ANALYSIS OF 50 CASES OF STROKE IN YOUNG ADULTS

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

for

M.D. Degree in General Medicine (Branch I)

The Tamilnadu
Dr. M.G.R. Medical University
Chennai
March 2009

ANALYSIS OF 50 CASES OF STROKE IN YOUNG ADULTS

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

for

M.D. Degree in General Medicine (Branch I)

The Tamilnadu
Dr. M.G.R. Medical University
Chennai
March 2009

Coimbatore Medical College
Coimbatore - 641 014
DEPARTMENT OF GENERAL MEDICINE
COIMBATORE MEDICAL COLLEGE HOSPITAL
COIMBATORE

CERTIFICATE

This is to certify that the Dissertation entitled **"Analysis of 50 cases of stroke in young adults"** herewith submitted by **Dr.N.SENTHILRANI.,** Post Graduate in General Medicine, Coimbatore Medical College to the Tamilnadu Dr.M.G.R.Medical University is a record of a bonafide research work carried out by her under my guidance and supervision from APRIL 2007 to SEP 2008.

Dr.M.RAMASAMY.M.D
Professor and Unit Chief
Department of Medicine.

Dr.K.UMAKANTHAN.M.D.,
Professor and Head of
Department of Medicine.

DEAN

DECLARATION

I solemnly declare that the dissertation titled **"Analysis of 50 Cases of Stroke in Young Adults"** was done by me at Coimbatore Medical College & Hospital during the period from April 2007 to September 2008 under the guidance and supervision of **Prof. Dr. K. Umakanthan M.D.,** and **Prof. M. Ramasamy M.D.**

This dissertation is submitted to the Tamilnadu Dr. MGR Medical University towards the partial fulfillment of the requirement for the award of MD Degree in General Medicine (Branch I)

Place: Coimbatore **Dr. N. SENTHILRANI**

Date:

Coimbatore Medical College

COIMBATORE, TAMILNADU, INDIA - 641 014

(Affiliated to The T.N. Dr. MGR Medical University, Chennai)

ETHICS COMMITTEE

CERTIFICATE

Name of the Candidate	:	Dr. N. Senthil Rani
Course	:	M.D. (General Medicine)
Period of Study	:	2006 - 2009
College	:	Coimbatore Medical College
Dissertation Topic	:	Analysis Of 50 Cases Of Stroke In Young Adults

The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation is accepted / ~~Not accepted~~ and you are permitted / ~~Not Permitted~~ to proceed with the above Study.

Coimbatore - 14.

Date : 5.3.2008

Secretary
Ethics Committee

ACKNOWLEDGEMENT

I express my regards and gratitude to Dean, Coimbatore Medical College for permitting and supporting me to conduct this study.

I am indebted to Professor and Head of Department of medicine, Dr.K.Umakanthan MD, Professor Dr.M.Ramasamy MD, and Unit chiefs – Department of medicine for their valuable guidance, encouragement, inspirations and support without which this effort would not have been possible.

I am extremely grateful for the whole hearted support and valuable feedback, suggestions and guidance from professor and Head of Department of Neuromedicine Dr.K.Govindarajan MD, DM.

I express my sincere thanks and gratitude to my assistant professors, Dr.S.Usha, Dr.T.Ravikumar MD, Dr.K.Jamunarani MD, Mr.P.S.Manshur MD, Dr.R.Ramakrishnan DM, Dr.M.Sacratis DM for their valuable guidance.

I wish to express my thanks to all coPGs of the department for their kind cooperation.

Last but not the least my gratitude and thanks to the patients and their attenders for their whole hearted co-operation, without which this study would not have been possible.

CONTENTS

INTRODUCTION

Stroke is one of the leading causes of morbidity and ranks next only to Coronary Artery Disease and Malignancy as the leading cause of mortality worldwide. At least 50 percent of the neurological disorders in a general hospital are due to stroke. As remarked by a renowned neurologist C.M.Fisher, neurology is learnt “Stroke by Stroke”.

Cerebrovascular diseases occur predominately in the middle and late years of life. The incidence of stroke increases with age; thus the disability affects many people in their “golden years”, a segment of the population that is growing rapidly in western countries. Categories of cerebrovascular diseases include ischaemia – infarction and intracranial haemorrhage . Many of the arterial and cardiac disorders underlying these diseases are preventable; the morbidity and mortality from cerebrovascular diseases has been diminishing in recent years, apparently because of better recognition and treatment of hypertension.

Most cerebrovascular diseases are manifest by the abrupt onset of a focal neurologic deficit. The deficit may remain fixed or may rapidly improve or progressively worsen. It is this abrupt onset of a non-occlusive and focal neurologic deficit that defines a stroke, or cerebrovascular accident (CVA).

Stroke in young adults is uncommon but by no means rare: some 4% of all strokes occur under the age of 40 years. There are a number of additional causes which should be specifically considered in young adults beyond those common in adults. These include neck trauma causing carotid dissection, alcohol intoxication, infarction in association with migraine, post-herpeszoster infarction, and cardiac causes such as

Rhematic heart disease, prolapsing mitral valve or atrial myxoma.

The term stroke denotes the sudden and dramatic development of a focal neurological deficit due to cerebrovascular disorder. The advent of imaging procedures such as computerized tomography, magnetic resonance imaging and carotid Doppler made the evaluation of stroke and its risk factors easier.

However young strokes pose a major socioeconomic challenge, occupational neurorehabilitational programme of stroke survivors and also have varying aetiology and prognosis in comparison with stroke elderly also. It is one of the major causes of disability and death among the adult population.

AIM OF THE STUDY

1. To evaluate the risk factors of Stroke in young adults.
2. To study the different mode of clinical presentation or types of Stroke in young adults.
3. To study the pathogenesis of young stroke with the aid of investigations, treatment outcome and prognosis.
4. Correlative study of the above modalities.

MATERIALS AND METHODS

The study was conducted in Coimbatore Medical College Hospital during the period of 2007 - 2008. The bed strength of this Hospital is 1020 and about 1200 patients are cared as inpatients, daily 4500 outpatients come to this hospital for treatment. This center is rendering medical services to a fairly large size of population nearly 10 lakhs, cater

ing to the population in and around Coimbatore and nearby areas of Kerala including Palghat. This being a post-graduate training center, the fulltime services of a team of qualified neurologist and experienced medical personnel are available round the clock. This study was possible because of full cooperation and enthusiasm of various departments like Neurology, Radiology and with available facilities of this college.

The study includes fifty patients of stroke in patients below the age of 40 years admitted in medical wards and Neurology ward. Ethics committee approval was obtained for conducting the study. Informed consent was obtained from all the patients. Patients in the paediatric age group are not included. A carefully elicited history and repeated clinical examinations were used in ascertaining the temporal profile of the disease and the probable area of brain that is affected. A detailed history taking was done for risk factors like Smoking, Alcohol, Rheumatic valvular heart disease, Tuberculosis and family history of cerebrovascular disease.

A clinical search for extra-cranial carotid artery narrowing, cardiac diseases which can give rise to cerebral embolism, evidence for generalized arterial disease, systemic

hypertension, diabetes mellitus, Rheumatic valvular heart disease and other risk factors were made.

Laboratory evaluation and other investigations :

After detailed history and meticulous neuromedical examination including palpation and auscultation of brachiocephalic and peripheral pulses, the relevance and priority of any laboratory test in acute stroke should be at the clinician's judgement. The value of careful ophthalmoscopic examination of the retina and its vasculature for disease and embolic fragments needs emphasis.

1. Urine examination for albumin and sugar.
2. Estimation of blood sugar and serum lipid profile levels for detecting diabetes and hyperlipidemia which are predisposing causes of atherosclerosis.
3. Serological test for syphilis was done in all patients.
4. Haemtological studies like complete hemogram with platelet count, ESR, bleeding / clotting and prothrombin time to find out conditions like polycythaemia, anaemia, thrombocytopenia and bleeding disorders were undertaken.
5. When elevated ESR was present, further test were made to identify the causes like systemic lupus erythematosus, subacute baterial endocarditis and tuberculous meningitis.
6. Two dimensional echocardiography for detecting cardiac source of emboli was done in all patients.

7. ECG for detecting rhythm disorders & underlying cardiac disorder were done.
8. CSF analysis was used to determine meningeal infection causing stroke.
9. Carotid angiography was done only in those patients in whom a vascular malformation, subdural haematoma, intracranial tumour or extracranial vascular disease was suspected.
10. CT Scan of brain was done to assess the lesion was infartion or haemorrhage and to locate the site of lesion.
11. HIV testing was done in all patients.
12. Sr.Homocystein level was estimated.
13. MRI Brain / MR Angiogram / MRVenogram – were done.
14. TEE was done.
15. Anti thrombin III level was estimated.

REVIEW OF LITERATURE

GENERAL CONSIDERATIONS[1-8]

DEFINITION

Stroke is defined as a focal (or at times global Neurological impairment of sudden onset, lasting more than 24 hr (or leading to death and of presumed vascular origin)[25].

The term Transient Ischaemic Attack (TIA) implies focal neurological deficit with complete recovery of cerebral function within 24 hrs. Some of the sub types of stroke include cerebral haemorrhage, cerebral infarction and sub arachnoid haemorrhage.

The normal functioning of the brain is dependant upon a relatively constant supply of oxygen, glucose and other nutrients derived from the blood perfusing it. Normal blood flow is brain to 55 – 70 ml/min. If for any reason the blood flow is critically reduced below 15 ml per 100g per minute, the resulting ischaemia with hypoxia when sufficiently prolonged, may cause death of neurons and glia (cerebral infarction).

The mean arterial blood pressure, cerebrovascular and tissue resistance, local metabolic products (pH, paO_2, etc) together with several known and unknown factors help to maintain the critical threshold of blood flow for energy metabolism. Further more the blood flow varies in different areas of the brain and auto regulation determines the regional blood flow to meet local metabolic need.

In regions of cerebral ischaemia there is “Paralysis of auto regulation” and the microvasculature in the ischaemic region is not reactive to pressure changes, vasoactive

agents and other forms of stimuli. The cerebral vasculature in this ischaemic zone becomes permeable to proteins, and fluid leaks in the vicinity leading to extracellular oedema. Such events also lead to local haemoconcentration and vascular stasis.

Hence cerebral infarction is not merely the result of ischaemia from occluded blood vessels but an end result of a series of highly complex ischaemia modifying events.

BLOOD SUPPLY OF THE BRAIN : (FIG : 1)

At rest the brain which is only 2 percent of total body weight receives 15 percent of the cardiac output and consumes about 25 percent of the total inspired oxygen. This rich blood supply is carried by two internal carotid and two vertebral arteries which anastamose at the base of brain to form the 'circle of Willis'. The carotid arteries supply the ɪrtion of the bra

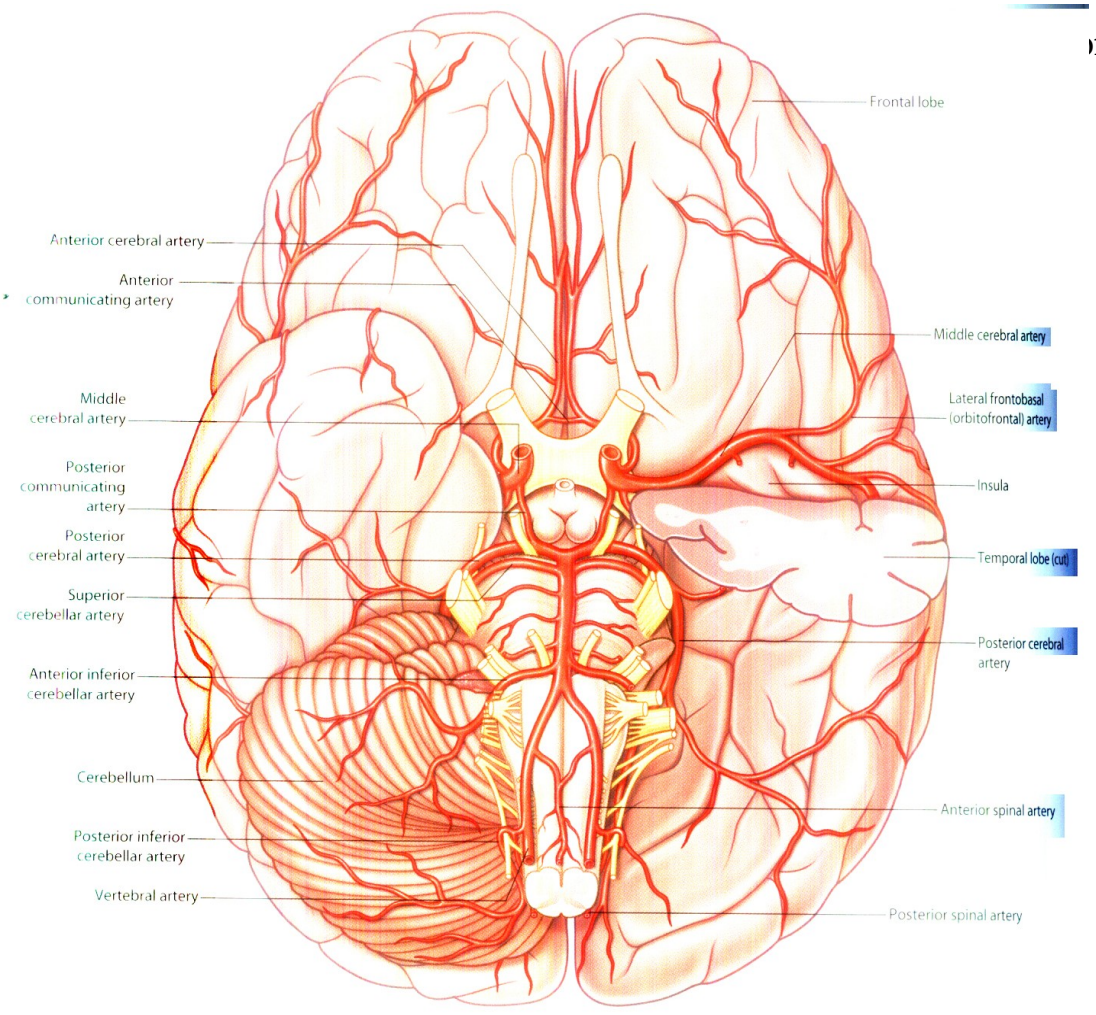


Figure : 1 - Base of Brain Showing Circle of Willis

The branches of the internal carotid artery are

a. The Opthalmic artery

b. Anterior cerebral artery

c. Middle cerebral artery

d. Anterior choroidal artery

e. Posterior Communicating artery

The vertebral artery which arises from the subclavian artery, enters foramen magnum and unites with the opposite vertebral artery at the pontomedullary junction to form the basilar artery. Vertebral artery gives rise to anterior and posterior spinal arteries, the posterior inferior cerebellar artery and small penetrating arteries to the medulla. The basilar artery ascends up to the pontomidbrain junction in the interpeduncular cistern and divides into the two posterior cerebral arteries. Numerous small branches penetrate the brainstem and cerebellum. It also gives rise to the anterior inferior cerebellar artery, the internal auditory artery and the superior cerebellar artery. The meninges are supplied by branches of the internal carotid, external carotid and vertebral arteries.

THE COLLATERAL BLOOD SUPPLY OF THE BRAIN

Normally each ICA provides blood to the anterior two thirds of the cerebral hemisphere on the ipsilateral side. There is little mixing of blood via the posterior communicating artery with the vertebrobasilar system. However collateral circulation

can develop distal to major artery occlusion. The development of collaterals is more effective if the vessel occlusion occurs insidiously rather than suddenly. But unlike the normal cerebral blood supply, the functional capacity of the collateral blood supply to respond to changes in perfusion pressure is limited.

COLLATERAL BLOOD FLOW MAY DEVELOP VIA

1. The circle of Willis. However about 50 percent of circles have one or more hypoplastic segments (usually one of the communicating arteries) and also since atheroma commonly affects the circle of Willis, the potential for collateral flow is not always good.
2. Around the orbit, branches of the ECA anastomose with branches of the Opthalmic artery if the ICA is severely stenosed.
3. Muscular branches of the vertebral artery in the neck distal to an obstruction of that artery, receive blood from occipital and ascending pharyngeal of ECA.
4. Leptomeningeal anastomoses on the surface of the brain may develop between cortical branches of the anterior, middle and posterior cerebral arteries.
5. Dural anastomoses can develop between meningeal branches of the precapillary bed of the vertebral arteries.
6. Parenchymal anastomoses occasionally develop in the precapillary bed of the perforating arteries supplying the basal ganglia (Moyamoya syndrome).

VENOUS DRAINAGE OF THE BRAIN :

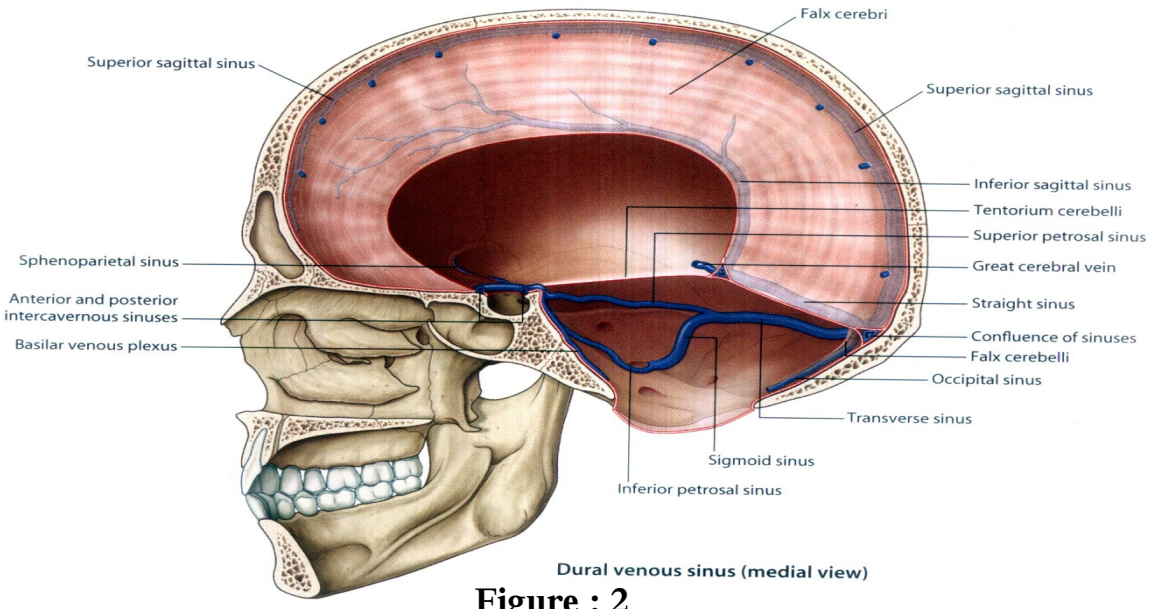


Figure : 2

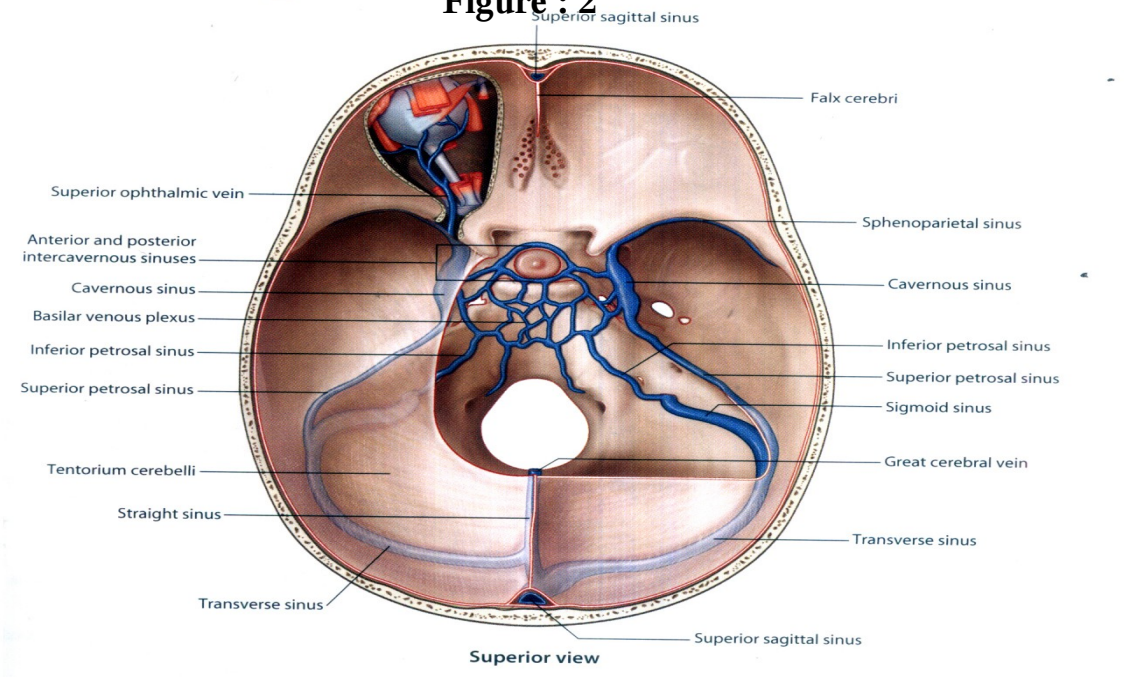


Figure 3 : Dural Venous Sinus (Superior View)

Venous blood flow peripherally via the superficial cerebral veins and centrally via the deep cerebral veins into the venous sinuses which in turn drain into the internal jugular vein (Fig 2,3). The cerebral veins are thin walled have no valves and the blood flow is often in the same direction as in the neighbouring arteries. There are numerous venous connections between the veins and dural sinuses as well as with the venous system in the meninges, skull, scalp and nasal sinuses so facilitating the propagation of thrombus or spread of infection between these vessels.

EPIDEMIOLOGY :

This lagged behind coronary heart disease because

1. Stroke is a disorder of late middle age and elderly where other diseases coexist.
2. Stroke pathologically is more diverse and may be due to intracerebral small vessel disease or embolism from the heart or primary intracerebral haemorrhage.

MORTALITY :

Mortality rises rapidly with age.

INCIDENCE :

Stroke in young adult is surprisingly common. Stroke incidence rises with age with about 75% of the stroke in adults occur between the age group of 26 to 40 years and about 25% occurring below the age of 26 years. Ischaemic stroke is much more common than haemorrhagic stroke.

GEOGRAPHICAL RACIAL AND SOCIAL INFLUENCES :

Primary intra cerebral haemorrhage is less common in Western countries than in Japan and China.

In India there is some evidence that stroke is particularly common in young people. During pregnancy stroke is commonly associated with CVT.

TRENDS IN MORTALITY AND INCIDENCE :

Incidence is declining with assumption that the prevalence of hypertension is less than it was and early diagnosis and treatment of hypertension may also be responsible. The prevalence of Rheumatic heart disease also is less than it was earlier.

SEASONAL AND DIURNAL VARIATION :

Stroke incidence and mortality is more in winter, the possible explanation being the effect of temperature, pollution and higher blood pressure in the winter. The increased mortality may also be due to complications of stroke such as pneumonia which is more common in winter.

Cerebral infarction occurs most commonly in the hour or two after waking in the morning. Subarachnoid haemorrhage is unlikely to occur during sleep. Circadian changes in physical activity, catecholamine level, blood pressure, blood viscosity and platelet aggregation may explain.

Intracerebral haemorrhage is more likely to occur during strenuous activity than during rest or sleep.

ETIOLOGY OF STROKE IN YOUNG ADULTS :

The range of potential etiologies for stroke in young adults is broader than that for older adults. Like in older adults, stroke in younger adults is typically categorized as primarily ischaemic or haemorrhagic . Ischaemic etiologies include cardioembolic, atherosclerotic disease[24], and nonatherosclerotic cerebral vasculopathies.

ETIOLOGY OF STROKE IN YOUNG ADULTS :

ISCHAEMIC [26-34]

- Cardiac disease

LARGE VESSEL DISEASE :

- Premature atherosclerosis
- Dissection (Spontaneous or traumatic)
- Inherited metabolic disease (Homocystinuria, Fabry's, Pseudoxanthoma elasticum, MELAS syndrome)
- Fibromuscular dysplasia
- Infection (bacterial, fungal, tuberculosis, syphilis, Lyme's disease)
- Vasculitis (Collagen vascular diseases – systemic lupus erythematosus, rheumatoid arthritis, Sjogren's syndrome, polyarteritis nodosa; Takayasu's disease, Wegener's syndrome, cryoglobulinemia, sarcoidosis, inflammatory bowel disease, isolated central nervous system angitis)
- Moyamoya disease
- Radiation

- ❖ Toxic (Drugs)

SMALL VESSEL DISEASE :

- ❖ Vasculopathy (infectious, noninfectious, microangiopathy)

HAEMATOLOGIC DISEASE :

- ❖ Sickle-cell disease
- ❖ Leukemia
- ❖ Hypercoagulable states
- ❖ Disseminated intravascular coagulation
- ❖ Thrombocytosis
- ❖ Polycythemia Vera
- ❖ Paroxysmal nocturnal haemoglobinuria
- ❖ Thrombotic thrombocytopenic purpura
- ❖ Venous occlusion

MIGRAINE[16]

HAEMORRHAGIC

Subarachnoid haemorrhagic (cerebral aneurysm)

Intraparenchymal haemorrhage .

- ❖ Arteriovenous malformation
- ❖ Neoplasm

- ❖ Haematological Disease
- ❖ Drug use
- ❖ Iatrogenic (peri-procedural)

CARDIAC DISEASE :[13, 14]

It is the most common cause of stroke in young adult.

It includes :

1. Congenital heart disease
2. Rheumatic valve disease
3. Mitral valve prolapse [9]
4. Patent foramen ovale
5. Endocarditis
6. Atrial myxoma
7. Arrhythmias
8. Cardiac Surgery
9. Cardiomyopathy
10. Prosthetic Valves [10]
11. Mural thrombi after myocardial infarct[17]

Of these, rheumatic mitral valvular disease with atrial fibrillation, prosthetic valves, subacute bacterial endocarditis and intracardiac tumour are universally recognized causes of emboli. The role of disorders such as non-valvular atrial fibrillation, myocardial infarction and mitral valve prolapse is less clear. In mitral valve

prolapse the cerebral symptoms may arise from non-septic or septic emboli and paroxysmal arrythmias associated with this condition may play a role. Cardiomyopathies are being increasingly recognized as a cause of embolic stroke in young adults. Cardiogenic cerebral emboli is the most frequent cause of stroke recurrence.

CARDIOGENIC EMBOLIC OCCLUSION :

The determination that a stroke is embolic in origin generally results from the demonstration of an appropriate cardiac abnormality in a patient with a stroke, featuring characteristics suggestive of an embolus. Clinical features of a stroke which suggest a possible cardiac embolic origin are –

1) Sudden onset with maximal neurological deficit, appearing immediately.
2) History of multiple episodes of TIA of different pattern. Multifocal cerebral infarctions especially in a patient with an associated systemic arterial occlusion, (eg) renal arteries or limb arteries.

PREMATURE ATHEROSCLEROSIS :

Though atherosclerosis is the major cause of stroke in the elderly age group, it does occur in young patients too, especially when predisposing factors like hypertension[12], diabetes and hyperlipidemia[18] are present. Young men who smoke heavily suffer atherosclerotic brain infarction six times more frequently than non-smokers[11]. Non modifiable risk factors include age, race and male sex. Genetics may play a role.

DISSECTION OF INTRACRANIAL CEREBRAL ARTERIES :

Dissection of intracranial cerebral arteries most often affect the middle cerebral and basilar arteries with the clinical presentation of acute infarction. These subintimal dissections of unknown cause develop in otherwise healthy individuals. Dissection of the major extracranial cerebral arteries may be spontaneous dissections, occur most often in patients younger than 40 years.

INHERITED METABOLIC DISEASES :

HOMOCYSTINURIA : [35-37, 49-51]

Homocystinuria predisposes to cerebral arterial or less often venous thrombosis as well as to thromboemboli in other organs. The mechanisms are not delineated but the estimated risk of stroke at a young age is between 10% and 16%. The diagnosis is usually made in childhood because of other stigmata such as thin Marfan-like appearance, malar flush, dislocated ocular lens, bony deformities, mental retardation and seizures.

FIBROMUSCULAR DYSPLASIA :

Fibromuscular dysplasia is a non-atheromatous vascular disorder, in which there is intimal and medial fibroplasia of the extracranial internal carotid artery and other large systemic vessels. The intracranial arteries are usually spared. The disorder is far more common in women. The cause is not understood.

CEREBRAL & MENINGEAL INFECTIONS :

Meningeal infections can result in cerebral infarction through development of

inflammatory changes in the vessel walls. Meningovascular syphilis and tuberculous infections are common causes. Pyogenic infections are infrequent causes. Another infection that may result in cerebral arteritis is mucormycosis. Other rare causes of cerebral infarction are fungus, typhus, schistosomiasis, falciparum malaria and trichinosis.

TAKAYASU'S ARTERITIS :

Takayasu's arteritis (pulsesless disease or aotic arch syndrome) is a giant cell arteritis that may result in narrowing and thrombosis of large branches of the aortic arch at their origins and aneurysmal formation, signs of ischaemia of the head and arms include cataracts, retinal and optic atrophy, transient monocular blindness, focal cerebral symptoms and hypertension in the legs with intermittent claudication in the arms (reverse coarctation).

MOYAMOYA DISEASE :

Moyamoya disease is a syndrome of stenosis of the vessels in and around the circle of Willis, with profuse telangiectatic collacteral vessels at the base of brain that have the angiographic appearance of a puff of smoke, Microaneurysms may develop and either infarction or subarachonoid haemorrhage may result.

DRUGS :

Drugs that have been associated with stroke include methamphetamines, LSD, heroin, oral contraceptives[15], and anticoagulants. Methamphetamine induces a necrotizing vasculitis that may result in intracerebral, subdural and subarachnoid

haemorrhages. LSD which is an ergot alkaloid produces arteriospasm and heroin produces allergic vascular hypersensitivity leading to cerebral infarction. The risk of stroke with the use of oral contraceptives is increased five to nine fold for thrombosis and two fold for haemorrhage.

VASCULOPATHY :

Apart from atherosclerosis there are other vascular diseases which can produce stroke in young. They are –

1) Arteritis due to –
    a) Collagen disorders.
    b) Infections – Meningovascular syphilis
        Tuberculous arteritis
        Pyogenic infection
        Fungal - Mucormycosis
    c) Takayasu's arteritis
    d) Drug induced vasculitis – methamphetamine
    e) Irradiation
2) Ruptured saccular aneurysm
3) Moyamoya disease
4) Dissection of intracranial cerebral arteries.
5) Fibromuscular dysplasia.

The collagen vascular disease most frequently causing stroke is systemic lupus erythematosus. There is an increased risk of thrombosis in patients with an immunoglobulin called “lupus anticoagulant”. The incidence of stroke is less in polyarteritis nodosa with 13% reportedly experiencing cerebral infarction or haemorrhage. Stroke is infrequent in scleroderma and rheumatoid arthritis.

SICKLE CELL DISEASE :

Sickle cell disease can cause ischaemic infarction, intracerebral haemorrhage, venous sinus and cortical vein thrombosis and subarachnoid haemorrhage. The overall incidence of stroke in sickle cell disease is between 6% to 15%. The risk of cerebral infarction is greatest in children with sickle cell anaemia. Stroke occurs infrequently in sickle cell patients older than 20 years, complications are more common in adults. In addition to small vessel occlusion from intravascular sickling, endothelial proliferation affects small arteries and arterioles and angiopathy may involve the anterior part of the circle of Willis. Stroke recurrence is common in sickle cell anaemia and may be avoided by periodic exchange transfusions aimed at keeping haemoglobin-S levels below 20%.

HYPERCOAGULABLE STATES :

1. Multiple Myeloma
2. Antiphospholipid antibody syndromes
3. Deficiency of antithrombin III or protein S or C
4. Resistance to activated protein C
5. Increased factor VIII

Hyperviscosity syndrome is most offen associated with multiple myeloma with an increase in lgG or lgA paraproteins which causes hyperviscosity, small vessel occlusion and multiple areas of infarction or haemorrhage.

DISSEMINATED INTRAVASCULAR COAGULATION :

Disseminated intravascular coagulation may result in either cerebral haemorrhage or thrombosis. It involves the consumption of coagulation factors and platelet and may be associated with carcinoma, disorders of peripartum and postpartum period and sepsis.

POLYCYTHEMIA :

In polycythemia, there is a predispositions of both arterial and venous thrombosis and retinal vein occlusion. Intracerebral and subarachnoid haemorrhages may occur occasionally. Paroxysmal nocturnal haemoglobinuria may result in cerebral venous thrombosis and is suspected in patients with chronic haemolytic anaemia, unexplained pain and multiple episodes of venous thrombosis at different systemic sites.

PLATELET AND COAGULATION DISORDERS :

Among the platelet and coagulation disorders resulting in stroke are chronic idiopathic thrombocytopenic purpura, thrombotic thrombocytopenic purpura, idiopathic thrombocytosis and disseminated intravascular coagulation. Chronic idiopathic thrombocytopenic purpura is three to four times more common in women than in men and may occasionally result in intracerebral haemorrhage.

The triad of thrombotic thrombocytopenic purpura includes thrombocytopenic purpura, haemolytic anaemia and focal cerebral signs. Seizures are also common. The

disorder may start in the second half of pregnancy simulating eclampsia. The small

Table 1. Causes of and Risk Factors Associated with Cerebral Venous Sinus Thrombosis.

Genetic prothrombotic conditions
Antithrombin deficiency
Protein C and protein S deficiency
Factor V Leiden mutation
Prothrombin mutation (the substitution of A for G at position 20210)
Homocysteinemia caused by gene mutations in methylenetetrahydrofolate reductase
Acquired prothrombotic states
Nephrotic syndrome
Antiphospholipid antibodies
Homocysteinemia
Pregnancy
Puerperium
Infections
Otitis, mastoiditis, sinusitis
Meningitis
Systemic infectious disease
Inflammatory disease
Systemic lupus erythematosus
Wegener's granulomatosis
Sarcoidosis
Inflammatory bowel disease
Behçet's syndrome
Hematologic conditions
Polycythemia, primary and secondary
Thrombocythemia
Leukemia
Anemia, including paroxysmal nocturnal hemoglobinuria
Drugs
Oral contraceptives
Asparaginase
Mechanical causes, trauma
Head injury
Injury to sinuses or jugular vein, jugular catheterization
Neurosurgical procedures
Lumbar puncture
Miscellaneous
Dehydration, especially in children
Cancer

r both

bone

5% of

enous

artum

to a

Other causes of stroke during pregnancy and peurperium are –

1. Venous sinus and cortical vein thrombosis.
2. Emboli from mural thrombi of peripartum cardiomyopathy.
3. Intracerebral and subarachnoid haemorrhage due to eclampsia and consumptive coagulopathies of peripartum period which may occur due to amniotic fluid embolism, premature separation of placenta, septic abortion, hydatidiform mole, intrauterine fetal death and uterine rupture.

INFLAMMATORY BOWEL DISEASES :

Chronic inflammatory bowel diseases such as ulcerative colitis and regional enteritis have been associated with hypercoagulable state and thrombocytosis predisposing to recurrent retinal artery branch occlusions, cerebral venous and arterial thrombosis.

ARTERIOVENOUS MALFORMATIONS OF THE BRAIN :[72-79]

Arteriovenous malformations of the brain are focal abnormal conglomerations of dilated arteries and veins within brain parenchyma, in which a loss of normal vascular organization at the subarteriolar level and a lack of a capillary bed result in abnormal arteriovenous shunting (fig : 4).

The most common presenting sign of an arteriovenous malformation is intracerebral haemorrhage (occurring in 42 to 72% of clinically apparent arteriovenous malformations[52-57]). Haemorrhage of arteriovenous malformations accounts for approximately 2% of all strokes[58-59].

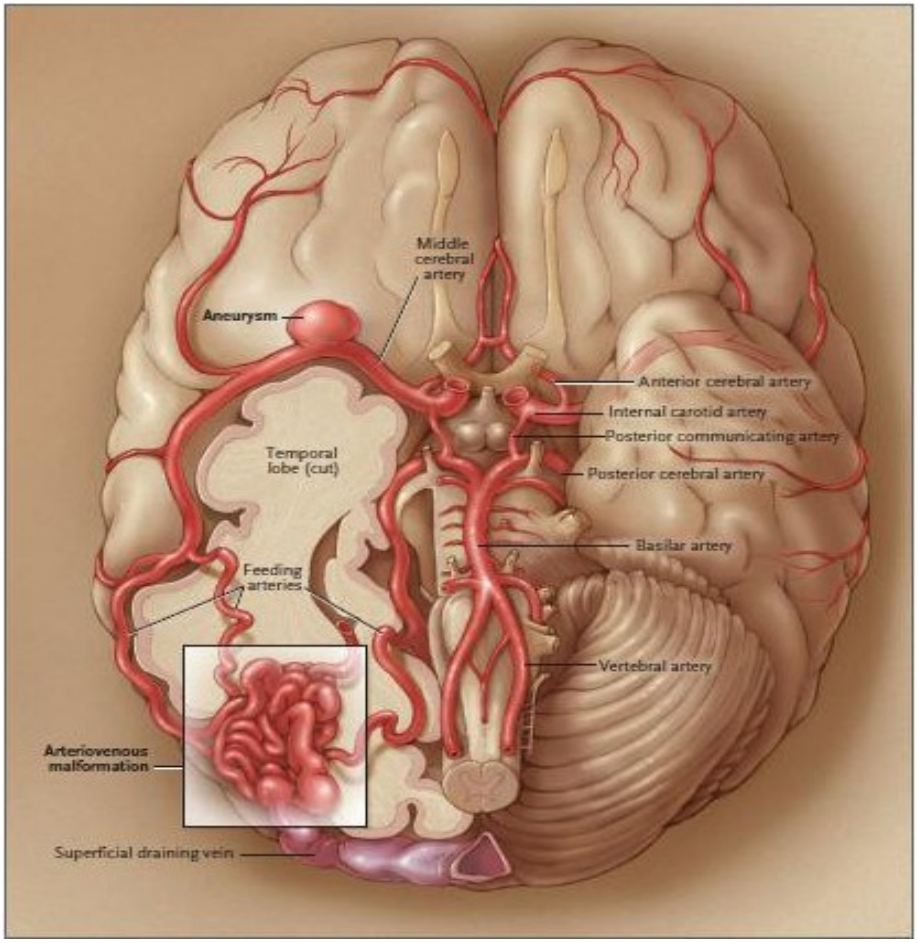


Figure : 4 Base of Brain Showing Arterio Venous malformation

The prevalence of arteriovenous malformation is estimated at approximately 0.01% of the general population, but reported rates range from 0.001% to 0.52%[61,62]. The lesions are thought to be congenital in origin. Although occasional cases are associated with other abnormalities (e.g., Osler-Weber-Rendu disease and the Sturge-Weber syndrome[63,64]).

The increased use of advanced imaging technologies has led to the identification of more arteriovenous malformations, including many that are asymptomatic[65].

The overall risk of haemorrhage of arteriovenous malformations is estimated at 2 to 4% per year[65].

After an initial haemorrhage, the annual risk of a subsequent haemorrhage has been reported to range from 4.5 to 34.4% [66-71].

In case series, reported rates of permanent weakness or paralysis, aphasia, and hemianopsia are 0 to 15%, and most report no deaths.

NEOPLASM CAUSING HAEMORRHAGIC STROKE :

1. Primary tomour in central nervous system
2. Metastatic Tomour
3. Leukaemia

HAEMATOLOGICAL DISORDER ASSOCIATED WITH STROKE INCLUDE :

1. Sickle-cell disease
2. Thrombocytopenia
3. Moyamoya Disease
4. Polycythemia.
5. Paraxysmal nocturnal haemoglobinuria.
6. Disorders of platelets & or blood coagulation.
7. Leukemias & Other Neoplasm.

DRUGS :

1. Warfarin
2. Amphetamines
3. Cocaine

4. Phenypropanolamine[20]

TEMPORAL PROFILE OF STROKE :

Depending upon the time course of the disease the strokes are classified as transient ischaemic attacks, reversible ischaemic neurological deficit, progressing stroke or stroke in evolution and completed stroke.

TRANSIENT ISCHAEMIC ATTACK (TIA) :

It is a sudden focal neurological dysfunction due to ischaemia of a portion of the brain and which resolves completely within 24 hours. Most of the episodes last usually less than 10 minutes but attacks lasting many hours are often documentedly associated with embolism. They may herald the oncoming vascular catastrophe. Previous history of TIAs are much more common but very rare in intracerebral haemorrhage. Recurrent attacks of the same pattern indicates that thrombosis is the possible cause, while multiple episodes of different pattern indicates embolism [4,5].

Symptoms of TIA vary depending upon the vascular territory involved. Symptoms due to involvement of carotid system are motor defects (weakness, paralysis or clumsiness) of the extremity or of both extremities of one side; sensory deficit (numbers of paraesthesia) of one or both extremities of one side, aphasia, amaurosis fugax and homonymous hemianopia. Symptoms due to involvement of vertebrobasilar system are motor defect of any combination of extremities upto quadriplegia or changing from one side to another in different attacks, sensory defects which are bilateral or changing from side to side in different attacks, bilateral loss of vision or

homonymous hemianopia and ataxia with or without vertigo.

REVERSIBLE ISCHAEMIC NEUROLOGICAL DEFICIT (RIND) :

It is a focal ischaemic event lasting longer than 24 hour but complete resolution of the deficit within three weeks. These episodes are also referred to as stroke with full recovery.

PROGRESSING STROKE OR STROKE IN EVOLUTION :

The temporal pattern of stroke is sometimes extended. Disability may increase by stepwise progression. Sudden deteriorations are interspersed with static intervals. Less commonly there is slow uninterrupted progression. The full extent of the patient's stroke conveys somewhat greater diagnostic uncertainty. A rapidly growing neoplasm or a subdural haematoma is more often a real differential diagnostic syndromes.

COMPLETED STROKE :

It is the stable focal ischaemic neurological deficit from which recovery occurs gradually over weeks and months.

SPECIFIC VASCULAR SYNDROMES :

Stroke syndromes are defined not only by their temporal profile but also by the vascular supply to the area of ischaemic brain.

INTERNAL CAROTID SYNDROME[7] :

The cervical portion of the carotid artery is a common site for both severe atheroma and thrombotic occlusion. About 30 percent of all occlusive lesions may be silent or asymptomatic. Symptoms include brief episodes of confusion, speech

difficulties, sensory paraesthesia with or without motor weakness on the opposite side. Ipsilateral amaurosis fugax, fleeting or semipermanent, alternating with or accompanied by a contralateral hemiplegia or sensory deficit, is pathognomonic of carotid artery syndrome, but it is noted in only 15 to 20 percent of the subjects.

The clinical manifestation may be similar to middle cerebral syndrome. Feeble carotid superficial temporal artery pulsation, dilated pupil and poorly pulsating retinal vessels (with or without optic atrophy) on the side of suspected carotid lesion and ocular or cervical bruits on the ipsilateral side may suggest the correct diagnosis.

In subjects with an old or silent occlusive carotid artery lesion on one side, a new lesion on the other side may prove catastrophic. Here, a physical findings of bilateral hemiplegia (quadriplegia) with coma can be mistaken for basilar artery syndrome.

MIDDLE CEREBRAL SYNDROME :

The cortical branches supply most of the lateral surface of the cerebral hemisphere, expect for the regions supplied by the anterior and posterior cerebral arteries. The areas of supply include the sensory-motor cortex, the motor and sensory speech centers, auditory area and visual radiation. The penetrating branches (lenticulo-striate arteries) supply the putamen, globuspallidus, genu and posterior limb of the internal capsule[2,5].

The clinical picture of middle cerebral artery occlusion is variable. Contralateral hemiplegia, hemianaesthesia with or without homonymous hemianopia and aphasia (dominant and non dominant) are the common outcome. However, occlusion of the

superior division results in contralateral hemiparesis with sensory deficit and expressive aphasia (Broca's aphasia) whereas Wernicke's aphasia (sensory aphasia) is frequent with lesion of the inferior division of dominant side. Monoplegic symptoms can occur with an occlusive lesion of a single cortical branch.

Several researchers have found that patients with Broca's aphasia were significantly younger than those with wernicke's aphasia. Occlusion of penetrating branches (lenticulo-striate arteries) has been blamed for a dense sensory motor hemiplegic syndrome (capsular hemiplegia), but significant sensory loss seldom occurs with such a occlusion, whereas pure motor hemiplegia is not uncommon.

ANTERIOR CHOROIDAL SYNDROME :

This artery supplies the posterior limb of the internal capsule, which carries the corticospinal and sensory fibres for the contralateral limb. This syndrome which represents a true "capsular hemiplegia" (dense hemiplegia hemianaesthesia and homonymous hemianopia), is rare[2,7].

ANTERIOR CEREBRAL SYNDROME :

This cortical branches mainly supply the medial superior surface of the frontal lobe and the parietal lobe upto the paracentral lobule. The penetrating branches supply the anterior limb of the internal capsule and part of the head of the caudate nucleus[7,8].

Anterior cerebral artery occlusion proximal to the anterior communicating artery, in subjects with a symmetrical circle of Willis, is frequently asymptomatic. Occlusion distal to the anterior communicating artery manifests itself by sensory motor paralysis of

the opposite lower extremity with mild weakness of the opposite shoulder. Mental changes, rectal and urinary incontinence, gait disturbances, apraxia and grasp and sucking reflexes may accompany the above findings.

Occlusion of an unpaired anterior cerebral artery (supplying both the hemispheres) results in a cortical type of paraplegia, with sphincter incontinence and a mental state in which the patient is alert but mute (akinetic mutism). Aphasia and hemianopia are never seen.

Occlusion of the penetrating branches and of the Heubner's artery is frequently blamed for ataxic tremor of the contralateral limbs (frontal ataxia) Apraxia, idiomotor dyspraxia of the limbs and gait may also be present.

POSTERIOR CEREBRAL SYNDROME [7,8]:

This artery supplies the medial and inferior aspects of the occipital and temporal lobes. Its branches also supply the mid-brain, cerebral peduncle and most of the thalamic and subthalamic regions.

Thrombotic occlusion of the posterior cerebral arteries is relatively rare. Contralateral homonymous hemianopia is a significant finding and this results from infarction of the primary visual area (calcarine cortex), the central vision is frequently spared, even in patients with bilateral disease (gun barrel vision). Other manifestations of visual dysfunction include illusory or distorted vision, visual object agnosia and various forms of dyslexia without dysgraphia. The pupillary reflexes are well preserved. Contralateral hemiplegia from a lesion of the cerebral peduncle (pedunclar hemiplegia)

and thalamic syndrome (Dejerine Roussy syndrome) may also be present. In the thalamic syndrome, there is varying degree of sensory loss to all modalities and spontaneous burning or agonizing pains are frequent (analgia dolorosa). Memory loss (amnesia) denotes a lesion of the medical temporal cortex. Contralateral involuntary choreoathetosis or ataxic tremors are rarely observed.

VERTEBRO-BASILAR SYNDROME [7,8] :

After traversing through the bony vertebral canals, both vertebral arteries unite intracranially to form the basilar trunk. Their short paramedian and long circumferential branches supply the entire brainstem, cerebellum and the vestibular apparatus. Ischaemic disorders, therefore manifest by episodes of vertigo, dizziness, diplopia, dysarthria, dysphasia, incoordination of gait and limbs and bilateral signs of sensory-motor deficit. Occipital headaches may be present.

Ipsilateral IIIrd nerve palsy (dilated pupil, ptosis and external strabismus) with contralateral hemiplegia (Weber's Syndrome) or with crossed cerebellar ataxia (Claude's syndrome) is diagnostic of mid-brain localization. Homolateral paralysis of the VIth and VIIth nerves (internal squint and facial palsy) with contralateral hemiplegia and hemianaesthesia (Millard-Gubler syndrome) is suggestive of a pontine lesion. Palatal Paralysis and ataxia of limbs, with impairment of posterior column sensation on same side of the body together with diminution of pain and thermal sense on the opposite limbs (Wallenberg's syndrome) indicate lateral medullary infarction[1,5,7].

Not infrequently quadriplegia with bilateral conjugate, lateral gaze palsy and mute

state with fully preserved consciousness has been described (Locked in syndrome) and suggests infarction of the basis pontis (sparing the tegmentum), from a midbasilar occlusion.

Occlusion of isolated cerebellar branches may produce dizziness, nausea, vomiting, nystagmus and appendicular or trunkal ataxia without sensory-motor deficit in any limb. Such a syndrome should be differentiated from cerebellar haemorrhage where emergency surgical decompression proves lifesaving.

LACUNAR SYNDROMES :

Occurs as a result of infarcts in the deep portions of cerebral hemispheres and brain stem due to occlusion of small perforating branches[4]. Pure motor hemiplegia is the most common lacunar syndrome due to an infarct in the posterior limb of internal capsule. There is no sensory deficit, visual field defect or aphasia.

ARTERITIS :

The clinical features of syphilitic and other forms of arteritis involving various cerebral arteries are in no way different from the neurovascular syndrome described under cerebral thrombosis. With specific serological tests, the diagnosis of meningovascular syphilis is not difficult.

CLINICAL PRESENTATION :

In stroke patients, symptoms and signs are in relation to the arterial circulation involved. The clinical assessment may be corroborated by diagnostic studies such as, computed tomography scanning or carotid angiography. However use of these and other

diagnostic techniques may be, the stroke syndrome continues to be characterised by the nature and time course of the patient's clinical findings. A careful analysis of history may help in differentiating stroke due to thrombosis, embolism or haemorrhage.

ONSET AND PROGRESSION OF STROKE :

Patient's activity during the onset of stroke indicates the possible cause of stroke. Intracerebral haemorrhage usually presents abruptly when the patient is awake and is prone to occur while he or she is engaged in physical exertion. Severe headache may precede the onset of haemorrhagic stroke by several hours and loss of consciousness is a usual feature.

In cerebral embolism the full blown picture of stroke evolves within a few seconds without any warning symptoms. In cerebral thrombosis the focal disability may occur at any time. The deficit evolves during the period of 1 or 2 days. Loss of consciousness occurs very rarely though drowsiness is common. Severe headache is unusual. Sometimes cerebral infarctions due to thrombosis and those due to embolism may be indistinguishable in terms of the time, course and nature of neurological findings.

FITS :

Epileptic fits, generalized or focal type may occur at the beginning or during the evolution of a stroke. Fits may occur in thrombosis, embolism or haemorrhage and it is not helpful in differentiating these conditions. Fits commonly occur in cerebral venous thrombosis.

SIGNS OF MENINGEAL IRRITATION :

Neck stiffness occurs when blood leaks into CSF in case of intracerebral haemorrhage or when there is rupture of saccular aneurysm producing subarachnoid haemorrhage. In our country since tuberculous meningitis is a common disease, it should be considered in stroke patients with signs of meningitis.

ARTERIAL PULSATIONS AND BRUIT :

A significant reduction in cerebral blood flow can occur due to atherosclerotic lesion narrowing the internal carotid artery or vertebral artery lumen by more than 60 to 80% of cross sectional area. Severe lesions of this kind are frequently seen in the internal carotid artery near the carotid sinus and in the vertebral channels coursing over the forehead and supra-orbital and supratrochlear arterial pulsations on the rim of the orbit suggest carotid occlusion. An additional sign of carotid occlusion is the presence of ocular bruit heard over the eye ball. Though vertebral artery is not accessible for palpation, its occlusion at its origin is suggested by a bruit heard over supraclavicular fossa.

OBSERVATION AND ANALYSIS

AGE DISTRIBUTION

The age distribution among 50 patients show that stroke in young adult are common in above 36 years of age.

Table – 2

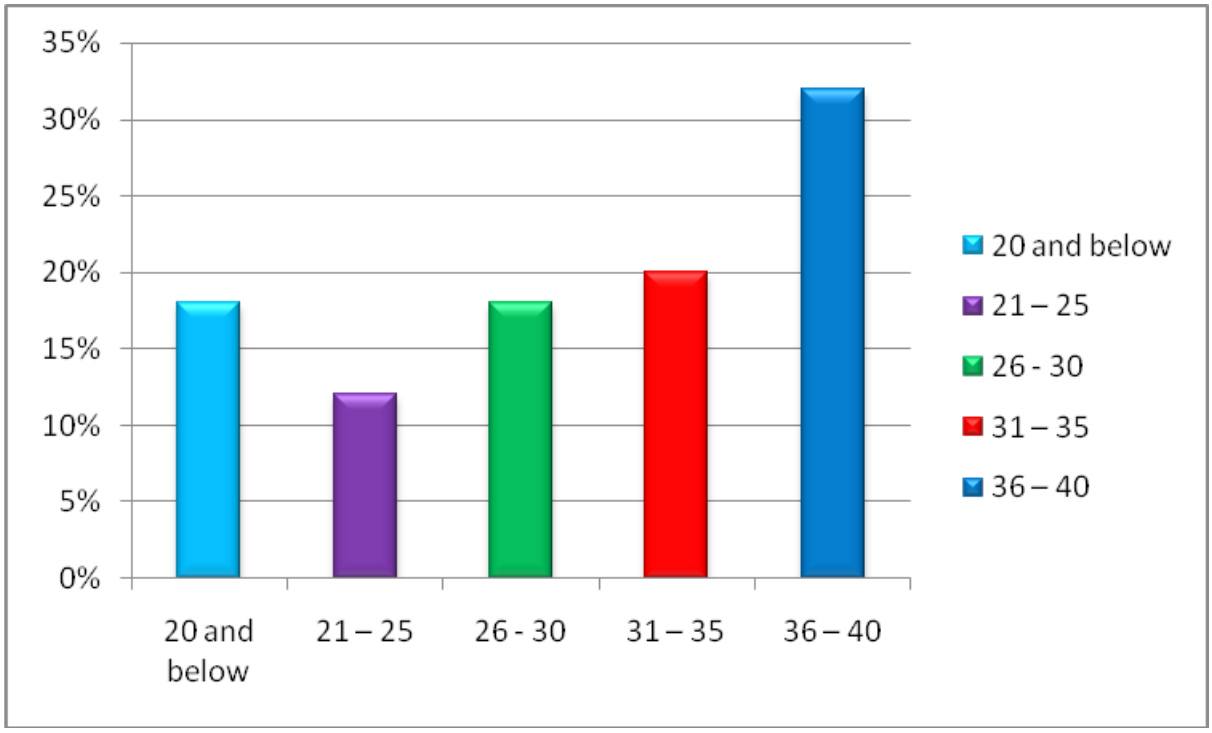
Age (in Years)	No of Cases	Percentage
20 and below	9	18%
21 – 25	6	12%
26 - 30	9	18%
31 – 35	10	20%
36 – 40	16	32%

30% of patients are less than 25 years of age

70% of patients are more than 25 years of age.

Age incidence of stroke in young adults is given in figure : 5

Figure 5 AGE INCIDENCE OF STROKE IN 50 YOUNG ADULTS



GENDER INCIDENCE OF YOUNG ADULTS WITH STROKE

IN 50 PATIENTS

Table – 3

Gender	No of Cases	Percentage
Male	32	64%
Female	18	36%
Total	50	

It is observed that the incidence of stroke in young adult is higher among male than female population. (fig : 6)

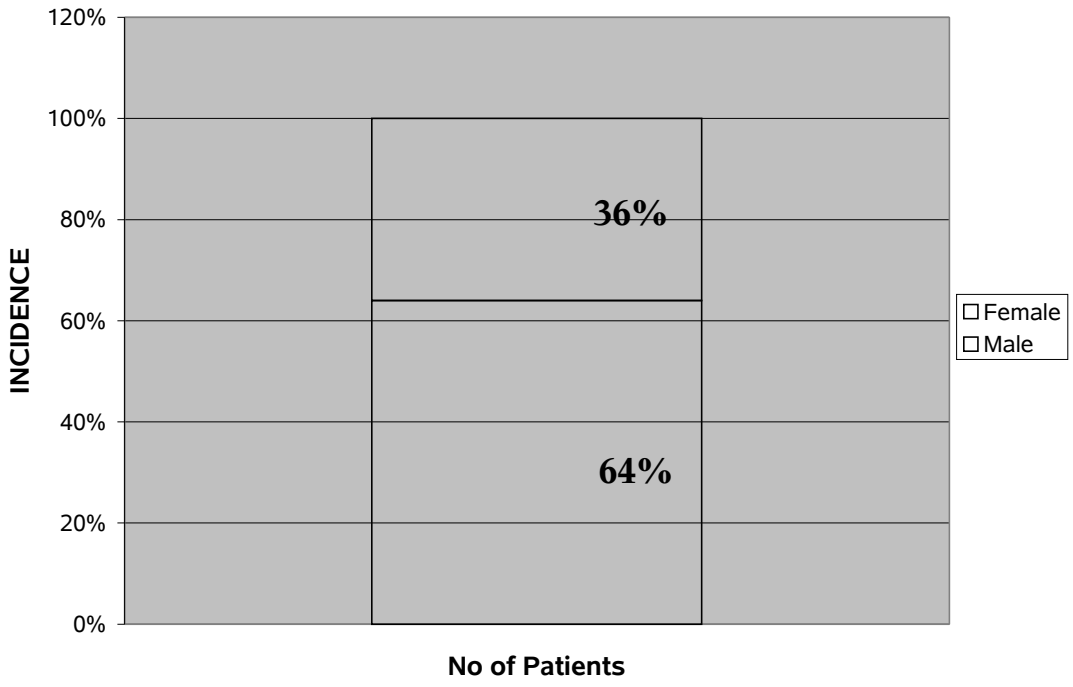


Figure : 6

PREVALENCE OF RISK FACTORS IN 50 YOUNG

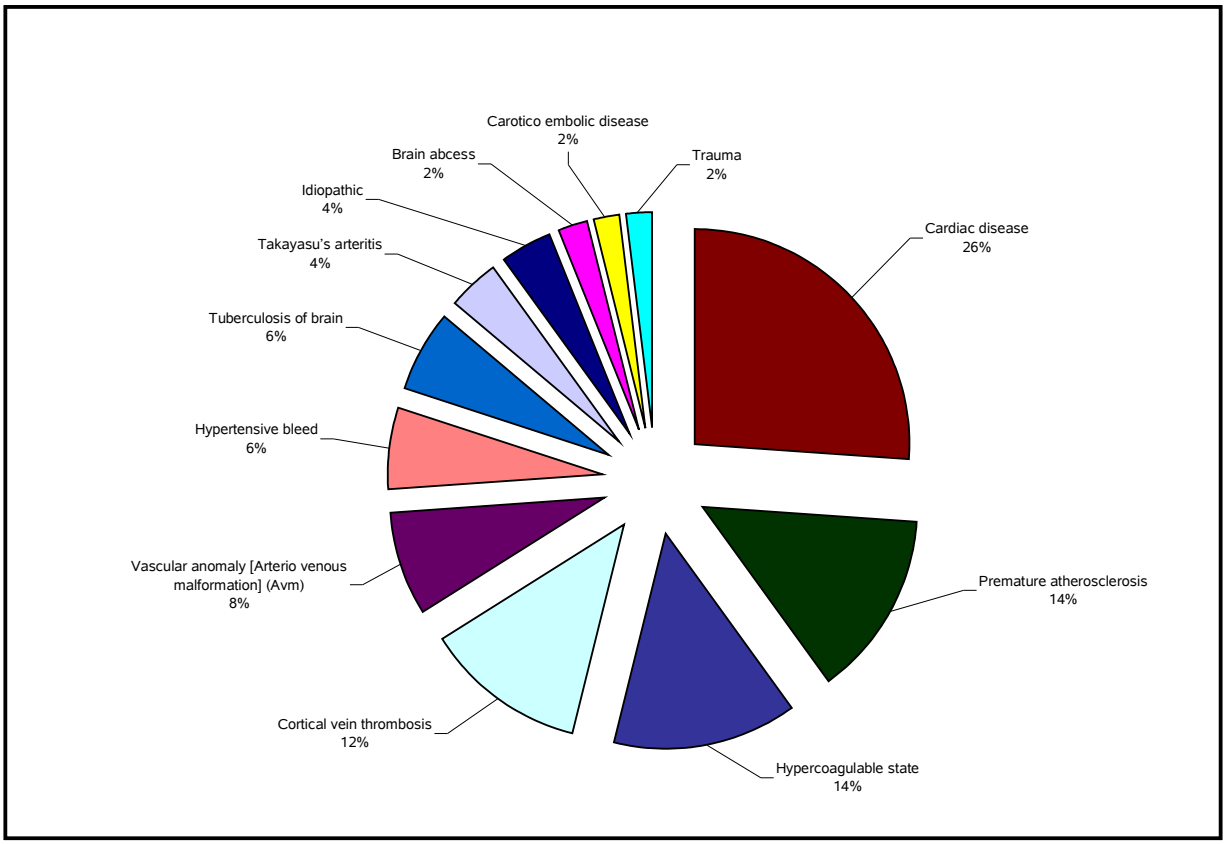
ADULTS WITH STROKE

Table – 4

Risk Factors	Number of cases	Percentage
Cardiac disease	13	26%
Premature atherosclerosis	7	14%
Hypercoagulable state	7	14%
Cortical vein thrombosis	6	12%
Vascular anomaly [Arterio venous malformation] (AVM)	4	8%
Hypertensive bleed	3	6%
Tuberculosis of brain	3	6%
Takayasu's arteritis	2	4%
Idiopathic	2	4%
Brain abcess	1	2%
Carotico embolic disease	1	2%
Trauma	1	2%

- Most of the patients (26%) who developed stroke are cardiac patients.
- Premature atherosclerosis is the risk factor for 14% of stroke patients.
- 4 Patients are having recurrent stroke. (Fig : 7)

Figure : 7 PREDOMINANT RISK FACTORS OF STROKE IN YOUNG ADULTS



TYPE OF STROKE (CLINICAL & CT SCAN & MRI EVALUATION)

Table – 5

Type of Lesion	Number of cases	Percentage
Infarction	33	66%
Haemorrhage	13	26%
Others	4	8%

Ischaemic – Infarction Stroke is more common than Haemorrhagic Stroke. (Fig : 8)

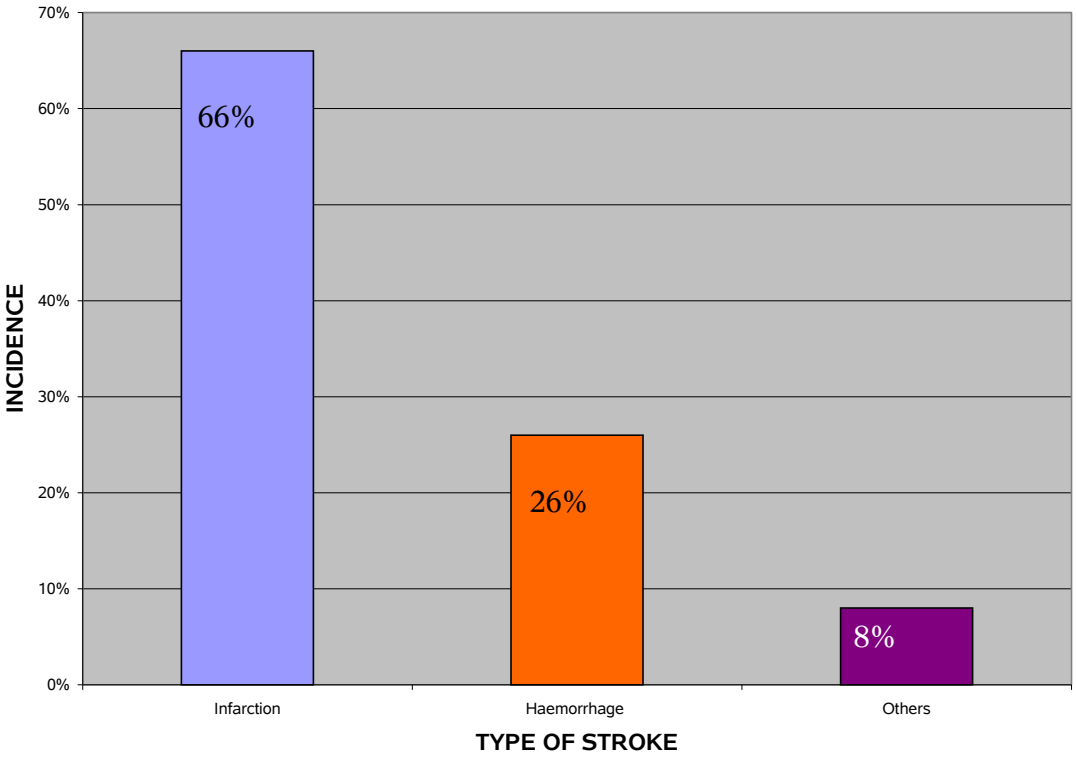


Figure : 8

ANALYSIS OF TREATMENT OUTCOME AND PROGNOSIS

TREATMENT OF STROKE INCLUDES :

1) Acute therapies designed to minimise brain infarction and life supportive measures.
2) Rehabilitative aimed at improving the quality of life.
3) Treatment aimed at preventing recurrence of strokes.

46 patients presented with completed stroke with fixed neurological deficits, admitted several hours to a few days after the onset of stroke, of which 33 patients had Brain Infarction & 13 patients had haemorrhagic stroke. Since there were no patients with stroke in evolution, anticoagulant therapy was not used to minimize brain infarction. Measures to reduce cerebral edema with intravenous mannitol, oral glycerol, frusemide and steroids were undertaken in fifty patients.

Anticoagulant therapy with intravenous heparin was started in 10 patients who had cardioembolic stroke with risk of recurrence. 3 patients with subacute bacterial endocarditis were treated with appropriate antibiotics. Anticoagulation therapy was not given to them.

3 patients with tuberculosis of Brain with stroke were treated with rifampicin, INH, ethambutol and pyrazinamide along with steroids. Blood pressure of the all hypertensive patients was controlled by oral administration of enalapril. 3 Hypertensive intracerebral bleed patients needed special attention to reduce blood pressure. Phenytoin

was used in 6 patient with focal seizures.

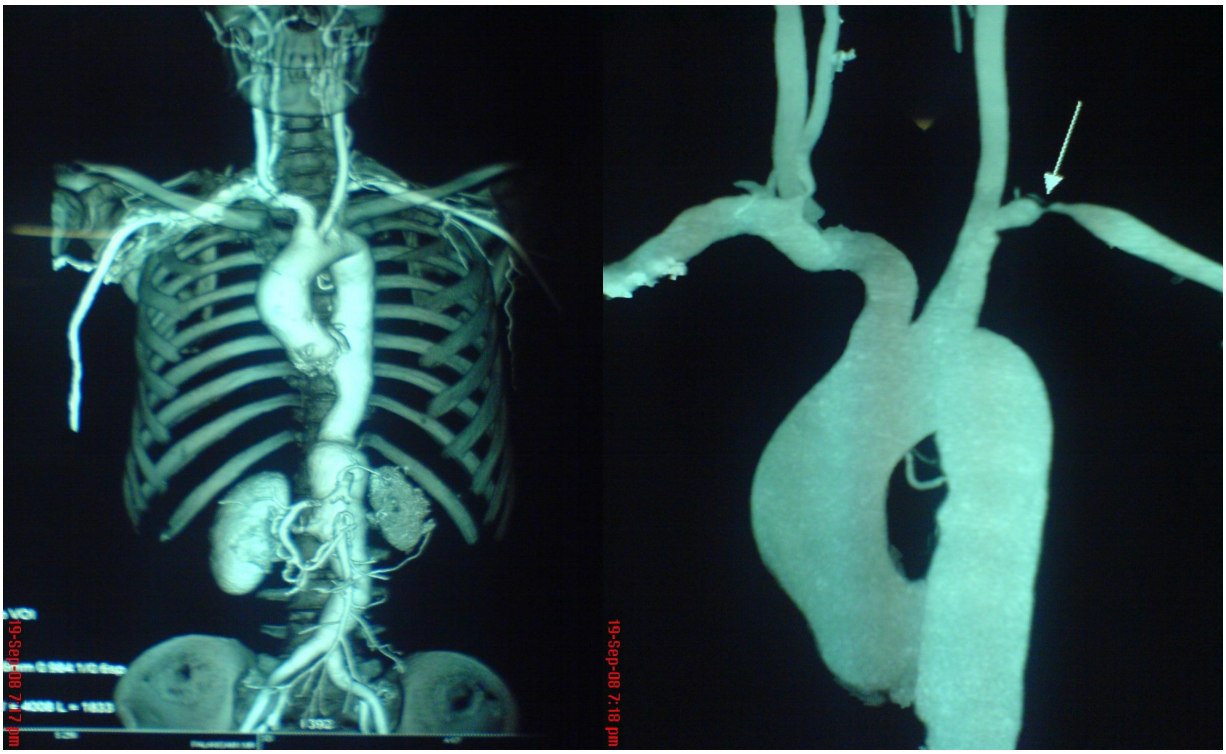
Antiplatelet therapy with low dose aspirin and dipyridamole was given in all patients who presented with thrombotic stroke.

Physical rehabilitative measures to maximize functional recovery were undertaken in all patients.

Prevention of recurrence of stroke was by long term antiplatelet therapy in those with thrombotic stroke, oral anticoagulant therapy in those with aseptic cardiogenic emboli and control of blood pressure in hypertensives. Prophylactic antibiotics before any minor or major surgical procedures in susceptible cardiac patients minimizes the risk of subacute bacterial endocarditis.

The prognosis of young adults with ischaemic stroke is better when compared with older patients. Out of 50 patients, 38 showed stable unchanged course over the first 7 days. 12 patients showed improvement in the first 7 days. None of the patients showed worsening after admission and no patient died.

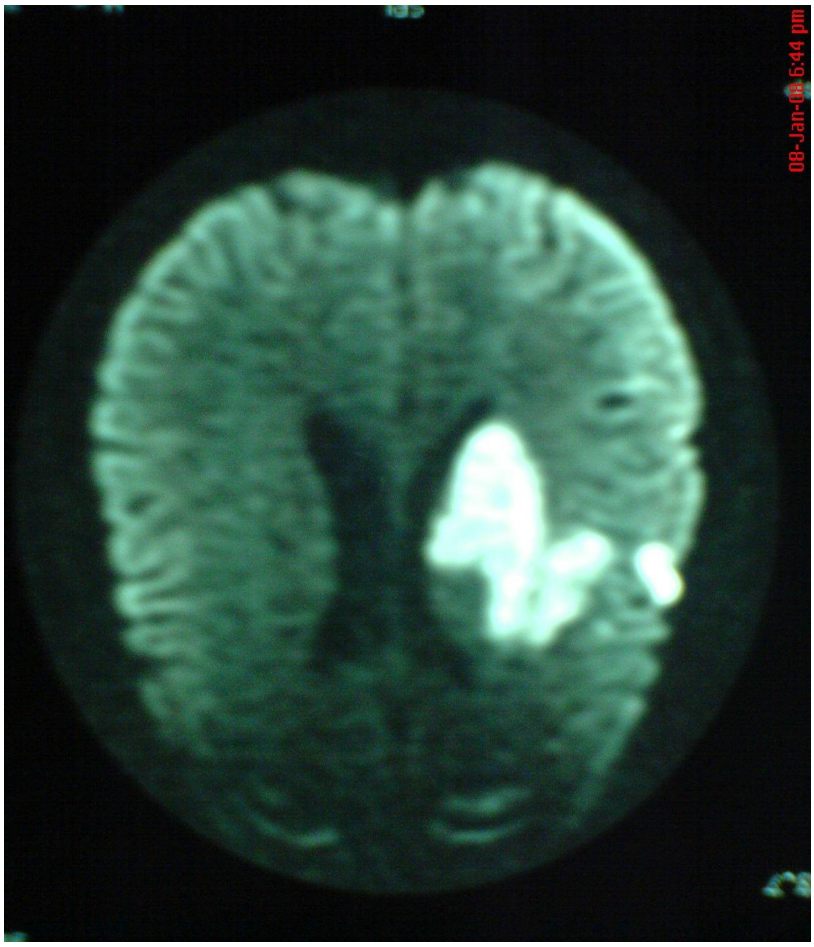
Figure : 9 - C.T. Angiogram Showing Total Occlusion Of (L) Subclavian Artery



(CASE NO : 20 MR.PALANISAMY - 32/M - IP.NO : 30617)

Figure : 10 - C.T. Scan Brain showing (L) MCA territory haemorrhage

(R) (L)



(Case No : 33 Mr. Seith Shake 30/M – IP NO.45623)

DISCUSSION

In this study, 50 young adults with stroke were included.

The incidence in men was 64% compared to 36% in women. This is in agreement with studies conducted by other centers.

In this study, the peak incidence of stroke was observed in the age group of 36-40 years which was 32%. It was 20% in the age group of 31-35 years. In a study conducted by P.M.Dalal et al[80], the incidence of stroke was maximum in the age group of 36-40 years which is similar to our study.

In our study 52% of patients were above the age of 30 years, compared is 48% patients in the above study.

Analysis of Risk factors :-

Hypertension :

Hypertension is one of the common risk factors associated with stroke. The incidence of hypertension was 18% in this study. In a study conducted by P.G.A.Sander Cock et al[81] this incidence was 32%.

Diabetes Mellitus :

Out of 50 patients studied, 2 patients had diabetes mellitus.

Hyperlipidemia :

7 Patients who had abnormal lipid profile were affected by premature atherosclerosis.

Transient Ischaemic attack :

Incidence of TIA was 10% in this study. In the study conducted by L.N.Jones the incidence of TIA was 14%.

Smoking :

In this study, out of 22 male patients, 20 were smokers. The remaining 18 patients were female non smokers. It became an important risk factor mainly due to premature atherosclerosis associated with it.

Cordiac Source of Emboli :

Cordiac source of emboli was observed in 26% of cases. In the study conducted by P.M.Dalal et al[80], the incidence of cordiac source of emboli was 20%. In our study, 10% of patients had atrial fibrillation. In the oxford shire community stroke project[81], the incidence of atrial fibrillation was 17%.

7 Patients had RHD, out of which 5 were in AF.

2 Patients had congenital heart disease (TOF).

Alcohol :

In this study, 32% of males were alcoholics, out of them one person had stroke followed by an alcoholic binge. Similar observation of stroke in young men after alcoholic binge, was noted by M.R.Wilkins, M.J.Kendall[82]. 8% of patients who presented with haemorrhagic stroke had arterio venous malformation in the cerebral vascular system. 4% of young patients with stroke had Takayasu's arteritis involving carotid & Subclavian artery.

Cervical arterial Bruit :

Cervical arterial bruit was observed in 2% of cases. In the Dalal et al[80] study, it was observed in 9% of cases.

Uncertain Causes :

Stroke due to undetermined causes was 6% in our study & in oxford shire community study[81], it was 5%. A definite risk factor was present in 70% of cases in our study but according to other observation it was 80%. More than one risk factors were noted in 30% of cases.

Serum Homocystein level was raised in 6 patients who had cerebral infarct. (Normal Range 3.7 – 13.9 μmol/L)

In our study, out of 50 patients, one had Antithrombin – III deficiency.

One patient developed metastatic brain abcess originated from congenital heart disease (TOF)

Types of Stroke :

In this study

- Ischaemic stroke was present in 66% of cases.
- Haemorrhagic stroke was present in 26% of cases but according to Harvard co-operative stroke registry study[12], the incidence was 15%.
- Cortical vein thrombosis was seen in 12% of cases & all the patients observed were female in post partum period. Similar observation was made by M.E. Yeolker[83].

According to Dalal et al study[80], which included 93 cases for a period of 5 years, the incidence of Ischaemic stroke was 80.60% and Haemorrhagic stroke was 12.50% when compared with this study, the incidence of Ischaemic stroke was 66% and haemorrhagic stroke was 26% in our study.

CONCLUSION

1. The incidence of stroke in young adults is more common in the age group between 36-40 years & males are more affected (64%) than females (36%).
2. Cardioembolic stroke is the commonest cause of stroke in young adults. Smoking is the most significant risk factor for stroke in young adults mainly due to the premature atherosclerosis associated with it.
3. Even though mitral value prolapse is consided as one of the major risk factors for stroke in young adults, in our study, out of 50 young patients who had stroke only 2 had MVP. But even in those cases MVP was not the cause for stroke.
4. TIA is experienced by 10% of patients.
5. All the patients who suffered from cortical vein thrombosis are females in puerperal period.
6. Among hypercoagulopathies causing stroke, hyperhomocysteinemia is the commonest followed by antithrombin III deficiency.
7. In about 6% of cases, no cause could be attributed.
8. The predominant mode of presentation of stroke is middle cerebral territory involvement. The commonest pathological type is (ischaemic stroke (66%)).
9. In vascular disease, takayasu's arteritis causes 4% of stroke & arteriovenous malformation causes 8% of stroke.

BIBLIOGRAPHY

1. NEUROLOGY IN CLINICAL PRACTICE – PRINCIPLES OF DIAGNOSIS AND MANAGEMENT – WALTER G.BRADLEY - 5TH EDITION.
2. BRAIN'S DISEASES OF THE NERVOUS SYSTEM - 11th EDITION.
3. PRINCIPLES OF NEUROLOGY – RAYMOND D.ADAMS - 8th EDITION.
4. HARRISION PRINCIPLES OF INTERNAL MEDICINE 17th EDITION – VOLUME 2.
5. DAVIDSON TEXT BOOK OF MEDICINE – 20th EDITION.
6. OXFORD TEXT BOOK OF MEDICINE – 3rd EDITION.
7. API TEXT BOOK OF MEDICINE - 8th EDITION – VOLUME 2.
8. GRAY'S TEXT BOOK OF ANATOMY – 37th EDITION
9. Martin A. Alpert a study of mvp with stroke. BMJ June 1993 vol.9 no.4 page 311, 312.
10. I.S.Anand, Y.Chandrasekar, prosthetic valve problem JAPI April 91 vol. 39 page 307.
11. Smoking/Donnan et al – smoking as a risk factor for cerebral ischaemia lancet 2 – 643.
12. Hypertension / Mohr JP Caplan LR et al – the Harvard prospective registry neurology 1978/28 754-762.
13. Nedea s – stroke due to cardiogenic embolism seminars in neurology 1986, 6, 277-280.

14. Mortality from cardiovascular diease among interregional migrants in England and Wales D P Strachan, D A Leon, and B Dodgeon BMJ 1995; vol 310:423-427.
15. Case – control study of oral contraceptives and risk of thromboembolic stroke: results from international study on oral contraceptives and health of young women Lothar A J Heinemann, Michael A Lewis, Margaret thorogood, Walter O Spitzer, Irene Guggenmoos-Holzmann, and Rudolf Bruppacher BMJ 1997; 315 : 1502 – 1504.
16. Case-control study of migraine and risk of ischamic stroke in young women Christophe Tzourio, Alain Tehindrazanarivelo, Serge Iglesias, Annick Alperovitch, Francois Chedru, Jacques D'Anglejan-chatillon, and Mariegermaine Bousser BMJ 1995; 310:830-833.
17. Relation between Troponin T concentration and mortality in patients presenting with an acute stroke : observational study P James, C J Ellis, R M L Whitlock, A R Mcneil, J Henley, and N E Anderson BMJ 2000; 320: 1502-1504.
18. Influence of total cholesterol, high density lipoprotein cholesterol, and triglycerides on risk of cerebrovascular disease : the Copenhagen city heart study E Lindenstrom, G Boysen, and J Nyboe BMJ 1994; 309:11-15.
19. Prognosis after transient monocular blindness associated with carotid-artery stenosis Benavente O., Eliasziw M., Streifer J.Y., Fox A.J., Barnett H.J.M.,

Meldrum H., the north american symptomatic carotid endarterectomy trial collaborators N EGL J Med 2001; 345: 1084-1090, Oct 11, 2001.

20. Phenylpropanolamine and the risk of haemorrhagic stroke Kernan W.N., Viscoli C.M., Broderick J.P., Brott T., Feldmann E., Morgenstern L.B., Wilterdink J.L., Horwotz R.I., N ENGL J MED 2000; 343: 1826-1832, Dec 21, 2000.
21. Caplan's stroke : a clinical approach Norris J.N. ENGL J MED 2000; 343:1899, Dec 21, 2000.
22. Pravastatin therapy and the risk of stroke Brett A. S., Meilof J.F., Uitdehaag B. M.I., Frechter O., White H.D., Simes R.J., Tonkin A.M., the lipid study group N ENGL J MED 2000; 343:1894-1896, Dec 21,2000.
23. Adverse cardiovascular and central nervous system events associated with dietary supplements containing ephedra alkaloids N ENGL J MED 2000; 343:1833-1838, Dec 21, 2000.
24. Stroke in patients with asymptomatic internal-carotid-artery stenosis Oldstein L.B., Howard G., Cohen S.N., Toole J.F., Tunick P.A., Karonzon I., Inzitari D., Eliasziw M., Barnett H.J.M. N ENGL J MED 2000; 343:1420-1421, Nov 9,2000. correspondence.
25. World Health Organisation the WHO stepwise approach to stroke surveillance – Geneva : WHO – 2005.
26. Hart R.G.Miller VT : cerebral infarction in young adult : a practical approach stroke 1983 : 14 : 110-114.

27. Grindal Ab, Cohen RJ, Saul RF, Taylor JR : cerebral infraction in young adult stroke 1978; 9 : 39-42.
28. Adams HP, Butler MJ, Biller J, Joffol GJ : non haemorrhagic cerebral infarction in young adults. ARCH NEUROL 1986; 43 : 793 – 796.
29. Radhakrishair K, Ashok PP, Sridharan R, Mousa ME : stroke in the young : incidence and pattern in Benghazi Libya. ACTA NEUROL SCAND 1986; 73:434-438.
30. Abraham J, Schetty G. Jose CJ : strokes in the young stroke 1971; 2 : 258 -267.
31. Hachinsky V, Norn's JW : the young stroke, in the acute stroke, Philadelphia, FA DAVIS, 1985, PP 141 – 164.
32. Snyder BD, Ramirez – Lassepas M : cerebral infarction in young adult : long term prognosis stroke 1980; 11: 149-153.
33. Stroke vol 19 no 8 August 1988; 982 – 986.
34. Estatisticas DA Saude 1972-1986. Lisboa, Portugal, Instituto National DE Estatistica, 1987.
35. Mudd SH, Shoubuf, Leuyhl, et al. the natural history of homocystinuria due to cystathione beta – synthase deficiency. AM I HUM GENET 1985; 37 : 1-3.
36. Hassan A, Hunt BJ, O Sullivan M, et al. Homocysteine is a risk factor for cerebral small vessal disease, acting via endothelial dysfunction. BRAIN 2004 : 127, 212-9.
37. Yaps, Boers GH, Wilcken B, et al. vascular outcome of patients with

Homocystinuria due to cystathione - bete synthase deficiency treated chronically, a multicantric observational study. ARTERIOSCLER THROMB VASC BIOL 2001; 21 : 2080 - 5.

38. Ringlestein, EB, Zeumerh, Argelou D : the pathogenesis of stroke from internal carotid artery occlusion. diagnostic and therapeutical implications stroke 1983 : 14 : 867 – 875.
39. Mohr JP, Gautier JC, Hier DB, stein rw : middle cerebral artery, in barnett hjm, mohr jp, stein bm, yatsu fm (eds) : stroke. new york, churchill, 1986, vol 1, p 409.
40. Sindermann F, Dichgons J, Bergleiter R : occlusion of the middle cerebral artery and its branches; Angiographic and clinical correlates, BRAIN 1969; 92 : 607 – 620.
41. (1) Basso A, Capitani E, Laiacona M, Luzzatti C : Factors influencing type and severity of aphasia, CORTEX 1980, 16 : 631 – 636.

(2) DE Renzi E, Faglioni P, Ferrari P : The influence of sex and age on the incidence and type of aphasia CORTEX 1980 : 16 : 627 – 630.

42. Obler Lk, Albert M, Good Glass H, Benson F : Aphasia type and aging. BRAIN LARG 1979; 6 : 318 – 322.
43. Kertesz A : Aphasia and associated disorders : taxonomy, localization and recovery, New York, GRUNE & STRATTON, 1979.
44. Eslinger P, Damasio AR : Age and type of aphasia in patients with stroke. J

Neurol Neurosurg Psychiatry 1981 : 44 : 377 – 381.

45. Blecic S, Bogousslavsky J. Stroke in young adults. In : Barnett HJM, Mohr JP, Stein BM, et al, eds. Stroke : pathophysilogy, diagnosis and management. 3rd ed. NEW YORK, NY : CHURCHILL LIVINGSTONE; 1998:1001-1012.
46. Wiebers DO, Feigin VL, Brown RD. Cerebrovascular disease in children and young adults. In : Handbook of stroke, Philadelphia, PA : LIPPINCOTT – RAVEN; 1997:237-243.
47. Stern BJ, Wityk RJ, Stroke in the young. In : Feldmann E, ed. Current diagnosis in neurology. St. Louis MO : MOSBY; 1994 : 34 – 40.
48. Biller J.strokes in the young in : Toole JF, ed. Cerebrovascular disorders. 5th ed Philadelphia, PA : LIPPINCOTT WILLIAMS AND WILKINS; 1999 : 283 – 316.
49. Mudd SH, Shovby F, Levy HL, et al. The natural history of Homocystinuria due to Cystathione beta-synthase deficiency. AM J HUM GENET 1985:37:1-31.
50. Hassan A, Hunt BJ, O'sullivan M, et al. Homocysteine is a risk factor for cerebral small vessel disease, acting via endothelial dysfunction, BRAIN 2004; 127:212-9.
51. Yap S, Boers GH, Wilcken B, et al. Vascular outcome of patients with Homocystinuria due to cystathione-beta synthase deficiency treated chronically; a multicenter observational study. ARTERIOSCLER THROMB VASC BIOL 2001; 21:2080-5.
52. Brown RD JR, Wiebers DO, Torner JC, O'Fallon WM.Frequency of intracranial

haemorrhage as a presenting symptom and subtype analysis: a population-based study of intracranial vascular malformations in OLMSTED COUNTRY, MINNESOTA. J NEUROSURG 1996; 85:29-32.

53. Apsimon HT, Reef H, Phadke RV, Popovic EA. A population-based study of brain arteriovenous malformation : Long – term treatment outcomes. Stroke 2002; 33:2794-800.
54. Crawford PM, West CR, Chadwick DW, Shaw MD, arteriovenous malformations of the brain; natural history in unoperated patients. J NEUROL NEUROSURG PSYCHIATRY 1986; 49:1-10.
55. Ondra SL, Troupp H, George ED, Schwab K. The natural history of symptomatic arteriovenous malformations of the brain: a 24 year follow-up assement. J NEUROSURG 1990;73:387-91.
56. Stapf C, Mast H, Sciacca RR, et al. Predictors of haemorrhage in patients with untreated brain arteriovenous malformation neurology 2006; 66:1350-5.
57. Stieg PE, Batjer HH, Samson D, eds. Intracranial arteriovenous malformations NEW YORK : Informa, 2007.
58. Perret G, Nishioka H. Report on the cooperative study of intracranial aneurysms and subarachnoid haemorrhage . Section vi. Arteriovenous malformations: an analysis of 545 cases of cranio-cerebral arteriovenous malformations and fistulae reported to the cooperative study. J NEUROSURG 1966;25:467-90.
59. Brown RD JR, Wiebers DO, Torner JC, O'Fallon WM. Incidence and prevalence

of intracranial vascular malformation in Olmsted county, Minnesota, 1965 to 1992. Neurology 1996;46:949-52.

60. Gross CR, Kase CS, Mohr JP Cunningham SC, Baker WE. Stroke in south Alabama : incidence and diagnostic features a population based study. Stroke 1984;15:249-55.
61. Mccormick WF. Pathology of vascular malformations of the brain. In: Wilson CB, Stein BM, eds. Intracranial arteriovenous malformations. Baltimore : Williams & Wilkins, 1984:44-63.
62. Al-Shahi R, Fang JS, Lewis SC, Warlow CP. Prevalence of adults with brain arteriovenous malformations : A community based study in Scotland using capture – recapture analysis. J NEUROL NEUROSURG PSYCHIATRY 2002; 73:547-51.
63. Kikuchi K, Kowada M, Sasajima H. Vascular malformations of the brain in hereditary haemorrhagic telangiectasia (Rendu-Osler-Weber disease). SURG NEUROL 1994;41:374-80.
64. Laufer L, Cohen A. Sturge-Weber syndrome associated with a large left hemispheric arteriovenous malformation. PEDIATR RADIO 1994; 24: 272-3.
65. Brown RD JR, Wiebers DO, Forbes GS. Unruptured intracranial haemorrhage in patients with cerebral arteriovenous malformation. STROKE 1998;29:931-4.
66. Hartmann A, Mast H, Mohr JP, et al. Morbidity of intracranial haemorrhage in patients with cerebral arteriovenous malformation. STROKE 1998;29:931-4.

67. Mast H, Young WL, Koennecke HC, et al. Risk of spontaneous haemorrhage after diagnosis of cerebral arteriovenous malformation. LANCET 1997;350:1065-8.

68. Redekop .G, Terbrugge K, Montanera W.Willinsky R.Arterial aneurysms associated with cerebral arteriovenous malformations; classification, incidence, and risk of haemorrhage , J NEUROSURG 1998;89: 539-46.

69. Pritz MB. Ruptured supratentorial arteriovenous malformations associated with venous aneurysms. ACTA NEUROCHIR (WIEN) 1994;128:150-62. [ERRATUM, ACTA NEUROCHIR (WIEN) 1994;131:314].

70. Spetzler RF, Hargraves RW, Mccormick PW, Zabramski JM, Flom RA, Zimmerman RS. Relationship of perfusion pressure and size to risk of haemorrhage from arteriovenous malformations. J NEUROSURG 1992;76:918-23.

71. Lasjaunias P, Piske R, Terbrugge K, Willinsky R.Cerebral arteriovenous malformations (C. AVM) and associated arterial aneurysms (AA) : Analysis of 101 c. AVM cases, with 37 AA in 23 patients. ACTA NEUROCHIR (WIEN) 1988; 91:29-36.

72. The international study of unruptured intracranial aneurysm investigators. Unruptured intracranial aneurysms — risk of rupture and risks of surgical intervention. N ENGL J MED 1998;339:1725-33. [ERRATUM, N ENGL J MED 1999;340:744.]

73. Wiebers DO, Whisnant JP, Huston J, et al. Unruptured intracranial aneurysms: natural history, clinical outcome, and risks of surgical and endovascular treatment. LANCET 2003;362:103-10.
74. Schievink WI. Intracranial aneurysms. N ENGL J MED 1997;336:28-40. [ERRATUM, N ENGL J MED 1997;336:1267.]
75. Endovascular repair of intracranial aneurysms. Medtech Insight. Vol. 7. No. 2. February 2005:47-52 (NEWSLETTER).
76. Wijdicks EF, Kallmes DF, Manno EM, Fulgham JR, Piepgras DG. Subarachnoid haemorrhage : Neurointensive care and aneurysm repair. MAYO CLIN PROC 2005;80: 550-9.
77. N ENGL J MED 2006;355, 9 Aug 31, 2006 AV Malformation of brain.
78. Av malformation of brain.N ENGL J MED 326, 26 June 28, 2007.
79. Av malformation of brain.N ENGL J MED 2005, 352 : 146 – 53.
80. P.M.Dalal, K.P.Dalal, A.C.Vyas / Stroke in young / in west central India, JAPI June 1998 vol.96, page 365.
81. P.G.A.Sander Cock, C.P.Warloo – Predisposing factors for CCA – the Oxford shire community stroke project, BMJ June 1989 vol-5 No.4 page 257.
82. Mr.Wilkins, M.J.Kendall. Stroke affecting young men after alcoholic binge. BMJ Jan 1986 Vol-1 No.11 page 869.
83. M.C.Yeolker CVT. JAPI May 1996 Vol 38, page 321.

ANNEXURES

PROFORMA

STROKE IN YOUNG ADULT

Name :	Age :	Sex :
Address :	Ward :	I.P.No .:
		D.O.A .:
		D.O.D. :

History of present illness :

HISTORY OF THE RISK FACTORS :

Hypertension / Diabetes Mellitus / Rhematic Heart disease / smoking / Alcohol intake / Previous History of TIA / RIND / Migraine / Drug intake / Head injury / other treatment.

FAMILY HISTORY :

Stroke / Hypertension / Diabetes mellitus

CLINICAL EXAMINATION :

GENERAL EXAMINATION :

Built	Anaemia	Jaundice
Clubbing	Pedeledema	

Gen-lymphadenopathy

FEATURE SUGGESTIVE OF CONGENITAL HEART DISEASE :

(Marfan / Syndactyly / Polydactyly / Hypertelurism / Lowset ears / Low hairline / Chest anomaly)

FEATURE SUGGESTIVE OF DYSLIPIDEMIA :

(Obesity / Arcus Juvinalis / Arcus Lipidus / Tenden Xanthoma / Xanthelasma)

Vital Signs :

Pulse rate Blood pressure

SYSTEMIC EXAMINATION :

CVS : RS

Carotid artery Bruite ABDOMEN

CNS EXAMINATION :

2. Higher function
3. Cranial Nerve examinations :
4. Examination of spinomotor system :
    a) Bulk of the muscle
    b) Tone of the Muscle
    c) Muscle power
    d) Reflexes
5. **Examination of sensory system :-**
6. **Examination of cerebellar system :-**

7. **Signs of increased intracranial tension**
8. **Examination of spine and cranium**
9. **Signs of Meningeal irritation**

LABORATORY INVESTIGATION :

1. Urine Examinations
2. Blood Examination

   Total leucocyte count

   Differential count

   Platelet count

   Bleeding time

   Clotting time

   Hb%

   ESR

   Sugar

   Lipid profile

   Blood culture

   Liver function test

   Renal function test

   VDRL :

   ELISA for HIV

   Sr. Electrolytes

CRP

3. ECG
4. CT Brain
5. ECHO
6. MRI Brain
7. Others

(MR Angiogram, MR Venogram

Sr.Homocystein Level

Anti thrombin III Level

ANA

Carotid Doppler study

TEE)

MASTER CHART

S No	Name	Age in yrs	Gender	IP.No	Clinical Presentation	Risk Factors				H/O TIAPrevious	ECG	ECHO	CT Brain	MRI Brain/ MRV/MRA	Diagnosis / Remarks
						AbuseSubstance	SHT	DM	RHD/CHD						
1.	Andal	37	F	2767	(L) Focal Seizure, (L) Hemiplegia				RHD	√	Normal sinus rhythm	MS-moderate MRmild/LVE/ smoke in LA+	(R) MCA territory infarct	(R) MCA territory infarct	Cardio embolic Stroke (RHD)
2.	Farija	36	F	2800	(L) Hemiplegia				RHD		RAE / AF	MS-Severe / Mild As / no Clot	(R) MCA territory infarct	(R) MCA territory infarct	Cardio embolic Stroke (RHD)
3.	Yogeswaran	40	M	3641	(L) Focal Seizure, Loc (L) Hemiplegia	AlcoholTobacco					Sinus bradycar dia	Normal Study	Recent hemorrhagic infarct in (R) frontoparietal region	Chronic thrombosis of anterior portion of superior sagittal sinus, (L) transverse sinus, Hemorrhage in (R) frontal lobe	C.V.T
4.	Ramakrishnan	22	M	4512	(R) hemiplegia Broca's aphasia				RHD		WNL	AR Severe / Bicuspid AV / vegetation in both cups +	(L) MCA territory infarct	(L) MCA territory infarct	Cardioembolic Stroke, (IE)

5.	Ananthi	27	F	8261	H/A (R) Hemiplegia & global aphasia						WNL	MVPs	Hemorrhage (L) thalamoganglionic area	Spasm of the circle of Hills, both MCA & ACA and (L) PCA and their branches & small aneurysm in ACA	Vascular anomaly (AVM)
6.	Thangaraj	35	M	8989	(R) focal seizure H/A (R) Hemiplegia	AlcoholTobacco					WNL	Normal Study	Hyperdence superior sagictal sinus	Partial thrombosis of the entire superior sagittal and transverse dural venous sinuses.	CVT/ Hyper Homocys-teinemia
7.	Sakthi	32	M	12617	(L) Hemiplegia						WNL	Normal Study	(R) MCA territory infarct	(R) MCA territory infarct	Idiopathic
8.	Ushadevi	33	F	12671	(L) Hemiparesis						WNL	Normal Study	(R) MCA territory infarct	(R) MCA territory infarct	Idiopathic
9.	Mallika	19	F	12803	(R) Hemiplegia				CHD (TOF)		(R) axis deviation RVH	VSD, pulmonary Stenosis, RVH	Brain abcess in (L) temporo parietal region	Brain abcess in (L) temporo parietal region, Surronding edema	Brainabcess with CHD
10.	Leo Peter	31	M	17610	(R) Hemiplegia Broca's aphasia	Tobacco Alcohol					RVH, Strain Pattern	Concentric LVH	(L) MCA territory infarct	(L) MCA territory infarct	Premature atherosclerosis

11.	Thulasimani	32	F	18122	(R) Hemiplegia, Global Aphasia						WNL	Normal Study	(L) MCA Territory Infarct	Near total occlution of proximal (L) CC, (L) SC Artery. Diffuse wall thickening of Aortic arch,	Takayasu's Arteritis
12.	Sivamani	40	M	20277	H/A, Vomitting, (R) Hemiplegia	√					NSR/ LVH	Concentric LVH	Brainstem hemorrhage- Ventricular extention	Brainstem hemorrhage- Ventricular extention	Hypertensive ICH
13.	Mahendran	24	M	20437	(L) Focal Seizure (L) Hemiplegia						WNL	Normal Study	(R) MCA Territory Infarct	Thrombosis (R) ICA, (R) MCA territory infarct. Carotid Doppler Study :- Thrombosis (R) ICA	Carotico Embolic Stroke
14.	Krishnan	38	M	22060	(R) Hemiplegia	Tobacco Alcohol	√				Sinus Brady-cardia	Concentric LVH	(L) MCA Territory Infarct	(L) MCA Territory Infarct	Premature Atherosclerosis
15.	Malaisamy	39	M	22142	(R) Focal Seizure (R) Hemiparesis, Global Aphasia	Tobacco Alcohol		√			WNL	Normal Study	(L) MCA Territory Infarct	(L) MCA Territory Infarct	Hyper coagulable State
16.	Rajkumar	17	M	22692	Fever, H/A, (L) Hemiplegia						Sinus Brady-cardia	MVPs MR-Mild	Tuberculoma in (R) Temporo Parietal Area	Tuberculoma in (R) Temporo Parietal Area	CNS – Tuber Culosis (Tuberculoma)

17.	Saravanan	30	M	23724	(R) Hemiplegia Broca's Aphasia	Tobacco Alcohol		√			WNL	Normal Study	(L) MCA Territory Infarct	(L) MCA Territory Infarct	Premature Atherosclerosis
18.	Lakshmi	39	F	25289	(R) Hemiplegia				CHD (TOF)		(R) Axis deviation RVH	Large VSD, Infundibular PS, RVH,Vegetation +	Infarct in (L) Coronaradiata (L) Lentiform nucleus.	Infarct in (L) Coronaradiata (L) Lentiform nucleus.	Cardioembolic Stroke (CHD)
19.	Perumalsamy	20	M	26428	Fever, (R) Hemiplegia				RHD		WNL	AV-thickened mild MVPs, Mild MR, Small Vegitation in MVcusp +	(L) MCA Territory Infarct	(L) MCA Territory Infarct	Cardioembolic Stroke (RHD)
20.	Palanisamy	32	M	30617	H/A, Vomiting (L) Hemiplegia	Tobacco Alcohol	√			√	(L) axis deviation LVH-Strain	Global Hypokinesia of LV	ICH – (R) ganglio Capsular Region	Total occlution of (L) CC & (L) SC artery. Abdominal Aortic Aneurycm	Takayasu's Arteritis
21.	Mariammal	38	F	30707	(L) Hemiplegia				RHD		AF	Tight Mitral Stenosis	(R) MCA Territory Infarct	(R) MCA Territory Infarct	Cardioembolic Stroke (RHD/AF)
22.	Abuthakir	33	M	31681	(L) Hemiplegia						WNL	Normal Study	(L) Cerebellar hemisphere, Vermis, Pontine-Infarct	(L) Cerebellar hemisphere, Vermis, Pontine-Infarct	Hyper Coagulable State (Antithrombin III deficiency)

23.	Murugan	22	M	32412	(R) Hemiplegia	Tobacco Alcohol	√	√			WNL	Normal Study	(L) MCA Territory Infarct	(L) MCA Territory Infarct	Premature Atherosclerosis
24.	Navamani	39	M	32419	(R) Hemiplegia	Tobacco Alcohol	√				WNL	Normal Study	(L) MCA Territory Infarct	(L) MCA Territory Infarct	Premature Atherosclerosis
25.	Selvam	39	M	33436	(R) Hemiplegia	Tobacco Alcohol	√				WNL	Normal Study	(L) MCA Territory Infarct	L) MCA Territory Infarct	Premature Atherosclerosis
26.	Veeran	35	M	34712	(L) Hemiplegia	Tobacco Alcohol					WNL	Normal Study	Fracture of (R) Frontal & Parietal bone & ICH	Fracture of (R) Frontal & Parietal bone & ICH	Traumatic Cerebral Bleed
27.	Rangan	39	M	39953	(R) Hemiplegia & Broca's Aphasia		√				Sinus brady cardia LVH	Concentric LVH, Diastolic dysfunction +	ICH in basal ganglion, intraventricular extension	ICH in basal ganglion, intraventricular extension	Hypertensive ICH
28.	Mariya Jenifer	13	F	41243	H/A, Fever (R) Hemiplegia						Sinus arrhyth-mia	Normal Study	Hypodense lesion in (L) Perasagittal region, midline shift	Thrombosis of (L) Transverse Sinus	C.V.T

29.	Radha	23	F	41363	(R)Hemiplegia				RHD	√	AF	Prosthetic Mitral Value Restenosis, MR	(L) Capsuloganglionic infarct, old (L)MCA infarct	(L) Capsuloganglionic infarct, old (L)MCA infarct	Cardioembolic Stroke (RHD/AF)
30.	Manikandan	19	M	41831	(L) Hemiplegia	Tobacco					WNL	Normal Study	(R) MCA Territory Infarct	(R) MCA Territory Infarct	Premature Atherosclerosis
31.	Mani	14	M	42110	H/A, Fever (L) Hemiplegia						WNL	Normal Study	Diffuse Cerebral Edema & Hydrocephalus	Diffuse Cerebral Edema, obstructive Hydrocephalus	CNS Tuberculosis (T.B.M)
32.	Neela	29	F	45610	(R) Hemiplegia & Broca's Aphasia	OCP					WNL	Normal Study	(L) MCA Territory Infarct	(L) MCA Territory Infarct	Idiopathic
33.	Seith Shake	30	M	45623	(R) Hemiplegia & Wernicke's Aphasia	Tobacco Alcohol	√				LVH	Concentric LVH	(L) MCA Territory Hemorrhage	(L) Basal Ganglionic & Capsular Hemorrhage No AVM	Hypertensive ICH
34.	Kumar	20	M	45780	H/A, Vomiting, altered sensorium (R) Hemiplegia						WNL	Normal Study	Hemorrhage in III, IV both lateral ventricle, minimal obstructive hydrocephalus	AVM in (L) lateral ventricle, arterial feeders from the PCA & MCA	A.V.M

35.	Indirani	32	F	47664	(R)Hemiplegia		√			√	WNL	MS-Moderate MR-Mild LAE Smoke in LA+	(L) MCA Territory Infarct, Old Infarct in (L) Temporo-parietal region	(L) MCA Territory Infarct, Old Infarct in (L) Temporo-parietal region	Cardioembolic Stroke (RHD)
36.	Sakkariyammal	37	F	50857	(R) Focal Seizure & (R) Hemiplegia						WNL	Normal Study	Hemorrhagic Infarct (R) Parieto-temporal & (L) Parietal Region	Thrombosis of the Superior sagittal, inferior sagittal and (R) transverse sinuses & hemorrhagic venous infarct - (L) parietal, (R) temporal region	C.V.T
37.	Lakshmi	30	F	51102	(R) Focal Seizure / (R) Hemiplegia						Sinus Tachy-cardia	Normal Study	(L) MCA Territory Hemorrhagic Infarct	(L) Temporoparietal Hemorrhagic Infarct & Thrombosis of (L) Transverse Sinus	C.V.T
38.	Mehraj Begam	36	F	52367	(L) Hemiplegia				RHD		AF	Tight MS LA Clot +	(R) MCA Territory Infarct	(R) MCA Territory Infarct	Cardioembolic Stroke (RHD / AF)
39.	Bakri	24	M	52610	H/A, Neckpain (L) Hemiplegia,	Tobacco Alcohol					WNL	Normal Study	ICH in (R) MCA Territory	Aneurysm in (R) MCA & Occipital AVM	A.V.M

40.	Rajan	29	M	52733	(R) Hemiplegia						WNL	Normal Study	(L) MCA territory infarct	(L) MCA territory infarct	Hyper coagulable State & Hyperhomocy-steinemia
41.	Devaraj	35	M	53789	(L) Hemiplegia	Tobacco Alcohol					WNL	Global hypokinesia of LV, mild (L) Ventricle dilatation, dilated cardio myopathy	(R) MCA territory infarct	Acute infarct in (R) frontal, fronto-temporal, parietal region & lentiform nucleus.	Cardioembolic stroke (Alcoholic C.M.P)
42.	Chinnaranga samy	39	M	53885	(L) Hemiplegia	Tobacco Alcohol					WNL	Patent foramen ovale	Normal study	(R) MCA territory infarct	Cardioembolic stroke (Patent formen ovale)
43.	Velmurugan	28	M	54487	(R) Hemiplegia & Broca's aphasia	Tobacco Alcohol					WNL	Normal Study	(L) MCA territory infarct	(L) MCA territory infarct	Hyper coagulable State & Hyperhomocy-steinemia
44.	Karupusamy	37	M	55113	(R) Hemiplegia & Broca's aphasia	Tobacco					WNL	Normal Study	(L) MCA territory infarct	(L) MCA territory infarct	Hyper coagulable State & Hyperhomocy-steinemia
45.	Sankar Kumar	27	M	59803	H/A (R) Hemiplegia	Tobacco				√	NSR LVH	Normal Study	(L) MCA territory infarct (Lacunar infarct)	(L) MCA territory infarct (Lacunar infarct)	Hyper coagulable State & Hyperhomocy-steinemia

46.	Muniyandi	19	M	61617	H/A (R) hemiparesis						WNL	Normal Study	Diffuse cerebral edema & Hydrocephalus	Diffuse cerebral edema & Hydrocephalus	CNS – Tuber Culosis (T.B.M)
47.	Yamuna	19	F	63647	(R) hemiplegia		PIH				WNL	Normal Study	ICH in (L) temporo parieto occipital region	Thrombosis of (L) transverse sinus & (L) sigmoid sinus	C.V.T
48.	Yesu	39	F	64566	(R) hemiparesis						WNL	Normal Study	(L) MCA territory infarct	Focal stenosis-distal segment of (L) MCA & watershed infarct-fronto – parieto - temporo - occipital region.	Vascular Malformation
49.	Senthil Kumar	30	M	64701	(R) hemiplegia & Wernicke's aphasia				RHD		AF	MS severe MR-trivial PHT severe LA clot +	(L) MCA territory infarct	(L) MCA territory infarct	Cardioembolic storke (RHD/AF)
50.	Pavithra	23	F	65412	(R) hemiparesis & global aphasia				RHD		LAE, RVH	Tight MS, LA clot +	(L) MCA territory infarct	(L) MCA territory infarct	Cardioembolic storke (RHD)

ABBREVIATION

H/A- Headache , F – Female R-Right

M – Male L-Left

AVM – Arterio Venous malformation

CVT – Cortical Vein thrombosis

IE – Infective Endocarditis

ICH – Intra Cerebral Hemorrhage

TBM – Tuberculous Meningitis

CC – Common Carotid

SC – Subclavian

SHT – Systemic Hypertension

DM – Diabetes Mellitus

RHD – Rhematic Heart disease

CHD – Congenital Heart disease

WNL – Within Normal Limit

LVH – Left Ventricular Hypertrophy

RVH – Right Ventricular Hypertrophy

LA – Left Atrium

ACA – Anterior Cerebral Artery

OCP – Oral Contraceptive Pills

MCA – Middle Cerebral Artery

LOC – Loss of Consciousness

TIA – Transient Ischaemic Attack

ECG – Electro Cardiogram

RAE – Right Atrial Enlargement

AF – Atrial Fibrillation

MS – Mitral Stenosis

AS – Aortic Stenosis

MR – Mitral Regurgitation

MVPs – Mitral Valve Prolapse Syndrome

MV – Mitral Valve

MRI – Magnetic resonance imaging

MRA – Magnetic resonance Angiogram

MRV - Magnetic resonance Venogram