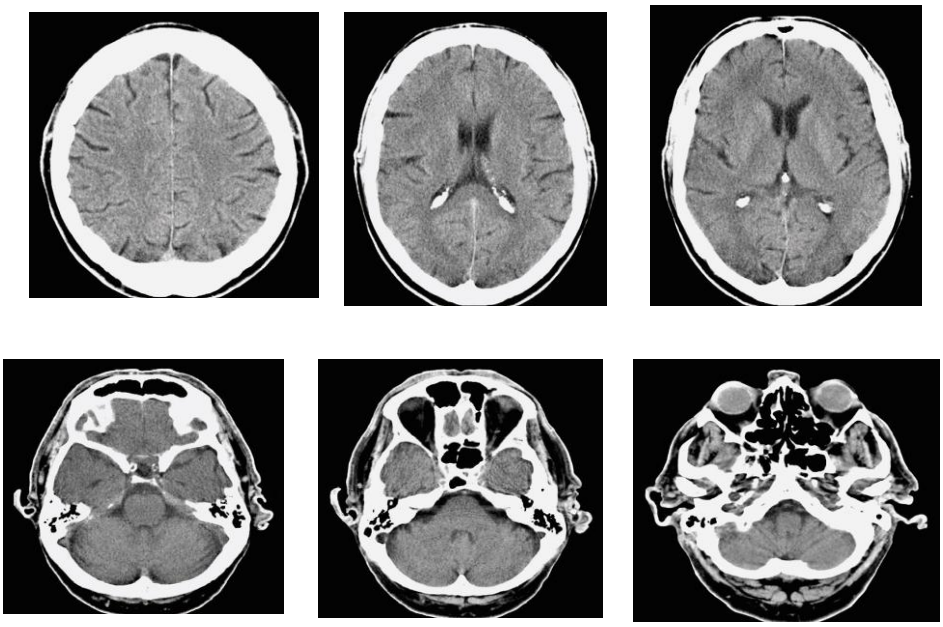
TRANS CRANIAL DOPPLER

PARAMETERS	**RT CS**	**LT CS**	**RT OA**	**LT OA**
PEAK				
MEAN				
DIAS				
PI				
RI				
HR				

IMPRESSION:

CT ANGIOGRAM:

CT BRAIN



FINDINGS:

MRI BRAIN

T1	T2	T2 FLAIR	DIFFUSION

MRA :

IMPRESSION:

NIHSS SCALE

ITEM	NAME	RESPONSE	SCORE		
IA	LOC	Alert Not alert,obtunded Unresponsive	0 2 3		
IB	LOC QUESTIONS	Answers both correctly Answers one correctly Answers neither correctly	0 1 2		
IC	LOC COMMANDS	Performs both facts correctly Performs one fact correctly Performs neither facts correctly	0 1 2		
2	GAZE	Normal Partial gaze palsy Total gaze palsy	0 1 2		
3	VISUAL FIELDS	No visual loss Partial hemianopia Complete hemianopia	0 1 2		
4	FACIAL PALSY	Normal Minor paralysis Partial paralysis Complete paralysis	0 1 2 3		
5	MOTOR ARM A.LEFT B.RIGHT	No drift Drift before 10 sec Fall before 10 sec No effort against gravity No drift movements	0 1 2 3 4		
6	MOTOR LEG A.LEFT B.RIGHT	No drift Drift before 5 sec Fall before 5 sec No effort against gravity No movement	0 1 2 3 4		
7	ATAXIA	Absent One limb Two limbs	0 1 2		
8	SENSORY	Normal Mild loss Severe	0 1 2		
9	LANGUAGE	Normal Mild aphasia Severe aphasia Mute or global aphasia	0 1 2 3		
10	DYSARTHRIA	Normal Mild Severe	0 1 2		
11	EXTINCTION / INATTENTION	Normal Mild Severe	0 1 2		
		TOTAL	42		

MASTER CHART

S.No	Age	Sex	Presentation	vascular territory	subtype	TIA	DM	SHT	Smoking	Alcohol	Dyslipidemia	Extracranial Carotid Stenosis	Intracranial Carotid Stenosis
1	65	M	R HP,GA	MCA	TI	Yes	Yes	Yes	Yes	Yes	Yes	E and F	Yes R
2	60	M	R HP,WA	MCA	TI	No	No	No	No	No	Yes	D	Yes L
3	55	M	R HP,GA	WS	EWS	No	No	No	Yes	Yes	Yes	B	Yes L
4	30	F	R HP	WS	IWS	No	No	No	No	No	No	D	No
5	36	M	L HP	MCA	TI	No	No	No	No	Yes	Yes	A	No
6	60	M	R HP	MCA	TI	No	No	Yes	Yes	Yes	Yes	D	No
7	55	M	L HP HS HA	MCA	TI	No	No	No	Yes	Yes	Yes	A	No
8	55	M	R HP GA	MCA	TI	No	No	No	Yes	Yes	Yes	H	NA
9	52	M	L HP HS	MCA	SC	No	No	No	Yes	Yes	Yes	A	No
10	75	M	L HP	MCA	SC	No	Yes	Yes	Yes	Yes	Yes	C	Yes R
11	50	M	R HP	MCA	SC	No	Yes	No	Yes	Yes	Yes	B and C	Yes both
12	57	M	BA	MCA	TI	No	No	No	Yes	Yes	No	C	No
13	65	M	R HP GA	MCA	TI	Yes	Yes	No	No	No	Yes	A and B	Yes L
14	63	M	R HA	MCA	SC	No	Yes	No	No	No	Yes	F	Yes L
15	45	M	L BMP	NORMAL	NI	No	Yes	No	Yes	Yes	Yes	E and F	Yes R
16	60	M	R HP	WS	EWS	No	No	No	Yes	No	Yes	B and C	No
17	46	M	R HP VL	MCA	SC	No	Yes	No	Yes	Yes	Yes	B	No
18	56	M	R HA	MCA	SC	No	Yes	Yes	Yes	Yes	Yes	D	No
19	56	M	TIA	NORMAL	NI	Yes	Yes	No	No	No	No	E and F	Yes R
20	61	M	L HP HS HA	MCA	TI	No	No	No	Yes	No	No	B	No
21	68	F	R HP SEIZURE	WS	EWS	Yes	No	Yes	No	No	Yes	D and E	No
22	58	F	R HP	ACA	SC	No	No	Yes	No	No	No	A and D	No
23	45	M	L HP	MCA	TI	No	No	No	Yes	No	Yes	A	Yes R
24	86	M	R HP GA	MCA	TI	Yes	Yes	Yes	Yes	Yes	Yes	D	No
25	60	M	R HP	MCA	TI	No	No	No	Yes	Yes	Yes	H	NA
26	65	M	TIA	NORMAL	NI	Yes	No	Yes	No	No	No	G	NA
27	52	M	L VL WA	WS	EWS	Yes	Yes	No	Yes	No	No	E and H	Yes Both
28	65	F	R HP HS	WS	IWS	No	Yes	Yes	No	No	Yes	B	Yes L
29	54	M	L BMP	ACA	SC	No	No	No	Yes	Yes	Yes	A	No
30	58	M	R HA	MCA	SC	No	No	Yes	Yes	Yes	Yes	D	No
31	70	F	R CMP	ACA	TI	No	Yes	Yes	No	No	Yes	I	Yes R
32	42	M	L HP	MCA	TI SC	Yes	Yes	No	Yes	Yes	Yes	G	NA
33	50	M	L HP	MCA	TI SC	No	Yes	No	No	No	Yes	G	NA
34	65	M	R HP	MCA	SC	No	No	Yes	No	No	Yes	E	Yes both
35	60	M	R HP	MCA	SC	No	No	Yes	No	No	Yes	E	Yes R
36	35	M	L VL	OA	TI	Yes	Yes	No	Yes	Yes	Yes	C and H	Yes R L-NA
37	55	F	R HP	MCA	SC	No	No	Yes	No	No	Yes	F	No
38	65	F	R HP,GA	MCA	TI	Yes	Yes	Yes	No	No	Yes	E and D	Yes both
39	60	M	R HP,WA	MCA	TI	No	Yes	No	No	No	Yes	D	Yes L
40	65	M	R HP,GA	WS	EWS	No	Yes	No	Yes	Yes	Yes	B	Yes L
41	65	F	R HP	WS	IWS	No	Yes	No	No	No	No	D	No
42	56	M	L HP	MCA	TI	No	Yes	No	No	Yes	Yes	A	No
43	66	M	R HP	MCA	TI	No	No	Yes	Yes	Yes	Yes	D	No
44	66	M	L HP HS HA	MCA	TI	No	No	No	Yes	Yes	Yes	A	No

45	55	M	R HP GA	MCA	TI	No	Yes	No	Yes	Yes	Yes	H	NA
46	57	M	L HP HS	MCA	SC	No	No	No	Yes	Yes	Yes	A	No
47	76	M	L HP	MCA	SC	No	No	Yes	Yes	Yes	Yes	C	No
48	54	M	R HP	MCA	SC	No	No	No	Yes	Yes	Yes	B and C	Yes both
49	62	M	BA	MCA	TI	No	No	No	Yes	Yes	No	C	No
50	66	M	R HP GA	MCA	TI	Yes	No	No	No	No	Yes	A and B	Yes L
51	65	M	R HA	MCA	SC	No	No	No	No	No	Yes	F	Yes L
52	47	M	L BMP	NORMAL	NI	No	Yes	No	Yes	Yes	Yes	E and F	Yes R
53	62	M	R HP	WS	EWS	No	Yes	No	Yes	No	Yes	B and C	No
54	47	M	R HP VL	MCA	SC	No	No	No	Yes	Yes	Yes	B	Yes L
55	58	M	R HA	MCA	SC	No	Yes	Yes	Yes	Yes	Yes	D	Yes L
56	59	M	TIA	NORMAL	NI	Yes	No	No	No	No	No	E and F	Yes R
57	62	M	L HP HS HA	MCA	TI	No	No	No	Yes	No	No	B	No
58	67	F	R HP	WS	EWS	Yes	Yes	Yes	No	No	Yes	D and E	Yes R
59	58	F	R HP	ACA	SC	No	No	Yes	No	No	No	A and D	Yes R
60	48	M	L HP	MCA	TI	No	No	No	Yes	No	Yes	A	Yes R
61	84	M	R HP GA	MCA	TI	Yes	No	Yes	Yes	Yes	Yes	D	Yes L
62	61	M	R HP	MCA	TI	No	Yes	No	Yes	Yes	Yes	H	NA
63	66	M	TIA	NORMAL	NI	Yes	Yes	Yes	No	No	No	G	NA
64	53	M	L VL WA	WS	EWS	Yes	No	No	Yes	No	No	E and H	Yes Both
65	66	F	R HP HS	WS	IWS	No	No	Yes	No	No	Yes	B	Yes L
66	55	M	L BMP	ACA	SC	No	Yes	No	Yes	Yes	Yes	A	Yes R
67	57	M	R HA	MCA	SC	No	No	Yes	Yes	Yes	Yes	D	Yes L
68	71	F	R CMP	ACA	TI	No	No	Yes	No	No	Yes	I	Yes R
69	44	M	L HP	MCA	TI SC	Yes	No	No	Yes	Yes	Yes	G	NA
70	55	M	R HP	MCA	TI SC	No	No	Yes	No	No	Yes	H	NA

A- Right less than 50% stenosis
B- Left less than 50% stenosis
C- Right 50%-69% stenosis
D- Left 50%-69% stenosis
E- Right 70%-99% stenosis
F- Left 70%-99% stenosis
G-100% stenosis
H- 100% stenosis
I - IMT Ratio more than 1

R- Right
L-Left
HP-Hemi Paresis
HS-Hemi Sensory Impairment
VL-Visual Loss
CMP-Crural Mono Paresis
BMP_Brachial monoparesis
HA-Hemi Ataxia
BA-Brocas Aphsis
WA_Wernickes aphasia
TIA-Transient Isheamic Attack
DM_Diabetes Mellitus

SHT-Systemic Hypertension
GA-Global aphasia
ACA-Anterior Cerebral Artery
MCA-Middle Cerebral Artery
OA -Ophthalmic Artery
TI -Territorial Infarct
WS-Water Shed Infarct
IWS-Internal -Water Shed Infarct
EWS-External -Water Shed Infarct
SC -Sub Cortical Infarct
NA-Not Applicable
NI - Negative Imaging

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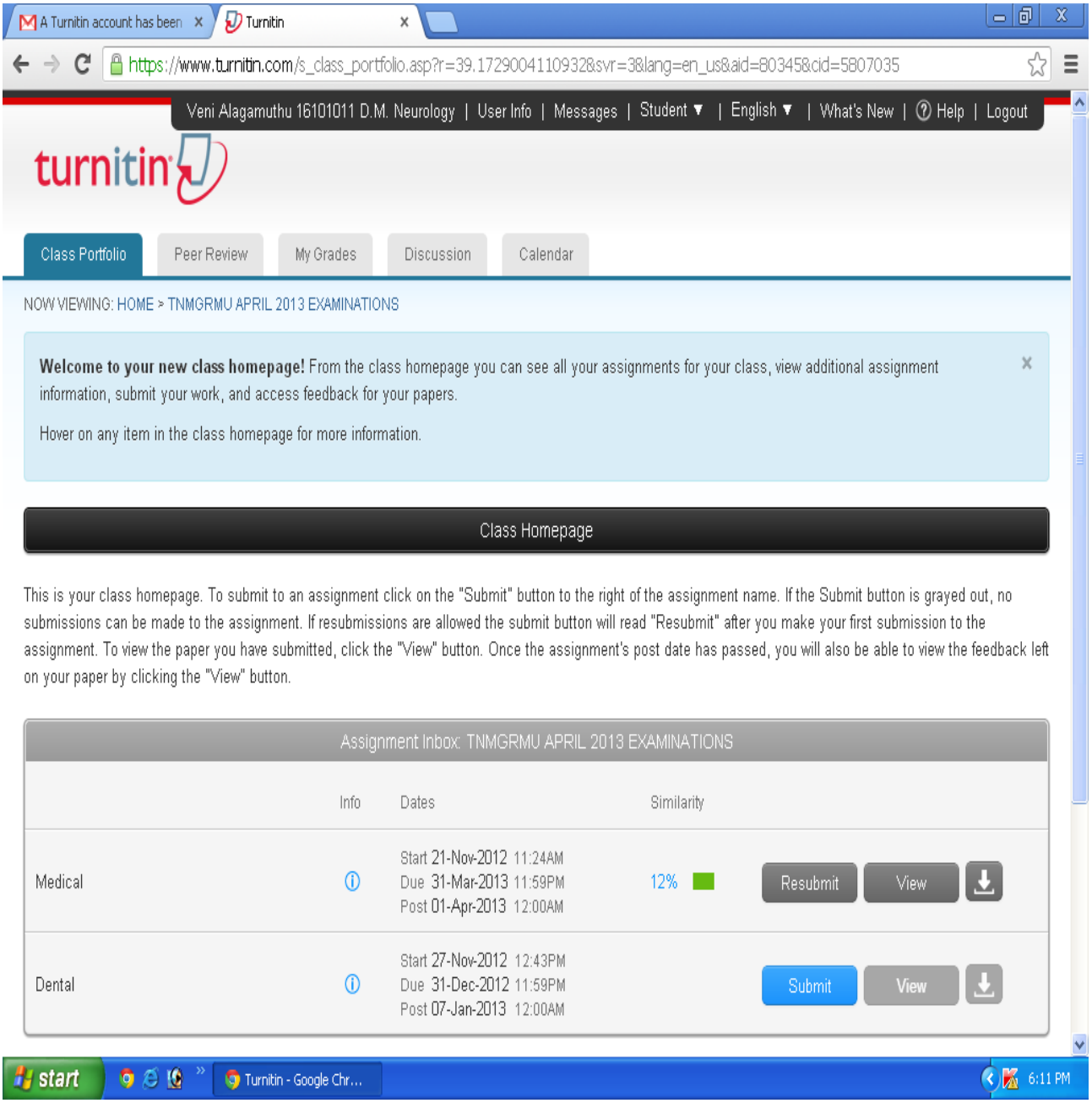
1 INTRODUCTION Stroke is defined as developing symptoms or and signs of global or focal, loss of cerebral function with symptoms lasting for more than 24 hours or leading to death with no apparent cause other than vascular origin.(1) Globally, the rate of occurrence of stroke is estimated as 400 - 800 per lakh of population. Around 57 lakh deaths occur due to stroke in a year. Approximately, 1.6 crore new acute strokes are reported yearly.(3) Worldwide, stroke mortality is high in Eastern Europe and Asia. In India, the rate of occurrence of stroke is estimated as 90 - 222 per lakh of population. Approximately, 14 – 16 lakhs new acute strokes are reported yearly. 12% of them occur in the age...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