PROSPECTIVE STUDY OF CORRELATION OF CLINICAL FEATURES,MRI BRAIN – MRA INTRACRANIUM,CAROTID VERTEBRAL ARTERIAL DOPPLER STUDY IN ISCHEMIC STROKE

Dissertation submitted to

The Tamil Nadu Dr. M.G.R Medical university, Chennai

In fulfilment of the requirements for the award of the degree of

Doctor of Medicine in General Medicine

Under the guidance of

Dr. S.SUJITH KUMAR, M.D.,

DEPARTMENT OF GENERAL MEDICINE

P.S.G INSTITUTE OF MEDICAL SCIENCES & RESEARCH, COIMBATORE

THE TAMIL NADU DR. M.G.R MEDICAL UNIVERSITY,

CHENNAI, TAMIL NADU

CERTIFICATE BY THE HOD AND DEAN OF THE INSTITUTION

This is to certify that the dissertation entitled, "**PROSPECTIVE STUDY OF CORRELATION OF CLINICAL FEATURES,MRI BRAIN,MRA INTRACRANIUM, CAROTID VERTEBRAL ARTERIAL DOPPLER STUDY IN ISCHEMIC STROKE** is the bonafide original research work of **Dr. SRINIVASAN T V** under the guidance of **Dr. SUJITH KUMAR S, M.D.**, Professor of Medicine, PSG IMS&R, Coimbatorein partial fulfilment of the requirements for the degree of Doctor of Medicine in General Medicine.

Seal and Signature of the HOD
Dr. JAYACHANDRAN.K, M.D.,
Professor & HOD,
Department of Medicine
PSG IMS&R, Coimbatore

Seal and Signature of the Dean
Dr. RAMALINGAM .S, M.D.,
Dean
PSG IMS&R, Coimbatore

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled "**PROSPECTIVE STUDY OF CORRELATION OF CLINICAL FEATURES,MRI BRAIN,MRA INTRACRANIUM, CAROTID VERTEBRAL ARTERIAL DOPPLER STUDY IN ISCHEMIC STROKE** "is a bonafide and genuine research work carried out by me under the guidance of **Dr.SUJITH KUMAR S , M.D.,** Professor of Medicine, PSG IMS&R, Coimbatore. This dissertation is submitted to The Tamil Nadu Dr. M.G.R Medical University in fulfilment of the University regulations for the award of MD degree in General Medicine. This dissertation has not been submitted for award of any other degree or diploma.

Signature of the Candidate

Dr. SRINIVASAN T V

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled, "**PROSPECTIVE STUDY OF CORRELATION OF CLINICAL FEATURES,MRI BRAIN,MRA INTRACRANIUM, CAROTID VERTEBRAL ARTERIAL DOPPLER STUDY IN ISCHEMIC STROKE** is the bonafide original work of **Dr. SRINIVASAN T V,** done under my direct guidance and supervisionin the Department of General Medicine, PSG Institute of Medical Sciences and Research, Coimbatore in fulfilment of the regulations by The Tamilnadu Dr. MGR Medical University, Chennai for the degreeof Doctor of Medicine in General Medicine.

Signature of the guide

Dr.SUJITH KUMAR S, M.D.,
Professor of Medicine,
Department of General Medicine,
PSG IMS&R, Coimbatore.

ACKNOWLEDGEMENTS

I would like to voice my deep sense of regards and respect to my guide and teacher **Dr. S.Sujith kumar**, Professor, Department of General Medicine for his valuable advice and guidance. I am very much thankful for his constant inspiration and timely suggestions without which this study would have not been completed.

I would also extend my gratitude to **Dr.K.Jayachandran**, Professor & head of Department, Department of General Medicine, for constant encouragement and structural support in carrying out this study.

I take this opportunity to thank **Dr.Madakasira Bheemarao Pranesh R** Professor Emeritus, Department of Neurology and **Dr .Gnanashanmugam** who had lended a very big hand with the expertise and valuable suggestions and never ending guidance that played an essential role in initiation and completion of this study.

I am very much thankful to Dr.Ramdoss and Dr.Balakrishnan from the Department of Neurology, for their keen enthusiasm and constant supervision during the study.

I also thank Dr.Sujaya Menon M.D, MRCP, Dr.L.S.Somasundaram M.D, Dr.Saravanan M.D, Dr.Tolstoy M.D and Dr.Murali M.D, Professors in Department of General Medicine for their constant support and encouragement.

My heartful thanks to Dr.Anith kumar M.D, MRCP, Dr.Denesh Narasimham M.D, Dr.Jagadeeshwaran M.D,Dr Shantini M.D DNB, Associate Professor, Department of General Medicine for support and guidance.

My heartful regards to Dr.Zeya Ansari, Dr.Velammal, Dr.Yoganathan, Dr.Sathish Assistant professor, Department of general medicine for their support.

I also extend my sense of gratitude to all my colleague post graduates and my friends for their constant help and cooperation during the study.

I also extend my thanks to all the staff of Department of general medicine, department of neurology, Intensive critical units for their help in carrying out the study.

Finally, I do have great regards and respect forall the patients and their relatives who cooperated for my study.

PSG Institute of Medical Sciences & Research
Institutional Human Ethics Committee

Recognized by The Strategic Initiative for Developing Capacity in Ethical Review (SIDCER)
POST BOX NO. 1674, PEELAMEDU, COIMBATORE 641 004, TAMIL NADU, INDIA
Phone : 91 422 - 2598822, 2570170, Fax : 91 422 - 2594400, Email : ihec@psgimsr.ac.in

To
Dr T V Srinivasan
Postgraduate
Department of General Medicine
PSG IMS & R
Coimbatore

Ref: Project No. 14/439

Date: April 17, 2015

Dear Dr Srinivasan,

Institutional Human Ethics Committee, PSG IMS&R reviewed and discussed your application dated 12.12.2014 to conduct the research study entitled "*Prospective study of correlation of clinical features, MRI Brain - MRA intracranium, carotid vertebral arterial doppler study in ischemic stroke*" during the IHEC meeting held on 22.12.2014.

The following documents were reviewed and approved:

1. Project Submission form
2. Study protocol
3. Informed consent form
4. Data collection tool
5. Permission letter from concerned Head of the Department
6. Current CVs of Principal investigator, Co-investigator
7. Budget

The following members of the Institutional Human Ethics Committee (IHEC) were present at the meeting held on 22.12.2014 at IHEC Secretariat, PSG IMS & R between 10.00 am and 11.00 am:

Sl. No.	Name of the Member of IHEC	Qualification	Area of Expertise	Gender	Affiliation to the Institution Yes/No	Present at the meeting Yes/No
1	Dr. P. Sathyan (Chairperson, IHEC)	DO, DNB	Clinician (Ophthalmology)	Male	No	Yes
2	Dr. S. Bhuvaneshwari (Member-Secretary, IHEC)	MD	Clinical Pharmacology	Female	Yes	Yes
3	Dr. S.Shanthakumari	MD	Pathology, Ethicist	Female	Yes	Yes
4	Mrs P Rama	M Pharm	Non-medical (Pharmacy)	Female	Yes	Yes

The study is approved in its presented form. The decision was arrived at through consensus. Neither PI nor any of proposed study team members were present during the decision making of the IHEC. The IHEC functions in accordance with the ICH-GCP/ICMR/Schedule Y guidelines. The approval is valid until one year from the date of sanction. You may make a written request for renewal / extension of the validity, along with the submission of status report as decided by the IHEC.

PSG Institute of Medical Sciences & Research
Institutional Human Ethics Committee

Recognized by The Strategic Initiative for Developing Capacity in Ethical Review (SIDCER)
POST BOX NO. 1674, PEELAMEDU, COIMBATORE 641 004, TAMIL NADU, INDIA
Phone : 91 422 - 2598822, 2570170, Fax : 91 422 - 2594400, Email : ihec@psgimsr.ac.in

Following points must be noted:

1. IHEC should be informed of the date of initiation of the study
2. Status report of the study should be submitted to the IHEC every 12 months
3. PI and other investigators should co-operate fully with IHEC, who will monitor the trial from time to time
4. At the time of PI's retirement/intention to leave the institute, study responsibility should be transferred to a colleague after obtaining clearance from HOD, Status report, including accounts details should be submitted to IHEC and extramural sponsors
5. In case of any new information or any SAE, which could affect any study, must be informed to IHEC and sponsors. The PI should report SAEs occurred for IHEC approved studies within 7 days of the occurrence of the SAE. If the SAE is 'Death', the IHEC Secretariat will receive the SAE reporting form within 24 hours of the occurrence
6. In the event of any protocol amendments, IHEC must be informed and the amendments should be highlighted in clear terms as follows:
   a. The exact alteration/amendment should be specified and indicated where the amendment occurred in the original project. (Page no. Clause no. etc.)
   b. Alteration in the budgetary status should be clearly indicated and the revised budget form should be submitted
   c. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval
   d. If the amendment demands a re-look at the toxicity or side effects to patients, the same should be documented
   e. If there are any amendments in the trial design, these must be incorporated in the protocol, and other study documents. These revised documents should be submitted for approval of the IHEC and only then can they be implemented
   f. Any deviation-Violation/waiver in the protocol must be informed to the IHEC within the stipulated period for review
7. Final report along with summary of findings and presentations/publications if any on closure of the study should be submitted to IHEC

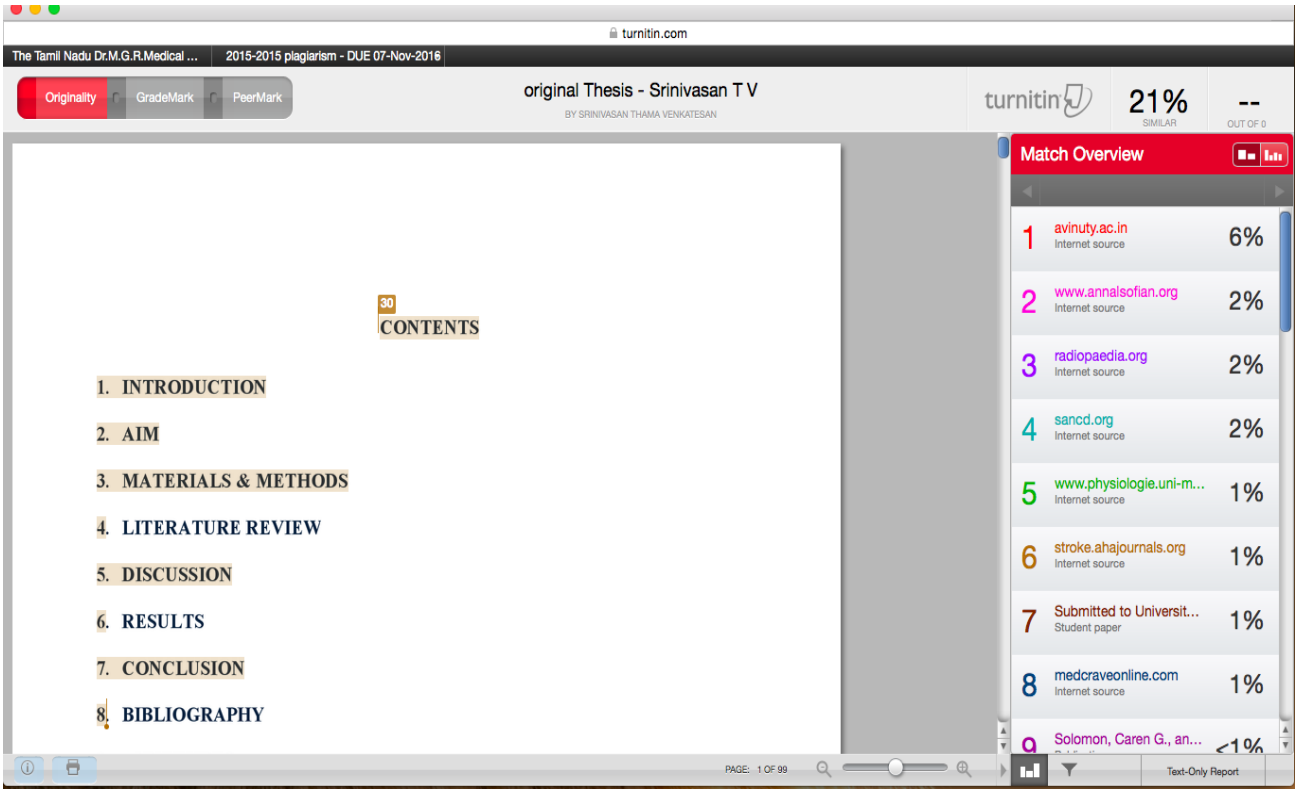
Kindly note this approval is subject to ratification in the forthcoming full board review meeting of the IHEC.

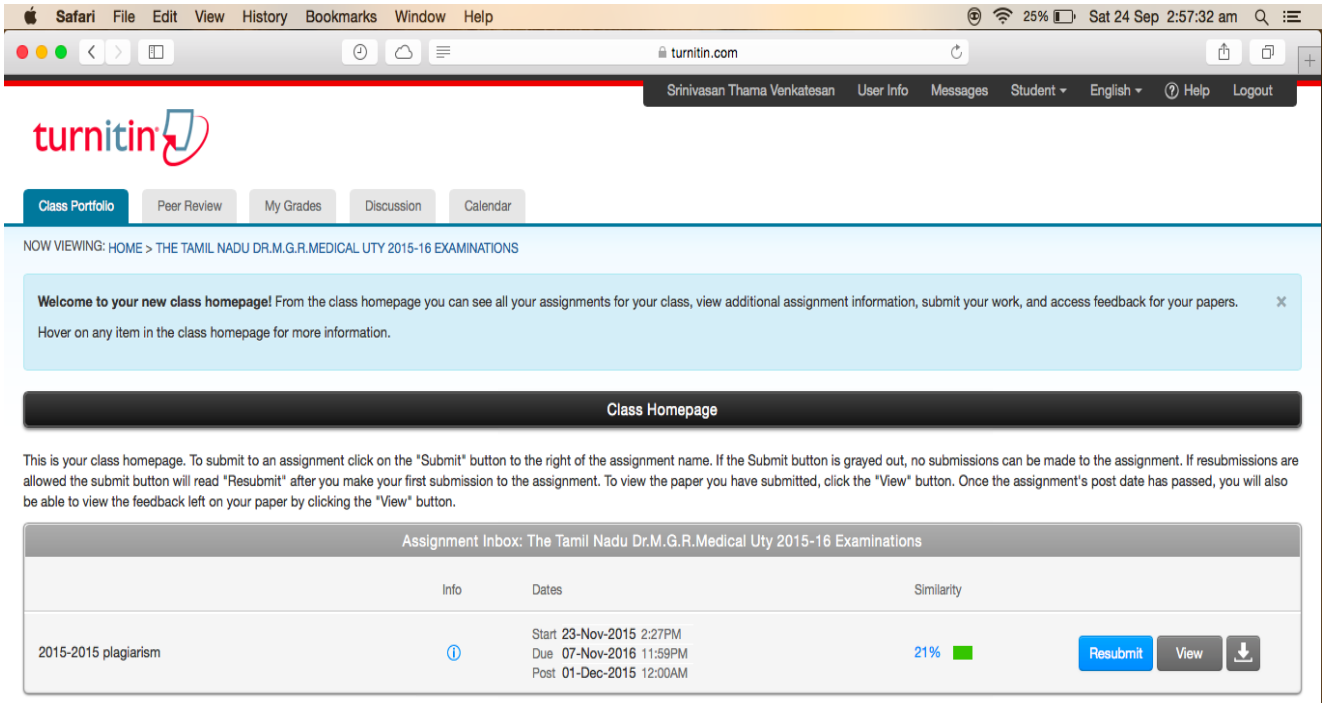
Thanking You,

Yours Sincerely,

SECRETARY
PSG IMS&R
COIMBATORE-641004.
INSTITUTIONAL HUMAN ETHICS COMMITTEE

Dr S Bhuvaneshwari
Member-Secretary
Institutional Human Ethics Committee


turnitin.com
The Tamil Nadu Dr.M.G.R.Medical ...
2015-2015 plagiarism - DUE 07-Nov-2016
Originality
GradeMark
PeerMark
original Thesis - Srinivasan T V
BY SRINIVASAN THAMA VENKATESAN
turnitin
21%
SIMILAR
--
OUT OF 0
30
CONTENTS
1. INTRODUCTION
2. AIM
3. MATERIALS & METHODS
4. LITERATURE REVIEW
5. DISCUSSION
6. RESULTS
7. CONCLUSION
8. BIBLIOGRAPHY
PAGE: 1 OF 99
Match Overview
1 avinuty.ac.in Internet source 6%
2 www.annalsofian.org Internet source 2%
3 radiopaedia.org Internet source 2%
4 sancd.org Internet source 2%
5 www.physiologie.uni-m... Internet source 1%
6 stroke.ahajournals.org Internet source 1%
7 Submitted to Universit... Student paper 1%
8 medcraveonline.com Internet source 1%
9 Solomon, Caren G., an... <1%
Text-Only Report


Safari File Edit View History Bookmarks Window Help
25% Sat 24 Sep 2:57:32 am
turnitin.com
Srinivasan Thama Venkatesan User Info Messages Student English Help Logout
turnitin
Class Portfolio
Peer Review
My Grades
Discussion
Calendar
NOW VIEWING: HOME > THE TAMIL NADU DR.M.G.R.MEDICAL UTY 2015-16 EXAMINATIONS
Welcome to your new class homepage! From the class homepage you can see all your assignments for your class, view additional assignment information, submit your work, and access feedback for your papers.
Hover on any item in the class homepage for more information.
Class Homepage
This is your class homepage. To submit to an assignment click on the "Submit" button to the right of the assignment name. If the Submit button is grayed out, no submissions can be made to the assignment. If resubmissions are allowed the submit button will read "Resubmit" after you make your first submission to the assignment. To view the paper you have submitted, click the "View" button. Once the assignment's post date has passed, you will also be able to view the feedback left on your paper by clicking the "View" button.
Assignment Inbox: The Tamil Nadu Dr.M.G.R.Medical Uty 2015-16 Examinations
Info
Dates
Similarity
2015-2015 plagiarism
Start 23-Nov-2015 2:27PM
Due 07-Nov-2016 11:59PM
Post 01-Dec-2015 12:00AM
21%
Resubmit
View

CONTENTS

TITLE

PROSPECTIVE STUDY OF CORRELATION OF CLINICAL FEATURES, MRI BRAIN – MRA INTRACRANIUM,CAROTID VERTEBRAL ARTERIAL DOPPLER STUDY IN ISCHEMIC STROKE

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Stroke is a worldwide concern. Approximately two hundred lakh people every year will suffer from stroke and of these fifty lakh will not survive[2,3]. In the developed nations, stroke is the leading cause for disability, second cause of dementia and third cause of death. Stroke is the second common cause of death and fourth leading cause of disability globally[1] .Stroke is an important cause for epilepsy, falls & depression in developed nations[4] and is a leading cause of functional impairments, with 20% of survivors required hospital and nursing care for a prolonged period more then 3 months duration and 15% - 30% of patient develop permanent disability[5].Stroke is a life-changing both financially and physically. Stroke is not only a traumatic experience to the patients but for their near and dear ones.

Global Stroke estimates:

- 400-800 strokes per 100,000[6]
- 57 lakh Deaths[7]
- 160 lakhs new acute strokes every year[1]
- 28,500,000 DALYs (disability adjusted life-year)[8]
- 28-30 day case fatality ranges from 17%-35%Stroke[9]

Morbidity and Mortality in India

- Prevalence 90-222 per 100,000[2]
- 102, 620 deaths [10]

Morbidity and Mortality in India (continued)

- 14,40,000-16,00,004 cases of new acute strokes per year [11,12]
- 6,398,000 DALY's[13]
- 12 percentage of strokes occur in the population aged <40 years [14]
- 28-30 day case fatality ranges from eighteen to forty one percentage[2,3]

A stroke is caused by acute interruption of blood supply to the brain region it supplies, usually because of a blood vessel bursts or blocked by a thrombus or emboli. This cuts off the supply of vital oxygen and nutrients to the brain, accumulation of free radicals causing damage to the brain tissue. An acute total occlusion of the vessels to the brain can cause death of the brain tissue within four to ten minutes. The WHO clinically defines "stroke as the rapid development of clinical signs and symptoms of a focal neurological disturbance lasting more than 24 hours or leading to death with no apparent cause other than vascular origin "[11]. Among CVA the Ischemic strokes constitutes 50%–85%.[9] Hemorrhagic strokes–Subarachnoid hemorrhage and intra-cerebral hemorrhage, account for 1%-7% and 7%-27% respectively of all strokes worldwide.[9] In a study done which is tabulated below, hemorrhagic stroke have significantly higher mortality when compared with ischemic stroke.

TABLE 1: Death rate (in percentage) @ One month, One year, and Five years after stroke

Source: [15]

	1 Month	1 Year	5 Years
Ischaemic stroke	10	23	52
Intra-cerebral hemorrhage	52	62	70
Subarachnoid haemorrhage	45	48	52

TABLE 2: TOAST CLASSIFICATION

TOAST Classification Of Subtypes Of Acute Ischemic Stroke

- **Large Vessel atherosclerosis (emboli/thrombus)**
- **Cardioembolic stroke**
- **Small-vessel occlusion(lacunar infarcts)**
- **Stroke related to other determined etiology**
- **Stroke - undetermined etiology**
  - **a. Two or more causes identified**
  - **b. Negative evaluation**
  - **c. Incomplete evaluation**

TABLE 3: National Institute of Health stroke scale (NIHSS)

NIHSS	Severity of stroke
0	Nil symptoms
1-4	Minor Stroke
5-15	Moderate Stroke
16-20	Moderate to Severe Stroke
21-42	Severe Stroke

In a study conducted in west China in 2012 which was later published in AHA, American heart study compared and analysed the anterior circulation involvement with the posterior circulation strokes.This study compared, the frequency of symptoms and signs associated with the 2 major vascular territories. Among the study population of 1174 consecutive patients with a diagnosis of posterior circulation (PC) involvement or anterior circulation (AC) involvement which was confirmed by magnetic resonance imaging in the Chengdu Stroke Registry. There was 302 posterior and the remaining 872 anterior circulation strokes. Homolateral hemiplegia (PC, 53.6% versus AC, 74.9%), central facial/lingual palsy (PC, 40.7% versus AC, 62.2%), and hemisensory deficits (PC, 36.4% versus AC, 34.2%) were the 3 most common symptoms and signs in PC and AC.

The signs with high specificity involving PC strokes were Horner's syndrome (PC 4.0% versus AC 0%), crossed sensory deficits (3.0% versus 0%;), quadrantanopia (1.3% versus 0%), oculomotor nerve palsy (4.0% versus 0%), and crossed motor deficits (4.0% versus 0.1%).In short MRI and other radiological imaging modalities was the key determining factors which helped in differentiating between the two different vascular territories.[80]

In another study conducted in National Cardiovascular CenterJapan among ischemic stroke patients,comparing the NIHSS and the clinical outcome using modified Rankins scale score < 2 at 3 months.Of 302 patients studied, NIHSS at baseline was correlated with the favorable outcome. NIHSS ≤5 for patients with posterior circulation stroke (sensitivity, 84%; specificity, 81%) and ≤8 for patients with anterior circulation stroke (sensitivity, 80%; specificity, 82%).[16]

Brain imaging plays a pivotal role in diagnosis, prognosis,management of stroke patients. Various modalities of imaging such as

- CT scan, CT angiography, CT perfusion studies
- MRI,MRA,MRI – DWI, MRI-PWI
- Carotid and vertebral duplex, transcranial Doppler (TCD), combined duplex and TCD

The sensitivity of standard noncontrast CT for brain ischemia increases after 24 hours. The presence of early signs in CT such as infarction implies a worse prognosis. The presence of the followingsigns is associated with an increased risk and poor prognosis[17]

Early signs of infarction include the following:

- Hypo attenuation of one-third or more of MCA
- Obliteration of Lentiform nucleus
- Cortical sulcal effacement
- Focal parenchymal hypo attenuation
- Loss of the insular ribbon or obscuration of the Sylvian fissure
- Hyperdense MCA sign
- Loss of gray-white matter differentiation in basal ganglia[18,19,20,21]

MRI have certain caveats such as takes time and not readily available like CT but have significantly higher sensitivity and specificity in the diagnosis of acute infarction in the first few hours after onset.[22]

- Diffusion-weighted imaging (DWI) / ADC:

Diffusion restriction may be seen within minutes following the onset of Ischemia correlates well with infarct core for a detailed discussion of DWI and ADC in stroke.

- T2-weighted imaging and FLAIR:

Less sensitive than DWI in the first few hours

Parenchymal change loss of normal signal void in large arteries may be visible immediately after 6-12 hours infarcted tissue becomes high signal.

Sulcal effacement and mass effect develop and become maximal in the first few days.

- T1

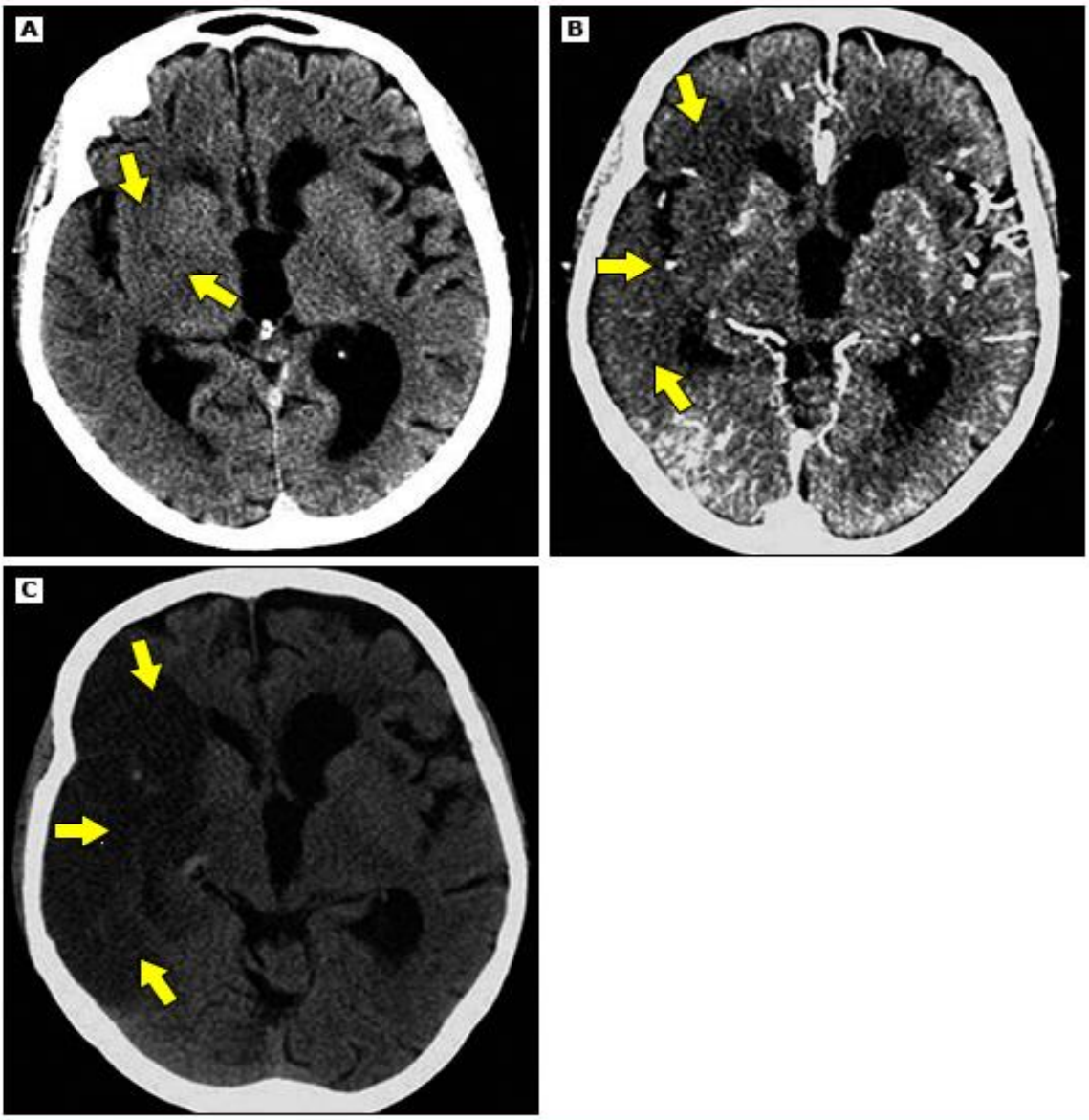
Low intensity roughly mirrors high T2 / FLAIR signal

Cortical laminar necrosis or pseudolaminar necrosis may be seen as a ribbon of intrinsic high T1 signal, usually after 2 weeks (although it can be seen earlier).

- SWI:

Highly sensitive in the detection of haemorrhage [22,23,24]

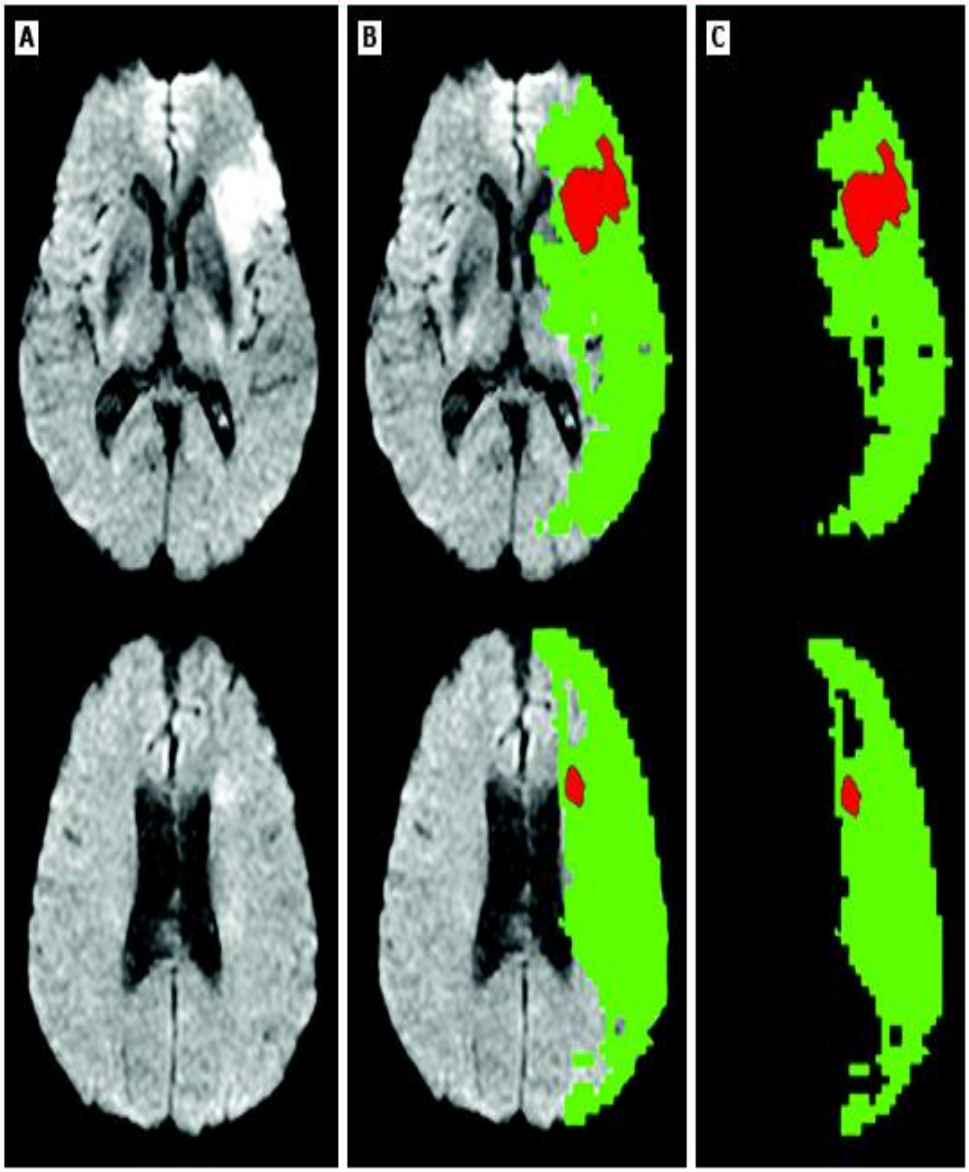
Acute ischemic stroke on CT angiography source image



An 88-year-old woman admitted with acute left hemiparesis and dysarthria 90 minutes after the onset of symptoms. Baseline noncontrast head CT (A) shows early ischemic changes in the right MCA territory (arrows). CTA-SI image (B) reveals hypoattenuation in the right MCA territory (arrows), which corresponds to the final infarct (C) on follow-up CT scan (arrows).

Figure 1

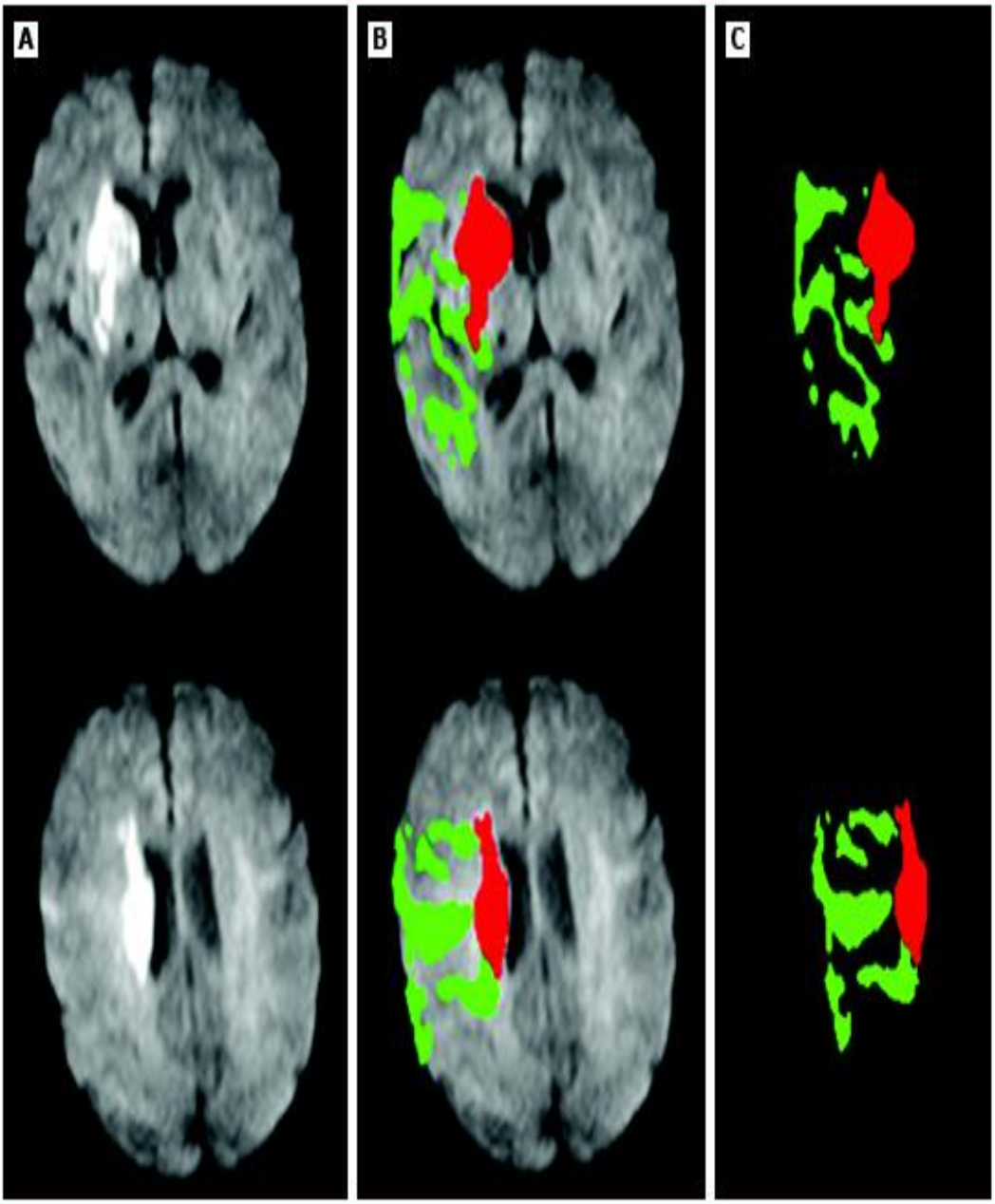
Classic pattern of DWI-PWI mismatch



Representative case with classic pattern of mismatch.
(A) Diffusion weighted-image (DWI).
(B) DWI abnormal lesion (shown in red) and hypoperfusion lesion (shown in green) superimposed on DWI.
(C) DWI abnormal lesion and hypoperfusion lesion with brain image (DWI) removed.

Figure 2

Nonclassic pattern of DWI-PWI mismatch



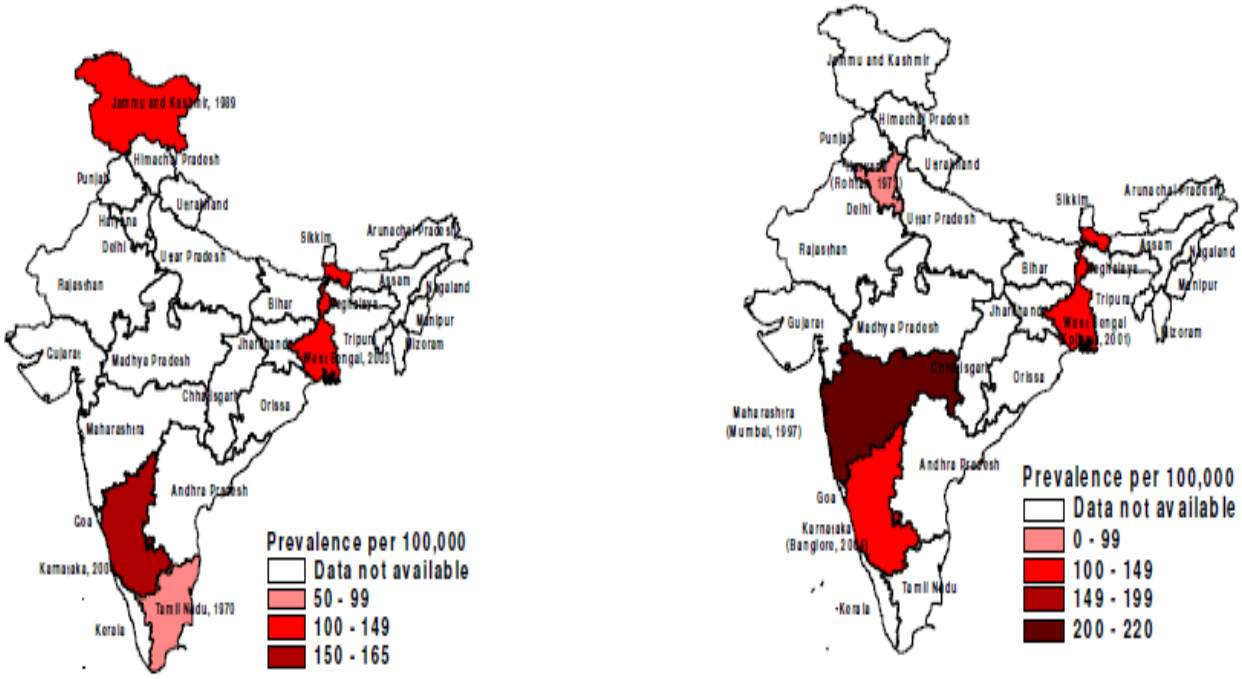
Representative case with nonclassic pattern of mismatch.
(A) Diffusion weighted-image (DWI).
(B) DWI abnormal lesion (red) and hypoperfusion lesion (green) superimposed on DWI. Note fragmentation of the pattern.
(C) DWI abnormal lesion and hypoperfusion lesion with brain image (DWI) removed.

Figure 3

Figure 4:

Crude prevalence rates for stroke Rural India 1970-2004 Vs. Urban India 1973-2004

RURAL **URBAN**



AIMS AND OBJECTIVES:

- Percentages of intracranial , extracranial or both involvement in stroke
- Percentages of anterior and posterior circulation strokes in PSGIMSR
- Most commonly involved arteries
- Correlation of infarcted area in the brain and MRA + CV doppler
- The acute, chronic or both nature of the brain infarcts

- Correlation of clinical presentations and infarcted areas
- Percentages of recurrent CVA
- Percentages of intracranial anomalies
- Percentage of thrombolysis in PSGIMSR
- NIHSS comparisons between right and the left sided brain involvement
- Percentage of smokers, alcoholics or both
- Percentages of DM, SHT, Dyslipidemia
- Incidental ANA-IF positivity and hyperhomocystenemias in PSGIMSR
- Decision regarding medical or surgical treatment based on the arterial studies
- Prevention of strokes

MATERIALS AND METHODOLOGY

Type of study: Prospective and clinical study

Duration Of Study: One year (June 2015 – June 2016)

Sample : 75 patients

Study volunteers - specific population & age group :

Patients between 20-80years admitted with ischemic stroke of both sexes

Location: PSG Hospitals, PSGIMS&R, and Coimbatore.

INCLUSION CRITERIA:

- Age between 20-80years
- Patients admitted in general medical ward/neurology ward with ischemic stroke

EXCLUSION CRITERIA:

- Age < 20years
- Age > 80years
- Venous strokes
- Cardio embolic strokes

METHODOLOGY:

The study is based on prospective collection of data of patients aged between 20-80 years diagnosed as ischemic stroke who got admitted in medical ward or neurology ward in a tertiary care centre where systematic computer coding for registry is used.

Patients admitted at PSG hospitals, PSGIMS&R, Coimbatore diagnosed with ischemic stroke confirmed with MRI –MRA imaging at admission and meeting the inclusion criteria as mentioned above, during the study period of June, 2015 to June, 2016 are taken into consideration for the study. A proforma was made which included detailed history, baseline NIHSS admission, clinical examination and requisite investigations available in the hospital. After informed consent from patients, history and risk factors attributable to the stroke are collected in detail. Investigations like complete hemogram, routine urine analysis, blood sugar, serum elecctrolytes, serum creatinine, blood urea, chest X-ray, FLP, ECHO, electrocardiogram, MRI – MRA intracranium brain, CV Doppler were done in all patients.

HbA1C was done if patient was found to be Diabetic based on RBS. Investigations like ANA- IF and serum Homocysteine was done in patients, later when PSG Primegrant of Rs One Lakh was awarded which I had applied earlier.

Statistical tools:

The data collected from the patients admitted with acute stoke and relevanr information was tabulated using Microsoft Excel. The patient was admitted in Neurology and General Medicine. The data are reported as the mean +/- SD or the median, depending on their distribution. Frequencies are reported / documented in percentages. The differences in quantitiative variables between groups were assessed by means of the unpaired t test. Non parametric test Wilcoxon signed test were use to assess the qualitative variables. To assess the difference in categoric variables

between groups chi square test was used. Two tailed test was considered significant for all statistical tests when p value is <0.05. All data were analysed with a statistical software package (SSPS version 16.0 for microsoft windows)

REVIEW OF LITERATURE

History and Background:

Stroke was first recognized by Hippocratespopularly known as father of medicine. The oath which is often taken by the medical graduates worldwide is named after Hippocrates. Initially it was termed as “apoplexy” in Greek language (meaning “struck down by violence”).

Johann Jacob Wepfer (1620–1695), the next well known person in the field of stroke. He studied on the corpses of deceased due to apoplexy, discovered that blood supply might be disrupted to the brain in the deceased either due to blocked arteries in few or bleed [25]

Rudolf Virchow, father of modern pathology was first to describe, thromboembolism was a significant factor causing stroke. During nineteenth century, he described the term thrombosis which can detached and be mobile to form embolus causing cardio-embolic stroke[26].

In the year 1928, apoplexy was subcategorised based on the cause. After which the term cerebrovascular accident has been come into norms. Recently in 2011, use of this term cerebrovascular accident have been discouraged due to reasons that the word *accident* insufficiently highlights the modifiability of the underlying risk factors[27,28]. Now it is used as cerebrovascular incident interchangeably.

DEFINITION AND CLASSIFICATION :

Although stroke more common in older adults, the same also occurs in lower incidence in neonates, infants, children and young adults age (< 40 Years), resulting in significant morbidity and mortality[29]. Stroke is a clinical syndrome which is classified broadly as:

- Ischaemic strokes – These are caused by sudden occlusion of arteries supplying the brain, either due to thrombus or embolli intacranially or extracranilly causing impaired blood supply to a region of brain causing infarction. It accounts for 50-85% of strokes worldwide[30].
- Haemorrhagic strokes – defined as bleed which occurs within substance of the brain, intracerebral haemorrhage or contained within the subarachnoid space[31].
- Transient ischemic attacks (TIAs) are defined as temporary neurological deficit, symptoms lasting less than 24hrs. It is due to inadequate cerebral or ocular blood supply as a result of arterial thrombus or embolism associated with arterial, cardiac or hematological disease, indeed awarning for impending stroke. Radiologically, there is no findings in MRI or CT.[32].

Epidemiology:

The All India registry of stroke observed that in various cities across India, Calcutta, Mumbai, and Trivandrum incidence of strokes were higher comparatively than United States (107 /100,000 per year), European population (61-111 / 100,000 per year), Australia (99/ 100,000 year) but similar to Chinese city - Changasha.

[33,34,35,36,37]

The crude prevalence rate was4.72 per 1,000 during the year 2003-04 in Calcutta. Stroke was the cause of death in 13% very similar to death due tocoronary artery disease (14%). The 30-day case fatality rate(CFR) in Indian city Calcutta was 41.08%,significantly higher when compared with Westernnations (17-33%)[33,34,35,36,37.] This reflects poor health facilities whencompared to the western countries.

Risk Factors

They are further classified into

- Modifiable Risks
- Non-modifiable Risks

Non modifiable risks:

Risk factors like age, gender, race/ethinicity and family genetics come under non modifiable risk factors.

1. The male : female sex ratio among affected individuals is 7:1 in India. Among the females high risk groups are oral contraceptives and pregnancy.[46,47]
2. Blacks are more commonly affected with stroke compared to whites
3. Genetic factors for hypertension, Von Willebrand factors, Sickle cell disease among family may also contribute to increased risk of stroke.

Modifiable risks:

Cigarette smoking, alcohol consumption, dyslipidemia, abdominal obesity, Sedentary life style, Hypertension, Diabetes mellitus, OCP, pregnancy, illicit drugabuse, migraine headache, prior stroke or TIA.

One interesting risk factor was previous infection with *Chlamydia pneumoniae*, raising the possibility of infection related immune reaction .[48,49]

Etiology:

A study from a hospital-based registry of Southern India has attributedmajority of strokes had large artery atherosclerosis, closely followed by lacunar causes and cardioembolic.

Rare causes such as

- Takayasusyndrome
- MoyaMoya disease
- Carotid dissection
- Hyperhomocysteinemia
- Anticardiolipin antibody
- Protein Sdeficiency
- Fibromuscular dysplasia
- Radiotherapy,
- MDS
- Sickle cell anemia
- Takayasu's arteritis
- Polyarteritisnodosa (PAN)
- Wegener's granulomatosis (WG)
- Behcet's disease
- Amyloidangiopathy andother genetic causes.

The remaining about 27% of the study population who had ischemic stroke was idiopathic. Among cardioembolic stroke, rheumatic heart disease (RHD) and ischemic heart disease (IHD) were majorcause.[38]

The Indian Collaborative Acute Stroke Study (ICASS); a multicentric study conducted among 2,162 admitted stroke patients across southern, northern, and western India; observed ischemic stroke in77%, hemorrhagic stroke in 22%, and unspecified stroke in 1% cases based on cerebral computed tomography (CT).[39] The extracranial carotid disease is the etiological factor in 25-26% and intracranial carotid disease in 30% of ischemic stroke cases.[40]

Stroke in young

In India, stroke below the age of 40 yrs was increasing a an alarming rate ranging anywhere between 15% - 30%.In a study at All India Institute of Medical Sciences (AIIMS), out of the 2,634 patients admitted with AIS, 440 patients (16.7%) were in the age group of 18-45 years and majority (83.4%) were male.[41] The rate of CVT was twelve times more common in India than western nations. In a stroke study reported that around 50% of total strokes in young women were related to pregnancy & puerperium, 95% of which were due toCVT.[42] MoyaMoya disease, not so common was reported in India[43]. Takayasu's arteritis[44] is often reported in Indian population in both the sexes. Stroke in children and adolescents due to Takayasu's arteritis was reported. In another study of young patients with hemorrhagic stroke,[45] again the subjects were predominantly males. The important risks in this study was hypertension, hypercholestremia, alcohol and anticoagulant usage. The various

etiologies in this study were hypertension, vascular malformation, coagulopathy, CVT, thrombocytopenia, vasculitis, and cryptogenic stroke[45].

Table 4: Genetic causes of stroke

TYPES:	GENE MUTATION	VESSEL AFFECTED
CADASIL	Notch 3 receptor	Small vessel disease
CARASIL	Notch 3 receptor	Small vessel disease
Fabry disease	α-galactosidase - A	Both large and small vessel disease
MELAS (maternal)	Transfer RNA	Complex
Marfan syndrome	Fibrillin	Cardioembolism and arterial dissection
Ehlers–Danlos syndrome – type IV	Collagen - III	

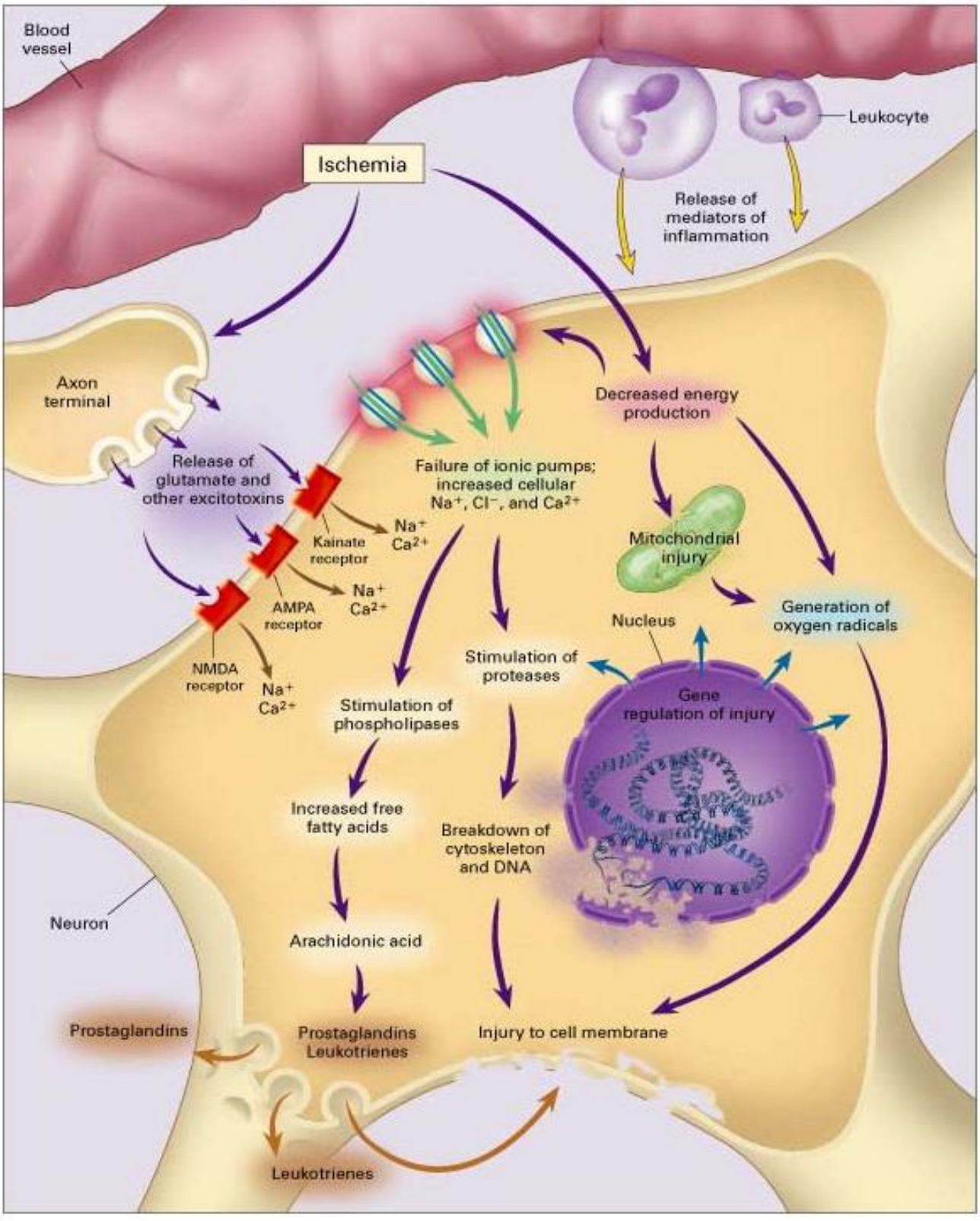


Figure 5: Pathophysiology of stroke

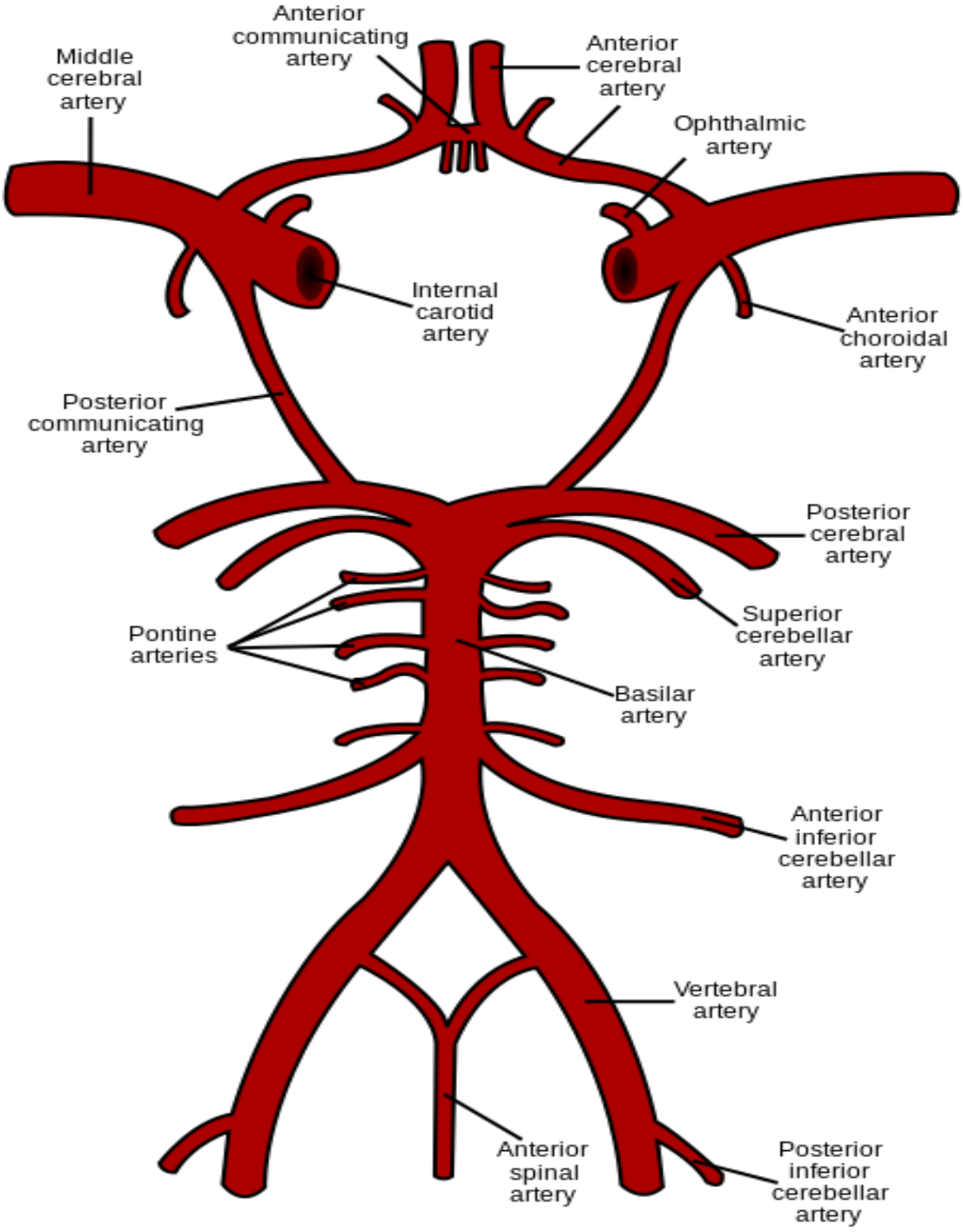


Figure 6: Circle of Willis

Table 5: Common Large-Vessel Ischemic Stroke Syndromes

Syndrome	Anatomy involved	Major symptoms	Vessels involved	Etiology
Left MCA	Left frontal/parietal cortex & sub cortical structures	Aphasia,right visual field cut,motor/sensory deficits,face>arm>leg weakness;left gaze prefernce	Left MCA or major branch,left ICA	Emboli/intrinsic atherothrombosis
Right MCA	Right frontal/parietal cortex & sub cortical structures	Neglect syndrome,agnosia,apraxia, left motor/sensory deficits,visual field deficit,right gaze preference	Right MCA or major branch,Rt. ICA	Same as left MCA
Left ACA	Left frontal and parasagittal area	Behaviour changes,Leg> arm weakness,speech disturbances	Left ACA	Emboli/intrinsic atherothrombosis
Right ACA	Right frontal and parasagittal areas	Behaviour changes,Leg> arm weakness	Right ACA	Same as left ACA
Brainstem	Pons/midbrain/medulla/cerebellum	Opthalmoplegia,bilateral motor deficits,ataxia/dysmeria,nausea,vomiting,vertigo,coma,altered mentation	Basilar artery	Embolism from proximal leision/atherothrombosis
PCA	Upper midbrain/occipital cortex/subcortex/thalamus/medial temporal lobes	Visual field cut,motor or sensory loss,seizures,gaze problems,CN III deficits	PCA,thalamic perforators	Embolism from proximal leision/atherothrombosis

Table 6: Common Lacunar Stroke Syndromes

Syndrome	Vessels Involved	Brain Location	Symptoms
Pure motor Hemiparesis	Lenticulostriate/pontine Perforator	Internal capsule, pons	Unilateral weakness only
Mixed motor/sensory	Lenticulosriate/ Thalamic perforator or Deep white matter vessel	Internal capsule, deep white matter, thalamus	Motor and sensory deficits
Pure sensory	Thalamic Perforator	Posterior thalamus	Loss of contralateral sensory modalities
Ataxic hemiparesis	Lenticulostriate /basilar/pontine/ Perforator	Internal capsule, basis pontis	Unilateral weakness with prominent ataxia
Dysarthria/clumpsy hand syndrome	Lenticulostriate or deep white matter vessel	Internal capsule, deep white matter	Prominent dysarthria with isolated hand weakness

Anatomy of blood vessels and clinical presentation:

- **ANATOMY OF CEREBRAL CIRCULATION:**

Brain is the highest perfused organ in the body. It receives about 20% of total circulation and also have maximum consumption of oxygen in the blood. It is mainly supplied by two pairs of large arteries – internal carotid arteries (ICA) and vertebral arteries.[78]

Internal carotid artery(ICA) supply about 3/5th of cerebrum. The two vertebral arteries join together to form basilar artery which supplies cerebellum and brain stem. These two arterial circulation join together with the help of communicating branches to form circle of willis.[77,79]

The internal carotid group produce three main vessel branches which include –

1. Ophthalmic artery – supplies the meninges, contents of orbit.
2. Anterior cerebral artery (ACA) – these are pair of arteries supplying the medial portions of frontal lobes along with prefrontal and supplementary motor cortex and superior medial parietal lobes. They are further subclassified into 5 smaller branches called callosal arteries as they also supply the carpus callosum. Due to collateral supply by anterior communicating artery, stroke due to ACA is very rare.
   - Clinical relevance –
     - Occlusion of ACA may cause following symptoms:
     - Contralateral lower limb upper motor neuron type (UMN) of weakness
     - Contralateral sensory loss in lower limb
     - Due to frontal lobe involvement – Behavioural abnormalities, cortical release reflexes – grasp reflex, sucking reflex, gegenhalten phenomenon.
     - Transcortical aphasia.
3. Middle cerebral artery (MCA) – It also a paired artery which supplies anterior temporal and insular cortices. They are connected to ACA with the help of anterior communicating branches and connected with PCA with the help of

posterior communicating branches. They are further divided into 4 parts or segments in their course of supply. They supply the bulk of lateral surface of the hemispheres along with speech areas (Broca's and Wernicke's areas)

- Clinical relevance –
  - Contralateral upper and lower limb UMN type of paralysis
  - Contralateral sensory loss over face and arm
  - If lenticulostriate branches of MCA are involved –
    - If involvement of dominant hemisphere → aphasia
    - Involvement of non-dominant hemisphere → contralateral neglect syndrome

4. Posterior cerebral artery – paired arteries which supplies the posterior part which includes occipital lobe. It is divided into 2 branches – cortical and ganglionic vessels.

- Clinical relevance –
  - Contralateral loss of pain and temperature
  - Contralateral homonymous hemianopia with macular sparing
  - Alexia and agraphia
  - Weber's syndrome – 3rd cranial nerve palsy &contralateral hemiplegia
  - Horner's syndrome

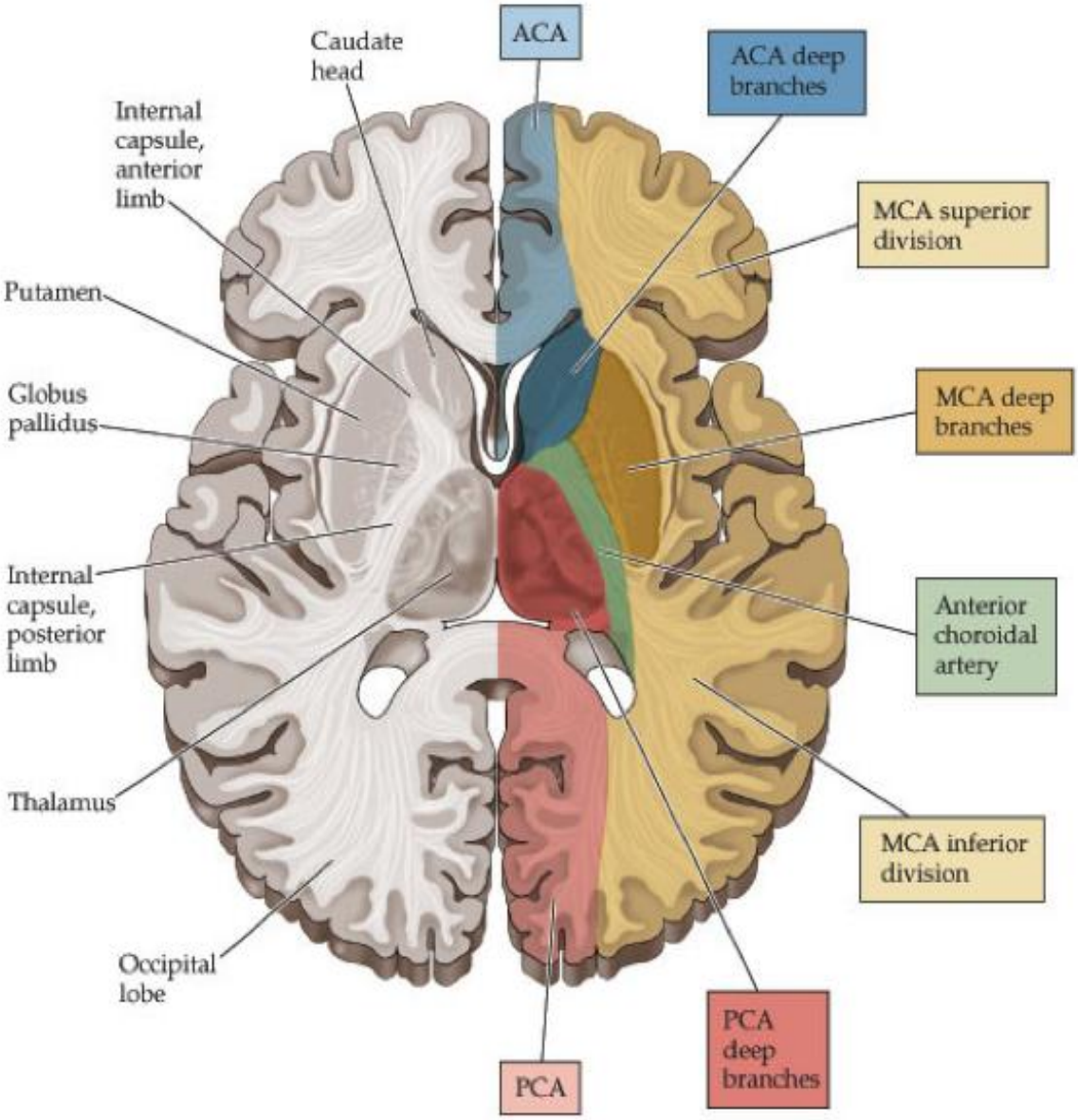


Figure 7: Vascular territories of Brain

Table 7: PCA Syndrome: (P1 territory)

Syndrome	Clinical features	Localization
Claude's Syndrome	CNIII + contralateral ataxia	Red nucleus / Cerebral penduncle
Weber's Syndrome	CN III + Hemiplegia	Medial mid brain /cerebral peduncle
Benedikt's Syndrome	CN III + hemiplegia + ataxia	Red nucleus / Medial mid brain
Subthalamic Nucleus	Contralateral hemiballismus	
Thalamic Dejerine-Roussy Syndrome	Contralateral hemisensory loss and agonizing pain	Thalamus

Table 8: Common Clinical findings and presentations - anatomical and vascular localization of Posterior Circulation

Vascular territory	Anatomical location	Stroke syndrome	Clinical findings
Unilateral PCA	Occipital lobe	Contralateral homonymous hemianopsia	Homonymous hemianopsia with macular sparing
	Dominant occipital lobe*plus* splenium of corpus callosum	Alexia without agraphia	Homonymous hemianopsia and alexia without agraphia
	Ventral occipital cortex; infracalcarine	Achromatopsia	Loss of color differentiation contralateral to the side of the lesion, can be associated with a quadrantanopsia
	Optic radiation OR supracalcarine	Inferior quadrantanopsia	Inferior quadrantanopsia
	Myers loop (temporal lobe) or infracalcarine	Superior quadrantanopsia	Superior quadrantanopsia
Bilateral PCA	Both occipital lobes	Cortical blindness	Bilateral cortical blindness with normal phthalmological findings
		Anton's syndrome	Cortical blindness with denial of blindness and confabulations or visual hallucinations
PCA–MCA border zone regions	Bilateral ventral–mesial occipital–temporal border zones	Prosopagnosia	Inability to recognize familiar faces and/or interpret facial expressions. Retained ability to identify with speech or unique feature (e.g., glasses, facial hair, tattoo, etc.)

	Bilateral occipital–parietal border zones	Balint's syndrome	Optic ataxia (inability to reach targets with visual guidance), oculomotor apraxia (inability to volitionally direct gaze), and simultagnosia (inability to synthesize objects within a visual field)
	Unilateral left temporal–parietal border zone	Transcortical sensory aphasia	Impaired comprehension, fluent speech but preserved repetition
PICA	Inferior posterior cerebellar hemisphere, inferior vermis, lateral medulla	Lateral medullary or Wallenberg syndrome Superior cerebellar artery syndrome	Vertigo, nausea, vomiting, ipsilateral facial numbness and dysmetria, Horner's syndrome, dysphagia, and ataxia dysphonia contralateral hemisensory loss below the face
SCA	Dorsolateral upper brainstem and cerebellum and superior cerebellar peduncle	Superior cerebellar artery syndrome	Ipsilateral limb ataxia, vertigo, nystagmus, dysarthria, and gait ataxia
AICA	Ipsilateral labyrinth, lateral pontine tegmentum and brachium pontis, ICP	Lateral pontine syndrome	Ipsilateral dysmetria, hearing loss, Horner's syndrome, choreiform dyskinesia, contralateral thermoanalgesia
Top of the BA	Midbrain, thalamus, and mesial temporal lobes and occipital lobes	Top of the basilar syndrome	Somnolence, peduncular hallucinosis, convergence nystagmus, skew deviation, oscillatory eye movements, Colliers sign (retraction and elevation of eye lids), vertical gaze paralysis
Mid-BA	Lateral and medial pons	Lateral mid-pontine syndrome	Ipsilateral loss of facial sensation and motor function of the trigeminal nerve, ipsilateral dysmetria

		Medial mid-pontine syndrome	Ipsilateral dysmetria, contralateral arm and leg weakness and gaze deviation
Pontine paramedian penetrators	Anteromedial pons	Dorsal mid-pontine syndrome	Ipsilateral nuclear facial palsy, horizontal gaze palsy, and contralateral arm and leg weakness
Short pontine circumferential arteries	Anterolateral pons	Superior medial pontine syndrome	Ipsilateral intranuclear ophthalmoplegia, palatal, facial, pharyngeal and/or ocular myoclonus, dysmetria, and contralateral arm and leg weakness, ocular bobbing
Proximal BA	Lower pons	Locked-in syndrome	Quadriplegia, horizontal gaze paralysis, bifacial, paralysis, and tongue and mandibular weakness. Awareness is spared
VA	Medulla and cervical spinal cord	Medial medullary or Dejerine syndrome (intracranial disease may lead to Wallenberg syndrome)	Contralateral arm and leg weakness, hemibody loss of tactile, vibration, position sense, ipsilateral tongue paralysis
Anterior spinal artery		Anterior spinal artery syndrome	Quadriparesis, bilateral pain and temperature loss, decreased sphincter tone, autonomic instability, and hyperreflexia. Proprioception spared

Table 9: Syndromes associated with respective vascular territories of Brain

• Middle cerebral artery	• Middle cerebral artery: complete
	• Middle cerebral artery: superior division
	• Middle cerebral artery: inferior division
	• Gerstmann syndrome
	• Ataxic hemiparesis
• Posterior cerebral artery	• Posterior cerebral artery: unilateral occipital
	• Balint syndrome
	• Cortical blindness (Anton syndrome)
	• Weber syndrome
	• Alexia without agraphia
	• Thalamic pain syndrome (Dejerine–Roussy syndrome)
• Anterior inferior cerebellar artery	• Lateral pontine syndrome (Marie–Foix syndrome)
• Posterior inferior cerebellar artery	• Lateral medullary syndrome (Wallenberg syndrome)
• Basilar artery	• Locked-in syndrome
	• Lateral pontine syndrome (Marie–Foix syndrome)
	• Ventral pontine syndrome (Raymond syndrome)
	• Ventral pontine syndrome (Millard–Gubler syndrome)
	• Inferior medial pontine syndrome (Foville syndrome)
	• Ataxic hemiparesis
	• Cortical blindness (Anton syndrome)
	• Medial medullary syndrome (Dejerine syndrome)
• Vertebral artery	• Medial medullary syndrome (Dejerine syndrome)
	• Lateral medullary syndrome (Wallenberg syndrome)
• Anterior spinal artery	• ASAS
• Posterior spinal artery	• PSAS [50]

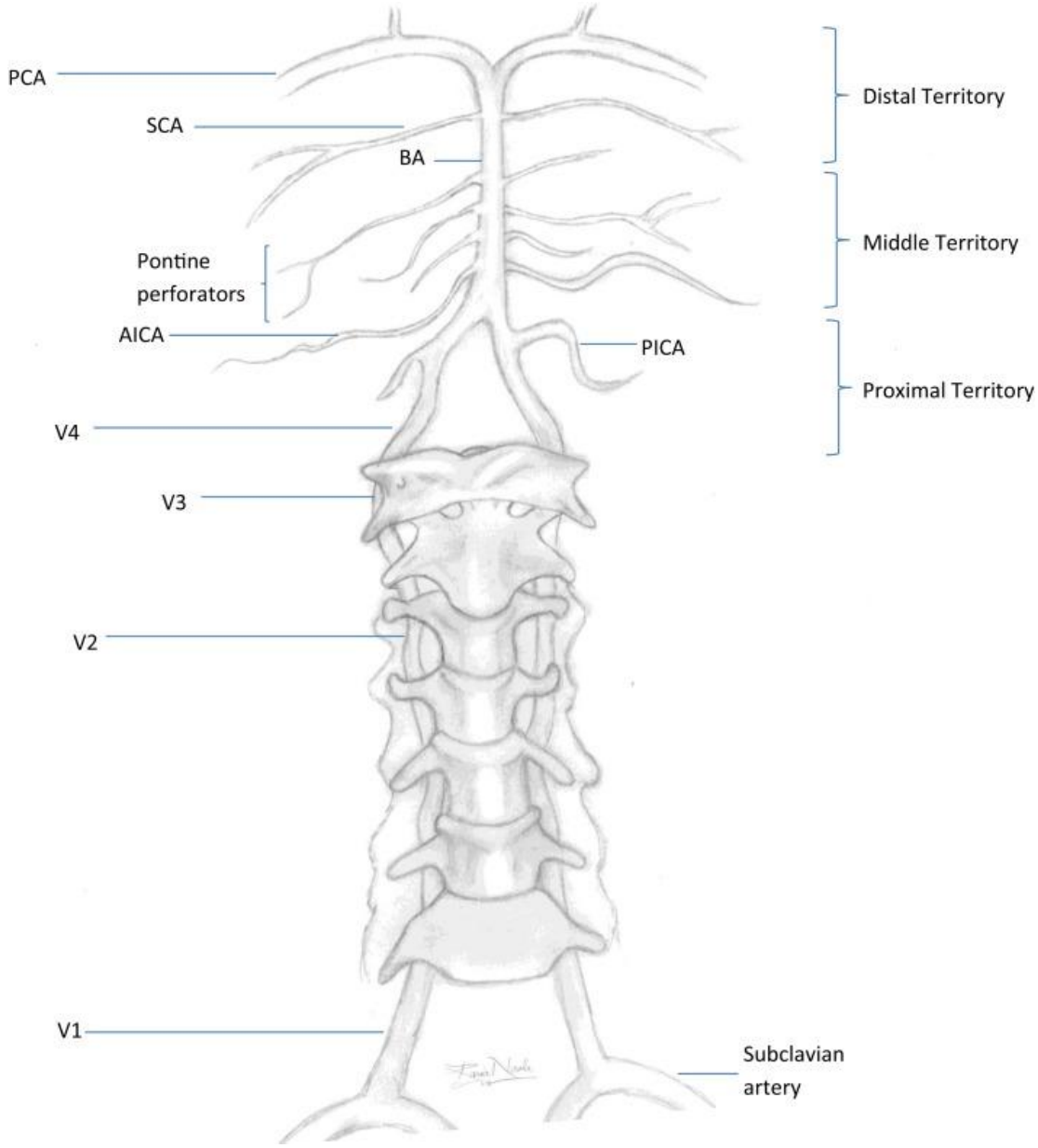


Figure 8

Vertebrobasilar system. PCA, posterior cerebral artery; SCA, superior cerebral artery; BA, basilar artery; AICA, anterior inferior cerebellar artery; PICA, posterior inferior cerebellar artery; V1–V4, segments of the vertebral artery. Proximal territory, areas supplied by the intracranial VAs and PICAs up to the VB junction; middle territory, BA and AICAs up to the SCAs; distal territory, rostral BA, SCAs, and PCAs.

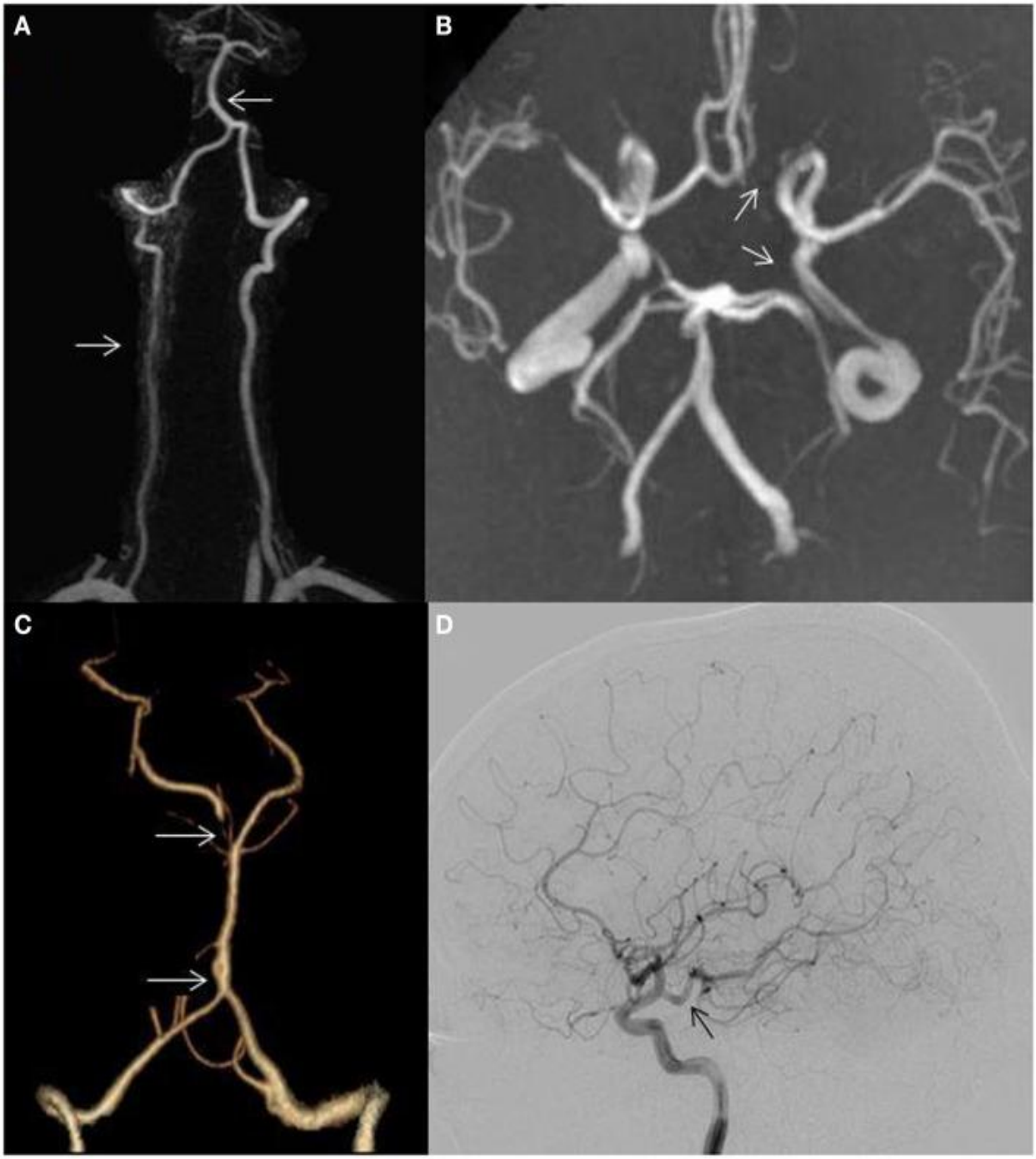


Figure 9: MRA (A) Hypoplastic right vertebral artery (bottom arrow); basilar artery displacement opposite to the dominant vertebral artery (top arrow); **(B)** incomplete circle of Willis, absent left posterior communicating artery (bottom arrow), absent left A1 segment (top arrow); **(C)** fenestration of the basilar artery (bottom arrow); hypoplastic right P1 segment (top arrow) and **(D)** posterior cerebral artery arising directly from the internal carotid artery (fetal variant, arrow).

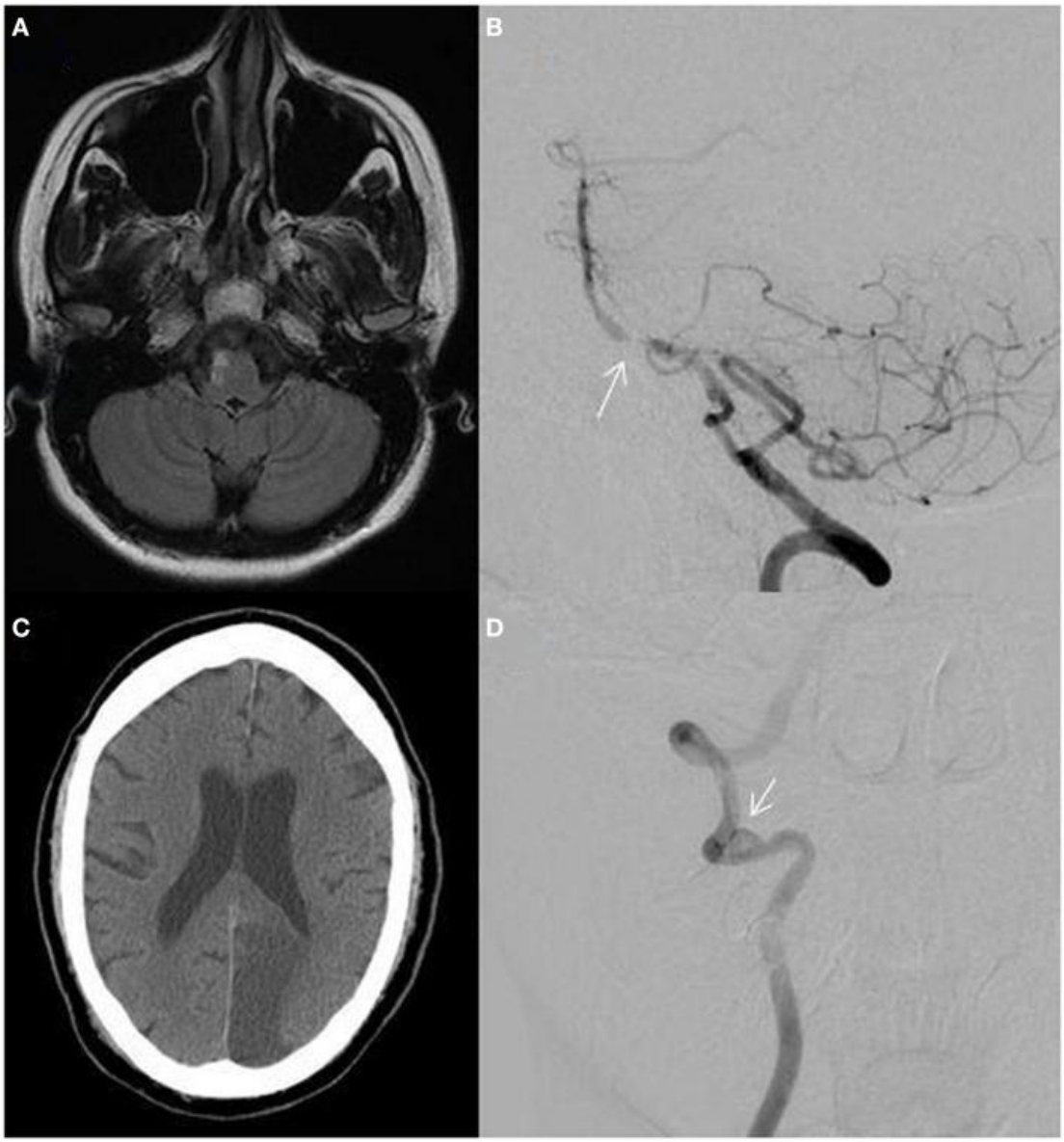


Figure 10 : MRI (A) MRI fluid-attenuated inversion recovery (FLAIR) sequence showing a right lateral medullary infarction in a 32-year-old woman with a hypoplastic right vertebral artery shown in Figure
Figure2A;
2A; **(B)** catheter angiogram of a 55-year-old African-American man showing stenosis at the vertebrobasilar junction (arrow); **(C)** non-contrast CT showing a left posterior cerebral artery territory infarction in a 60-year-old man with atrial fibrillation; **(D)** catheter angiogram showing a right distal vertebral dissecting aneurysm with intraluminal thrombus (arrow) in a 19-year-old man presenting with vertigo, ataxia, and a right cerebellar infarction.
post-content

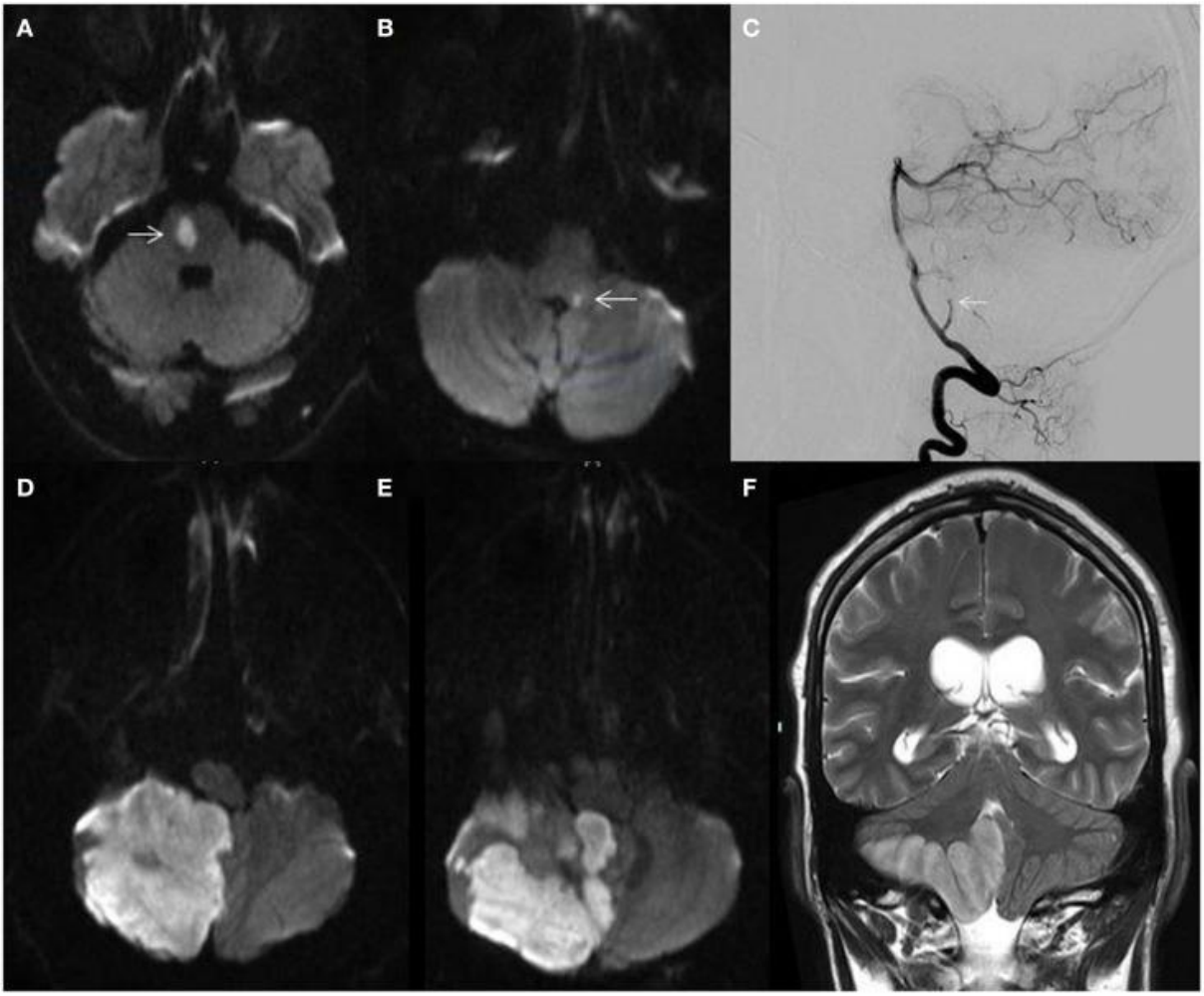


Figure 11: (A) MRI diffusion-weighted image (DWI) demonstrating a right ventral pontine infarction (arrow) in a 62-year-old man with fluctuating left sided weakness; **(B)** MRI-DWI showing a small dorsal left medullary infarction (arrow) in a 58-year-old man with hypertension and hyperlipidemia presenting with acute isolated vertigo; **(C)** catheter angiogram showing cut-off of the right posterior inferior cerebellar artery (arrow); **(D,E)** MR DWI showing massive right cerebellar hemispheric and vermian infarction; **(F)** MRI T2-weighted sequence demonstrating right cerebellar infarction with edema and mass effect.
post-content

Management :

First to recognise even the subtle clinical presentation is very important in the management of CVA. Recognising the hemiparesis would not be great deal, but mild sensory disturbances, gait disturbances, double vision, blurring of vision, giddiness, difficulty in swallowing, seizures in young females, TIA's, deviation of angle of mouth. Detailed history taking including the assessment of risk factors and clinical examination is the key in stroke management.

Rapid assessment by various scoring systems gives a broad idea of severity. The various scoring systems are NIHSS, mRS. CT plays a important role in management of CVA, it helps in ruling out hemorraghic strokes. Treatment differs totally once heamorraghic stroke is ruled out. MRI with MRA with diffusion and perfusion weighted image helps in assesing the ischemic penumbra in ischemic stroke, which is reversible.

The goals in the initial phase include:

- Attention and protection of airway, breathing, and circulation
- Assess for any reversible condition which closely mimics stroke
- Determining eligibility for thrombolytic therapy

Routine baseline investigations (TABLE 10):

Complete haemogram

Blood sugars	Hypo or hyper glycemia
Urine routine	Diabetes , infection
Serum electrolytes & ABG	Hyponatremia or hypokalaemia or hyperkalaemia
Renal function tests	Renal failure
Fasting lipids	Dyslipidemia
Homocysteine levels	Homocystinemia
Serology	Vasculitis , Infections, HIV, VDRL
ECG	Left ventricular hypertrophy (LVH), Atrial fibrillation, arrhythmias, AMI
Echocardiography	Infective endocarditis, atrial myxoma
ESR, CRP	Autoimmune causes-vasculitis, SLE
To do (optional)	
ANA profile, APLA	APLA syndrome, SLE, vasculitis
Coagulation profile	Protein C and S deficiency, anti thrombin III deficiency, hyperfibrinogenemia.
Genetic studies (optional)	CADASIL, MELAS, CARASIL.,etc

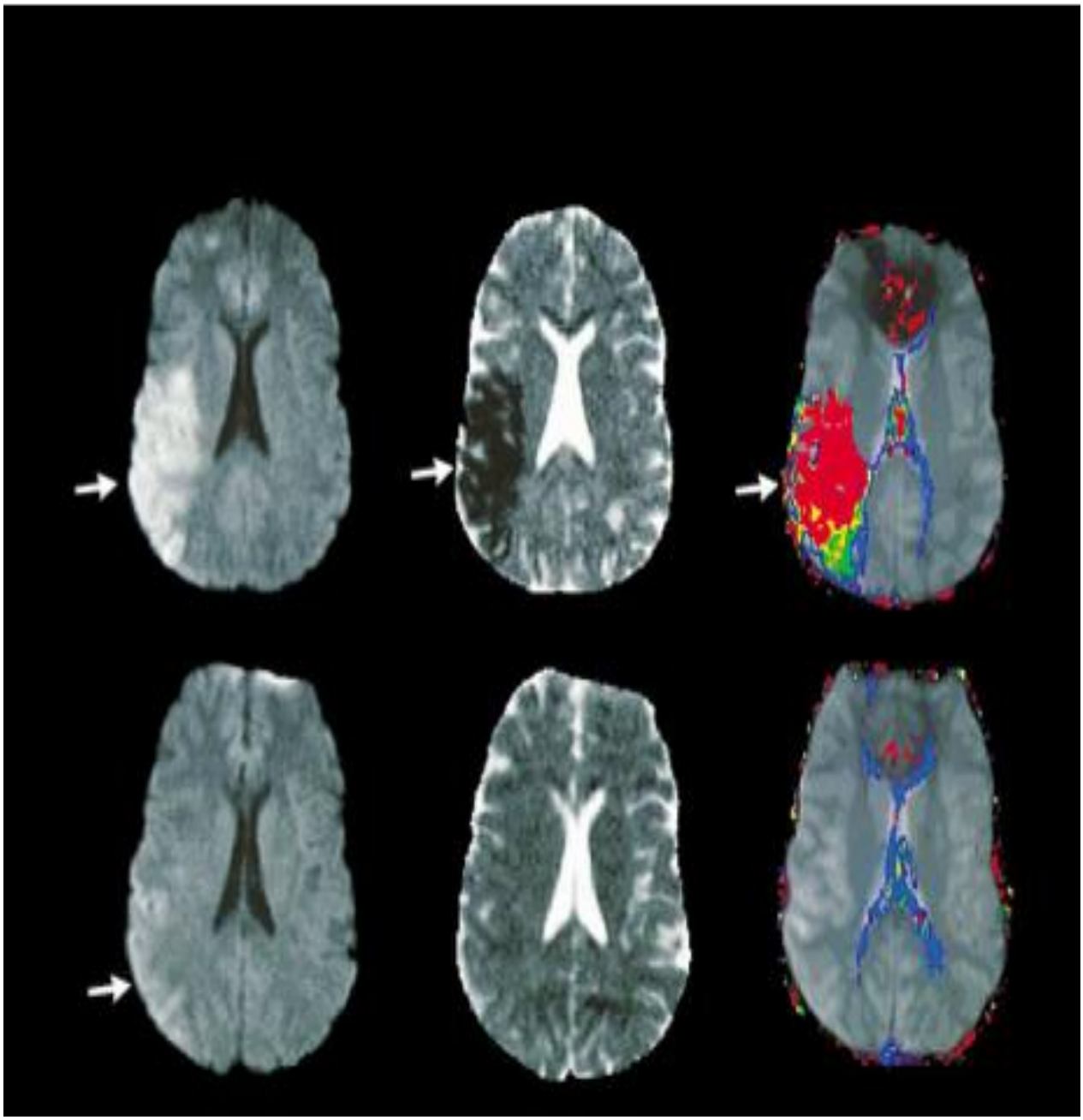


Figure 12. MRI Study Showing Improvements in Diffusion and Perfusion Abnormalities in the Right Cerebral Hemisphere after Intra-arterial Administration of Tissue Plasminogen Activator in a 27-Year-Old Woman with Left Hemiparesis. The top row shows abnormalities (arrows) in a diffusion-weighted image, an image constructed with the apparent diffusion coefficient, and a perfusion-weighted image from a representative scan obtained 2.5 hours after the onset of symptoms, before thrombolytic therapy was begun. In the perfusion-weighted images, red represents a delay in the delivery of the bolus of eight or more seconds, yellow a delay of six to less than eight seconds, green a delay of four to less than six seconds, and blue a delay of two to less than four seconds. Recanalization of the posterior division of the right middle cerebral artery occurred four hours after the onset of symptoms. The bottom row shows corresponding images obtained three hours after vessel recanalization following intra- arterial administration of tissue plasminogen activator. A substantial decrease in the size of the lesion (arrow) on the diffusion- weighted image and complete resolution of the lesion on the image constructed with the apparent diffusion coefficient and on the perfusion-weighted image can be seen. The neurologic deficit, as measured by the score on the National Institutes of Health Stroke

- Patients should be maintained with oxygen if hypoxic and should be maintained at 94%[52]
- **Differential diagnosis** are Migraine aura,seizureswith postictal paresis (Todd's paralysis),aphasia or neglect, CNS tumors or abscess, conversion disorder, hypertensive encephalopathy, head trauma, MELAS, multiple sclerosis, PRES, Spinal cord disorder such as compressive myelopathy, spinal dural arteriovenous fistula , SDH, syncope, systemic infection, Toxic metabolic disturbance such as hypoglycemia exogenous drug intoxication, transient global amnesia, viral encephalitis, Wernicke's encephalopathy.
- To watch for signs of endocarditis, cholesterol emboli, purpura, ecchymoses, or evidence of recent surgery or other invasive procedures, particularly if reliable history is not forthcoming. The funduscopic examination may be helpful if there are cholesterol emboli or papilledema. The head should be examined for signs of trauma. A tongue laceration may suggest a seizure.
- Fluids – most of the patient present with volume depletion, administration of isotonic saline is sensible, avoidance of hypotonic solutions is advisable since it can worsen cerebral edema.
- Both hypo and hyperglycemia have poor outcomes, maintaining RBS 140 – 180 mg /dl is advisable.[52-57]
- Supine position is preferred for nonhypoxic patients with acute ischemic stroke who are able to tolerate lying flat[52]
- Patients with AIS, symptoms may be provoked when there is decrease in preload Like standing, sitting, or elevating the head of the bed. [58,59]

- 30 degrees head elevation is recommended for patient who are prone for aspiration, cardiac and pulmonary issues, elevated intracranial pressure. Otherwise keeping the bed flat for 24 hrs. is advised.
- Hyperthermia worsens ischemic process , therefore it should be controlled with antipyretics.
- According to the latest guidelines when not choosing thrombolytic therapy, the blood pressure reduction should be made only when the blood pressure exceeds 220/120 mmHg or earlier in the following conditions active IHD , heart failure, aortic dissection, hypertensive encephalopathy, acute renal failure, or pre-eclampsia/eclampsia [60,61]
- Blood pressure while thrombolysing a patient needs proper management <185 mmHg of systolic and diastolic of 110 mmHg for at least 24 hrs. .[60,61]

There is some evidence from small randomized controlled trials suggesting that early initiation of selective serotonin-reuptake inhibitors (SSRIs) after ischemic stroke for patients with hemiparesis but without depression enhances motor recovery and reduces dependency [62,63]. However, it is unclear if this apparent benefit occurs through a reduction in poststroke depression or some other mechanism.

Prevention of the following complications: MI, CHF, Dysphagia, Aspiration pneumonia, Urinary infection, DVT, Pulmonary embolism, Dehydration, Malnutrition, Pressure sores, joint complications and contractures.

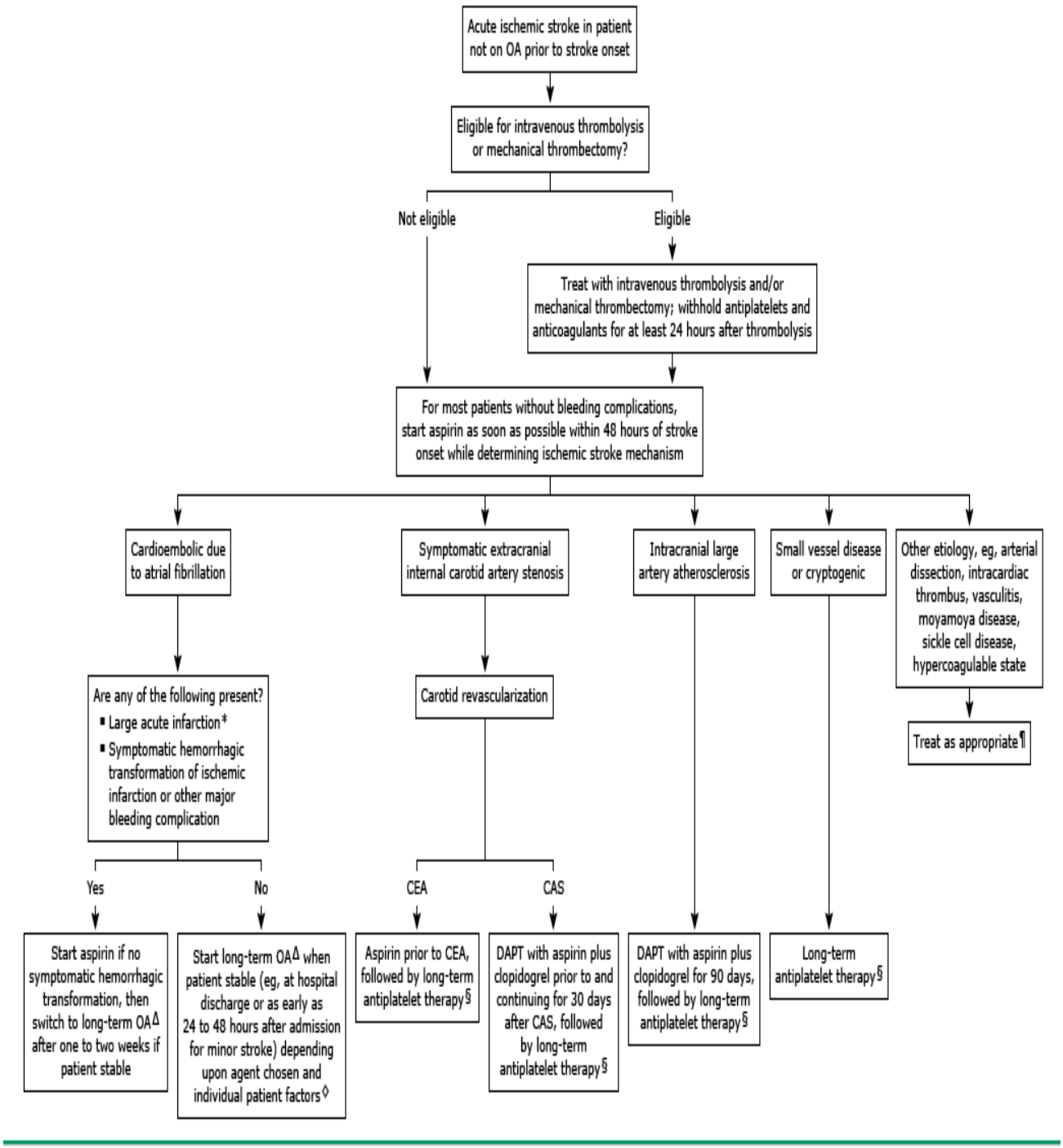
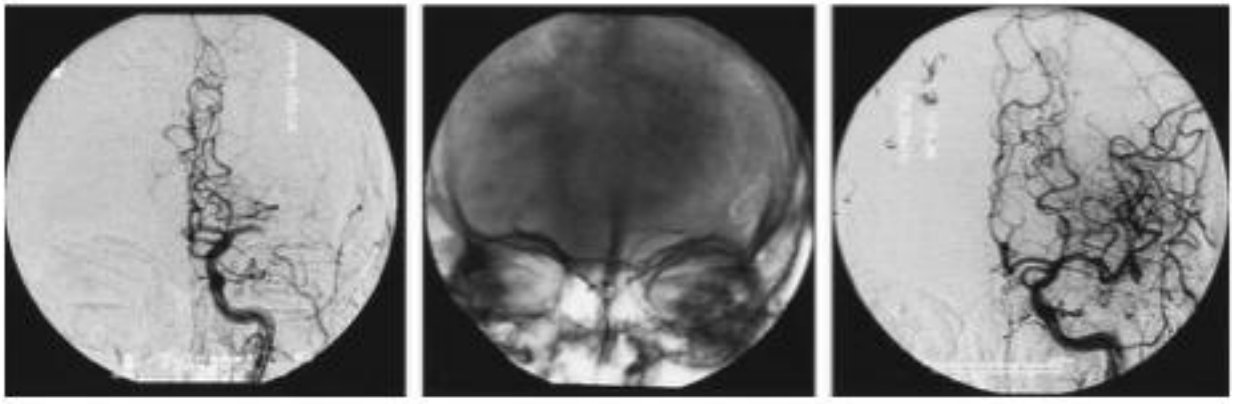


Figure 13: General approach to anti thrombotic therapy in AIS

❖ ***Early fibrinolysis :***

According to AHA/ASA guidelines for the management of AIS, indications for thrombolysing a patient with fibrinolytics to restore the blood flow to the brain and early resolution or neurological deficits. For this purpose, most commonly used fibrinolytic drug is r-tPA (recombinant tissue plasminogen activator). Streptokinase which has a major role in AMI, has high incidence of complications in acute AIS. Hence this drug is of not on regular use for AIS[69,70,71]. Individual presented to ED must be identified by inclusion and exclusion criteria where patient is a feasible candidate for fibrinolysis. They must be thrombolysed within a window period of maximum of 4.5hrs from the onset of symptoms.



2 Hours, 20 Minutes 4 Hours, 20 Minutes

Figure 14. Cerebral Arteriograms in a Patient with Dysphasia and Right Hemiplegia. A cerebral arteriogram shows the placement of the microcatheter tip just proximal to the embolic occlusion in the trunk of the left middle cerebral artery (arrow, left-hand panel). Intraarterial administration of r-tPA was initiated 2 hours and 20 minutes after the onset of symptoms. The center panel shows the position of the microcatheter after infusion of r-tPA over a two-hour period (arrow). The final cerebral arteriogram (right-hand panel) demonstrates complete recanalization of the left middle cerebral artery. The microcatheter tip can be seen at the distal end of the trunk of the right middle cerebral artery (just proximal to the arrow).

The 0.9 mg per Kg of body weight of rt- PA is approved by FDA. Firstly, bolus of 10% of the dose should be given over one minute, with remaining dose of 90% to be given over sixty minutes. Weight should be determined reliably. In a study in Japan, treatment with a lower dose of0.6 mg per kilogram r-tPA concluded that it had similar efficacy. 3rd generation plasminogen activators, such as tenecteplase and desmoteplase, are more fibrin specific than 2nd generations r-tPA and cause less activation of systemic complications.[69] Symptomatic ICH occurs in 1.7 to 8.0% of treated patients.[70,71,72,73] Administration of cryoprecipitate and platelets, for symptomatic hemorrhage[74,75,76]

- 10 units of cryoprecipitate (to increase fibrinogen and factor VIII
- 6-8 units of platelets to be given
- In patient receiving UFH consider giving protamine 1mg for every 100 U UFH

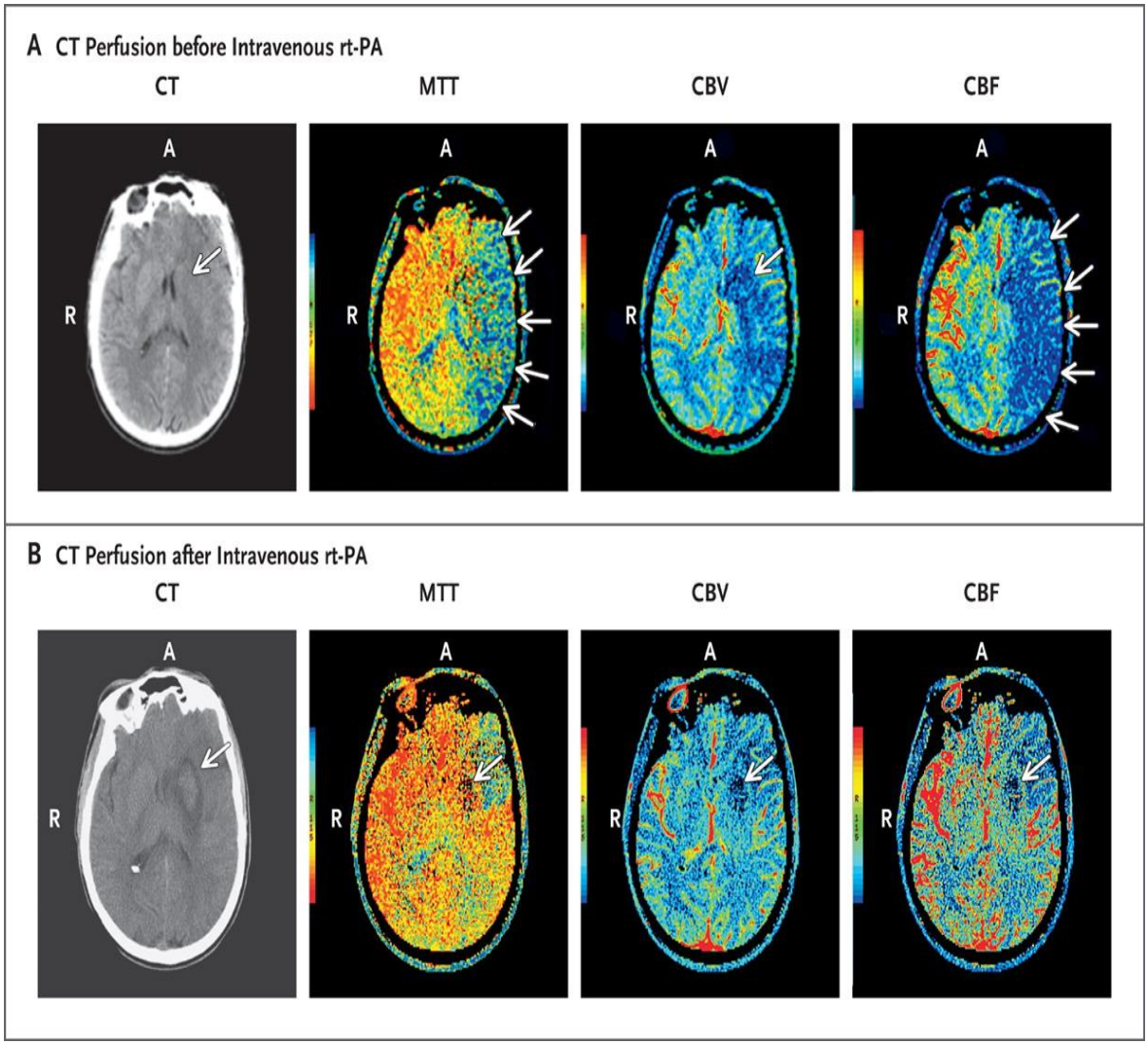


Figure 15. CT Perfusion Imaging in a Patient with Stroke, before and after Thrombolysis with Recombinant Tissue Plasminogen Activator (rt-PA). Standard CT images without contrast material (left two images) and CT perfusion images obtained during the first pass of an intravenous bolus of iodinated contrast material (right six images) are shown. Images were obtained before (Panel A) and after (Panel B) thrombolysis with rt-PA. Mathematical algorithms are used to create, from the perfusion data, maps of the mean transit time (MTT) (the difference in time between arterial inflow and venous outflow), cerebral blood volume (CBV), and cerebral blood flow (CBF). The CT scan without contrast material that was obtained before thrombolysis shows hypodensity in the area of the left caudate nucleus (arrow), with loss of definition between gray matter and white matter. The scan obtained after thrombolysis shows a central area of hyperdensity in the area of the left caudate nucleus, which is consistent with hemorrhage within the infarct zone, surrounded by an area of hypodensity (arrow), which is consistent with infarction. The CT perfusion maps in Panel A, obtained before thrombolysis, show prolonged MTT (arrow), decreased CBV (arrow), and decreased CBF (arrow) in the left hemisphere. There is some degree of mismatch between the CBV map (which emphasizes irreversible injury, primarily in the area of the left caudate nucleus) and the

CBF map (which shows an extensive area of abnormality throughout the left hemisphere, indicating tissue at risk). The CT perfusion maps in Panel B, obtained after thrombolysis, show some improvement in MTT, CBV, and CBF in most areas, although the focus of hypo perfusion in the area of the left caudate nucleus persists (arrows), which is consistent with the area of infarction shown on the CT scan obtained without contrast material. In these images, the color spectrum indicates the spectrum of values for each quantity. On the MTT map, the areas of fastest transit time appear red and those of slowest transit time appear blue. On the CBV and CBF maps, the area of greatest blood volume or blood flow appear red and those of least blood volume or blood flow appear blue. Although quantitative values can be assigned to these data, CT perfusion images are usuall $y interpreted qualitatively by comparing areas of normal with areas of abnormal perfusion. Areas without detectable perfusion are black. A denotes anterior, and R right.

INCLUSION AND EXCLUSION CRITERIA FOR THROMBOLYSIS IN AIS:

INCLUSION CRITERIA :

- Onset of symptoms < 3 hours before beginning treatment
- Onset time is defined as either the witnessed onset of symptoms or the time last known normal
- Age ≥18 years

EXCLUSION CRITERIA

- Significant head trauma or prior stroke in previous 3 months
- Symptoms suggestive of any history of haemorrhage stroke
- Intracranial neoplasm, arteriovenous malformation, or aneurysm
- Recent intracranial or intra-spinal surgery
- Arterial puncture at a non compressible site in the last seven days
- Elevated blood pressure (systolic >185 mm Hg or diastolic >110 mm Hg)
- Active internal bleeding
- Blood glucose concentration < 50mg/dl

- Acute bleeding diathesis, including but not limited to: Platelet count <1,00,000/mm³
- Heparin received within 48 hours, resulting in abnormally elevated aPTT greater than the upper limit of normal
- Current use of anticoagulant with INR >1.7 or PT >15 seconds and current use of direct thrombin inhibitors or direct factor Xa inhibitors with elevated sensitive laboratory tests (such as aPTT, INR, platelet count, and ECT; TT; or appropriate factor Xa activity assays)
- CT demonstrates multilobar infarction (hypodensity >1/3 cerebral hemisphere)

RELATIVE EXCLUSION CRITERIA :

- Only minor or rapidly improving stroke symptoms (clearing spontaneously)
- Seizure at onset with postictal residual neurological impairments
- Major surgery or serious trauma within previous 14 days
- Recent gastrointestinal or urinary tract haemorrhage (within previous 21 days)
- Pregnancy
- MI in the past three months

To extend IV tPA to 4.5 hours from symptom onset/last known normal, the following additional criteria MUST be met:

- Patient is < 80 years of age
- Patient does not have a history of both diabetes AND stroke

- Patient is not taking Warfarin (Coumadin) or any other anticoagulant regardless of INR/coagulation results
- NIHSS is < 25
- Written informed consent obtained from patient and/or family – required when IV tPA given within the 3-4.5 hour window.

❖ **Anti Coagulants:**

Anticoagulation is not recommended in all the patients with AIS in emergency condition. Drugs most commonly used are low molecular weight heparin (LMWH) or unfractionated heparin (UFH)[64]. They are to prevent venous thromboembolism. Only with following situations, anticoagulation therapy is indicated :

- Conditions with potential high risk of early cardiogenic reembolization
- Symptomatic dissection of arteries supplying the brain
- Symptomatic extra-cranial or intra-cranial atherosclerotic stenosis
- Basilar artery occlusion before or after intra-arterial pharmacological or mechanical thrombolysis
- Known hypercoagulable states
- Cerebral venous sinus thrombosis

- Anticoagulants should not be given for 24 hrs. post thrombolysis
- Early anticoagulation should be avoided when potential contraindications to anticoagulation are present, such as a large infarction (NIHSS >15), uncontrolled hypertension, or other bleeding conditions.

❖ ***Anti-thrombotic agents :***

They are mainly used for secondary prevention of stroke. Drugs most commonly used are aspirin. Clopidogrel and extender release dipyridamole. Newer drugs such dabigatran, apixaban and rivaroxaban are being studied for anticoagulation comparing with warfarin for primary end point. According to guidelines, aspirin should be given within 48hrs (Preferably within 24 hrs.) of onset of stroke to prevent mortality and primary end point.

Endovascular techniques – intra-arterial fibrinolysis, thrombo-embolectomy, suction thrombectomy, angioplasty and revascularisation are few among known endovascular procedures. The main aim of these is to recanalise the thrombosed vessel to improve the blood flow. For this a team of skilled neurologists, interventional radiologists, anaesthesiology, nursing and technical support for optimal success.

❖ **Neuro protection:**

Several novel neuro-protective agents like citicholine, traxoprodil, ONO-2506, magnesium, SSRI, DP-b99g naloxone, gangliosides, nimodipine, *N*-methyl-D-aspartate–receptor antagonists, antibodies to adhesion molecules, and free-radical scavengers and NXY-059 have been identified. They limit the infarct size and improve functional outcome 65. They act as free radicals scavengers and inhibit further occurrence of neuronal cell death.

❖ **Rehabilitation centres :**

The main aim of these centres is to improve the quality of life of stroke survivor by improving the skills to do the daily day to day activities. There were four recommendation and they are:

First, diagnostic evaluation, initial treatment, preventive therapy, and rehabilitation services should be provided in a coordinated setting.

Second, measures to prevent deep-vein thrombosis should be implemented.

Third, prevention of recurrent stroke and of complications of stroke should be given high priority.

Fourth, surveillance for the development of depression is important, since major depression occurs in 10 to 30 percent of patients who survive a stroke.[66,67,68] Dysphagia, urinary incontinence, immobility, focal weakness, aphasia, a tendency to fall, injury to the skin, bowel irregularity or fecal incontinence, and shoulder injury have to be assessed and treated. A team of neurologist, psychiatrist, occupational therapist, speech language therapists, dietician and social workers play an important role in achieving the desired result.

RESULTS:

In this study, about 75 consecutive cases admitted with AIS, which met inclusion and exclusion criteria are taken into the study. At presentation, along with complete history taking, relevant clinical examination, scoring was done, based on NIHSS at admission.

The following objectives were studied :

- Percentages of intracranial, extracranial or both involvement in stroke
- Percentages of anterior and posterior circulation strokes in PSGIMSR
- Most commonly involved arteries
- Correlation of infarcted area in the brain and MRA + CV doppler
- The acute, chronic or both nature of the brain infarcts
- Correlation of clinical presentations and infarcted areas
- Percentages of recurrent CVA
- Percentages of intracranial anomalies
- Percentage of thrombolysis in PSGIMSR
- NIHSS comparisons between right and the left sided brain involvement
- Percentage of smokers, alcoholics or both
- Percentages of DM, SHT, Dyslipidemia
- Incidental ANA-IF positivity and hyperhomocystenemias in PSGIMSR
- Decision regarding medical or surgical treatment based on the arterial studies
- Prevention of strokes

GENDER:

Among the study population of 75 patients, 62 (83%) were males and 13 (17%) were females suffered with AIS. Males are invariably more in number than females in this study.

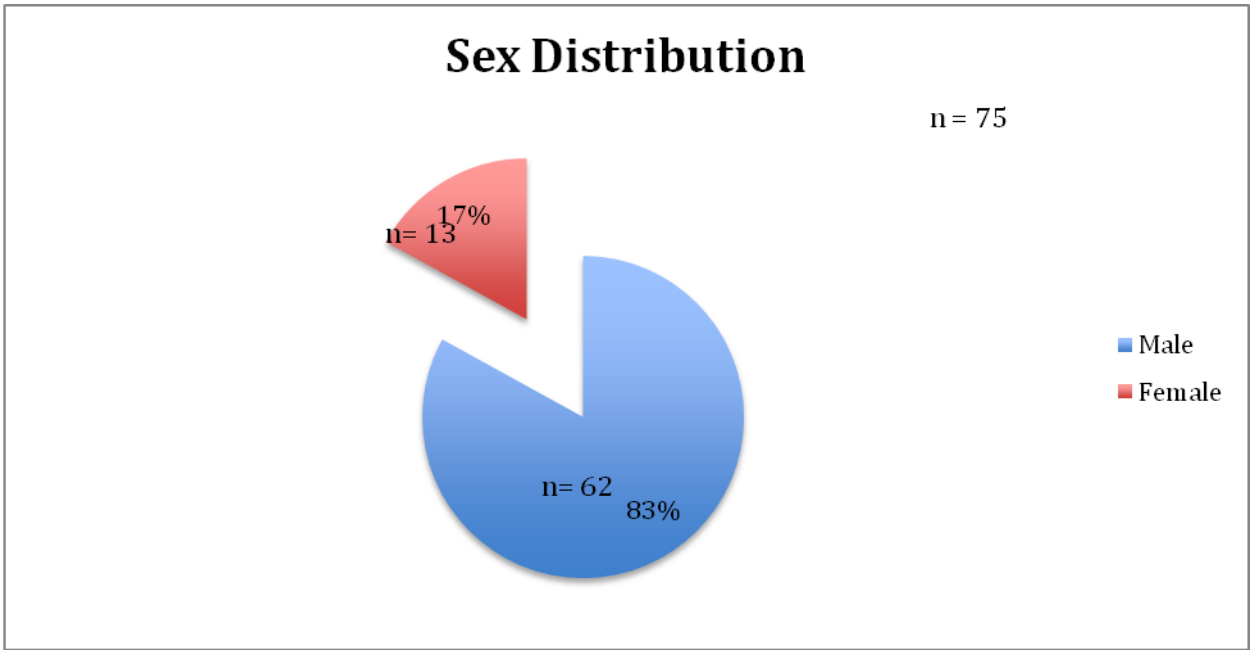


Figure 16

AGE:

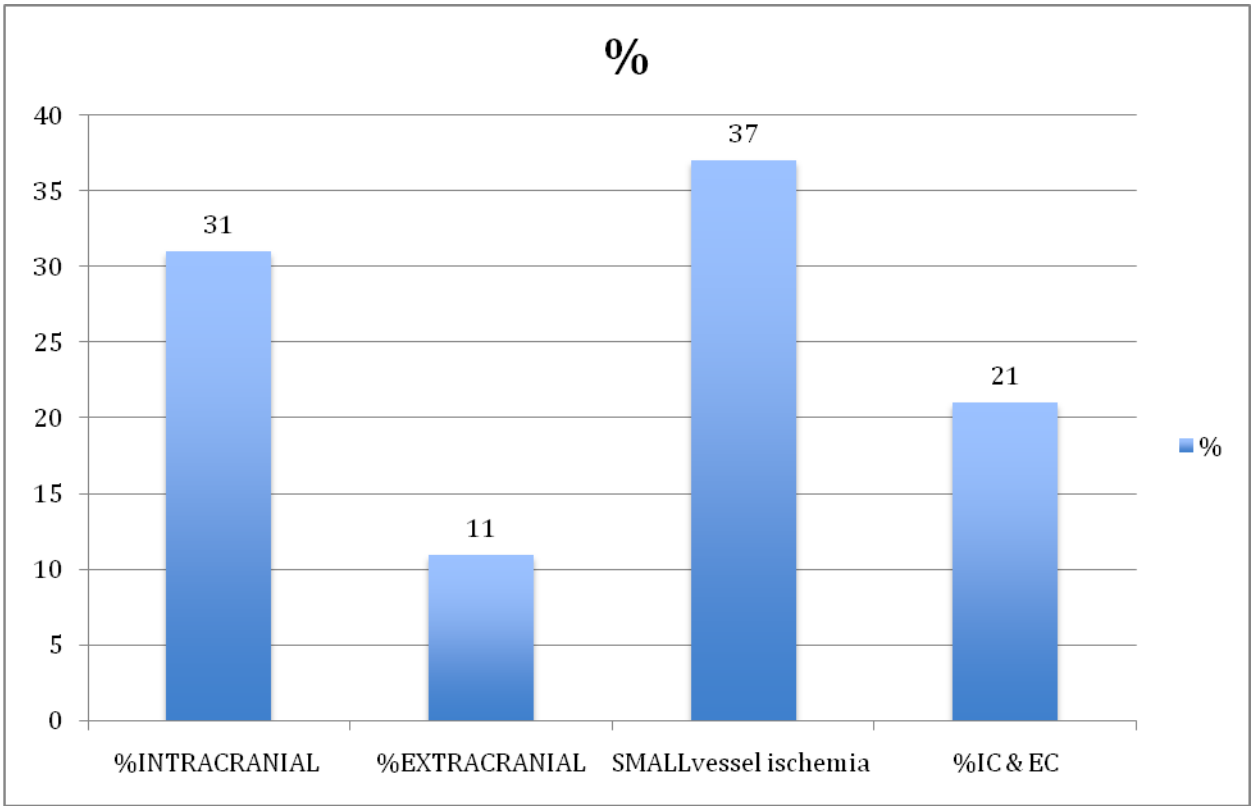
Among the age groups involved, most commonly patients were aged more than 50 years of age group constituting 77% of total patients in this study. Out of which 47 were males and 11 females. The mean age in this study population was 59 years. The young stroke population in this study was 5%.

Table 11:

Age Distribution

	SEX			
Age	**Male**	**Female**	**Total**	**(%)**
< 30	**0**	**0**	**0**	**0%**
31 – 40	**4**	**0**	**4**	**5%**
41 – 50	**11**	**2**	**13**	**18%**
>50	**47**	**11**	**58**	**77%**
Total	**62**	**13**	**75**	**100%**

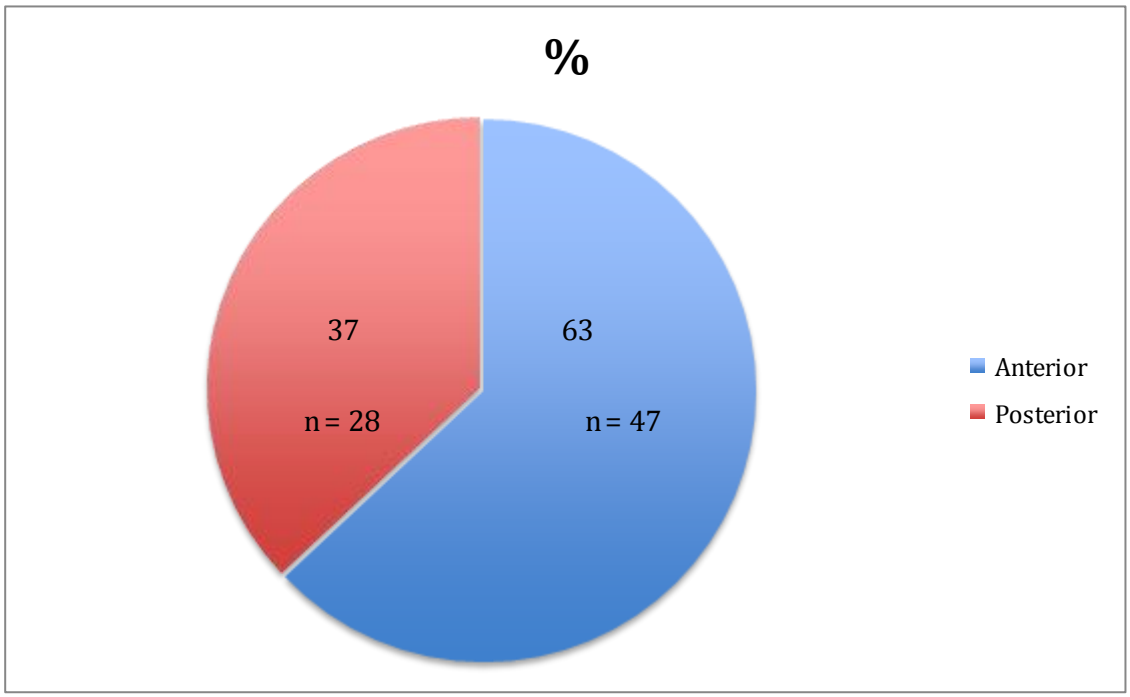
Figure17: Extracranial, intracranial or both



In this study of 75 patients,31% of the patients had intracranial obstruction due to atherothrombotic process which was classically seen in MRA - IC. Eleven percentage of the patients had only extracranial occlusion which was seen in CV doppler studies. Twenty one percentage of the study population had both intracranial and extracranial leisions and 37% had normal IC & EC studies, probably related to small vessel ischemia. Extracranial occulution that carotid artery disease constitutes 10-20% of the strokes, stroke risk is directly proportional to the degree of occlution. Carotid endarterectomy should be considered within 2 weeks if carotid stenosis of more than 70% involvement.There is less benefit of end carotidectomy and carotid

stenting in patients with stenosis of carotids 50 to 69% and in asymptomatic patients, and there is literally no benefit in patients with stenosis of less than 50%.

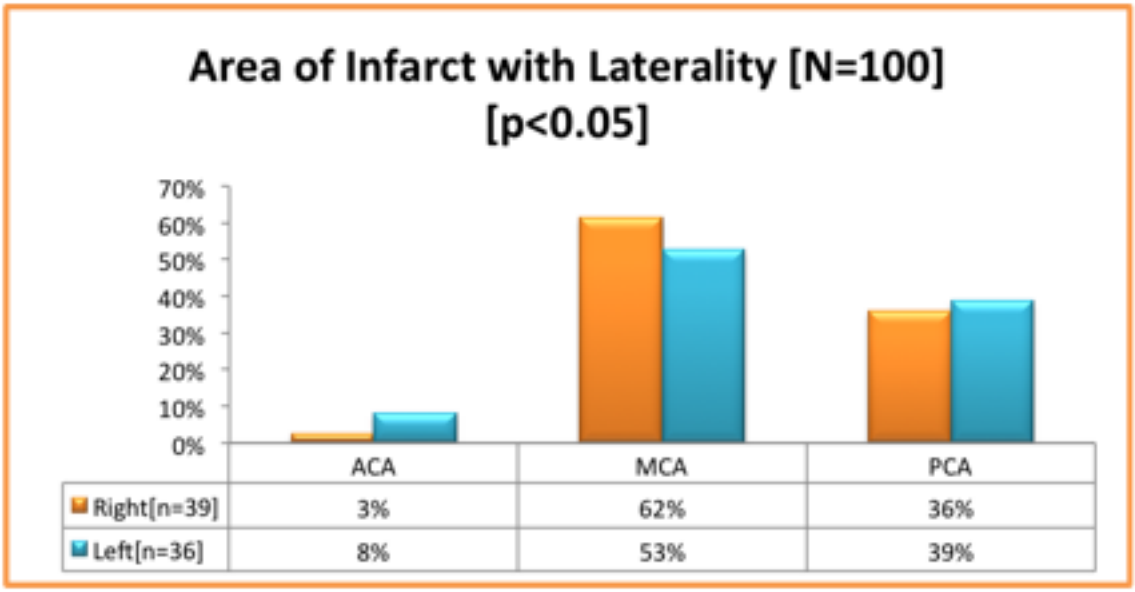
Figure 18: Anterior and posterior circulation involvement



In this study population,fourty seven out of seventy five patients had anterior circulation stroke and rest twenty eight patients had posterior circulation involvement. All patient was investigated with MRI –MRA IC and CV doppler studies. Thus anterior circulation was predominant than posterior circulation.

Table 12: Most commonly involved arteries

ASSOCIATION OF AREA OF INFRACT WITH LATERALITY			
	Laterality		
Area	**Right**	**Left**	**Total**
ACA	1	3	4
MCA	24	19	43
PCA	14	14	28
Total	**39**	**36**	**75**



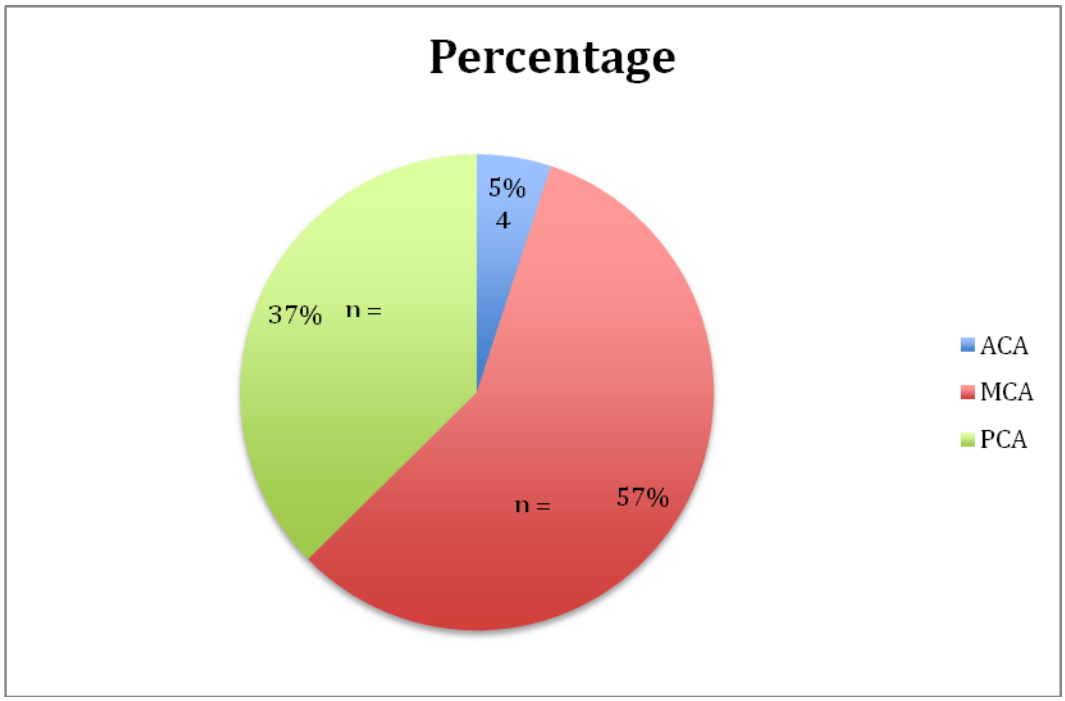
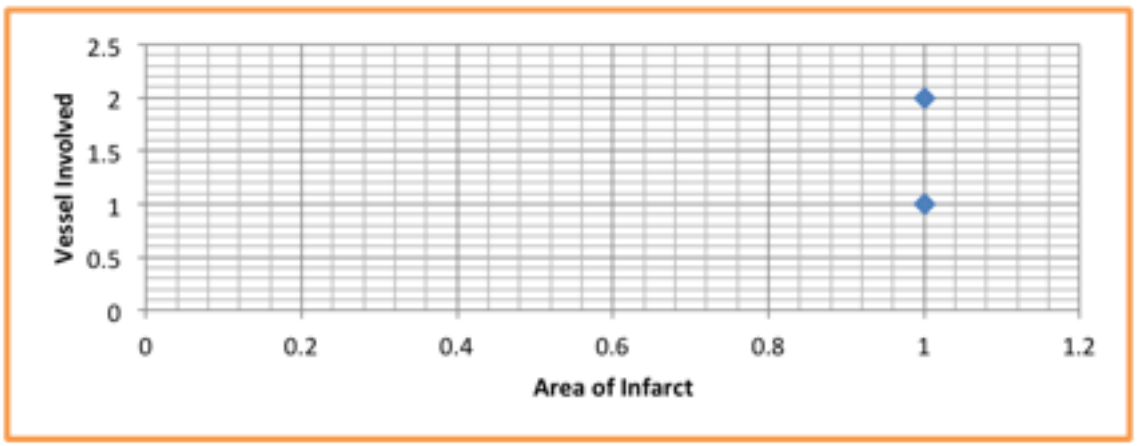


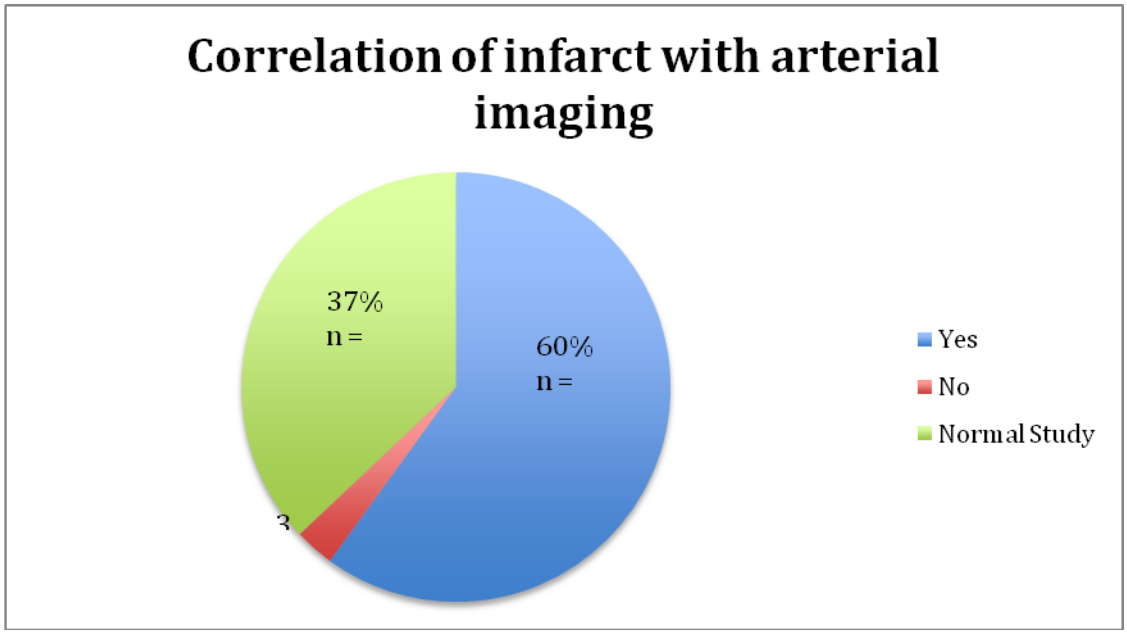
Figure 19: Most commonly involved arteries

Among the vascular supply to the brain, the most commonly involved arteries in this study population was MCA. Fourty three out of seventy five patients had MCA involvement. Among the 43 patient who had MCA involvement, 24 patients had involvement in the right side and 19 patients in the left side MCA.PCA was involved in twenty eight patients.ACA was involved in 4 patients, since it has a collateral blood supply.

Figure 20: Correlation of infarcted area in the brain and MRA + C V doppler

CORRELATION OF AREA OF INFARCT AND MRA + C V Doppler



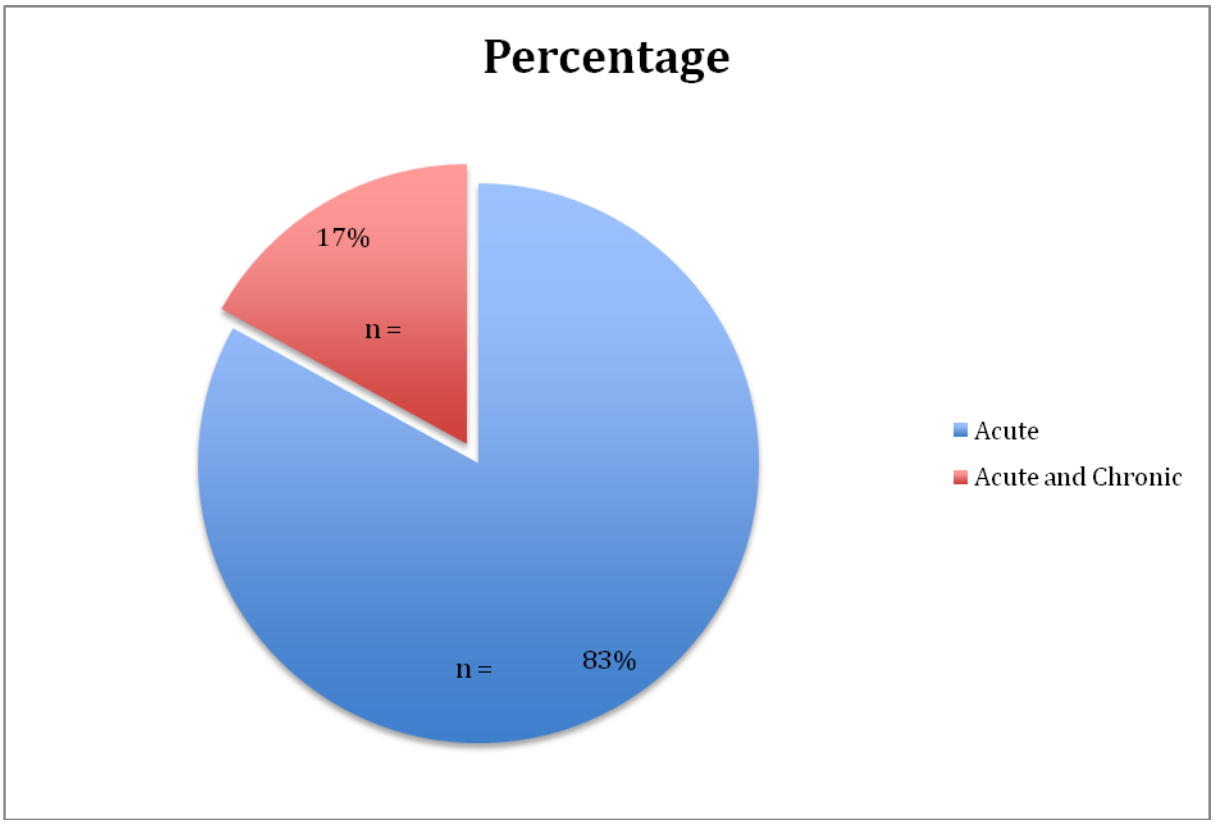


Wilcoxon Signed Ranks Test		
Frequencies		
		N
Vessel - Infarct	Negative Differences[a]	0
	Positive Differences[b]	30
	Ties[c]	45
	Total	75
a. Vessel < Infarct		
b. Vessel > Infarct		
c. Vessel = Infarct		

Test Statistics[b]	
	Vessel - Infarct
Z	-5.477[a]
Asymp. Sig. (2-tailed)	<0.001
a. Based on negative ranks.	
b. Wilcoxon Signed Ranks Test	

Fourty five out of seventy five patients comprising sixty percentage of the study population had positive correlation of location of the infarct in the brain and either MRA & C –V Doppler findings. Thirty seven percentage of the study population had normal MRA & C-V Doppler findings. Only in 3% of the study population there was no correlation of the same. The normal study of the vessels are included in the positive differences.

Figure 21: The acute, chronic or both natures of the brain infarcts



Among the study population eighty three percentage of the population had acute infarct in the brain and remaining seventeen percentage had both acute & chronic infarcts. Sixty two out of seventy five patients had acute infarcts and remaining thirteen patient had both acute and chronic infarcts in the brain.

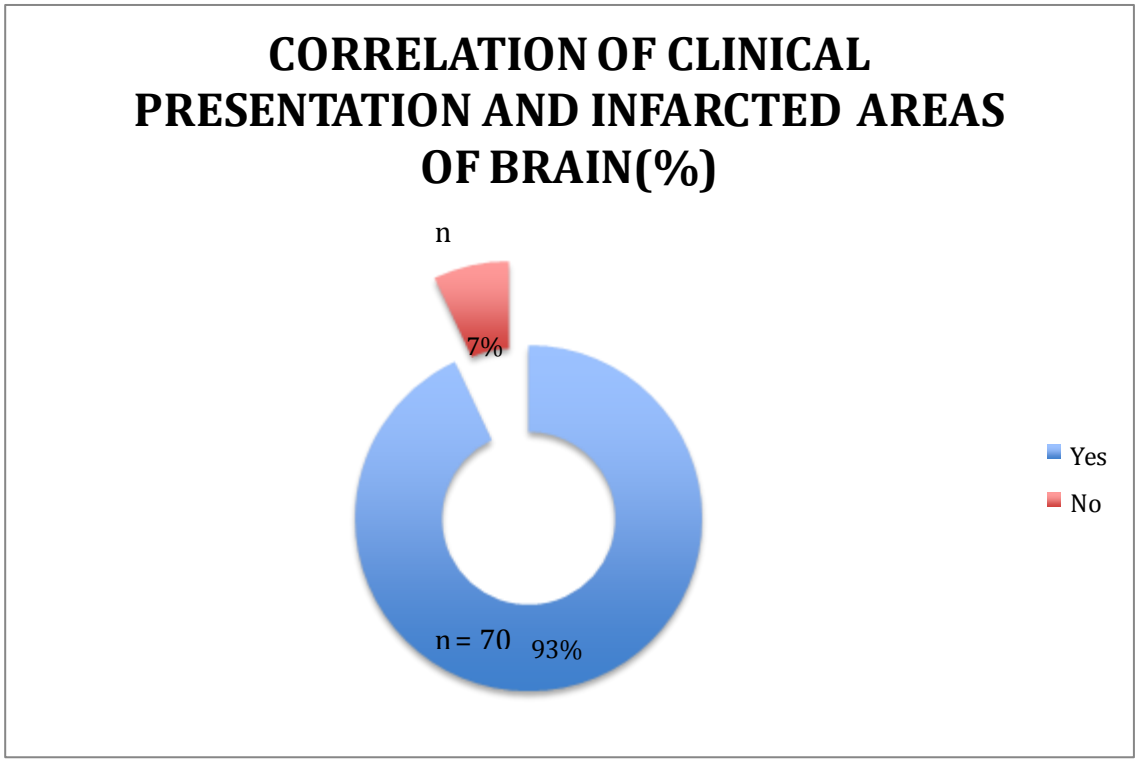
Table 13: Clinical Presentations

HEADACHE	**6**
SLURRING OF SPEECH	**33**
GAIT	**16**
GUIDINESS	**17**
DEVIATION OF MOUTH	**33**
WEAKNESS	**51**
VOMITING	**8**
URINARY INCONTINENCE	**1**
DECREASED SENSATION	**12**
TOTAL LOSS OF SPEECH	**4**
DOUBLE VISION	**5**
HICCUPS	**1**
BLURRING OF VISION	**1**
ALTERED SENSORIUM	**1**
SEIZURES	**3**
PIN PRICK SENSATION	**1**
PAIN	**1**
DYSPHAGIA	**1**
LOC	**2**
TINGLING SENSATION	**1**

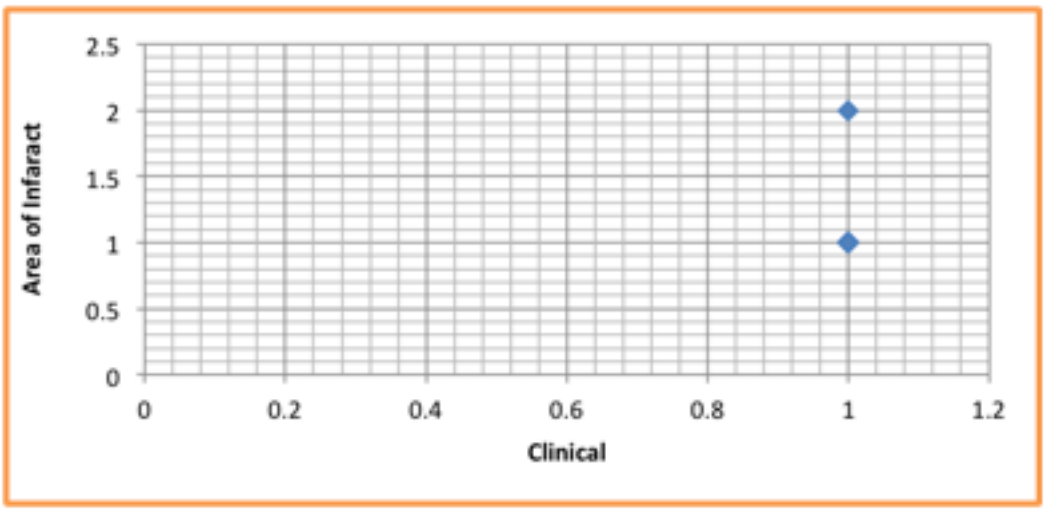
The most common symptoms was weakness, slurring of speech, deviation of angle of Mouth, gait disturbances, giddiness and decreased sensation. Other symptoms were headache, seizures, vomiting, headache, loss of consciousness, loss of speech, pain, dysphagia, double vision, blurring of vision, hiccups, altered sensorium, incontinence. Mainly to differentiate between anterior vs. posterior circulation with history, following symptoms are very specific for posterior circulation stroke if present.

The symptoms, which are specific for posterior circulation involvement, are vomiting, giddiness, gait disturbances, double vision. Presentations with seizures mostly had cortical involvement. The non specific symptoms which poorly indicates whether it is anterior or posterior involvement are sensory symptoms, weakness and slurring of speech.

Figure 22: Correlation of clinical presentations and infarcted areas



CORRELATION OF CLINICAL VS AREA OF INFARCT



Wilcoxon Signed Ranks Test

Frequencies

		N
Area - Clinical	Negative Differences[a]	0
	Positive Differences[b]	5
	Ties[c]	70
	Total	75

a. Area < Clinical

b. Area > Clinical

c. Area = Clinical

Test Statistics[b]

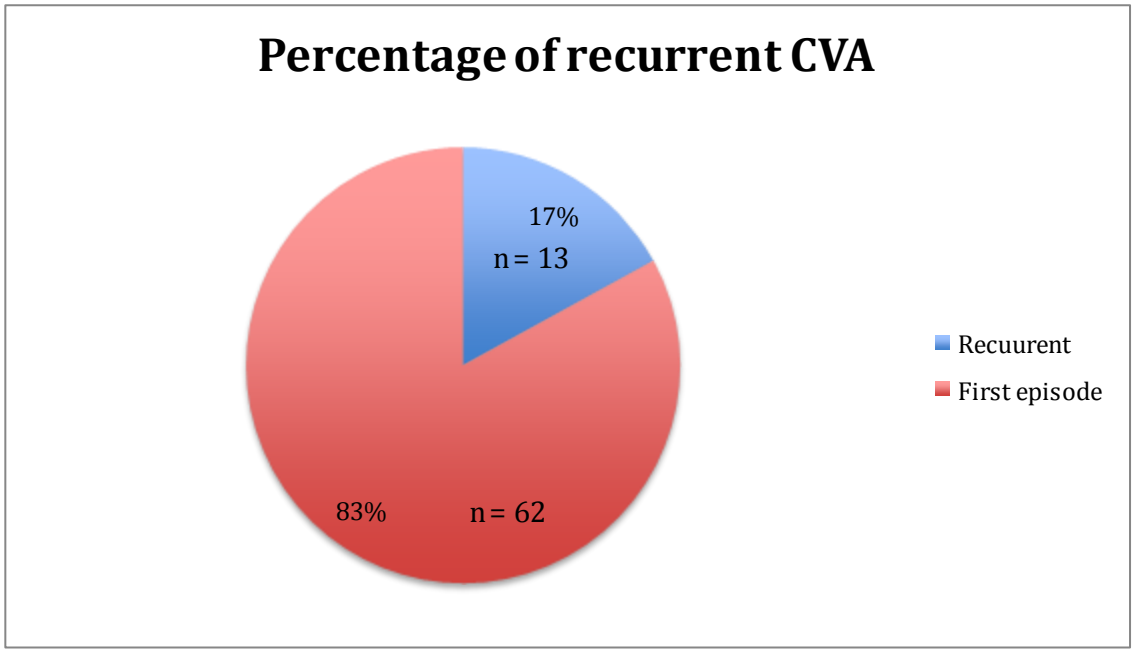
	Area - Clinical
Z	-2.236[a]
Asymp. Sig. (2-tailed)	<0.05

a. Based on negative ranks.

b. Wilcoxon Signed Ranks Test

The clinical presentations was correlating with infarct in MRI in seventy of seventy five patient in this study population which constitute 93 percentage. Only seven percentage of the study population there was no correlation due to bilateral involvement.

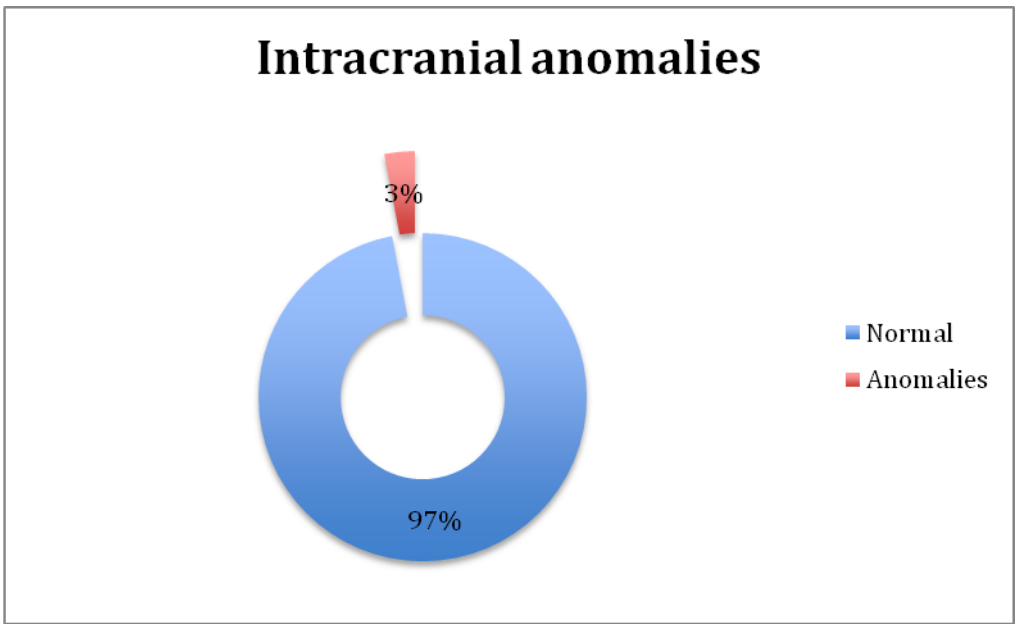
Figure 23: Percentages of recurrent CVA



In this study population, percentage of recurrent CVA was nearly 17%which accounted for thirteen out of seventy five patients. The cause for recurrent CVA was smoking, alcohol and poor compliance of medications. The mean age of recurrent CVA was 62 yrs and average NIHSS was 6.5 which was significantly higher than patients who had CVA for the first time.

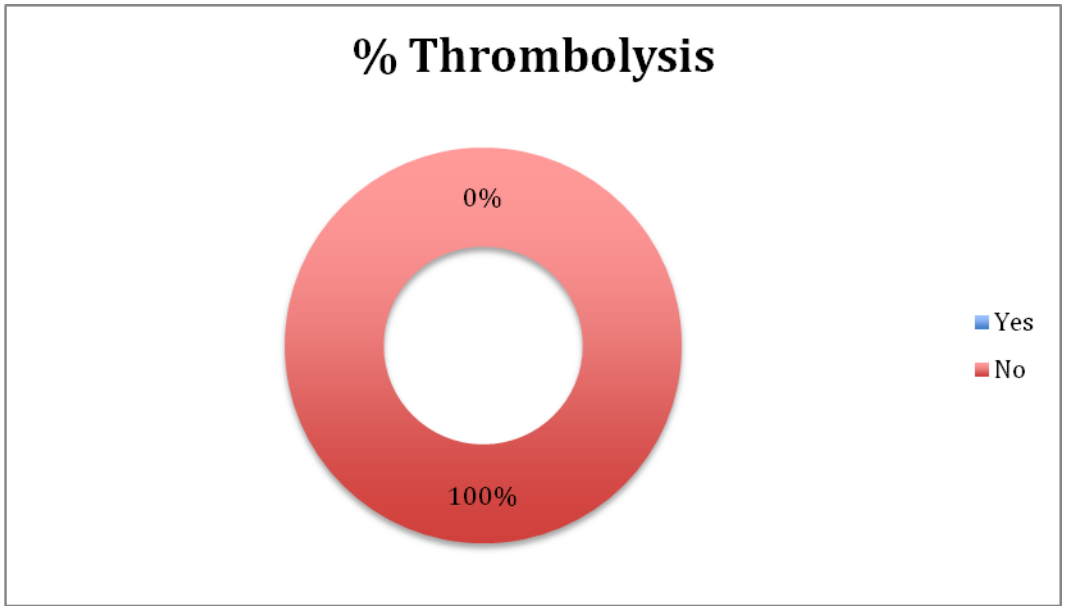
Relapse of alcohol, hard to kickoff smoking, poor adherence of medications was prime important in the stroke recurrence. Nicotine patches, chewing gums which had nicotine, psychiatric counseling and rehabilitation to prevent alcohol relapse.

Figure 24: Percentages of intracranial anomalies



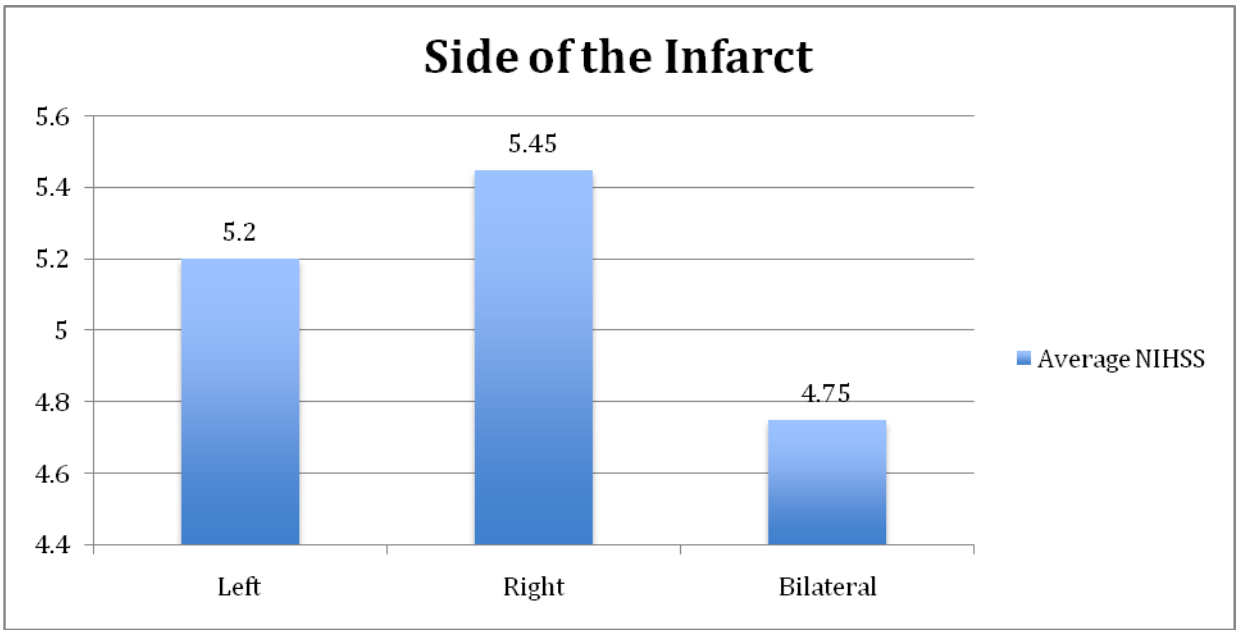
In the study population, 2 patients which constitutes of 3% had congenital anomalies. They were Frontal arteriovenous malformation and fetal PCA.

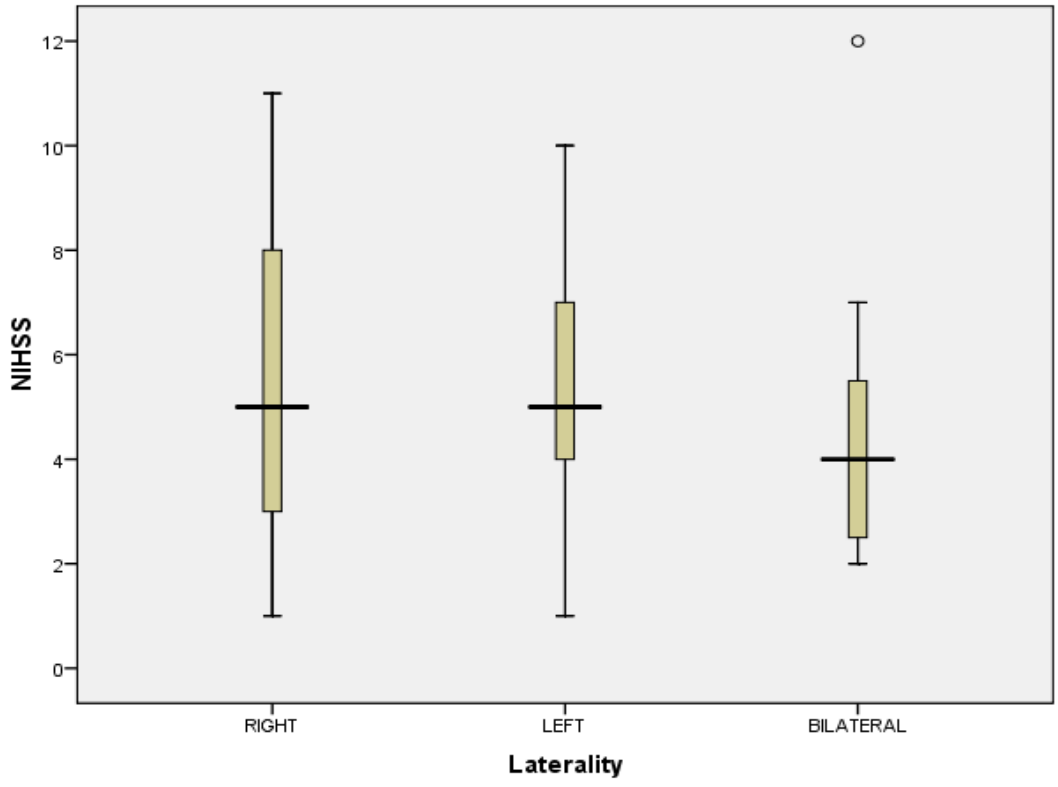
Figure 25: Percentage of thrombolysis in PSGIMSR



In this study poppulation none of the patients was thrombolysed.

Figure 26: NIHSS comparisons between right and the left sided brain involvement

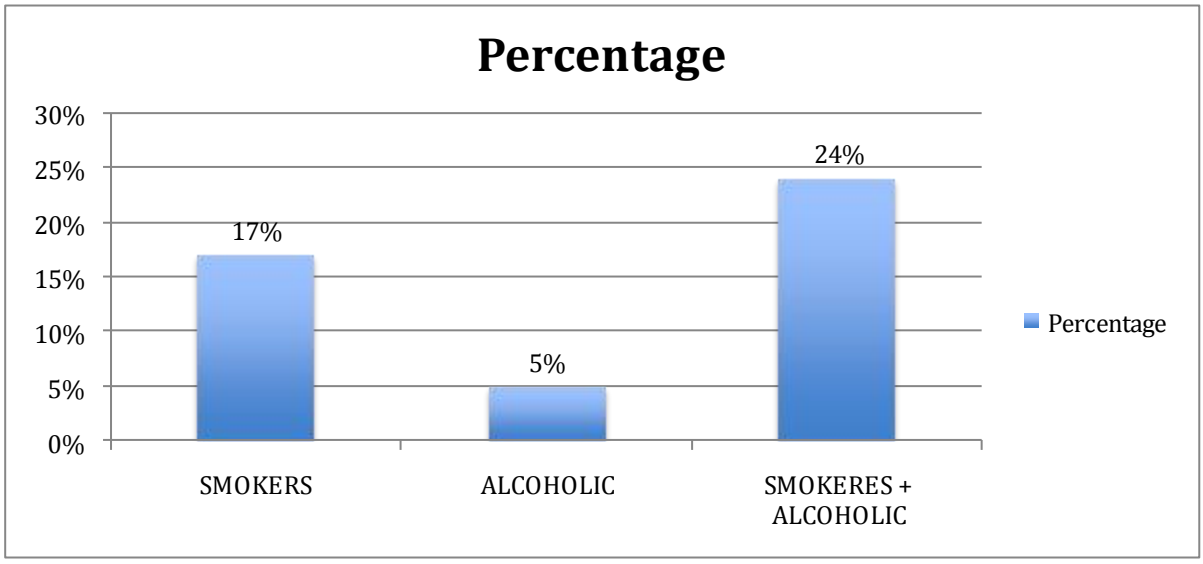




Mean NIHSS score with Laterality							
			95% CI for Mean				
	Mean	SD	Lower	Upper	Minimum	Maximum	Sig
Right	5.5	2.9	4.5	6.5	1	11	
Left	5.2	2.3	4.4	6.0	1	10	< 0.05
Bilateral	4.8	3.3	2.0	7.5	2	12	
Total	5.3	2.7	4.7	5.9	1	12	

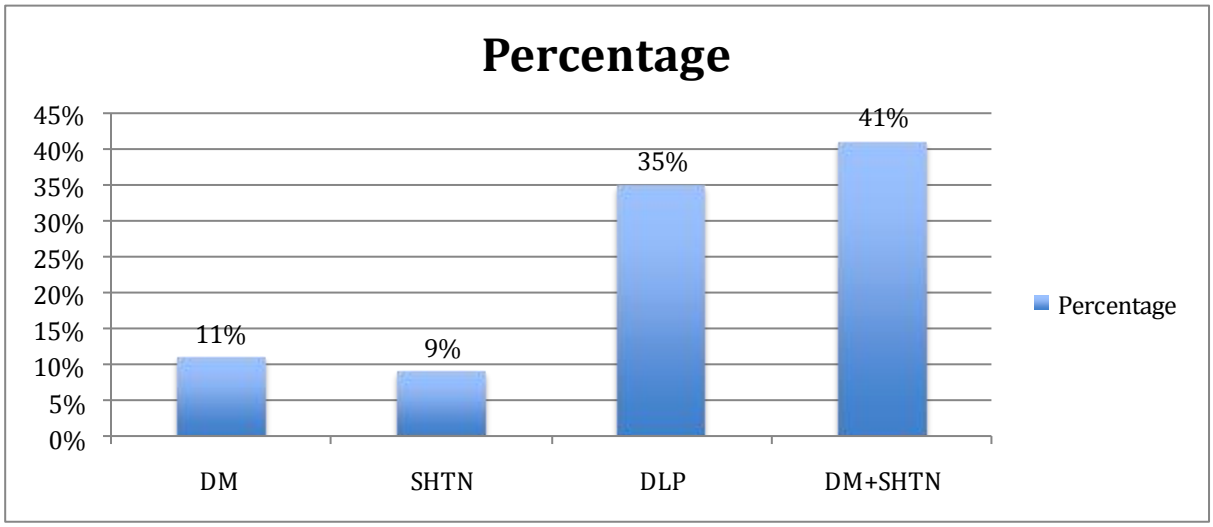
In this study population NIHSS was more in right sided brain involvement when compared with left and bilateral involvement. The average NIHSS for right, left, bilateral invelvement was 5.5, 5.2, 4.8 respectively. The maximum NIHSS in this study population was 12 which was in the bilateral involvement.

Figure 27: Percentage of smokers, alcoholics or both



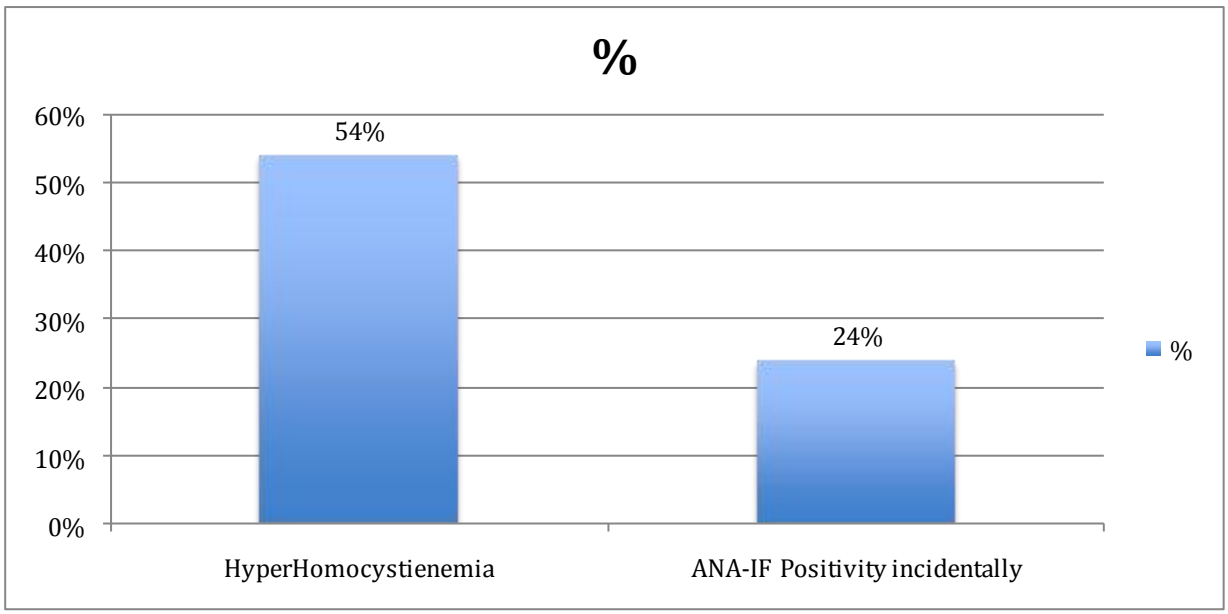
The percentage of smokers, alcoholics and both was 17%, 5% and 24% respectively. When put together totally 46% of the study population was abusing either alcohol, tobacco or Both.

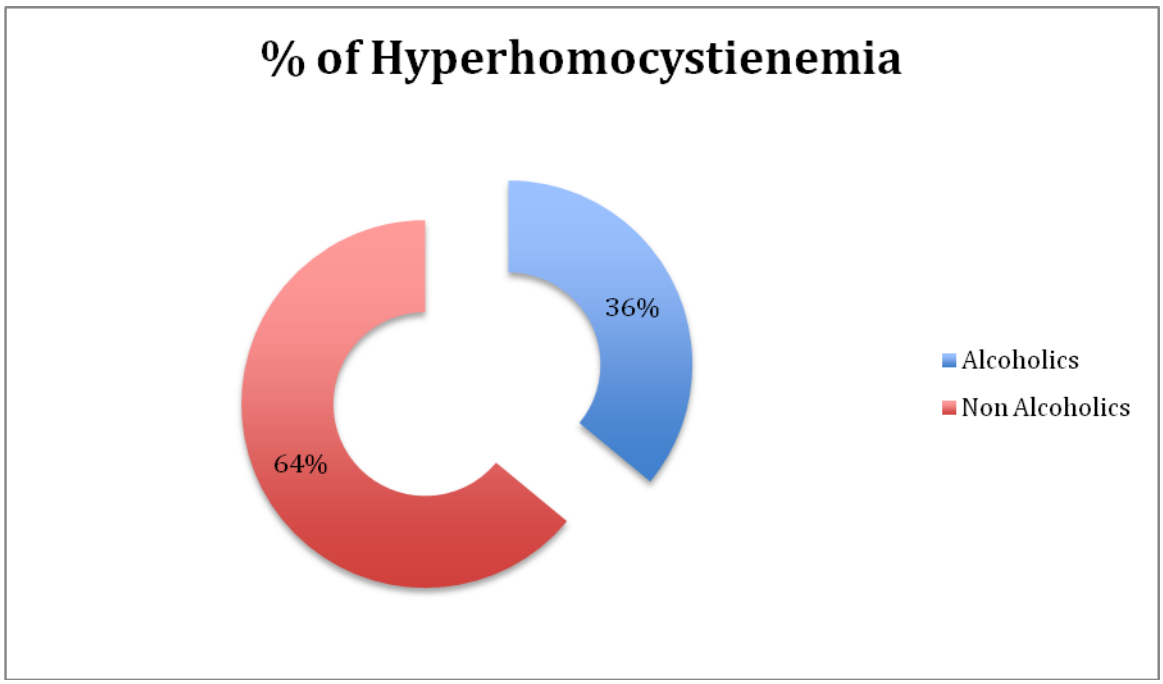
Figure 28: Percentages of DM, SHT, Dyslipidemia



In this study population the percentage of diabetic mellitus type 2, systemic hypertension, dyslipidemic, DM + systemic hypertension was 11%, 9%, 35% and 41% respectively. Totally 61% of the study population was either DM or hypertensive or both. The percentage of dyslipidemia was 35% for this study population.

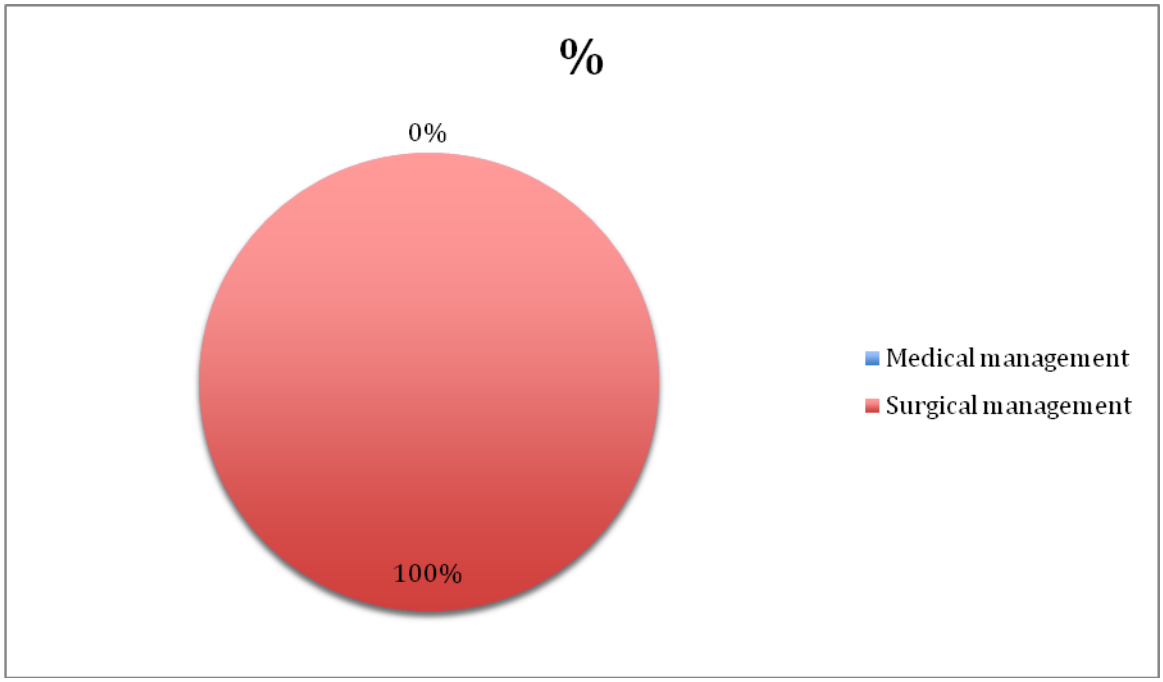
Figure 29 & 30: Incidental ANA-IF positivity and hyperhomocystenemias in PSGIMSR





Total of fourty six patients was studied with fasting serum homocystiene and ANA –IF. This blood sample was taken along with other investigations to prevent extra needle prick which would lead to inconvenience. Vitamins such as B12,B6 and folate play a vital role in the homocystiene methionine cycle, thus alcholics commonly have lower levels on folate and hence the increased homocystiene levels. Serum homocystiene was done in patients irrespective of any connective tissue disorder history. In this study population increased homocystiene and incidental ANA –IF positivity was 54% and 24% respectively. Serum homocystiene was elevated in 36% of the alcoholics and 64 % of non alcoholics. The highest value was 160 against <12 of the normal reference range. Serum homocystiene was elevated in acute stroke, there is correlation made in few studies that higher the homocystiene calues higher the mortality and recurrence of stroke. Incidental ANA- IF positivity in 24% of the study population raises concern whether it should be done routinely in all stroke patients and the same should be repeated to confirm the autoimmune etiology 6 weeks later.

Figure 31: Decision regarding medical or surgical treatment based on the arterial studies



Prevention of strokes :

Stroke prevention have to be endorsed at all levels primary, secondary and tertiary levels. Primary prevention is by banning of tobacco and alcohol and stop abusing the same.

Secondary prevention is by controlling hypertension, DM, lipids and taking antiplatelets dietary modification, exercise and statin therapy. Tertiary prevention is prevention of diabilty of stroke. Identifying the etiology play an important role in the prevention of strokes, especially the cardioembolic strokes. The use of oral anticoagulants play a important role in prevention of CVA.

DISCUSSION

Stroke among adults rapidly increasing in the recent times. Proper clinical history, risk factors, investigations, proper management of stroke is the need of the hour for AIS patients.In this study, 75 patients of acute ischemic stroke:

- Male population was more when compared with females.
- The mean age was 59 years
- Predominantly anterior circulation was involved
- Right MCA was the most common artery involved
- Positive correlation for clinical presentation and area of infarct
- Positive corelation for areas of infarct and the vessels involved
- Most common clinical presentation was weakness and slurring of speech
- 3% intracranial anamolies
- Smoking, alcoholism and dyslipidemia have been found to be significantly associated with stroke and its recurrence.
- Poor control of DM, systemic hypertension was associated with sroke recurrence
- Hyper-homocystinemia has significant association with the causation of stroke. 56% (only in 46 patients) had significantly elevated levels of homocystiene. The levels ideally have to be assessed 6 weeks later, for ideally diagnosing hyperhomocystinemia.

- The proportion of elevated levels of homocystiene was more in the non alcoholics when compared with alcoholics.
- 24% had incidental ANA- IF positivity (out of 46 patientts) but none of them had history of connective tissue disease. This incidental finding have to rechecked 6 weeks later.
- Recurrence of stroke though was seen in 17%, but no mortality and less morbidity has been observed among these patients. NIHSS was comparatively higher than patients who had stroke for the first time.
- None of the pateints was thrombolysed due to varied reasons like exclusion criteria, after explaining the risks involved and mainly the time lapse in reaching the hospital.
- Right sided infarct had higher NIHSS
- None of the patients was treated surgically.

CONCLUSION

I thank PSGIMSR for the grant of one lakh Rupees for the study of ANA- IF and serum Homocystiene in AIS patients. Good clinical examination plays a important role in localising the leision despite many caveats.

- Positive correlation for clinical presentation and area of infarct
- Positive corelation for areas of infarct and the vessels involved
- Elevated serum homocystiene and Incidental positivity of ANA-IF have to reassessed 6 weeks later.
- Awareness regarding golden time have to made available among the masses
- It is team work rather one person job required in the management of AIS patients.
- Prevention at every levels is important, Prevention is always better than cure.

BIBLIOGRAPHY

1. Strong K, Mathers C, Bonita R. Preventing stroke: saves lives around the world. Lancet Neurol 2007; 6: 182-7.
2. PM Dalal, Madhumita Bhattacharjee. Stroke Epidemic in India: Hypertension-Stroke Control Programme is Urgently Needed. JAPI. Vol 55. October 2007.
3. Dalal P, Bhattacharjee M, and Vairale J, Bhat P. UN millennium development goals: can we halt the stroke epidemic in India? Ann Indian Acad Neurol 2007; 10: 130-6.
4. Marc Fisher MD, Bo Norrving. 1st Global Conference on Healthy. Lifestyles and Non communicable diseases Control. Moscow, April 28-29, 2011.
5. Steinwachs DM, Collins-Nakai RL, Cohn LH, Garson A Jr, Wolk MJ.J The future of cardiology: utilization23 and costs of care. Am Coll Cardiol. 2000 Apr; 35(5 Suppl B):91B-98B.
6. Banerjee T, Das S. Epidemiology of stroke in India. Neurology Asia 2006; 11: 1-4
7. Sridharan S et al. Incidence, types, risk factors and outcome of stroke in a developing country: the Trivandrum Stroke Registry. Stroke 2009; 40: 1212-18.
8. Mackay J, Mensah G, Mendis S, Greenlund K. Atlas of heart disease and stroke. WHO 2004.
9. Feigin V, Lawes C, Bennet D, Barker Cello S, Parag V. Worldwide stroke incidence and early case fatality in 56 population based studies: a systematic review. Lancet Neurology 2009; 8 (4): 355- 369.

10. Baridalyne Nongkynrih, BK Patro, Chandrakanta S Pandav. Current Status of Communicable and Non- communicable Diseases in India. JAPI vol 52 Feb 2004.
11. Mackay J, Mensah G, Mendis S, Greenlund K. Atlas of heart disease and stroke. WHO 2004.
12. Murthy J. Thrombolysis for stroke in India: Miles to go..... Neurology India 2007; 55 (1) 3-5.
13. Prabhakaran D, Ajay V. Non-communicable Disease in India: A perspective. Centre for Chronic Disease Control discussion report for the WHO 2009, New Delhi, India.
14. Shah B, Mathur P. Workshop Report on Stroke Surveillance in India. Division of Non communicable Diseases, Indian Council of Medical Research, New Delhi 2006.
15. Posterior Versus Anterior Circulation Infarction How Different Are the Neurological Deficits? Wen-Dan Tao, MD; Ming Liu, MD, PhD; Marc Fisher, MD; De-Ren Wang, MD; Jie Li, MD; Karen L Furie, MD, MPH; Zi-Long Hao, MD; Sen Lin, MD; Can-Fei Zhang; Quan-Tao Zeng; Bo Wu, MD, PhD.
16. Baseline NIH Stroke Scale Score predicting outcome in anterior and posterior circulation strokesS. Sato, MD, K. Toyoda, MD, T. Uehara, MD, N. Toratani, MD, C. Yokota, MD, H. Moriwaki, MD, H. Naritomi, MD and K. Minematsu, MD.
17. Early signs of brain infarction at CT: observer reliability and outcome after thrombolytic treatment--systematic review. Wardlaw JM1, Mielke O

18. Loss of the insular ribbon: another early CT sign of acute middle cerebral artery infarction Truwit CL, Barkovich AJ, Gean-Marton A, Hibri N, Norman D.
19. Sensitivity and prognostic value of early CT in occlusion of the middle cerebral artery trunk von Kummer R, Meyding-LamadéU, Forsting M, Rosin L, Rieke K, Hacke W, Sartor K.
20. Evaluation of early computed tomographic findings in acute ischemic stroke Marks MP, Holmgren EB, Fox AJ, Patel S, von Kummer R, Froehlich J.
21. Middle cerebral artery territory infarction and early brain swelling: progression and effect of age on outcome Wijdicks EF, Diringer MN.
22. Allen LM, Hasso AN, Handwerker J et-al. Sequence-specific MR Imaging Findings That Are Useful in Dating Ischemic Stroke. Radiographics. 2012;32 (5): 1285-97.
23. Pressman BD, Tourje EJ, Thompson JR. An early CT sign of ischemic infarction: increased density in a cerebral artery. AJR Am J Roentgenol. 1987;149 (3): 583-6
24. Srinivasan A, Goyal M, Al azri F et-al. State-of-the-art imaging of acute stroke. Radiographics. 2006;26 Suppl 1 : S75-95
25. Lyden P, Lu M, Jackson C, Marler J, Kothari R, Brott T, et al. Underlying structure of the National Institutes of Health Stroke Scale: results of a factor analysis. NINDS tPA Stroke Trial Investigators. Stroke. 1999;30(11):2347–54
26. Rouhl RPW, van Oostenbrugge RJ, Knottnerus ILH, Staals JEA, Lodder J. Virchow-Robin spaces relate to cerebral small vessel disease severity. J Neurol. 2008;255(5):692–6

27. Finger S, Stone JL. Chapter 14 Landmarks of surgical neurology and the interplay of disciplines. Handbook of Clinical Neurology. 2009. p. 189–202.
28. Zeman A. Neurology is psychiatry--and vice versa. Pract Neurol [Internet]. 2014;14(3):136–44
29. Kokotailo RA, Hill MD. Coding of stroke and stroke risk factors using international classification of diseases, revisions 9 and 10. Stroke. 2005;36(8):1776–81
30. Jamieson DG. Diagnosis of Ischemic Stroke. Am J Med. 2009;122(4 SUPPL. 2).
31. Smith SD, Eskey CJ. Hemorrhagic stroke. Radiologic Clinics of North America. 2011. p. 27–45.
32. Uchiyama S. New Definition and Concept of TIA. Rinsho Shinkeigaku. 2010;50(11):904–6.
33. Brown RD, Whisnant JP, Sicks JD, O'Fallon WM, Wiebers DO. Stroke incidence, prevalence, and survival: Secular trends in Rochester, Minnesota, through 1989. Stroke. 1996;27:373–80
34. Rothwell PM, Coull AJ, Giles MF, Howard SC, Silver LE, Bull LM, et al. Change in stroke incidence, mortality, case-fatality, severity, and risk factors in Oxfordshire, UK from 1981 to 2004 (Oxford Vascular Study) Lancet. 2004;363:1925–33
35. Kolominsky-Rabas PL, Sarti C, Heuschmann PU, Graf C, Siemonsen S, Neundoerfer B, et al. A prospective community-based study of stroke in Germany — The Erlanger Stroke Project (ESPro): Incidence and case fatality at 1, 3, and 12 months. Stroke. 1998;29:2501–6

36. Syme PD, Byrne AW, Chen R, Devenny R, Forbes JF. Community-based study of stroke incidence in a Scottish population: The Scottish Borders Stroke Study. Stroke. 2005;36:1837–43
37. Tsiskaridze A, Djibuti M, van Melle G, Lomidze G, Apridonidze S, Gaurashvili I, et al. Stroke incidence and 30-day case fatality in a suburb of Tbilisi: Result of the first prospective population-based study in Georgia. Stroke. 2004;35:2523–8
38. Kaul S, Sunitha P, Suvarna A, Meena AK, Uma M, Reddy JM. Subtypes of ischemic stroke in a metropolitan city of south India (one year data from hospital based stroke registry) Neurol India. 2002;50:S8–14.
39. Dalal PM. Burden of stroke: Indian perspective. Int J Stroke. 2006;1:164–6
40. Wasay M, Khatri IA, Kaul S. Stroke in South Asian countries. Nat Rev Neurol. 2014;10:135–43
41. Dash D, Bhashin A, Pandit AK, Tripathi M, Bhatia R, Prasad K, et al. Risk factors and etiologies of ischemic strokes in young patients: A tertiary hospital study in north India. J Stroke. 2014;16:173–7
42. Bansal BC, Gupta RR, Prakash C. Stroke during pregnancy and puerperium in young females below the age of 40 years as a result of cerebral venous sinus thrombosis. Jpn Heart J. 1980;21:171–83
43. Gosalakkat JA. Moyamoya disease: A review. Neurol India. 2002;50:6–10
44. Sharma BK, Sagar S, Sing AP, Suri S. Takayashu arteritis in India. Heart Vessels Suppl. 1992;7:37–43

45. Kalita J, Goyal G, Kumar P, Misra UK. Intracerebral hemorrhage in young from a tertiary neurological centre in North India. J Neurol Sci. 2014;336:42–7. [PubMed]
46. Sethi PK. Stroke - Incidence in India and Management of Ischaemic stroke. Neurosci Today [Internet]. 2002;6(3):139–43. Available from: http://openmed.nic.in/1753/01/SGRH_p202-206_EDIT.pdf
47. Das S, Hazra A, Ray BK, Ghosal M, Banerjee TK, Roy T, et al. Burden among stroke caregivers: Results of a community-based study from Kolkata, India. Stroke. 2010;41(12):2965–8.
48. Thom DH, Grayston JT, Siscovick DS, Wang SP, Weiss NS, Daling JR. Association of prior infection with Chlamydia pneumoniae and angiographically demonstrated coronary artery disease. JAMA. 1992;268:68–72. [PubMed]
49. Melnick SL, Shahar E, Folsom AR, Grayston JT, Sorlie PD, Wang SP, et al. Past infection by Chlamydia pneumoniaestrain TWAR and asymptomatic carotid atherosclerosis. Atherosclerosis Risk in Communities (ARIC) Study Investigators. Am J Med. 1993;95:499–504
50. Stroke syndromes and clinical managementJ.S. Balami, R.L. Chen, A.M. B
51. Clinical Presentation and Diagnosis of Cerebrovascular Disease Mark J. Alberts
52. Jauch EC, Saver JL, Adams HP Jr, et al. Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 2013; 44:870.

53. Weir CJ, Murray GD, Dyker AG, Lees KR. Is hyperglycaemia an independent predictor of poor outcome after acute stroke? Results of a long-term follow up study. BMJ 1997; 314:1303.
54. Bruno A, Biller J, Adams HP Jr, et al. Acute blood glucose level and outcome from ischemic stroke. Trial of ORG 10172 in Acute Stroke Treatment (TOAST) Investigators. Neurology 1999; 52:280.
55. Capes SE, Hunt D, Malmberg K, et al. Stress hyperglycemia and prognosis of stroke in nondiabetic and diabetic patients: a systematic overview. Stroke 2001; 32:2426.
56. Dave JA, Engel ME, Freercks R, et al. Abnormal glucose metabolism in non-diabetic patients presenting with an acute stroke: prospective study and systematic review. QJM 2010; 103:495.
57. Béjot Y, Aboa-Eboulé C, Hervieu M, et al. The deleterious effect of admission hyperglycemia on survival and functional outcome in patients with intracerebral hemorrhage. Stroke 2012; 43:243.
58. Toole JF. Effects of change of head, limb and body position on cephalic circulation. N Engl J Med 1968; 279:307.
59. Caplan LR, Sergay S. Positional cerebral ischaemia. J Neurol Neurosurg Psychiatry 1976; 39:385.
60. Jauch EC, Saver JL, Adams HP Jr, et al. Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the Am

61. Lee M, Ovbiagele B, Hong KS, et al. Effect of Blood Pressure Lowering in Early Ischemic Stroke: Meta-Analysis. Stroke 2015; 46:1883.
62. Chollet F, Tardy J, Albucher JF, et al. Fluoxetine for motor recovery after acute ischaemic stroke (FLAME): a randomised placebo-controlled trial. Lancet Neurol 2011; 10:123.
63. Mead GE, Hsieh CF, Hackett M. Selective serotonin reuptake inhibitors for stroke recovery. JAMA 2013; 310:1066.
64. Chong JY, Mohr JP. Anticoagulation and platelet antiaggregation therapy in stroke prevention. Curr Opin Neurol. 2005;18(1):53–7.
65. Meldrum BS. Cytoprotective therapies in stroke. Curr Opin Neurol. 1995;8(1):15–23.
66. Gresham GE, Duncan PW, Stason WB, et al. Post-stroke rehabilita- tion. Clinical practice guideline, no. 16. Rockville, Md.: Agency for Health Care Policy and Research, May 1995. (AHCPR publication no. 95-0662.)
67. Gresham GE. Rehabilitation of the stroke survivor. In: Barnett HJM, Mohr JP, Stein BM, Yatsu FM, eds. Stroke: pathophysiology, diagnosis, and management. 3rd. ed. Philadelphia: Churchill Livingstone, 1998:1389- 401.
68. Pohjasvaara T, Erkinjuntti T, Ylikoski R, Hietanen M, Vataja R, Kaste M. Clinical determinants of poststroke dementia. S
69. The Multicenter Acute Stroke Trial — Europe Study Group. Throm- bolytic therapy with streptokinase in acute ischemic stroke. N Engl J Med 1996;335:145-50.

70. Multicentre Acute Stroke Trial — Italy (MAST-I) Group. Randomised controlled trial of streptokinase, aspirin, and combination of both in treat- ment of acute ischaemic stroke. Lancet 1995;346:1509-14.
71. Donnan GA, Davis SM, Chambers BR, et al. Streptokinase for acute ischemic stroke with relationship to time of administration: Australian Streptokinase (ASK) Trial Study Group. JAMA 1996;276:961-6.
72. Tanswell P, Modi N, Combs D, Danays T. Pharmacokinetics and pharmacodynamics of tenecteplase in fibrinolytic therapy of acute myocardial infarction. Clin Pharmacokinet 2002;41:1229-45.
73. Albers GW, Bates VE, Clark WM, Bell R, Verro P, Hamilton SA. Intravenous tissue-type plasminogen activator for treat- ment of acute stroke: the Standard Treatment with Alteplase to Reverse Stroke (STARS) study. JAMA 2000;283:1145-50.
74. Hill MD, Buchan AM. Thrombolysis for acute ischemic stroke: results of the Canadian Alteplase for Stroke Effective- ness Study. CMAJ 2005;172:1307-12.
75. Wahlgren N, Ahmed N, Dávalos A, et al. Thrombolysis with alteplase for acute ischaemic stroke in the Safe Implementa- tion of Thrombolysis in Stroke-Monitor- ing Study (SITS-MOST): an observational study. Lancet 2007;369:275-82. [Erratum, Lancet 2007;369:826.]
76. The National Institute of Neurologi- cal Disorders and Stroke rt-PA Stroke Study Group. Tissue plasminogen activa- tor for acute ischemic stroke. N Engl J Med 1995;333:1581-8.
77. Broderick J, Connolly S, Feldmann E, et al. Guidelines for the management of spontaneous intracerebral hemorrhage in adults: 2007 update: a guideline from the

American Heart Association/American Stroke Association Stroke Council, High Blood Pressure Research Council, and the Quality of Care and Outcomes in Research Interdisciplinary Working Group. Stroke 2007;38:2001-23.

78. Rasler F. Emergency treatment of hemorrhagic complications of thrombol- ysis. Ann Emerg Med 2007;50:485.
79. Goldstein JN, Marrero M, Masrur S, et al. Management of thrombolysis-associated symptomatic intracerebral hemor- rhage. Arch Neurol 2010;67:965-9.
80. Zunt JR. ADAMS AND VICTOR'S PRINCIPLES OF NEUROLOGY. Neurology. 2010. p. 1400–1400
81. Peacock J. Harrison's Neurology in Clinical Medicine. First Edition. Yale J Biol Med. 2006;79(1):39–40.
82. Stevens R. Gray's Anatomy for Students. Ann R Coll Surg Engl. 2006;88(5):513–4.
83. Posterior Versus Anterior Circulation Infarction How Different Are the Neurological Deficits? Wen-Dan Tao, MD; Ming Liu, MD, PhD; Marc Fisher, MD; De-Ren Wang, MD; Jie Li, MD; Karen L Furie, MD, MPH; Zi-Long Hao, MD; Sen Lin, MD; Can-Fei Zhang; Quan-Tao Zeng; Bo Wu, MD, PhD

PROFORMA

- Name
- Age
- Sex
- Address
- Occupation
- Chief complaints
- H/o cocaine abuse or any other substance abuse or use of oral contraceptives
- H/o smoking, alcohol, diabetes, hypertension, seizure disorder, bleeding disorders, migraine, thrombosis, rheumatic heart disease, ischaemic heart disease, severe LV dysfunction, arrhythmias (mainly atrial fibrillation)
- H/o head trauma
- Family history of thrombosis, hyperlipidaemia, hypertension, diabetes

CLINICAL EXAMINATION

- General examination
- CNS examination:
  - Speech – dysarthria or aphasia (motor / sensory/ global)
  - Cranial nerves
  - Weakness of limb – 1 UL or 1 UL and 1 LL or 4 limbs
  - Power – UL and LL
  - Sensory
  - Cerebellar

- Carotid and subclavian bruits, murmurs
- Peripheral pulses
- NIHSS on admission
- mRS on admission and after 3 months

- Routine investigations:

- Complete blood count, Diabetic profile
- Serum electrolytes, urine routine and microscopy
- ECG, ECHO, serum homocysteine levels
- CT- brain plain, MRI brain with angiography

- Other investigations:
- ANA IF and Antiphospholipid antibodies

- If needed:
- Complete coagulation work up
- Genetic testing

ABBREVIATIONS

AIS – Arterial ischemic stroke

WHO - World Health Organization

TOAST - Trial of Org 10172 in Acute Stroke Treatment (TOAST) criteria.

ANA – Anti nuclear antibody profile

APLA – Anti-phospholipid antibody

CT – Computed tomography

MRI – Magnetic resonance imaging

NIHSS - National Institute of Health stroke scale

mRS – modified rankin scoring

TIA - Transient ischemic attacks

DALY - disability life adjusted years

LDL-C - low-density lipoprotein cholesterol

TGL - Triglycerides

HDL-C - High-density lipoprotein cholesterol (HDL-C)

BMI – Body mass index

SBP – Systolic blood pressure

DBP – Diastolic blood pressure

FBS - fasting blood glucose

HbA1c - glycosylated haemoglobin

DM – Diabetes mellitus

MA – Migraine headache with aura

ECMO – Extracorporeal membrane oxygenation

RHD - Rheumatic heart disease

DCM - Dilated cardiomyopathy

AMI – Acute myocardial infarction

IE – Infective endocarditis

AF – Atrial Fibrillation

CAD – Coronary artery disease

LVH – Left ventricular hypertrophy

LAE – Left atrial enlargement

CBS – Cystathione b-synthase

PV – Polycthemia vera

ET - Essential thrombocythemia

PAN – Polyarteritis nodosa

WG – Wegener's granulomatosis

CNS - Central nervous system

CADASIL – Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy

CARASIL – Cerebral autosomal recessive arteriopathy with subcortical infarcts and leukoencephalopathy

MELAs – Mitochondrial myopathy, encephalopathy, lacto-acidosis and stroke

EDS – Ehlers Danlos syndromes

OI – Osteogenenis imperfect

ADPKD - Autosomal dominant polycystic kidney disease (ADPKD)

LDS - Loeyz Dietz syndrome

AD – Autosomal dominant

COW – Circle of willis

ACA – Anterior cerebral artery

MCA – Middle cerebral artery

PCA – Posterior cerebral artery

UMN – Upper motor neuron type

ED – emergency department

HIV – human immunodeficiency virus

ECG – Electrocardiogram

ECHO – Echocardiography

AHA/ASA – American heart association / American stroke association

r-tPA – Recombinant tissue plasminogen activator

NINDS – National institute of Neurological Disorders and Stroke

aPTT – Activated partial thromboplastin time

INR – International normalised ratio

PT – Prothrombin time

UFH – Unfractionated heparin

LMWH – Low molecular weight heparin

DWI - Diffusion weighted image

PWI - Perfusion weighted image

PCI - Posterior circulation infarct

ACI - Anterior circulation infarct

TCD - Transcranial doppler

ADC - Apparent diffusion coefficient

FLAIR - Fluid-attenuated inversion recovery

SWI - Susceptibility weighted imaging

ICA - Internal carotid artery

SCA - Superior cerebral artery

BA - Basilar artery

AICA - Anterior inferior cerebellar artery

PICA - Posterior inferior cerebellar artery

V1–V4 - Segments of the vertebral artery

VDRL - Veneral disease research laboratory

SSRI - Selective serotonin-reuptake inhibitors

ED - Emergency department

MTT - Mean transit time

CBV - Cerebral blood volume

CBF - Cerebral blood flow

IC - Intracranial

EC - Extracranial

PSG Institute of Medical Science and Research, Coimbatore
Institutional Human Ethics Committee
INFORMED CONSENT FORMAT FOR RESEARCH PROJECTS

(strike off items that are not applicable)

I / We (write name of the investigator(s) here), Dr.SRINIVASAN .T V, am carrying out a study on the topic: A PROSPECTIVE STUDY OF ETIOLOGY AND CORRELATION OF CLINICAL FEATURES WITH MRI-MRA & CAROTID VENOUS DOPPLER IN ISCHEMIC STROKE PATIENTS as part of my / our research project being carried out under the aegis of the Department of: GENERAL MEDICINE

(*Applicable to students only*): My / our research guide is: Dr.SUJITH KUMAR S MD,
Co-guide: Dr.MADAKASIRA BHEEMARAO PRANESH R
Dr.GNANASHANMUGAM
The justification for this study is:

Considering the disease burden and the morbidity and mortality associated with ischemic stroke in patients,to analyse whether strokes are intracranial or extracranial in origin by MRI-MRA & CV DOPPLER and whether to manage medically or surgically

The objectives of this study are:

Primary Objective: To study whether strokes are intracranial, extracranial or both in origin.
Secondary Objective: To analyse which areas of the brain is most commonly affected. To determine clinical correlation with the area of infarct in the brain.

Sample size: 75

Study volunteers / participants are (specify population group & age group): Patients admitted with ischemic stroke.

Location: PSG Hospitals, Coimbatore.

We request you to kindly cooperate with us in this study. We propose collect background information and other relevant details related to this study. We will be carrying out:

Initial interview (specify approximate duration):_____40_____ minutes.

Data collected will be stored for a period of __TWO__ years. We will / will not use the data as part of another study.

Health education sessions: Number of sessions: _____. Approximate **duration** of each session:

_____ minutes.

Clinical examination (Specify details and purpose): To assess the general condition and systemic examination

No. of times examined: ONCE

Whether blood sample collection is part of routine procedure or for research (study) purpose: NIL

1. Routine procedure 2. Research purpose

Specify **purpose**, discomfort likely to be felt and side effects, if any: __NIL___

Whether blood sample collected will be stored after study period: No, it will be destroyed

Whether blood sample collected will be sold: No

Whether blood sample collected will be shared with persons from another institution: No

Medication given, if any, duration, side effects, purpose, benefits: NIL

Benefits from this study: A PROSPECTIVE STUDY OF CORRELATION OF CLINICAL FEATURES, MRI-MRA, CAROTID VENOUS DOPPLER IN ISCHEMIC STROKE PATIENTS.

Risks involved by participating in this study: NIL

How the **results** will be used: Study will be submitted to Dr. MGR Medical University as thesis in post graduate course in general medicine.

If you are uncomfortable in answering any of our questions during the course of the interview, **you have the right to withdraw from the interview / study at anytime.** You have the freedom to withdraw from the study at any point of time. Kindly be assured that your refusal to participate or withdrawal at any stage, if you so decide, will not result in any form of compromise or discrimination in the services offered nor would it attract any penalty. You will continue to have access to the regular services offered to a patient. You will **NOT** be paid any remuneration for the time you spend with us for this interview / study. The information provided by you will be kept in strict confidence. Under no circumstances shall we reveal the identity of the respondent or their families to anyone. The information that we collect shall be used for approved research purposes only. You will be informed about any significant new findings - including adverse events, if any, – whether directly related to you or to other participants of this study, developed during the course of this research which may relate to your willingness to continue participation.

Consent: The above information regarding the study, has been read by me/ read to me, and has been explained to me by the investigator/s. Having understood the same, I hereby give my consent to them to interview me. I am affixing my signature / left thumb impression to indicate my consent and willingness to participate in this study (i.e., willingly abide by the project requirements).

Signature / Left thumb impression of the Study Volunteer / Legal Representative:

Signature of the Interviewer with date: Witness:

Contact number of PI: 08754189611

Contact number of Ethics Committee Office: 0422 2570170 Extn.: 5818

ஒப்புதல் படிவம்

தேதி :

---------- ஆகிய நான், **PSG** மருத்துவக் கல்லூரியின் ---------- துறையின் கீழ், ----------
---
---------- என்ற தலைப்பில் ஆய்வு மேற்கொள்ள உள்ளேன்.

என் ஆய்வு வழிகாட்டி:

ஆய்வு மேற்கொள்வதற்கான அடிப்படை:

ஆய்வின் நோக்கம்:

ஆய்வில் பங்கு பெறும் நபர்களின் எண்ணிக்கை:

ஆய்வு மேற்கொள்ளும் இடம்:

ஆய்வின் பலன்கள்:

ஆய்வினால் ஏற்படும் அசௌகரியங்கள் / பக்க விளைவுகள்:

இந்த ஆய்வில் கிடைக்கும் தகவல்கள் ---------- வருடங்கள் பாதுகாக்கப்படும். இவை வேறு எந்த ஆய்விற்கும் பயன்படுத்தப் பட மாட்டாது. எந்த நிலையிலும் உங்களைப் பற்றிய தகவல்கள் யாருக்கும் தெரிவிக்கப்பட மாட்டாது. அவை இரகசியமாக வைக்கப்படும்.

இந்த ஆய்வில் பங்கேற்க ஒப்புக்கொள்ளுவதால் எந்த விதமான பலனும் உங்களுக்குக் கிடைக்காது. எந்த நேரத்தில் வேண்டுமானாலும் ஆய்விலிருந்து விலகிக்கொள்ளும் உரிமை உங்களுக்கு உண்டு. ஆய்விலிருந்து விலகிக்கொள்வதால் உங்களுக்கு அளிக்கப்படும் சிகிச்சையில் எந்த வித மாற்றமும் இருக்காது.

இந்த ஆராய்ச்சிக்காக உங்களிடம் சில கேள்விகள் கேட்கப்படும் / சில இரத்த மாதிரிகள் அல்லது திசு மாதிரிகள் எடுக்கப்படும்.

மேலும், இந்த ஆய்வில் பங்கு கொள்வது உங்கள் சொந்த விருப்பம். இதில் எந்த விதக் கட்டாயமும் இல்லை. நீங்கள் விருப்பப் பட்டால், இந்த ஆய்வின் முடிவுகள் உங்களுக்குத் தெரியப் படுத்தப்படும்.

ஆய்வாளரின் கையொப்பம் :
தேதி :

<u>ஆய்வுக்குட்படுபவரின் ஒப்புதல்:</u>

நான் இந்த ஆராய்ச்சியின் நோக்கம் மற்றும் அதன் பயன்பாட்டினைப் பற்றி தெளிவாகவும், விளக்கமாகவும் தெரியப்படுத்தப் பட்டுள்ளேன். இந்த ஆராய்ச்சியில் பங்கு கொள்ளவும், இந்த ஆராய்ச்சியின் மருத்துவ ரீதியான குறிப்புகளை வரும் காலத்திலும் உபயோகப்படுத்திக் கொள்ளவும் முழு மனதுடன் சம்மதிக்கிறேன்.

ஆய்வுக்குட்படுபவரின் பெயர், முகவரி :

கையொப்பம் :
தேதி :

ஆய்வாளரின் தொலைபேசி எண்:

மனித நெறிமுறைக் குழு அலுவலகத்தின் தொலைபேசி எண்: 0422 2570170 Extn.: 5818

S.NO	SEX	AGE	NIHSS	correlation of clinical vs MRI	correlation of MRI with MRA & CVD	DIABETIC	S.HTN	ANA	HOMOCYSTIENE	RISK FACTORS
1	M	50	4	Yes	Yes	DM	NIL			SMOKER & ALCOHOLIC
2	M	86	5	Yes	Yes	DM	HTN			Nil
3	M	57	2	Yes	NO	NIL	NIL			SMOKER,
4	M	63	2	Yes	NO	DM	HTN			smoker,
5	M	64	4	Yes	Yes	DM	HTN			NIL
6	F	50	2	Yes	Yes	DM	HTN			NIL
7	F	61	6	Yes	Yes	DM	NIL			NIL
8	M	58	2	Yes	NO	DM	NIL			SMOKER ALCOHOLIC
9	M	60	4	Yes	Yes	NIL	HTN			NIL
10	M	55	9	NO	NO	NIL	NIL			ALCOHOL
11	M	76	1	Yes	Yes	NIL	NIL			SMOKER
12	M	71	1	Yes	Yes	NIL	NIL			Nil
13	F	58	8	Yes	Yes	DM	HTN			NIL
14	M	68	3	Yes	Yes	DM	HTN			NIL
15	M	70	5	Yes	Yes	DM	HTN			NIL
16	M	52	2	Yes	Yes	NIL	NIL			ALCOHOLIC,SMOKER
17	M	57	1	Yes	NO	NIL	NIL			SMOKER
18	M	63	4	NO	Yes	DM	HTN			SMOKER
19	F	54	6	Yes	NO	DM	HTN	POSITIVE	29	Nil
20	F	71	7	Yes	NO	DM	HTN	NEGATIVE	8	Nil
21	M	47	6	NO	NO	DM	HTN	NEGATIVE	10	SMOKER ALCOHOLIC
22	M	66	4	Yes	Yes	NIL	NIL	POSITIVE	34	EX SMOKER
23	M	58	7	Yes	Yes	NIL	NIL	NEGATIVE	68	SMOKER
24	m	79	11	Yes	NO	DM	HTN	ANA IF NEGATIVE	H -22.72	alcholic
25	M	60	6	Yes	Yes	NIL	HTN	ANA IF NEGATIVE	H -17.9	ALCOHOLIC,SMOKER
26	M	70	5	Yes	Yes	NIL	HTN	ANA IF NEGATIVE	HC 18	ALCOHOLIC,SMOKER
27	M	52	7	Yes	NO	DM	HTN	ANA IF NEGATIVE	32	ALCOHOLIC,SMOKER
28	M	63	6	Yes	Yes	DM	NIL	ANA POSITIVE	HC 12	EX SMOKER
29	M	69	10	Yes	Yes	DM	HTN			EX SMOKER
30	M	59	2	Yes	NO	DM	HTN			SMOKER ALCOHOLIC

31	M	54	8	Yes	NO	DM	NIL			NIL
32	M	64	9	Yes	Yes	DM	HTN			SMOKER ALCOHOLIC
33	M	47	1	Yes	NO	DM	HTN			NIL
34	M	31	3	Yes	Yes	NIL	NIL			NIL
35	m	52	7	Yes	Yes	NIL	NIL	NEGATIVE	27	SMOKER ALCOHOLIC
36	M	55	6	Yes	NO	NIL	NIL	NEGATIVE	20	Nil
37	F	70	2	Yes	NO	DM	HTN	NEGATIVE	5.85	Nil
38	M	43	4	Yes	Yes	NIL	HTN	NEGATIVE	95	SMOKER ALCOHOLIC
39	M	45	8	Yes	NO	NIL	NIL	NEGATIVE	160	NIL
40	M	72	7	Yes	Yes	DM	HTN	NEGATIVE	7.88	Nil
41	M	46	7	NO	Yes	DM	HTN	NEGATIVE	14.8	NIL
42	F	60	4	Yes	Yes	DM	HTN	negative	16.1	Nil
43	F	47	3	Yes	NO	NIL	NIL	positive	21	NIL
44	M	75	8	Yes	Yes	DM	HTN	NEGATIVE	14	Nil
45	M	61	8	Yes	NO	DM	HTN	POSITIVE	15	Nil
46	M	69	5	Yes	NO	DM	HTN	POSITIVE	13	SMOKER
47	M	58	4	Yes	NO	NIL	HTN	NEGATIVE	22	Nil
48	M	30	4	Yes	Yes	NIL	NIL	NEGATIVE	61	Nil
49	M	32	7	Yes	Yes	NIL	NIL	NEGATIVE	19.8	Nil
50	M	70	5	Yes	Yes	NIL	NIL	NEGATIVE	19	SMOKER
51	M	50	4	Yes	NO	DM	S.HTN	POSITIVE	19	Nil
52	M	73	10	Yes	Yes	DM	S.HTN	sample rec	19	NIL
53	F	58	9	Yes	Yes	NIL	NIL	NEGATIVE	5.9	Nil
54	M	61	8	Yes	NO	NIL	NIL	NEGATIVE	81	NIL
55	M	50	4	Yes	Yes	DM	NIL			ALCOHOL
56	M	48	12	Yes	Yes	DM	HTN	NEGATIVE	6	ALCOHOLIC,SMOKER
57	M	67	5	Yes	Yes	NIL	NIL	NEGATIVE	10	NIL
58	M	58	3	Yes	NO	NIL	NIL	NEGATIVE	16	NIL
59	M	60	3	Yes	NO	NIL	NIL	NEGATIVE	32	ALCOHOLIC,SMOKER
60	F	62	6	Yes	Yes	DM	HTN	POSITIVE	15	NIL
61	M	54	5	Yes	Yes	DM	HTN	NEGATIVE	16	ALCOHOLIC,SMOKER
62	M	52	5	Yes	NO	DM	HTN			Nil
63	M	74	9	Yes	Yes	NIL	HTN	POSITIVE	44	alcholic
64	M	46	10	Yes	Yes	DM	HTN	NEGATIVE	24	ALCOHOLIC,SMOKER
65	M	69	4	Yes	NO	NIL	NIL	POSITIVE +++	51	NIL

66	M	65	10	Yes	Yes	DM	NIL	NEGATIVE	21	ALCOHOLIC,SMOKER
67	M	45	2	Yes	NO	NIL	NIL	NEG	50	ALCOHOLIC,SMOKER
68	F	80	2	Yes	NO	DM	Nil	NEG	13	Nil
69	F	65	3	Yes	NO	NIL	HTN	POSITIVE ++	20	Nil
70	M	35	5	Yes	Yes	NIL	NIL	NEG	13	SMOKER
71	M	74	3	Yes	NO	DM	NIL	NEG	12	NIL
72	F	79	7	Yes	Yes	NIL	HTN	NEGATIVE	32	NIL
73	M	64	4	NO	Yes	DM	HTN			SMOKER
74	M	39	6	Yes	Yes	NIL	NIL			ALCOHOLIC,SMOKER
75	M		5	Yes	Yes	NIL	NIL			ALCOHOLIC,SMOKER