A DISSERTATION

ON

CAROTID DOPPLER ULTRASONOGRAPHY EVALUATION IN PATIENTS WITH STORKE

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CERTIFICATE

This is to certify that the dissertation entitled **"CAROTID DOPPLER ULTRASONOGRAPHY EVALUATION IN PATIENTS WITH STROKE"** is a bonafide work done by **Dr. KAMARUL JAMAN.A.** in **M.D BRANCH I GENERAL MEDICINE** at Government Mohan Kumaramangalam Medical College, Salem, to be submitted to The Tamil Nadu Dr.M.G.R Medical University, in partial fulfillment of the University Rules and Regulation for the award of M.D Degree Branch I General Medicine, under my supervision and guidance, during the academic period from June 2004 to March 2007.

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DECLARATION

I solemnly declare that this dissertation **"CAROTID DOPPLER ULTRASONOGRAPHY EVALUATION IN PATIENTS WITH STROKE"** was prepared by me at Government Mohan Kumaramangalam Medical College and Hospital, Salem under the guidance and supervision of **Prof. Dr. K. Sathyamoorthy M.D**, Professor of Medicine, Govt. Mohan Kumaramangalam Medical College and Hospital Salem.

This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the university regulations for the award of the degree of M.D. Branch 1 General Medicine.

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CONTENTS

INTRODUCTION

Stroke is defined as rapid onset of focal neurological deficit resulting from diseases of cerebral vasculature and its contents[1]. Community surveys in India have shown a crude prevalence rate for hemiplegia in the range of 200 per 100,000 persons, nearly 1.5% of all urban hospital admission, 4.5% of all medical and around 20% of neurological cases.[1]

Stroke is one of the leading causes of mortality and morbidity. Ischemic infarcts account for 80-85% of all strokes. Stroke related neurological disability has a major impact on the patient, family members and caretakers. 17% will remain institutionalized and 25-30% will need moderate to total assistance for activities of daily living[2].

Carotid atherosclerosis is one of the main risk factor for ischemic stroke which accounts for 5-10% of all ischemic stroke[3]. Carotid endarterectomy has been shown to effectively reduce the risk of developing stroke in patients with significant stenosis i.e. 70% or more.

Various noninvasive vascular techniques can now be used to evaluate Carotid atherosclerosis which includes Carotid Doppler Ultrasonography, transcranial Doppler, CT angiography, Magnetic Resonance angiography, contrast enhanced Magnetic Resonance angiography.

Among the tests Carotid Doppler ultrasound is a noninvasive, cost effective and easy available mode of investigation to identify the patients with significant stenosis and other changes due to atherosclerosis.

Since the noninvasive tests are used for preliminary screening before doing Carotid endarterectomy the value of the tests lie in their ability to accurately identify patients with significant stenosis. However newer studies of older technologies and the emergence of new noninvasive tests justify a revaluation of published data about noninvasive testing of Carotid arteries.

The purpose of this study is to estimate retrospectively the various changes in Carotid arteries by Doppler Ultrasonography in patients who had already suffered a stroke, which in turn determine the value of using Carotid Doppler Ultrasonography as a modality of test to screen for atherosclerosis and the risk of developing stroke in people with risk factors for stroke.

AIMS OF THE STUDY

1. To study the role of Carotid Doppler Ultrasonography in the diagnosis and management of stroke patients.

2. To study retrospectively the various abnormalities in Carotid arteries in patients who had suffered stroke.

3. To determine the usefulness of doing Carotid Doppler Ultrasonography as a screening procedure in predicting the chance of developing stroke in persons having risk factors for stroke.

HISTORY

The modern era of blood vessel imaging began in 1929 when Forssmann injected himself with contrast medium through a large bore catheter. Although recognized to be a hazardous procedure the diagnostic ability of angiography was quickly appreciated and a whole new field of neurosurgery rapidly emerged. Dott in Edinburg wrapped a cerebral aneurysm in 1932. Eastcott in London performed the first Carotid endarterectomy in 1954. While angiographic techniques have continued to improve so the appreciation of risks. When the benefits of treatment are minimal such as in patients with low grade Carotid stenosis the risk of angiography can outweigh its benefits. This has led to the development of less invasive modalities to image the blood vessel which includes Carotid Doppler Ultrasonography, magnetic resonance angiography and CT angiography[4].

Carotid ultrasound was initially performed with continuous wave Doppler. The next major advance was the elegant combination of real time imaging with Doppler information called Duplex Ultrasonography. The most recent development has been colour flow Doppler Ultrasonography whereby flow information is colour coded and superimposed on the gray scale images[4].

STROKE

A stroke or cerebrovascular accident is defined as abrupt onset of a neurological deficit that is attributed to a focal vascular cause. Thus the definition of stroke is clinical, and laboratory studies indicating brain imaging are used to support the diagnosis. The clinical manifestations of stroke are highly variable because of the complex anatomy of the brain and its vasculature. Cerebral ischemia is caused by reduction in blood flow that lasts longer than several seconds. Neurologic symptoms manifest within seconds because neurons lack glycogen and energy failure is rapid. When blood flow is restored quickly brain tissue can recover fully and the patient's symptoms are only transient. This is called transient ischemic attack. Typically the signs and symptoms of transient ischemic attack last for 5 to 15 minutes but by definition it should be less than 24 hours. If the cessation of blood flow lasts for more than a few minutes, infarction or death of the brain tissue occurs. If the signs and symptoms last for more than 24 hours it is termed as stroke[3].

PATHOPHYSIOLOGY OF ISCHEMIC STROKE

Acute occlusion of an intracranial vessel causes reduction in blood flow to the brain region it supplies. The magnitude of flow reduction is a function of collateral blood flow and this depends on individual vascular anatomy and the site of occlusion. A fall in cerebral blood flow to zero causes death of brain tissue within 4 to 10 minutes. Values less than 16 to 18 ml/100 gram tissue per minute cause infarction within an hour and values less than 20ml/100gram

tissue per minute cause ischemia without infarction unless prolonged for several hours or days. If the blood is restored prior to a significant amount of cell death the patient may experience only transient symptoms. Tissue surrounding the core region of infarction is ischemic but reversibly dysfunctional and referred to as the ischemic penumbra. The penumbra may be imaged by perfusion-diffusion imaging with magnetic resonance imaging. The ischemic penumbra will eventually infarct if no change in flow occurs, hence saving the ischemic penumbra is the goal of thrombolytic therapy and newer therapies are under investigation[3].

Cellular death occurs via two distinct pathways.

1. A necrotic pathway in which cellular cytoskeletal breakdown is rapid principally due to energy failure of the cell.
2. An apoptotic pathway in which cells become programmed to die.

Ischemia produces necrosis by starving neurons of glucose which in turn results in failure of the mitochondria to produce ATP. Without ATP, membrane ion pumps stops functioning and neurons depolarize, allowing intracellular calcium to rise. Cellular depolarization also causes glutamate release from synaptic terminals. Excess extracellular glutamate produces neurotoxicity by agonizing post synaptic glutamate receptors that increase neuronal calcium influx. Free radicals are produced by membrane lipid degradation and mitochondrial dysfunction. Free radicals cause catalytic destruction of membranes and likely damage the other vital functions of the cells. Lesser

degrees of ischemia as are seen within the ischemic penumbra, favor apoptotic cellular death causing cells to die days to weeks later. There are no clinically proven strategies that alter these ischemic cascades despite extensive clinical study[3].

RISK FACTORS FOR ISCHEMIC STROKE

Identification and control of modifiable risk factors is the best strategy to reduce the burden of the stroke, as the total number of strokes could be reduced substantially by these means.

ATHEROSCLEROSIS-RISK FACTIORS

Older age, family history of thrombotic stroke, diabetes mellitus, hypertension, tobacco smoking and abnormal blood cholesterol are either proven or probable risk factors for ischemic stroke, largely by their link to atherosclerosis. Risk of second stroke is strongly influenced by prior stroke or transient ischemic attack, depending on cause. Many cardiac conditions predispose to stroke, including atrial fibrillation and recent myocardial infarction. Oral conrtraceptives increases the stroke risk, and certain inherited and acquired hypercoagulable states predispose to stroke. Hypertension is the most significant of the risk factors in general and all hypertension should be treated[3].

THE PATHOGENESIS OF ATHEROSCLEROSIS

Atherosclerosis is the leading cause of death and disability in the developed world. Atherosclerosis manifests itself focally not only in space, as just described, but in time as well. Atherogenesis in humans typically occurs over a period of many years, usually many decades. Growth of atherosclerotic plaques probably does not occur in a smooth linear fashion, but rather discontinuously, with periods of relative quiescence punctuated by periods of rapid evolution. After a generally prolonged 'silent' period, atherosclerosis may become clinically manifest.

INITIATION OF ATHEROSCLEROSIS

Fatty Streak Formation

An integrated view of experimental results in animals and study of human atherosclerosis suggests that the "fatty streak" represents the initial lesion of atherosclerosis. The formation of these early lesions of atherosclerosis most often seems to arise from focal increases in the content of lipoproteins within regions of the intima. This accumulation of lipoprotein particles may not result simply from an increased permeability, or "leakiness", of the overlying endothelium. Rather, these lipoproteins may collect in the intima of arteries because they bind to constituents of the extracellular matrix, increasing the residence time of the lipid-rich particles within the arterial wall. Lipoproteins that accumulate in the extracellular space of the intima of arteries

often associate with proteoglycan molecules of the arterial extracellular matrix, an interaction that may promote the retention of lipoprotein particles by binding them and slowing their egress from the intima.

Lipoprotein particles in the extracellular space of the intima, particularly those bound to matrix macromolecules, may undergo chemical modifications. Accumulating evidence supports a pathogenic role for such modifications of lipoproteins in atherogenesis. Two types of such alterations in lipoproteins bear particular interest in the context of understanding how risk factors actually promote atherogenesis: oxidation and nonenzymatic glycation.

LIPOPROTEIN OXIDATION

Lipoproteins sequestered from plasma antioxidants in the extracellular space of the intima become susceptible to oxidative modification. Oxidatively modified low-density lipoprotein (LDL), rather than being a defined homogenous entity, actually comprises a variable and incompletely defined mixture. Both the lipid and protein moieties of these particles can participate in oxidative modification. Modifications of the lipids may include formation of hydroperoxides, lysophospholipids, oxysterols, and aldehydic breakdown products of fatty acids. Modifications of the apoprotein moieties may include breaks in the peptide backbone as well as derivatization of certain amino acid residues. A more recently recognized modification may result from local hypochlorous acid production by inflammatory cells within the plaque, giving rise to chlorinated species such as chlorotyrosyl moieties. Considerable

evidence supports the presence of such oxidation products in atherosclerotic lesions.

NONENZYMATIC GLYCATION

In diabetic patients with sustained hyperglycemia, nonenyzmatic glycation of apolipoproteins and other arterial proteins likely occurs that may alter their function and propensity to accelerate atherogenesis.

LEUKOCYTE RECRUITMENT

After the accumulation of extracelluar lipid, recruitment of leukocytes occurs as a second step in the formatuion of the fatty streak. The white blood cell types typically found in the evolving atheroma include primarily cells of the monouclear lineage: monocytes and lymphocytes. A number of adhesion molecules or receptors for leukotypes expressed on the surface of the arterial endothelial cell likely participate in the recruitment of leukocytes to the nascent fatty streak. Constituents of oxidatively modified LDL can augment expression of leukocyte adhesion molecules. The example illustrates how the accumulation of lipoproteins in the arterial intima may link mechanistically with leukocyte recruitment and subsequent events in lesion formation.

Once adherent to the surface of the arterial endothelial cell via interaction with adhesion receptors, the monocytes and lymphocytes penetrate the endothelial layer and take up residence in the intima. In addition to products of modified lipoproteins, cytokines (a class of protein mediators of inflammation) can regulate the expression of adhesion molecules involved in

leukocyte recruitment. For example the cytokines interleukin 1 (IL-1) or tumor necrosis factor (TNF-) induce or augment the expression of leukocyte adhesion molecules on endothelial cells.

FOAM CELL FORMATION

Once resident within the intima, the mononuclear phagocytes differentiate into macrophages and transform into lipid-laden foam cells. The conversion of monoculear phagocytes into foam cells requires the uptake of lipoprotein particles by receptor mediated endocytosis. One might suppose that the well-recognized “classical” receptor for LDL mediates this lipid uptake. Patients or animals lacking effective LDL mediates this lipid uptake. Patients or animals lacking effective LDL receptors due to genetic alterations (e.g. familial hypercholesterolemia), however, have abundant arterial lesions and extrarterial xanthomata rich in macrophage-derived foam cells.

Macrophages taking up modified lipoproteins, much like intrinsic vascular wall cells, may elaborate cytokines and growth factors that can further signal some of the cellular events in lesion complication.

ATHEROMA EVOLUTION AND COMPLICATIONS

Involvement of arterial smooth-muscle Cells

Although the fatty streak commonly precedes the development of a more advanced atherosclerotic plaque, not all fatty streaks progress to form complex atheromas. Why do some fatty streaks progress to form complex

atheromas. Why do some fatty streaks progress to fibrous lesions while others do not? By what mechanisms do fatty streaks evolve into more complex lesions? While accumulation of lipid-laden macrophages is the hallmark of the fatty streaks, accumulation of fibrous tissue typifies the more advanced atherosclerotic lesion. The smooth-muscle cell synthesizes the bulk of the extra cellular matrix of the complex atherosclerotic lesion. Hence, arrival of smooth-muscle cells and their elaboration of extracellular matrix probably provides a critical transition, yielding a fibrofatty lesion in place of a simple accumulation of macrophage-derived foam cells.

BLOOD COAGULATION

In addition to locally produced mediators, atherogenic risk factor signals related to blood coagulation and thrombosis likely contribute to atheroma evolution and complication. Current evidence suggests that fatty streak formation begins underneath a morphologically intact endothelium. In advanced fatty streaks, however, microscopic breaches in endothelial integrity may occur. Microthrombi rich in platelets can form at such sites of limited endothelial denudation, due to exposure of the highly thrombogenic extraceullar matrix of underlying basement membrane.

MICROVESSELS

As artherosclerotic lesions advance, abundant plexi of microvessels develop in connection with the artery's vasa vasorum. These newly developing microvasucular networks may contribute to lesion complication in several

ways. These blood vessels provide an abundant surface area for leukocyte trafficking and may serve as the portal of entry and exit of white blood cells from the established atheroma. The plaques microvessels may also furnish foci for intraplaque hemorrhage. Like the neovessels in the diabetic retina, microvessels of the plaque may be friable and prone to rupture and produce focal hemorrhage.

PLAQUE EVOLUTION

Traditionally, atherosclerosis research has focused much attention on proliferation of smooth-muscle cells, yet these cells actually replicate rather slowly in complicated atherosclerotic lesions. Estimates of the rate of smooth-muscle cell division in such lesions at a given time show a replicative rate below 1%. Such observations do not exclude bursts of proliferative activity at certain junctures in the history of an artheroma, perhaps in association with local thrombin generation due to microvascular hemorrhage or formation of a microthrombus at a site of localized endothelial denudation.

Thus, during the evolution of the atherosclerotic plaque, a complex balance between entry and egress of lipoproteins and leukocytes, cell proliferation and cell death, extra cellular matrix production and remodeling, as well as calcification and neovascularization contribute to lesion formation.

PLAQUE INSTABILITY AND RUPTURE

Patholgoic studies afford considerable insight into the microanatomic substrate underlying the “instability” of plaques that are not criticially stenotic. A superficial erosion of the endothelium of a frank plaque rupture or fissure usually produces the thrombus that causes episodes of unstable angina pectoris or the occlusive and relatively persistent thrombus that causes acute myocardial infarction. In the case of carotid atheroma, a deeper ulceration that provides a nidus for formation of platelet thrombi may underlie the unstable syndromes that cause transient ischemic attacks.

Rupture of the plaque’s fibrous cap permits contact between coagulation factors in the blood with highly thrombogenic tissue factor expressed by macrophage foam cells in the plaque’s lipid rich core.

Not all atheroma exhibit the same propensity to rupture. Pathologic studies of culprit lesions that have caused acute myocardial infarction reveal several characteristic features. Plaques that have proved vulnerable tend to have thin fibruous caps, relatively large lipid cores, and a high content of macrophages. Morphonmetric studies of such culprit lesions show that macrophages and T lymphocytes predominate at the site of plaque rupture. On the other had, sites of plaque rupture contain relatively few smooth-muscle cells. The cells that concentrate at sites of plaque rupture bear markers of inflammatory activation.

Inflammatory mediators may actually regulate processes that govern the integrity of the plaque's fibrous cap and hence its propensity to rupture. For example, the T cell-derived cytokine IFN-, found in atherosclerotic plaques and required to induce the HLA-DR present at sites of rupture, can inhibit growth and collagen synthesis of smooth-muscle cells. Cytokines derived from activated macrophages such as TNF- α or IL – 1 in addition to T cell – derived IFN-γ can elicit the expression of genes that encode proteolytic enzymes that can degrade the extracellular matrix of the plaque's fibrous cap. Thus inflammatory mediators can impair collagen synthesis required for maintenance and repair of the fibruous cap and trigger degradation of extraceullar matrix macromolecules, processes that weaken the plaque's fibrous cap and enhance its vulnerability to rupture. In contrast to vulnerable plaques, those with a dense extracellular matrix and relatively thick fibrous cap without substantial tissue factor-rich lipid cores seem generally resistant to rupture and unlikely to provoke thrombosis.[3]

BLOOD SUPPLY OF THE BRAIN

Two internal Carotid and the two vertebral arteries carry the total arterial supply of the brain[5].

1. INTERNAL CAROTID ARTERIES

Internal Carotid arteries on either side which bifurcates from the common Carotid artery at C3 – C5 level is divided into four segments

The first two segments are extra cranial namely Carotid bulb and cervical segment.

The third segment is the internal Carotid artery segment within the petrous temporal bone.

The distal two segments are the cavernous and intracranial (supraclinoid) internal Carotid artery segments.

CAROTID BULB

The Carotid bulb includes the distal 2 to 4cms of the common Carotid artery, the bulbous dilatation at the internal Carotid origin and 2 to 4 cms segment beyond the bifurcation.

Flow at the common Carotid bifurcation is complex and flow distal to the bulb is normally laminar. Flow reversal within the posterior bulb is normal.

CERVICAL SEGMENT

The ascending or cervical internal Carotid artery typically has neither narrowing nor dilatations, almost never branches and does not taper. At its origin the internal Carotid artery initially lies posterolaterally to the external Carotid artery then it crosses behind and then becomes medial to the external Carotid artery.

In about 10% of cases the internal Carotid artery originates from the common Carotid bifurcation medial to the external Carotid artery.

CERVICAL INTERNAL CAROTID ARTERY ANOMALIES

Anomalous external Carotid branches sometimes arise from the cervical internal Carotid artery. Persistent embryonic vessels may sometimes anastomose with the veterbrobasilar system.

PETROUS SEGMENT

The intraosseous segment begins where the internal Carotid artery enters the Carotid canal in the petrous temporal bone. Within the temporal bone the internal Carotid artery first makes an anteromedial right angle bend, then bends again as it courses cephalad to enter the cavernous sinus. The intrapetrous internal Carotid artery has tympanic branches that supply the middle ear[5].

CAVERNOUS SEGMENT

The cavernous segment begins where the internal Carotid artery exits from the Carotid canal at the petrous apex and terminates at its entrance into the intracranial subarachnoid space adjacent to the anterior clinoid process.

The cavernous internal Carotid artery has several small but important branches

1. The meningohypophyseal artery
2. The superior hypophyseal trunk
3. The inferolateral trunk
4. The ophthalmic artery
5. The posterior communicating artery
6. The anterior choroidal artery
7. Two terminal branches
    a. Anterior cerebral artery
    b. Middle cerebral artery

2. VERTEBRAL ARTERIES

The vertebral arteries usually originate from their respective subclavian arteries. The left vertebral artery is usually the dominant vessel (50 -60% of cases). The vertebral arteries course cephalad to enter the transverse foramina typically at C6, and then ascend directly to C2 where they turn laterally, then superiorly through the C1 vertebral foramina. After looping posteriorly along

the atlas each vertebral artery passes superomedially through the foraman magnum within the posterior fossa usually anterior to the medulla. The vertebral arteries then unite to form the basilar artery[5].

BRANCHES

From vertebral artery[5]

1. Posterior spinal artery
2. Anterior spinal artery
3. Posterior inferior cerebellar artery
4. Medullary branches

From Basilar artery[5]

1. Anterior inferior cerebellar artery
2. Labyrinthine artery
3. Superior cerebellar artery
4. Pontine branches
5. Two terminal posterior cerebral arteries

DOPPLER ULTRASOUND OF THE CAROTID ARTERIES

Doppler ultrasound techniques are based on the Doppler equation which was described by Christian Johann Doppler in 1842. The underlying principle is that when sound or light waves are moving between a transmitter and a receiver which are stationary in relation to each other then the receiver will register the same frequency as the transmitter emitted. If there is relative

movement towards each other then the receiver will register a slightly higher frequency (shorter wavelength) than was transmitted; conversely if there is relative motion apart, then the receiver will register a slightly lower frequency (longer wavelength). These small changes are known as Doppler shifts and can easily be measured by modern ultrasound equipment through direct comparison of the returning frequency with the transmitted frequency[6].

The derivation of the Doppler equation used in medical ultrasound is

$$Fd= Ft- Fr= 2FtvcosQ/c$$

Fd- is the frequency or Doppler shift, Ft is the transmitted frequency, Fr is the received frequency. v is the velocity of the reflector (usually blood in the vessels). Q is the angle between the direction of flow of blood and c is the mean velocity of sound in tissues, 1540m/s. using modern ultrasound equipment the only variable which is unknown is the velocity of the reflecting blood cells[6].

The basic information obtained with Doppler is quite limited. It shows

1. If there is moving blood present, which way it is going and how fast it is moving.
2. Some information on the character of flow can be deduced such as presence of turbulent flow and decreased diastolic flow.
3. Doppler shifts are given in units of frequency-kilohertz.

4. The velocity of flow in centimeters or meters per second can only be calculated and displayed if an angle correction is applied using a cursor on the image of the vessel.

TYPES OF DOPPLER EQUIPMENT

CONTINUOUS – WAVE DOPPLER (CW DOPPLER)

This is the simplest type of equipment. The probe contains two transducer crystals one transmits continuously and the other receives continuously. The Doppler shift is calculated and displayed as an audio signal. This type of equipment is used in vascular clinics to locate arterial pulses, measure perfusion pressure, test for venous reflux, etc[6].

DUPLEX DOPPLER

These machines combine real time imaging with pulsed Doppler. This allows the operator to identify a specific segment in a particular vessel and to place the gate, or sample volume, at a specific location so that the source of the Doppler signal is known. In addition to transmitting the Doppler information as an audio signal, it can also be displayed as a spectral trace, or wave form scrolling across the screen[6].

Vessels have different wave forms or Doppler signatures which depend primarily on the size of the vessel and the type of capillary bed they are supplying

Internal Carotid artery supplies the relatively low resistance cerebral circulation and therefore has high diastolic flow in comparison to the external Carotid artery which supplies the higher resistance circulation of the scalp and face resulting in significantly lower diastolic flow.

The waveform characteristics can change significantly in response to physiological stimuli as shown by the increased diastolic flow that is seen in femoral arteries on exercising the leg muscles.

COLOUR DOPPLER

In colour Doppler systems the pulses along each scan line are divided on return to the transducer, some are used to provide imaging information and the rest are used to calculate the mean Doppler shift within small pixels of the image. The mean shift information is then coded on a colour scale and displayed as a colour map over the grayscale image. The choice of colours is arbitrary: usually shades of blue and red are used to represent flow towards and away from the transducer, with paler shades of the colour representing higher velocities[6].

POWER DOPPLER

Small volumes of blood moving slowly produce a weak signal, which is difficult to define from background noise. One way to improve this situation is to integrate the energy from all the shift information in both directions together, thus increasing the overall power of the Doppler information and sensitivity of the system, but at the expense of losing directional and velocity information.

Power Doppler techniques are therefore good for showing areas of flowing blood, particularly when it is moving slowly, or in small vessels. Because of its higher sensitivity, it is more prone to movement artifact from respiratory motion, or bowel activity. Some manufacturers provide Doppler imaging using a combination of power Doppler for sensitivity associated with colour Doppler for directional information[6].

THE NORMAL CAROTID ARTERIES

The longitudinal view of the normal Carotid wall demonstrates two nearly parallel echogenic lines; the inner line is the lumen-intima interface and the outer line is the media-adventitia interface. The distance between these lines is the combined thickness of intima and media (I-M complex). Measurement of the intima medial thickness is feasible with today's high resolution ultrasound machines. Normally this is less than 0.8mm or .08cm[7]. Thickening of I-M complex more than 0.8 mm represents early changes of atherosclerosis. The intimal reflection should be straight, thin and parallel to the adventitial layer.

Undulations and thickening indicate plaque deposition or more rarely fibromuscular hyperplasia.The common Carotid lies immediately adjacent to the jugular vein but the two vessels are easily differentiated. The Carotid artery exhibits pulsatile flow pattern whereas the jugular vein shows continuous low velocity signal.

The external Carotid artery can be distinguished from the internal Carotid artery by four features[6].

1. It is more anterior than the internal Carotid artery.
2. It has visible branches. The internal Carotid artery has no branches in the neck.
3. It has less diastolic flow than the internal Carotid artery.
4. Tapping the superficial temporal artery as it passes over the zygoma induces fluctuations in the waveform of external Carotid artery but not the internal Carotid artery.

FLOW CHARACTERISTICS ON COLOUR DOPPLER IMAGE

Laminar flow is apparent in normal common Carotid artery and Internal Carotid artery as manifested by gradations of shades of colour from the periphery to the center of the vessel. This can be appreciated on longitudinal as well as transverse images. A tortuous vessel or bifurcation of the vessel may produce flow disturbances that vary in severity in proportion to the curvature or angular measurements of the vessel. Flow disturbance may be manifested by mixtures of shades of colour, all flowing cephalad or mixtures of colours representing forward or reversed flow[7].

Normal flow disturbances occur at the Carotid bulb. Aspects of Carotid pulsality that assist with the identification of external Carotid artery and

internal Carotid artery are also manifested by the Doppler image. The common Carotid artery and internal Carotid artery exhibit a continuous flow pattern with antegrade flow in diastole indicated by persistence of colour throughout the entire cardiac cycle. External Carotid artery shows cessation or marked diminution of diastolic flow and this is indicated by the disappearance of colour during the diastolic portion of the cardiac cycle[7].

The internal Carotid artery and external Carotid artery have distinct spectral waveforms. The external Carotid artery shows a sharp velocity rise during systole and a rapid fall during diastole, approaching zero or transient reverse direction. This flow pattern is due to the high resistance vascular bed of the facial musculature supplied by the external Carotid artery. The internal Carotid artery supplies the low resistance circulation of the brain. Thus it shows large quantity of forward flow in diastole. Percussion of the superficial temporal artery (temporal artery tap) often results in a serrate distortion of the Doppler waveform in the external Carotid artery. The common Carotid artery waveform is a composite of the internal Carotid artery and external Carotid artery waveforms but the common Carotid artery more often closely resembles the internal Carotid artery flow pattern and diastole is generally above the base line[7].

PLAQUE CHARECTERISATION

Plaque morphology may be apparent in the Carotid and different types of plaques may be identified. On gray scale imaging plaque is seen as

echogenic material that encroaches on the arterial lumen and produces a flow void. Plaque echogenicity depends on its composition

LOW ECHOGENICITY PLAQUE

These are fibro fatty plaques containing large amount of lipid material. These may be difficult to identify on gray scale imaging due to their faint echogenicity. The problem is lessened with colour Doppler imaging since a flow void is visible even if the plaque is not.

MODERATELY ECHOGENIC PLAQUE

These are fibrous plaques in which collagen is a prominent component.

STRONGLY ECHOGENIC PLAQUE

These plaques show posterior acoustic shadowing secondary to calcifications in the areas of hemorrhage and necrosis. Acoustic shadowing from the plaque may obscure the arterial lumen and wall opposite the plaque and thus may prevent acquisition of Doppler information[7].

Plaque texture is classified as being homogeneous or heterogeneous. Homogeneous plaque has a uniform echo pattern and smooth surface. Heterogeneous plaque has a more complex echo pattern and contains at least one or more focal sonolucent areas representing intraplaque hemorrhage. Sonographic findings suggestive of plaque ulceration include a focal depression or break in the plaque surface or anechoic area within the plaque,

which extends to the plaque surface without an intervening echo between the vessel lumen and the anechoic region. Colour flow Doppler and power Doppler ultrasound may demonstrate slow moving eddies of colour within an anechoic region in the plaque, which suggest ulceration; these findings are highly accurate in predicting plaque ulceration. Pulsed wave Doppler traces from within the ulcer crater show low velocity dampened waveforms. The cephalo-caudal extent of plaque is reliably visualized with longitudinal images. The thickness of plaque, as well as severity of luminal narrowing should be measured from transverse sections. In addition it is useful to describe plaque as circumferential or non circumferential[7].

COLOUR DUPLEX EVALUATION OF CAROTID STENOSIS:

The detection of Carotid stenosis and occlusions with colour Duplex sonography relies mainly on the combination of B mode and colour encoded flow imaging. The B mode image defines the outer boundary of the vessel wall and the lumen reducing material while the colour image demonstrates the flow pattern. Doppler frequency analysis serves mainly to confirm the imaging findings and may be necessary for quantification.

The severity of the Carotid stenosis may be evaluated by measuring the diameter or area of residual lumen and diameter or area of the original lumen. Thus the percentage of luminal reduction can be calculated. The accurate measurement of the Carotid Stenosis is dependent on good quality images and on the attainment of the true cross section of the vessel. Cross sectional images

of diagnostic quality cannot always be obtained if there are tortuous vessels and calcified plaques. In such cases severity of stenosis must be estimated from Doppler spectral information[8-11].

Colour Doppler ultrasound facilitates Doppler spectral analysis by rapidly identifying areas of flow disturbances. The highest velocity shifts can frequently be identified by colour flow Doppler aliasing. Colour Doppler ultrasound facilitates this by placing the pulsed wave Doppler sample volume in the region of the most striking colour abnormalities.

Power Doppler ultrasound showing better edge definition and relative angle dependent flow imaging offers the potential for better visual assessment of degree of stenosis[8-11.]

DETERMINING DEGREE OF STENOSIS

Carotid stenosis usually begins to cause velocity changes when the stenosis exceeds 50% diameter reduction (reduction of 70% cross sectional area)[12]. Flow velocity increases as severity of stenosis increases. Velocity increases are focal and more pronounced immediately distal to a stenosis. The point of maximum velocity can be easily determined on a colour Doppler image.

Commonly used methods of acoustic estimation of the degree of stenosis include the following:[13]

1. Measurement of peak systolic velocities and end diastolic velocities

2. Measurement of ratios (e.g., internal Carotid artery peak systolic velocity /Common Carotid peak systolic velocity)
3. Some laboratories characterize degree of stenosis in terms of exact percentages. A range (e.g. 50-69% stenosis) is probably more accurate. The ranges and measurements vary from laboratory to laboratory. Factors that affect measurements include the equipment used, the person performing the ultrasound, and the sites sampled for measurement (e.g. the distal internal Carotid artery often has higher velocities than the proximal internal Carotid artery). When possible, laboratories should perform their own correlations with angiographic measurements for quality control.

A consensus conference in 2003 of the Society of Radiologists in Ultrasound recommended some criteria for estimating stenosis which is used in this study.[13]

VERTEBRAL ARTERIES

The vertebral Duplex scan can provide useful information in selected circumstances. The vessels can be imaged to only to a limited extent because of the anatomy and the insonation window available. The flow pattern can be altered by nonspecific kinking of the vessel and the tortuosity[14]. There is often asymmetry in the diameter of the two vessels in which case the left is usually larger. The main items of information that can be gathered on these vessels include the fact that both are present, the direction of flow in them, and whether

the flow is normal or damped, occasionally a stenosis in the artery may be demonstrated. A stenosis, or absent segment, in one vessel is not usually of clinical significance as the basilar circulation can be maintained from the other artery. If reversed flow is demonstrated, it is a sign of an occluded, or severely stenotic subclavian artery. In some patients, exercise of the ipsilateral arm muscles may be required to produce reversed flow[6].

ARTIFACTS IN ULTRASOUND

At least 18 artifacts have been identified in Carotid ultrasound; most of them occur during imaging[13].

The common artifacts include the following:

- Reverberations: If 2 or more reflectors are in the sound path, multiple reflections (i.e. reverberations) occur and may result in unreal images on the screen.
- Refraction: A refracting object "bends" the ultrasonic waves so that a reflector is improperly positioned on the screen.
- Shadowing: A strong reflector (e.g. calcified plaque) reduces the quantity of ultrasound that is intended to pass beyond it; the object behind the reflector is "shadowed"; in the case of a plaque, the artifact proves to be helpful in identifying it.
- Enhancing: Enhancing is the opposite of shadowing (e.g. gallbladder).

- Aliasing: This occurs during spectral analysis and colour flow imaging. During spectral analysis, the spectrum is "wrapped" around the screen so that the top of the waveform is seen at the bottom. This problem can be corrected by increasing the pulse repetition frequency (PRF) (aliasing occurs when the highest Doppler shift is greater than half the PRF), increasing the Doppler angle, shifting the baseline, lowering the emitted frequency, or using a continuous wave device.

OTHER NONINVASIVE INVESTIGATIONS.

Transcranial Doppler Ultrasound (TCD)

Since Carotid Doppler can only look at the Carotid artery in the neck, before the artery enters the brain, another ultrasound technique has been developed to look at the blood vessels in the brain. A TCD probe is placed either over the eye, at the back of the neck, or on the temple to look at various arteries. The TCD measures the speed of blood flow which can help determine the status of the underlying vessels. It takes 30-60 minutes to complete and is painless.

Magnetic Resonance Angiography (MRA)

During an MRI, the scanner can also obtain pictures of the blood vessels. Blood is always moving inside an open blood vessel and an MRI can detect the movement and produce a picture of a blood vessel.

The MRA can assess arteries of the brain from where they start in the neck (i.e. the Carotid origin), all the way to up and into the brain. MRA technology is improving but it is still not the best way to look at the arteries. However, its advantage is that it can be done at the same time as an MRI scan, adding about 10 minutes to the scan time.

CT Angiography (CTA)

Similar to an MRI, a CT scanner can obtain an angiogram as well. However, an injection is required for a CTA so that the injected material (contrast dye) flowing through the brain arteries can be scanned, thereby producing a picture of the blood vessel. A CTA is somewhat more limited than an MRA because a CT scanner can only look at a portion of the blood vessel. A CTA adds about 15 minutes to a CT scan and there is minor discomfort during the dye injection.

Digital Subtraction Angiography (DSA)

This is the best way to look at the brain's blood vessels. To obtain a DSA, a patient lies on a bed and a tube (catheter) is placed in a groin blood vessel after freezing the groin with local anesthetic. The catheter is then moved

upwards, into the various arteries of the neck which go up to the brain. Liquid (contrast dye) is then injected into the arteries and X-ray pictures are taken. The entire procedure takes 1 hour and is done with the patient awake. Since a catheter is put into an artery in the groin, the patient needs to lie flat on their back afterwards for 6 hours while the groin is healing. There is a small risk of stroke during DSA (1%) so that although DSA is very safe, doctors only obtain DSA when necessary.

MATERIALS AND METHODS

The study was conducted in Department of Medicine, Government Mohan Kumaramangalam medical college, Salem in the year 2005-2006.

INCLUSION CRITERIA

Patients who suffered ischemic stroke in the anterior circulation of the brain as confirmed by CT scan brain.

EXCLUSION CRITERIA

1. Patients who suffered stroke due to intracerebral hemorrhage.
2. Patients who suffered stroke due to head injury.

METHODS

In this study patients who are admitted with history of sudden onset of neurological illness are subjected to CT scan brain. Among them patients who had suffered ischemic stroke in the anterior circulation are selected and they were further evaluated in the following ways.

1. Detailed history taking for the evaluation of risk factors such as hypertension, diabetes mellitus, smoking, previous TIA, previous stroke and coronary artery disease.

2. Complete physical examination to know the type of cerebrovascular accident (for right / left hemiplegia) and Carotid artery examination for the presence of bruit.
3. Laboratory investigation which include blood sugar, serum total cholesterol, ECG and Carotid Doppler Ultrasonography

The details of proforma in Annexure 1.

METHOD OF EXAMINATION OF CAROTID ARTERIES

Carotid artery examination is performed with the patient in supine position, with the neck slightly extended and the head turned away from the side being examined. A 5 MHz transducer is used.

Gray scale examination begins in the transverse projection. The transducer is applied either from the anteromedial or lateral side of the sternocledomastoid muscle. Scans are obtained along the entire course of the cervical Carotid artery from the supraclavicular notch cephalad to the angle of mandibule. Origin of the common Carotid artery is identified by the inferior angulation of the transducer. The Carotid bulb is identified by looking for mild widening if the common Carotid artery at its bifurcation. Any anomalies in the bifurcation and the tortuosity are identified**.** The longitudinal view of the normal Carotid arterial wall demonstrated two parallel echogenic lines. Inner line is lumen-intima interface. The outer line is media-adventitia interface. The distance between these lines is measured and the thickness is called Intimal-

medial thickness. The artery is then searched for the presence of plaques and the plaques are classified into four types based on its echogenicity.[6]

Type1 - Plaques have a thin rim over the surface but are predominantly anechoeic.

Type2 - Plaques have less than 25%echogenic components.

Type3 - Plaques have less than 25%hypoechoic components

Type4 - Plaques are predominantly echogenic.

The examination plane necessary for optimal longitudinal scans of the Carotid artery to perform Doppler spectral analysis is determined by the course of the vessels demonstrated on the transverse study. Images are obtained to display the relationship of both branches of the Carotid bifurcation to the visualized plaque disease and the extent of the plaque is measured.

The percentage of stenosis is assessed by three methods

1. North American symptomatic Carotid endarterectomy trial method

1- N/D x100

N- Narrowest portion

D- distal normal internal Carotid artery

2. European Carotid surgery trial method

1-N/E x100

N- narrowest portion

E- estimate of unseen Carotid artery bulb wall

3. common Carotid artery method

1-N/C 100

C- Common Carotid artery

Commonly used methods of acoustic estimation of the degree of stenosis include the following:

- Measurement of peak systolic velocities and end diastolic velocities
- Measurement of ratios (e.g. Internal Carotid artery peak systolic velocity /Common Carotid artery peak systolic velocity)

A consensus conference in 2003 of the Society of Radiologists in Ultrasound recommended the following criteria for estimating stenosis:[13]

The same criteria is used in our study.

As per the criteria it is classified as

- Normal: Internal Carotid artery peak systolic velocity <125 cm/s and no plaque or intimal thickening is visible.
- <50% stenosis: Internal Carotid artery peak systolic velocity <125 cm/s and plaque or intimal thickening is visible.

- 50-69% stenosis: Internal Carotid artery peak systolic velocity is 125-230 cm/s and plaque is visible.
- >70% stenosis to near occlusion: Internal Carotid artery peak systolic velocity >230 cm/s and visible plaque and lumen narrowing are seen
- Near occlusion: A markedly narrowed lumen is seen on colour Doppler ultrasound.
- Total occlusion: No detectable patent lumen is seen on grayscale ultrasound, and no flow is seen on spectral, power, and colour Doppler ultrasound.

RESULTS

The study included 50 patients

The age distribution of the patients is shown in Table 1

TABLE-1

Age group	Number of patients
Less than 40	3
41-50	11
51-60	22
61-70	11
71-80	3

The sex distribution of the patients is shown in Table 2.

TABLE - 2

Age	Male	Female
Less than 40	3	0
41-50	7	4
51-60	12	10
61-70	6	5
71-80	3	0
Total	31	19

The various risk factors which contributed to stroke are shown in table-3

TABLE-3

Risk factor	Male	Female
Hypertension	18 (58%)	13 (58%)
Diabetes mellitus	13 (41%)	17 (89%)
Smoking	26 (83%)	---
Dyslipedemia	20(64%)	13(68%)

The relationship between serum cholesterol level and stroke is shown in Table-4

TABLE-4

Total number	Raised total cholesterol	Normal total cholesterol
50	33(66%)	17(34%)

The number of risk factors and its association with stroke is shown in Table-5

TABLE – 5

Number of risk factors	**Number of patients**
1	8
2	13
3	20
4	6

The presence and absence of Carotid bruit is shown in TABLE-6

TABLE-6

Total number of patients	Bruit present	Bruit absent
50	5(10%)	45 (90%)

The various types of plaques seen are given in this Table 7.

TABLE-7

Type of plaques	Number of patients
No plaques with normal IM thickness	3 (6%)
Increased IM thickness (No plaques)	21(42%)
Type 1	12(24%)
Type 2	4(8%)
Type 3	9(18%)
Type 4	1(2%)

The percentage of stenosis detected by Doppler spectral analysis are shown in table 8.

TABLE – 8

Percentage of Stenosis	Number of Patients
More than 70%	1 (2%)
50 – 70%	10 (20%)
Less than 50%	36 (72%)
Normal	3 (6%)

The number of patients showing changes in contralateral Carotid artery are shown in table 9

TABLE-9

Total number of patients	Showing changes in the contralateral carotid artery	Showing no changes in the contralateral side
50	22(44%)	28(56%)

The number of patients showing changes in ECG are shown in table-10

TABLE - 10

Total Number of patients	Showing Abnormality in ECG	Showing No abnormality
50	22(44%)	28(56%)

Chart - 1
The Age Distribution of the Patients

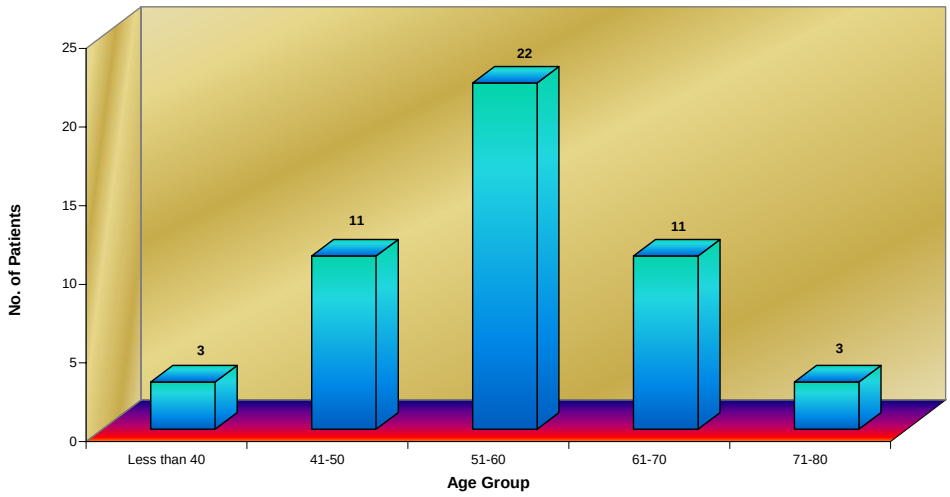


Chart - 2
The sex distribution of the patients

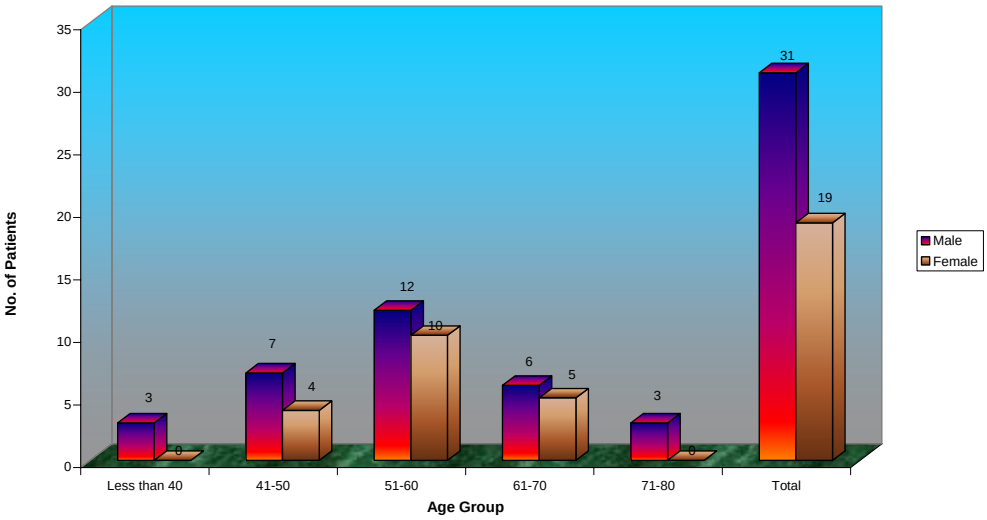


Chart - 3
The various risk factors which contributed to stroke

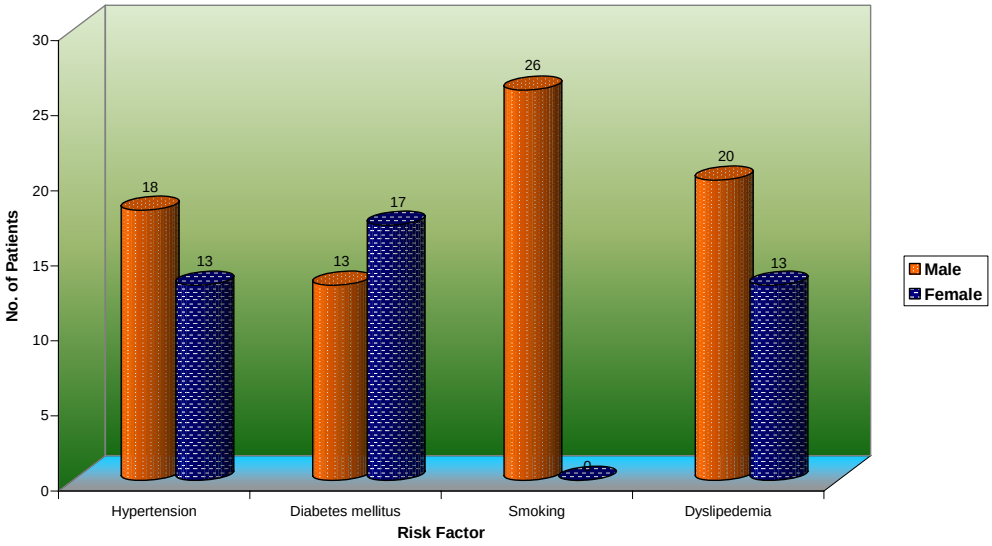


Chart - 4
The number of risk factors and its association with stroke

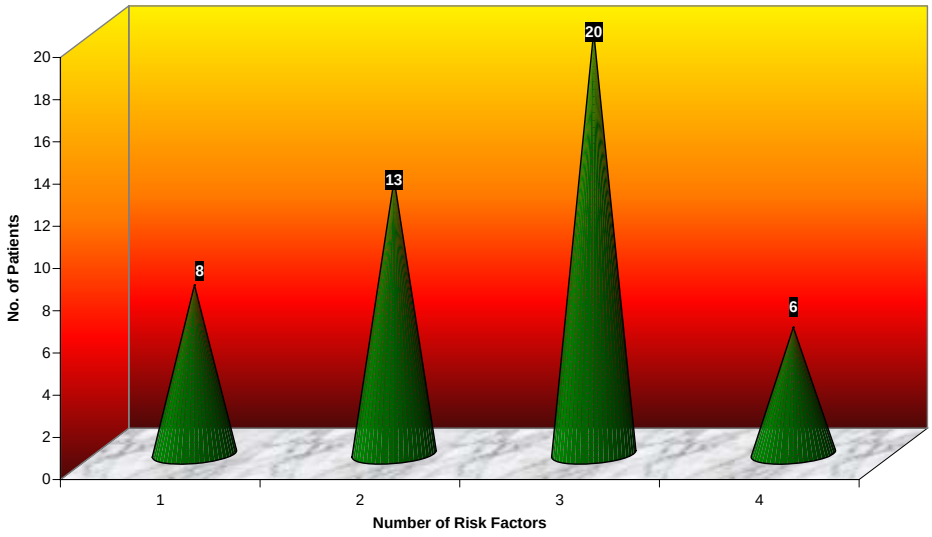
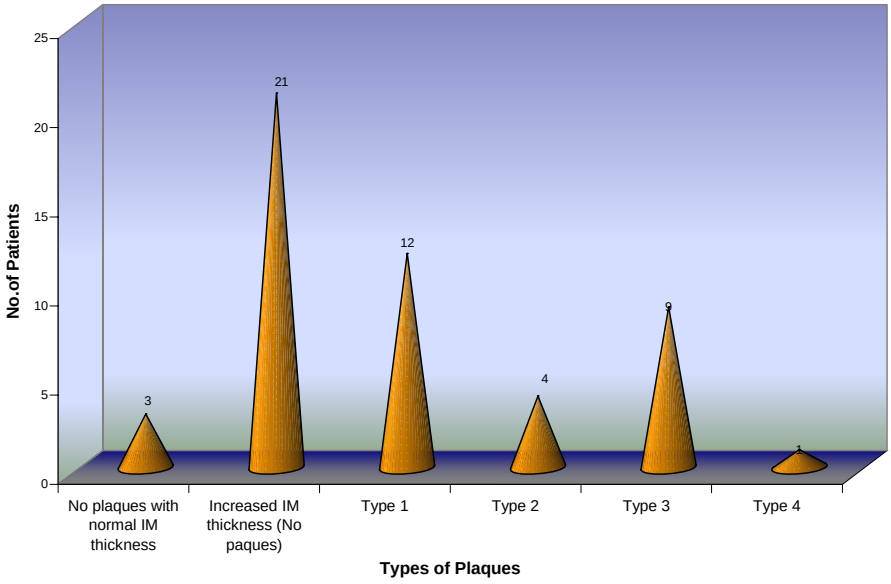


Chart - 5
Various types of Plaques



DISCUSSION

This study retrospectively assessed the changes in the Carotid arteries by means of Doppler ultrasound in patients who had suffered stroke. In this study 50 patients were included of which 31 were males and 19 were females. The study group is selected among the patients who were admitted with history of sudden onset of weakness of right or left half of the body. The selected patients were subjected to CT scan brain. Among them patients who were having infarct in anterior circulation of the brain, were further evaluated for the identification of risk factors which included hypertension, diabetes, smoking and hypercholesterolemia. The patients with hemorrhagic stroke and those patients with definite history of head injury were excluded. The patients were then subjected to Carotid Doppler Ultrasonography both on the ipsilateral and also on the contra lateral side of the lesion shown in CT scan brain. As per the age and sex of the patients as shown in **table-1 & table-2.** 44% of them were between 51-60 years with almost equal number of males and females. 72% of them were above 50 years and 28% were below 50 years. Most of the patients below 50 years are males. Hence this study shows that male sex and increased age are the important risk factors for the development of stroke. This has been observed in other studies also[3]. As per the other risk factors are concerned as shown in **table-3** hypertension was present equally in 58% of the males and females studied. Hence this study confirmed that hypertension is one of the major risk factor for the development of stroke and control of hypertension will reduce the risk of developing stroke[15]. Numerous randomized placebo-

controlled trials have also demonstrated that lowering blood pressure in patients with hypertension prevents both hemorrhagic and ischemic strokes (relative risk [RR] reduction, 35 to 45%[16-19]. The stroke prevention benefits of antihypertensive drug therapy are continuous across the usual range of blood pressures, and the relative benefits for each mm Hg reduction in blood pressure are similar regardless of the baseline systolic blood pressure (i.e., whether the systolic blood pressure is 170 mm Hg or 150 mm Hg).Thus, there does not appear to be a J curve in antihypertensive drug efficacy[20].

In this study diabetes as a risk factor was present in 89% of females and 41% of males. Hence this study observes that patients with diabetes are at increased risk for all forms of ischemic stroke and also are more likely to have hypertension and hyperlipidemia. However, no high-quality evidence supports reduction of stroke risk through improved glucose control[21-23]. The three major randomized trials that have tested the glucose-control hypothesis demonstrated no significant reductions in the risk of ischemic stroke or any other macro vascular outcome[24-25]. Nonetheless, several guidelines recommend tight glucose control to reduce the development or progression of micro vascular complications in patients with type 1 or type 2 diabetes.

Because hypertension, hyperlipidemia, and type 2 diabetes (or at least glucose intolerance) frequently coexist, which is also evident in this study as shown in **table-5** in which 50% of them are having more than one risk factor it is important to screen patients with any one of these risk factors for the other

factors and to institute aggressive risk-factor modification for all three conditions to prevent a wide variety of atherosclerotic events. In particular, aggressive blood pressure reduction (to a target of less than 130/80 mm Hg) is important in patients with diabetes.[26]

In this study smoking as a risk factor was present in 83% of males and none of the female patient in the study were smokers[27-29].

While obesity, lack of regular aerobic exercise, excessive alcohol intake, and smoking all increase the risk of stroke, no high-quality randomized trials have evaluated the effects that modifications of these factors have on stroke risk. However, given the strength of observational data and the overall health benefits of weight loss, alcohol restriction, regular aerobic physical activity, and smoking cessation, these lifestyle modifications should be discussed and encouraged[30-31].

In this study hypercholesterolemia was present in 64% of males and 68% of the females as shown in **table-4**. Information from other studies also suggests that higher total and low-density lipoprotein (LDL) cholesterol levels are associated with an increased risk of ischemic stroke[32-35]. While most individual trials of lipid-lowering therapies (e.g., resins, fibrates, dietary measures) have not shown a decreased risk of stroke, a meta-analysis of 11 trials found that treatment with statins (3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors) is associated with a 25 percent reduction (95 percent CI, 14 to 35 percent) in the risk of fatal and nonfatal stroke.[36] It has also been

observed in our study that 34% of the patients having normal cholesterol level even though they are having evidence of atherosclerosis as shown by Carotid Doppler Ultrasonography as increased intimal medial thickness. Hence it is not clear whether reduction of cholesterol level has any effect on the cervical or intracranial atherothrombotic process or on stroke risk. However it requires further studies.

It has been observed in this study as shown in **table-5** that the auscultation of Carotid arteries shows bruit in only 10% of patients this may be because most of them are having stenosis of less than 50%.It has been shown in other studies that neck bruits do not reliably predict presence or absence of underlying occlusive Carotid disease[37]. Cervical bruits may be due to other causes such as transmitted cardiac murmurs, anatomic variations, tortuosity, and hyper dynamic states. Studies looking at the relationship between Carotid bruit and corresponding stenosis have used different methodologies which limits comparability (populations with different prevalence of vascular disease, interobserver variability among clinicians about auditory characteristics of the bruit, different methods of imaging and different definition of Carotid lesion severity). Depending on the method of assessment, the predictive value of a Carotid bruit for ipsilateral moderate to severe stenosis ranges from approximately 16% to 75%. Patients with asymptomatic bruits are less likely to have an underlying stenosis than patients with symptomatic bruits. According to some, among patients with asymptomatic neck bruits, 17% had a >75% stenosis, while in patients with both Carotid stroke and bruits, 60% had

a>75% stenosis. Conversely, many patients with a high grade stenosis do not have a cervical bruit[37]. Hence it is said that Carotid bruit do not reliably predict or exclude carotid occulusive disease.

Regarding the types of plaques and its morphology detected by Carotid Doppler Ultrasonography in our patients as shown in **table-7**, 3 of them had normal intimamedia thickness and without any plaques. 21 of them showed increased carotid intima-media thickness without any visible plaque. 12 of them showed type 1 plaque. 4 of them showed type 2. 9 of them showed type 3 and one of them showed type 4. Hence it is observed that 47 of our patients showed changes in the carotid arteries due to atherosclerosis which include increased carotid intima-media thickness and various types of plaques. Out of them 21 patients [42%] showed only increased intima-media thickness without any plaques. Hence increased intima-media thickness alone without any visible plaques can be taken as one of the risk factor for developing storke. This has been proved in other studies[38-40]. In one study conducted by S.K. Mukherjee, A.K. Basu from Department of Medicine, Kolkatta Medical College, also it is observed that carotid intima-media thickness is a good predictor of ischemic stroke.

Eventhough there are various methods to detect the degree of stenosis such as NASCET [North American Symptomatic Carotid Endarterectomy Trial] method. ECST (Euorpean Carotid Surgery Trial] method in our study degree of stenosis is measured as per the recommendation arrived in consensus

conference of the radiologist in 2003. Based on the recommendation peak systolic velocity and end diastolic velocity is measured in internal carotid artery and peak systolic velocity in measured in common carotid artery and the ratio is measured. It has also been proved in other studies that measured peak systolic velocity, end diastolic velocity and carotid index. Correlate very well with measuring the residual lumen diameter.[41-42] Based on that our study shows as in **table-8** that no stenosis is present in 3 of the patients, 36 patients showed less than 50% stenosis, 10 patients showed 50 to 70% stenosis and one patient showed more than 70% stenosis. As per the NASCET & ECST trial surgery is indicated for persons showing more than 70% stenosis.[43-45] However most of our patients who had suffered stroke showed only 50-69% of stenosis and less than 50% stenosis. Hence it is observed that even patient with lesser degree of stenosis are also having increased risk of stroke. Hence they should be given aggressive medical therapy for their risk factors beside surgery for symptomatic individual with stenosis of 50 – 69%.

It has also been observed in our study as per **table-9** 44% of the study group showed evidence of atherosclerosis in the contralateral carotid artery also. Hence it is observed that more than the stenosis the study of physical and mechanical properties of vessel wall and plaque characteristics by ultrasound tissue Doppler imaging is of more value in detecting the risk of developing stroke. This has proved in various studies.[46-47] In one pilot study conducted by Kumar V. Ramnarine, Tim Harristone & Others. Published in Cardio Vascular Ultrasound in the year 2003. Arterial wall motion were successfully extracted

in 91% of cases within the carotid bifurcation and plaque region with help of tissue Doppler ultrasound. According to that study the Arterial wall motion showed a wide variation and had poor correlation with severity of stenosis. Hence it is suggested that arterial wall motion abnormality detection will give more value in detecting the chance of plaque rupture and status of arterial wall which many predict the risk of stroke. However it requires further studies.

In our study we also correlate the findings in the ECG and the findings in the carotid Doppler and 44% of our patients as show in table-10 showed both intima-media thickness and changes in ECG consistent with coronary artery disease. Hence carotid intima-media thickness can be taken as an independent predictor of both coronary artery as well as cerebrovascular disease. This has been proved in various studies[48-50] like the one conducted by Kusum D Jashnani, Rohit R Kulkarni, Jaya R Deshpande, Department of Pathology, TN Medical College and BYL Nair Ch Hospital, Mumbai which is an autopsy study where the carotid intima-media thickness and coronary atherosclerosis were compared and it found very good correlation between the two.

However it requires further studies to substantiate the value of carotid Doppler in predicting the risk of developing stroke.

CONCLUSION

1. Doppler Ultrasonography of Carotid arteries shows significant changes due to atherosclerosis in the patients who suffered stroke.
2. Abnormalities in the vessel wall is more pronounced than the flow disturbances due to stenosis.
3. Since most of the patients showed changes in the contra lateral sides and ECG changes suggestive of ischemic heart diseases it is taken as an evidence of generalized vascular disease due to atherosclerosis.
4. Arterial wall motion abnormalities and plaque characteristics by tissue Doppler imaging is more useful in predicting the risk of stroke than detecting flow pattern abnormalities.
5. Carotid Doppler Ultrasonography cannot substitute for angiography as the sole preoperative tests for Carotid endarterectomy.
6. However it can be used as a screening test for the detection of the asymptomatic Carotid disease in patients with risk factors for stroke.

BIBLIOGRAPHY

1. P.M. Dalal, Cerebrovascular disorders, API text book of medicine, 7th edition page 796.
2. Mackey A, Cote R, Battista RN. Asymptomatic Carotid disease. In: Canadian Task Force on the Periodic Health Examination. Canadian Guide to Clinical Preventive Health Care. Ottawa: Health Canada, 1994; 692-704.
3. Wade S. smith, S. Claiborne Johnston, J. Donald Easton; Carotid atherosclerosis, cerebrovascular diseases Harrison's Principles of Internal Medicine 16th edition chapter 349, page 2377.

   Peter Libby, The Pathogensis of Ataerosclerosis Harrison's Principles of Internal Medicine, 16th Edition, Volume II, Page 1425-29.
4. R.J. Sellar, Imaging blood vessels of the head and neck; Neurological investigations; Journal of neurology, neurosurgery and psychiatry BMJ 1997
5. Osborn Anne G Cerebral vasculature; Normal vascular anatomy and pathology chapter 6 & 11 Diagnostic Neuroradiology page (117-123) & (330-332).
6. David Sutton and Roger Gregson; Doppler ultrasound of the arteries, Text book of radiology and imaging by David Sutton sixth edition volume1 page 719-729.
7. RajaGopal, KV Bhushan, N Lashkar, Shekar Banavali, Nitin Kumar Singh; Pictorial Essay: Colour Duplex Evaluation of Carotid Occlusive Lesions ; Indian Journal Radiology Imaging 2000; 10:
8. Erricson SJ, Mewissen MW, Foley WB, et al. Stenosis of the internal Carotid artery: assessment using colour Doppler imaging compared with angiography. AJR 1989; 152: 1299-1305.
9. Middleton WD, Foley WD,Lawson TL. Colour flow Doppler imaging of Carotid artery abnormalities. AJR 1988; 150: 419-425
10. Zwiebel WJ, Knighton R. Duplex examination of Carotid arteries. Seminars in Ultrasound, CT and MR 1990; 11: 97-135.
11. Griwing B, Driesner F, Kallwellis G et al. Cerebrovascular disease assessed by colour flow and power Doppler Ultrasonography. Stroke 1996; 27: 95-100.
12. Carrol BA. Carotid sonography. Radiology 1991; 178: 303-313.
13. Grant EG, Benson CB, Moneta GL, et al: Carotid artery stenosis: grayscale and Doppler ultrasound diagnosis--Society of Radiologists in Ultrasound consensus conference. Ultrasound Q 2003 Dec; 19(4): 190-8Medline
14. Roger E Kelley; Noninvasive cardiovascular studies, Neurology in clinical practice (principles of diagnosis and management) page 499 chapter38 second edition

15. MacMahon S, Peto R, Cutler J, Collins R, Sorlie P, Neaton J, et al. Blood pressure, stroke, and coronary heart disease. Part 1, prolonged differences in blood pressure: prospective observational studies corrected for the regression dilution bias. Lancet 1990;335:765-74.

16. Perry HM Jr, Davis BR, Price TR, Applegate WB, Fields WS, Guralnik JM, et al. Effect of treating isolated systolic hypertension on the risk of developing various types and subtypes of stroke: the Systolic Hypertension in the Elderly Program (SHEP). JAMA 2000;284:465-71.

17. Psaty BM, Smith NL, Siscovick DS, Koepsell TD, Weiss NS, Heckbert SR, et al. Health outcomes associated with antihypertensive therapies used as first-line agents. A systematic review and meta-analysis. JAMA 1997;277:739-45.

18. 18.Rodgers A, MacMahon S, Gamble G, Slattery J, Sandercock P, Warlow C. Blood pressure and risk of stroke in patients with cerebrovascular disease. The United Kingdom Transient Ischaemic Attack Collaborative Group. BMJ 1996; 313:147.

19. Wright JM, Lee CH, Chambers GK. Systematic review of antihypertensive therapies: does the evidence assist in choosing a first-line drug? CMAJ 1999; 161:25-32.

20. McAlister FA. Using evidence to resolve clinical controversies: is aggressive antihypertensive therapy harmful? Evidence-Based Med [United Kingdom] 1999;4:4-6.

21. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. The Diabetes Control and Complications Trial Research Group. N Engl J Med 1993;329:977-86.

22. Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). UK Prospective Diabetes Study (UKPDS) Group. Lancet 1998;352:837-53.

23. Meinert CL, Knatterud GL, Prout TE, Klimt CR. A study of the effects of hypoglycemic agents on vascular complications in patients with adult-onset diabetes. II. Mortality results. Diabetes 1970;19 (suppl):747-830.

24. Meltzer S, Leiter L, Daneman D, Gerstein HC, Lau D, Ludwig S, et al. 1998 clinical practice guidelines for the management of diabetes in Canada. Canadian Diabetes Association. CMAJ 1998;159(suppl 8):S1-29.

25. Woolf SH, Davidson MB, Greenfield S, Bell HS, Ganiats TG, Hagen MD, et al. Controlling blood glucose levels in patients with type 2 diabetes mellitus. An evidence-based policy statement by the American Academy of Family Physicians and American Diabetes Association. J Fam Pract 2000;49: 453-60.

26. Benson RT, Sacco RL. Stroke prevention: hypertension, diabetes, tobacco, and lipids. Neurol Clin 2000; 18:309-19.

27. Law M, Tang JL. An analysis of the effectiveness of interventions intended to help people stop smoking. Arch Intern Med 1995; 155:1933-41.

28. Rice VH, Stead LF. Nursing interventions for smoking cessation. Cochrane Database Syst Rev 2001; (3):CD001188.

29. 29.Kawachi I, Colditz GA, Stampfer MJ, Willett WC, Manson JE, Rosner B, et al. Smoking cessation and decreased risk of stroke in women. JAMA 1993; 269:232-6.

30. Goldstein LB, Adams R, Becker K, Furberg CD, Gorelick PB, Hademenos G, et al. Primary prevention of ischemic stroke: a statement for healthcare professionals from the Stroke Council of the American Heart Association. Circulation 2001;103: 163-82.

31. Gorelick PB, Sacco RL, Smith DB, Alberts M, Mustone-Alexander L, Rader D, et al. Prevention of a first stroke: a review of guidelines and a multidisciplinary consensus statement from the National Stroke Association. JAMA 1999; 281:1112-20.

32. Cholesterol, diastolic blood pressure, and stroke: 13,000 strokes in 450,000 people in 45 prospective cohorts. Prospective studies collaboration. Lancet 1995;346:1647-53.

33. Benfante R, Yano K, Hwang LJ, Curb JD, Kagan A, Ross W. Elevated serum cholesterol is a risk factor for both coronary heart disease and thromboembolic stroke in Hawaiian Japanese men. Implications of shared risk. Stroke 1994; 25:814-20.

34. Byington RP, Davis BR, Plehn JF, White HD, Baker J, Cobbe SM, et al. Reduction of stroke events with pravastatin: the Prospective Pravastatin Pooling (PPP) Project. Circulation 2001;103:387-92.

35. Iso H, Jacobs DR Jr, Wentworth D, Neaton JD, Cohen JD. Serum cholesterol levels and six-year mortality from stroke in 350,977 men screened for the multiple risk factor intervention trial. N Engl J Med 1989;320:904-10.

36. Bucher HC, Griffith LE, Guyatt GH. Effect of HMGcoA reductase inhibitors on stroke. A meta-analysis of randomized, controlled trials. Ann Intern Med 1998;128:89-95

37. 37.Mackey A, Cote R, Battista RN. Asymptomatic Carotid disease. In: Canadian Task Force on the Periodic Health Examination. Canadian Guide to clinical preventive health care. Ottawa: Health Canada, 1994; 692-704.

38. Kitamura A, Iso H – Carotid intima media thickness and plaque characteristics as a risk factor for stroke in Japanese elderly men. Stroke 2004;35;2788-94.

39. Hatsukami TS, Ferguson MA- Carotid plaque morphology and clinical events. Stroke 1997;28;95-100.

40. Rosfors S, Hallerstam S, Jensen-Urstad K, Zettering M, Carlstrom C – Relationship between intima media thickness in common Carotid artery and atherosclerosis in the Carotid bifurcation.

41. Call GK, Abbott WM, Macdonald NR, Megerman J. Davis KR, Heros RC, Kistler JP. Correlation of continuous-wave Doppler spectral flow analysis with gross pathology in Carotid stenosis. *Stroke.* 1988; 19:584-588.

42. Kistler JP, Lees RS, Miller A, Crowell RM, Roberson G. Correlation of spectral phonography and Carotid angiography with gross pathology in Carotid stenosis. *N Engl J Med.* 1981; 305:417-419.

43. North American Symptomatic Carotid Endarterectomy Trial Collaborators. Beneficial effect of Carotid endarterectomy in symptomatic patients with high-grade Carotid stenosis. *N Engl J Med.* 1991; 325:445-453.

44. European Carotid Surgery Trialists' Collaborative group. MRC European Carotid Surgery Trial: interim results for symptomatic patients with severe (70-99%) or with mild (10-29%) Carotid stenosis. *Lancet.* 1991;337:1235-1243.

45. Mayberg MR, Wilson SE, Yatsu F, Weiss DG, Louis M, Hershet LA, Colling C, Eskridge J, Deykin D, Winn HR, for the Veterans Affairs Cooperative Studies Program 309 Trialist Group. Carotid endarterectomy and prevention of cerebral ischemia in symptomatic Carotid stenosis. *JAMA.* 1991; 266:3289-3294

46. Laurent S, Katsahian S, Fassot C, Tropeano AI, Gautier I, Laloux B, Boutouyrie P: Aortic stiffness is an independent predictor of fatal stroke in essential hypertension *Stroke* 2003, 34(5)**:**1203-1206

47. Oliver JJ, Webb DJ: Noninvasive assessment of arterial stiffness and risk of atherosclerotic events; *Arteriosclerosis Thromb Vasc Biol* 2003, 23**:**554-566.

48. Jadhav UM, Kadam NN. Carotid intima-media thickness as an independent predictor of coronary artery disease. Indian Heart Journal 2001;53;458-462

49. Jadhav UM. Non invasive early prediction of atherosclerosis by Carotid intima media thickness. Asian Journal clinical cardiology 2001; 4:24-28.

50. Hodis HN, Mack WJ, LaBree L, Selzer RH, Liu CR,Liu CH< etal. The role of Carotid arterial intima-media thickness in predicting clinical coronary events. Ann Intern Med 1998: 128:262-269

PROFORMA

1. NAME

2. AGE

3. SEX

4. I.P N.O

5. DATE OF ADMISSION

6. DETAILED HISTORY REGARDING RISK FACTORS

HYPERTENSION	YES/NO
DIABETES MELLITUS	YES/NO
DYSLIPIDEMIA	YES/NO
SMOKING	YES/NO
CORONARY ARTERY DISEASE	YES/NO
PREVIOUS TIA	YES/NO
PREVIOUS STROKE	YES/NO

7. COMPLETE PHYSICAL EXAMINATION

TYPE OF CEREBROVASCULAR ACCIDENT : RIGHT/LEFT HEMIPLEGIA

PRESENCE OF CAROTID BRUIT YES/NO

BLOOD PRESSURE

INVESTIGATIONS

BLOOD SUGAR

TOTAL CHOLSTEROL

ELECTROCARDIOGRAPHY

CT BRAIN

CAROTID DOPPLER ULTRASONOGRAPHY

SONOGRAPHER OBSERVATIONS

Carotid Doppler Sonographer Observations

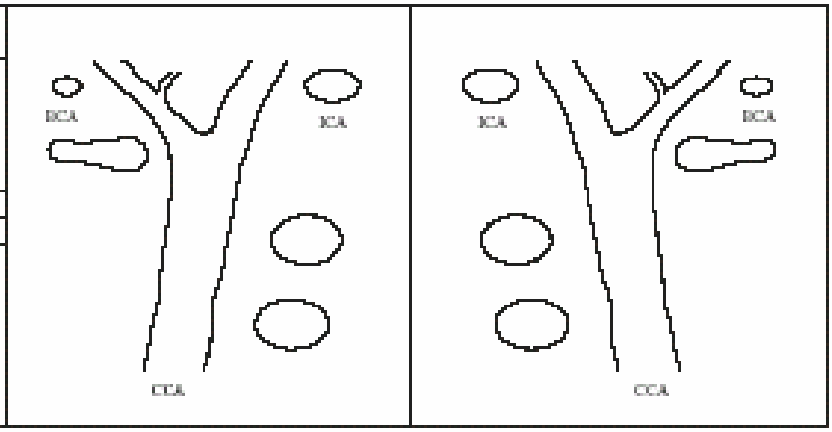
Proforma

Recast 2005 after Grant et al

NAME		
MRN		
CASE No.	Date.../.../...	DOB.../.../...

BIFURCATION		Normal		High		Low
TORTUOSITY		Minimal		Moderate		Good
TECHNICAL		Poor		Good		Excellent

Plaque Characteristics
Echogenic
Hypoechoic
Mixed
Calcified
Surface
Smooth
Irregular



VELOCITIES IN THE CAROTID SYSTEM

RIGHT		PSV (cm/s)	EDV (cm/s)
CCA	MID		
	DISTAL		
	BULB		
ECA	PROX		
	DISTAL		
ICA	PROX		
	MID		
	DISTAL		
ICA/CCA			
VERT			

LEFT		PSV (cm/s)	EDV (cm/s)
CCA	MID		
	DISTAL		
	BULB		
ECA	PROX		
	DISTAL		
ICA	PROX		
	MID		
	DISTAL		
ICA/CCA			
VERT			

GRADING OF INTERNAL CARTOID STENOSIS *(Edward GRANT et al. Radiology 2003; 226: 543-564)*

DIAMETER REDUCTION	<50%	50-69%	>70%	Near Occlusion	Occlusion
PSV	<125 cm/s	125-230 cm/s and visible plaque	>230 cm/s	>230 cm/s and visible markedly decreased lumen	No patent lumen and no flow on colour, spectral, or power Doppler
EDV				>140 cm/s	
ICA/CCA			>4.0 supportive		

SONOGRAPHER
Date.../.../...

MASTER CHART

S. No.	IP No.	Age (yrs)	Sex	Risk Factors				Bruit		CTSAN	Carotid Doppler Ultrasonographic						ECG Evidence of Ischemia
											Ipsilateral Side			Contralateral Side			
				HT	DM	Smoking	Dyslipidemia	Yes	No		IM Thickness	Plaque	Percentage of Stenosis	IM Thickness	Plaque	Percentage of Stenosis	
1	15605	63	Male	Yes	No	Yes	Yes		No	L MCA infart	0.11cm	Type1 plaque	<50%	Normal	Noplaque	No stenosis	No
2	16689	58	Male	Yes	Yes	Yes	Yes		No	L MCA infart	0.12cm	Type1 plaque	<50%	0.10cm	Type1 plaque	<50%	Yes
3	17152	63	Male	Yes	No	Yes	Yes		No	R parietal infart	Normal	No plaque	Normal	Normal	Noplaque	No stenosis	Yes
4	17659	70	Male	No	Yes	Yes	No		No	L parietal infart	0.12cm	No plaque	<50%	Normal	Noplaque	No stenosis	No
5	18291	55	Male	No	Yes	Yes	Yes		No	L parietal infart	Normal	No plaque	Normal	Normal	Noplaque	No stenosis	No
6	16601	60	Male	Yes	No	Yes	Yes		No	R parietal infart	0.12cm	Type1 plaque	<50%	0.10cm	Type1 plaque	<50%	Yes
7	18923	70	Female	Yes	Yes	No	No		No	R MCA infart	0.13cm	Type3 plaque	50-69%	0.12cm	Type1 plaque	<50%	Yes
8	21082	67	Male	Yes	Yes	Yes	Yes	Yes		R MCA infart	0.14cm	Type 3 plaque	50-69%	0.11cm	Type1 plaque	<50%	Yes
9	37785	60	Female	Yes	Yes	No	Yes		No	R parietal infart	0.11cm	Type1 plaque	<50%	Normal	Noplaque	No stenosis	No
10	22304	70	Male	Yes	Yes	Yes	Yes	Yes		Ltemporal infart	0.13cm	Type3 plaque	50-69%	0.11cm	Type1 plaque	<50%	Yes
11	22208	40	Male	No	Yes	Yes	Yes		No	L parietal infart	0.10cm	Type1 plaque	<50%	Normal	Noplaque	No stenosis	Yes
12	23084	60	Male	Yes	Yes	Yes	Yes	Yes		Lcapsular infart	0.15cm	Type3 plaque	50-69%	0.13cm	Type2 plaque	<50%	No
13	21250	80	Male	Yes	Yes	Yes	Yes		No	Rcapsular infart	.0.11cm	No plaque	<50%	Normal	Noplaque	No stenosis	Yes

S. No.	IP No.	Age (yrs)	Sex	Risk Factors				Bruit		CTSAN	Carotid Doppler Ultrasonographic						ECG Evidence of Ischemia
											Ipsilateral Side			Contralateral Side			
				HT	DM	Smoking	Dyslipidemia	Yes	No		IM Thickness	Plaque	Percentage of Stenosis	IM Thickness	Plaque	Percentage of Stenosis	
14	24313	54	Male	Yes	Yes	Yes	Yes		No	R cr infart	0.10cm	No plaque	<50%	Normal	Noplaque	No stenosis	Yes
15	24102	60	Male	No	No	Yes	No		No	L MCA infart	0.15cm	Type3 plaque	50-69%	0.12cm	Type1 plaque	<50%	No
16	25121	72	Male	No	No	Yes	No		No	L parietal infart	0.12cm	No plaque	<50%	Normal	Noplaque	No stenosis	No
17	25021	80	Male	No	No	No	No		No	Both MCA infart	0.11cm	No plaque	<50%	Normal	Noplaque	No stenosis	No
18	21221	67	Male	No	No	Yes	No		No	Ltemporal infart	0.13cm	No plaque	<50%	0.11cm	Noplaque	<50%	No
19	21241	38	Male	No	No	No	No	Yes		R MCA infart	0.14cm	Type4 plaque	>70%	0.12cm	Type2 plaque	<50%	No
20	24211	70	Male	Yes	No	Yes	Yes		No	L MCA infart	0.16cm	Type3 plaque	50-69%	0.14cm	Type2 plaque	<50%	Yes
21	21249	47	Male	Yes	No	No	No		No	R MCA infart	0.09cm	No plaque	Normal	Normal	Noplaque	No stenosis	No
22	24292	45	Male	Yes	No	Yes	Yes		No	R parietal infart	0.12cm	No plaque	<50%	Normal	Noplaque	No stenosis	Yes
23	21947	55	Male	Yes	Yes	No	Yes		No	L MCA infart	0.12cm	No plaque	<50%	Normal	Noplaque	No stenosis	Yes
24	21241	60	Female	Yes	Yes	No	Yes		No	R parietal infart	0.11cm	Type1 plaque	<50%	Normal	Noplaque	No stenosis	No
25	27329	70	Male	No	No	Yes	No		No	R MCA infart	0.13cm	Type1 plaque	<50%	0.10cm	Type1 plaque	<50%	No
26	27416	40	Male	No	No	Yes	Yes	Yes		L MCA infart	0.11cm	No plaque	<50%	Normal	Noplaque	No stenosis	No
27	24121	67	Female	Yes	No	No	No		No	L parietal infart	0.13cm	Type1 plaque	<50%	0.11cm	Type1 plaque	<50%	Yes
28	19712	60	Female	Yes	Yes	No	Yes		No	Rtemporal infart	0.15cm	Type3 plaque	50-69%	0.13cm	Type1 plaque	<50%	No

S. No.	IP No.	Age (yrs)	Sex	Risk Factors				Bruit		CTSAN	Carotid Doppler Ultrasonographic						ECG Evidence of Ischemia
											Ipsilateral Side			Contralateral Side			
				HT	DM	Smoking	Dyslipidemia	Yes	No		IM Thickness	Plaque	Percentage of Stenosis	IM Thickness	Plaque	Percentage of Stenosis	
29	25121	52	Male	Yes	Yes	No	No		No	L parietal infart	0.12cm	Type1 plaque	<50%	Normal	Noplaque	No stenosis	Yes
30	24251	42	Male	No	No	Yes	Yes		No	Rfrontoparietalinfart	Normal	No plaque	Normal	Normal	Noplaque	no stenosis	No
31	21241	50	Male	No	No	Yes	No		No	R MCA infart	0.11cm	Type3 plaque	50-69%	0.11cm	Type1 plaque	<50%	No
32	24124	50	Female	Yes	Yes	No	No		No	L MCA infart	0.15cm	Type2 plaque	<50%	0.11cm	Type1 plaque	<50%	No
33	24129	55	Female	No	Yes	No	No		No	Ltemporal infart	0.11cm	No plaque	<50%	Normal	Noplaque	No stenosis	No
34	29641	43	Female	No	Yes	No	Yes		No	Ltemporal infart	0.13cm	Type1 plaque	<50%	0.12cm	Type1 plaque	<50%	No
35	20726	50	Male	Yes	Yes	Yes	Yes	Yes		Rtemporal infart	0.14cm	Type1 plaque	<50%	0.12cm	Type1 plaque	<50%	No
36	21421	60	Female	No	Yes	No	Yes		No	Lparietal infart	0.13cm	Type1 plaque	<50%	0.10cm	Type1 plaque	<50%	Yes
37	33207	68	Female	No	Yes	No	No		No	Lparietal infart	0.12cm	No plaque	<50%	Normal	Noplaque	No stenosis	Yes
38	33434	57	Female	Yes	Yes	No	Yes		No	L MCA infart	0.12cm	Type2 plaque	<50%	0.10cm	Type1 plaque	<50%	Yes
39	31425	70	Female	No	Yes	No	Yes		No	L parietal infart	0.13cm	No plaque	<50%	Normal	Noplaque	No stenosis	Yes
40	34251	60	Female	Yes	Yes	No	Yes		No	R MCA infart	0.14cm	Type3 plaque	50-70%	0.11cm	Type1 plaque	<50%	Yes
41	34250	70	Female	Yes	Yes	No	No		No	Rparietal infart	0.12cm	Type2 plaque	<50%	Normal	Noplaque	no stenosis	No
42	34252	45	Female	Yes	Yes	No	No		No	Rparietal infart	0.11cm	No plaque	<50%	Normal	Noplaque	No stenosis	No
43	34253	60	Female	Yes	Yes	No	Yes		No	L MCA infart	0.12cm	Type1 plaque	<50%	Normal	Noplaque	No stenosis	Yes

S. No.	IP No.	Age (yrs)	Sex	Risk Factors				Bruit		CTSAN	Carotid Doppler Ultrasonographic						ECG Evidence of Ischemia
											Ipsilateral Side			Contralateral Side			
				HT	DM	Smoking	Dyslipidemia	Yes	No		IM Thickness	Plaque	Percentage of Stenosis	IM Thickness	Plaque	Percentage of Stenosis	
44	34747	60	Male	Yes	No	Yes	Yes		No	Lfrotoparietal infart	0.11cm	No plaque	Normal	Normal	Noplaque	No stenosis	Yes
45	34337	50	Male	Yes	No	Yes	Yes		No	Rparietal infart	0.14cm	Type2 plaque	<50%	0.11cm	Type1 plaque	<50%	Yes
46	38621	52	Male	Yes	Yes	Yes	Yes		No	L MCA infart	0.15cm	Type2 plaque	50-69%	0.13cm	Type2 plaque	<50%	Yes
47	24293	60	Female	Yes	No	No	Yes		No	L MCA infart	0.11cm	No plaque	Normal	Normal	Noplaque	No stenosis	No
48	24258	45	Female	Yes	Yes	No	Yes	Yes		Rparietal infart	0.13cm	Type3 plaque	<50%	0.11cm	Type1 plaque	<50%	No
49	21288	60	Male	No	No	Yes	Yes		No	Rparietal infart	0.10cm	No plaque	<50%	Normal	Noplaque	No stenosis	Yes
50	21425	58	female	No	Yes	No	Yes		No	Ltemporal infart	0.15cm	Type 2 Plaque	<50%	0.13cm	Type2 plaque	<50%	No

IP No. - Inpatient Number

HT - Hypertension

DM - Diabetes Mellitus

R - Right

L - Left

LMCA - Left Middle Cereberal Artery

RMCA - Right Middle Cereberal Artery

IM - Intima-media

Type 1 Plaque - Plaques have a thin rim over the surface but are predominantly anechoeic.

Type 2 Plaque - Plaques have less than 25%echogenic components

Type 3 Plaque - Plaques have less than 25%hypoechoic components

Type 4 Plaque - Plaques are predominantly echogenic