

**A STUDY ON MICROALBUMINURIA IN
NON DIABETIC RECENT ISCHEMIC
STROKE PATIENTS**

Dissertation submitted for

**MD Degree (Branch-I)
General Medicine**



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CERTIFICATE

This is to certify that this dissertation titled **A STUDY ON MICROALBUMINURIA IN NON DIABETIC RECENT ISCHEMIC STROKE PATIENTS**” submitted by **DR. B.GOWRIPATHY** to the faculty of General Medicine, The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of MD degree Branch I (General Medicine) is a bonafide research work carried out by his under our direct supervision and guidance.

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DECLARATION

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This is submitted to the Tamil Nadu Dr .M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of MD degree Branch I (General Medicine).

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INTRODUCTION

Cerebrovascular diseases include one of most common and devastating disorders worldwide. These include ischemic stroke, haemorrhagic stroke, AV malformations, and intracranial aneurysms. This causes one of the major causes of disability. There are major risk factors for the stroke which can be prevented by awareness and prevention programs and warning signals by the public and stress on the preventive training in medical education will reduce the stroke prevention gap.

There is a growing interest in the pathogenesis of ischemic stroke because nearly fifty percent of cerebrovascular disease risk factors identified were traditional risk factors. Hence leads to search for new risk factors and treatment. The markers of inflammation like C- reactive protein, intracellular adhesion molecule-1, lipoprotein associated phospholipase A2, increased white blood cell count, endothelial nitric oxide synthase, infectious etiologies like Chlamydiae pneumonia, Helicobacter pylori and cytomegalovirus, Homocysteine, Tissue factor Fibrinogen,

Lipoprotein (a), Small dense LDL, cytokine transforming growth factor, etc, have been identified as new risk factors for stroke. Another upcoming risk factor for the growing list is said to be microalbuminuria.

Microalbuminuria has been associated with disease like diabetic nephropathy, hypertension with left ventricular hypertrophy and renal insufficiency etc. Microalbuminuria has been associated with risk factors for stroke like diabetes, hypertension aging, history of myocardial infarction, obesity, smoking. But there was meager knowledge in connection as independent risk factor for stroke or as a predictor of stroke outcome. So there were many studies in the world to demonstrate the microalbuminuria as a risk factor for stroke and predictor of stroke outcome in non diabetic individuals.

AIMS AND OBJECTIVES

1. To determine the presence of microalbuminuria in the recent ischemic cerebrovascular disease who are non – diabetic individuals.
2. To study the prognostic significance of microalbuminuria in recent ischemic cerebrovascular individuals.

REVIEW OF LITERATURE

ISCHEMIC STROKE

DEFINITION

Stroke

A stroke or cerebrovascular accident is defined by abrupt onset of neurological deficit that is attributable to focal vascular cause. Thus the definition of stroke is clinical and laboratory studies including the brain imaging support the diagnosis.

Transient Ischemic Attack

TIA is defined that neurological signs and symptoms resolve within 24 hours regardless whether there is imaging evidence of new permanent brain injury.

TIA per se does not impose any lasting burden on the individual or the society. But it serves as warning signal for later occurrence of stroke and thus may form the basis of high risk prevention strategy.

Stroke in Evolution

Is a progressive neurological deficit developing over few hours or days, which evolves to Completed stroke after a few hours or days.

Completed Stroke

Is a stroke syndrome in which the deficit is prolonged and often permanent causing demonstrable parenchymal damage.

Small Vessel Stroke

Is the infarction following atherothrombotic or lipohyalinotic occlusion of a small artery in the Brain

CLASSIFICATION

Oxfordshire stroke sub – classification

A. Anterior Circulation Syndrome (total)

It is a stroke involving a big cortical area either MCA or both MCA AND ACA territories.

It consists of:

- cerebral dysfunction
- Homonymous visual field defect.
- Both motor and sensory deficit involving the same side either arm, face and leg.

B. Anterior Circulation Syndrome (partial)

- It includes a moderate form of stroke involving MCA or ACA territory.
- These people will have 2 out of 3 criteria of

The total anterior circulation syndromes or involving Cerebral dysfunction only or both motor /sensory defect

confined than those defined as total anterior circulation syndrome

C. Lacunar Syndrome

- includes a subcortical stroke due to the occlusion of penetrating vessel
- involvement of either higher mental function or loss of consciousness rules out lacunar stroke.

D. Posterior Circulation Syndrome

- involvement of same side cranial nerve palsies and opposite side motor and sensory defects.
- Motor and sensory defects of both the sides.
- Defects in conjugate eye movements with cerebral dysfunction not involving the pyramidal tract of the same side.
- Isolated homonymous visual field defects.

2 Hachinske and Norris classification

A. presumed stroke

Presumed TIA

B. Anatomic classification

- a. By vascular supply
 - Carotid
 - Vertebrobasilar
- b. By location
 - Supratentorial
 - lobar
 - Ganglionic/thalamic
 - Infratentorial
 - Cerebellar
 - Brain stem

C. Etiological classification:

- a. By result
 - Cerebral Infarct
 - Arterial
 - Arteriolar
 - Venous
 - Cerebral haemorrhage
 - Parenchymal
 - Subarachnoid
- b. By cause
 - Ischemia
 - Embolism
 - Extra cranial
 - vascular disease
 - Haemorrhage
 - Hypertension
 - Amyloid
 - Angiopathy
 - Vascular malformation
 - Aneurysm

EPIDEMIOLOGY

Stroke is one of the leading cause of morbidity in adults worldwide. The morbidity and mortality associated with stroke are Global.

- 400-800 strokes per 100,000
- 5.7 million deaths
- 16 million new acute stroke every year
- Disability adjusted life year (DALYS) around 28 lakhs
- 4 week case fatality rate ranges from 18% - 36%

INDIA

- Prevalence 90- 222 per 100,000
- 102,620 million deaths.
- In each year the incidence of strokes cases were 1.4 to 1.6 million
- 6,398,000 DALYs
- The incidence of stroke below 40 years were around 12%.
- 4 week case fatality rate ranges from 17%- 36%

THE INCIDENCE AND PREVALENCE OF STROKE IN INDIA

The data of stroke were more restricted and confined to frequent bias in sample size and vague eligibility criteria. But prevalence rate conducted at 2004 by Gourie devi at Karnataka was

Urban – 136 per 100,000

Rural - 165 per 100,000

The annual stroke occurrence in India by Sridharan (2009) at Trivandrum

Urban – 116 per 100,000

Rural – 124 per 100,00

by Bhattacharya (2005) at West Bengal.

RISK FACTORS FOR STROKE

1. Age :

It is one of the strongest risk factor for stroke. It found to be 25 times more common in people aged 75- 85 years than in people aged 45- 55 years.

2. Gender:

There is marginal rise of male preponderance in middle and old age.

3. Blood pressure :

Hypertensive patients are strongly associated with stroke risk it correlates with both the systolic and diastolic BP. There is a relative risk of cerebrovascular disease in hypertension both in men and women.

4. Smoking:

There is a definite risk for stroke . It has been established that there is a dose – response relationship, affecting both sexes in all age groups .

5. Serum lipids :

There is a relationship between serum lipoprotein (a)

6. Diabetes Mellitus:

Patients with diabetes have twice the risk for acquiring the stroke when compared to non diabetic individuals. Stroke in diabetics will be very fatal.

7. Hypercoagulable states:

Increased fibrinogen, increased plasma factor VII coagulant activity, decreased fibrinolytic activity, raised von Willebrand factor and raised haematocrit are all risk factors for stroke.

8. Atrial fibrillation

Is one of the most common cause of cardio-embolic strokes when particularly with patients associated with diabetes, hypertension, smoking, h/o previous peripheral vascular diseases.

9. Alcohol :

Moderate consumption of alcohol may be protective for ischemic stroke . But it also raises the blood pressure, alters lipid

profile, increases the chance of AF and cardiomyopathy which may increase the risk of stroke.

10. Diet:

Omega 3 fatty acids and unsaturated fatty acids in take may reduce stroke risk. Increase in salt in take increases blood pressure and stroke risk.

11. Vascular disease:

Coronary artery disease, asymptomatic peripheral vascular disease and TIA are all associated with increased stroke risk.

12. Hereditary factors:

Connective tissue disorders such as Marfans syndrome, MVP, Fibromuscular dysplasia, Pseudoxanthoma elasticum, Ehlers- danlos syndrome, haematological disorders like protein C deficiency, dysfibrinogenemia, antithrombin III deficiency, sickle cell disease, familial hypercholesterolemia, homocysteinemia, cardiac myxoma, Fabrys disease are linked with higher risk for stroke.

ETIOLOGY OF ISCHEMIC STROKE

COMMON

UNCOMMON

Thrombosis	Hypercoagulable disorders
Lacunar stroke	Protein S deficiency
Large vessel thrombosis	Factor V leiden
Dehydration	Antithrombin deficiency
Embolic occlusion	Antiphospholipid syndrome
Artery-to- artery	Protein C deficiency
Arterial dissection	Systemic malignancy
Carotid bifurcation	Sickle cell anaemia
Aortic arch	Prothrombin G 20210 Mutation
Cardioembolic	Polycythemia vera
Atrial fibrillation	Homocysteinemia
Mural thrombosis	Thrombocytopenic purpura
Dilated cardiomyopathy	Systemic lupus erythematosus
Myocardial infarction	Beta Thalssemia
Valvular lesions	Dysproteinemias
Mitral stenosis	Oral contraceptives
Mechanical valve	Nephrotic syndrome
Infective endocarditis	Inflammatory bowel disease

Paradoxical embolism	Disseminated Intravascularcoagulation
Atrial septal aneurysm	Venous sinus thrombosis
Spontaneous ECHO contrast	Fibromuscular dysplasia
	Vasculitis
	Primary CNS vasculitis
	Sytemic vasculitis(PAN, Wegener's,Takayusu,s Giant cell arteritis) Meningitis (Syphilis, fungal , Herpes Zoster)
	Cardiogenic
	Mitral valve calcification
	Itracardiac tumour
	Atrial myxomas
	Libman 's sacks endocarditis
	Subarchnoid haemorrhage vasopasm
	Drugs- cocaine, amphetamine
	Eclampsia
	Moyamoya disease

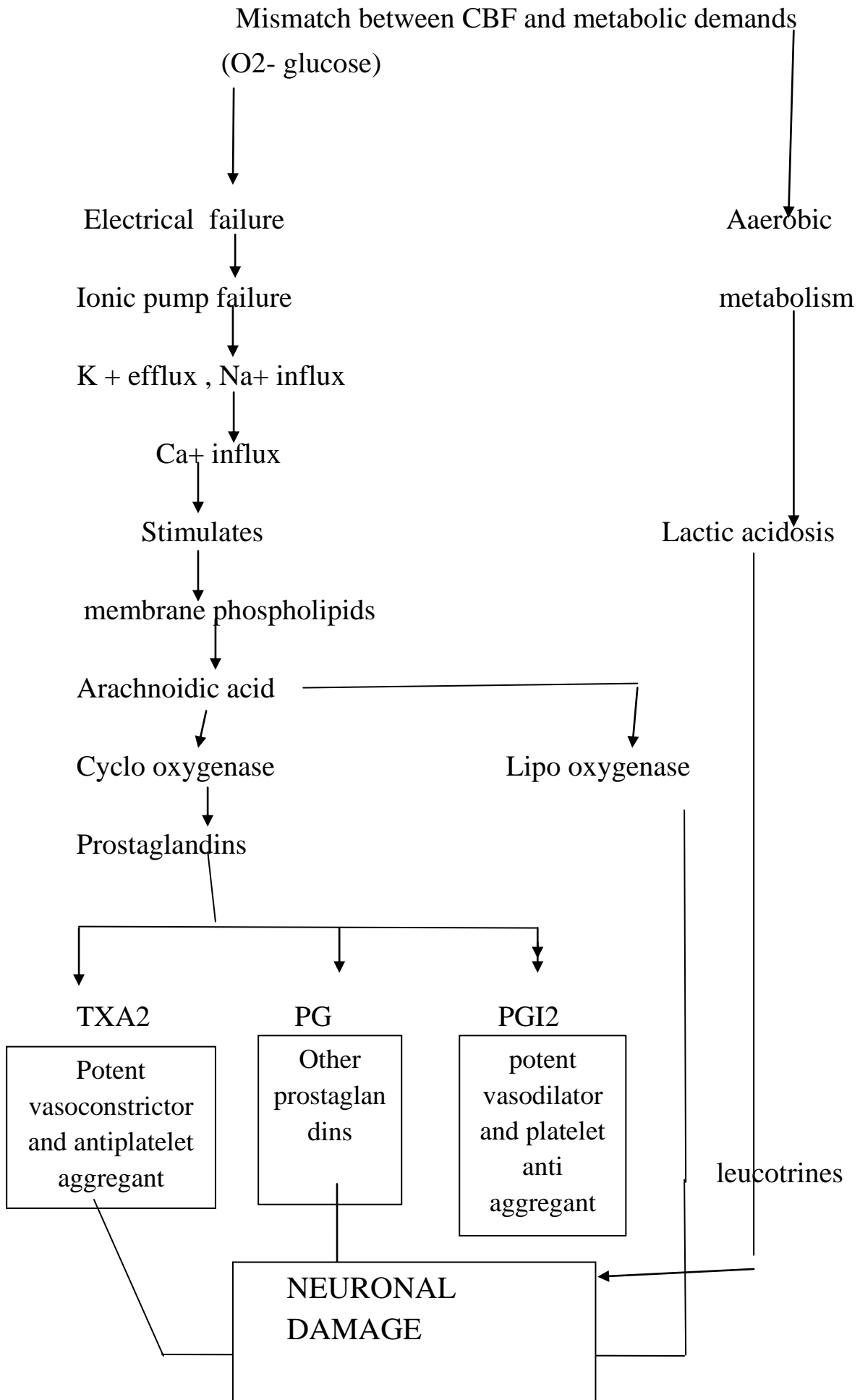
Pathophysiology of stroke:

More than any other organ, the brain depends from moment to moment on an adequate supply of oxygenated blood .

Cerebral infarction comprises of mainly two pathological processes one a loss in the supply of oxygen and glucose secondary to vascular occlusion and the other an array of changes in the cellular metabolism consequent o collapse of energy producing processes , ultimately with the disintegration of cell structures and their membranes ,a process termed as apoptosis. Some cellular processes leading to neuronal death and are not irrevocable and may be reversed by early intervention of restoration of blood flow

ISCHEMIC CASCADE

A significant fall in the cerebral blood flow produce cascade of events which if unchecked lead to the production and accumulation of toxic compounds and apoptosis (programmed cell death)



Role of neurotransmitters

In addition to the cascade outlined above one of the amino acid excitatory neurotransmitters, Glutamate in excess is a powerful neurotoxin which plays an important role in ischemic brain damage.

Approach to stroke patients

- The rapid evaluation of patients presenting with stroke is important as it may lead to prompt treatment such as thrombolysis
- An adequate history from an attenders is essential
- Once provisional diagnosis is made, brain imaging study is essential to determine whether the etiology is ischemic or haemorrhagic.

INVESTIGATION

1. CT SCAN :

It identifies or excludes the haemorrhage as the cause of stroke. It helps to identify extraparenchymal haemorrhages, neoplasms, abscess, and other conditions masquerading as stroke.

2. MRI :

This clearly shows the exact size of the infarcted areas of the brain which includes the cortex and posterior fossa. Diffusion weighted imaging is more sensitive for early brain infarction than standard MR sequence or CT.

3. MRI arteriography

is the confirmatory investigation for detecting stenoses of cerebral arteries and diagnosing and detecting pathologies such as aneurysmal dilation, vasospasms, fibromuscular dysplasias, vascular causes, intramural thrombi, arteriovenous fistula and collateral channels of blood flow.

4. Ultrasound technique:

Stenosis at the origin of the internal carotid artery can be identified and characterized by Duplex ultrasound . Transcranial Doppler can assist in thrombolysis and improve large artery recanalization following rtPA administration.

5. Perfusion techniques:

MR perfusion can be MR diffusion imaging is to identify the ischemic penumbra as the mismatch between the two images .The ability to image ischemic penumbra helps more judicious use selection of patients who may or may not benefit from acute interventions such as thrombolysis, thrombectomy or other investigational purposes.

6. Echocardiogram:

To identify any cardiac source of embolism.

TREATMENT:

1. Medical support

- securing the airway to protect from aspiration and hypoventilation.

- blood pressure should be lowered if there is malignant hypertension or concomitant myocardial ischemia or if blood pressure is more than 180/110mm hg and thrombolytic therapy is anticipated.

- if brain oedema is seen then it can be reduced with mannitol

- maintenance of body temperature to prevent hyperthermia

- Blood glucose should be monitored and maintained at 110mg/dl, if necessary by using insulin infusion.

2. Thrombolysis

Administer IV rtPA 0.9 mg /kg (max 90mg) 10% given as bolus and the remaining dose within 1 hour.

INDICATION AND CONTRAINDICATION FOR THROMBOLYSIS

INDICATION	CONTRAINDICATION
<p>Clinical diagnosis of stroke</p> <p>Onset of symptoms to the time of drug administration within 3 hours.</p> <p>CT scan showing no haemorrhage or edema of > 1/3 of MCA territory</p> <p>Age > 18 years</p> <p>Consent by patient or surrogate</p>	<p>Sustained BP . 185/110 mmhg despite treatment</p> <p>Platelet < 100,000, HCT < 25%, blood glucose < 50 or >400 g/dl</p> <p>Use of heparin within 48 hrs and prolonged PTT and elevated INR</p> <p>Rapidly improving symptoms</p> <p>Prior stroke or head injury within 3 months, prior intracranial Haemorrhage</p> <p>patients undergone surgery within 2 weeks</p> <p>symptoms of mild stroke</p> <p>Any bleeding from GIT within 2 weeks deeply comatose and stuporous or patients suffered from recent myocardial infarction.</p>

4. Antiplatelet:

Aspirin within 48 hours of stroke occurred decreases both recurrence of stroke and death minimally. Aspirin, clopidogrel and combination of aspirin and dipyridamole are used.

5. Anticoagulation:

There are many trials have fail to prove the benefit from anticoagulation. But there are indications in conditions such as carotid artery bidissection, Basilar artery thrombosis, cardio embolic stroke.

6. Neuroprotection :

It is the concept of providing a treatment that prolong brain tolerance to ischemia.

7. Stroke centres and rehabilitation:

Team that provide emergency 24 hour evaluation of acute stroke patients for acute medical management , thrombolysis and endovascular treatment which are essential ingredients of primary

and comprehensive stroke centre . It also include early physical, occupational speech therapy.

THE PROGNOSIS OF ACUTE STROKE

The outcome of an individual who had recovered from stroke depends on certain factors

1. Age :

The survival is better in younger than the older people, more in men than in women

2. Comorbid conditions:

There are factors such as diabetes, dyslipidemia, hypertension, atrial fibrillation, smoking, alcohol, previous h/o stroke leads to recurrent stroke and also influence on outcome and survival of the individual. Other diseases such as COPD,

Parkinsons disease, peripheral vascular disease, polyneuropathy have effect on functional recovery.

Lesion related factors

Survival is poor in anterior circulation infarcts than intracerebral or subarachnoid haemorrhage. Coma at onset of stroke makes is an important predictor of 4 weeks survival. There are

symptoms such as seizures, brain stem dysfunction, B/L pyramidal signs carries an higher risk of mortality.

3. Specific therapy

Good supportive care such as cardiac and respiratory support may reduce the mortality.

4. Biochemical factors:

Recent studies suggests that microalbuminuria has been associated with poor outcome. Lipoprotein (a) found to be independent risk factors for vascular diseases. Higher blood glucose at the onset of stroke even in non diabetic individuals predict the poor survival.

STROKE SCALES

Acute assessment stroke scales

1. Canadian neurological scale
2. European stroke scale
3. Glasgow coma scale
4. Hemispheric stroke scale
5. National institute of health stroke scale

Functional assessment scale

1. Modified Rankins scale
2. Barthel index
3. Glasgow outcome scale
4. Berg balance

NATIONAL INSTITUTES OF HEALTH STROKE SCALE

LEVEL	SCORE
stages of consciousness	
Conscious	0 point
somnolent	1 point
patient responds to deep painful stimuli	2 points
deeply comatose	3 points
orientatation	
tells about his age and date	0 points
able to answer one question perfectly	1 point
not able to answer any question properly	2 points
response to verbal	

obeys 2 verbal speech	0 points
obeys 1 verbal speech	1 point
not obeying to any verbal speech	2 points

movement of eyes

perfect movement of both the eyes	0 points
moderate paresis to 1 one side	1 point
severe gaze palsy to 1 side	2 points

visual impairment

Normal vision	0 points
homonymous hemianopia (moderate)	1 point
homonymous hemianopia (total)	2 points
total visual impairment	3 points

motor function (face)

Normal face	0 points
Minimal one sided facial palsy	1 point
Moderate one sided facial palsy	2 points
total facial palsy either 1 or 2 sides	3 points

motor function of the upper limb (scores 8 points)

no paralysis	0 points
slight decrease in movement in upper limb	1 point
minimal strength against gravity	2 points

moderate strength against gravity but moves limbs 3 points

complete paralysis 4 points

motor function of the lower limbs (score 8 points)

no paralysis 0 points

slight paralysis of lower limb 1 point

moderate paralysis against gravity 2 points

moderate strength against gravity but moves
the limb 3 points

complete paralysis 4 points

ataxia of limb (not in presence of paralysis)

normal ataxia 0 points

present in 1 limb 1 point

present in 2 limbs 2 points

sensory modalities

normal 0 points

sensory loss from minimal to moderate 1 point

sensory loss is complete 2 points

speech

speech is normal 0 points

minimal to moderate speech impairment 1 point

speech impairment is severe 2 points

speech loss is complete 3 points

Articulation

Normal 0 points

Minimal to moderate articulation impairment 1 point

Severe articulation impairment 2 points

Neglect

normal 0 points

minimal to moderate visual or sensory neglect 1 point

severe visual and sensory neglect 2 points

MODIFIED RANKINS SCALE

SCORE SEVERITY OF FUNCTIONAL LOSS

1 normal

2 symptoms present but no impairment

3 minimal impairment with no assistance
required

4 impairment is moderate but can move

5 severe impairment

6 expired

MICROALBUMINURIA

DEFINITION

Microalbuminuria defined as the range in between urinary excretion of albumin of 20 – 200 $\mu\text{g}/\text{min}$ or 30- 300mg/24 hours. The low- normal albuminuria is defined as a morning excretion less than 10mg/l. High normal albuminuria is in between 10 – 20mg/l.

MECHANISM

The correlation between the microalbuminuria and vascular permeability leads to higher sensitivity during any inflammatory process such as cerebrovascular disease.

Any small changes in the vascular gets amplified by the kidney. Overall kidney receives $\frac{1}{4}$ the of cardiac output. The amount of albumin that enters t the kidney all through out the day is 70 kg. The glomerular filtrate that reaches the renal tubules is 7g/24 hr. Most of the albumin taken up by the proximal tubule taken up by the mechanism such as high affinity low capacity endocytotic process leading to only 10-30mg/24hr appears in the urine. The

amount of albumin filtered over 24 hours is 7 gm . If 1% change in the systemic vascular permeability due to inflammatory stimulus would lead to extra 70 mg of albumin during the filtration process. Since tubular mechanism for reabsorption of albumin were near saturation would lead to change in the albumin excretion from range of 30 mg / hr to more than 100 mg/ hr.

Both charge and size selectivity are properties of endothelium. The negative charge is confined to glomerular membrane by its consistent glycoprotein plays a major role in restricting the permeability of anionic proteins. So the loss of charge selectivity in the glomerulus leads to microalbuminuria not only in diabetic individuals but also in non diabetic individuals.

Other mechanisms of microalbuminuria as follows

1. Impaired arterial dilatory capacity:

In clinically normal individuals slight increase in albuminuria excretion leads to impaired dilatation of arteries. This decrease in arterial dilatory response was seen in both in vivo and in vitro. This

reduced arterial dilatory capacity may lead to higher cardiovascular risk in subjects with increased urinary albumin excretion.

2. Hyperhomocysteinemia:

This is seen as a major risk factor for atherosclerosis. The patients who are associated with hyperhomocysteinemia leads to higher risk of cardiac and cerebrovascular disease.

3. Sytemic Transvascular albumin leakage;

In vivo the rate of transcapillary escape of albumin is an overall measurement of vascular permeability. Transcapillary rate of albumin is defined as the fraction of the intravascular mass of albumin going through the vascular bed per unit time. Hence microalbuminuria directly proportional to transvascular leakiness of albumin which in turn may allow more amount lipid insudation into the larger arteries leads association of microalbuminuria to atherogenesis.

4. Hyperinsulinemia:

Hyperinsulinemia and Microalbuminuria are the two major factors of metabolic syndrome. There are studies shows that insulin may cause smooth muscle cell proliferation which in turn

accelerate the LDL binding in smooth muscle cells and cholesterol synthesis in monocytes.

5. Role of sialic acid:

There are studies that reveals sialic acid influence many haematological factors, deposition of lipids in the arteries and transvascular permeability. The higher level of serum sialic acid acts as a predictive value of atherosclerotic vascular disease in patients especially without diabetes mellitus with an elevated urinary albumin excretion.

6. The elevated VWF concentration and prothrombotic factors:

There are many studies that shows prothrombotic factors such as vWF, factor VIIc, and fibrinogen are higher in patients with type 1 DM as well as hypertensive patients who are associated with microalbuminuria.

SIGNIFICANCE OF MICROALBUMINURIA

The importance of microalbuminuria indicates different vascular permeability and its diagnosis shows not only renal disease but also higher risk of cerebrovascular diseases. There were two landmark studies in the early 80's which showed the importance of microalbuminuria and independently predict the development of nephropathy in diabetic individuals and increasing renal impairment. Therefore, different studies were conducted to highlight the importance of microalbuminuria in various conditions:

ADA guidelines for Diabetic Nephropathy

Stages	Albuminuria cut –off values	Clinical characters
Micro albuminuria	20-199 mcg/min	Abnormal nocturnal fall in BP and rise in BP level
	30-200 mcg/24 hrs	Increased Triglyceride, total and LDL cholesterol. Higher incidence of metabolic syndrome component.
		Endothelial dysfunction.
		Linked with retinopathy of diabetic individuals and amputation and CVD
Micro albuminuria	>200 mcg/min	Hypertension
	>300 mcg/24 hr	Increased triglycerides, total and LDL cholesterol.
	>300 mg/gm	Asymptomatic Myocardial ischemia
		Progressive GFR decline

ADA recommended microalbuminuria as a risk factor for cardiovascular diseases in patients with diabetes.

2. Studies have shown that the prevalence of microalbuminuria is enhanced in hypertensive subjects, in particular in those with blood pressure characteristics that are associated with enhanced cardiovascular risk, such as salt sensitivity and an abnormal diurnal blood pressure rhythm. Microalbuminuria possibly identifies at an early stage, hypertensive patients with an elevated risk of developing the well-known cardiovascular and renal hypertensive complications.

3. There are many studies that have shown the association of microalbuminuria and risk factors for atherosclerosis such as diabetes hypertension and dyslipidemia in the normal population. Studies have shown that the importance of microalbuminuria as predicts more mortality among the patients aged more than 70 years.

4. Microalbuminuria is detected in earlier stage of Acute Myocardial Infarction and is considered as an independent factor that predicts of early mortality. Microalbuminuria had been found to be directly proportional to the size of the infarct.

5. There is one study shown that microalbuminuria distinguished aseptic meningitis from bacterial meningitis with specificity of 94%- Roine et al
6. One study had shown that microalbuminuria significance Predicting a of 36 months prognosis in patients who had suffered from acute coronary artery disease - Spyridon k et al
7. There are studies that has shown early rise in urinary albumin concentration is useful in differentiating between myocardial infarct from Angina- Gosling et al
8. Microalbuminuria had been found to be associated with wide variety of inflammatory conditions like, surgery, rheumatoid arthritis, inflammatory disorder
9. There was a significant correlation between carotid artery thickness and microalbuminuria which reveals that microalbuminuria may be a significant marker in the development of atherosclerosis in carotid artery which indicates a possible linkage between atherothrombotic stroke and microalbuminuria.

10. Shearman et al found that microalbuminuria peaked in 36 hours after admission in individuals with acute pancreatitis and that has serious complications developed later only those with higher values of microalbuminuria .
11. Pallister et al found that microalbuminuria levels after 8 hours of admission in trauma victims predicted the development of ARDS with apposite predictive value of 85% and negative predictive value 95%.

STUDIES RELATING MICROALBUMINURIA and ISCHAEMIC STROKE

Eventhough microalbuminuria is associated with risk factors of cerebrovascular disease including hypertension, previous h/o myocardial infarction, diabetes, aging and left ventricular hypertrophy but there are few data regarding microalbuminuria predicting independent risk factor for stroke . There are several studies that has been done and proved association of ischemic stroke and microalbuminuria.

1. Damsgaard EM et al followed 216 people who had been selected as control subjects for diabetics during a sytemic

screening for diabetes mellitus among all people aged between 60-75 years living in municipality of Fredericia, Denmark between Feb 1981 and Dec 1987. Their clinical and biochemical examination were found median urinary excretion rate of albumin is 7.53mcg/min. 8 of those rate below median died compared to 23 with a rate equal to or greater than the median. The median albumin excretion rate is the 31 who died was 15 mcg/min and cardiovascular disease was the main cause of death in both groups.

2. Yudhkin et al used Islington Diabetes survey in 1988 to study urine albumin excretion and found that urinary albumin excretion had skewed distribution with maximum rate of 191.9 mcg/min . There was significant correlation between albumin excretion rate and systolic BP, diasolic BP and 2 hour blood glucose but not with age , sex or BMI.
3. Mlack B et al studied the frequency of microalbuminuria in patients with and without diabetes (138/160) randomly selected from a stratified sample comparable with known diabetes by age, sex and profession in metlika county, Slovenia between

1994-1998. The groups were examined in the same way and mortality was followed over 5 years. Albuminuria was significantly high in diabetes, hypertension, coronary artery disease, dyslipidemia, peripheral vascular disease. The albuminuria was frequent in those who died in the observed 5 year period.

4. Heikke Meietinen et al had shown that study conducted during 1982 and 1990 had found increased excretion of urinary albumin in 25 % of non diabetics and 58% of NIDDM subjects . The morbidity and mortality were more in patients both in NIDDM and non diabetic patients with albuminuria than in those patients without albuminuria

5. Nancy B. Beamer et all had shown in that study done in Oregan Health sciences university Hosspital in Portland conducted around 1999 found that microalbuminuria seen in in acute ischemic CVA individuals three times more than in individuals with risk factors and not detected in healthy individuals .On follow up period of 1.5 ± 0.9 years 1/5 th of patients with ischemic CVA 14% were with risk factors for stroke is 0% in healthy individual volunteers

had vascular end points with events being as frequent in patients with microalbuminuria (32%) as in patients without microalbuminuria .

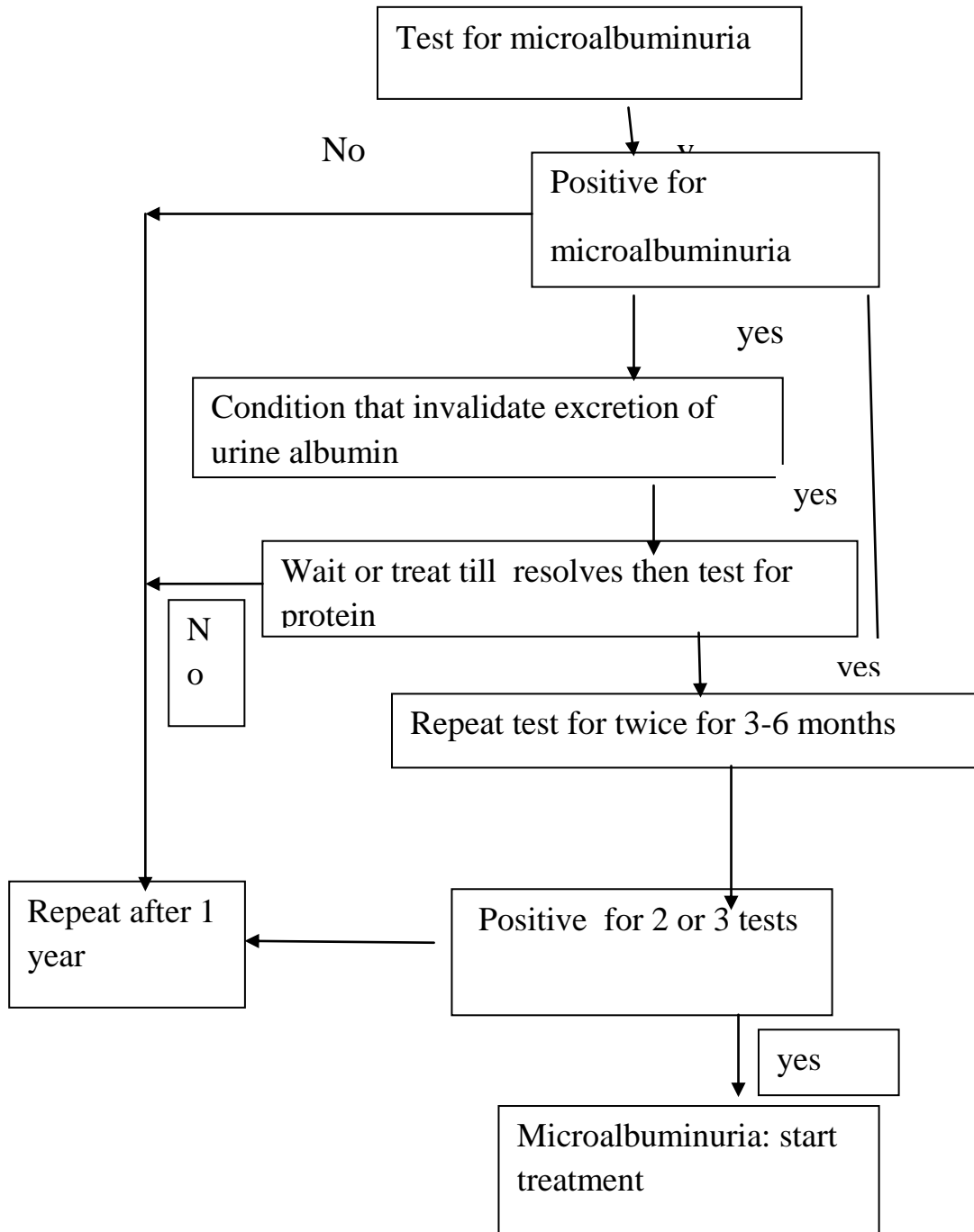
6. Yuyun MF et al had conducted population based on cohort study in Britain among 23,630 subjects between 40- 79 years and followed up for 7 years (93 – 97) with albuminuria tested. There are 246 strokes were occurred. Then age adjusted incidence of stroke elevated significantly among all categories of baseline albuminuria. So they had identified that microalbuminuria independently associated with 50% increased risk of stroke in general population.

7. Hans.L .Hillege et al had done study in Groningen , Netherlands around 2001 and had found that elevated levels of microalbuminuria more frequent with aging, male sex , smoking, hyperlipidemia, hypertension, diabetes. Eventhough Micro and macroalbuminuria were found more in diabetic and hypertensive patients that are independently associated with cardiovascular risk factors.

8. Turaj et al had shown on a study on 52 patients in Jagiellonian university Caracow, Poland within 24 hours after stroke. Microalbuminuria were found in 24 out of 52 stroke patients (46.1%) and 5 out of 37 controls (13.5%) ($p < 0.05$) . Then 3 months mortality was more in subjects with micro albuminuria than when compared to subjects without micro albuminuria (45.8% vs 7.1%). The individuals with micro albuminuria scored lesser scores in Scandinavian stroke scale than in individuals without microalbuminuria both at the time of admission and later.

Diagnostic criteria of diabetic nephropathy according to ADA

Algorithm for microalbuminuria



Treatment of microalbuminuria

1. Control of hypertension

Studies have shown secondary prevention of hypertension effectively decreases albuminuria. The treatment of choice was Angiotensin converting enzyme inhibitors and Angiotensin receptor blocker for controlling microalbuminuria. The cut off point for blood pressure should be <130/80 mm hg for diabetics and < 140/90 mm hg for non diabetic individuals.

2. Control of blood glucose

Aggressive diabetic control effectively decreases the risk of developing microalbuminuria in patients especially with diabetic individual.

3. Treatment of hyperlipidemia

The use of statins has many indication such as to modify thrombus formation, plaque stabilization, inflammatory response, endothelial dysfunction. It also has effective reduction of microalbuminuria.

4. Protein restriction

The normal intake of protein is 0.8g/kg/d.in patients with renal diseases. There are studies that shown restriction of protein intake decreases hyperfiltration and intraglomerular pressure there by reducing the progression of microalbuminuria.

5. Smoking

Smoking should be stopped in patients as it not only protective against cardiovascular disease but also decreases the progression of microalbuminuria

TESTS FOR MICROALBUMINURIA

The albumin in the urine is one of the risk factors for cardiovascular and cerebrovascular diseases. Hence the detection of albumin in the urine depends on the tests to identify microalbuminuria. Traditional qualitative tests cannot detect small quantities of albumin in the urine. The various methods are as follows

Dipstick method

Chemically impregnated dipstick contain methylred and bromophenol blue buffering salts . The later dissolve on contact with urine and protein in the urine lowers pH turning it green . It was traditionally known to detect albuminuria $> 300\text{mg /dl}$. hence not advocated for screening of microalbuminuria

1. Semiquantative analysis:

Chemical precipitation test :

5 drops of 20% sulphosalicylic acid is added to 3 ml of urine in one test tube . This test tube is compared to test tube of untreated urine held against the dark background immediately and turbidity is taken to indicate proteinuria

Immunoprecipitation test :

Micral test :

It is based on color shift on monoclonal antibody in human albumin labeled with gold. Here gold labeled antibody optically read immunoassay detects microalbuminuria. A specimen of the urine sample pass via wick fleece into the conjugate fleece.

Any albumin present in the urine binds itself specifically to the gold labeled antibodies. Excess antibodies are bound by immobilized albumin in the capture matrix .only antibodies bound to albumin for capture matrix the urine sample and pass through

- Lapiuex agglutination tests (inhibin test): titre is adapted in away that drop of antihuman albumin falls one by one and attach all connection places of albumin in urine in concentration over 30mg /l and more.

2. Quantitative analysis:

There are many reactions for measuring albumin concentration in urine but all of them use the same principle ;immunochemical reaction between antibody to antihuman albumin and albumin. Difference is only the preparing the reaction, Elisa, immunoturbidimetry,Nephelometry and Electrochemiluminescence RIA.

All these methods are mostly well balanced and good analytical precision, sensitivity and detection area. A quantitative procedure can be done by using immunological test methods on standard automated analyzer.

Radioimmunoassay

It is one of the gold standard for estimation of microalbuminuria. It is the double antibody technique where albumin the sample has to compare with the fixed amount of ^{125}I labeled albumin for the binding sites of specific antibodies. Bound and free albumin is separated by separated addition of a second antibody immunoabsorbent followed by configuration and decanting . The radioactivity in the pellet is measured a C- counter . Albumin concentration with sample inversely proportional to radioactivity . The sensitivity RIA method was 0.3 mg/ml.

Microalbumin – turbidimetric

Estimation of microalbumin in urine in our case is done by

Latex turbidimetric

PRINCIPLE :

1. Its a slide agglutination tests
2. It depends on the turbidity of the solution
3. The albumin in a sample of urine gets reacts with specific antibody coated with latex particles. The turbidity measured with a

spectrophotometer and its absorbency is proportional to albumin concentration

Material required :

Thermostatic bath at 37c Spectrometer or thermostable at 37 c with a 540 \pm 20 nm filter.

Procedure:

The working reagent is warmed and the photometer to 37

1. Mix well and record the absorbances immediately (A1) and 2 minutes (A2) after the sample addition this gives the final result on microalbuminuria.

METHODOLOGY

Source of data

The patients admitted both in the department of medicine and department of Neurology in Government Rajaji Hospital affiliated to Madurai medical college between January 2012 to May 2012 were taken in our study. Ethical committee approval was obtained

Collection of datas

1. Prospective observational study
2. There were 50 patients enrolled in the study diagnosed and clinically confirmed by CT Scan brain

Inclusion criteria

1. Patients were diagnosed as stroke according to WHO criteria which includes patients of any age and both sexes within 48 hours onset of symptoms.
2. Ischemic infarct confirmed by CT scan
3. Informed consent obtained from the patient

Exclusion criteria

1. Patients with haemorrhagic stroke
2. Patients with diabetes who is on antidiabetics or Fasting blood glucose > 126mg/dl or Random blood glucose > 200 mg /dl
3. Any abnormal urinalysis
4. Patients who are on antihypertensives or with cut off value for blood pressure > 140mm hg in systolic and >90 mm hg in diastolic.

Patients were taken detailed history, proper clinical examination and required laboratory investigations done.

The severity of the stroke scale were assessed using NIHSS and functional assessment were done by using mRs.

In the above patients following investigation were done as follows

1. CT scan were done to confirm ischemic infarct.
2. urinalysis were done to exclude proteinuria, leucocyturia, haematuria, glucosuria.

3. Cardiac evaluation were done using ecg, echo and chest x ray. The albumin excretion was assessed by using turbidimetry tests in the urine sample. spot sample obtained . microalbuminuria is defined such as urinary albumin excretion between 20- 200 mg dl.
4. After the patients gets discharged from the hospital subjects were again tested 6 weeks later to determine the outcome of the stroke including the mortality by functional assessment scales using Modified Rankins scales.
5. 50 age and sex matched controls were selected among the subjects with > 6 months old who had suffered from stroke and were coming for follow up .
6. The control subjects were tested for risk factors for stroke and estimate the presence of microalbuminuria using latex turbidimetry tests, plasma glucose, lipid profile

RESULTS AND OBSERVATION

Study design

Our study was done comparing 50 recent ischemic stroke patients as cases and as 50 old stroke patients as controls and showed the following findings .

TABLE 1

AGE DISTRIBUTION

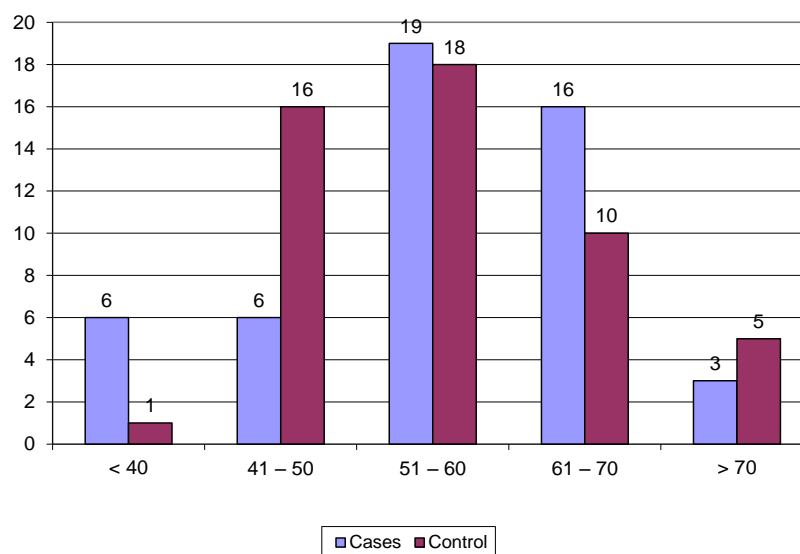
Age in years	Cases	Controls
<40	6	1
41-60	25	34
>60	19	15
Total	50	50
Mean	55.80	55.82
SD	12.79	9.83
p value	0.998	Not significant

TABLE-2

Age distribution by microalbuminuria

Age in years	< 20	20- 200
< 40	3	3
41-60	15	10
>60	12	7
Total	30	20

AGE DISTRIBUTION



The youngest patient among the cases was 18 years and oldest was 78 years. The youngest patient was among controls was 39 years and old patient was 80 years.

The mean age cases were 55.80 ± 12.79 and in controls were 55.89 ± 9.83 there is not much difference in age. The majority of the cases and controls were found in the age group of 41-60 years of age . The age is not statistically significant as $p= 0.998$

TABLE-3

Sex distribution

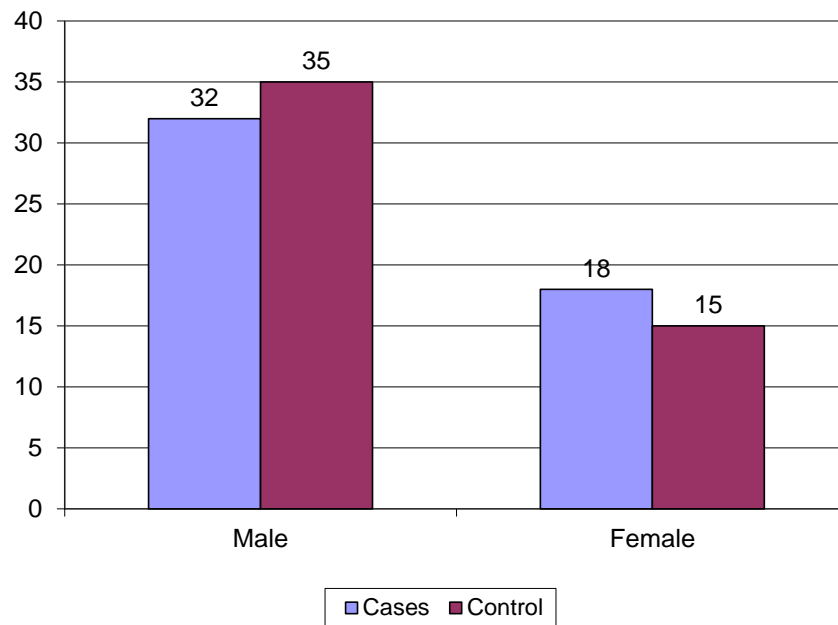
Sex	Cases	Control
Male	32	35
Female	18	15
Total	50	50

Table - 4

Sex distribution by microalbuminuria

Sex	<20	20-200
Male	18	14
Female	12	6
Total	30	20

SEX DISTRIBUTION



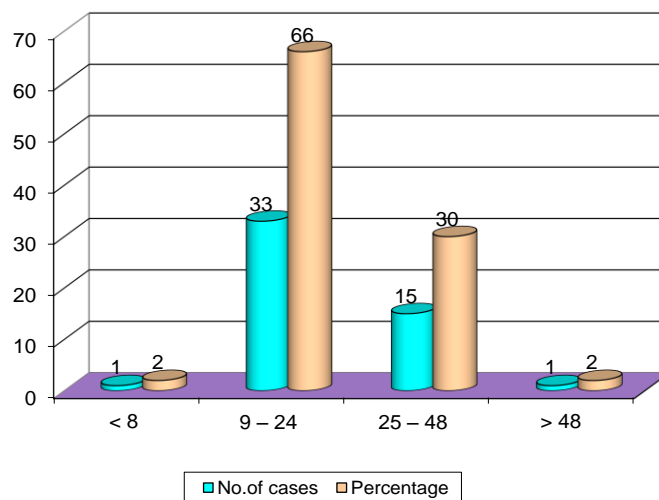
As such there is no more difference in the sex distribution between male and female Incidence of stroke were more in males than females but that was not statistically significant

Table – 5

Duration of Symptoms in hours

Duration of Symptoms in hours	No of cases	Percentage
< 8	1	2
9-24	33	66
24-48	15	30
>48	1	2
Total	50	100

DURATION OF SYMPTOMS



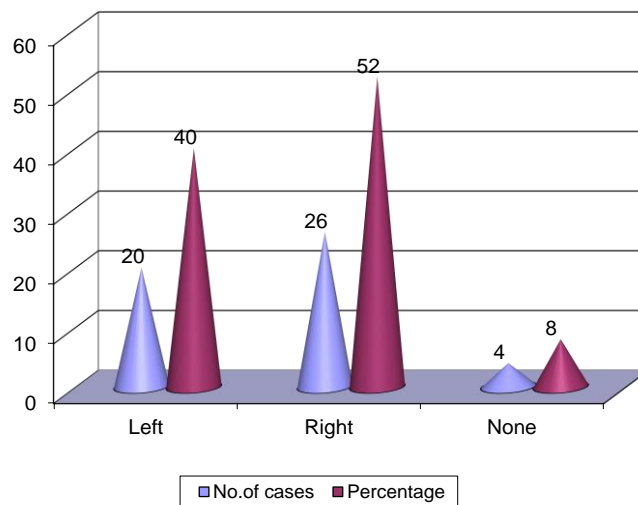
In our study in cases majority of the subjects had duration of symptoms within 9-24 hours is 33(66%) where as 15 patients had symptoms within 24-48 hours (30%) and more than 48 hours only 1% had presented with symptom

Table -6

Paucity of movements

Paucity	No of cases	percentage
Left	20	40
Right	26	52
None	4	8
Total	50	100

PAUCITY OF MOVEMENT



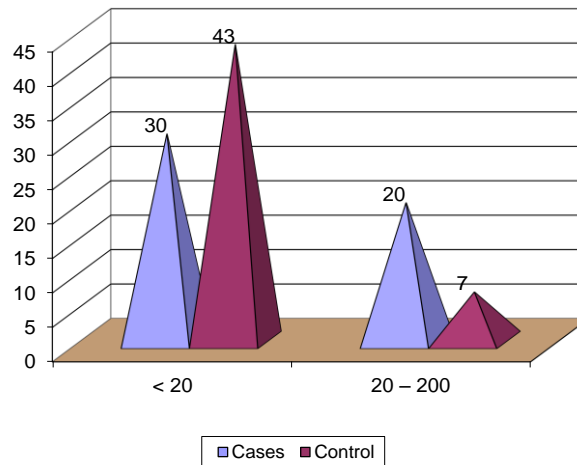
Among the case 20(40%) were had left sided hemiparesis and 26(52%) were had right sided hemiparesis and 4(8%) had no focal neurological deficit.

Table – 7

Microalbuminuria

Microalbuminuria	Cases	Control
<20	30	43
20-200	20	7
Total	50	50

MICRO ALBUMINERIA



Inference: The patients with recent ischemic stroke were 2.578 times more likely to have microalbuminuria with $p= 0.0447$.

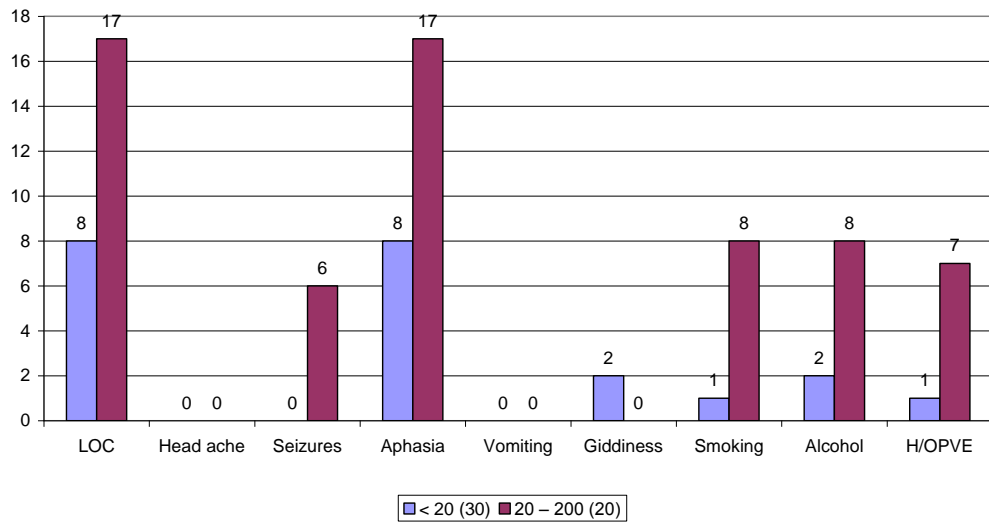
Microalbuminuria were found in 20 (40%) patients with recent ischemic stroke where as 7(14%) . Therefore patients with recent ischemic stroke were 2.58 times high likely to have microalbuminuria as compared to controls.

TABLE- 8

Presenting factors associated with microalbuminuria

Presenting factors	<20(30)	20-200(20)	P - value	percentage
LOC	8	17	0.041	50
HEADACHE	0	0	0.100	0
SEIZURES	0	6	0.019	12
APHASIA	8	17	0.041	50
VOMITING	0	0	0.100	0
GIDDINESS	2	0	0.690	4
SMOKING	1	8	0.019	18
ALCOHOL	2	8	0.083	20
H/O PVE	1	7	0.034	16

PRESENTING FACTORS



Among the presenting factors loss of consciousness, seizures, aphasia, smoking, h/o previous vascular diseases were higher among the cases than the controls and also statistically significant. Hence this implies that microalbuminuria correlates with the severity of the stroke.

Table – 9

	Mean + SD		'p' value
	< 20 (30)	20 – 200 (20)	
Cholesterol	196.6 ± 12.23	194.1 ± 15.06	0.516 NS
HDL	45.3 ± 4.76	45.2 ± 4.22	0.910 NS
LDL	105.5 ± 7.40	110.1± 9.01	0.057 NS
Triglyceride	198.7 ± 10.28	196.5 ±5.42	0.378 NS

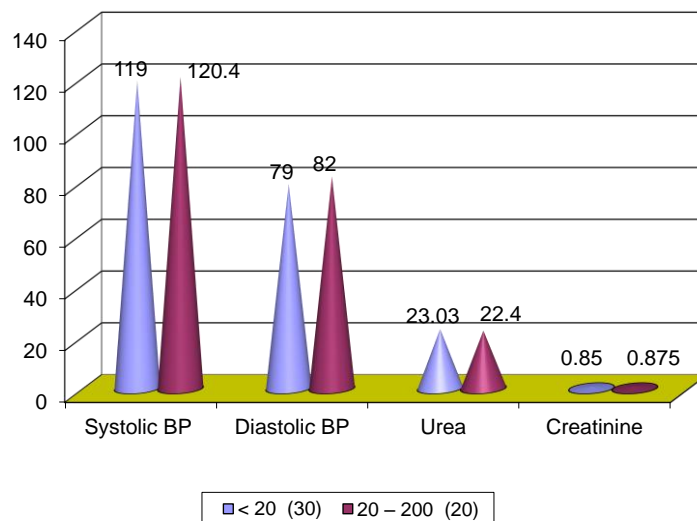
Among the cases the Total cholesterol, HDL, Triglyceride were lower among the patients with microalbuminuria and LDL was higher but it is not statistically significant.

Table 10

Blood Pressure and Renal parameters

	Mean + SD		'p' value
	< 20 (30)	20 – 200 (20)	
Systolic BP	119.0 ±13.22	120.4 ±12.61	0.710 NS
Diastolic BP	79.0 ±10.62	82.0 ± 7.68	0.283 NS
Urea	23.03 ± 3.61	22.40 ± 3.82	0.555 NS
Creatinine	0.850 ± 0.104	0.875 ± 0.116	0.432 NS

BLOOD PRESSURE AND RENAL PARAMETERS



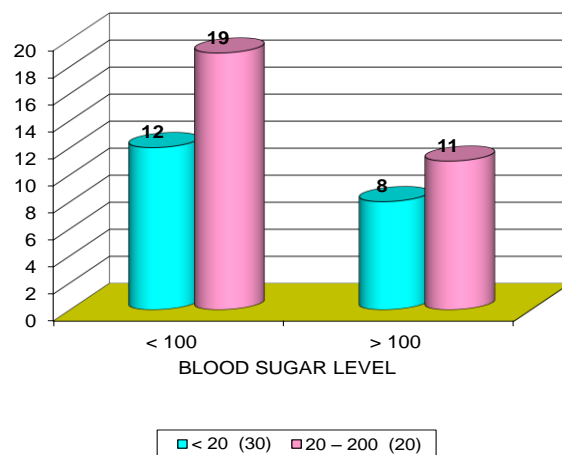
Among the blood pressure diastolic blood pressure higher than the systolic pressure but it is not statistically significant . Renal parameters were not significant.

TABLE-11

Blood Sugar

Blood sugar level	< 20 (30)	20 – 200 (20)	'p' value
< 100	12	19	0.906 Not significant
> 100	8	11	0.908 Not Significant
Total	20	30	

MICROALBUMINURIA VS BLOOD SUGAR



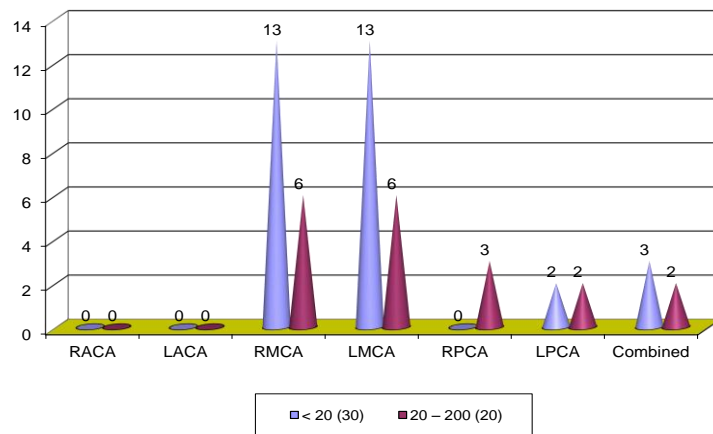
The blood sugar in the stroke patients were not significant in patients both with and without microalbuminuria.

TABLE- 12

CT Scan results

CT Scan results	< 20 (30)	20 – 200 (20)	Percentage (%)
RACA	0	0	0
LACA	0	0	0
RMCA	13	6	38
LMCA	13	6	38
RPCA	0	3	6
LPCA	2	2	8
Combined	4	2	12

CT SCAN RESULTS



According to CT scan results predominant lesion were the middle cerebral artery infarct in study accounts to 38% each left and right sided lesions

TABLE-13

NIHSS score

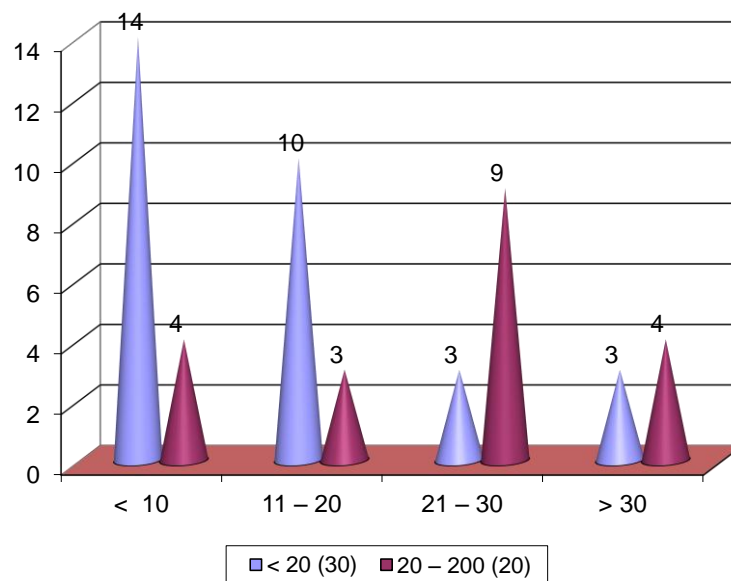
NIHSS	< 20 (30)	20 – 200 (20)
< 10	14	4
11 – 20	10	3
21 – 30	3	9
> 30	3	4
Total	30	20

TABLE-15

NIHSS score vs Microalbuminuria

NIHSS	< 20 (30)	20 – 200 (20)	‘p’ value
< 20	24	6	0.109 Not significant
> 20	6	14	0.045 Significant
Total	30	20	

NIHSS



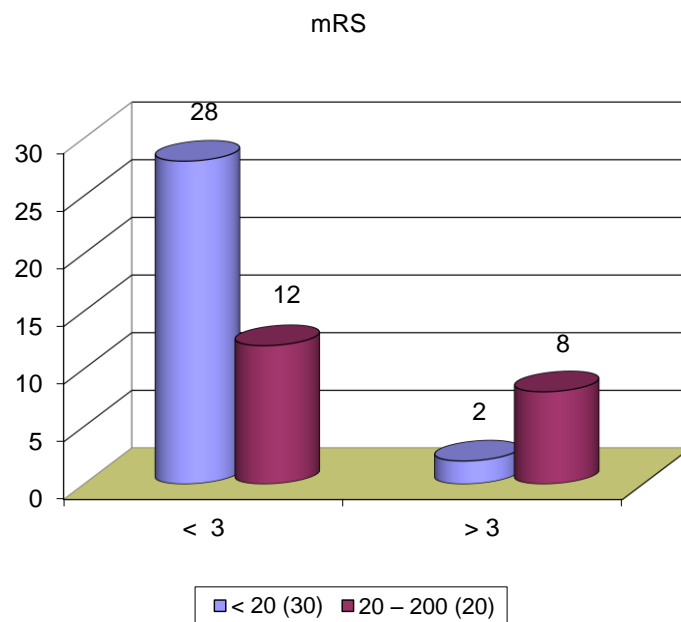
Severity of the stroke scale were measured using NIHSS .

The severity of the stroke scale were higher in patients with microalbuminuria and it was statistically significant

TABLE-16

Modified Rankins Scores

mRS	< 20 (30)	20 – 200 (20)	'p' value
≤ 3	28	12	0.608 Not significant
> 3	2	8	0.035 Significant
Total	30	20	



The functional assessment scale was done using Modified Rankins index patients with microalbuminuria were found higher scores which indicates patients with microalbuminuria were found to have more neurological deficit than patients without microalbuminuria .

DISCUSSION

Our study is a comparative study with 50 acute ischemic stroke subjects as cases and 50 old stroke subjects as controls were taken to

1. To estimate the incidence of microalbuminuria in acute ischemic Stroke.
2. The difference in various laboratory investigations done in subjects with and without microalbuminuria.
3. The correlation of National institute of health stroke scales and Modified Rankins scale in presence of microalbuminuria

Presence of microalbuminuria

	Microalbuminria in cases	Microalbuminuria in controls
Beamer et al	29%	10%
Slowik A et al	46.7%	16.7%
Turaj et al	46.1%	16.7%
Our study	40%	14%

In our present study we found that age and sex matched controls were having similar presenting and pre disposing factors , subjects with acute ischemic stroke were 2.58 times more likely to present with microalbuminuria .The present study we found 40% of the patients with microalbuminuria had incidence of stroke and 14% of patients were found in the controls. The above findings were found to be similar to the studies conducted by others such as Beamer et , Slowik et al and turaj et al.

Age and microalbuminuria

	Microalbuminuria present	Microalbuminuria absent	Control
Turaj et al	73.3±11.6	66.0±12.0	65.2±5.5
Our study	55.80±12.79	55.82±9.83	

The studies such as Turaj et al had shown that age had influence on the neurological deficit. older the age severity of the deficit is increased, But in our study there is no correlation between

the age and microalbuminuria and was not statistically significant.

Sex and microalbuminuria

Turaj et al	Microalbuminuria present	Microalbuminuria absent
Males	12(50%)	14(50%)
Females	12(50%)	14(50%)

Our study

Our study	Microalbuminuria present	Microalbuminuria absent
Males	18(36%)	14(28%)
Females	12(24%)	6(12%)

In our study there was difference in between male and female but not statistically significant but similar findings were not noted in turaj et al

H/o of previous vascular disease

	Microalbuminuria present	Microalbuminuria absent
Turaj et al	9(37.5%)	16(57.2%)
Our study	7(14%)	1(2%)

There is incidence of stroke is more in patients without microalbuminuria in turaj et al but in our study there is a significant difference was there 7(14%) in patients with microalbuminuria and 1(2%) in patients without microalbuminuria and it was statistically significant.

SUMMARY

1. The subjects were taken consists of 50 acute ischemic stroke patients of age 55.80 years as cases with 32 males and 10 females.
2. The controls were age and sex matched of 50 old stroke patients
3. The cases and controls were matched with presenting factors
4. Among the case the 26(52%) were right sided hemiparesis and 20(40%) were left sided hemiparesis.
5. Among the CT scan results predominant lesions were middle cerebral artery .
6. Patients with microalbuminuria has higher incidence with presenting factors such as aphasia, loss of consciousness.

H/o peripheral vascular events were higher in patients with micro albuminuria which is statistically significant

H/o smoking also found to be higher in patients with micro albuminuria which is statistically significant.

7. In our study with recent ischemic stroke 20 patients (40%) had Micro albuminuria and among the controls only 7 patients had microalbuminuria. Hence patients with recent ischemic stroke were

2.58 times were higher incidence of microalbuminuria than without microalbuminuria and was found statistically significant with $p= 0.447$.

8. There are 17/20 subjects with microalbuminuria (85%) had loss of consciousness whereas 8/30 subjects without microalbuminuria had loss of consciousness. Therefore microalbuminuria were found to be associated with severity of the stroke.
9. The Total cholesterol were 194.1 ± 15.06 LDL were 110.1 ± 9.01 , HDL were 45.3 ± 4.76 and triglycerides 196.5 ± 5.42 were found in subjects with microalbuminuria whereas total cholesterol were 196.6 ± 12.23 , LDL 105.5 ± 7.40 , HDL 45.3 ± 4.76 , triglycerides 198.7 ± 10.28 . This difference were not statistically significant.
10. The NIHSS scores were found to have high in subjects with microalbuminuria as compared to subjects without microalbuminuria. Hence microalbuminuria had significant correlation with the severity of the stroke .
11. Functional assessment grading were done using modified rankins scale found to have higher scores in subjects with microalbuminuria than without microalbuminuria

CONCLUSION

There are many studies that have documented microalbuminuria as a risk factor for acute ischemic stroke. Our study demonstrated microalbuminuria in (40%) of non diabetic acute ischemic stroke and is consistent with previous study associating microalbuminuria in atherosclerotic vascular disease.

In our study measuring the presence of microalbuminuria was found to be a strong predictor of stroke outcome after 6 weeks of stroke. The poor neurological deficit during the course of the disease correlates with microalbuminuria . This study gives the indirect prediction of microalbuminuria as a independent risk factor for stroke outcome.

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PROFORMA

Patient profile

NAME:

OP/IP NO:

AGE:

DOA:

SEX:

DOD:

OCCUPATION:

ADDRESS:

PRESENTING COMPLAINTS

1. Paucity of movements of one side of the body

Yes/No –

Duration –

2. Loss of consciousness

Yes/No-

Duration-

3. Seizures

Yes /No –

Duration –

Type -

4. Other symptoms

PAST HISTORY

Diabetes mellitus-

HTN -

Renal insufficiency-

Liver disease-

H/O vascular events-

FAMILY HISTORY

Diabetes mellitus-

HTN-

Cerebrovascular accidents-

PERSONAL HISTORY

Appetite-

Diet-

Sleep-

Bladder movement-

Bowel movement-

Smoking –

Alcohol intake-

OBSTETRIC HISTORY

(In females)

GENERAL PHYSICAL EXAMINATION

Built – poor/moderate/well

Nourishment – poor/moderate/well

Pallor-

Icterus-

Cyanosis-

Clubbing-

Lymphadenopathy-

Oral cavity-

Oedema-

Weight-

Height-

Pulse-

B.P-

JVP-

RR-

Temperature-

1. CNS EXAMINATION:

Done by using NIHSS scoring less severe < 20

More severe > 20

2.CARDIOVASCULAR SYSTEM

Inspection –

Palpation –

Percussion –

Auscultation –

3. RESPIRATORY SYSTEM

Inspection –

Palpation –

Percussion-

Auscultation-

4.ABDOMEN

INVESTIGATIONS

1. Urine analysis

Sugar -

Albumin –

Microscopy -

2. Complete Haemogram

Hb% -

TC-

DC-

ESR-

3. RBS

Blood Urea –

Serum Creatinine-

4. Fasting Lipid profile

TC –

HDL –

LDL –

TG –

5. Chest X –ray PA view

6. 12 lead ECG

7. Echocardiogram

8. C. T Brain

9. Turbidimetry test for Microalbuminuria

Functional assessment chart

10. Modified Rankins criteria

Less severe < 3

More severe > 3

KEY TO MASTER CHART

OP/IP No - Out patient /In patient Number

M - Male

F - Female

N - No

Y - Yes

Territory involved

1. RACA – Right anterior cerebral artery infarct

2. LACA – Left anterior cerebral artery infarct

3. RMCA – Right middle cerebral artery infarct

4. LMCA – Left middle cerebral artery infarct

5. RPCA – Right posterior cerebral artery infarct

6. LPCA – Left posterior cerebral artery infarct

LOC – Loss of consciousness

CBF - cerebral blood flow

Seizures

1. Generalised tonic clonic seizures

2. Right focal seizures

3. Left focal seizures.

ANTIPLAGIARISM CERTIFICATE

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A STUDY ON MICROALBUMINURIA IN NON DIABETIC RECENT ISCHEMIC STROKE Dissertation

submitted for MD Degree (Branch-I) General Medicine The Tamil Nadu Dr.M.G.R. Medical University

Chennai- 600 032. Madurai Medical College, Madurai APRIL - 2013 CERTIFICATE

This is to certify

that this dissertation titled A STUDY OF MICROALBUMINURIA IN NON DIABETIC RECENT

ISCHEMIC STROKE ” submitted by DR. B.GOWRIPATHY to the faculty of General Medicine, The

Tamil Nadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award

of MD degree Branch I (General Medicine) is a bonafide research work carried out by his under our

direct supervision and guidance. Dr. Moses K Daniel, MD.,...

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Department of Medicine

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Govt. Rajaji Hospital,
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Institutional Review Board / Independent Ethics Committee.

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Sub: Establishment-Govt. Rajaji Hospital, aMadurai-20-
Ethics committee-Meeting Agenda-communicated-regarding.

The Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held at 11.00 Am to 1.00Pm on 28.06.2012 at the Dean Chamber, Govt. Rajaji Hospital, Madurai. The following members of the committee have been attended the meeting.


1. Dr.N.Vijayasankaran,M.ch(Uro.) 094-430-58793 0452-2584397	Sr.Consultant Urologist Madurai Kidney Centre, Sivagangai Road,Madurai	Chairman
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11.Shri. S.sivakumar,M.A(Social) Mphil 093-444-84990	Sociologist, Plot No.51 F.F, K.K Nagar, Madurai.	Member

Following Projects were approved by the committee

Sl. No	Name of P.G.	Course	Name of the Project	Remarks
1. ✓	Dr. Gowripathy	M.D Gen med	Microalbuminuria in non-diabetics with ischemic strokes.	Approved

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