PREVALENCE OF CAROTID ARTERY STENOSIS IN ACUTE ISCHAEMIC STROKE PATIENTS

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DECLARATION

I solemnly declare that this dissertation entitled **"PREVALENCE OF CAROTID ARTERY STENOSIS IN ACUTE ISCHAEMIC STROKE PATIENTS"** was done by me at Madras Medical College and Govt. General Hospital, during 2006-2009 under the guidance and supervision of **Prof. K.RAGHAVAN**, **M.D**. This dissertation is submitted to the Tamil Nadu Dr.M.G.R. Medical University towards the partial fulfillment of requirements for the award of M.D. Degree in General Medicine (Branch-I).

Place:

Date:

Dr. K. KAMATCHI

M.D.General Medicine,
Post Graduate,
Institute of Internal Medicine,
MMC, Chennai-3.

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CONTENTS

INTRODUCTION

Stroke remains the second leading cause of death world wide[1], after Ischaemic Heart Disease. 85% of stroke cases are due to infarction and 15% are due to haemorrhage. Carotid athero sclerosis remains an important cause of ischaemic stroke[2].

Carotid atherosclerosis occurs in patients with atherosclerotic risk factors like diabetes mellitus, hypertension, smoking and hyperlipidemia. The internal carotid artery is the commonest site of atherosclerosis next to abdominal aorta, followed by the common carotid artery. The extra cranial part of internal carotid artery is the commonest site of atherosclerosis than the intracranial part of internal carotid artery.

Carotid atherosclerosis leads to plaque formation and these plaques gradually increase in size and cause stenosis. Atherosclerotic plaques interrupt the endothelium and then ulcerate. As the endothelium is breached, platelets adhere to the wall and a Hemostatic plug is formed. This platelet nidus initiates coagulation cascade and an occlusive thrombus is formed.

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

Atherosclerotic plaques and stenosis can be detected by non-invasive ultrasound imaging of the carotid arteries which has high sensitivity and specificity in detecting carotid artery stenosis.

Patients with carotid artery stenosis are at higher risk of development of stroke and recurrence of stroke after a stroke/TIA.

In this study we attempted to find out the prevalence of carotid artery stenosis in ischaemic stroke patients and whether they are prone for recurrence or not so that aggressive secondary preventive measures can be directed to those patients.

Carotid artery stenosis can be assessed by means of non invasive high - resolution B-mode ultrasonography of the carotid arteries.

Carotid ultrasonography combines B mode ultrasound image with a Doppler ultrasound assessment of blood flow velocity. These plaques alter the blood flow haemodynamics and increase the systolic flow velocity. With this increased systolic flow velocity stenosis can be detected and severity can be assessed, and this can be helpful in our management protocol for ischaemic stroke patients with carotid artery stenosis as a cause.

AIMS & OBJECTIVES

1. To find out the prevalence of carotid artery stenosis in acute ischaemic stroke patients.

2. To find out whether there is any association between carotid artery stenosis and risk factors such as diabetes mellitus, Hypertension, Hyperlipidemia, smoking and age.

REVIEW OF LITERATURE

Cerebrovascular accident is an important health problem world wide[3]. The annual incidence of stroke is 0.2 to 2.5/1000 population. World wide approximately 20 million suffer from stroke each year, of these 15 million survive, while the other 5 million become disabled by stroke[5]. The global burden of stroke is 9.4 million deaths/year[1]. In India, incidence of stroke is 33/1,00,000 population[4].

Stroke is defined as an abrupt onset of neurological deficit that is attributable to a focal vascular cause[6]. It may be either due to infarct of the brain tissue or haemorrhage into or around the brain.

Stroke may be due to different causes and pathology.

CAUSES OF STROKE

1. Atherosclerotic thrombosis
2. Embolism
3. TIA's
4. ICH

5. SAH

6. Hypertensive haemorrhage

7. Head trauma

8. Metastatic brain tumour

9. Amyloidangiopathy

10. Trauma and dissection of carotid and basilar arteries

11. Dissecting aortic aneurysm.

CAUSE OF ISCHAEMIC STROKE

COMMON CAUSES

ATHERO THROMBOSIS

- Lacunar Stroke (Small Vessel)
- Large vessel thrombosis
- Dehydration
- Embolic occlusion

ARTERY - TO - ARTERY

- Carotid arteries
- Aortic arch
- Arterial dissection

CARDIO EMBOLIC

1. Atrial fibrillation

2. Mural thrombus

3. Myocardial infarction

4. DCM (Dilated Cardiomyopathy)

5. Valvular lesion

   Mitral stenosis / Mitral regurgitation

   Mechanical valve

   Bacterial endocarditis

6. Paradoxical embolus

   ASD

   Patent foramen ovale

7. Atrial septal aneurysm

8. Spontaneous echo contrast

UNCOMMON CAUSES

1. Hypercoagulable disorders

   Protein C deficiency

   Protein S deficiency

Antithrombin III deficiency

Antiphospholipid syndrome

Factor V leiden mutation

Prothrombin G20210 mutation

Systemic malignancy

Sickle cell anemia

B- Thalassemia

Polycythemia vera

Systemic lupus erythematosus

Homocysteinemia

Thrombotic thrombocytopenic purpura

DIC

Dysproteinemias

Nephrotic syndrome

Inflammatory bowel disease

Oral contraceptives

2. Venous sinus thrombosis

3. Fibromuscular dysplasia

4. Vasculitis

5. Cardiogenic

6. SAH – Reactive secondary vasospasm

7. Drugs cocaine

8. Moyamoya disease

9. Eclampsia

Ischaemic stroke is responsible for 85% of cases of stroke. It may be due to athero thrombosis (53%) of cerebral vessels, which may be further classified into athero thrombosis of large vessels (33%), small vessel thrombosis (Lacunar strokes 20%) and embolic (32%).

Similar to atherosclerosis occurring in coronary arteries, athero sclerosis and thrombosis of intracranial cerebral blood vessels and extracranial carotid arteries occurs. Atherosclerosis of the carotid arteries leads to plaque formation and stenosis of the carotid arteries.

NORMAL CAROTID ANATOMY

1. The aortic arch gives rise to the innominate artery, the left common carotid artery, and the left subclavian artery. The innominate artery bifurcates into the right

common carotid artery and the right subclavian artery. The left common carotid artery arises from the aortic arch in 70% of People and from the innominate artery in 20%.

2. The aortic arch can be classified into 3 types based on the distance of the orgin of the great vessels from the top of the arch. The widest diameter of the left common carotid artery is used as reference unit. If all of the great vessels originate within one diameter length from the top of the arch it is classified as Type I arch. In Type II arch, all great vessels originate within two diameters length from the top of the arch. In Type III arch the great vessels originate beyond two diameter length from the top of the arch.

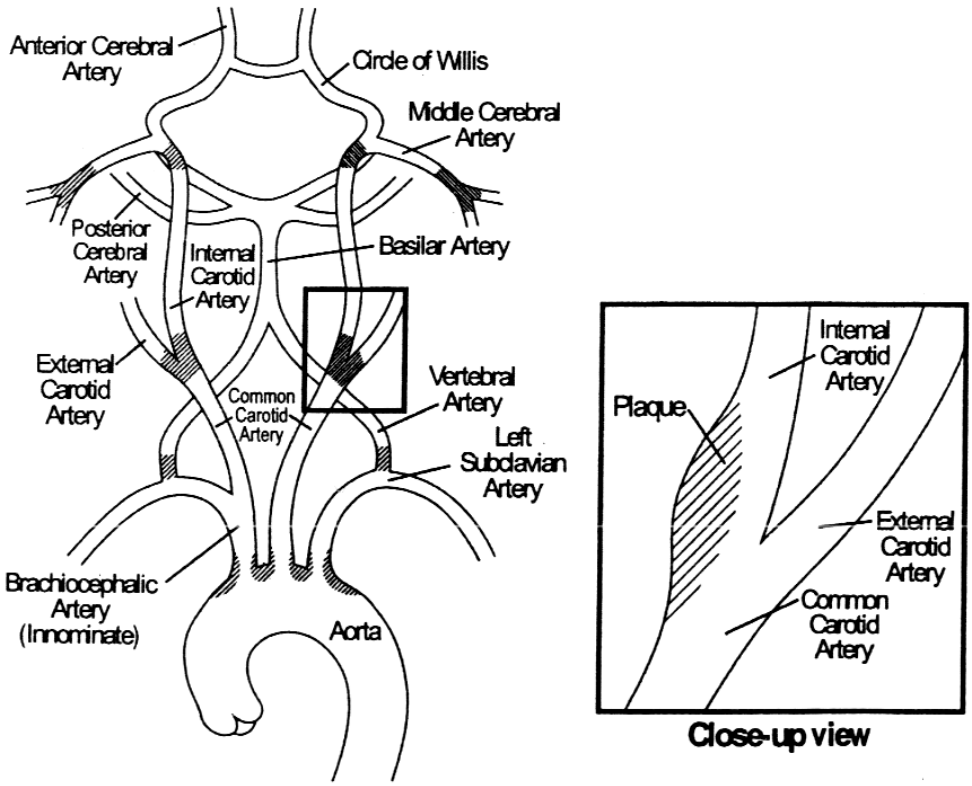
3. The common carotid arteries divide into the internal and external carotids at the C4 - C5 level in 50% of patients. In approximately 40% of the patients the bifurcation is higher and lower in the remainder.

4. The internal carotid artery gives the ACA, MCA and posterior communicating artery which participate in the

formation of circle of Willis. Stenosis and occlusion of ICA/CCA causes ischaemic stroke.

5. ECA supplies facial muscles and thyroid and its occlusion is rare.

COMMON SITES OF CAROTID ATHEROSCLEROSIS



Normal anatomy of the aortic arch, great vessels, and circle of Willis. The shaded areas depict the areas most prone for the development of atherosclerosis.

RISK FACTORS FOR STROKE

UNMODIFIABLE RISK FACTORS

Age	:	M > 45, F > 55 years
Gender	:	Male
Race	:	African -American men
Family History of Previous stroke		

MAJOR MODIFIABLE RISK FACTORS [6]

- Smoking
- Hypertension (BP>140/90 mm Hg)
- Carotid stenosis
- Hyperlipidemia (high LDL and Low HDL)
- Diabetes mellitus
- Atrial fibrillation
- TIA
- Ischaemic Heart Disease
- Physical inactivity
- Obesity
- Atherogenic diet

NOVEL RISK FACTORS [7]

- Alcohol
- Increased fibrinogen levels and increased platelet count
- Increased Haemotocrit
- Dehydration
- Anticardiolipin antibodies
- OCP
- Pregnancy
- Homocysteine
- Lipoprotein (a)
- D.Dimer
- Proinflammatory factors (CRP)
- Chronic infection - CMV, H. pylori
- Lipoprotein
- Microalbuminuria
- Insulin resistance

RISK FACTORS FOR CAROTID ATHEROSCLEROSIS

- Smoking
- Age > 45 Yrs
- Gender: Under 75 years M > F
  Over 75 years F > M
- Hypertension
- Diabetes mellitus
- Hyperlipidemia
- Obesity
- Chronic infection with CMV, HSV

PATHO PHYSIOLOGY OF CAROTID ATHEROSCLEROSIS AND PLAQUE FORMATION

Atherosclerotic carotid disease is a common cause of cerebral ischaemia [12,13]. Atherosclerotic carotid disease develops at branch points and bends and regions of disturbed blood flow especially at the first 2 cm from the origin of CCA, Bifurcation of CCA, ICA. The extra cranial carotid artery is far more commonly involved than intracranial part [10,11].

Plaques are more often localised in the ICA than the external carotid artery and tends to extend from the outer wall of the carotid sinus into the CCA. These fibrous plaques are considered as the hallmark of advancing atherosclerosis and they occur in carotid arteries as early as 25 to 40 Yrs. There is a linear increase in the risk of stroke as the stenosis increases to more than 70%.

A recurrent injury to the intima is the important step in the initiation of atherosclerotic lesion. The effects of injury are influenced by turbulent flow, HT induced shearing and vibration forces, hyperlipidimia. stress, and sedentary life style. Platelets adhere to the injured exposed endothelium and circulating plasma lipids enter the lesion [8,9]. Smooth muscle cells migrate from media to intima where they proliferate and leading to the formation of atheromatous plaque.

MECHANISM BY WHICH A CAROTID PLAQUE CAUSES CEREBRAL ISCHAEMIA AND INFARCT

1. Carotid plaque is highly vascularised. Rupture of this vascular plaque can result in plaque hemorrhage, fissure, erosion and ulceration with subsequent complete vessel obstruction or distal atherothrombo embolism into large

arteries or smaller arteries causing hemisphere or lacunar infact. This mechanism accounts for most cerebrovascular events due to carotid disease. When critical plaque size and reduction in lumen are reached the occlusive process accelerates. Reduced lumen and plaque bulk creates local turbulence in blood flow [14,15]. Progressive increase in the size of plaque leads to stenosis or occlusion.

2. Larger plaques can result in high grade stenosis or obstruction with subsequent ischaemic stroke in watershed areas due to a reduction in blood flow.

Arteriographic and necropsy studies show that occlusion of the cerebral and extracerebral arteries are the frequent cause of ischaemia and infarction.

These atheromatous plaques cause thickening of the wall and stenosis. Platelet material mixed with fibrin complexes superimpose on the atheroma and convert the stenosis into a thrombotic occlusion of the lumen.

HAEMODYNAMICALLY SIGNIFICANT OR SEVERE STENOSIS

A stenosis to become haemodynamically significant depends on the reduced cross - sectional area of the blood vessel, length of stenosis, velocity of blood flow and blood viscosity. Of these the cross sectional area is the most important. The most common method is the percentage of reduction of vascular lumen, which correlates with the reduction of cross sectional area of the blood vessel.

PATHOPHYSIOLOGY OF CEREBRAL ISCHAEMIA AND ISCHAEMIC STROKE

It has two process

- Loss of supply of oxygen and glucose secondary to vascular occlusion.
- An array of changes in cellular metabolism consequent to collapse of energy producing process.

VASCULAR FACTORS [16]

At the centre of the ischaemic stroke is a zone of infarction. This occurs when cerebral blood flow decreases below 10-12ml/100gm/mt of brain tissue. At this point K+ increases and ATP, creatine

phosphate are depleted. This is reversible if circulation is restored to normal.

Disturbances of calcium ion homeostasis and accumulation of free fatty acids interfere with full recovery. These free fatty acids destroy the phospholipids of neuronal membranes. Prostoglandins, leukotrienes and free radicals accumulate and intracellular proteins and enzymes are denatured, cells swell resulting in cytotoxic edema.

Penumbra is a zone which is marginally perfused and there are viable neurons. By elevating the systemic blood pressure or improving the flow properties of blood in small vessels, there is improvement in the flow in penumbra and this helps to salvage the brain tissue.

METABOLIC FACTORS

The excitatory neurotransmitters glutamate and aspartate are released by ischaemic cells. They excit the neurons and produce intracellular influx of Na+ and Ca++. These are said to be responsible for irreversible cell injury [17,18,19].

Increased availability of glutamate opens membranes and increases Na+ and Ca++ influx into cells. Large influxes of Na+ followed by entry of Cl- Ions and water causes cell swelling at both NMDA and non NMDA receptors. Changes in ionic concentration of Na+, K+ and Ca++, release O_2 free radicals, release of excitatory neuro transmitters [18,19]. Further damage of cells leads to more local biochemical changes and causes more neuronal damage. The process of ischaemia then becomes irreversible despite reperfusion of tissues. Ischaemia may cause programmed cell death referred to as apoptosis.

ANATOMICAL SITES OF INFARCTS CAUSED BY CAROTID OCCLUSION

1. MASSIVE HEMISPHERE INFARCTION

These infarcts occupy the entire area of supply of MCA or ACA. They account for about 55% of infarcts caused by occlusion of either internal carotid or common carotid artery.

2. PARASYLVIAN INFARCTION

These infarcts involve the cortical area around the fissure of sylvius and the basal ganglia and internal capsule.

3. WATER SHED INFARCTION

Infarcts occurs in the cortex and along the junction of the anterior and middle cerebral arteries following carotid occulusion.

4. DISTAL OR TERMINAL INFARCTION

Small ischaemic lesion scattered through out the white matter in the territory supplied by the peripheral branches of MCA.

CLINICAL MANIFESTATIONS OF CAROTID ARTERY OCCLUSION

The clinical manifestations can be classified into

1. Completed stroke
2. Stroke in evolution
3. Transient ischaemic attack/TIA
4. Reversible ischaemic neurological defecit /RIND

1. COMPLETED STROKE

A completed stroke can be defined as a neurological deficit which has persisted for a considerable time. The occlusion of internal carotid artery in some cases presents as an acute hemiplegia that involves the face and arm known as faciobrachial monoplegia, but it can present also as hemiplegia, the commonest form. The hemiplegia is often accompanied by cortical sensory loss and hemianopia.

When the dominant hemisphere is involved it presents with aphasia along with the above mentioned feature. The pathognomonic feature of carotid occlusion is ipsilateral mono ocular blindness with contralateral hemiplegia.

Neck should be examined for bruit over the carotid artery which is heard in carotid occlusion.

2. STROKE IN EVOLUTION

Patient may develop hemiplegia evolving step by step over several hours followed by some recovery and recurs some hours later characterised by dense hemiplegia which persists. This is known as stuttering hemiplegia.

3. TRANSIENT ISCHAEMIC ATTACK

It is characterised by a neurological deficit lasting less than 24 hours (usually 5-15mts). It is referred as mini/transient stroke. The typical carotid TIA's are brief lasting only 7-10mts, from which patients recovers such that at the end of 24 hours there is no focal neurological deficit.

Patients who suffer carotid stroke from extracranial carotid source have a known prior TIA incidence of 50-70%.

It presents as weakness/numbness of part or all of the contralateral body with or without speech disturbances depending upon whether the dominant hemisphere is involved. The deficit is due to ischaemia to a portion of the cerebral cortex either because of embolism or perfusion failure.

4. TRANSIENT MONO OCULAR BLINDNESS

Transient mono ocular blindness also known as amaurosis fugax is an important manifestation of carotid artery disease. They are brief mono ocular visual obscurations described by patients as 'fog' or 'cloud'. It is a painless loss of vision. It lasts lesser than 10 minutes vision is fully restored following an attack. It is a shortest TIA.

Transient mono ocular blindness is a relatively infrequent type of TIA. In a few cases of carotid occlusion with completed strokes the stroke had been preceded by one or more TIA's.

5. REVERSIBLE ISCHAEMIC NEUROLOGIC DEFICT

RIND is characterised by an ischaemic event in which the deficit usually occurs over a 24 to 72 hour period, but it may take as long as 1 week to resolve.

OXFORDSHIRE COMMUNITY STROKE PROJECT CLASSIFICATION OF STROKE

It classifies stroke according to clinical features in to

1. TACI (Total Anterior Circulation Infarct)
2. PACI (Partial Anterior Circulation Infarct)
3. LACI (Lacunar Infarct)
4. POCI (posterior Circulation Infarct)

TACI

It is characterised by all of these

a) Higher function dysfunction

- Dysphasia
- Visuospatial defects

b) Homonymous hemianopia

c) Motor/sensory deficit

- >2/3 of face/arm/leg

PACI

It is characterised by any one of these

a) Two out of three as TACI

1) Higher function dysfunction

-Dysphasia

-Visuospatial defect

2) Homonymous hemianopia

3) Motor /sensory deficit

b) Higher function Dysfunction alone

c) Limited motor/sensory deficit

LACI

It is characterised by any one of these

a) Pure motor stroke (>2/3 face/Arm/Leg)

b) Pure sensory stroke (>2/3 face/Arm/Leg)

c) Sensory motor stroke (>2/3 face/Arm/Leg)

d) Ataxic hemiparesis

LACI

Must have none of these

a) New dysphasia

b) New visuospatial defect

c) Proprioceptive sensory loss only

d) No vertebro basilar features

POCI

It is characterised by any of these features

a) Ipsilateral cranial Nerve palsy and contralateral motor/ sensory deficit

b) Bilateral motor or sensory deficit

c) Conjugate eye movement problems

d) Cerebellar dysfunction without ipsilateral long tract signs

e) Isolated homonymous hemianopia

STOKE EVALUATION

IMPORTANT POINTS IN HISTORY

a) Time of onset and rate of progression

b) Risk factors

1. Previous stroke, TIA
2. HT
3. DM
4. Ischaemic Heart Disease
5. Peripheral vascular disease
6. Hyperlipidemia
7. Smoking
8. Alcohol

IMPORTANT POINTS ON EXAMINATION

a) Pulse rate and rhythm

b) Blood pressure

c) Heart sounds

d) Neck bruit

e) Peripheral pulses

f) Funduscopy

1. An assessment for the presence of a cervical bruit is an important part of the physical examination but should not be relied on as the sole marker for the presence of carotid disease.

   In the North American Symptomatic Carotid Endarterectomy Trial (NASCET)[20] the presence of a cervical bruit had an approximately 60% sensitivity and specificity for high grade carotid stenosis.

2. In the Framingham study[21], the presence of a carotid bruit in asymptomatic patients doubled the risk of stroke, but most of these strokes occurred in vascular beds different from that of the carotid bruit. The presence of a bruit may be a general marker for patients at higher risk of cerebrovascular events.

3. All patients should have an evaluation of the carotid arteries after a stroke or TIA. The risk of a second stroke is elevated for several years after the first stroke or TIA. Symptomatic patients with 70% or more stenosis have an 8% risk of stroke at 30 days and a 13% annual incidence of stroke.

4. The risk of stroke in asymptomatic patients increases as the degree of carotid stenosis increases. Asymptomatic patients with 60% or more stenosis have a stroke risk of approximately 2% per year. Asymptomatic patients with 80% or more stenosis have an annual risk of 5% per year [22].

STROKE EVALUATION

NEUROLOGIC ASSESSMENT

a) Consious level

b) Higher functions

1. Orientation
2. Memory
3. Speech
4. Language
5. Attention

c) Visual fields

d) Eye movements

e) Swallowing

f) Limb power

g) Trunk control

h) Gait

i) Coordination

j) Sensory testing

Important investigations to be carried out in stroke patients		*Important Conditions to be looked for*
a) Complete blood count:		Anemia, polycythemia, leukaemia, thrombocytopenia
b) ESR	:	Vasculitis, infective endocardits, hyper viscosity
c) Plasma glucose	:	Diabetes, hypoglycaemia
d) Plasma cholesterol	:	Hypercholesterolemia
e) Syphilis serology	:	Syphilis, anticardiolipin antibody
f) Urine analysis	:	DM, renal disease

g) ECG	:	LVH, arrythmias, Myocardial ischaemia/infarct
h) Echo	:	For valvular abnormalities, vegetation, SEC, Lt. atrial clot, LVEF, etc…
i) CT-Brain	:	To diagnose infarct or haemorrhage
j) Carotid doppler	:	To detect athero sclerotic plaques and carotid stenosis
k) MRI Brain	:	To detect early infarct

COMPUTED TOMOGRAPHY [23]

CT scan plays an important role in the diagnosis of cerebral infarction. It is well established that CT scan can distinguish between an ischaemic stroke, haemorrhagic stroke as well as haemorrhagic infarction.

The classic neuropathologic process that occurs during the evolution of an infarction is well reflected by the CT scan. The

radiologic imaging characteristics are divided into four stages and are dependant on the time from the onset of stroke.

The 4 stages are

1. Hyper acute : <24 Hrs
2. Acute : 24 hrs - 7 days
3. Subacute : 8-21 days
4. Chronic : > 21 days

MRI

MRI can detect ischaemia as early as 1 hour from the onset of stroke. It helps in identifying and locating the infarct in all areas of brain including the posterior fossa which cannot be easily detected by CT scan. Perfusion and diffusion weighted images help in identifying the salvageable ischaemic penumbra and hence the patient can be thrombolysed (within the golden period-3hours).

INVESTIGATIONS FOR DIAGNOSING CAROTID ARTERY STENOSIS

Both invasive and non invasive investigations are available to diagnose carotid artery stenosis.

1. CAROTID ANGIOGRAPHY

Carotid angiography with digital subtraction angiography is the gold standard investigation for assessment of carotid atherosclerosis[24]. It allows the simultaneous assessment of the aortic arch, subclavian arteries, vertebral arteries and intracranial circulation. It enables the accurate measurement of stenosis and the presence of collateral circulation.

Carotid stenosis can be quantified by the NASCET (North American Symptomatic Carotid Endartectomy Trialists) criteria[20] and ECST (European Carotid Surgery Trialists Collaborative Group) criteria.

According to NASCET criteria, the normal reference internal carotid diameter is the maximum diameter of the ICA distal to the lesion.

According to the ECST criteria the normal reference diameter is the estimated position of the external wall of the carotid sinus.

The NASCET criteria has less variability and it is widely used.

Limitations of carotid angiography are - it is an invasive method. Patients renal function should be normal because a contrast is injected.

The procedural complication like neurologic events (such as stroke) can occur in 1-4% of the patients.

MAGNETIC RESONANCE ANGIOGRAPHY

CEMRA (Contrast Enhanced MRA) is a sensitive (80-90%) and specific (60-90%) test for carotid disease.

Its advantages are slightly better accuracy than duplex USG, its reproductivity and the ability to visualise the entire carotid tree along with intracranial circulation.

Its limitations are
- high cost
- inability to study critically ill and claustrophobic patients.
- or patients with metallic implants or pacemakers.

DUPLEX ULTRA SOUND

It is the most widely used method for the detection and quantification of carotid artery disease. This non-invasive test should be the first study ordered to assess carotid disease.

It has a sensitivity of more than 80% and a specificity of more than 90% (26%). It is non-invasive and less expensive and it is portable. Limitations include-inability to image intra cranial disease and assess collateral flow.

Occasional inaccuracy in distinguishing high-grade stenosis from complete occlusion and the need for an experienced sonographer.

DOPPLER SONOGRAPHY IN THE DIAGNOSIS OF CAROTID ARTERY DISEASE [27]

It is a non-invasive ultra sound technique used for the detection of extracranial and intracranial arterial disease. Continuous and pulsed wave Doppler sonography assess the haemodynamics according to the Doppler shift. Special scanners for studying superficial arteries like carotid and femoral arteries have been developed. For studying carotid arteries, transducers with 4-5 mhz are used.

PRINCIPLES OF DOPPLER STUDIES

An ultrasound beam insonating a blood vessel is partially reflected by red blood cells. If these are moving there is a change in the frequency, if the blood flow is towards the probe and a decrease if it is away from it. This is the Doppler effect and the changes in frequency (difference between Received and Transmitted) is referred to as the Doppler shift.

Doppler Equation

$$FD = fr\text{-}fo = \frac{2foV\cos\theta}{C}$$

FD = Doppler frequency shift

Ultra sonic Doppler equipment is used for detecting and evaluating blood flow. An ultrasonic transducer is placed in contact with the skin surface, it transmits a beam whose frequency is fo. The received frequency fr will differ from fo when echoes are picked up from moving scatterers such as the RBC's. The Doppler frequency is defined as the difference between the received and transmitted frequencies.

"C" is the speed of sound

"V" is the flow velocity

"θ" is the angle between the direction of flow and the axis of the ultrasound beam, looking toward the transducer. It is called the "Doppler angle". If the angle is known and frequency shift measured, blood flow velocity can be calculated.

B-MODE IMAGING IN CAROTID ARTERY DISEASE

Ultrasound imaging is done using "Pulse echo techniques". B-mode display is used in imaging.

Atherosclerotic and ageing changes of the vascular system is well reflected by the structures of the carotid arteries [28]. Atherosclerotic changes are also seen in femoral and carotid arteries in familial hypercholesterolemia. (Arterio sclerothrombosis 1993; 1401-1411).

This artery is well accessible by ultrasonic investigation and it is the main vessel supplying the brain. Carotid artery structure, thickness and percentage of stenosis are good indicators for estimation of risk of stroke.

The main changes in the carotid artery can be observed in the wall structure, which is divided into inner layer (intima), middle layer (media) and outer layer (adventitia). Sclerotic damage of the

vascular system manifests itself by thickening of intima layer and media layers and further increase in thickness along with fat and fibrous tissue deposition leads to atherosclerotic plaque formation and carotid stenosis.

Both A and B scanning methods can be used for ultrasonic investigation. B scan is used more frequently, since it gives overall view of the artery, measure of intima-media thickness, the presence of stenosis and the presence of plaques [28,29]. The intimal and medial layers comprises of endothelial cells, connective tissue and smooth muscle and it is the site of lipid deposition and plaque formation (30).

The normal IMT (intima media thickness) in adults range between 0.5-0.9 mm and an IMT of 0.9 or more is abnormal and it is associated with sonographically visible plaque. Small carotid plaques are very commonly present in individuals older than 50 years and the prevalence of plaque increases with age to a high of 80% for men between the ages of 80-100 years.

EVALUATION OF PLAQUE

Plaque Extent and Severity

Having detected a plaque, its extent and severity has to be described. By using precise ultrasound images longitudinal and circumferential extent of the plaque and its thickness can be estimated. These are assessed by transverse images.

CATEGORIES OF ATHEROSCLEROTIC PLAQUES

UNCOMPLICATED OR STABLE PLAQUE

It is uniform in composition and is covered by a sub intimal fibrous cap.

COMPLICATED OR UNSTABLE PLAQUE

Its architecture is not uniform and is characterised by plaque necrosis, haemorrhage into the plaque, calcification, thinning or disruption with embolisation and occlusion of cerebral arteries and causing ischaemia or infarction.

PLAQUE CHARACTERISATION

The primary role of carotid sonography is the detection and assessment of carotid stenosis

LOW ECHOGENICITY

A fibro fatty plaque which contains large amount of lipid is low in echogenicity. It is associated with elevated levels of LDL, plaque ulceration and increased risk of cerebral ischaemic symptoms.

MODERATE ECHOGENICITY

When the collagen and cellular content increases it becomes moderately echogenic. It is less associated with cerebral ischaemic symptoms.

STRONG ECHOGENICITY

Dystrophic calcification occurs in the plaque and such calcification generates strong reflections. It has a very low risk of cerebral ischaemic symptoms.

ULTRASOUND ASSESSMENT OF CAROTID STENOSIS

Carotid stenosis is being assessed by PSV (peak systolic velocity) and EDV (end diastolic velocity) of blood flow in carotid arteries. Colour flow imaging accurately identifies ICA occlusion than grey scale imaging. Haemodynamic quantification of the severity of stenosis is achieved by analysis of Doppler spectral wave form.

As a stenosis develops PSV first becomes elevated. PSV is a principal measure of stenosis severity. EDV rises as the stenosis becomes severe. EDV is a good marker of high grade stenosis.

THE SOCIETY OF RADILOGISTS IN ULTRASOUND CONSENSUS CRITERIA FOR GRADING CAROTID STENOSIS

Stenosis Range	***PSV***	***EDV***	***Plaque***
Normal	<125cm/sec	<40cm/sec	None
<50%	<125cm/sec	<40cm/sec	<50% of diameter reduction
50-69%	125-230cm/sec	40-100cm/sec	>50% of diameter reduction
>70%	>230cm/sec	>100cm/sec	>50 of diameter reduction
Near occlusion	Low or Undetectable	Variable	Significant stenosis and detectable lumen
Complete Occlusion	Undetectable	Not applicable	Significant stenosis and no detectable lumen

STAGES OF CAROTID ARTERY STENOSIS

1. A mild stenosis (upto 50%) is characterised by local increase of peak and mean flow velocities.

2. A moderate stenosis (50-69%) shows a distortion of the normal pulsatile flow in addition to local increase of peak and mean frequencies. Peak systolic flow deceleration are found in the post stenotic segment.

3. A severe stenosis (>70%) produces marked increase in peak velocities.

4. A subtotal stenosis (>95%) is characterised by a small signal of variable frequencies that decrease once a stenosis becomes pseudo occlusive.

5. With complete internal carotid artery occlusion, in which case no signal is detectable, the spectra of common carotid artery are dampened and retrograde flow may occur.

According to Goldstein LB, Adams et al [31] the incidence of stroke increases as the percentage of stenosis increases. That is >60%stenosis, stroke risk is 2% per year and with >80% stenosis, the stroke risk is 5% per year.

CEA (Carotid End Arterectomy) and aspirin is advised in selected patients (medically stable patients expected to live >5years) with ≥ 60% stenosis, performed by a surgeon with surgical morbidity/mortality rate <3%. Asymptomatic patients with <60% stenosis should be treated with aspirin along with treating other risk factors for stroke.

In ACAS (Asymptomatic Carotid Atherosclerosis Study)[32] There was 53% relative risk reduction in CVA/related death and absolute reduction of 5.9% over 5 years in patients with carotid stenosis (60-90%) treated with CEA+aspirin 325mg/day.

In ACST (Asymptomatic Carotid Surgery Trial)[33] which is the largest prospective study (upto May 2004) 3120 patients with ICA stenosis >60% were subjected to CEA vs Medical therapy with aspirin and their stroke risk was 6.4% with CEA and stroke risk was 11.8% with medical therapy alone.

SYMPTOMATIC CAROTID ARTERY STENOSIS

By symptomatic carotid artery stenosis we mean patients with carotid artery stenosis who have experienced an attack of TIA/stroke.

In these patients (with symptomatic carotid artery stenosis), with significant stenosis (>70%) the risk of recurrent stroke is 8% at 30 days and an annual incidence of stroke is at a rate of 13% per year.

Mayberg et al in Veteran affairs co-operative studies programme 309 trialists [34] group - JAMA have recommended the following strategy for patients with symptomatic stenosis.

Symptomatic Stenosis

<50% Stenosis : No significant benefit of surgery
patients should be treated with aspirin 325mg/day.

50-69% Stenosis : Patients with greater degree of stenosis, those greater than 75 years of age, Men, patients with recent (<2 weeks) stroke (rather than TIA) and patients with hemispheric symptoms benefit (than transient mono ocular blindness) from CEA.

>70% Stenosis : with recent TIA/ischaemic stroke in the last 6 months, CEA by a surgeon with a perioperative morbidity and mortality <6% is recommended.

For patients in whom the stenosis is difficult to access surgically and in those with medical conditions that which greatly increase the risk of surgery such as radiation induced stenosis, arterial dissection and fibro muscular hyperplasia, carotid angioplasty and stenting is recommended.

THE RISK OF STROKE AND THE SEVERITY OF CAROTID STENOSIS IN SYMPTOMATIC PATIENTS IN THE NASCET TRIAL[20]

ICA stenosis	***Medical group(aspirin)***	***Surgical group (CEA within 6months)***	***NNT***
70-99%	26%	12.9%	8
50-69%	22.2%	15.7%	15
<50%	18.7%	14.9%	26

NNT: Number Needed to Treat to prevent one stroke annually.

CLEAR INDICATIONS FOR CEA

- Symptomatic men and women aged 80 years or younger with 70% or greater carotid stenosis if surgical risk for stroke and death is 6 to 7% or less.

- Asymptomatic men and women aged 80 years or younger with a 80% or greater carotid stenosis if surgical risk for stroke and death is 3% or less.

POSSIBLE INDICATIONS FOR CEA

- Symptomatic stenosis > 50% with risk factors.
- Asymptomatic stenosis >60%.

CEA IS CONTRAINDICATED IN

- Symptomatic / Asymptomatic stenosis <50%.
- Asymptomatic stenosis <60%.

MATERIALS AND METHODS

Place of study	:	Institute of Internal Medicine and Barnard Institute of Radiology GGH, Chennai.
Collaborative Department	:	Radiology
Study design	:	Cross sectional hospital based prevalence study
Study Sample	:	75 Patients
Ethical committee clearance	:	Obtained
Period of study	:	January'08 to June'08

INCLUSION CRITERIA

1. Age : 18-80 years
2. Sex : Both Gender
3. Patients with acute stroke of less than 2 weeks duration.
4. Patients with acute stroke with CT brain showing infarct.
5. Patients with or without known H/o DM/HT/ hyperlipidemia.
6. Patients with risk of accelerated atherogenesis like smoking.
7. Patients with or without past H/o of CVA/CAD.

EXCLUSION CRITERIA

NEUROLOGICAL

1. Duration of stroke > 2 weeks
2. Patients with Haemorrhagic stroke
3. Patients with H/o head injury

SYSTEMIC ILLNESS

1. Haemodynamically unstable patients
2. Malignancy
3. Unconscious patients
4. HIV
5. Stroke due to infections like TB
6. Metabolic emergencies
7. Poor general condition
8. Other systemic illness

All patients with ischaemic stroke of acute onset admitted in the medical wards of GGH between Jan'08 and June'08 were included in the study. Examination was carried out as soon as the patients were admitted.

All vitals were recorded and careful methodical examination of the central nervous system carried out recording all the physical signs in order.

Cardiovascular system was carefully examined, arterial pulses including carotid, internal carotid, radial and all other peripheral pulses were examined.

In all the cases, with the help of close relatives the preceding symptoms and risk factors were enquired.

Investigations like Hb% TC,DC, ESR, platelet count, Fasting blood sugar, urea, Serum Creatinine, fasting lipid profile, urine analyses, CXR, ECG, Echo, CT-Brain and Carotid Doppler were done for all patients.

All patients were subjected to CT scan brain study and colour Doppler study of extracranial carotid arteries and vertebral arteries.

Systolic and diastolic velocity of blood flow, carotid intimal medial thickness, presence of atheromatous plaque and thrombus was looked for and then the percentage of stenosis of the affected arteries were calculated.

The Doppler instrument used in the study was Toshiba. It has a triplex scanning system comprising of

1. High resolution B-mode imaging
2. Pulsed wave Doppler sonography
3. Colour Doppler flow imaging

CAROTID DOPPLER ULTRA SOUND EXAMINATION

Patients are made to lie in supine position for examining the carotid arteries and the examiner is seated at or next to the patients head. Transducer positions are used accordingly to examine the carotid arteries in long axis planes, which shows the CCA, ICA and carotid bifurcation best. The images are viewed and then recorded.

STATISTICAL ANALYSIS

Statistical analysis was carried out for 75 subjects. Age, Presence of diabetes, hypertension, Smoking, Alcoholism and hyperlipidemia were analysed in patients with and without carotid stenosis admitted for acute ischaemic stroke. The statistical significance was calculated using Chi- square test.

Statistical significance was taken when P value was <0.05. Statistical analysis were carried using standard formulae. Microsoft Excel 2007 and SPSS (Statistical Package for Social Sciences) Version 13.0 softwares were used for data entry and analysis.

OBSERVATION

We included 75 patients with acute ischaemic stroke in our study and all of them had a carotid Doppler done.

Table 1: Prevalence of Carotid Stenosis in Acute Ischaemic stroke patients

Total No. of patients in whom carotid Doppler was done	***Patients with carotid stenosis***	***Percentage***	***Patient without carotid stenosis***
75	35	46%	40

Table 2: Patient characteristics

Characteristics	***Present***	***Absent***
Diabetes	27	48
Hypertension	39	36
Smoking	37	38
Increased total cholesterol	41	34
Increased LDL	31	44
Decreased HDL	16	59
Increased TGL	40	35

Table 3: Age distribution of carotid stenosis in stroke patients

Age group	*Total*	*Carotid Stenosis*	*No stenosis*
<50	31	9	22
50-59	32	18	14
>60	12	8	4

Table 4: Percentage of patients with carotid stenosis in different age groups

Age	*Percentage of Patients*
<50	29%
50-59	56%
>60	66%

P= 0.03046

It was found that percentage of patients with carotid stenosis was increasing with increase in age and there was a statistically significant association between age and carotid stenosis (P<0.05)

In our study out of 20 young stroke patients between 15-45 years, 5 patients had carotid stenosis. The prevalence of stenosis was about 25% in those patients.

Table 5: Sex distribution of carotid stenosis

Sex	*Total*	*Stenosis*	*No stenosis*
Male	60	32	28
Female	15	3	12
Total	75	35	40

P=0.02064

Table 6: Percentage of Male and Female patients with carotid stenosis

Sex	*Percentage of patients*
Male	53%
Female	20%

P<0.001

It was found that more male patients had carotid stenosis than female patients and it was statistically significant.

RISK FACTOR ANALYSIS IN CAROTID STENOSIS PATIENTS

Table 7: Correlation between diabetes mellitus and carotid stenosis

	Total	***Stenosis***	***No Stenosis***
DM	27	20	7
NON - DM	48	15	33
Total	75	35	40

P=0.00036

There was a correlation between DM and Carotid stenosis. More DM patients had carotid stenosis than Non-DM patients and it was statistically significant.

Table 8: Correlation between Hypertension and Carotid stenosis

	Total	***Stenosis***	***No Stenosis***
HT	39	29	10
NON - HT	36	6	30
Total	75	35	40

P<0.001

Prevalence of carotid stenosis was more in Hypertensives than in normotensives and it was statistically significant.

Table 9: Correlation between Smoking and Carotid stenosis

	Total	***Stenosis***	***No Stenosis***
Smokers	37	28	9
Non – Smokers	38	7	31
Total	75	35	40

P<0.001

Prevalence of carotid stenosis was more in smokers than in non-smokers and it was statistically significant.

Table 10: Correlation between patients with increased cholesterol and Carotid stenosis

	Total	***Stenosis***	***No Stenosis***
Cholesterol (>200mg/dl)	41	25	16
Cholesterol (<200mg/dl)	34	10	24
Total	75	35	40

P=0.006

Prevalence of carotid stenosis was more in patients with increased cholesterol than in patients with decreased cholesterol and it was statistically significant.

Table 11: Carotid stenosis in patients with increased TGL

	Total	***Stenosis***	***No Stenosis***
TGL >150	41	28	12
TGL <150	34	7	28
Total	75	35	40

P<0.001

Prevalence of Carotid stenosis was more in patients with increased TGL than in patients with decreased TGL and it was statistically significant.

Table 12: Prevalence of carotid stenosis in Low HDL patients

	Total	***Stenosis***	***No Stenosis***
HDL >40	59	22	37
HDL <40	16	13	3
Total	75	35	40

P=0.002

Prevalence of Carotid stenosis was more in patients with Low HDL than in patients with Increased HDL and it was statistically significant.

Table 13: Carotid stenosis in patients with increased LDL

	Total	***Stenosis***	***No Stenosis***
LDL >130	31	25	6
LDL <130	44	10	34
Total	75	35	40

P<0.001

Prevalence of Carotid stenosis was more in patients with increased LDL than in patients with decreased LDL and it was statistically significant.

Table 14: Degree of carotid stenosis in ischaemic stroke patients

Degree of Stenosis	***No. of cases***	***Percentage***
Mild Stenosis (<50%)	13	17%
Moderate Stenosis (50-69%)	12	16%
Severe or Significant Stenosis (>70%)	10	13%
No Stenosis	40	54%

The prevalence of mild stenosis and moderate stenosis were more than severe stenosis. In our study 2 cases with severe stenosis had carotid bruit.

Table 15: Carotid stenosis on Right side and Left side

Right	*Left*
17	18

P=0.8658

There was no particular side preponderance of carotid stenosis and it was more or less equal on both sides and there was no statistical significance between the two sides.

Table 16: Site of carotid stenosis

Side	*ICA*	*CCA*	*Total*
Right	12	5	17
Left	13	5	18
Total	25	10	35

P=0.914

It was found that carotid stenosis was more on internal Carotid arteries both on Right and Left side than common carotid arteries, but it was not statistically significant.

DISCUSSION

In our study we have found that the prevalence of carotid stenosis in acute ischaemic stroke patients is about 46%, consistent with studies done by Oliviero et al [70]. In their study the prevalence of carotid stenosis was about 43% in ischaemic stroke patients.

The percentage of patients with significant stenosis (>70%) was about 13% which is associated with the recurrence of stroke. The prevalence of significant stenosis in studies conducted in Western population is about 14% [36] and 21% [35]. This variation could be due to racial differences [41,42]. Extracranial carotid artery stenosis is more commoner in whites and men [43]. The prevalence of significant stenosis in a study conducted by M.M.Singh et al was about 32% [37].

In our study the prevalence of moderate stenosis was about 16%, mild stenosis was 17% and 54% of stroke patients had no carotid stenosis. The prevalence of asymptomatic carotid stenosis (>50%) in a study conducted in asymptomatic carotid stenosis patients by P.P.Mineva et al was 6.4% [44]

AGE AND CAROTID STENOSIS

We found in our study the percentage of patients who had carotid stenosis, increased with increase in age [38]. The prevalence in patients <50 years, 50-69 years, >60 years was about 29%, 56%, and 66% respectively. In a study conducted by K.Rajamani et al [38] showed increasing incidence of carotid stenosis with increase in age in African American men. Carotid stenosis in keeping with atherosclerotic diseases, increases with age. The risk of carotid atherosclerosis increases after 45 years of age.

SEX AND CAROTID STENOSIS

We found that the prevalence of carotid stenosis was more in males (53%) than females (20%) which was consistent with studies conducted by Jacob et al [39]. It is also shown by Ralph et al [40] that carotid stenosis was commoner in males (43%) than females.

RISK FACTORS OF CAROTID ATHEROSCLEROSIS

1. Diabetes Mellitus and Carotid Stenosis

Carotid artery stenosis was more common in diabetics (74%) than in non-diabetics (31%) and it was statistically significant. K.Rajamani et al [38] have shown in their study that carotid stenosis was more common in diabetics (22%).

2. *Hypertension and Carotid Stenosis*

In our study we found that hypertension was one of the risk factors for carotid stenosis and the prevalence of carotid stenosis was more in hypertensives (74%) than in normotensives (16%) consistent with studies done by Duncan et al, Sutton et al [(63)]. They, in their study, found that asymptomatic carotid stenosis was found in 25% of adults with hypertension, than those without hypertension. Hypertension accelerates carotid atherosclerosis and stenosis [(45-53)]. The predictors of carotid stenosis were systolic BP > 160 mmHg and in isolated Systolic Hypertension patients when diastolic BP was <75 mmHg there was a strong correlation with carotid stenosis.

3. *Smoking and Carotid Stenosis*

In our study we found that smoking acts as a risk factor for carotid stenosis. More smokers (75%) had carotid stenosis than non-smokers (18%), which is also shown by H.R. Muller et al [(54)]. Smoking as a risk factor for carotid stenosis in *Journal of neurology* 1990, Page No. 97-102. [(54)] [(55-62)]

4. Hyperlipidemia and carotid stenosis

In our study the prevalence of patients with increased cholesterol (> 200mg/dl), increased TGL (>150mg/dl), decreased HDL (< 40 mg/dl) and increased LDL (>130 mg/dl) were 61%, 70%, 81% and 80% respectively. The prevalence in patients with decreased cholesterol (<200mg/dl), decreased TGL (<150mg/dl), Increase HDL (>40mg/dl) and decreased LDL (<130mg/dl) were 29%, 20%, 37% and 22% respectively.

Prevalence of carotid stenosis, just like coronary atherosclerotic disease, increases with Hyper cholesterolemia (>200mg/dl) and Increased LDL (> 150mg/dl) and Increased TGL (>130mg/dl) and decreased HDL (<40mg/dl). They are associated with extra cranial large vessel atherosclerosis and also coronary atherosclerosis. Carotid atherosclerosis leads to increase in IMT and plaque formation and stenosis [(68)]. Extracranial carotid atherosclerosis is associated with major brain vessel occlusion, leading to infarct of brain tissue [(64)].

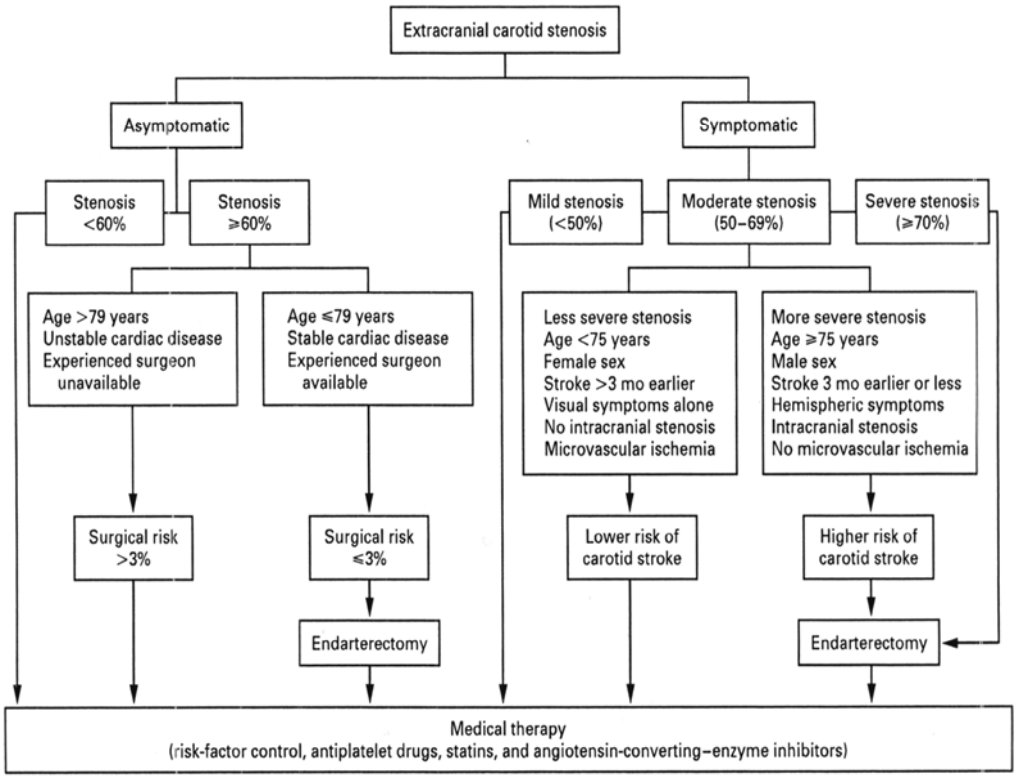
4. Site and carotid stenosis

In our study carotid stenosis was found at the bifurcation of CCA, and the origin of ICA. Carotid stenosis was equal on both

sides and was more on ICA than CCA. ICA stenosis was found in 71% of patients and CCA stenosis was found in 28% of patients.

We have found that age, male sex, smoking, HT, DM and Hyperlipidemia are associated with increased rate of carotid stenosis. In our study every patient with carotid artery stenosis had one or the other risk factor for carotid atherosclerosis. In other words, there was no patient with carotid artery stenosis, without any risk factor in our study. Hence asymptomatic patients with these risk factors should be screened for carotid stenosis to prevent stroke [(65)].

ALGORITHM FOR MANAGEMENT OF EXTRA CRANIAL AROTID STENOSIS



The algorithm is based on the Guidelines of the American Heart Association and the National Stroke Association

SUMMARY

1. 75 Acute ischaemic stroke patients with CT-Brain showing infarcts were taken for the study.

2. Age, Sex, History of DM, HT, Smoking, FBS and Fasting lipid profile were recorded for the subjects.

3. They were subjected to Doppler Ultrasonography of the carotid arteries which is a cost effective and non-invasive method to detect atherosclerotic plaque, carotid stenosis and to measure the degree of stenosis.

4. 35 patients had carotid stenosis. The prevalence of carotid stenosis in our study was 46%. The prevalence of mild, moderate and severe stenosis were 17%, 16% and 13% respectively.

5. The distribution of carotid stenosis was equal on both sides and ICA (71%) was the common site of atherosclerotic plaque and stenosis.

6. There was a statistically significant correlation between increasing age, male gender, smoking, DM, HT, and Hyperlipidemia and the prevalence of carotid stenosis.

CONCLUSIONS

1. Carotid stenosis is one of the common causes of ischaemic stroke.

2. About 46% of ischaemic stroke patients had carotid stenosis in our study.

3. The prevalence of carotid stenosis increases with increase in age, male gender, smoking, DM, HT & Hyper lipidemia.

4. DM, HT, Smoking & Hyperlipidemia act as risk factors for carotid stenosis. Hence patients with DM, HT & Hyperlipidemias should have their carotid arteries screened to detect asymptomatic carotid stenosis and if present, should have their blood glucose, blood pressure and lipids under control and should be started on antiplatelet drugs and statins for plaque regression and for primary prevention of stroke.

5. Patients with stroke who have carotid stenosis (symptomatic carotid stenosis) are prone for recurrence of stroke. They should be advised to control the risk factors for carotid stenosis and should be started on anti platelet drugs and statins. Carotid endarterectomy should be done in selected cases for secondary prevention of stroke.

6. A simple, non invasive screening procedure like Doppler sonography of the carotid arteries in high risk individuals could therefore have profound diagnostic and therapeutic implications in predicting and preventing a potentially fatal and devastating stroke.

FIGURE 1

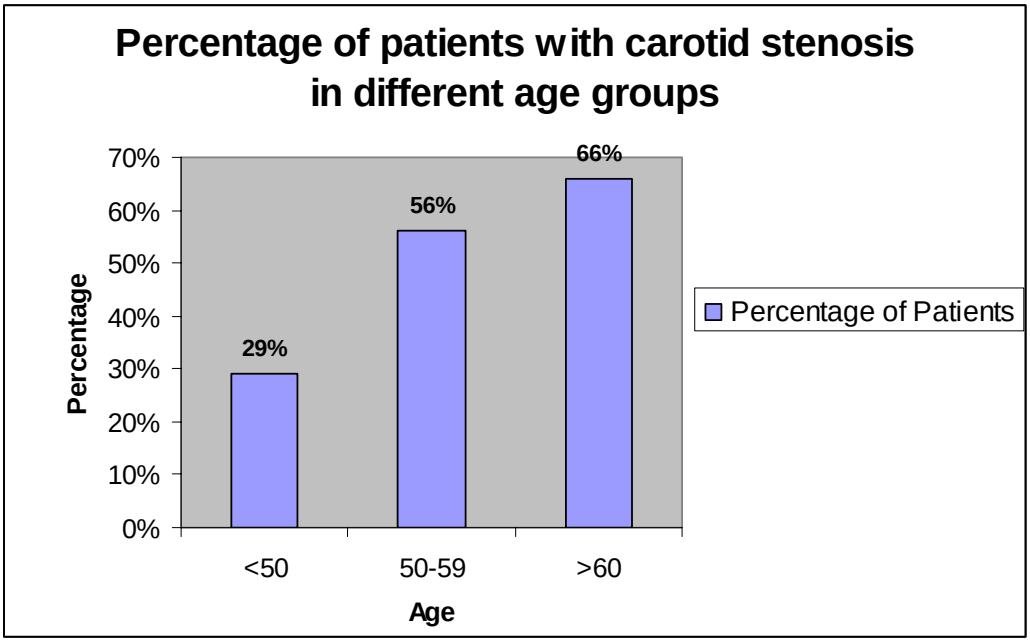
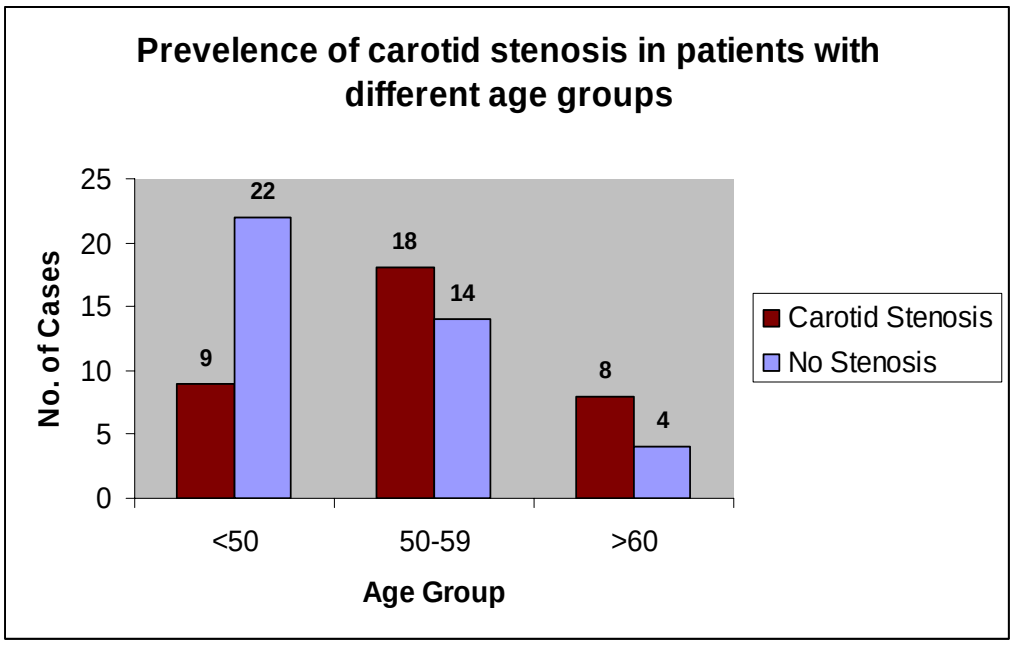


FIGURE 2



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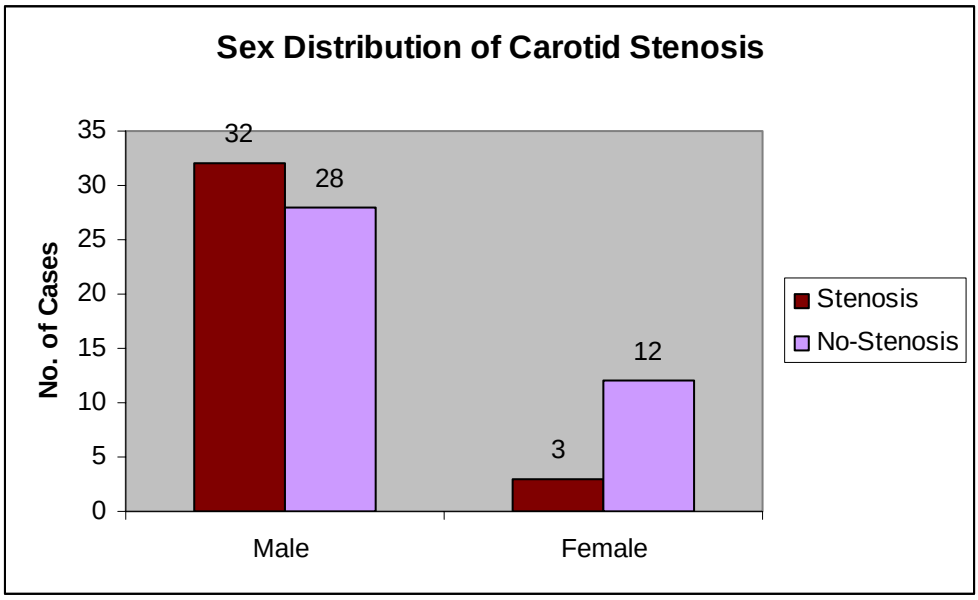

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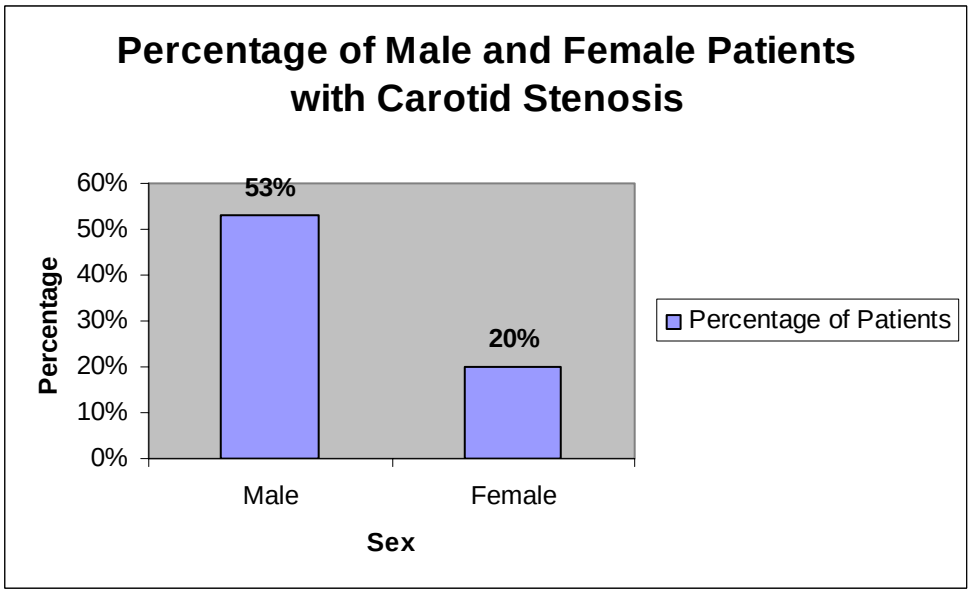

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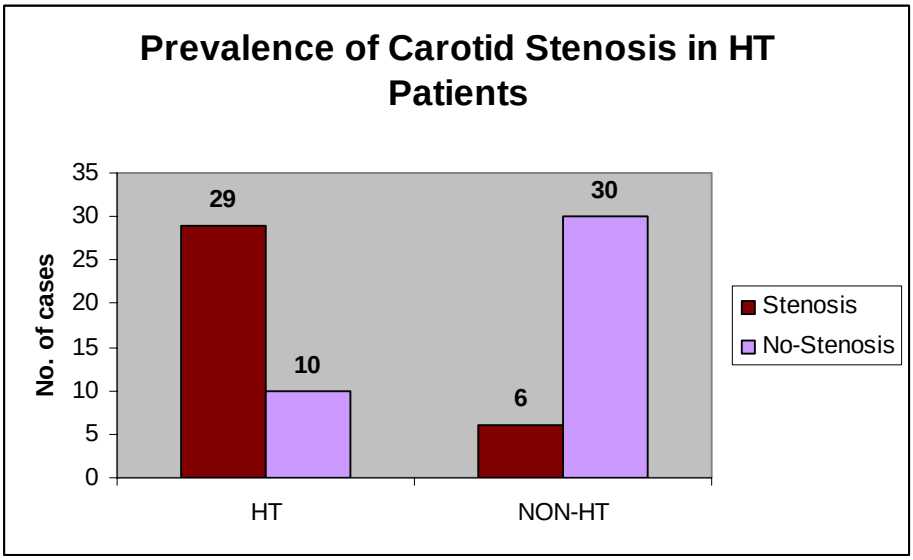

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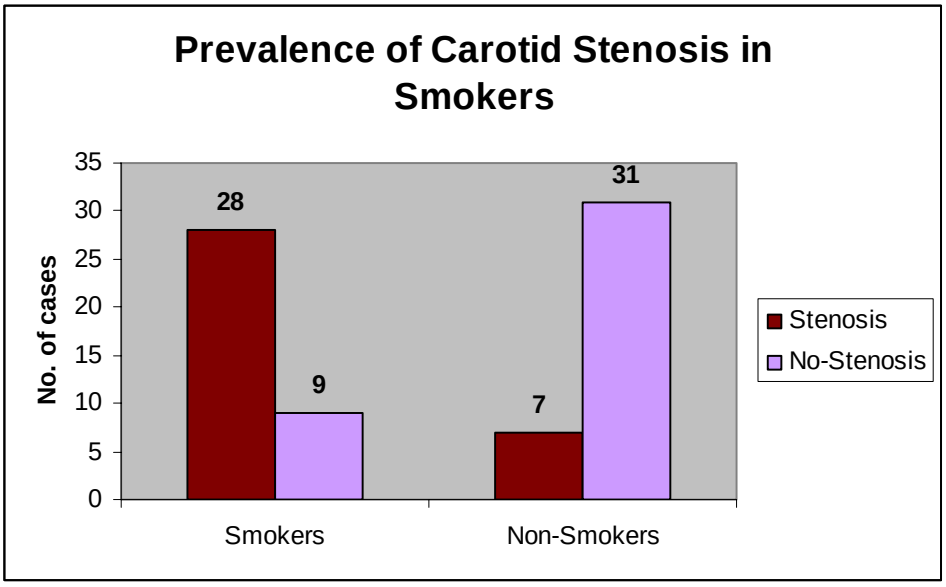

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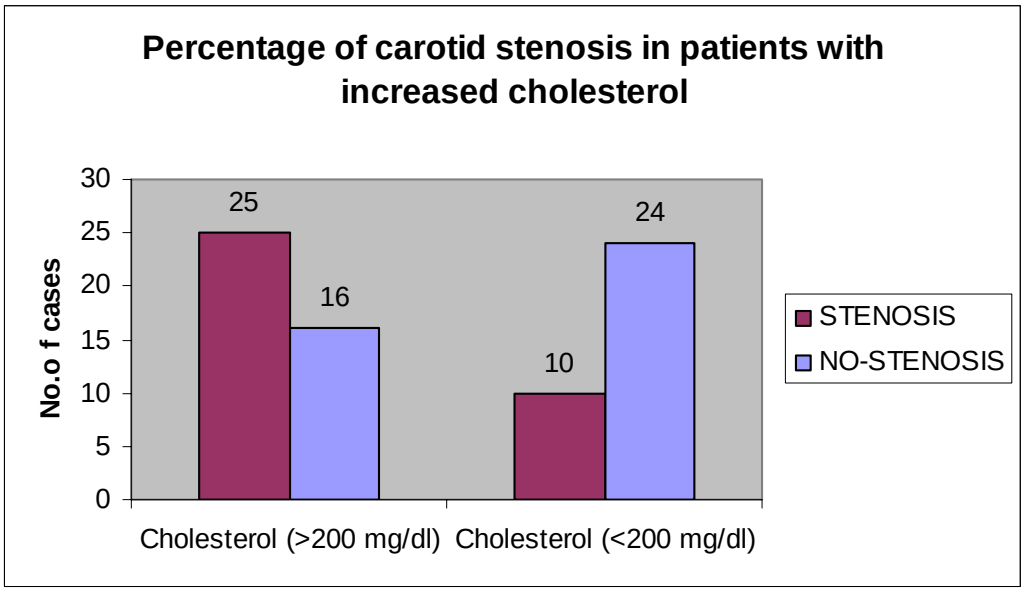

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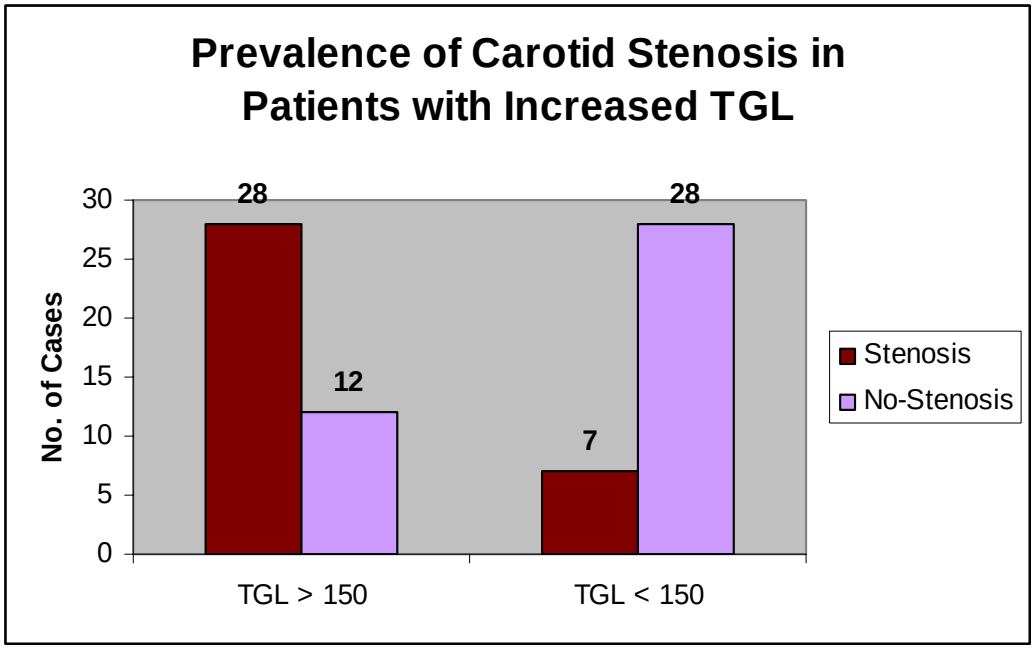


FIGURE 9

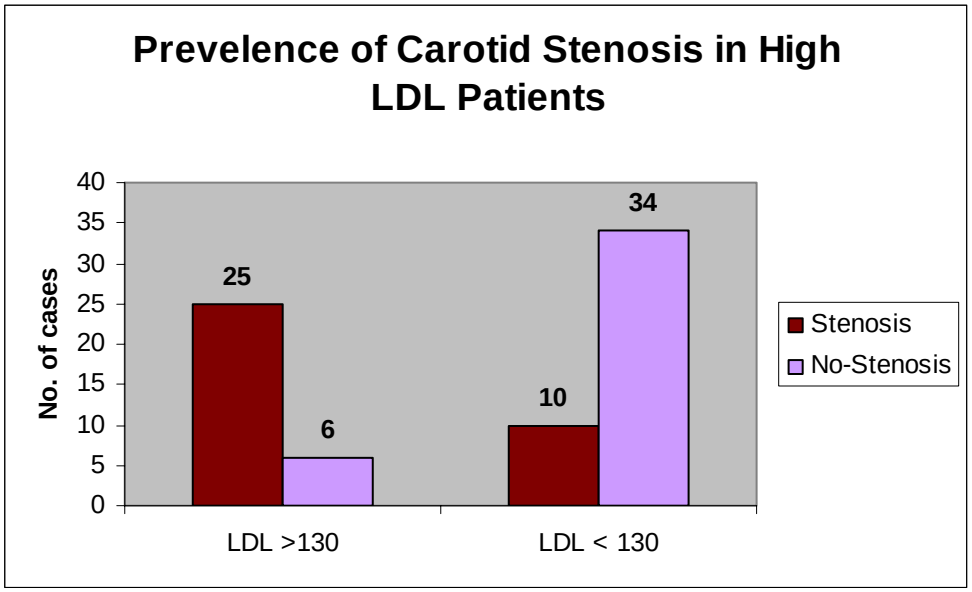


FIGURE 10

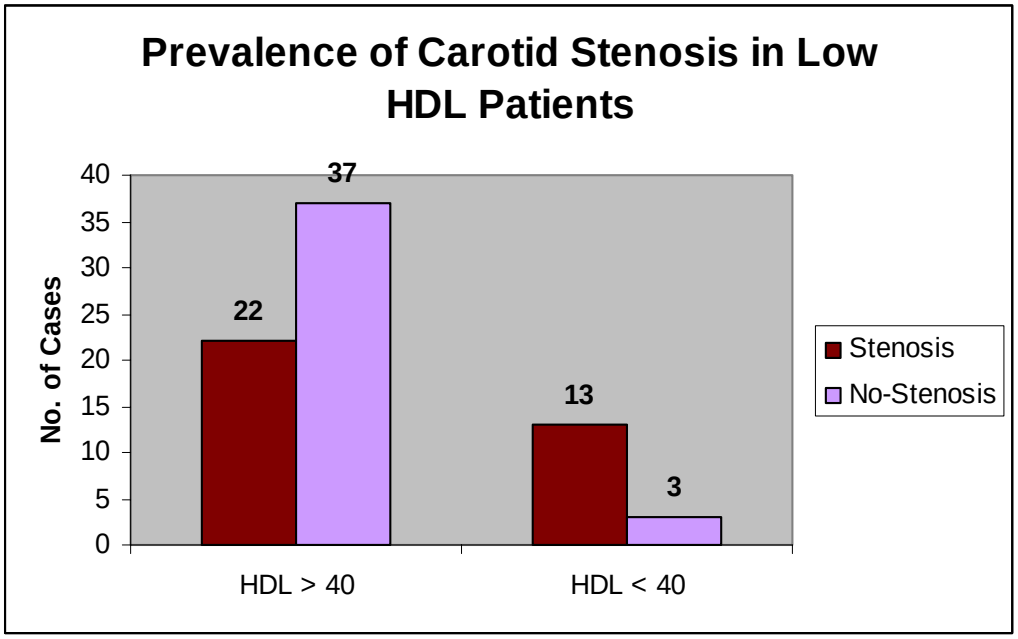


FIGURE 11

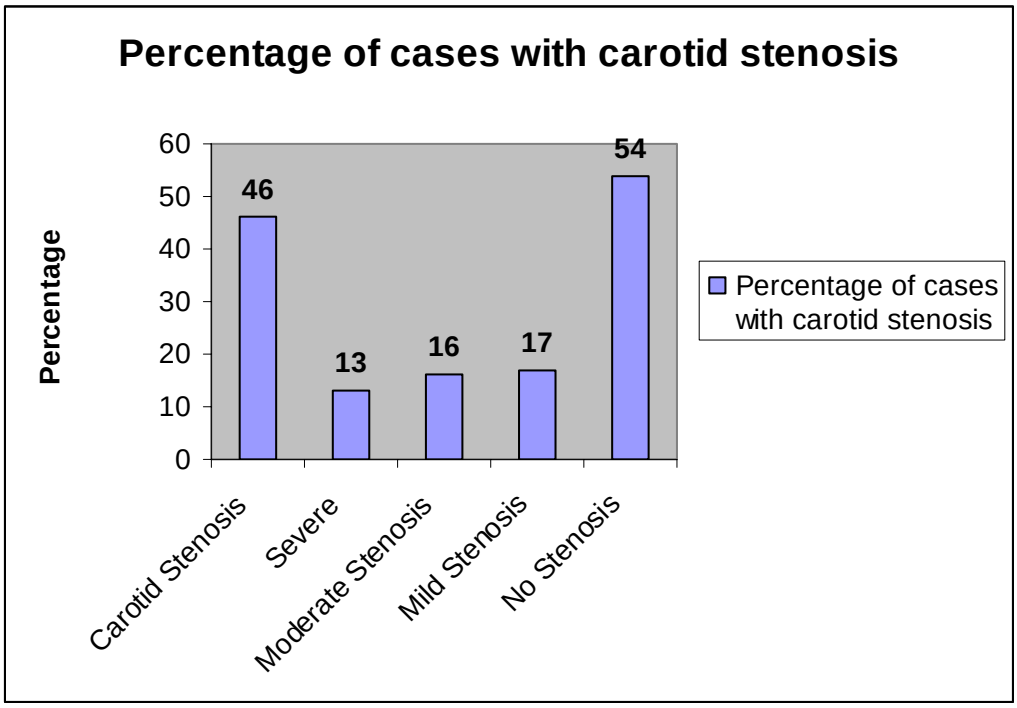


Figure 12

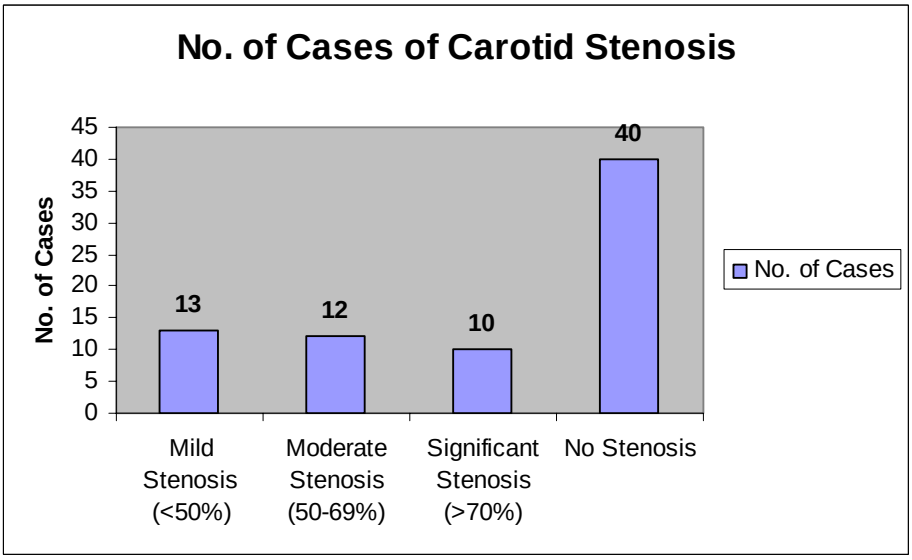


Figure 13

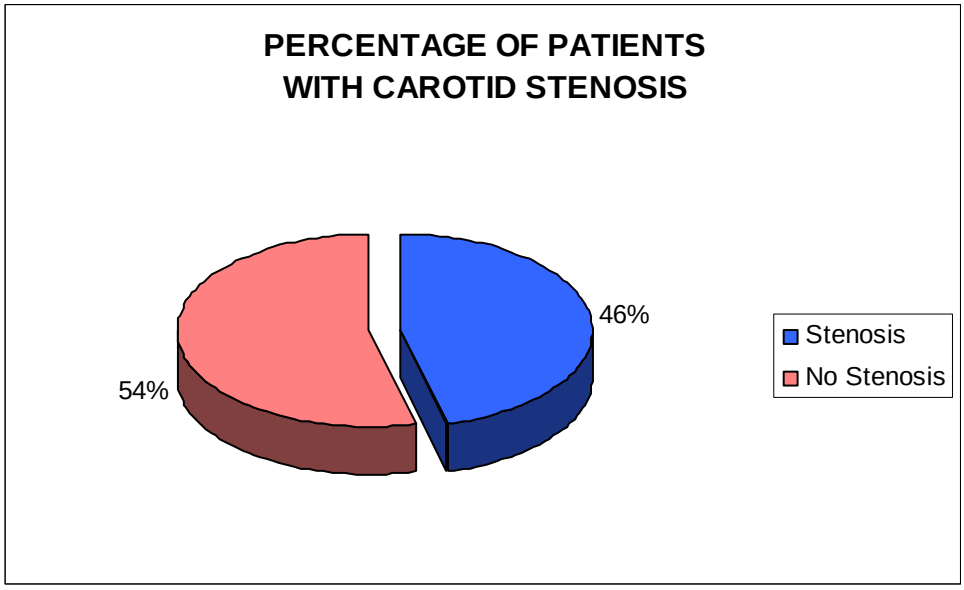
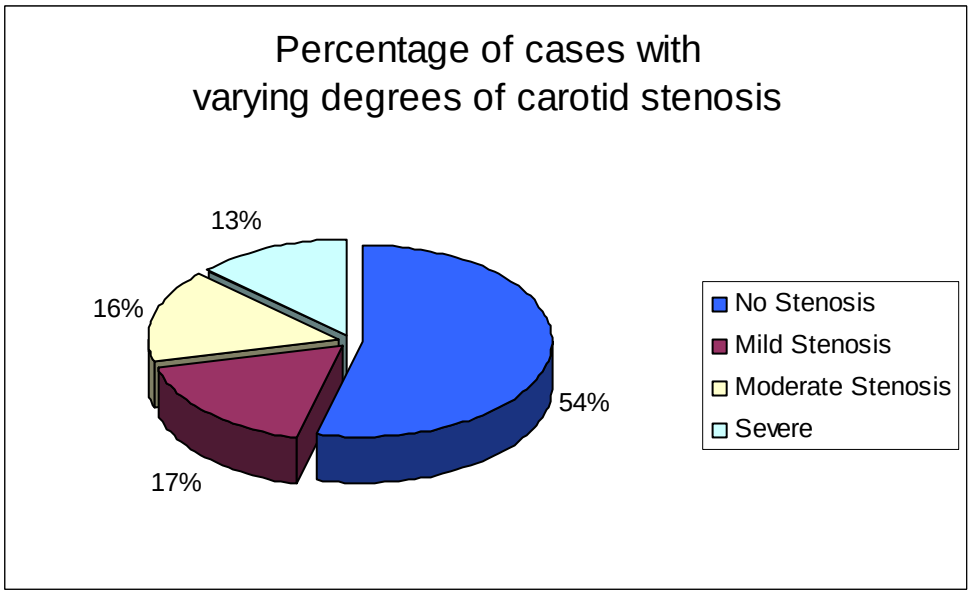


FIGURE 14



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LIST OF TABLES

LIST OF FIGURES

PROFORMA

Name: Age: Sex:

D.O.A.: D.O.D.: IP. No.:

Diagnosis:

DURATION OF ILLNESS:

- H/O weakness:
- H/O slurring of speech:
- H/O deviation of angle of mouth:
- H/O sensory disturbances:
- H/O bladder/bowel incontinence:
- H/O seizures:
- H/O diplopia:
- H/O dysarthria:
- H/O dysphagia:
- H/O dyspnoea:
- H/O LOC:
- H/O headache:

PAST HISTORY:

- DM:
- Hypertension:
- CAD:
- TIA:
- CVA:
- Seizures:

PERSONAL HISTORY:

- Smoking:
- Alcohol:
- Tobacco chewing:

DRUG HISTORY:

- Aspirin:
- Anti coagulants:
- Anti hypertensives:
- OHA:
- OCP:
- AED:

EXAMINATION:

Consciousness:
Orientation:
Pulse:
BP:
CVS:
RS:
P/A:
CNS:
HMF:
Cranial nerves:
V/A:
V/F:
EOM:
Sensation over face:
Seventh nerve Palsy: R: L:
Gaze preference:

SPEECH:

Comprehension:
Fluency:
Word output:
Paraphasias:
Naming:
Reading:
Writing:

Upper limb:

		Right	Left
Motor	Bulk		
	Tone		
	Power		
	DTR		
	Superficial reflexes		
Sensation	Pain		
	Touch		
	Temperature		
	Vibration		

Lower limb:

		Right	Left
Motor	Bulk		
	Tone		
	Power		
	DTR		
	Superficial reflexes (plantar)		
Sensation	Pain		
	Touch		
	Temperature		
	Vibration		
Signs of incoordination			

INVESTIGATION:

CBC:
Hb:
TC:
DC:
ESR:
PCV:
Platelets:
Urea:
Fasting blood sugar:
Creatinine:
Electrolytes: Na: K:
ECG:
Echo:
Chest X-Ray:
Lipid profile:
Total cholesterol:
TGL:
HDL:
LDL:
CT Brain:

CAROTID DOPPLER:

IMT:

	Right	Left
Common carotid		
Internal carotid		

Percentage of stenosis:

	Right	Left
Common carotid		
Internal carotid		
External carotid		

Artery	Right (cm/s)		Left (cm/s)	
	PSV	EDV	PSV	EDV
Common carotid (proximal)				
Common carotid (distal)				
Internal carotid				
External carotid				
Vertebral				

Impression:

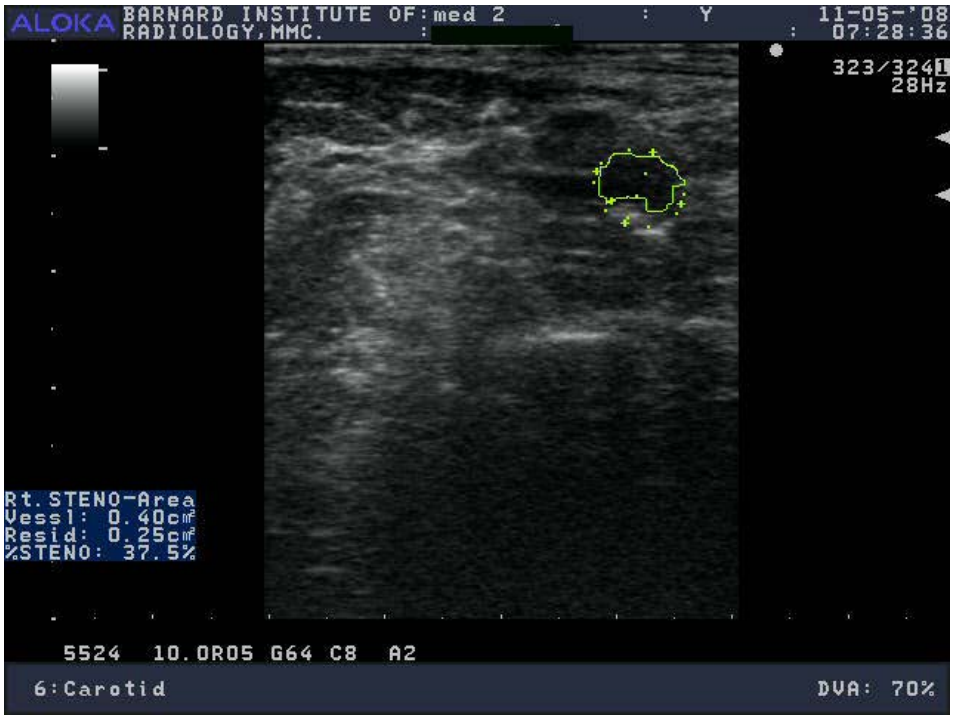
MASTER CHART

S.No	Name	Age	Sex	DM	HT	Smoking	LDL mg/dl	HDL mg/dl	TGL mg/dl	T.Chol mg/dl	Carotid Stenosis				Carotid IMT mm
											Stenosis	Side	Site	Severity	
1.	Purushothaman	54	M	Y	Y	N	143	40	170	217	A				1.1
2.	Sarangan	48	M	N	N	N	181	45	200	266	A				0.7
3.	Sekar	48	M	N	N	N	125	46	122	195	A				1.15
4.	Krishnan	59	M	Y	Y	Y	146	33	134	215	P	R	ICA	Mild	0.6
5.	Chandra Sekar	46	M	N	N	Y	152	42	99	213	A				1
6.	Indrani	66	F	Y	N	N	91	48	141	159	A				0.8
7.	Durai Samy	70	M	Y	Y	Y	134	39	230	210	P	R	CCA	Severe	0.82
8.	Appavu	70	M	N	Y	Y	144	42	170	220	P	R	ICA	Severe	0.65
9.	Kaliammal	55	F	Y	Y	N	122	50	135	199	A				0.80
10.	Antony Samy	58	M	Y	Y	Y	133	42	268	228	P	L	ICA	Severe	1.1
11.	Sekar	45	M	N	Y	Y	139	41	150	210	A				0.8
12.	Lakshmi	49	F	N	N	N	116	43	142	187	A				0.73
13.	Giri	32	M	N	N	Y	130	42	193	210	A				0.4
14.	Majith Bhai	45	M	N	Y	N	146	40	172	220	P	L	ICA	Mild	0.9
15.	Ramesh	32	M	N	N	Y	131	42	180	203	P	L	ICA	Mild	0.95
16.	Elumalai	44	M	Y	N	Y	138	38	150	210	P	L	CCA	Mod	0.6
17.	Krishnan	40	M	N	N	N	121	45	154	192	A				0.8
18.	Jayammal	56	F	Y	Y	N	150	34	184	230	P	R	CCA	Mod	0.7
19.	Thangavel	54	M	Y	Y	Y	152	46	194	236	P	L	CCA	Severe	1.3
20.	Palaiyammal	57	F	N	Y	N	181	47	170	262	P	R	ICA	Mild	0.8
21.	Jeevabagyam	75	M	Y	Y	Y	133	38	196	220	P	R	ICA	Mod	1.1
22.	Sathyamoorthy	74	M	Y	Y	Y	133	47	172	194	P	L	ICA	Severe	1
23.	Rasool	46	M	Y	N	Y	117	41	194	196	A				0.9

S.No	Name	Age	Sex	DM	HT	Smoking	LDL mg/dl	HDL mg/dl	TGL mg/dl	T.Chol mg/dl	Carotid Stenosis				Carotid IMT mm
											Stenosis	Side	Site	Severity	
24.	Kothandam	59	M	N	Y	Y	144	44	210	230	P	L	CCA	Severe	0.9
25.	Pachaiappan	84	M	N	Y	Y	154	36	192	234	P	L	ICA	Mild	2.2
26.	Balamurugan	36	M	N	N	Y	143	43	187	223	P	L	ICA	Mild	0.9
27.	Ross	55	M	Y	Y	Y	75	41	157	143	P	R	ICA	Mod	0.8
28.	Manohari	48	F	Y	N	N	145	36	120	225	P	L	ICA	Mod	1.1
29.	Mani	50	M	N	N	Y	96	42	134	164	A				0.9
30.	Ganesan	47	M	N	N	N	103	33	144	174	A				0.9
31.	Govindasamy	58	M	Y	Y	Y	132	44	174	210	A				0.9
32.	Raju	43	M	N	N	Y	119	45	154	194	A				0.8
33.	Mohan	65	M	N	Y	N	113	47	172	194	A				0.7
34.	Kothandam	52	M	N	Y	Y	130	44	182	210	P	L	ICA	Mod	0.9
35.	Pandurangan	59	M	Y	Y	Y	113	47	184	196	P	L	CCA	Mod	1.1
36.	Jamil Basha	48	M	N	N	Y	111	38	166	182	P	R	ICA	Mod	0.8
37.	Chandra sekar	58	M	N	Y	N	99	44	147	172	A				0.8
38.	Subramani	55	M	N	Y	Y	156	44	172	234	P	R	ICA	Sever	0.7
39.	Munusamy	70	M	N	Y	Y	185	49	164	263	P	R	ICA	Sever	0.7
40.	Govinda Raj	53	M	N	N	Y	63	42	89	112	A				0.7
41.	Sivakumar	49	M	N	Y	Y	106	34	124	174	A				1.5
42.	Munusamy	59	M	N	N	Y	136	37	174	207	P	L	ICA	Mod	1.3
43.	Kaliammal	58	F	N	N	N	100	42	154	172	A				0.9
44.	Fathima Bee	37	F	N	N	N	118	53	96	190	A				0.65
45.	Muniammal	46	F	N	N	N	91	46	237	184	A				1.1
46.	Veera Ragagan	58	M	Y	Y	Y	108	42	120	174	A				0.5
47.	Ettiammal	50	F	N	N	N	113	52	119	188	A				0.9
48.	Muniammal	60	F	N	N	N	110	36	124	178	A				0.8
49.	Senthil Kumar	36	M	Y	Y	Y	149	37	184	232	P	R	CCA	Mild	0.9

S.No	Name	Age	Sex	DM	HT	Smoking	LDL mg/dl	HDL mg/dl	TGL mg/dl	T.Chol mg/dl	Carotid Stenosis				Carotid IMT mm
											Stenosis	Side	Site	Severity	
50.	Kumar	35	M	N	N	Y	120	44	144	192	A				1.2
51.	Kalaiarasi	35	F	N	N	N	95	42	142	163	A				0.7
52.	Thiyagarajan	53	M	Y	Y	Y	163	40	157	232	P	R	CCA	Severe	1.2
53.	Baskaran	42	M	N	N	N	101	45	135	173	A				0.7
54.	Jayakumar	42	M	N	N	Y	110	43	155	182	P	L	CCA	Mild	0.9
55.	Mani	40	M	N	N	Y	87	45	110	145	A				0.8
56.	Parthasarathy	50	M	Y	Y	Y	128	36	154	202	P	L	ICA	Mod	1.5
57.	Renuka	32	F	N	N	N	96	42	132	164	A				0.5
58.	Madhavan	52	M	N	N	N	127	47	144	202	A				0.9
59.	Venugopal	70	M	Y	Y	N	108	40	154	178	P	R	ICA	Mod	1
60.	Mani	40	M	N	N	Y	109	43	162	184	A				0.8
61.	Kannappan	46	M	N	N	Y	98	45	145	172	P	L	CCA	Mild	0.8
62.	Parthasarathy	55	M	N	Y	N	162	37	152	234	P	R	ICA	Mod	1.2
63.	Chinnadurai	59	M	N	N	N	120	51	142	203	A				0.9
64.	Shankar	45	M	N	Y	Y	101	47	144	176	P	R	ICA	Mild	0.8
65.	Govindasamy	56	M	N	Y	N	98	40	132	164	A				0.9
66.	Saravana Perumal	56	M	N	Y	N	84	40	132	150	P	R	ICA	Severe	1.1
67.	Durai Rajan	40	M	N	N	Y	129	44	144	203	A				0.7
68.	Vanniya Nathan	47	M	N	Y	N	90	46	142	164	A				0.8
69.	Unnamalai	58	M	N	N	N	192	40	162	256	P	L	ICA	Mild	0.8
70.	Dharmalingam	55	M	N	N	N	96	43	134	165	A				0.8
71.	Sekar	45	M	N	N	Y	83	43	134	165	A				0.9
72.	Ramasamy	55	M	Y	Y	Y	84	42	146	154	A				0.6
73.	Kumari	54	F	Y	Y	N	84	46	132	156	P	L	ICA	Mild	0.7
74.	Nagarathinam	55	M	Y	N	Y	183	36	269	272	P	L	ICA	Mild	0.8
75.	Periyasamy	70	M	N	N	Y	135	41	146	205	A				0.9

Y – Yes, N – No, P – Present , A – Absent, R – Right, L - Left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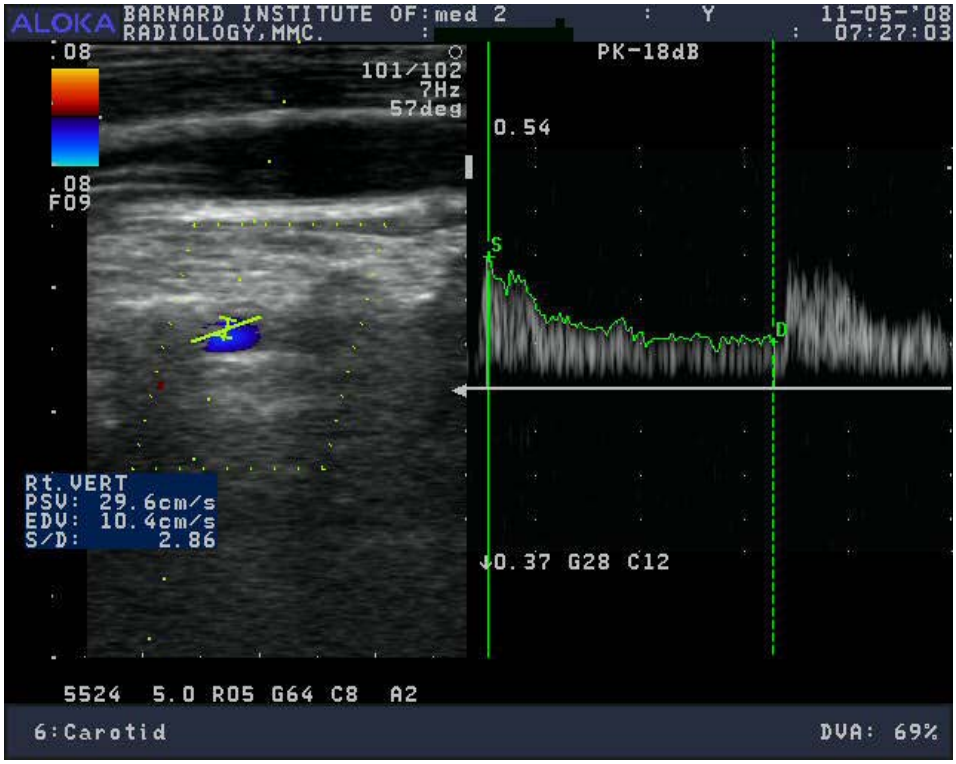
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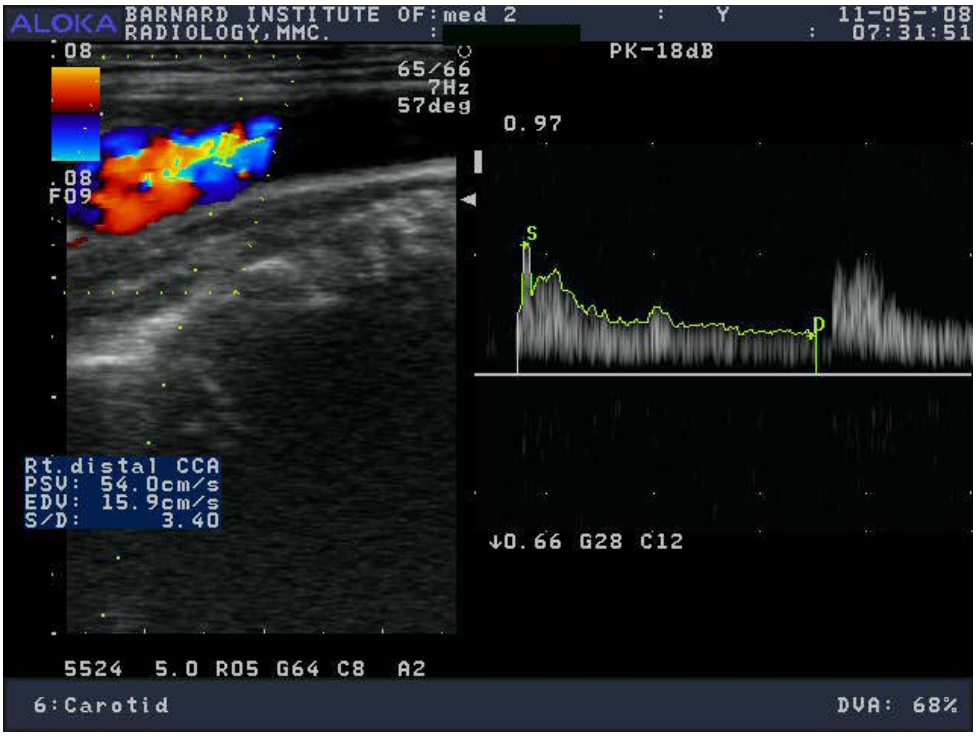
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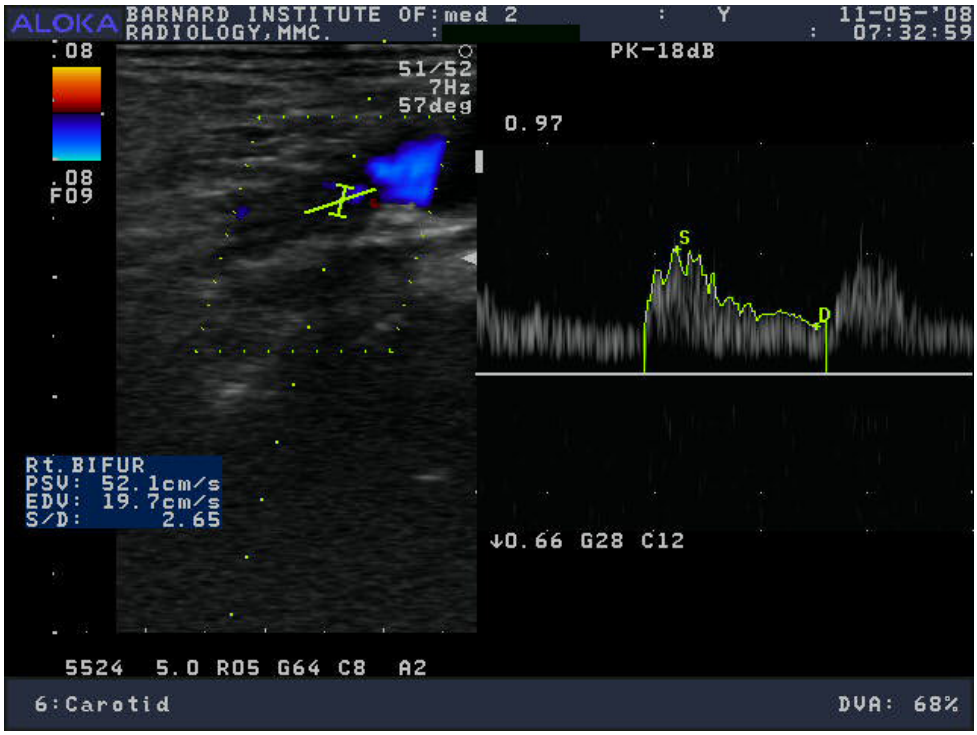
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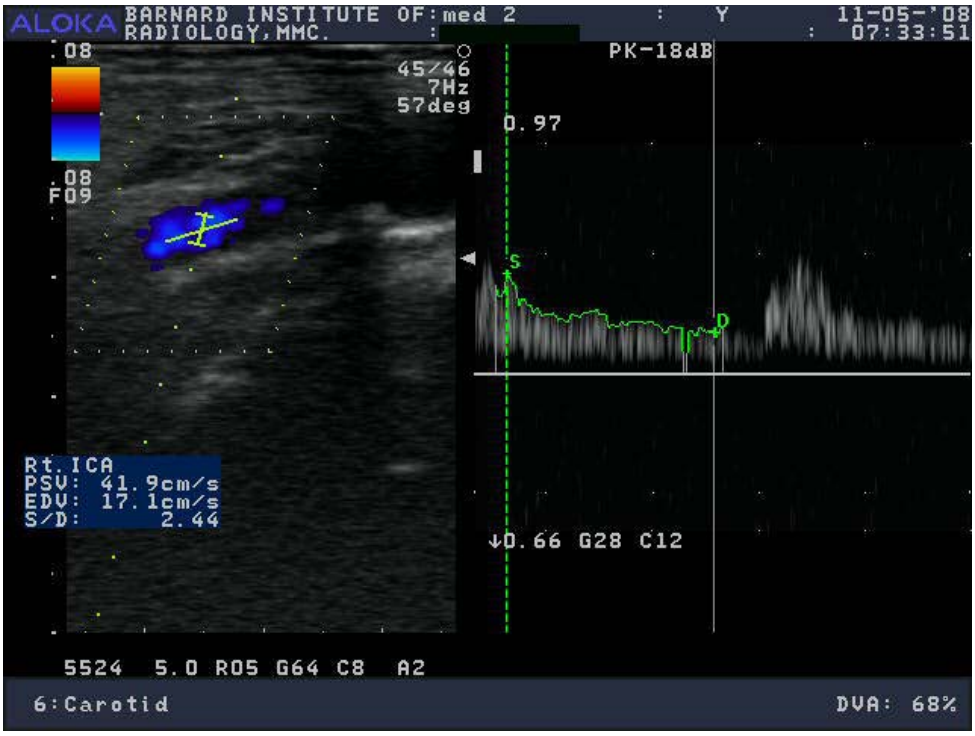
DVA: 68%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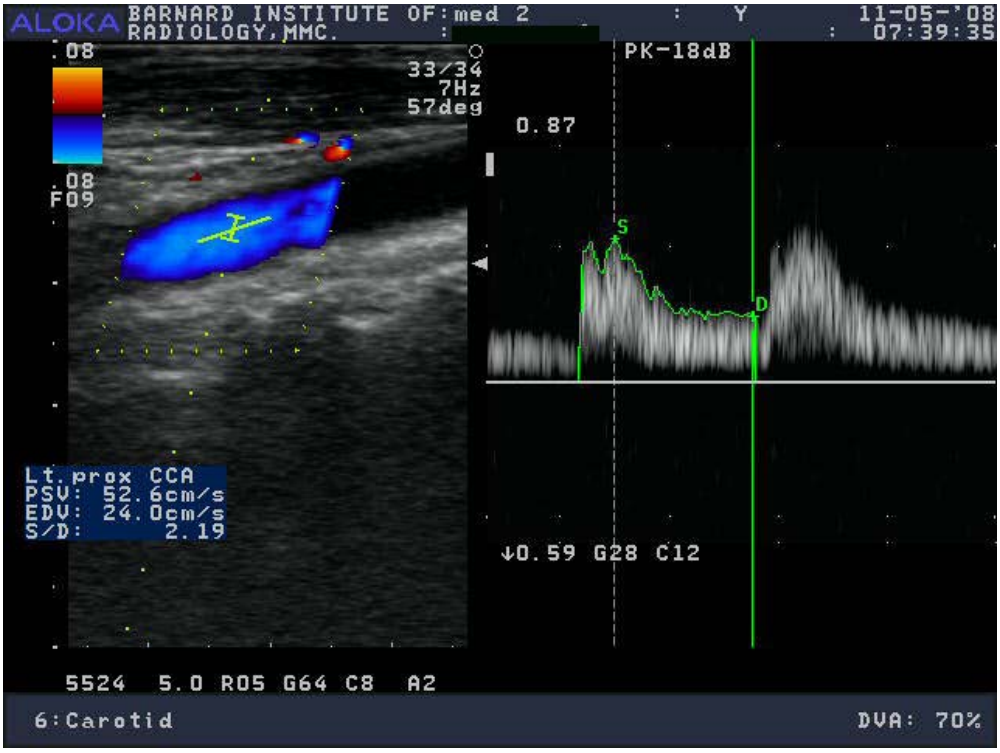
DVA: 68%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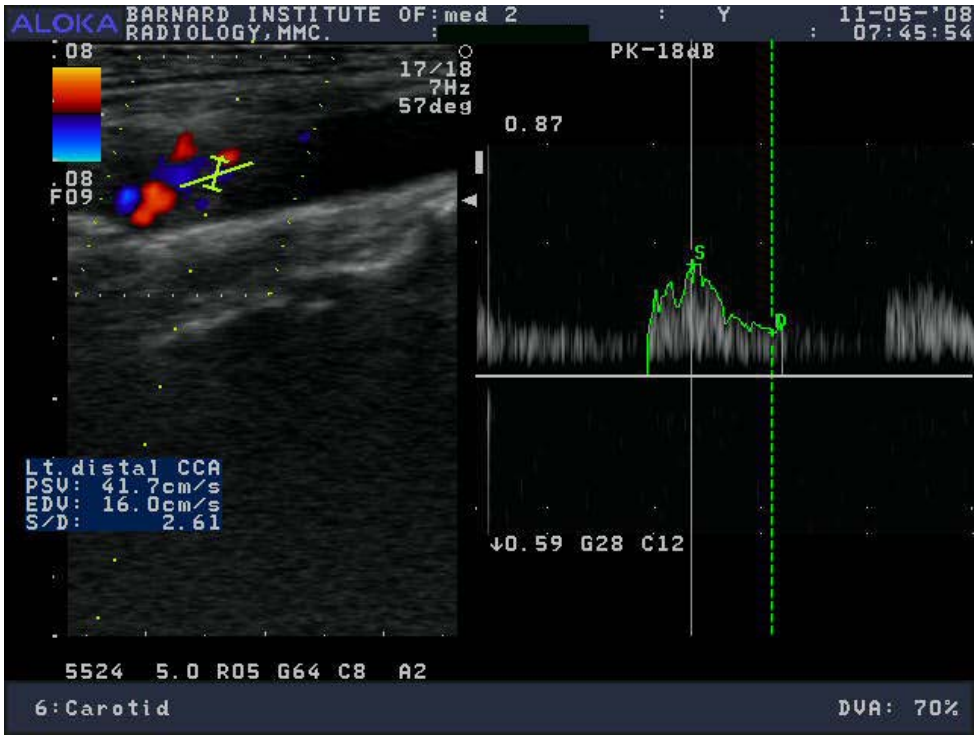
6:Carotid
DVA: 70%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EDV: 16.0cm/s
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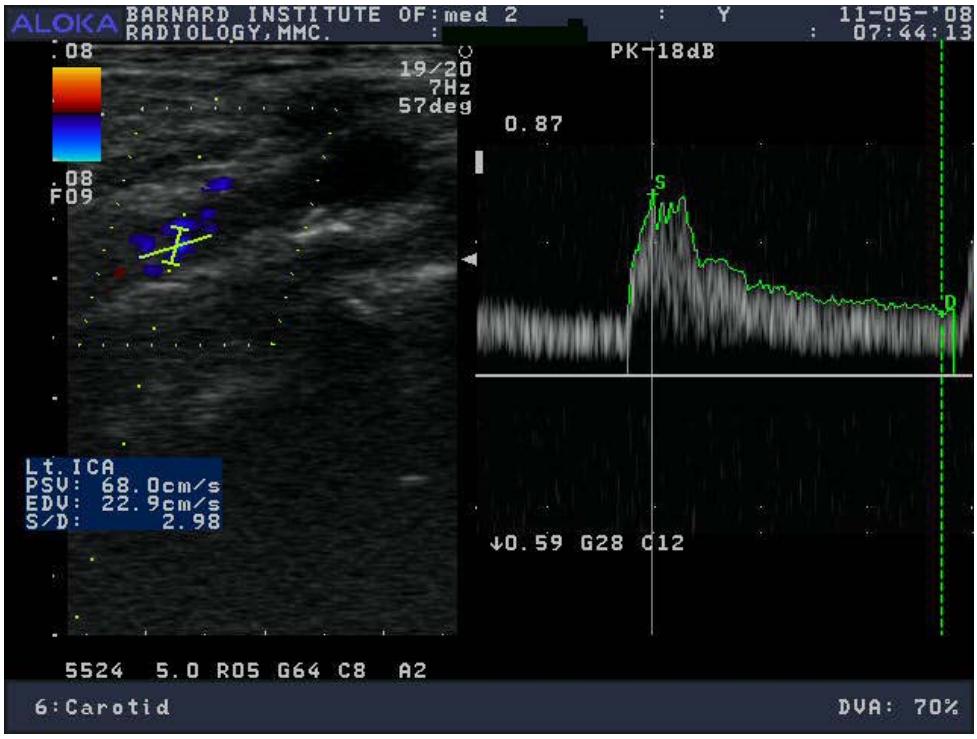
6:Carotid
DVA: 70%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Lt.ICA
PSV: 68.0cm/s
EDV: 22.9cm/s
S/D: 2.98
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6:Carotid
DVA: 70%