CEREBRO VASCULAR ACCIDENTS: RISK FACTOR PROFILE IN MIDDLE AGED AND ELDERLY

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CERTIFICATE

This is certify that dissertation entitled **"CEREBRO VASCULAR ACCIDENTS: RISK FACTOR PROFILE IN MIDDLE AGED AND ELDERLY"** Submitted by **Dr. J. THIRUMALAI RAJAN** to the Tamil Nadu Dr. M.G.R Medical University, Chennai, is in partial fulfillment of the requirement for the award of M.D Degree Branch – I (General Medicine) and is a bonafide research work carried out by him under direct supervision and guidance.

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DECLARATION

This is consolidated report on **"CEREBRO VASCULAR ACCIDENTS: RISK FACTOR PROFILE IN MIDDLE AGED AND ELDERLY"** based on at Govt. Rajaji Hospital, Madurai, during the period August 2005 to August 2006.

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INTRODUCTION

Stroke is a major cause of mortality and morbidity with disability and social dependence. It is the third commonest cause of death after heart disease and cancers[1].

Cerebral infarction is responsible for about 85% of all strokes and primary Intra cerebral hemorrhage for 7%, rest is formed by sub arachnoid hemorrhage and others[2].

Due to severe morbidity and mortality of stroke and limited effective therapies, research has mainly focused on prevention by modification of risk factors.

Though dyslipidemia is a established risk factor in ischaemic heart disease, its relationship with stroke is much weaker if it exists at all some studies have shown that cholesterol is negatively associated with ICH[1].

The commonest cause of death in stroke patients is subsequent coronary events. So cholesterol reduction is helpful in atleast reducing the risk of further coronary events. So lipid estimation is important in stroke patients.

AIM OF THE STUDY

1. To find out the relative frequencies of various risk factors associated with first ever stroke in middle aged and elderly patients.

2. To study the pattern of lipid abnormalities in stroke patients.

REVIEW OF LITERATURE

DEFINITION OF STROKE

A stroke or cerebrovascular accident is a rapidly developing clinical symptoms and / or signs of focal and at times global (applied to patients in deep coma and to those with subarachnoid haemorrhage) loss of cerebral function, with symptoms lasting for more than 24hrs or leading to death, with no apparent cause other than that of vascular origin.

ANATOMY OF CEREBRAL CIRCULATION

ARTERIES OF THE BRAIN

Two internal carotid arteries and 2 vertebral arteries supply the brain. The 4 arteries lie within the substance of subarachnoid space and their branches anastamose on the inferior surface of the brain to form the circle of Willis.

INTERNAL CAROTID ARTERY:

The left common carotid artery arises directly from the aorta and the right common carotid from the innominate artery. The internal carotid artery begins at the bifurcation of the common carotid artery. This ascends the neck and perforates the base of the skull by passing through the carotid canal of the temporal bone. The artery then runs horizontally forward through the cavernous sinus and emerges on the medial side of the anterior clinoid process by perforating the duramater. It now enters the subarachnoid space by piercing the arachnoid matter

and turns posteriorly to the region of the medial end of the lateral cerebral sulcus. Here it divides into the anterior and middle cerebral arteries[4].

BRANCHES OF THE CEREBRAL PORTION

OPHTHALMIC ARTERY:

The ophthalmic artery arises from the internal carotid artery. It enters the orbit through the optic canal below and lateral to the optic nerve. It supplies the eye and other orbital structures and its terminal branches supply the frontal area of the scalp, the ethmoid and frontal sinuses and the dorsum of the nose.

POSTERIOR COMMUNICATING ARTERY:

The posterior communicating artery is a small vessel that originates from the internal carotid artery close to its terminal bifurcation. The posterior communication artery runs posteriorly above the oculomotor nerve to join in posterior cerebral artery to form part of the circle of Willis.

CHOROIDAL ARTERY:

The choroidal artery is a small branch that passes posteriorly, close to the optic tract, enters the inferior horn of lateral ventricle and ends in the choroid plexus. It gives off small branches to the cruscerebri, lateral geniculate body, optic tract and the internal capsule.

ANTERIOR CEREBRAL ARTERY:

It runs forward and medially superior to the optic nerve and enters the opposite side by means of the anterior communicating artery. It curves backward

over the corpus callosum. The cortical branches supply the whole of the surface of the cerebral cortex as far back as the parieto-occipital sulcus. They also supply a strip of cortex about 1 inch wide on the adjoining lateral side. The anterior cerebral artery thus supplies the leg area of the precentral gyrus. A group of central branches pierces the anterior perforated substance and helps to supply the parts of the lentiform and caudate nuclei and the internal capsule.

MIDDLE CEREBRAL ARTERY:

It is the largest branch of the internal carotid artery, runs laterally in the lateral cerebral sulcus. Cortical branches supply the entire lateral surface of the hemisphere, except for the narrow strip supplied by the hemisphere, which are supplied by the anterior cerebral artery, the occipital pole and the inferolateral surface of the posterior cerebral artery. This artery thus supplies the whole motor cortex except the leg area. Central branches enter the anterior perforated substance and supply the lentiform and caudate nuclei and the internal capsule.

VERTEBRAL ARTERY

The vertebral artery, a branch of the first part of subclavian artery, ascends in the neck by passing through the foramina in the transverse processes of the upper 6 cervical vertebrae. It enters the skull through the foramen magnum and pierces the duramater and arachnoid to enter the subarachniod space. It then passes upward, forward and medially on the medulla oblongata. At the lower border of the pons, it joins vessel of the opposite side to form the basilar artery.

BRANCHES OF THE CRANIAL PORTION

MENINGIAL BRANCHES.

These are small branches and they supply the bone and dura and the posterior cranial fossa.

POSTERIOR SPINAL ARTERY

The posterior spinal artery may arise from the vertebral artery or the Posterior inferior cerebellar artery. It descends on the posterior surface of the spinal cord close to the posterior roots of the spinal nerves. Radicular arteries that enter the vertebral canal through the intervertebral foramina reinforce the branches.

ANTERIOR SPINAL ARTERY

It is formed from a contributory branch from each vertebral artery near its termination. The single artery descends on the anterior surface of the medulla oblongata and the spinal cord and is embedded in the piamater along the anterior median tissue. The radicular arteries that enter the vertebral canal through the vertebral foramen reinforce the artery.

POSTERIOR INFERIOR CEREBELLAR ARTERY

This largest branch of the vertebral artery passes on an irregular course between the medulla and cerebellum. It supplies the inferior surface of the vermis, central nuclei of cerebellum and the under surface of the cerebral hemisphere. It also supplies the medulla oblongata and the choroidal plexus of the fourth ventricle.

MEDULLARY ARTERIES.

These are very small branches that are distributed to the medulla oblongata.

BASILAR ARTERY

The basilar artery, formed by the union of the 2 vertebral arteries, ascends in the groove on the anteriors surface of the pons. At the upper border of the pons, it divides into 2 posterior cerebral arteries.

BRANCHES OF THE BASILAR ARTERY

PONTINE ARTERIES.

These are numerous small arteries that enter the substance of the pons.

LABYRINTHINE ARTERY

This is a long narrow artery that accompanies the facial and the Vestibulocochler nerves into the internal acoustic meatus and supplies the internal ear. It often arises as a branch of the anterior inferior cerebellar artery.

ANTERIOR INFERIOR CEREBELLAR ARTERY

This artery passes posteriory and laterally and supplies the anterior and inferior part of the cerebellum, a few branches pass to pons and upper part of the medulla.

SUPERIOR CEREBELLAR ARTERY

This arises close to the termination of the basilar artery. It winds around the cerebral peduncle and supplies the superior surface of the cerebellum. It also supplies the pons, the pineal gland and the superior medullary velum.

POSTERIOR CEREBRAL ATERY

This curves laterally and backwards around the mid brain and is joined by the posterior communicating branch of the internal carotid artery. Cortical branch supply the inferolateral and medial surface of the temporal lobe and lateral and medial surface of the occipital lobe. Thus it supplies the occipital lobe. Central branch pierce the brain substance and supply part of the thalamus, the lentiform nucleus, the midbrain, the pineal gland and the medial geniculate body. A choroidal branch enters the inferior horn of the lateral ventricle and supplies the choroid plexus.

CIRCLE OF WILLS

The circle of Willis lies in the interpeduncular fossa at the base of the brain. It is formed by anastomosis between the two internal carotid arteries and the two vertebral arteries. The anterior communicating, posterior cerebral, and the basilar arteries all contribute to the circle. The circle of Willis allows blood that enters either by the internal carotid or the vertebral arteries to be distributed to any part of either cerebral hemisphere. The cortical and central branches arise form the circle and supply the brain substance.

ARTERIES TO SPECIFIC BRAIN AREAS

Mainly the medial and lateral striate central branch of the middle cerebral artery supply the corpus striatum and the internal capsule; the central branches of the anterior cerebral artery supply the remainder of the structures.

The posterior cerebral, posterior communication and basilar arteries supply the thalamus.

The posterior cerebral, superior cerebellar, and basilar arteries supply the mid brain.

The basilar and anterior, inferior and superior cerebellar arteries supply the pons.

The vertebral, anterior and posterior spinal, posterior inferior cerebellar and basilar arteries supply the medulla oblongata.

VEINS OF THE BRAIN

EXTERNAL CEREBRAL VEINS

The superior cerebral veins pass upward over the lateral surface of the cerebral hemisphere and empty into the superior sagittal sinus. The superficial middle cerebral veins drain the lateral surface of the cerebral hemisphere; it runs inferiorly in the lateral sulcus and empties into the cavernous sinus. The deep middle cerebral vein drains the insula and is joined by the anterior cerebral vein and striate veins to from the basilar vein. The basilar vein ultimately joins the great cerebral vein, which in turn drains into the straight sinus.

INTERNAL CEREBRAL VEINS:

There are two internal veins, and the union of the thalamo striate vein and the choroidal vein at the interventricular foramen forms them. These two veins form the great cerebral vein that drains into the straight sinus.

EPIDEMOLOGY OF CEREBROVASCULAR DISEASES IN INDIA[10]

The burden of stroke on the community is best reflected by its incidence. Two incident studies[5] conducted in the late sixties and early seventies have been reported. The first population-based study was conducted in Vellore in two phases. In the first phase (1968 – 1969) a population of 2,58,576 in and around vellore was surveyed to detect cases of hemiplegia. In the secound phase (1969-1971) the population was kept under surveillance for two years and attempts were made to record all cases of hemiplegia. An incidence of 13/1,00,000 population per year was observed. The secound study was carried out as a part of WHO collaborative study in Rohtak. Haryana between 1971-1974. an annual incidence of 33/1,00,000 population was noted.

In India prevalence vary from region to region, Gowribidanur, and Rohtak had a prevalence of 40-60-1,00,000 population, whereas the Eastern part of India showed a prevalence of 100-270/1,00,000.

Stroke incidence varies among different countries. In WHO Monica project has shown high prevalence and mortality among Finnish population. According to this study incidence of stoke varies from 100/1,00,000 in Germany to 290/1,00,000 population of men in Finland.

CLASSIFICATION OF STROKE[4]

Stroke can be classified in various ways. Suggested classification of stroke

1. ANATOMICAL CLASSIFICATION

A. By vascular supply.

a. Carotid

b. Vertebrobasilar

B. By location

a. Supratentorial

i. Lobar

ii. Ganglionic / thalamic

b. Infratentorial

i. Cerebellar

ii. Brain stem

2. ETIOLOGICAL CLASSIFICATION

A. ischemic

B. Hemorrhagic

a. Intra cerebral hemorrhage

b. Subarachnoid hemorrhage

Ischaemic Strokes

A. with cerebral infarction

1. Cerebral thrombosis with or without atherosclerosis.

2. cerebral embolism (congenital heart disease and acquired vascular disease, cardiomyopathy, myocardial infarction, endocarditis, prolapsed mitral valve etc.)

3. Lacunar Strokes (deep, small cerebral infarcts)

4. Cerebral venous thrombosis and cortical thrombophlebitis.

5. Arteritis (syphilitic, tuberculous, rheumatic, takayasu's disease, collagen dieases etc.)

6. Blood diseases (polycythaemia, sickle cell anaemia, thrombotic states, hyperproteinaemia etc.)

7. Arterial hypotension and anoxic encephalopathy.

8. Dissecting aneurysms of brachio-cephalic vessels.

9. Angiographic complications

10. Infarction of undetermined cause.

B. with cerebral ischamia

1. Transient ischaemic attacks (platelet – fibrin) microemboli associated with atheroma, etc)

2. Arterial hypotension or haemodynamic crisis (e.g. massive gastrointestinal bleeding).

3. With cardiac arrythmias (artial fibrillation, complete heart block, sick-sinus syndrome etc).

4. With migraine.

5. Local embolism from athero-plaque and paradoxical embolism.

6. Undetermined source.

C. Idiopathic and rare types (drugs and oral contraceptives, disseminated intravascular coagulopathy, consumption coagulopathy, cerebral malaria, Behcet's syndrome, congophillic angiopathy, homocystinuria, hyperviscosity syndrome, paraporteinaemia etc.)

Haemorrhagic strokes

1. Hypertensive cerebral haemorrhage.
2. Ruptured aneurysm (saccular, mycotic, etc.)
3. Ruptured angioma (arterial, venous or mixed)
4. Trauma
5. Blood dyscrasias (leukemia, purpura, hyperviscosity syndrome, other bleeding diathesis)
6. Complication of anticoagulant therapy.
7. Bleeding in brain tumours
8. Miscellaneous causes(arteritis, bleeding in haemorrhagic infarct, etc.)
9. From undetermined source.

I. Strokes of undetermined origin

1. Moyamoya disease
2. Fibromuscular dysplasia.
3. Binswanger's Subcortical arteriosclerotic encephalopathy.
4. Winiwater-Buerger disease (thromboangitis obliterans-cerebral form)]
5. Aortic syndrome (non-inflammatory)
6. leukoaraiosis

RISK FACTORS FOR STROKE[11]

They can be grouped into modifiable. Some of them are as follows.

❖ **Age**

The incidence of stroke increase dramatically with advancing age, and increasing age is an important risk factor for stroke.

❖ **Sex**

Male sex is associated with increased risk of stroke.

❖ **Race**

The rate of cerebral infraction is higher in blacks than in whites. This could be partially explained by the higher prevalence of diabetes and hypertension in them.

❖ **Blood pressure**[12]

Hypertension is the single most dominant risk factor for both ischaemic and hemorrhagic stroke. Hypertension predisposes to ischemic stroke by aggravating atherosclerosis. In India, one ICMR multicentric stroke study on risk factors found hypertension, smoking, diabetes and low haemoglobin as risk factors. According to them 40% of stroke can be attributed to systolic blood pressure more than 140mm of mercury. Eastern stroke and coronary heart disease collaborative research found that blood pressure is an important risk factor in eastern Asian population. They found that 3mm of hg reduction in diastolic blood pressure should decrease the number of stroke by about a third. The SHEP study has shown a 36% reduction in non-fatal stroke events over 5 years in the age group 60 years and above with isolated systolic hypertension treated with active medication.

❖ **Diabetes Mellitus[13]**

Diabetes mellitus increases the risk of cerebrovascular disease 2-4 fold compared with risk in nondiabetics[7]. Stroke secondary to diabetes may be caused by cerebrovascular atherosclerosis, cardiac embolism, and rheological abnormalities. Diabetic patients with retinopathy and autonomic neuropathy are particularly at high risk for atherosclerosis.

❖ **Cardiovascular diseases**

Angina or myocardial infarction is clearly associated with stroke. ECG abnormalities reflecting hypertension and coronary artery disease are risk factors for stroke. Rheumatic heart disease and cardiac failure are other risk factors.

❖ **Claudication**

Caludication is a high risk factor for both myocardial infarction and stroke, presumably reflecting atheromatous disease in different parts of the circulation.

❖ **Cigarette smoking**

The relative risk is about 1.5. There is a dose dependent relationship between smoking and stroke. Cigarette smoking is a factor both in men and women. ICMR has shown that smoking is a risk factor both in the young and the elderly.

❖ **Atrial fibrillation**

Most frequent cardiac source of embolism to brain is artial fibrillation[8] either rheumatic or non rheumatic. Atrial fibrillation is associated with increased risk for stroke accounting for 6-24% of all ischaemic strokes.

❖ **Carotid artery disease**

Asymptomatic carotid artery disease less than 75% carries a stroke risk of 1.3% annually. With stenosis greater than 75% combined transient ischaemic attack and stroke rate is 1.5% per year. Carotid and supraclavicular arterial bruit are strongly related to stenosis of underlying arteries.

❖ **Transient ischameic attacks**

A TIA patient has an excess risk of stroke by about 5-10 times greater than a non TIA patient.

❖ **Lipid abnormalities[14]**

Increasing levels of total plasma cholesterol, LDL and decreased levels of HDL are strong risk factors for coronary artery disease than stroke. In a study conducted in NIMHANS they found that low HDL cholesterol is a risk factor in stroke group among various lipid fractions. Lipoprotein ‘a’ has been found to be an independent risk factor for stroke.

❖ **Other risk factors**

Family History, Abdominal obesity, plasma fibrinogen, homocysteine, dietary measures, snoring, cornel arcus, psychological factors, diagonal ear lobe crease, physical inactivity, maternal history of stroke, left ventricular

hypertrophy, peripheral vascular disease, alcohol consumption, oral contraceptives, plasma factor seven coagulant activity, hematocrit etc are some of the risk factors.

PATHOPHYSIOLOGY OF ACUTE ISCHEMIC STROKE:

Cerebral infarction basically comprises two pathophyiological processes.

1.A loss of supply of oxygen and glucose secondary to vascular occlusion.

2. An array of changes in cellular metabolism consequent to the collapse of energy producing processes, with a disintegration of cell membranes.

Normal cerebral blood flow at rest in normal adult brain is 55ml/100g/minute. When the blood flow decreases to 18ml/100g/mt cell death can result. These thresholds mark the upper and lower limits for the ischaemic penumbra. Ischaemic penumbra is the condition of the ischaemic brain between these 2 flow threshold in which there are some neurons that are functionally silent but are structurally intact and potentially salvageable.

The recognition of penumbra has focused attention on the development of treatment to minimize or to even reverse the damage effects of the ischaemic brain by drugs when started within a short period after the occlusion (therapeutic window).

PET studies have detected penumbral tissues up to 72 hours post stroke. It may comprise up to 50% of final volume of infarction up to 17hrs after ischaemic insult and the out come of stroke patient depend upon this proportion.

Ischaemia impairs cellular energy production and depletion of ATP, which is followed by a failure of ATP dependent ion transport system leading to cellular efflux to potassium and influx of sodium and water. This ischaemic depolarization causes presynaptic voltage sensitive calcium channels to open and allow calcium to enter the cell. Intracellular calcium stimulates neural transmitters, most notably excitatory amino acid glutamate. Extra cellular glutamate then activates 2 postsynaptic receptors NMDA receptor and AMPA receptors. Massive release of glutamate causes depolarization of adjacent neurons, triggering further neurotransmitter release increasing their energy requirement and tripping them into energy failure. This is the basis for the so-called excitotoxic cell injury. Intracellular calcium triggers activation of enzymes, which affects the structure and function of cellular constituent. Stimulation of phospholipase leads to an inflammatory reaction through leucotriene production and stimulation of NO synthase, which causes build up of nitric oxide and free redicals.

CLINICAL FEATURES OF STROKE.

It depends upon the site of lesion and type of stroke, ischaemic or hemorrhagic. Distinction between types of stroke is possible by history taking physical examination and investigation.

HISTORY:

a. Onset and progression.

Ischaemic stroke is likely to occur during early morning hour or during sleep, patient may wake up with paralysis. Intracerebral hemorrhage and

subarachiod hemorrhage are likely to occur during strenuous activity. Sudden onset is seen in embolic and hemorrhagic stroke, while onset may be prolonged in thrombotic stroke. Fluctuating course favours ischaemic stroke. If worsening is acted from the beginning, a progressive stenosis of large vessel has to be considered, althought it can be also occur in lacunar infarct. A protracted progression over 2-4 days is usually due to cerebral edema. Progression is usual in hemorrhage.

b. Level of consciousness

stupor and coma is common in hemorrhagic than ischaemic stroke. Hemispheric infarction rarely begins with coma. But patients may become comatose after 24 to 48 hrs

c. Raised ICT features

headache and vomiting are features favouring hemorrhagic stroke than ischemic stroke.

d. Nuchal rigidity.

It is particularly seen in subarahnoid hemorrhage. It can occur in cerebral hemorrhage also.

e. Blood pressure.

A history of hypertension may be there in both ischaemic and hemorrhagic stroke. Both ischaemic and hemorrhagic stroke can cause elevated blood pressure at the time of presentation. A very high diastolic BP at the time of stroke and 24hrs after that favors hemorrhagic stroke.

f. Atheroma markers.

History of diabetes mellitus, coronary artery disease or peripheral vascular disease favors ischaemic stroke very strongly.

g. History of TIA or previous stroke

This favors ischaemic stroke.

h. Heart disease

Atrial fibrillation, Rheumatic heart disease and cardiac failure are often associated with ischaemic stroke.

i. Fundoscopy

subhyloid haemorrhage is highly specific for sub arachniiod haemorrhage. Micoremboli may be seen in carotid stroke.

CLINICAL MANIFESTATIONS OF ISCHAEMIC STROKE

The typical ischaemic stroke presents with abrupt onset of focal neurological deficit and is characterized clinically by the mode of onset and subsequent course.

A transient ischaemic attack is arbitrarily defined as a neurological deficit lasting less than 24hrs (usually 5-20mts). It often portends an impending stroke. The pathophysiological mechanisms for TIA are a low flow in an artery due to a tight stenosis or occlusion or an embolism.

A completed stroke or cerbral infarction typically evolves into maximum deficit in a few hours. It is sometimes heralded by TIA’s.

In stroke in evolution the focal ischaemia worsens from minute to minute or hour to hour. There are usually stepwise incremental increase in neurological deficit occurring over several hours.

The commonest sites of affection are

a. the origin of the ICA within its first 2 cms
b. siphon of ICA
c. proximal segments of MCA and ACA

- **Occlusion of ICA** – Patient may have amaurosis fugax before the occurrence of contralateral hemiparesis.
- **Occlusion of MCA stem - Resulting** in contra lateral hemiplegia, hemianaesthesia and global aphasia (dominant), apraxia and anosognosia (nondominanat).
  - **Occlusion of inferior division of MCA-** Wernickes aphasia without weakness often with superior quadrantanopia. If nondominanat hemisphere, hemi neglect and spatial agnisia.
  - **Occlusion of ACA-** Occlusion of Precommunal segment is well tolerated because of collateral flow. If both post communal segments arise from a single ACA patient suffers profound abulia (delayed motor and verbal response), bilateral pyramidal signs and paraplegia.
  - **Occlusion of PCA[1]-** Occlusion of PCA causes serious neurological defiits as important structures are involved (brain stem). Thalamic

syndrome, thalamoperforate syndrome, Weber's syndrome are some of the known syndromes.

- **Vertebral artery and posterior inferior cerebellar artery lesions-** This includes the well- known lateral medullary syndrome. Medial medullary syndrome causes 12^{th} nerve lesion and contra lateral hemiplegia.
- **Basilar artery**-Complete basilar syndrome results in bilateral long tract signs with variable cerebellar, cranial nerves and other segmental abnormalities of brain stem.
- **Occlusion of superior cerebellar artery**-Ipsilateral cerebellar ataxia, contralateral hemianaestheia, ipsilateral Horner's etc.
- **Occlusion of Anterior inferior cerebellar artery** – Ipsilateral cerebellar ataxia, Ipsilateral Horners syndrome, conjugate gaze palsy, contra lateral loss of pain temperature etc.,

CLINICAL MANIFESTATIONS OF HEMORRHAGIC STROKE

Intracerebral haemorrhage

Common risk factors for intracerebral haemorrhage are hypertension, AV malformation, bleeding diathesis, amyloid angiopathy and drug induced.

Common anatomical sites of ICH are

Putamen 35%

Lobar 30%

Thalamus 20%

Caudate nucleus, Pons, cerebellum & sub thalamus

Putamen –Contra lateral hemi paresis, hemi sensory loss and homonymous hemianopia due to extension to adjacent structures. When hemorrhages are large, drowsiness gives way to stupor and coma, bilateral Babinsky sign and decerebrate rigidity.

Lobar haemorrhage- Amyloid angiopathy causes single and recurrent lobar hemorrhage and is probably the commonest cause in elderly. Most hemorrhages are small and cause restricted clinical symptoms. Large hemorrhages may cause stupor and coma secondary ro compression of the thalamus or midbrain.

Thalamic haemorrhage- It also produces hemiplegia due to pressure or dissection into the adjacent internal capsule. Downward and inward deviation of eyes, convergence retraction and nystagmoid movements, Conjugate gaze palsy etc occurs.

Cerebellar haemorrhage- Variable degree of alertness, skew Deviation of the eye, small reactive pupil, ocular bobbing and cerebellar signs occur.

SUBRACHNOID HEMORRHAGE

Cause of sub arachnoid haemorrhage are ruptured berry aneurysms, AV malformation, extension of ICH, etc

Clinical features of SAH:

- **Before rupture of aneurysm.**

II,III, IV, VI and ophthalmic division of 5th cranial nerves may be involved due to compression by aneurysm of internal carotid artery within the cavernous sinus. Isolated 3rd nerve palsy may be seen with the aneurysm of PCA.

- **After rupture of aneurysm.**

SAH due to rupture of aneurysm often occurs during exertion. Patient presents with headache, altered sensorium, meningial irritation, seizures and rarely focal neurological deficits.

DIAGNOSIS OF STROKE SUBTYPE USING IMAGING

The basic aims of imaging, in patients who have symptoms of stroke are

- To document the presence or absence of haemorrhage. This information is critical as the treatment of the two vary.
- To determine the location and extend of brain damage.
- To asses the current and impending herniation.
- To exclude other entities, which may mimic stroke syndrome
- To find out the cause of stroke.

Acute infarcts are more frequently visible on MRI than on CT scans. On admission approximately 90% of the MRI are positive compared to 60% in CT scans in acute infarcts.

COMPUTERIZED TOMOGRAPY OF ISCHAEMIC STROKE

The CT appearance of cerebral infarction is time dependent. Although the findings may be detected within 6-8 hours of onset, CT may be normal up to 24 hours.

- Hyper acute infarct (less than 12 hours)
    - Normal 50-60%
    - Hyper dense artery 25-50%
    - Obscuration of lentiform unclei
- Acute (12-24 Hours)
    - Loss of grey white interfaces (insular ribbon sign, obscuration of cortex medullary white matter border)
    - Low density basal ganglia.
    - Sulcal effacement
- 1-3 days
    - increasing mass effect.
    - Wedge shaped low-density area that involves both grey and white matter
    - Hemorrhagic transformation may occur.
- 4-7 days
    - Gyral enhancement
    - Mass effect, edema persist

- ❖ 1-8 weeks
    - contrast enhancement persists.
    - Mass effect resolves
- ❖ months to years
    - Encephalomalacic change
    - Volume loss and
    - Rarely calcification.

As mentioned above early finding on CT may be sudden loss of grey white matter contrast and effacement of adjacent subarachnoid spaces. However by 24 hours the abnormal low attenuating areas becomes obvious. Specifically insular ribbon sign has been defined as an early specific sign of MCA infarction. The early findings on noncontrast CT are the result of development of cytotoxic edema. Occasionally CT scan through the supra sellar cistern may show hyper dense MCA indicative of thrombus within the artery. Mass effect and decreased attenuation increases due to combination of cytotoxic and vasogenic edema. Mass effect form cytotoxic edema is maximum between 3-10 days, and may lead to herniation . Mass effect completely resolves by 3 weeks.

Lacunar infarction- These are secondary to arterial disease affecting the deep penetrating vessels of the brain. These arteries may demonstrate tiny foci of stenosis caused by micro atheroma or lipohyalinosis. Because of the small size CT may miss the infarct.

COMPUTERIZED TOMOGRAPHY OF HEMORRHAGIC STROKE

Noncontrast CT scan is always the initial modality of choice for patients presenting with finding suggestive of intracerebral haemorrhage.

- **Subarachnoid haemorrhage-** Plain scan is the modality of choice in acute subarachnoid haemorrhage. It is visualized as an area of increased density within the sucli, cisterns and fissures of brain. In the first 24 hours CT has sensitivity of 90% in detection of subrarchnoid haemorrhage and 50% after 1 week.
- **Intraparenchymal haemorrhage-** Appearance of uncomplicated intracrebal haemorrhage on CT scan is comparatively straightforward. Small petechial hemorrhages or thin linearclots adjacent to calvarium may be difficult to detect. The haematocrit of acute retracted clot is around 90% and the globin content of hemoglobin has high mass density. So the acute intracerebral bleed appears hyper dense on CT. As the haematoma absorbs the margins become fussy and the attenuation of clot progressively decreases, eventually becoming isodense with the brain. The decrease in mass effect takes place more slowly. During this period ring enhancement develops at the original margins of the haematoma and, may persist for months. Chronic haemorrhage is chacterized by hypo density with respect to brain. It will eventually disappear completely leaving a small area of low attenuation or leave a porencephalic cavity isodense with CSF.

- **Hypertensive haemorrhage-** Preferentially involves putamen, thalamus, external capsule, and the pons. Nearly two-thirds of intracerebral haematomas are located in the cerebral ganglia. Clot extension into the ventricular system occurs in about half the cases of hypertensive ICH and is associated with poor prognosis. Lobar white matter haemorrhage is seen in 15-20% of ICH. Some of these spontaneous hemorrhages are due to amyloid angiopathy, but most are hypertensive. Cerebellum is a relatively common site of hypertensive ICH.

MAGNETIC RESONANCE IMAGING OF ISCHEMIC STROKE

Acute infarcts are identified more often and localized more distinctly on MRI as compared to CT. the earliest MRI findings are vascular flow related abnormalities. These include absence of normal flow void and slow flow with intravascular arterial enhancement. These signs can be detected within minutes of symptom onset. Intravascular enhancement is seen nearly in three quarters of acute cortical infarct. Other early MRI findings include hyper intense signal on T2 weighted images, which may not be observed within 8 hours. On both initial and follow up examinations the T1 weighted images are less sensitive. The two earliest signs of acute infarction – intravascular and meningeal enhancement begin to diminish after two to four days. Parenchymal contrast enhancement ensue and may persist for weeks. Edema become more prominent and appears hypo internse on

T1 weighted images and hyper internse on T2 weighted images. Mass effect increases and then gradually decreases.

MAGNETIC RESONANCE IMAGING OF HEMORRHAGIC STROKE

The MRI of intracerebral haemorrhage is a complex and controversial subject. The MRI signals of hyper acute haemorrhage are due to protein rich water content. At this stage clots are typically isointense with grey matter on T1weighted images and hyper intense on T2 weighted images. Acute haemorrhage appears moderately hypo intense on T2 weighted images but isointense on T1. In early sub acute stage of haematoma the clot maybe hypo intense on T2 weighted images while in late sub acute stage becomes hyperintense on T1 and T2 weighted images.

NORMAL LIPID METABOLISM[15]

Lipids in plasma has two sources

a. Exogenous – form diet
b. Endogenous – form liver

Exogenous Tri Acyl Glycerol are carried in chylomicrons. Chylomicrons are formed after a load of Tri Acyl Glycerol is absorbed in the intestine. They are released by the process of reverse pinocytosis and find their way into the lymphatic drainig the intestine. TAG is acted upon by lipo protein lipase (LPL) in the endothelium of capillaries and is converted to free fatty Acid (FFA) and

Glycerol. FFA is either transported to tissues or bound to albumin. Chylomicrons (CM) are converted to CM remnants, which is metabolized in the liver.

Endogenous TAG are carried as VLDL which are systhesised in the liver and secreted into space of Disse. VLDL enters sinusoids. Newly secreted VLDL (nascent VLDL) contains little apolipoproteins C & E. The full component of apo –C and apo-E are taken up by tranfer from HDL. Once VLDL (& CM) have entered the circulation. Lipo protein lipase acts on VLDL to release FFA into tissues and VLDL remnants (IDL) are formed. IDL is mainly converted to LDL small proportion of IDL is taken up by the liver via LDL receptor. Most LDL appears to be formed from VLDL, but a small proportion is secreted by the liver directly.

HDL is synthesized and secreted from both liver and intestine. A major function for HDL is to act as a repository for apo C and apo E that are required in the metabolism of CM & VLDL.

Nascent HDL has discoid PL bilayers containing apolipoproteins and free cholesterol. Lecithin cholesterol acyl Transferase and its activator apo A-I bind to the disc. LCAT converts surface PL ands free cholesterol to lysolecithin and cholesterol Ester. CE moves to the interior of the bilayer. The reaction continues generating a non polar core that pushes the bilayer apart until a spherical, pseudomicellar. HDL is formed. As cholesterol in HDL becomes esterifed, it creates a concentration gradient and draws in cholesterol from tissues and other lipoproteins. It becomes less dense formingHDL 2 which is thought to deliver

cholesterol to liver. Thus HDL features prominently in this Reverse Cholesterol transport, the process by which tissue cholesterol is transported to liver.

Cholesterol Ester tranfer proteins facilitates transfer of CE form HDL to VLDL, IDL , LDL and allow TAG to transfer in opposite direction.

HYPER- LIPOPROTEINEMIAS

A number of diseases cause elevations in the concentration of one or more lipoprotein classes in the plasma. In general, thess abnormalities are detected by the finding of an elevated concentration of Triycerides or cholesterol in the fasting plasma, a condition called hyperlipidemia.

The diagnosis of hyperlipidemia is based on both the clinical features and laboratory findings.

I. clinical features of Hyperlipidemia:

1. Coronary heart disease and peripheral vascular disease
2. Lipid deposition in soft tissues
    - Tendon xanthomata
    - Plamar xanthomata
    - Xanthelasma
    - Corneal arcus
    - Eruptive xanthomata
    - Lipaemia retinalis
    - Acute pancreatitis

II. Laboratory Evidence:

As a working rule, hyper lipoproteinemia is considered to be present.

- When the plama cholesterol exceeds 200 mg/ dl (or)
- When the triglyceride level exceeds 150 mg/ dl

CLASSIFICATION

Hyperlipidemias can be classified into

- Primary, due to hereditary defects in Lipoprotein metabolism.
- Secondary, manifestation of some other conditions

PRIMARY HYPERLIPIDEMIAS

This condition can be classified by the WHO/ Friedrickson system, as given below:

Genetic Disorder	Biochemical Defect	Lipoprotein Elevation	Typical Clinical Findings
Familial Lipoprotein Lipase deficiency	Deficiency of Lipoprotein Lipase	Chylomicron(1)	Eruptive xanthomas, pancreatitis
Familial Lipoprotein CII deficiency	Deficiency of Apo-CII	Chylomicron & VLDL (1 or 5)	Pancreatitis
Familial type III hyper lipoproteinemia	Abnormal apo- E of VLDL	Chylomicron remanants and IDL (3)	Plamar tuberous xanthomas, premature atherosclerosis
Familial hyper cholesterolemia	Deficiency of LDL receptor	LDL (2a, rarely 2b)	Tendon xanthomas, premature atherosclerosis

Familial hyper triglyceridemia	Unknown	VLDL (4, rarely 5)	Eruptive xanthomas, premature atherosclerosis
Multipole Lipo-protein type	Unknown	LDL & VLDL (2a, 2b, 4 rarely 5) hyperlipidemia	

Secondary hyperlipidemias

A number of clinical disorders, produce secondary hyper lipoproteinemias.

Common causes are,

- Diabetes mellitus
- Hypothyroidism
- Nephrotic syndrome
- Alcohol consumption
- Chronic renal failue
- Collagen renal failure
- Drugs
    - Thiazide diuretics
    - Beta blockers
    - Gluco – cortocoids
    - Oral contraceptives

DYSLIPIDEMIA, ATHEROGENESIS AND STROKE[3]

Premature arteriosclerosis may be associated with lipoprotein abnormalities, hyperbetalipoprotinemia is frequently associated with premature atherosclerosis whether genetically determined as in familiar hypercholesterolemia, familial combined hyperlipidemia or polygenichyper cholesterolemia or whether secondary manifestation of hypothyoridism, Nephrotic syndrome or high cholesterol and saturated fat intake.

Increased VLDL level may increase risk for premature atherosclerosis when associated with other risk factors such as diabetes and in patients who smoke and are hypertensive. Recently it has been emphasized that HDL cholesterol seems to confer protection against the development of premature atherscerosis.

There are two important theories putforth for the pathogenesis of Atherosclerosis.

They are **1. LIPID THEORY**

2. The Injury – Healing Theory

Lipid theory Emphasizes the crucial role of lipids particularly cholesterol esters in catalysing atheroma formation. The Injury heading theory stresses the importance of Endothelial damage that triggers an exaggerated proliferative response of myointimal cells from the arterial media. Abnormally high cholesterol esters may damage the arterial endothelium and thereby initiate and sustain proliferative response.

THE ROLE OF DIETARY LIPIDS IN ATHEROSCLEROSIS[3]:

An associatation between elevated plasma levels of cholesterol and dietary increase in cholesterol, saturated fattyacids and reduced polyunsaturated fattyacids has been made to implicate a dietary role in atherosclerosis. To support this argument the dietary moderation is associated with reduced concentration of plasma cholesterol and decreased incidence of coronary artery diseases and stroke due to atherosclerosis. Dietary manipulation to reduce cholesterol and saturated fatty acids and to increase poly unsaturated fattyacids have been found to reduce the stroke rates as shown by SALONEN et al 38% reduction of stroke in men and 50% reduction in women.

HDL's promotion of cholesterol efflux and disposal at the liver appears to be critical in averting Atherosclerosis. This so called **reverse cholesterol transport** to rid of excess tissue cholesterol accomplished this task by the acceptor nature of the protein structure. Critical to this function is APOPROTEIN AI, which may be a more sensitive inverse marker of atherosclerosis. Accelerated atherosclerosis associated with normal cholesterol concentration, but low HDL support the value of this reverse transport system in maintaining cholesterol homeostasis.

In familial hypercholesterolemic patients, cellular receptor for LDL are absent, modified LDL is taken up in this patient cells which assumes cellular supply of cholesterol. But its excess is taken up by monocyte derived macrophages. This scavenging activity may be impaired in stroke patients. In

addition stroke patients circulating monocytes display increased retroendocytosis of LDL cholesterol, which may be highly atherogeinc. With low level of LDL superimposed upon an endothelial injury stimulus such as hypertension, smoking oral contraceptives damage can occur, but healing is expected. On the other hand with elevated level of LDL cholesterol, injurious process is continued resulting in accelerated Atherogenesis.

ROLE OF CHOLESTEROL REDUCTION IN PREVENTING RECURRENT STROKE AND OTHER SERIOUS VASCULAR EVENTS

The relationship between plasma cholesterol and Ischaemic stroke is unclear and it is therefore difficult to predict what lowering plasma cholesterol might do to stroke risk. But on the other hand, a systemic review of the relationship between base line cholesterol and the risk of subsequent coronary heart diseases has shown that the association is strong. Because TIA patients and survivors of Ischaemic stroke are at high risk of coronary events, it seems reasonable to reduce their plasma cholesterol, if not to reduce the risk of stroke, atleast to reduce the risk of coronary events.

However recent trials did show a trend towards a reduction in non fatal plus fatal strokes, which was marginally statistically significant.

It is prudent to advise all survivors of Ischaemic strokes to reduce their dietary intake of saturated fat. The rational for suggesting dietary cholesterol reduction is survivors of Ischaemic stoke and TIA is simply that their cerebrovascular events immediately identifies them as being at higher risk for all

vascular events including coronary events than normal people. The result of the **SCANDENAVIAN SIMVASTATIN SURVIVAL STUDY GROUP** suggest that any patients with a history of Ischaemic stroke or TIA and a previous myocardial infarction and a cholesterol level more than 200 mgs % is likely to benefit from cholesterol reduction with a statin.

MATERIALS & METHODS

This study was conducted in 40 ischaemic and 15 Hemorrhagic CVA patients who were admitted in Dept of medicine of Govt. Rajaji Hospital, Madurai. 50 Age-Matched, Sex-Matched persons were taken as controls

Design of the study :- Prospective Analytical study

Period of Study:- 1yr (August 2005 to August 2006)

Ethical Approval :- Obtained

Consent :- Informed consent was obtained from the subjects

Selection of patients:-

A total number of 55 patients (30 males, 25 Females) with age ranging from 40 to 90 yrs were included.

In ischaemic CVA group, the patients presenting with acute hemiplegia with or without aphasia of more than 24hrs duration with CT evidence of hypodense lesion (more the 1.5cms) were included.

In hemorrhagic CVA group, the patients presenting with Acute hemiplegia with or without aphasia with or without loss of consciousness of more than 24hrs duration with CT- evidence of hyperdense lesion were included.

50 (26 males, 24 Females) age matched and sex matched non diabetic, non hypertensive, non smoker, non alcoholic were taken as controls.

Exclusion criteria :-

Patients presenting with stroke with the following conditions were excluded from this study.

1. Recurrents stroke, stroke in less than 40 yrs of age
2. Systemic illness likely to cause stroke (Ex. Vasculitis, hematological disorders, infection)
3. Female patients on OCP
4. Drug abuse
5. brain stem stroke
6. stroke due to causes like trauma, brain tumour

This patients were evaluated on the basis of proforma on the following guidelines. Hypertension was defined as systolic BP >140mm of hg, diastolic BP> 90mm of hg or both on 2 separate occasions or the use of anti hypertensive medication at any time before the onset of stroke. Patients were diagnosed as diabetic if fastic blood glucose level were 126 mg/ dl or higher after an overnight fast on more than one occasions or random glucose level 200 mg/dl or higher on more than one occasion patients was also labeled as diabetic on the history of diabetes confirmed in patient's medical record or the patients was taking insulin or on oral hypoglycemic agents. Cut off values for each class of lipids were taken as abnormal as follows.

Total cholesterol	:	> 200mg%
LDL cholesterol	:	>130 mg%

Trig lycerides	:	>150 mg%
HDL cholesterol	:	<40mg%
T.C / HDL Ratio	:	<4.5
Normal BMI[19]	:	<23
Normal W/H Ratio Males	:	1.0
Female	:	0.8

A Detailed clinical history and neurological Examination was done. Routine investigations like blood Hb/ sugar urea, creatinine, urine analysis, TC, DC, ECG were done. CT Brain was taken in all patients. ECHO was done only in thrombotic stroke group populations. Carotid artery Doppler study was done in selected 15 thrombotic group patients only.

COLLECTION OF BLOOD SAMPLES FOR LIPID ANALYSIS:

All the blood samples were taken after 12hrs fasting. This was drawn from the cubital vein, serum separated and the lipoprotein analysis was done within 6hrs of withdrawal of blood.

Enzymatic assay CHOD PAP method was used for estimation of cholesterol and TGL. Calculation of LDL-C by FRIEWALDS FORMULA

$$\text{LDL in mg\%} = \text{T.C} - \text{HDL} - \frac{\text{TGL}}{5}$$

STATISTICAL ANALYSIS

The collected data was analysed using Epidemiological Information package – 2002 developed by centres for disease control an prevention, Atlanta in collaboration with World Health Organisation. Chi – Square test used for tests of

significance. These data was compared with published literature in consultation with guide a multivariant analysis.

LIMITATIONS OF STUDY

1) Total cases are smaller in number

2) Carotid artery Doppler study cann’t be done in all cases.

RESULTS AND OBSERVATION

In our study 55 stroke patients (40 ischemic, 15 hemorrhagic) and 50 controls were taken and studied.

In the study population age group range from 61.6 ± 6.8

In the control population age group range from 59.9 ± 7.7

Total Stroke cases (A)
- A1 = Ischemic stroke group
- A2 = Hemorrhagic stroke group

B = control group

Abnormal lipid level cut offs[16]:-

T.C	>	200mg%
TGL	>	150mg%
HDL	<	40mg%
LDL	>	130mg%
T.C / HDL Ratio	>	4.5

Table: 1

Age Group	A1		A2		Total A		B	
	No	%	No	%	No	%	No	%
40 – 49	2	5	1	6.7	3	5.5	6	12
50 – 59	7	17.5	7	46.7	14	25.5	15	30
60 – 69	25	62.5	6	40	31	56.4	25	50
70 to above	6	15	1	6.7	7	12.7	4	8
Total	40	100	15	100	55	100	50	100
Mean	62.6		59.1		61.6		59.9	
SD	6.7		6.3		6.8		7.7	

Total no of patients = 55. Majority of patients in both study and control group lie between 60 and 69yrs. Almost same age group of patients were selected in both groups (P = (0.1068)).

Table:2

Sex	A1		A2		A		B	
	No	%	No	%	No	%	No	%
Female	18	45	7	46.7	25	45.5	24	48
Male	22	55	8	53.3	30	545	26	52
P value for	A1 & A2 = 0.8466 A1 & B = 0.9435 A2 & B = 0.8383							
	A2 & B = 0.9479							

Total No of patients = 105

Male = 56

Female = 49

Both sexes are equally distributed in the study and control population.

Table: 3

TC	A1		A2		A		B	
	No	%	No	%	No	%	No	%
Normal	18	45	9	60	27	49.1	48	96
Abnormal	22	55	6	40	28	50.9	2	4
Mean	209.1		197.1		205.8		164.3	
SD	41.2		48.2		43.1		24.6	

P for 1. A & B = 0.0001 – Significant
2. A1 & B1 = 0.0001 – Significant
3. A2 & B = 0.0013 – Significant

The mean cholesterol was increased above the normal range in ischemic stroke group patients

(Statistically Significant – $P < 0.0001$).

Table: 4

TGL	A1		A2		A		B	
	No	%	No	%	No	%	No	%
Normal	15	37.5	7	46.7	22	40	36	72
Abnormal	25	62.5	8	53.3	33	60	14	28
Mean	184.3		182.3		183.7		157.8	
SD	81.3		74.5		78.8		42.6	

'P' value between

A & B = 0.0019 – Significant

A1 & B = 0.0022 – Significant

A2 & B = 0.1317 Not Significant

Mean TGL was increased above the control group in both the type of strokes.

Table: 5

HDL	A1		A2		A		B	
	No	%	No	%	No	%	No	%
Normal	18	45	2	13.3	20	36.4	30	60
Abnormal	22	55	13	86.7	35	63.6	20	40
Mean	38.1		36.7		37.7		41.3	
SD	5.3		12		7.6		7.2	

A & B = 0.0259 – Significant

A1 & B = 0.8137– Not Significant

A2 & B = 0.004 Significant

Mean HDL was decreased in both the types but statistically Significant in hemorrhagic stroke group (P Value = 0.004)

Table: 6

LDL	A1		A2		A		B	
	No	%	No	%	No	%	No	%
Normal	14	35	9	60	23	41.8	47	94
Abnormal	26	65	6	40	32	58.2	3	6
Mean	134.4		125.7		132		95.1	
SD	37		36.3		36.7		23.1	

'P' value between

A & B = 0.0001 – Significant

A1 & B = 0.0001 – Significant

A2 & B = 0.0033 – Significant

Mean LDL was increased in ischemic stroke group stroke patients (P value< 0.0001)

Table: 7

VLDL	A1	A2	A	B
Mean	36.7	36.5	36.6	28.1
SD	15.6	15	15	8.6

Table: 8

TC/HDL Ratio	A1		A2		A		B	
	No	%	No	%	No	%	No	%
Normal	6	15	2	13.3	8	14.5	40	80
Abnormal	34	85	13	86.7	47	85.5	10	20
Mean	5.4		5.3		4.1		4.1	
SD	1.2		1.2		0.9		1.9	

'P' value between

A & B = 0.0001 – Significant

A1 & B = 0.0001 – Significant

A2 & B = 0.0001 – Significant

Mean TC/HDL Ratio was increased in both the types of stroke

(Statistically significant P = 0.0001)

Table: 9

DM	A1		A2	
	NO	%	No	%
Present	11	27.5	2	13.3
Absent	29	72.5	13	86.7
+				
Total = 23%				

Table: 10

HT	A1		A2	
	No	%	No	%
Yes	24	60	10	66.7
No	16	40	5	33.3
+				
Total = 62%				

Table 11

IHD	A1		A2	
	No	%	No	%
Yes	24	60	6	40
No	16	40	9	60
+				
Total = 54%				

Table 12

History of TIA

History of TIA	A1		A2	
	No	%	No	%
Yes	4	10	0	0
No	36	90	15	100
+				
Total = 7.2%				

Table 13

Smoking	A1		A2		A		B	
	No	%	No	%	No	%	No	%
Yes	17	42.5	7	13.3	24	43.6	-	-
No	23	57.5	8	86.7	31	56.4	50	100
+								
43.6%								

Table 14

Alcoholism	A1		A2	
	No	%	No	%
Yes	15	37.5	7	46.7
No	25	62.5	8	53.3
+				
Total = 40%				

Table 15

Family History

Family History	A1		A2	
	No	%	No	%
Yes	4	10	1	6.7
No	36	90	14	93.3
+				
Total = 9%				

AF was present in 7.3% and Heart Failure in 7.2%

>70% Stenosis of carotid artery was found in 2 patients of the selected 15 ischemic stroke patients.

Table 16

Correlation between Lipid Abnormalities and sex

Ischemic stroke patients

Sex	Total patients	T.C	TGL	HDL – C	LDL - C
Male	22	13	13	9	14
Female	18	9	12	13	12

Hemorrhagic Stroke patients

Sex	Total patients	T.C	TGL	HDL – C	LDL - C
Male	8	2	4	6	2
Female	7	4	4	7	4

Table 17

Correlation between lipid Abnormalities and age group

Ischemic stroke patients

Age Group	Total patients	T.C	TGL	HDL – C	LDL - C
40-49	2	-	1	-	1
50-59	7	4	4	5	5
60-69	25	16	16	12	17
70 and above	6	4	4	5	3

Hemorrhagic Stroke patients

Age Group	Total patients	T.C	TGL	HDL – C	LDL - C
40-49	1	-	1	1	-
50-59	7	3	4	5	3
60-69	6	3	3	6	3
70 and above	1	-	-	1	-

Table 18

Risk factors	A1					A2				
	Abnormal cases in					Abnormal cases in				
	TC	TGL	HDL	LDL	TC/HDL	TC	TGL	HDL	LDL	TC/HDL
Smoking(22)	9	10	9	11	13	2	4	7	2	5
Alcoholism (22)	7	7	6	8	12	2	4	5	2	5
DM (13)	8	8	7	8	10	-	1	1	1	2
HT (34)	24	17	14	18	21	4	6	9	4	10
IHD (30)	14	16	12	15	19	-	2	5	1	6
TIA (4)	3	2	2	3	3	-	-	-	-	-
Family History (5)	3	2	1	3	3	1	1	1	-	1
AF (4)	2	4	-	2	4	-	-	-	-	-
Heart Failure (4)	3	3	3	4	4	-	-	-	-	-

DISCUSSION

A complete analysis of 40 ischemic stroke patients, 15 hemorrhagic stroke patients and 50 control patients was done.

Ischemic group = Males 22

Females 18

Hemorrhagic Group = Males 8

Female 7

Controls ---------- Males 26

Females 24

Ischemic stroke constituted 72.3% of cases, Hemorrhagic stroke 27.7% including 2 SAH

In Men

Ischemic stroke Constituted 73.3% of cases, hemorrhagic stroke 26.6% including one SAH

In Women

Ischemic Stroke = 72%

Hemorrhagic Stroke = 28% including one SAH

There findings age almost comparable with that of liaquat[17] a, All Rajehs et al studies. In this study hypertension was the most common risk factor for stroke. 62% were hypertensive. Among these patients 60% were known HT. Only 30% of these known HT were taking anti hypertensive drugs on regularbasis while

remaining 70% had poor compliance. The remaining 40% patients were diagnosed HT after frequent recording of BP.

This finding is comparable with Basu[20] AK et al study where they have found HT in 87.5% patients.

40% Patients has BMI> 23 while 36% had W/H Ratio abnormally BMI > 23 is at risk weight for developing cardio vascular diseases.

Smoking was the risk factor in 43.6%. This is higher than that of liaquat[20] A study (21%).

Alcoholism was present in 40% of patients.

Ischemic heart diseases was present in higher percentage of patients (54%). In Basu[20] AK et al study it was 35%.

Diabetes was present in 23%. This is almost comparable with that of Basu AK et al study. The frequency of intra cerebral hemorrhage is low in diabetic as compared to ischemic stroke.

History of TIA was present in 7.2% of cases while family History was positive in 9% of cases.

AF was present in 7.3% while Heart failure was present in 7.2%. Both the group developed ischemic strokes and they recovered well. >70% carotid artery stenosis was present in 2 of the 15 selected ischemic stroke patients. One patient showed both hypercholesterolemia and hypertriglyceridemia and the other showed hypertriglyceridemia alone

Lipid abnormalities in ischemic stroke group Vs controls:

T.C

T.C was increased in 22 out of 40 patients (55%) with thrombotic stroke in contrast to 2 patients in the control group (4%) and the average T.C was 209.1±41.2 in contrast to controls (164.3±24.6)

In garg RK[21] et al study the mean total cholesterol was 172.05±40.68% in contract to controls (151.85±32.98 mgs%)

TGL

TGL was increased in 25 out of 40 patients (62.5%). Mean TGL value is 184.3 in contrast to controls in whom the value was 137.8. In garg et al study the values were 162.26, 107.50 for cases and controls respectively.

HDL – Cholesterol

HDL was decreased in 22 of 40 thrombotic cases. The mean value were 38.1 and 41.3 in cases and controls respectively.

LDL – Cholesterol

LDL was increased in 26 patients (65%). The mean value were 134.4, 95% respectively.

This is supported by garg et al[21] study and Basu[20] et al study.

VLDL –Cholesterol

The mean VLDL value were 36.7 and 28.1 mg% in cases and control respectively

T.C/ HDL Ratio

The mean value were 5.4 and 4.1 cases and controls respectively.

In conclusion there was a significant increases in T.C TGC, LDL, TC/ HDL ratio in ischemic stroke group patients[24,25].

Lipid abnormalities in hemorrhagic group Vs controls

T.C

T.C was increased in 6 out of 15 pts. (40%). The mean value were 19.7 and 164.3 in cases, controls, respectively.

TGL

The Mean values

Cases = 182.3

Controls = 137.8

TGL elevated in 8 patients (53%)

HDL

Mean value were 36.7 and 41.3 in cases, control, respectively.

HDL was decreased in 13 out of 15 pts (86%)

LDL

Mean value

Cases = 125.7

Control = 95.1

VLDL

Mean values were 36.5 and 28.1 in hemorrhagic group and control group respectively.

T.C/ HDL Ratios:

Average value were 5.3±1.2, 4.1±0.9 in case, and controls respectively.

In conclusion, we found HDL is reduced significantly in hemorrhagic group patients (86%)[23].

CORRELATION BETWEEN OF OTHER RISK FACTORS AND LIPID ABNORMALITIES

In age group most of the lipid abnormalities are found in 60-90 group in ischemic stroke patients.

In hemorrhagic stroke patients the numbers are almost same in 50-59 and 60-69 age groups.

According to sex distribution, more females showed low HDL in both ischemic and hemorrhagic stroke group than males.

Among diabetics (13 out of 55) in ischemic group (11 out of 40) , 8 patients showed elevated T.C, 8 showed elevated TGL, 8 showed elevated LDL values. 7 patients showed decreased HDL values.

In hemorrhagic group (2 out of 15) 1 patients showed elevated T.C, 1 showed elevated LDL and 1 showed decreased HDL.

Among Hypertensives (34 out of 55) in ischemic stroke (24 out of 40) 24 showed increased T.C, 17 showed increased TGL, 17 showed increased LDL. 14 patients showed decreased HDL values.

In hemorrhagic group (10 out of 15) 4 patients showed elevated T.C, 6 showed elevated TGL, 4 showed elevated LDL. 9 patients showed decreased HDL values.

Among IHD patients (30 out of 55) in ischemic group, lipid abnormalities were almost similar in number. Bur in hemorrhagic stroke group (6 out of 15) patients, the predominant abnormality is low HDL values.

17 out of 40 patients in ischemic stroke group were smokers. 9 of them showed decreased HDLvalues and 11 showed increased LDL values. Out of 5 smokers in hemorrhagic stroke group 7 showed decreased HDL value and 2 showed elevated LDL values. In alcoholics also LDL values are more abnormal than the HDL values.

CONCLUSION

The following conclusions were derived from the study

1. Main risk factors for stroke are hypertension, diabetes mellitus, hyperlipidemia, smoking and obesity. Stroke can be prevented by modification of these risk factors. There is a genuine need for health education programmes on stroke.
2. Hyper cholesterolemia is a risk factor in ischemic Stroke patients.
3. Low HDL values were found to be associated with hemorrhagic strokes.

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CEREBRO VASCULAR ACCIDENTS: RISK

FACTOR PROFILE IN MIDDLE AGED & ELDERLY

NAME:

AGE: SEX:

IP NO: OCCUPATION:

HEIGHT: WEIGHT:

BMI: WAIST/HIP RATIO:

HISTORY:

ONSET:

NEROLOGICAL DEFICIT:

ASSOCIATED SYMPTOMS:

H/O HEADACHE H/O SEIZURES H/O VOMITING

H/O LOC H/O TRAUMA H/O SUGGESTIVE OF CARDIAC AETIOLOGY

PAST HISTORY:

H/O DM H/O HT H/O IHD

H/O PT H/OTIA

PERSONAL HISTORY:

DIETARY HABITS:

SMOKING:

ALCOHOLISM:

FAMILY HISTORY:

GENERAL EXAMINATION ;

VITAL SIGNS:

PULSE:

BP: RR:

CNS:

- GLASCOW COMA SCALE
- CRANIAL NERVE PALSY
- HEMIPLEGIA / HEMIPARESIS
- HEMIANAESTHESIA
- MENINGEAL SIGNS

CVS:

RS:

ABDOMEN:

INVESTIGATIONS:

URINE ALB: SUGAR: DEP:

BLOOD TC: DC: Hb %:

BLOOD UREA: mg/dl SUGAR: mg/dl

SERUM CREATININE: mg/dl

SERUM ELECTROLYTES:

ECG:

CHEST XRAY PA VIEW:

LIPID PROFILE:

SERUM CHOLESTEROL:

TRIGLYCERIDES:

HDL CHOLESTEROL:

LDL CHOLESTEROL:

VLDL CHOLESTEROL:

CT BRAIN:

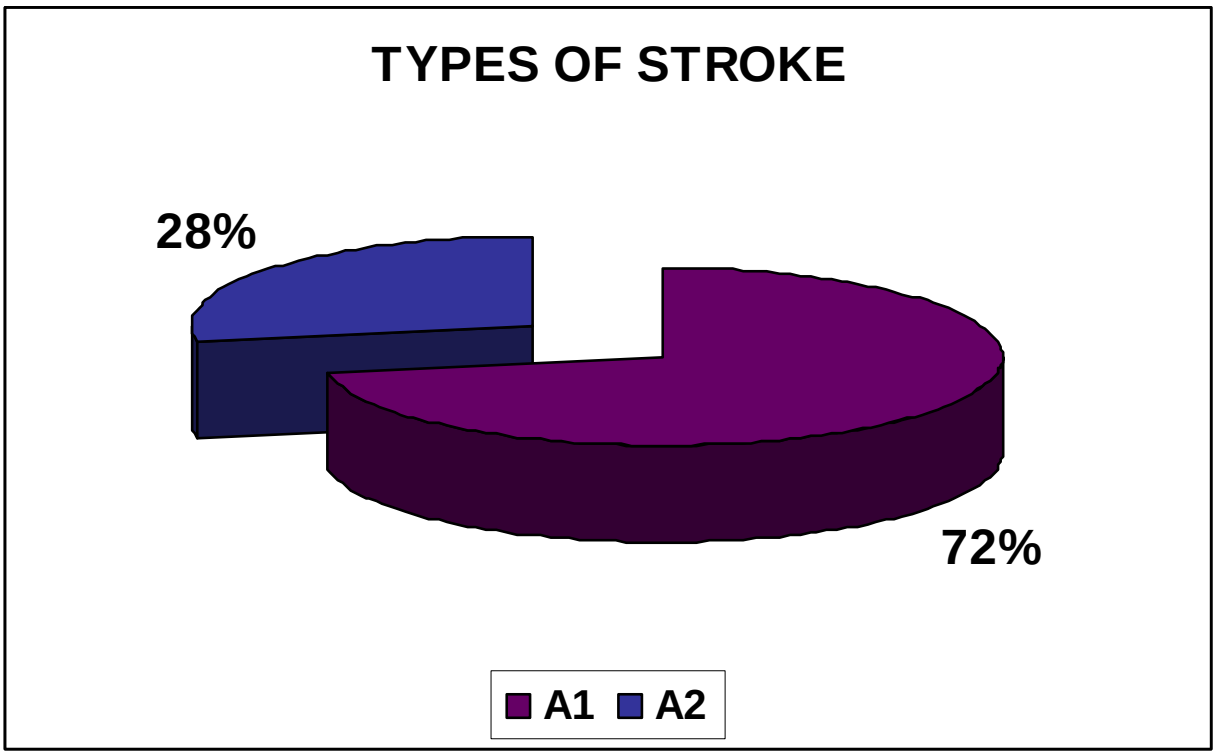
SPECIAL INVESIGATIONS:

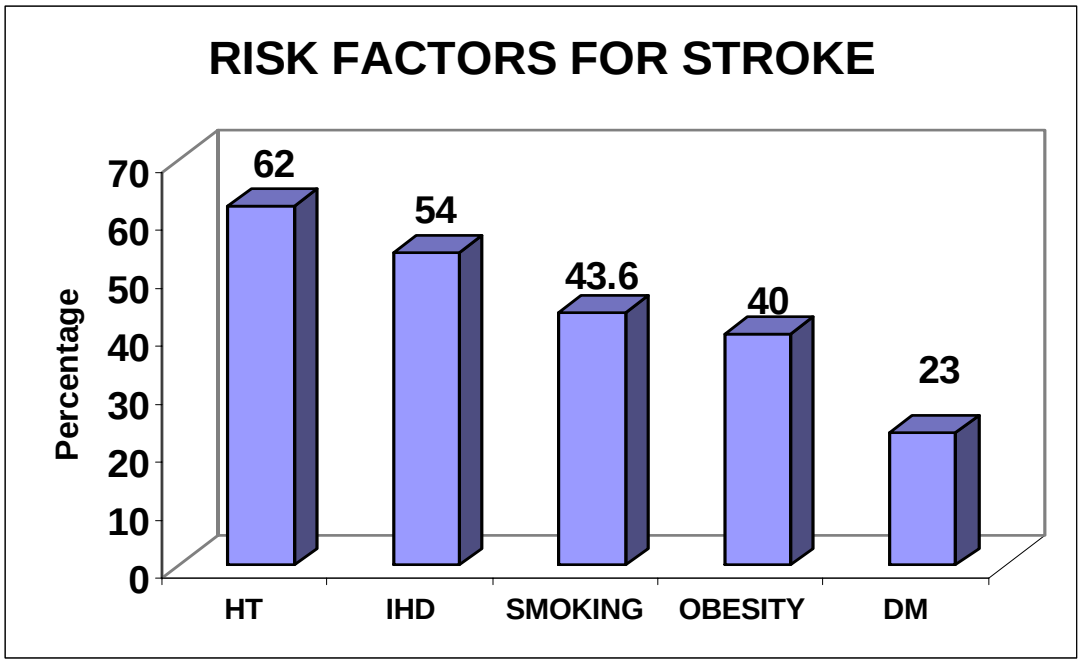
ECHO:

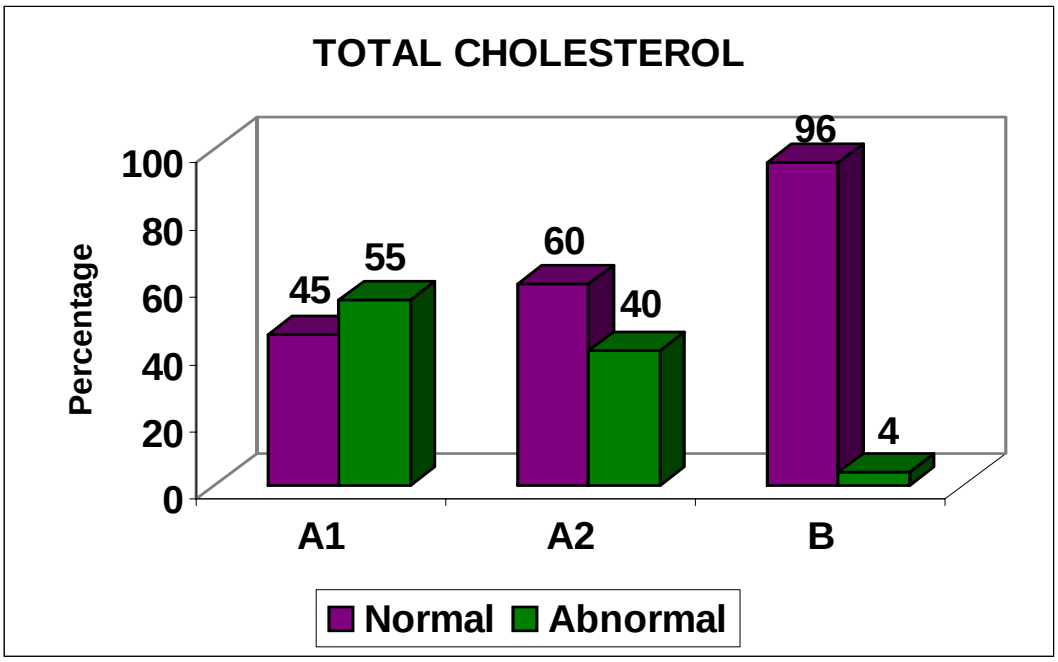
CAROTID ARTERY DOPPLER:

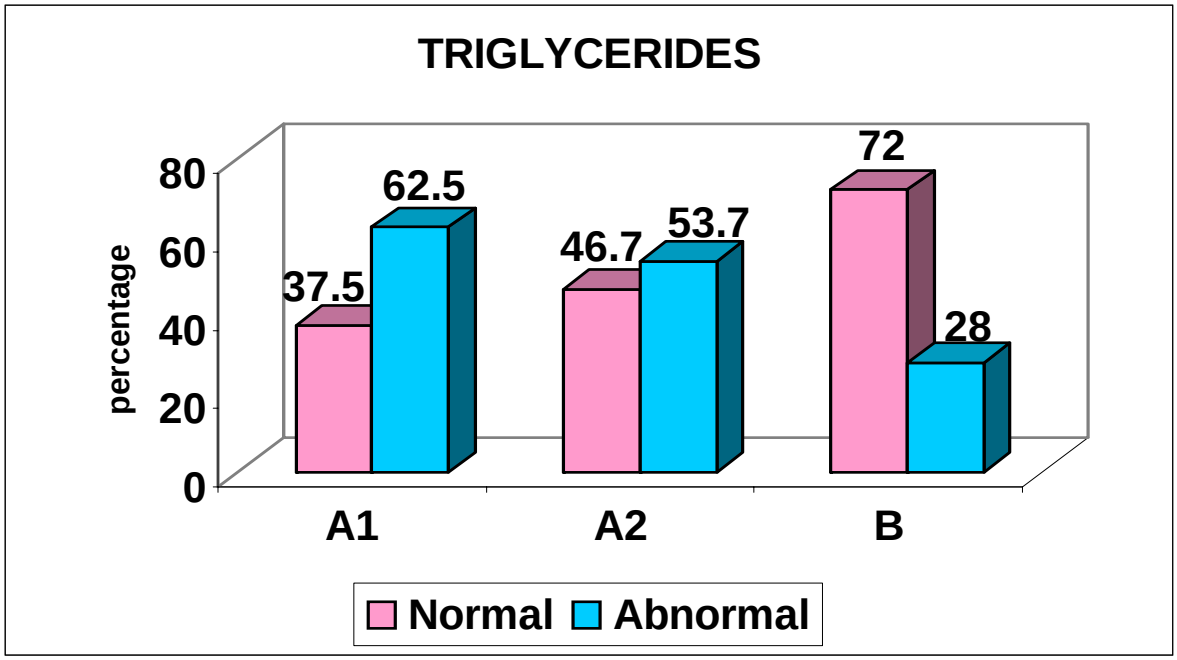
ABBREVIATIONS

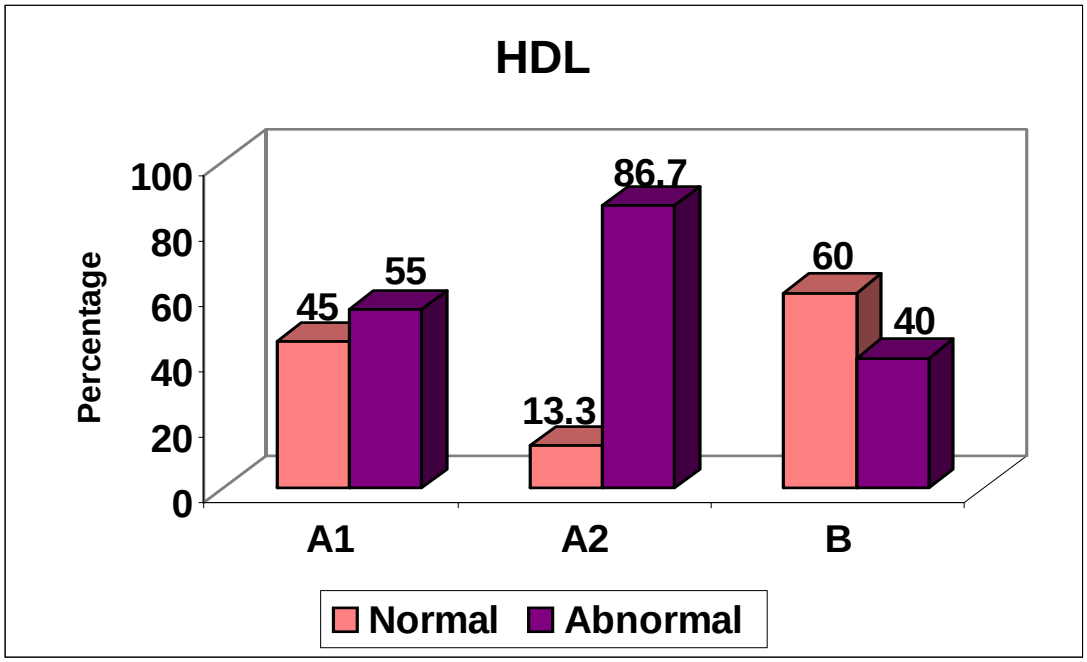
CVA	-	Cerebro vascular Accidents
TIA	-	Transient Ischemic attacks
CT	-	Computed Tomography
TC	-	Total Cholesterol
TGL	-	Triglycerides
HDL	-	high density Lipoproteins
LDL	-	Low density Lipoproteins
VLDL	-	Very low density Lipoproteins
HT	-	Hypertension
DM	-	Diabetes Mellitus
BMI	-	Body Mass Index
W/H Ratio	-	Waist/Hip Ratio
IHD	-	Ischemic heart disease
AF	-	Atrial Fibrillation
CM	-	Chylo Microns
OCP	-	Oral contraceptive pills
SAH	-	Sub arachnoid hemorrhage

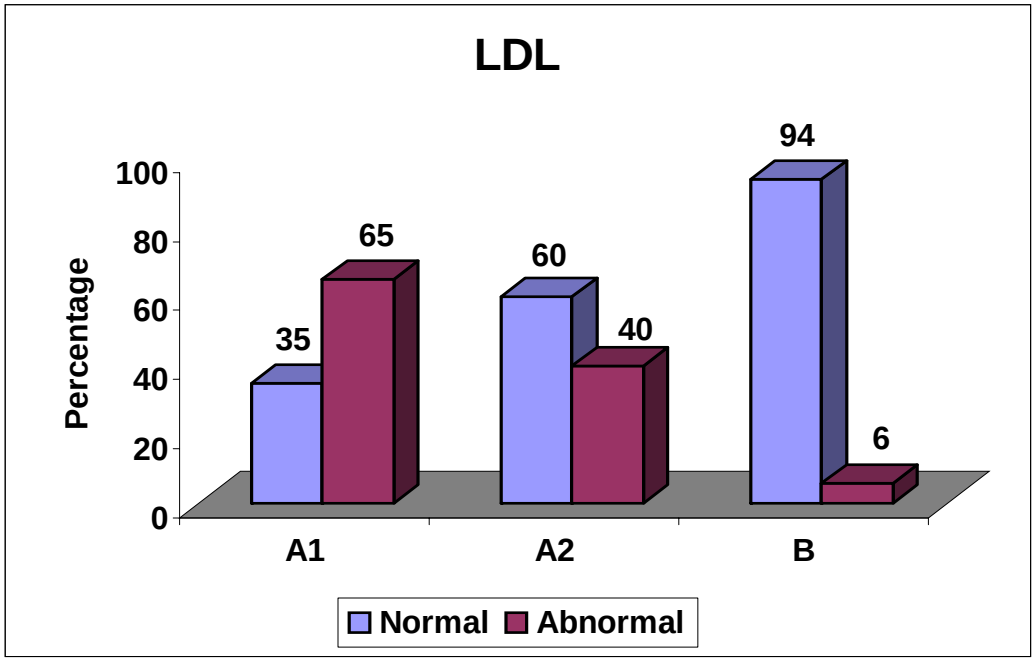

TYPES OF STROKE
28%
72%
A1
A2


RISK FACTORS FOR STROKE
Percentage
70
60
50
40
30
20
10
0
62
54
43.6
40
23
HT
IHD
SMOKING
OBESITY
DM


TOTAL CHOLESTEROL
Percentage
100
80
60
40
20
0
45
55
60
40
96
4
A1
A2
B
Normal
Abnormal


TRIGLYCERIDES
percentage
80
60
40
20
0
37.5
62.5
46.7
53.7
72
28
A1
A2
B
Normal
Abnormal


HDL
Percentage
100
80
60
40
20
0
45
55
13.3
86.7
60
40
A1
A2
B
Normal
Abnormal


LDL
Percentage
100
80
60
40
20
0
35
65
60
40
94
6
A1
A2
B
Normal
Abnormal