" MICROALBUMINURIA IN NON DIABETIC ACUTE ISCHAEMIC STROKE – PREVALENCE AND STROKE SEVERITY"

A Dissertation Submitted to

THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY

CHENNAI

In Partial Fulfillment of the Regulations

for the Award of the Degree of

M.D. (GENERAL MEDICINE) - BRANCH – I

GOVERNMENT KILPAUK MEDICAL COLLEGE

CHENNAI-10

April – 2016

BONAFIDE CERTIFICATE

This is to certify that **"MICROALBUMINURIA IN NON DIABETIC ACUTE ISCHAEMIC STROKE – PREVALENCE AND STROKE SEVERITY"** is a bonafide work performed by **Dr. UMALAKSHMI PREMNATH** , post graduate student, Department of Internal Medicine, Kilpauk Medical College, Chennai-10, under my guidance and supervision in fulfillment of regulations of the Tamil Nadu Dr. M.G.R Medical university for the award of M.D. Degree Branch I (General Medicine) during the academic period from July 2013 to April 2016.

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DECLARATION

I **Dr. UMALAKSHMI PREMNATH** hereby certify that i am the sole author of this study "**MICROALBMINURIA IN NON-DIABETIC ACUTE ISCHEMIC STROKE –PREVALENCE AND STROKE SEVERITY**" done at Kilpauk Medical College. I certify that this is the true copy my thesis,including any final revisions, as approved by my ethical committee done under the guidance of Prof .**Dr. T. RAVINDRAN MD,DNB , Professor of Medicine.**

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ACKNOWLEDGEMENT

I sincerely thank **Prof. R. NARAYANBABU, M.D.,DCH.,** Dean, Kilpauk Medical College, Chennai for permitting me to utilize the facilities needed for this dissertation work.

I am extremely grateful to **Prof.Dr.S.USHA LAKSHMI, M.D.,FMMC.,** Professor and Head of the Department of Internal Medicine, Kilpauk Medical College and Hospital for permitting me to carry out this study and for her constant encouragement and guidance.

I owe my sincere thanks and gratitude to my Chief **Prof.Dr.T.RAVINDRAN M.D.,DNB.** Professor of Medicine, Kilpauk Medical College for his esteemed guidance and valuable suggestions in all the stages of this dissertation.

I also express my sincere gratitude to **Prof. Dr. C.HARIHARAN M.D., Prof.Dr. C.KULOTHUNGAN,M.D., Prof.Dr.S.SURENDRAN,M.D.,** for their help and guidance rendered during the entire period of my work.

I whole heartedly express my sincere thanks to **Prof. DEVI MEENAL ,** M.D(Radiodiagnosis), Head of Department,Department of Radiology, Kilpauk Medical College, Chennai for his valuable guidance throughout my dissertation work.

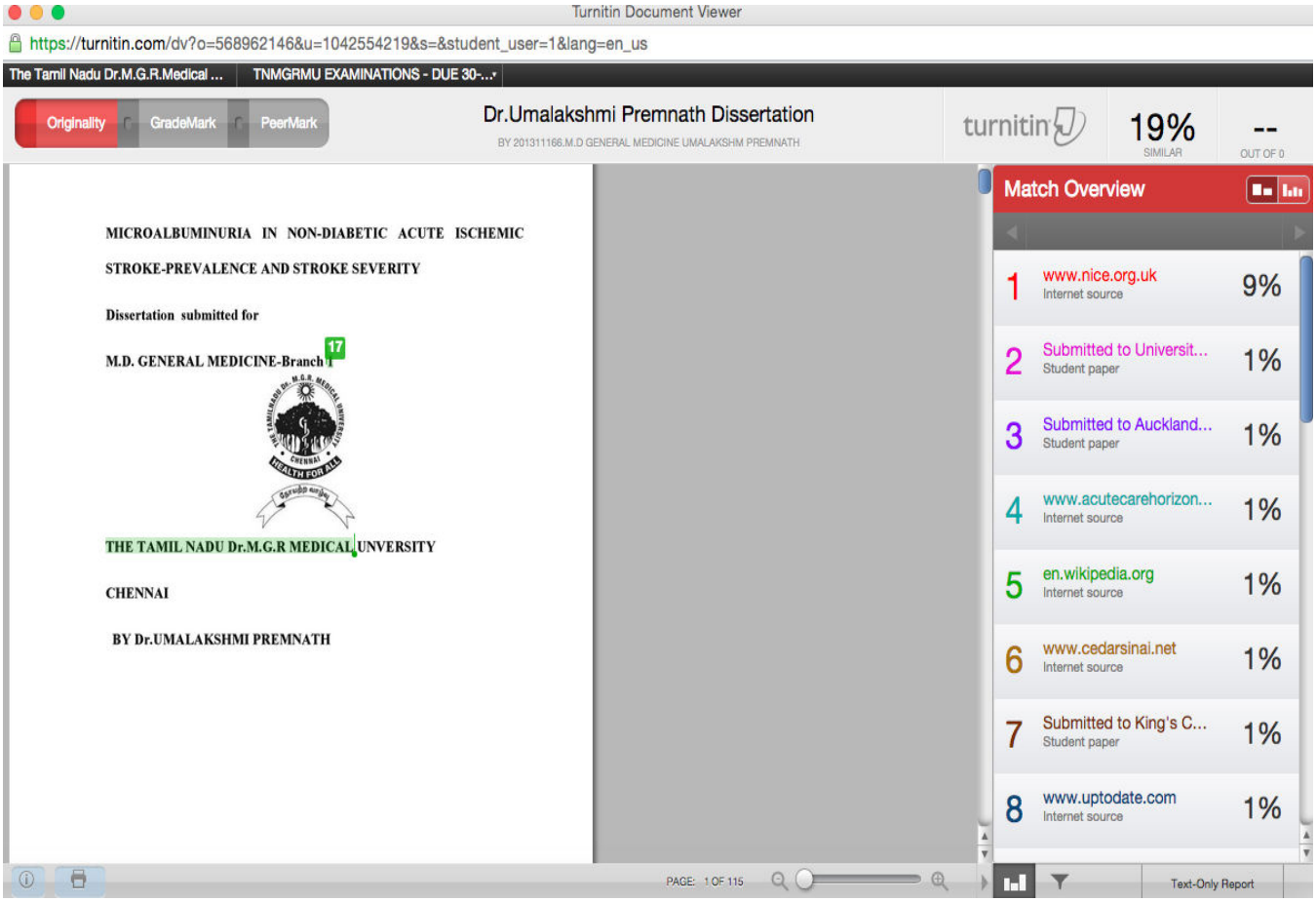
I whole heartedly express my sincere thanks to **Prof.. Dr. Lalitha M.D.,** Professor and Head, Department of Biochemistry, Kilpauk Medical College, Chennai for her valuable guidance and also for allowing me to use the Biochemistry Laboratory for the measurement of urine albumin .

I wish to thank **Dr.D.Venkteswarlu**, Registrar, **Dr.Rajan Babu.M.D,D.M, Dr. Panneer Selvam G, M.D.,** Assistant Professors, Department of Medicine, Kilpauk Medical College for their valuable suggestions and help rendered throughout this work. I also thank **Dr. Murugapandian, M.D.,** DM, Assistant Professor Department of Neurology, Kilpauk Medical College, for his valuable guidance and support throughout my dissertation work.

I also extend my thanks to all the laboratory technicians in the Biochemistry Department for their valuable support throughout my dissertation work.

I also thank my parents, my spouse, colleagues, friends and staff of our hospital, for their support for this work.

Last but not the least, with sincere gratitude, I thank all the patients who contributed so much to this study without whom this study could not have been possible.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STROKE-PREVALENCE AND STROKE SEVERITY
Dissertation submitted for
M.D. GENERAL MEDICINE-Branch I
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INTRODUCTION

Microalbuminuria though a relevant screening tool outside, rarely reported with very sparse literature in our setting.Dipstick analysis is a useful tool in screening of urinary abnormalities ,as itt detects hematuria,protenuria,specific gravity,leukocyturia.Microalbuminuria is defined as the urinary albumin excretion rate of 30-300mg/dl or 20-200microgram/dl n a randomly collected urine sample.

Microalbuminuria is said to be an indicator of widespread vascular endothelial damage and it reflects the transcapillary leakage of albumin.

It has been described to be a major factor in the causation of cardiovascular .renal,peripheral vascular disease and cerebrovascular diseases,especially when associated with conditions with Diabetes Mellitus,Systemic Hypertension.The prevalence ,progression and regression of microalbumnuria in these diseases was studied and landmark trials have confirmed its significance.Apart from these conditions it has also been proved to be important in other conditions like malignancy,meningitis where it acts as an important prognostic marker.

In this study we have made an attempt to find out the relation between microalbuminuria and stroke and an association has been found due to its relation with atherosclerosis responsible for the thickness in the intima and media of the carotid artery.

REVIEW OF LITERATURE

Microalbuminuria is defined as an excretion of albumin between 30-300mg/24 hrs or 20-200micrograms/minute.Among the modifiable risk factors causing microalbminuria are diabetes, hypertension, obesity, smoking ,hyperlipidemia, high salt or protein diet,oral contraceptives or hormone replacement therapy. Among the non-modifiable risk factors are race/ethnicity, male gender, older age and low birth weight. The mechanism responsible for this is the abnormal permeability of the glomerulus to albumin.KDIGO has replaced this term microalbuminuria with the term moderately increased albuminuria.2-3 measurements over a 2-3 month period is necessary to make a diagnosis of microalbuminuria.

To overcome the variations in calculating in the spot-check samples ,amount of albumin in the urine against the concentration gradient is used.Hence ,an albumin/creatitine ratio measured as >3.5 mg/mmol in males or >2.5mg/mmol n females equals 30-300mg/24 hrs.

An early morning sample is preferred as the patient must refrain from heavy exercise 24 hours before the test.

It has been used as a prognostic marker of kidney disease in

1.post-streptococcal glomerulonephritis

2.diabetes mellitus

3.Systemic hypertension

4.In intensive care setting it can be used as an indicator of overall mortality ,multiorgan dysfunction and acute respiratory failure if it s seen rising after 24 hours of admission.

5.It is also used as a risk factor for venous thromboembolism.

6.It is said to be a prognostic marker in meningitis,malignancy,hypertension.Also found useful in assessing response in diabetes,unilateral nephrectomy or renal scarring.

7.It has also found to be an indicator of glomerular injury,particularly in patients with sickle cell anemia prone to nephropathy.In sickle cell nephropathy,structural and functional abnormalities including hypoasthenuria,proteinuria with or without nephrotic syndrome,incomplete distal renal tubular acidosis,immune complex glomerulonephritis and progressive renal failure.The renal manifesations occur due to the microvascular occlusion of the renal vasculature by the sickle

cells,particularly in the absence of collateral circulation due to factors like hypoxia,acidosis and hypertonicity.

8.There has also said an inverse relation between low birth weight and microalbuminuria stating that factors in utero or in early childhood also claim to cause albumnuria in adult life.

Microalbuminuria and Chronic Kidney Disease

In diabetic and the non-diabetic population,microalbuminuria or proteinuria is associated with CKD. Heavy proteinuria is associated with a faster rate of progression to CKD.Is has been posutalated that those treated with antiproteinuric drugs like ACE inhibitors or ARBs are benefitted as the rate of progression to CKD is retarded.The tendency for natural progression is high when the proteinuria exceeds 1g/dl.

There is an independent association between rate os urine albumin excretion and presence and severity of CVD,including the non-diabetic and non-hypertensive population.

The common pathway linking microalbuminuria to CKD or CVD is diffuse endothelial and vascular dysfunction.

Renin Angotensin Aldosterone System has been implicated to be the common links between systemic hypertension,proteinuria/microalbuminuria and CVD.

An important single predictor of GFR decline is proteinuria and hence therapies that reduce proteinuria is helpful n decreasing GFR.

It has been proved from The Modification Of Diet (MDRD) and Ramipril Efficacy In Nephropathy (REIN) that by reducing the proteinuria to less than 1g/day there has been a signficant decline in GFR from 4-10 ml/min/year to 1-2ml/min/year

The recommended time interval for testing for proteinuria is 2-3 months for those with nephrotic range proteinuria and 6 months for those with subnephrotic range proteinuria.

Current guidelines suggest low-blood pressure goal is required only for those with greater proteinuria although lowering the blood pressure will help to lower the rate of proteinuria and might probably reduce the further cardiovascular risk.

Drugs useful in retarding proteinuria are

1.ACE inhibitors

2.ARBs.ACE inhibitors may be added to ARBs.

3.Renin Inhibitor.

Rationale behind this is that dual therapy will help to help to bring about the effect by optimizing the antiproteinuric effect through RAAS blockade.

However this dual therapy may not help to reduce the cardiovascular risk as does either of the drugs alone.This has been proved by the ONTARGET(ongoing Telamsartan alone and in combination with Ramipril Global Endpoint Trial).

Even in cases of resistant hypertension,non-dihydropyridines are recommended if adequate RAAS is present or eGFR<30ml as non –DHP have antihypertensive and antiproteinuric effects in contrast to dihydropyridines which might cause proteinuria.

There has been studies revealing analysis of Losartan used in treament of microalbminuria and hyperentension.A study called LIFE(losartan intervention for endpoint reduction in hypertension) was done for patients detected to high blood pressure (160-200/95) and an electrocardiographic evidence of left ventricular hypertrophy using a cut-off vale of > or equal to 3.5mg/mmol. Showed a prevalence of 26%.

It has been proved in many studies that those with metabolic syndrome (those with smoking,obesity,and insulin resistance) as satisfying the criteria by Adult Treament Panel III had higher range of microalbumnuria and left ventricular hypertrophy when compared to those without.

A higher the components of metabolic syndrome, more would be the prevalence of microalbuminuria and higher would be the target end organ damage.

C-reactive protein is also a sensitive marker of inflammation and a good indicator of cardiovascular outcome.

An increasing concentration of CRP from 0.2 -10mg/dl resulted in a marked steepening of the curve between albuminuria and arterial pressure with a potentiating effect when the mean arterial pressure are above 90mmHg.

Studies have also proven that in patients with essential hypertension there has been no detectable decrease in glomerular filtration rate among the normoalbminurics and microalbuminurics.The administration of captopril did not bring about the vasodialatory response in microalbminuric patients,which suggested that microalbminuria was an early marker of intra-renal vascular dysfunction.

A study called Hoorn study was done to find out the risk of microalbuminuria as a marker for atherosclerosis,which showed that an albumin/creatinine ratio of more than 2mg/mmol ad peripheral arterial disease ,which was analysed by the ankle-brachial index were associated with a fourfold increase in cardiovascular mortality that was more in hypertensive than in non-hypertensive population.

Evidence also proves that there is a greater cardiovascular morality among the patients with higher microalbuminuria among the diabetic population.In the PREVEND international trial those who were treated with fosinopril had a much lesser degree of coronary events,as much as 40 percent, when compared with those who were not treated.

Statins,renin inhibitors and glycosaminoglycans had also showed a reduction in microalbuminuria.

Studies have also shown that there is a tendency for higher body mass index,lesser reduction in blood pressure and a greater increase in glucose and uric acic acid in albuminuria progressors.Population studies did not reveal any relation between GFR(glomerular filtration rate) and microalbuminuria., but a study called PREVEND(prevention of renal and vascular end-stage disease),estimation of GFR by 24-hour creatinine clearance tended to be higher n those with higher albuminuria whereas reduction in creatinine clearance was associated with macroalbumnuria for that age and sex.

CEREBROVASCULAR DISEASES

Defined as an abrupt onset of a focal neurological deficit.It is common among the elderly population and its incidence increases with age.It includes some of the common disorders namely, ischemic stroke,hemorrhagic stroke,anomalies like arteriovenous malformations and intracranial aneurysms.

Ischemic Stroke

A decrease in cerebral blood flow causes death of the brain tissue in 4 to 10 minutes.Death occurs in an hour if the supply reduces by 16 to 18 ml/100 g tissue per minute and within several hours or days if the flow reduces by <20ml/100 g tissue per minute.Penumbra refers to the region surrounding the core and is reversibly dysfunctional.Perfusion-diffusion imaging wih CT or MRI will help to image the penumbra.

There are 2 pathways responsible for cerebral infarction

2.apoptotic-programmed cell death.

CAUSES OF ISCHEMIC STROKE

It occurs either due to insitu thrombosis or due to embolus from a proximal source ,either an artery or from the heart.

CAUSES

Thrombosis

Lacunar stroke

Large vessel thrombosis

Dehydration

Embolic Occlusion

Artery to artery

Carotid bifurcation

Aortic arch

Aortic dissection

Cardioembolic

Atrial fibrillation

Mural thrombus

Myocardial infarction

Dialated cardiomyopathy

Valvular lesions

Mitral stenosis

Mechanical valves

Bacterial endocarditis

Parodoxical embolus

Atrial septal defect

Patent foramen ovale

Atrial septal aneurysm

Spontaneous echo contrast

Others

Hypercoagulable states-protein c and protein s and antithrombin deficiency

Antiphospholipid antibody syndrome

Factor V leiden mutation

TTP

Homocysteinemia

DIC

Dysproteinemia

Nephrotic syndrome

Oral contraceptives

Inflammatory bowel disease

Venous sinus thrombosis

Stroke of undetermined origin

Multiinfarct dementia in lacunar syndrome

Moyamoya disease

Fibromuscular dysplasia

Binswanger's subcortical arteriosclerotic encephalopathy

Winiwarter buerger disease

Aortic arch syndrome

Risk factors

Hypertension

Atrial fibrillation

Diabetes

Smoking

Hyperlipidemia

Carotid stenosis

The normal functions of the brain is dependent on the oxygen supply and the glucose perfusing it.The critical threshold for the energy metabolism is maintained by the pH,partial pressure of oxygen and carbondioxide,cerebrovascular resistance and the mean arterial blood pressure.The is paralysis of autoregulation and the microvasculature is nonreactive to the changes occurring making it more permeable to protein and fluid leaks and also causing hemoconcentration and vascular stasis.Due to brain hypoxia,energy is depleted and hence formation of ATP doesn't occur.Among the factors that increase the neuronal injury are

increased cytosolic leaks due to increased intracellular calcium concentration due to loss of activity of sodium-potassium pump,acidosis and release of free radicals.Other ischemia promoting factors are exposure of the vascular endothelium to raised homocysteine(>100micromol/l) that leads to reduced levels of nitric oxide,increased levels of adhesion molecules and procaogulant factors like thrombomodulin,protein C,tissue plasminogen activator and plasminogen activator inhibitor.

A mechanism said to be responsible for this has been that at the site of atheromatous plaque ,endothelial injury occurs and there is attachment of platelet to the exposed subendothelial collagen ,at the site of which phospholipids and cholesterol has been released and phospholiase A2 and cycloxygenase converts to potent platelet aggregator thromboxane A2.

However a well developed vascular system is present to provide collateral circulation to the brain even in the presence of ischemia namely the carotid and the vertebral arteries .An extracranial anastomosis between the cervical branches of the ipsilateral external carotid,subclavian,and the vertebral arteries also provides an anastomosis.

Lacunar infarcts are also common among the diabetics and hypertensives .They are common in the deep white matter,basal ganglia,internal capsule,cerebellum,corona radiata .They are 150-300mm n size.

Internal carotid artery can also cause stroke especially in the cervical portion that is prone to atherosclerosis and is also a site for embolic episodes.However ,it may be partly offset by the anastomosis between ophthalmic and external carotid and partly by the superficial and the deep anastomosis from the opposite side.Symptoms of confusion ,dysarthria ,dyslexia sensory symptoms with or without motor weakness of the opposite side may occur.

Diagnosis of stroke

CT brain –it is important in ruling out a a hemorrhage.A CT scan in the first 6 hours may not show any difference.It is also useful to decide on the line of management whether antiplatelets or anticoagulants.It is also helpful in differentiating an ischemic infarction or a cerebral hematoma from other simulators like tumour or a subdural hematoma.It helps to detect small infarcts including lacunae(0.5cm). However brainstem lacunae may be difficult to detect.

CT findings

Hyperacute<12hours-normal(50-60%),hyperdense artery sign(25-50%),obscuration of lentiform nucleus.

Acute(12-24hours)-low density basal ganglia,loss of grey-white matter interface(insular ribbon sign),sulcal enhancement.

1-7 days-mass effect.wedge-shaped low density area involving grey and white matter,hemorrhagement transformation,gyral enhancement.

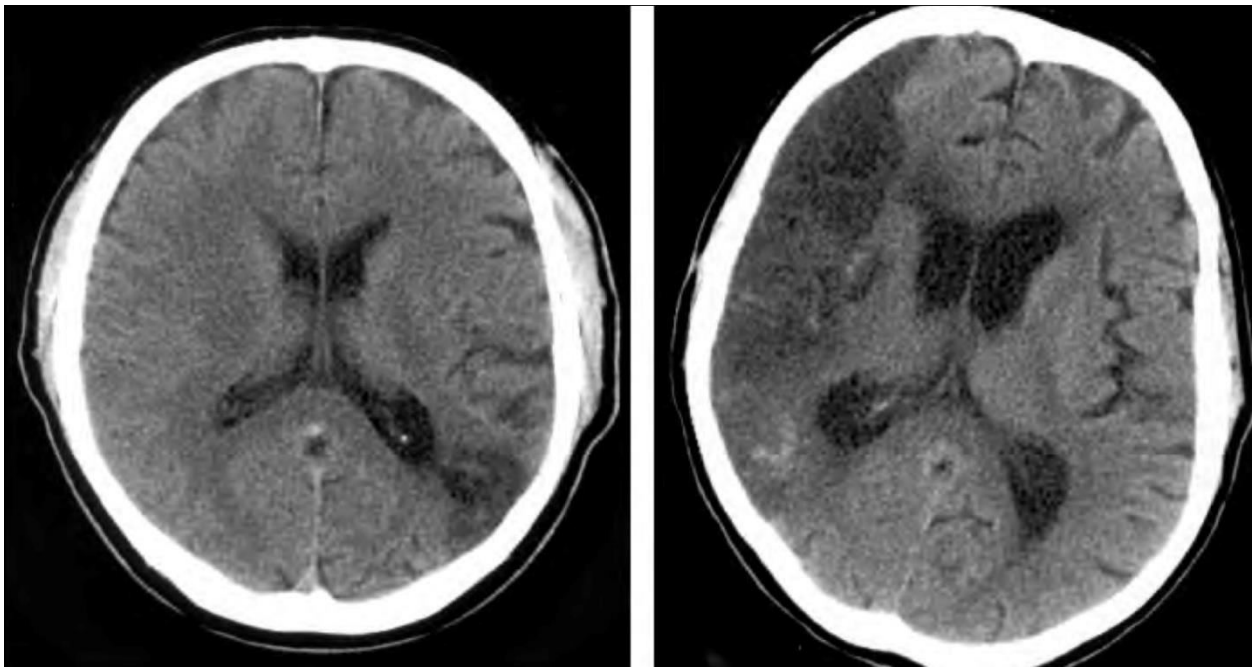
1-8weeks-contrast enhancement persists ,mass effect resolves.

Months-years-encephalomalacic change,volume loss,rarely calcification.

MAGNETIC RESONANCE DIFFUSION WEGHTED AND PERFUSION WEIGHTED IMAGING-revealed infarcts faster than the conventional T2 weighted imaging did .DWI was based on the Brownian Movement Of Water.Acute infarcts had lower ADC(apparent diffusion coefficients)than the noninfarcted regions did due to the energy failure and the subsequent cytotoxic edema.The DWI shows the ischemic core and PWI indicates the impaired perfusion in the core and in the surrounding penumbra,thereby complementing the DWI. Hence it played a role in making an anatomic diagnosis early and helped in

implementing stroke interventions earlier in order to save the penumbra. In Perfusion Weighted imaging,the volumes of the regions with TTP (time to peak)delay was calculated and has been found invarious trials that those with a TTP more than 6 was associated with a more lesional enlargement in the follow up scans between the first and the following. MRA with MRI helps to find out he extent and severity of the lesions and is helpful in taking decisions like thrombolytic therapy.

Duplex sonography and transcranial Doppler studies are also helpful in evaluating the extra and intracranial anastomosis ,the stenotic area and the superimposed thromboemboli.A Digital Subtraction Angiography will be belp to detect the precise degree of narrowing and the collateral circulation if a surgical procedure is planned .It is important to carefully weigh the risk and benefits of DSA in comatose patients and in those with neurological deficits.



TREATMENT

After maintenance of the vital signs ,patency of the fluid,electrolytes,prevention of complications like seizures,aspiration,bedsores and thrombophlebitis has to be looked into.Ischemic tissue infarction with break of the blood brain barrier retains fluid contributing to cerebral edema.By a phenomenon called “cerebral squeeze” the swollen tissue also squeezes on the adjacent viable neuronal tissue and may even herniate. Hence measures to reduce the cerebral blood pressure like judicious fluid restriction,osmotic diuretics like 3%mannitol and 10%glycerol is helpful.Blood pressure also has to be reduced.Sodium nitroprusside is often used in hypertensive crises (BP>240/130mm hg)however parenteral use of enalapril

,labetalol or frusemide is also advocated .In massive cerebral or cerebellar infarction causing brainstem ,decompressive craniotomy may be used.

SPECIFIC THERAPIES

Atherosclerotic risk factors

Diabetes mellitus,systemic hypertension,obesity,smoking,dyslipidemias ,family history of stroke,old age,oral contraceptives, hormone replacement therapies are said to proven risk factors of stroke. Several trials like SPARCL(stroke prevention by aggressive reduction in cholesterol levels)showed that there was a considerable reduction in stroke in those who were suffering from TIA and the incidence of stroke.Another trial namely JUPITER(justification for the use of statins in prevention,an intervention trail evaluating Rosuvasatin)low LDL caused by elevated CRP levels also benefitted from it.

Antiplatelets namely aspirin,clopidogrel was commonly used.Ticlopidine was used earlier but now has been banned due to side-effects like reversible neutropenia and diarrhea.

Aspirin prevents the conversion of arachidonic acid to thromboxane A 2.Dipyridamole prevents the enzyme phosphodiesterase and therefore platelet aggregation.Platelets also release prostacyclin which prevents the aggregation of platelets.

Ticlopidine inhibits platelet aggregation by interfering with ADP induced transformation of glycoprotein 2b-3a receptors on the surface of the platelets.

Anticoagulants like heparin an warfarin have also found use in strokes occuring due to a cardioembolic cause like atrial fibrillation.If the patient worsens on anticoagulating a immediate re-evaluation has to be done with a second CT to ascertain the cause as to whether there is an intracranial bleed or an extension of the infarct.

Heparin should be monitored by APTT (it must be kept upto two times the control).An intravenous bolus of 100 units /kg stat followed by 1000units /hour infusion for 24 hours must be given. Warfarin may be given to keep the INR between 2-3 baring in mind the complications that occur f not properly monitored.

THROMBOLYTIC THERAPY

Several measures had been tried to remove the clot lysis and to enable recanalisation.Available thrombolyic agents are streptokinase,recombinant tissue plasminogen activator,urokinase.

Several trials have been done.One such trial was the PROACT(prolyse in acute cerebral thromboembolism trial).It was done to test the efficacy and recanalisation by giving prokinase 6mg or placebo which was given within 6hours within the onset of stroke ,that was confirmed by a CT Brain.

Recanalisation was found in middle cerebral artery better than in internal carotid or basilar arteries.It was not found to be superior or inferior to intravenous thrombolysis.In this study co-adminisration was heparin was done and was found that it was associated with a small risk of bleeding.

There were trials done to estimate the use of streptokinase(1.5million units)which was given within 4 hours of onset of stroke .It was found to be associated with more incidence of hemorrhages and bleeding and hence the trials were withheld.

Another trial namely the European Cooperative acute stroke study was done to see the efficacy of r-TPA 1.1mg/kg,of which 10 percent was given as an intravenous bolus and the remaining within the next one hour .Those with a CT evidence of

diffuse cerebral swelling,effacement of the sulci,involvement of more than 33percent of the middle cerebral artery territory or parenchymal hypodensity were excluded.The outcome as suggested by the Modifed Rankin Score was 2 in the subgroup treated with the medication which showed a better neurological outcome as compared with he placebo.

A study namely the National Institute Of Neurological Disorders and Stroke r-tpa stroke study was done to evaluate the efficacy of r-tpa at a dose of 0.9 mg /kg ,of which 10 percent was given as an intravenous bolus dose and the remaining over an hour . within 3 hours of onset of stroke.Those suffering from ataxia alone ,dysarthria alone ,minimal weakness or sensory loss alone were excluded from the study as were those with other contraindications like high blood pressure >185/110mmhg ,prolonged prothrombin time beyond 15 seconds or an INR>1.7, thrombocytopenia or an abnormal hematocrit of below 25 percent or a blood glucode level of either below 50 or above 200 or improving neurological symptoms ,seizure at the beginning of the stroke or a gastrointesinal or urinary bleeding in the preceeding 21 days or a history of recent myocardial infarction.

However in neonates or in children the efficacy and safety of using the thrombolytic therapy for acute ischemic stroke was yet to be studied.

Use of thrombolytic agents for other purposes in children like arterial ,right atrial or caval thrombosis, in pulmonary embolism,in Blalock-Taussig shunt ,in thrombosed dialysis shunts and in cerebral venous thrombosis was done.The dose used is 0.5mg/kg in children.

Certain precautions to be taken before administering were that central venous access,arterial punctures,or insertion of nasogastric tubes within 24 hours had to be refrained.Indwelling catheters should be avoided during the drug infusion till 30 minutes after the infusion ends.

In case bleeding occurs,the thrombolytic therapy may be stopped depending on whether the bleeding points can be controlled with local pressure at the arterial or venous sites.

The bleeding patient's blood should be tested for hemoglobin,,hematocrit,partial thromboplastin time ,INR,platelet count and fibrinogen.After crossmatching blood may be transfused(4 units of packed cells and 4-6 units of cryoprecipitate or fresh frozen plasma).

Endovascular mechanical thrombectomy has also gained importance these days as an adjunctive measure or for those in whom thrombolytics are contraindicated.

One such device is MERCI (mechanical embolus removal in cerebral ischemia)which has brought good results by restoring the patency within 8 hours of onset of then symptoms.The Penumbra Pivotal Stroke Trial is another mechanical device which is also FDA approved like the MERCI.

Many scales have been used for the diagnosis

STROKE DIAGNOSTC SCALES:

Face Arm Speech Test

Cincinnati Prehospital Stroke Scale

Los Angeles Prehospital Stroke Screen

Recognition Of Stroke In Emergency Room Scale Comparison Studies

STROKE IMPAIRMENT SCALES

National Institute Of Health Stroke Scale

Pediatric National Institute Of Health Stroke Scale

European Stroke Scale

Canadian Neurological Scale

Scandinavian Stroke Scale

DISABILITY SCALES

Barthel Index

Functional Independence Measure

Instrumental Activities Of Daily Living

HANDICAP SCALES

Modified Rankin Scale

1a. Level of Consciousness: 0- **Alert;** keenly responsive. ———

= 1-**Not alert**; but arousable by minor stimulation to obey, answer, or respond.

= 2-**Not alert**; requires repeated stimulation to attend, or is obtunded and

requires strong or painful stimulation to make movements (not stereotyped).

= 3-Responds only with reflex motor or autonomic effects or totally unresponsive, flaccid, and areflexic.

1b. LOC Questons : 0-**Answers** both questions correctly. ———

: 1-**Answers** one question correctly.

: 2-**Answers** neither question correctly.

1c. LOC Command s:. 0-**Performs** both tasks correctly. ———

1-**Performs** one task correctly.

2-**Performs** neither task correctly.

2. Best Gaze: 0-**Normal.** ———

1-**Partial gaze palsy;** gaze is abnormal in one or both eyes, but forced deviation or total gaze paresis is not present.

2-**Forced deviation,** or total gaze paresis not overcome by the oculocephalic maneuver.

3. Visual: o-No visual loss. _____

1-Partial hemianopia.

2-Complete hemianopia.

3-Bilateral hemianopia (blind including cortical blindness).

4. Facial Palsy: 0-Normal symmetrical movements. _____

1-Minor paralysis (flattened nasolabial fold, asymmetry on smiling).

2-Partial paralysis (total or near-total paralysis of lower face).

3-Complete paralysis of one or both sides (absence of facial movement in the upper and lower face).

5. Motor Arm: 0-No drift; limb holds 90 (or 45) degrees for full 10 seconds. _____ _____

1--Drift; limb holds 90 (or 45) degrees, but drifts down before full 10 seconds;doesn't hit bed.

2. some effort against gravity

Limb cannot get to or maintain (if cued) 90 (or 45) degrees, drifts down to bed,

3-No effort against gravity; limb falls.

4-No movement.

= Amputation or joint fusion, explain: _____

5a-Left Arm

5b-Right Arm

6. Motor Leg 0-No drift; leg holds 30-degree position for full 5 seconds. ———

1-Drift; leg falls by the end of the 5-second period but does not hit bed.

2-Some effort against gravity; leg falls to bed by 5 seconds, but has some effort against gravity.

3-No effort against gravity; leg falls to bed immediately.

4-No movement.

= Amputation or joint fusion, explain:

_____

5a-Left Leg

5b-Right Leg

7.Limb Ataxia

0-Absent. _____

1-Present in one limb.

2-Present in two limbs.

= Amputation or joint fusion, explain:

_____

8. Sensory

0-Normal; no sensory loss. _____

1-Mild-to-moderate sensory loss; patient feels pinprick is less sharp or is dull on the affected side; or there is a loss of superficial pain with pinprick, but patient is aware of being touched.

2-Severe to total sensory loss; patient is not aware of being touched in the face,

arm, and leg.

9. Best Language	=	0-No aphasia; normal. 1-Mild-to-moderate aphasia; some obvious loss of fluency or facility of comprehension, without significant limitation on ideas expressed or form of expression. Reduction of speech and/or comprehension, however, makes conversation about provided materials difficult or impossible. For example, in conversation about provided materials, examiner can identify picture or naming card content from patient's response. 2-Severe aphasia; all communication is through fragmentary expression; great need for inference, questioning, and guessing by the listener. Range of information that can be exchanged is	———

limited; listener carries burden of communication. Examiner cannot identify materials provided from patient response.

3-Mute, global aphasia; no usable speech or auditory comprehension.

10. Dysarthria:

0-normal

1-**Mild-to-moderate dysarthria;** patient slurs at least some words and, at worst, can be understood with some difficulty.

2-**severe dysarthria;** patient's speech is so slurred as to be unintelligible in the absence of or out of proportion to any dysphasia, or is mute/anarthric.

= **Intubated** or other physical barrier, explain:____

11.Extinction and Sensory

0-**No abnormality.**

= 1-**Visual, tactile, auditory, spatial, personal inattention** or extinction to bila simultaneous stimulation in one of the sen

Inattention

modalities.

2-**Profound hemi-inattention or extinction**
more than one modality; does not recogn
own hand or orients to only one side of spac

_____

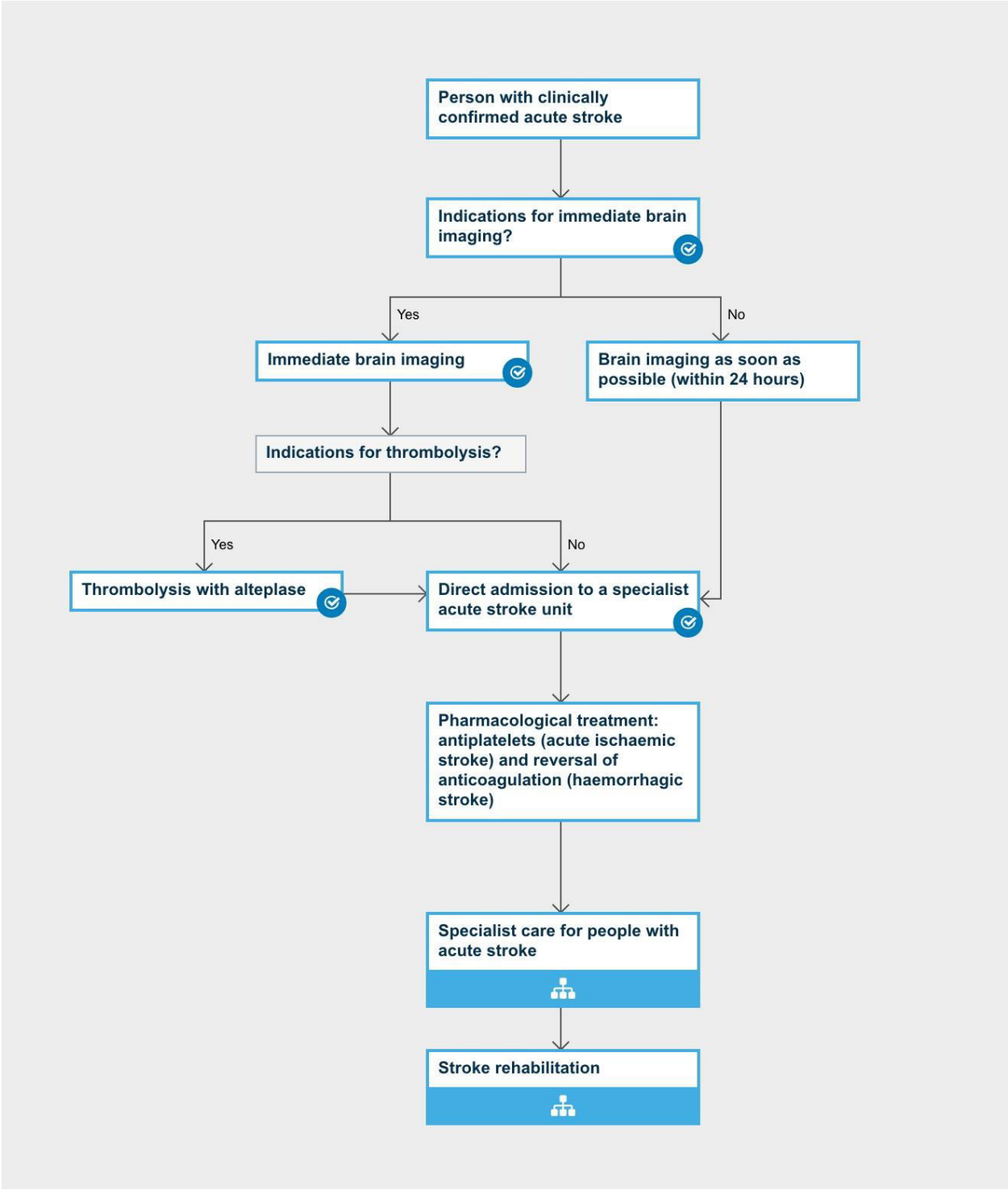
NIHSS

- SCALE
- 0-no stroke symptoms
- 1-4-minor symptoms
- 5-15-moderate disabilty
- 16-20-moderate to severe disability
- 21-42-severe diability.

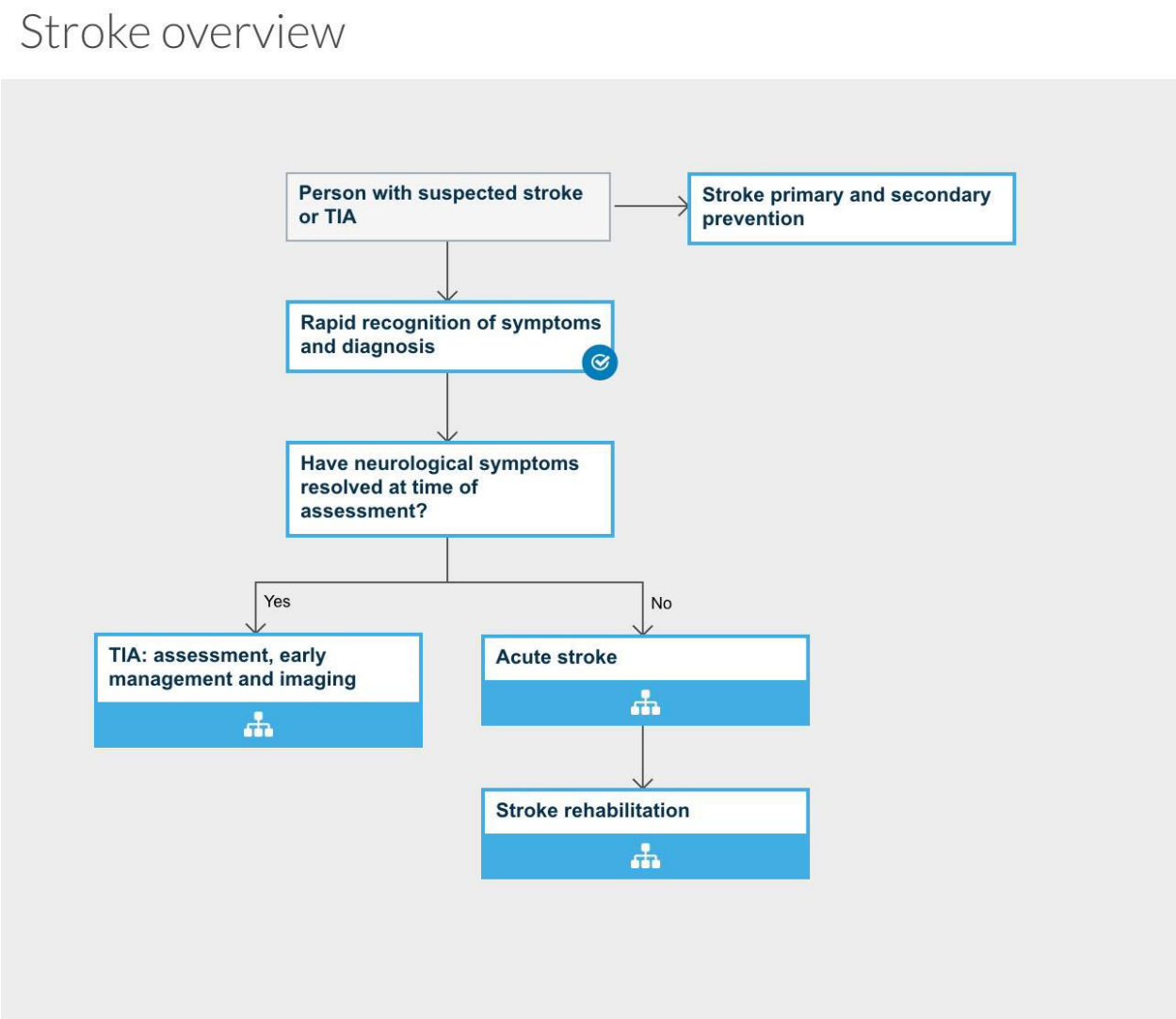
MODIFIED RANKN SCALE

- 0-no symptoms
- 1-no significant diasbility.able to carry out all usual activities,despite some symptoms
- 2-slight disability.able to look after own affairs without any assistance,but unable to carry out all previous activities.
- 3-moderate disability.requires some help but able to walk unassisted.
- 4-moderately severe disability.unable to attend o own bodily needs without assistance,and unable to walk unassisted.
- 5-severe disability.requires constant nursing care and attention.
- 6-dead

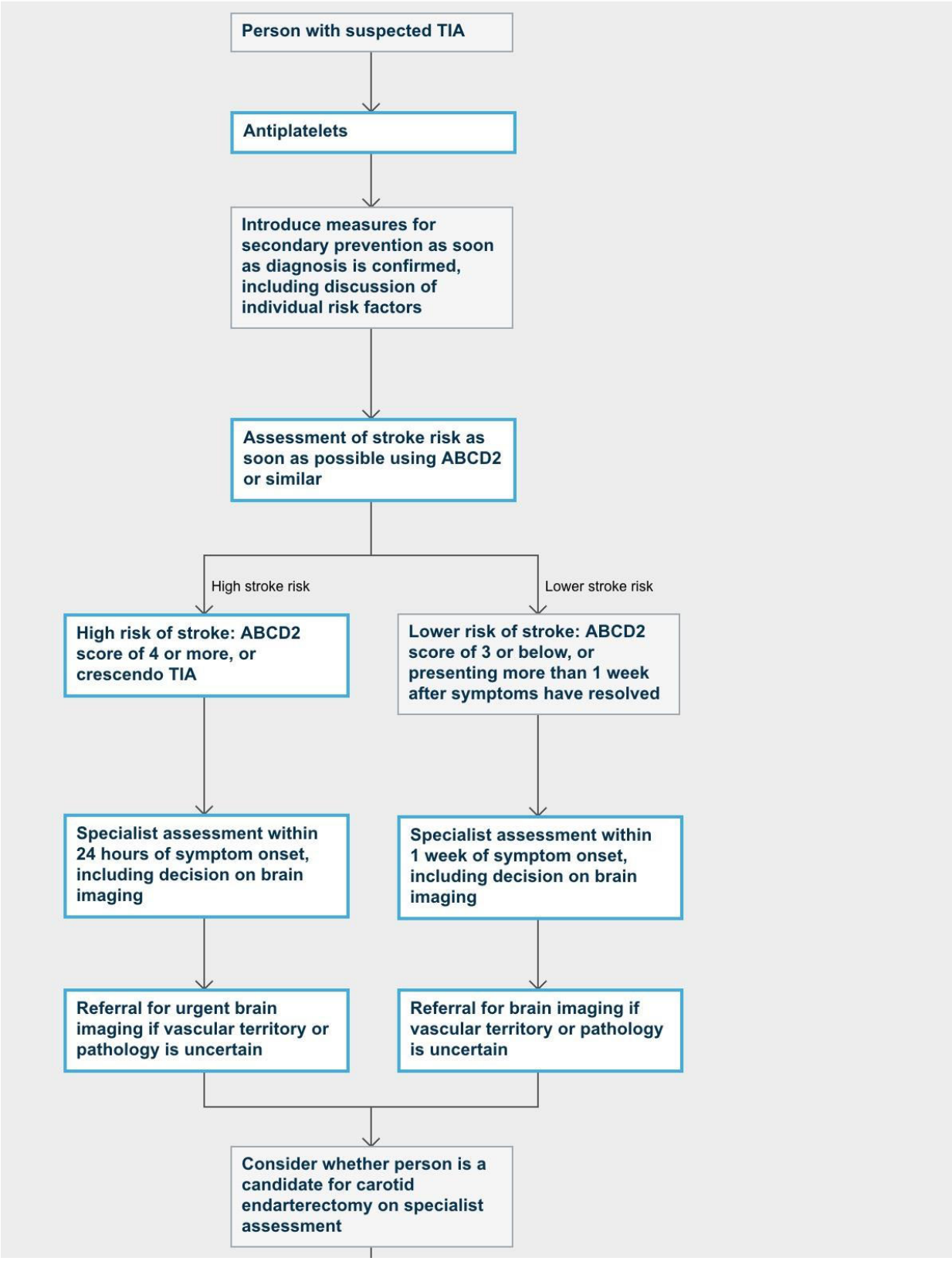
Acute stroke



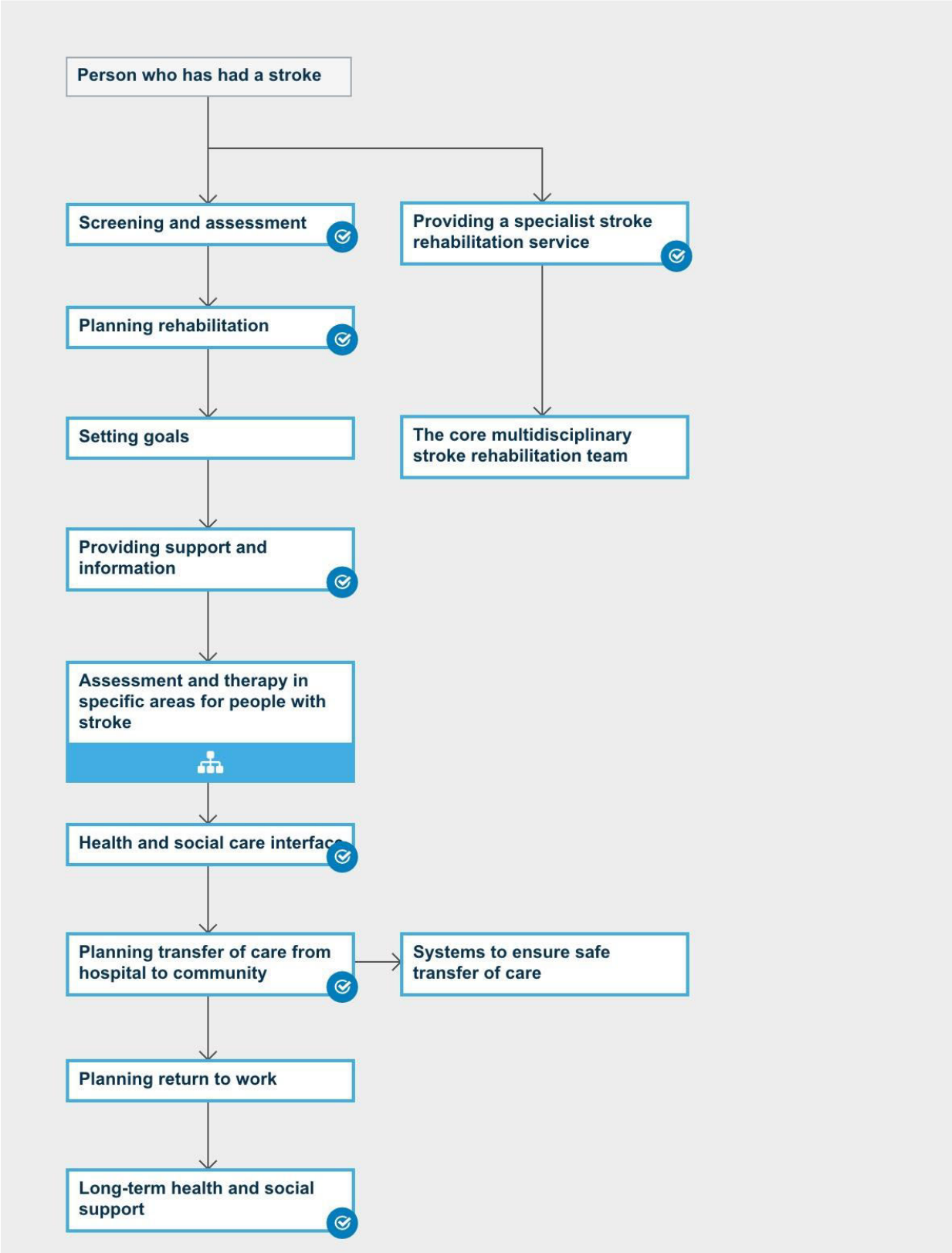
The above is the approach towards a case of confirmed stroke,indications for imaging ,need for specialist care followed by stroke rehabiltation.



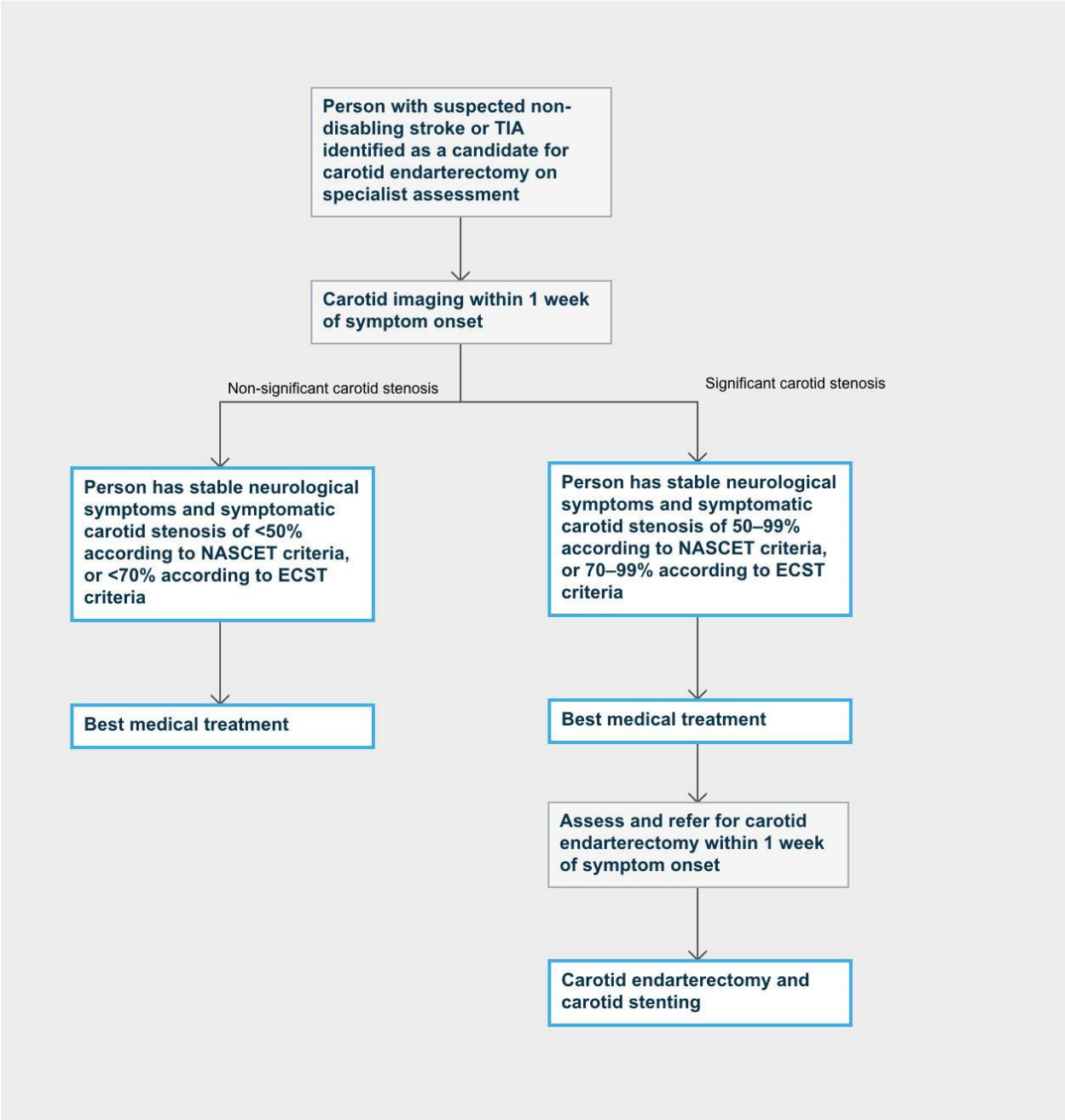
TIA: assessment, early management and imaging



Stroke rehabilitation



Carotid imaging and carotid endarterectomy for people with TIA or non-disabling stroke



The above is the flow chart showing an approach towards a case suspected of having a transient ischemic attack.

Rapid recognition of symptoms and diagnosis

There is evidence that rapid treatment improves outcome after stroke or TIA. The recommendations in this section cover the rapid diagnosis of people who have had sudden onset of symptoms that are indicative of stroke and TIA. How to identify risk of subsequent stroke in people who have had a TIA is also covered.

"Prompt recognition of symptoms of stroke and TIA

> In people with sudden onset of neurological symptoms a validated tool, such as FAST (Face Arm Speech Test), should be used outside hospital to screen for a diagnosis of stroke or TIA.
>
> In people with sudden onset of neurological symptoms, hypoglycemia should be excluded as the cause of these symptoms.
>
> People who are admitted to accident and emergency (A&E) with a suspected stroke or TIA should have the diagnosis established rapidly

using a validated tool, such as ROSIER (Recognition of Stroke in the Emergency Room).

Assessment of people who have had a suspected TIA, and identifying those at high risk of stroke

People who have had a suspected TIA (that is, they have no neurological symptoms at the time of assessment [within 24 hours]) should be assessed as soon as possible for their risk of subsequent stroke using a validated scoring system such as ABCD2.

People who have had a suspected TIA who are at high risk of stroke (that is, with an ABCD2 score of 4 or above) should have:

- aspirin (300 mg daily) started immediately
- specialist assessment and investigation within 24 hours of onset of symptoms
- measures for secondary prevention introduced as soon as the diagnosis is confirmed, including discussion of individual risk factors.

People with crescendo TIA (two or more TIAs in a week) should be treated as being at high risk of stroke, even though they may have an ABCD2 score of 3 or below.

People who have had a suspected TIA who are at lower risk of stroke (that is, an ABCD2score of 3 or below) should have:

- aspirin (300 mg daily) started immediately
- specialist assessment[10] and investigation as soon as possible, but definitely within 1 week of onset of symptoms
- measures for secondary prevention introduced as soon as the diagnosis is confirmed, including discussion of individual risk factors.

People who have had a TIA but who present late (more than 1 week after their last symptom has resolved) should be treated as though they are at lower risk of stroke.

Imaging in people who have had a suspected TIA or non-disabling stroke

While all people with symptoms of acute stroke need urgent brain scanning, there is less evidence to recommend brain scanning in those people whose symptoms have completely resolved by the time of assessment. This section contains recommendations about which people with suspected TIA need brain imaging and the type of imaging that is most helpful.

Some people who have had a stroke or TIA have narrowing of the carotid artery that may require surgical intervention. Carotid imaging is required to define the extent of carotid artery narrowing. Sections 1.2.3 and 1.2.4 cover the optimum timing of carotid imaging, and the selection of appropriate patients for, and timing of, carotid endarterectomy. The use of carotid stenting was also reviewed by the GDG. However, no evidence for early carotid stenting was found on which the GDG felt they could base a recommendation. For more information, see chapter 6 of the full guideline.

Suspected TIA – referral for urgent brain imaging

People who have had a suspected TIA (that is, whose symptoms and signs have completely resolved within 24 hours) should be assessed by a specialist (within 1 week of symptom onset) before a decision on brain imaging is made.

People who have had a suspected TIA who are at high risk of stroke (for example, an ABCD2 score of 4 or above, or with crescendo TIA) in whom the vascular territory or pathology is uncertain should undergo urgent brain imaging (preferably diffusion-weighted MRI [magnetic resonance imaging]).

People who have had a suspected TIA who are at lower risk of stroke (for example, an ABCD2 score of less than 4) in whom the vascular territory or pathology is uncertain should undergo brain imaging (preferably diffusion-weighted MRI).

Type of brain imaging for people with suspected TIA

People who have had a suspected TIA who need brain imaging (that is, those in whom vascular territory or pathology is uncertain) should undergo diffusion-weighted MRI except where contraindicated, in which case CT (computed tomography) scanning should be used.

Early carotid imaging in people with acute non-disabling stroke or TIA

All people with suspected non-disabling stroke or TIA who after specialist assessment are considered as candidates for carotid endarterectomy should

have carotid imaging within 1 week of onset of symptoms. People who present more than 1 week after their last symptom of TIA has resolved should be managed using the lower-risk pathway.

Specialist stroke units

All people with suspected stroke should be admitted directly to a specialist acute stroke unit following initial assessment, either from the community or from the A&E department.

Brain imaging for the early assessment of people with acute stroke

Brain imaging should be performed immediately for people with acute stroke if any of the following apply:

- indications for thrombolysis or early anticoagulation treatment
- on anticoagulant treatment
- a known bleeding tendency
- a depressed level of consciousness (Glasgow Coma Score below 13)

- unexplained progressive or fluctuating symptoms
- papilloedema, neck stiffness or fever
- severe headache at onset of stroke symptoms.

For all people with acute stroke without indications for immediate brain imaging, scanning should be performed as soon as possible.

Pharmacological treatments for people with acute stroke

Urgent treatment has been shown to improve outcome in stroke. This section contains recommendations about urgent pharmacological treatment in people with acute stroke.

Thrombolysis with alteplase

Alteplase is recommended for the treatment of acute ischaemic stroke when used by physicians trained and experienced in the management of acute stroke. It should only be administered in centres with facilities that enable it to be used in full accordance with its marketing authorization.

Alteplase should be administered only within a well organised stroke service with:

- staff trained in delivering thrombolysis and in monitoring for any complications associated with thrombolysis

- level 1 and level 2 nursing care staff trained in acute stroke and thrombolysis

- immediate access to imaging and re-imaging, and staff trained to interpret the images.

Aspirin and anticoagulant treatment

People with acute ischaemic stroke

All people presenting with acute stroke who have had a diagnosis of primary intracerebral haemorrhage excluded by brain imaging should, as soon as possible but certainly within 24 hours, be given:

- aspirin 300 mg orally if they are not dysphagic or

- aspirin 300 mg rectally or by enteral tube if they are dysphagic.

Thereafter, aspirin 300 mg should be continued until 2 weeks

after the onset of stroke symptoms, at which time definitive long-term antithrombotic treatment should be initiated. People being discharged before 2 weeks can be started on long-term treatment earlier.

Any person with acute ischaemic stroke for whom previous dyspepsia associated with aspirin is reported should be given a proton pump inhibitor in addition to aspirin.

Any person with acute ischaemic stroke who is allergic to or genuinely intolerant of aspirin should be given an alternative antiplatelet agent.

Anticoagulation treatment should not be used routinely for the treatment of acute stroke.

People with acute venous stroke

People diagnosed with cerebral venous sinus thrombosis (including those with secondary cerebral haemorrhage) should be given full-dose anticoagulation treatment (initially full-dose heparin and then warfarin [INR 2–3]) unless there are comorbidities that preclude its use.

People with stroke associated with arterial dissection

People with stroke secondary to acute arterial dissection should be treated with either anticoagulants or antiplatelet agents, preferably as part of a randomised controlled trial to compare the effects of the two treatments.

People with acute ischaemic stroke associated with antiphospholipid syndrome

People with antiphospholipid syndrome who have an acute ischaemic stroke should be managed in same way as people with acute ischaemic stroke without antiphospholipid syndrome.

Reversal of anticoagulation treatment in people with haemorrhagic stroke

Clotting levels in people with a primary intracerebral haemorrhage who were receiving anticoagulation treatment before their stroke (and have elevated INR) should be returned to normal as soon as possible, by reversing the effects of the anticoagulation treatment using a combination of prothrombin complex concentrate and intravenous vitamin K.

Anticoagulation treatment for other comorbidities

People with disabling ischaemic stroke who are in atrial fibrillation should be treated with aspirin 300 mg for the first 2 weeks before considering anticoagulation treatment.

In people with prosthetic valves who have disabling cerebral infarction and who are at significant risk of haemorrhagic transformation, anticoagulation treatment should be stopped for 1 week and aspirin 300 mg substituted.

People with ischaemic stroke and symptomatic proximal deep vein thrombosis or pulmonary embolism should receive anticoagulation treatment in preference to treatment with aspirin unless there are other contraindications to anticoagulation.

People with haemorrhagic stroke and symptomatic deep vein thrombosis or pulmonary embolism should have treatment to prevent the development of further pulmonary emboli using either anticoagulation or a caval filter.

Statin treatment

Immediate initiation of statin treatment is not recommended in people with acute stroke.

People with acute stroke who are already receiving statins should continue their statin treatment.

Maintenance or restoration of homeostasis

A key element of care for people with acute stroke is the maintenance of cerebral blood flow and oxygenation to prevent further brain damage after stroke. This section contains recommendations on oxygen supplementation, maintenance of normoglycaemia, and acute blood pressure manipulation.

Blood sugar control

People with acute stroke should be treated to maintain a blood glucose concentration between 4 and 11 mmol/litre.

Provide optimal insulin therapy, which can be achieved by the use of intravenous insulin and glucose, to all adults with type 1 diabetes with threatened or actual stroke. Critical care and emergency departments should have a protocol for such management.

Blood pressure control

Anti-hypertensive treatment in people with acute stroke is recommended only if there is a hypertensive emergency with one or more of the following serious concomitant medical issues:

- hypertensive encephalopathy
- hypertensive nephropathy
- hypertensive cardiac failure/myocardial infarction
- aortic dissection
- pre-eclampsia/eclampsia
- intracerebral haemorrhage with systolic blood pressure over 200 mmHg.

Blood pressure reduction to 185/110 mmHg or lower should be considered in people who are candidates for thrombolysis.

Aspiration pneumonia is a complication of stroke that is associated with increased mortality and poor outcomes.

In people with dysphagia, food and fluids should be given in a form that can be swallowed without aspiration, following specialist assessment of swallowing.

The study group was divide dinto two groups those with and those without microalbumnuria and they have been compared in terms of age,gender,systolic or diastolic blood pressure,NIHSS,Modified Rankin Scale,LVH.

AIMS OF THE STUDY

1. To estimate the prevalence of microalbuminuria in non-diabetic acute ischemic stroke.

2. To assess the stroke severity using NIHSS and MODIFIED RANKIN SCALE.

METHODS AND MATERIALS

STUDY GROUP:

Patients with new onset acute ischemic stroke within 24 hours of presentation to Kilpauk Medical College Hospital

Study Design:

Descriptive study

Place of study :

Government Kilpauk Medical College Hospital

Period Of Study:

December 2014-June 2015. (6 months)

Collaborating Departments

The study was done with the help of Department of Biochemistry for various biochemical parameters and Departments of Radiology for Computed Tomography of brain to confirm an acute ischemic stroke.

Selection Of Study Subjects:

The study subject was selected from a total of 182 patients admitted with acute ischemic stroke presenting within 24 hours of onset.They were carefully analysed for the exclusion criteria and the sample size was shrunk to 53.

INCLUSION CRITERIA:

The patient profile studied included both female and male patients between the age group between 25-83 years admitted within 24 hours of onset of acute ischemic stroke as confirmed bv Computed Tomography.

EXCLUSION CRITERIA:

1.Positive urine analysis including hematuria,proteinuria,pyuria,glycosuria.

2.Systemic Hypertension.

3.Kidney diseases both congenital and acquired.

4.Diabetes Mellitus.

5.Liver disease

6.Chronic inflammatory intestinal Disease.

7.Neoplasm

8.Any sign of infection.

9.Acute coronary event or Coronary artery disease.

10.Other congenital or endocrine disorders.

11.Inflammatory rheumatic disease.

12.Dyslipdemia

13.Those on NSAIDS or immunosuppresants.

14.Fever

Details of study subjects

All patients at the time of admission had their history taken and underwent a detailed checkup.

The severity was calculated using the NIHSS (National Institute Of Health Stroke Scale).

The patient or caregiver were interviewed to establish the past medical or personal history.

All patents had their blood pressure and electrocardiogram taken.

Acute ischemic stroke was confirmed using Computed Tomography Of Brain.

Blood glucose levels,hematocrit,WBC ,blood urea and serum creatinine and electrolytes and the lipid profile of the patients were taken.

The spot urine albumin excretion was calculated and expressed in mg/dl.

NIHSS was calculated on day 1 and Modified Rankin Scale was calculated on day 14 of diagnosis.

STUDY DETAILS

In the study ,the subjects were divided into 2 groups ,those with microalbumnuria GROUP A and those without microalbuminuria-GROUP B.

These two groups were compared in terms of their age,gender,systolic and diastolic blood pressure,ECG evidence of left ventricular hypertrophy,Modified Rankin Scale and NIHSS .

SIGNFICANT CORELATION

Urine Albumin Excretion and Age

Urine albumin Excretion and Gender

Urine albumin Excretion and NIHSS

Urine albumin excretion and Modified Rankin Scale

Urine albumin excretion and Left Ventricular Hypertrophy

Urine albumin Excretion and Systolic and Diastolic blood pressure.

OBSERVATION AND ANALYSIS

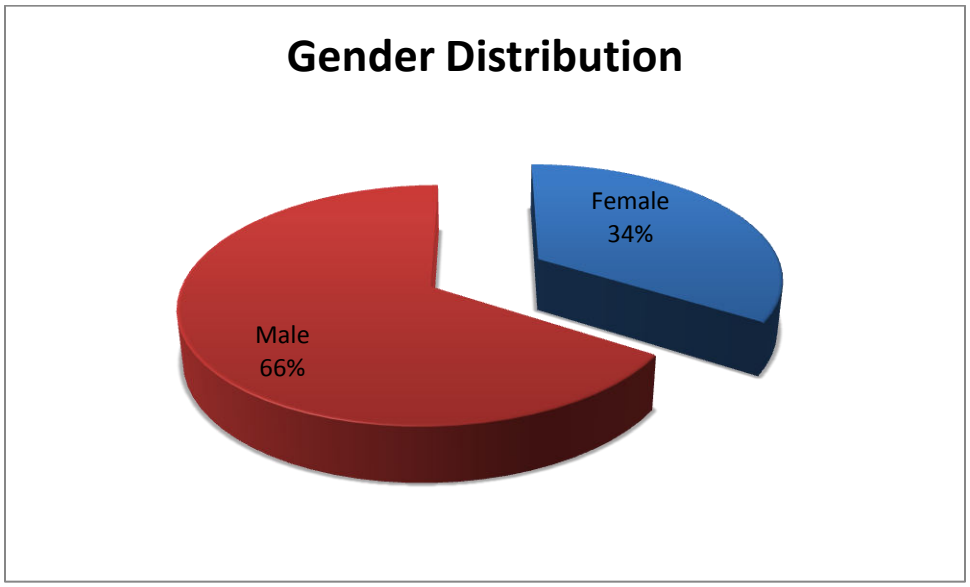
The study group has been divided into 2 groups

A-patients with microalbuminuria.

B-patients without microalbuminuria.

The two groups have been compared on the basis on age,gender.left ventricular hypertrophy,systolic and diastolic blood pressure,NIHSS and Modified Rankin Scale .

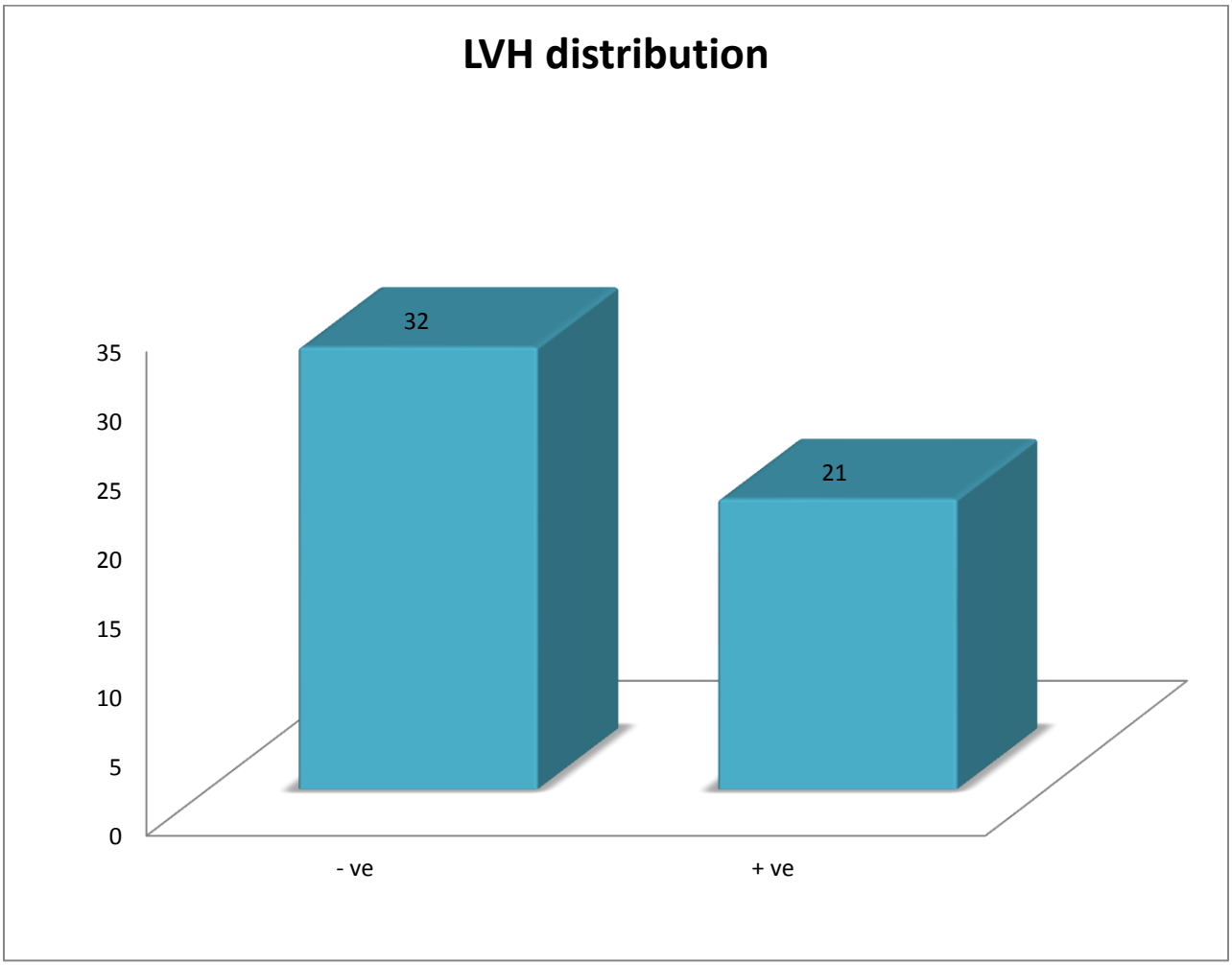
	Frequency	Percent
Female	18	34.0
Male	35	66.0
Total	53	100.0



The above diagram shows that in the study there was a majority of male of 66% and females constituted only 34%.

LVH

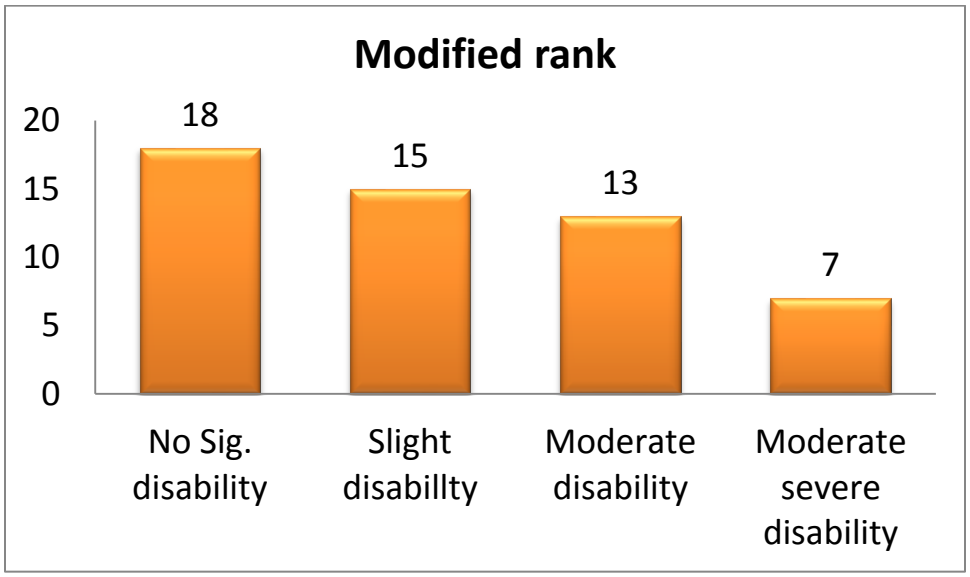
		Frequency	Percent		
Valid	- ve	32	60.4		
	+ ve	21	39.6		
	Total	53	100.0		



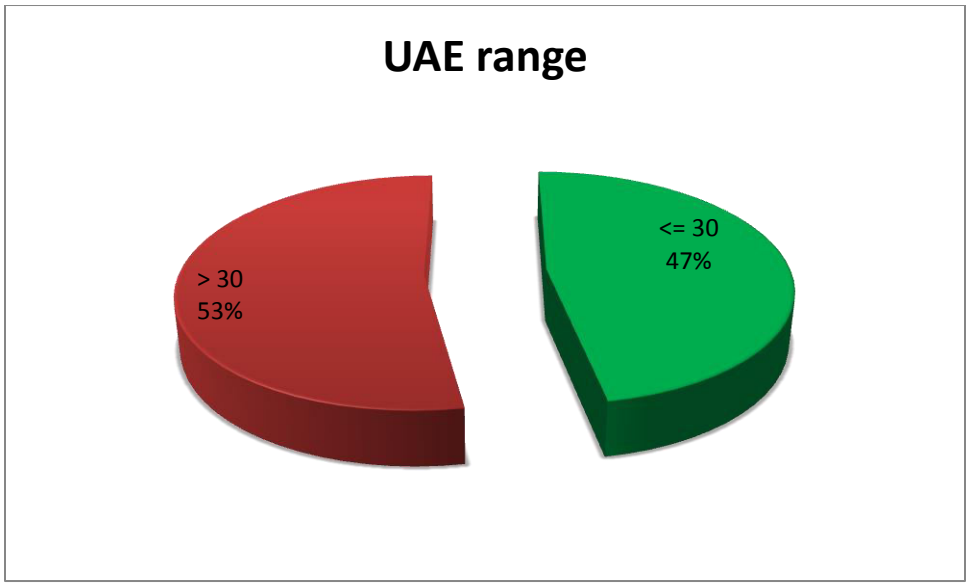
The above histogram showed that left ventricular hypertrophy was present in 39.6% who had microalbuminuria and it was present in 60.4% of those who did not have microalbuminuria.

MODIFIED RANKIN SCALE

	Frequency	Percent		
No Sig. disability	18	34.0		
Slight disabillty	15	28.3		
Moderate disability	13	24.5		
Moderate severe disability	7	13.2		
Total	53	100.0		



The above diagram shows that in the study group,18 suffered from no signifcant disability ,15 from slight disability,13 from moderate and 7 from moderate to severe disability.

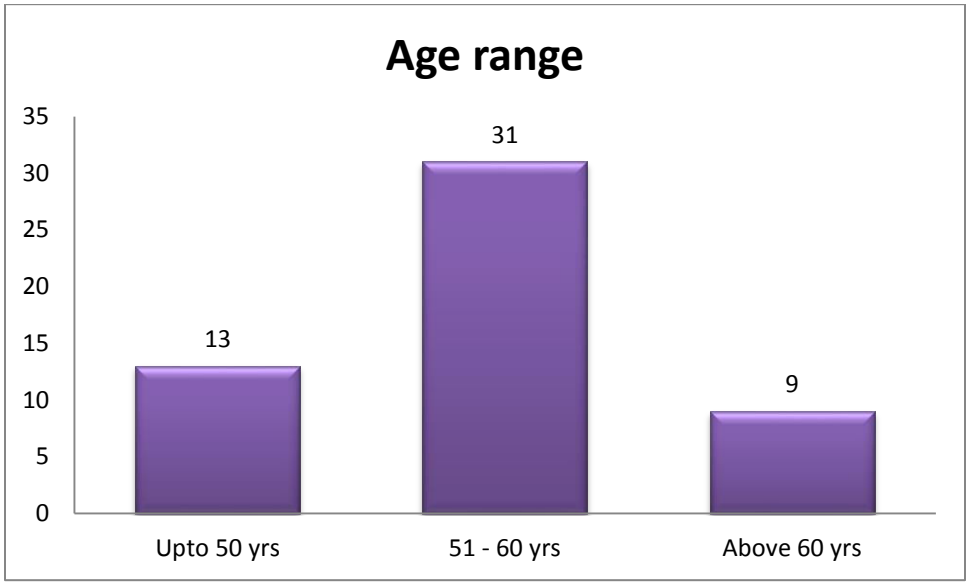


UAErange

		Frequency	Percent		
Valid	<= 30	25	47.2		
	> 30	28	52.8		
	Total	53	100.0		

In the study group,52.8 percent were microalbumnurics,and 47.2 percent were non-microalbuminurics

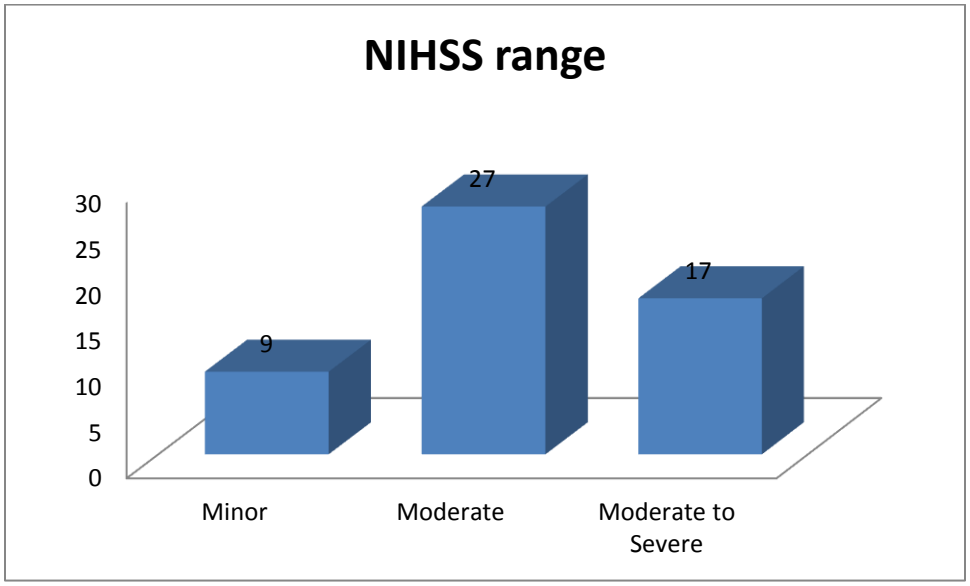
Age Range :



	Frequency	Percent
Upto 50 yrs	13	24.5
51 - 60 yrs	31	58.5
Above 60 yrs	9	17.0
Total	53	100.0

In the group,13 subjects were below 50 years of age,31 were between 51-60 years,and 9 were above 60 years of age.

NIHSS RANGE :



	Frequency	Percent
Minor	9	17.0
Moderate	27	50.9
Moderate to Severe	17	32.1
Total	53	100.0

In the study group,9 had only minor disability,27 had moderate ,17 had moderate-severe disability.

Descriptive Statistics:

	N	Minimum	Maximum	Mean	Std. Deviation
AGE	53	43	80	55.77	7.617
SBP	53	124	176	147.92	13.020
DBP	53	70	110	90.94	10.916
NIHSS	53	1	20	10.79	5.415
UAE	53	6	184	54.91	47.126
	53				

GENDER = Female

Descriptive Statistics[a]

	N	Minimum	Maximum	Mean	Std. Deviation

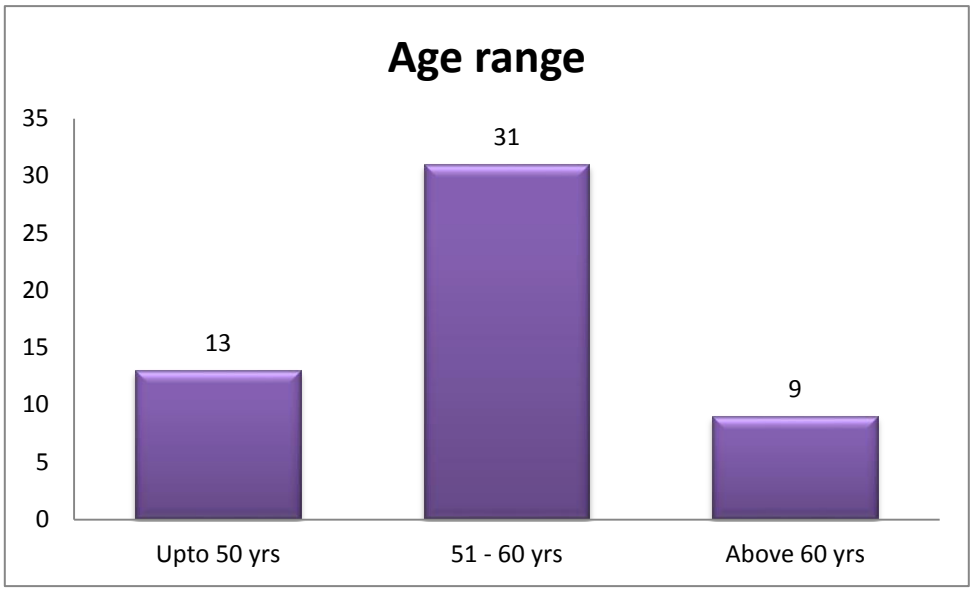
AGE	18 18	45	80	58.22	9.687

GENDER = Female

	N	Minimum	Maximum	Mean	Std. Deviation
AGE	35 35	43	70	54.51	6.085

a. GENDER = Male

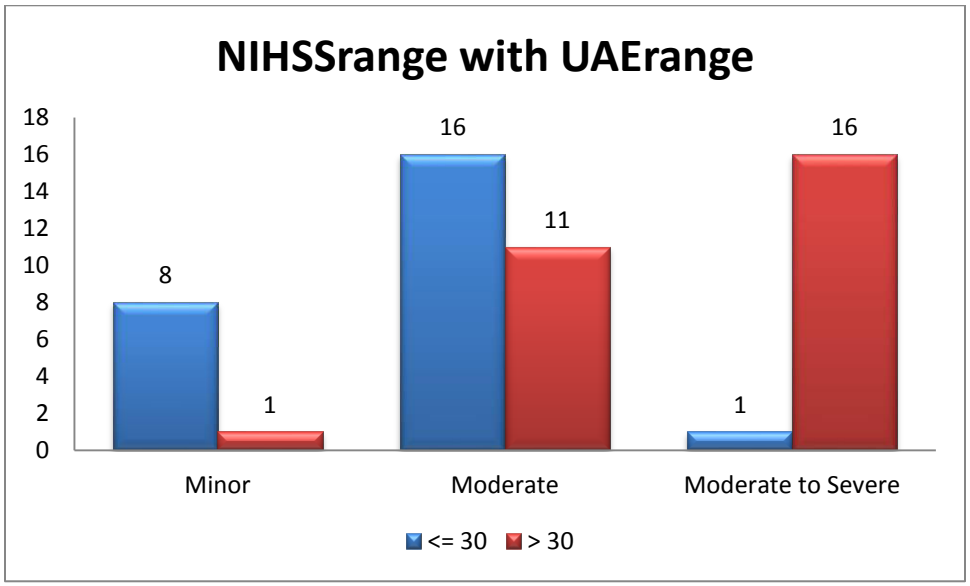
He mean age among the female population was 58.22 with a standard deviation of 9.687.and among the male population was 54.51 with a standard deviation of 6.085.



NIHSSrange vs UAErange

			UAErange		Total
			<= 30	> 30	
NIHSSrange	Minor	Count	8	1	9
		% within UAErange	32.0%	3.6%	17.0%
	Moderate	Count	16	11	27
		% within UAErange	64.0%	39.3%	50.9%
	Moderate to Severe	Count	1	16	17
		% within UAErange	4.0%	57.1%	32.1%
Total		Count	25	28	53
		% within UAErange	100.0%	100.0%	100.0%

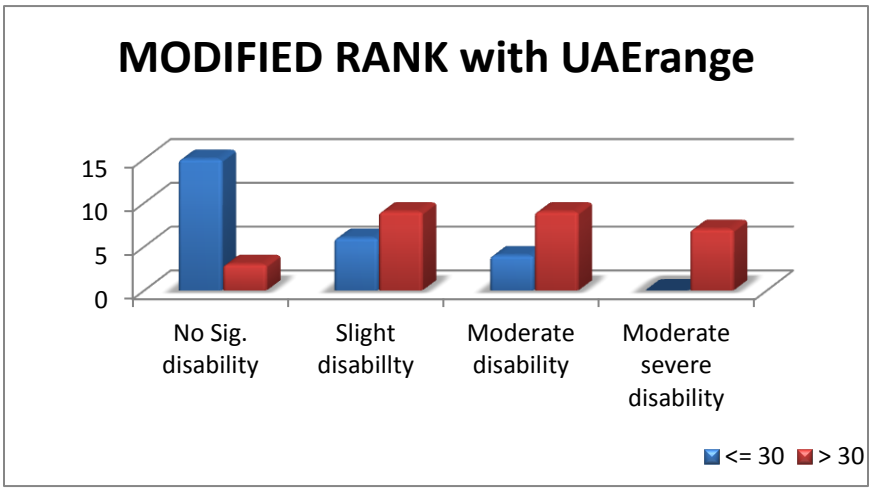
	<= 30	> 30
Minor	8	1
Moderate	16	11
Moderate to Severe	1	16



From the above study 8 among the non-microalbumnurics and 1 microalbumnuric suffered from minor disability.16 among the non-microalbumnurics and 11 microalbumnurics suffered from moderate and 1 from non-microalbummuric and 16 from microalbumnuric suffered from severe disability.This shows a statistically significant relation exists between NIHSS and urine albumin excretion with a p value of .000

			UAErange		
			<= 30	> 30	Total
MODIFIED RANKIN	No Sig. disability	Count	15	3	18
		% within UAErange	60.0%	10.7%	34.0%
	Slight disabillty	Count	6	9	15
		% within UAErange	24.0%	32.1%	28.3%
	Moderate disability	Count	4	9	13
		% within UAErange	16.0%	32.1%	24.5%
	Moderate severe disability	Count	0	7	7
		% within UAErange	0.0%	25.0%	13.2%
Total		Count	25	28	53
		% within UAErange	100.0%	100.0%	100.0%

	<= 30	> 30
No Sig. disability	15	3
Slight disabillty	6	9
Moderate disability	4	9
Moderate severe disability	0	7

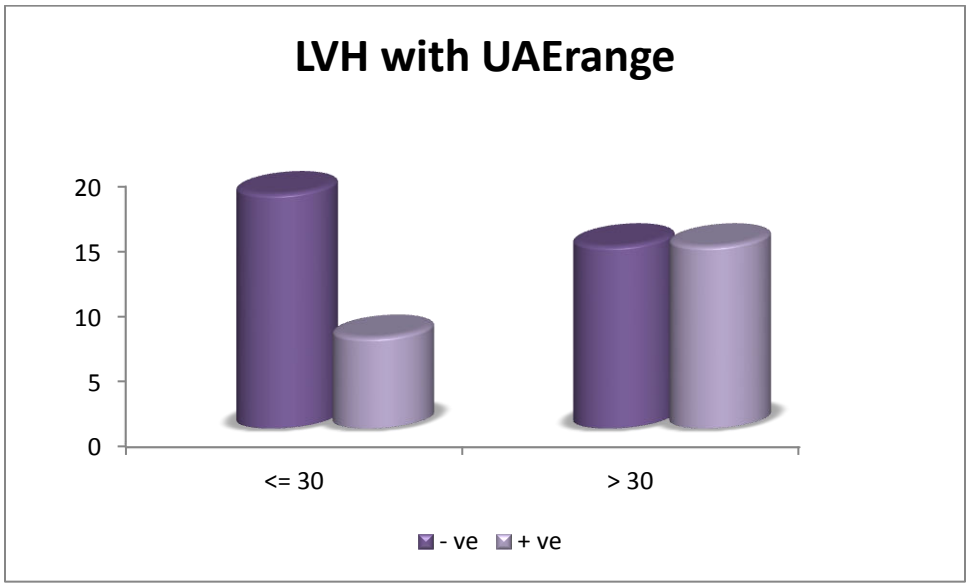


The above table shows that a statistical significance exists between modified Rankin Scale and urine albumin excretion with a p value of .001.

LVH vs UAE range :

			UAErange		
			<= 30	> 30	Total
LVH	- ve	Count	18	14	32
		% within UAErange	72.0%	50.0%	60.4%
	+ ve	Count	7	14	21
		% within UAErange	28.0%	50.0%	39.6%
Total		Count	25	28	53
		% within UAErange	100.0%	100.0%	100.0%

	<= 30	> 30
- ve	18	14
+ ve	7	14



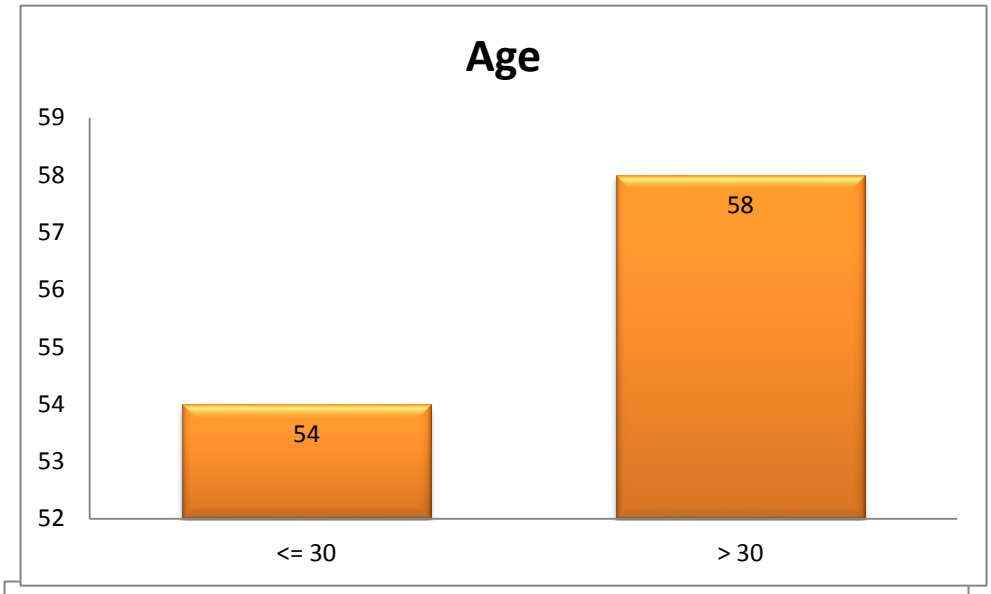
From the above diagram it is evident that no significant relation exists between left ventricular hypertrophy and urine albumin excretion with a p value of only .102

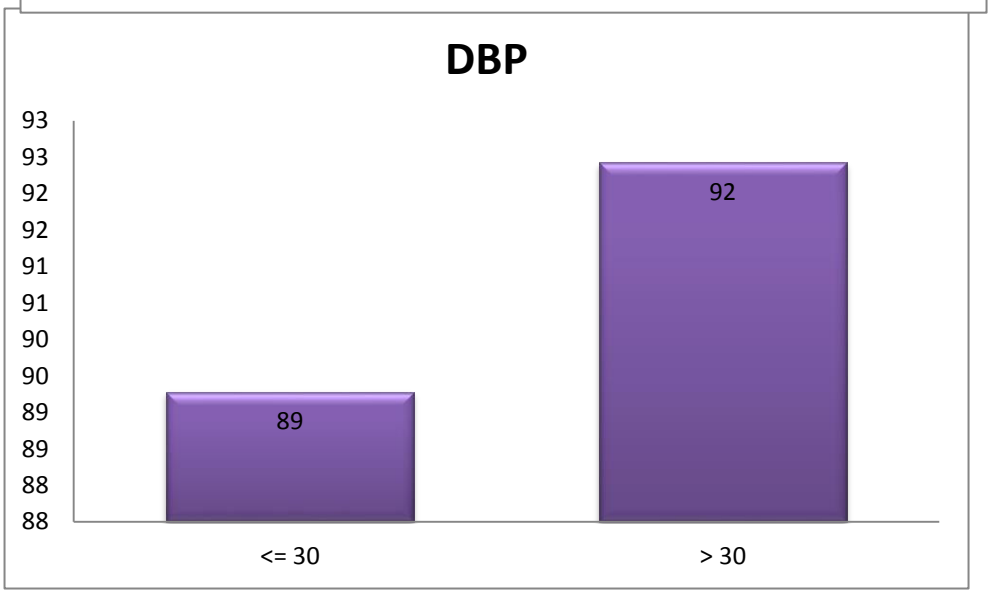
GENDER vs UAErange

			UAErange		
			<= 30	> 30	Total
GENDER	Female	Count	9	9	18
		% within UAErange	36.0%	32.1%	34.0%
	Male	Count	16	19	35
		% within UAErange	64.0%	67.9%	66.0%
Total		Count	25	28	53
		% within UAErange	100.0%	100.0%	100.0%

	<= 30	> 30
Female	9	9
Male	16	19

No statistically significant correlation exists between gender and urine albumin excretion with a p value of .767


Age
59
58
57
56
55
54
53
52
54
58
<= 30
> 30


DBP
93
93
92
92
91
91
90
90
89
89
88
88
89
92
<= 30
> 30

SBP

<= 30	146
> 30	150

DBP

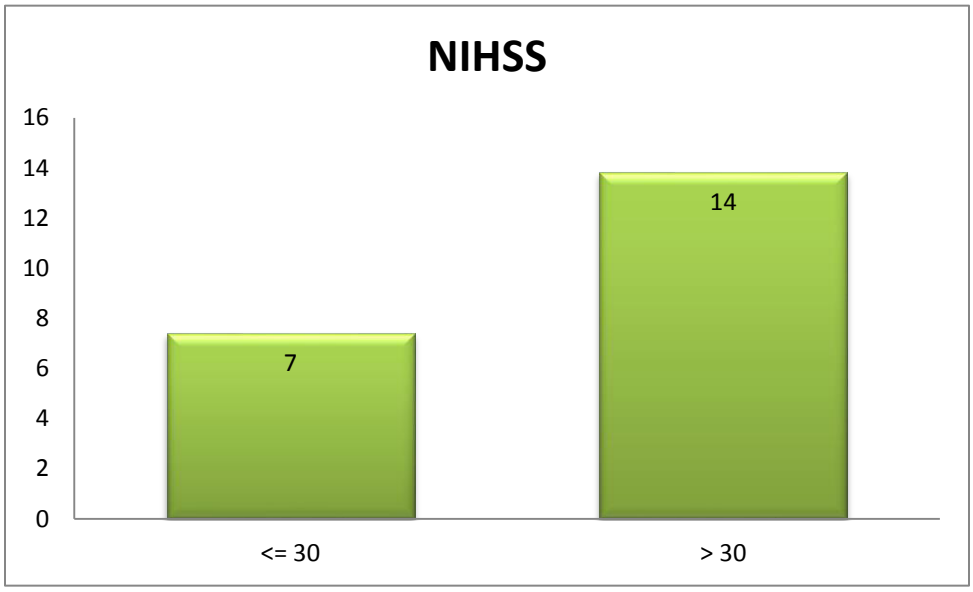
<= 30	89
> 30	92

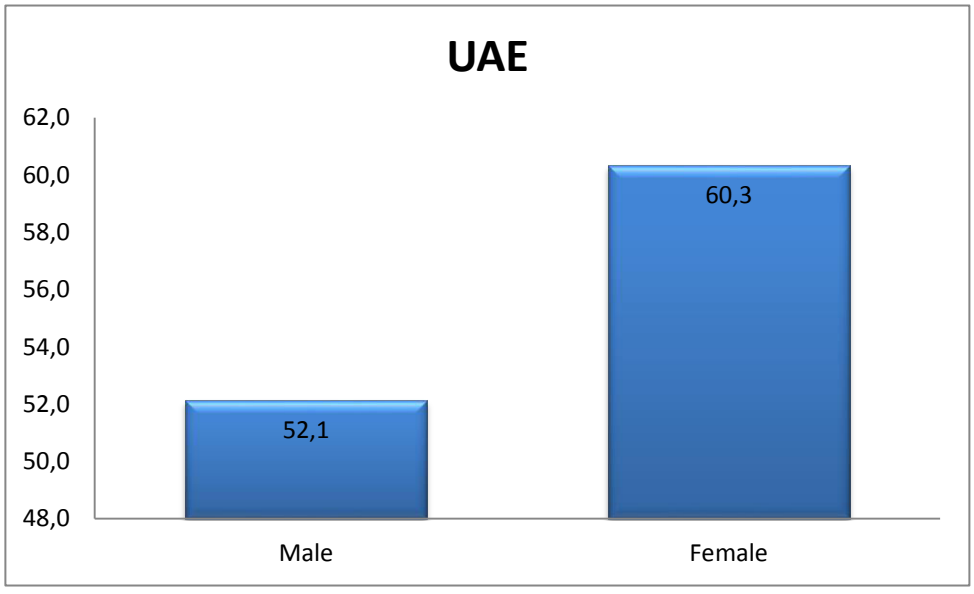
From the above study no significant corelation exists between systolic or diastolic blood pressure and urine albumin excretion.

However there was a statistically significant corelation between age and microalbuminuria with a p value of .001.

NIHSS

<= 30	7
> 30	14


NIHSS
16
14
12
10
8
6
4
2
0
7
14
<= 30
> 30


UAE
62,0
60,0
58,0
56,0
54,0
52,0
50,0
48,0
52,1
60,3
Male
Female

	UAE
Male	52.1
Female	60.3

However a significant corelation exists between NIHSS and urine albumin excretion.

GENDER		N	Mean	Std. Deviation	Std. Error Mean
UAE	Male	35	52.11	38.097	6.440
	Female	18	60.33	61.992	14.612

UAErange		N	Mean	Std. Deviation	Std. Error Mean
AGE	<= 30	25	53.64	5.338	1.068
	> 30	28	57.68	8.857	1.674
SBP	<= 30	25	145.76	11.537	2.307
	> 30	28	149.86	14.141	2.672
DBP	<= 30	25	89.28	12.661	2.532
	> 30	28	92.43	9.065	1.713
NIHSS	<= 30	25	7.40	4.193	.839
	> 30	28	13.82	4.546	.859

DISCUSSION

The relation between microalbumnuria and cerebrovascular diseases has been studied in the west and there seems to have been an association between the two.

This study has been undertaken to see the relevance in our population.A total of 53 patients was taken as the sample size after applying the exclusion criteria.

STRENGTH OF THE STUDY

There has been an association found between microalbuminuria and other conditions like systemic hypertension,diabetes mellitus and coronary artery diseases,But an association between microalbumnuria and cerebrovascular diseases without the presence of other risk factors was yet to be ascertained.So,the crux of the study is to determine whether microalbuminria is individually a risk factor.

LIMITATION OF THE STUDY

As the study was carried out in a tertiary care centre ,it cannot be ascertained if the results of the study can be reproduced even in a periphery care setting.

Another drawback of the study was that since the stroke severity grading was to be done in two days some patients did not respond to the study and some had died .Those who died were not included in the study.

PREVALENCE OF MICROALBUMINURIA :

The prevalence of microalbuminuria in non-diabetic acute ischemic stroke was said to be 52.8percent.

Previous studies were done to show an asssociaton between stroke and microalbuminuria using Glasgow coma scale and was found to be 47 percent.

The prevalence of microalbuminuria in stroke is similar to that reported in other conditions like diabetes in which a prevalence of 40 percent was reported. In hypertensives,it was said to be present in 37percent.However the incidence is said to be relatively higher in causing ischemic stroke.

PROGNOSTIC SIGNIFICANCE OF MICROALBUMINURIA

In earlier studies relation between ischemic stroke and microalbumnuria was studied using the Glasgow Coma Scale and was found to be significant.It was found that lower score was found in those with microalbumnuria.

Other scale used earlier was Scandinavian Stroke Scale . Hence measurement of microalbuminuria was important and treatment was considered important in preventing strokes.

GENDER AND URINE ALBUMIN EXCRETION

From the study it seems evident that there seems no relation between gender and urine albumin excretion is those diagnosed with acute ischemic stroke.In this study we had 66 percent of the individuals and 34 percent as females.

AGE AND MICROALBUMINURIA

Contrary to what we expected we had a higher population in the age group between 51-60 figuring 31.Below 50 years we had only 13 individuals and above 61 years we had a total population of 9.The relation between albumin excretion and age was found to be significant with a mean age of 53 among the non microalbuminurics and 58 among the microalbumnurics showing a statistical significance.However there was a significant association has been found between age and microalbminuria.

BLOOD PRESSURE AND MICROALBUMINURIA IN STROKE

Blood pressure has always been a confounding factor in stroke because of a certain degree of dysautonomia at the time of stroke which cause an erroneous result showing a falsely elevated or depressed blood pressure.In this study there seems to be no difference between systolic or diastolic blood pressure and microalbuminuria qualitatively or quantitatively. Among the

microalbumbnurics,150 mm of hg was the mean systolic blood pressure and 146 mm of Hg among the non-microalbminurics.

Among the microalbmnurics,mean diastolic blood pressure was 92mm of hg and 89 mm of Hg among the nonmicroalbumnurics.

MICROALBMINURIA AND LEFT VENTRCULAR HYPERTROPHY

In this study there seems to be no association between microalbuminuria and left ventricular hypertrophy.

MICROALBUMINURA AND MODIFIED RANKIN SCALE

This scale was used to fnd out the stroke severity on day 15 from the diagnosis of ischemic stroke.It was found that 3 among all the patients suffered from no significant disability,9 of them suffered from slight disability,9 from moderate and 7 from moderate to severe disability.Among the nonmicroalbuminrcs,15 of the patients had no significant disability,6 had slight disability and 4 from moderate disabilty.And from the study,it has been concluded that

a significant association was found between modified rankin scale and microalbuminuria .

NIHSS AND MICROALBUMINURIA

Among the microlalbumnurics ,1 patient suffered from minor disability,11 from moderate disability and 16 from moderate o severe disability.Among the nonmicroalbumnurics 8 suffered from minor significant disability,16 from moderate disabily and 1 from moderate to severe disability. And hence from ths study it could be concluded that there was a statistical significance between NHSS and microalbuminuria.

CONCLUSION

Prevalence of microalbuminuria in acute ischemic stroke was found out to be 52.8 percent from this study.

Urine albumin excretion had a very significant statistical association with Modified Rankin Scale and a higher rate of albumin excretion was associated with higher score.

Urine albumin excretion had a significant statistical corelation with NIHSS and a high urine albumin excretion was associated with higher score.

There also seemed to be a significant association between microalbuminuria and age from this study.

No significant association was found between microalbuminuria and systolic or diastolic blood pressure from this study.

No significant association has been found between microalbuminuria and gender from this study .

No significant association has been found out between microalbuminuria and left ventricular hypertrophy,

SUMMARY

Microalbumnria has long been associated with nephropathy due to various causes and an early indicator of renal involvement including in conditions like malignancy and sickle cell disease.This study has been shown to be significant as studies regarding its importance in cerebrovascular diseases has been carried out in the west but far and few in our country. Hence a study to relate is occurance in stroke has been studied.The sample size was chosen after applying the exclusion criteria t rule out other causes of microalbuminuria and hence a direct association to find out the stroke severity using NIHSS scale on the day of diagnosis of the ischemic stroke and Modified Rankin Score on day 15 after diagnosing stroke was done. A comparison between urine albumin excretion and age ,systolic and diastolic blood pressure,gender ,left ventricular hypertrophy,NIHSS and Modifed Rankin was done and was found that a statistical significance was present between microalbuminuria and NIHSS ,Modified Rankin.

Hence microalbuminuria was individually found to be a risk factor in ischemic stroke but studies concerning if treating microalbuminuria would be helpful n preventing further strokes are yet to be reported.

Further studies are required to see if microalbuminuria can be used as a predictor of vascular accidents of heart,brain,kidney and other disease processes where blood vessels are involved.

ANNEXURES

A- ABBREVIATIONS

CT-computed tomography.

UAE-urine albumin excretion.

NIHSS-national institute of health stroke scale.

MRS-modified Rankin Scale

DWI-diffusion weighted imaging.

PET-positron emission tomography

B-MASTERCHART

sl no.	name	AGE	GENDER	SBP	DBP	LVH	NIHSS	mod.rank	UAE
1	S	62	M	164	110	+	17	3	114
2	S	60	M	150	94	-	3	1	18
3	A	53	M	124	70	-	16	1	86
4	S	53	M	130	80	+	2	1	20
5	I	52	M	164	110	+	16	3	25
6	C	57	M	130	80	-	5	2	56
7	D	58	M	1489	90	+	15	2	60
8	J	51	M	138	80	-	1	1	6
9	Y	60	M	176	98	+	17	2	104
10	U	60	M	130	70	-	2	1	13
11	L	54	M	170	100	+	18	4	124
12	D	54	M	154	96	+	3	1	17
13	K	64	M	154	100	-	4	2	18
14	U	70	M	166	98	+	16	2	88
15	R	60	M	140	92	-	4	2	15
16	P	64	M	136	84	-	9	1	38
17	A	54	M	130	80	-	12	4	71
18	J	43	M	154	92	+	4	3	45
19	S	57	M	170	110	+	5	2	17
20	M	45	M	160	100	-	5	1	22
21	G	45	M	154	90	-	16	2	40
22	S	47	M	154	90	+	14	3	24
23	B	45	M	150	90	-	5	1	20
24	K	50	M	136	70	-	4	1	18
25	S	54	M	140	92	+	17	3	54
26	S	54	M	130	80	-	12	3	24
27	L	56	M	140	94	+	7	3	50
28	K	53	M	128	70	-	10	1	22
29	M	60	M	130	80	-	17	4	100
30	M	56	M	136	76	-	9	1	19
31	R	48	M	154	96	+	20	4	110
32	S	50	M	160	100	+	17	3	96

33	M	59	F	140	82	-	8	1	18
34	K	60	F	154	104	+	7	1	12
35	T	52	M	154	100	-	16	2	56
36	S	48	F	160	110	+	7	2	7
37	Z	50	M	148	98	-	16	4	110
38	H	45	F	154	100	-	10	1	8
39	I	57	M	150	96	-	18	4	124
40	R	53	F	170	100	+	18	3	130
41	U	49	F	140	90	-	12	1	10
42	N	80	F	164	100	+	16	3	160
43	R	52	F	148	96	-	12	1	12
44	K	75	F	166	102	+	18	4	170
45	K	54	F	138	86	-	12	2	14
46	S	71	F	150	100	-	14	3	184
47	J	54	F	148	98	-	14	2	90
48	H	55	F	142	80	-	7	3	17
49	S	50	F	130	80	-	10	3	94
50	M	58	F	144	76	-	11	2	14
51	K	64	F	140	86	-	7	2	48
52	M	67	F	144	84	-	10	2	44
53	M	54	F	156	90	+	7	1	54

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D- PROFORMA

Name

Age

Sex

IP/OP NO:

Marital Status:

Educational Status:

CLINICAL STATUS:

Duration Of Weakness of limbs:

c/o fever or any symptoms s/o infection:

c/o any cardiac complaints s/o any cardiac event:

PAST HISTORY:

Diabetes:

Hypertension:

Ischemic heart disease;

Kidney disease,congenital or acquired:

Liver Disease:

Chronic inflammatory gastrointestinal disease:

Dyslipidemia:

Rheumatic Inflammatory disease:

DRUG HISTORY:

Nonsteroidal anti-inflammatory drugs:

Immunosuppresants:

PERSONAL HISTORY:

Smoking:

Alcohol:

Diet:

FAMLY HISTORY:

Diabetes:

Hypertension

PERSONAL HISTORY:

Exposure to sexually transmitted diseases:

EXAMINATION:

Height (metres):

Weight(kilograms):

Body Mass Index:

VITAL SIGNS:

a)Pulse rate:

b)respiratory rate

c)blood pressure:

d)temperature:

SYSTEMIC EXAMINATION:

Cardiovascular status:

Neurological status:

Respiratory status:

INVESTIGATIONS:

Urinealbumn,sugar,deposits:

Blood urea:

Serum creatinine:

Liver function tests:

Serum electrolytes:

Complete hemogram :

Chest X-Ray(Posteroanterior view):

NIHSS SCORING(ON DAY 1):

	SCORE
Level of consciousness	0-3
Horizontal eye movement	0-2
Visual field test	0-3
Facial palsy	0-3
Motor arm(right and left)	0-8
Motor leg(right and left)	0-8
Limb ataxia	0-2

Sensory	02
Language	0-3
speech	0-2
Extinction and inattention	0-2

Total score

SCORE	STROKE SEVERITY
0	No stroke symptoms
1-4	Minor stroke
5-15	Moderate stroke
16-20	Moderate-severe stroke
21-42	Severe stroke

INVESTIGATIONS:

Microalbuminuria:

CT Brain plain:

Modified rankin scale(assessed after 15 days for functional outcome):

0-no symptoms

1-no significant disability.Able to carry out all ususal activities,despite some symptoms.

2-slight disability.Able to look after own affairs withot assistance,but unable to carry out all previous activities.

3-moderate disability.Requires some help,but able to walk unassisted.

4-moderate to severe disability-unable to attend to own bodily needs and unable to walk unassisted.

5-severe disability-Requires constant nursing care and attention,bedridden and incontinent.

6-dead.

E- ETHICAL COMMITTEE CLEARANCE CERTIFICATE

INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Protocol ID. No.6/02/2015 Dt:01/02/2015
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "Microalbuminuria in non-diabetic acute ischemic stroke – prevalence and stroke severity"- For Project Work submitted by Dr.Umalakshmi Premnath, Post Graduate in MD (GM), Govt. Kilpauk Medical College, Chennai.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

CHAIRMAN, 27/3/15
Ethical Committee
Govt. Kilpauk Medical College, Chennai

27/3/15

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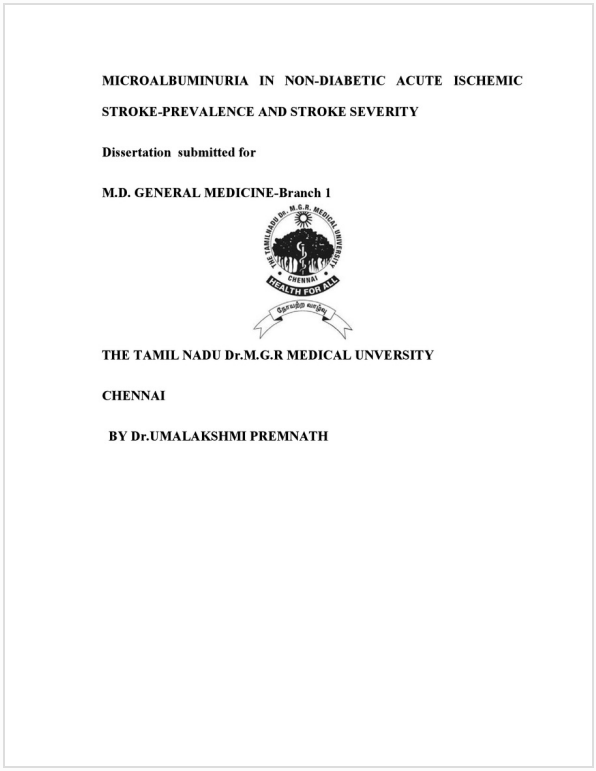

MICROALBUMINURIA IN NON-DIABETIC ACUTE ISCHEMIC STROKE-PREVALENCE AND STROKE SEVERITY

Dissertation submitted for

M.D. GENERAL MEDICINE-Branch 1

THE TAMIL NADU Dr.M.G.R MEDICAL UNVERSITY

CHENNAI

BY Dr.UMALAKSHMI PREMNATH