DISSERTATION ON

CLINICAL AND RADIOIMAGING STUDY IN POSTERIOR CIRCULATION STROKE

Submitted in partial fulfilment of

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CERTIFICATE

This is to certify that this dissertation entitled **"CLINICAL AND RADIOIMAGING STUDY IN POSTERIOR CIRCULATION STROKE"** submitted by **Dr. VENKATESH KUMAR A** appearing for M.D. Branch I General Medicine Degree examination in March 2007 is a bonafide record of work done by him under my direct guidance and supervision during the academic year 2004-2007, in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

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DECLARATION

I solemnly declare that the dissertation titled "**CLINICAL AND RADIOIMAGING STUDY IN POSTERIOR CIRCULATION STROKE**" is done by me at Madras Medical College & Govt. General Hospital, Chennai during 2005-2006 under the guidance and supervision of Prof. V.K. Rajamani, M.D.

The dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University towards the partial fulfilment of requirements for the award of M.D. Degree (Branch I) in General Medicine.

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ABBREVIATIONS AND ACRONYMS

AICA - ANTERIOR INFERIOR CEREBELLAR ARTERY

ASA - ANTERIOR SPINAL ARTERY

CAD - CORONARY ARTERY DISEASE

CSF - CEREBROSPINAL FLUID

CT - COMPUTED TOMOGRAPHY

ICA - INTERNAL CAROYID ARTERY

JNC - JOINT NATIONAL COMMITTEE

MRI - MAGNETIC RESONANCE IMAGING

PCA - POSRERIOR CEREBRAL ARTERY

PET - POSITRON EMISSION TOMOGRAPHY

PICA - POSTERIOR INFERIOR CEREBELLAR ARTERY

PPRF - PARAPONTINE RETICULAR FORMATION

INO - INTERNUCLEAR OPTHALMOPLEGIA

RHD - RHEUMATIC HEART DISEASE

SCA - SUPERIOR CEREBRAL ARTERY

TIA - TRANSIENT ISCHEMIC ATTACKS

VBI - VERTEBRO BASILAR INSUFFICIENCY

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INTRODUCTION

STROKE is defined ‘as abrupt onset of neurological deficit that is attributable to focal vascular cause’[1]. Stroke syndromes are classified into 1. Anterior circulation stroke and 2.Posterior circulation stroke based on the blood supply. The internal carotid artery and its branches comprise the anterior circulation. Stroke syndromes of the posterior circulation account for approximately 20% of all strokes[7,8].

The posterior circulation consists of the paired vertebral arteries, the basilar artery, the paired posterior cerebral arteries and their branches . These arteries, through short penetrating branches and circumferential branches, supply the brainstem (medulla, pons, and midbrain), the thalamus, the hippocampus, the cerebellum, and parts of the occipital and temporal lobes including the visual cortex. There is marked variability in the exact arterial anatomy of the posterior circulation[2].

The posterior circulation is constructed quite differently from the anterior circulation and consists of vessels from each side which unite to form midline arteries that supply the brainstem and spinal cord . Within the posterior circulation, there is a much, higher incidence of asymmetric, hypoplastic arteries, of variability of supply and of retention of fetal circulatory patterns.

The posterior circulation, unlike the intracranial portions of the anterior circulation, is prone to atherosclerosis much as are other

systemic arteries[3]. In the case of one vertebral artery being occluded, collateral flow comes from the opposite vertebral artery, from muscular cervical artery branches, and from posterior communicating artery[4,5].

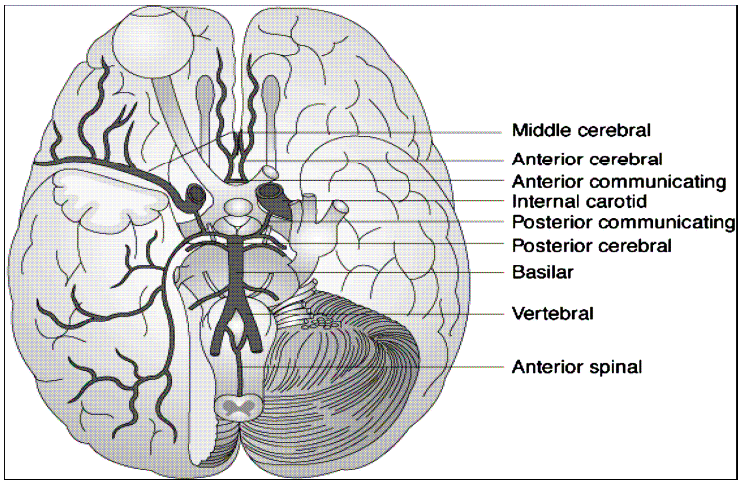


Figure 1 : Blood supply of Brain

The vertebral arteries originate from the subclavian arteries and course through the vertebral foramina of C6-C2, around the atlas, and through the foramen magnum. At the pontomedullary junction both vertebral arteries join to create the basilar artery. The vertebral arteries supply the medulla, the pons, and the cerebellum. The distal vertebral arteries and the basilar artery give rise to the arteries that supply the cerebellum: the superior, anterior, posterior, and inferior cerebellar arteries. The vertebral arteries are prone to atherosclerotic changes at their origin, and at the junction with the basilar artery.

The basilar artery and its branches supply the pons and the cerebellum. Distally the basilar artery divides into the posterior

cerebral arteries and then the posterior communicating artery in the Circle of Willis. Atherosclerotic lesions can occur anywhere along the trunk of the basilar artery.

The posterior cerebral artery (PCA) supplies portions of the midbrain, the thalamus, the hippocampus, the medial temporal lobe, and the occipital lobe, including the visual cortex.

Posterior circulation ischemia can range from fluctuating brainstem symptoms, caused by intermittent insufficiency of the posterior circulation (so-called VBI), to the “locked-in syndrome," which is caused by basilar artery or bilateral vertebral artery occlusion[3].

AIMS AND OBJECTIVES

- To study the various clinical patterns of posterior circulation stroke.
- To analyze various risk factors for stroke in these patients.

REVIEW OF LITERATURE

'Patients who present to physicians and hospitals with symptoms that suggest posterior circulation ischemia are handled differently from patients who have symptoms that suggest anterior circulation disease in the great majority of medical facilities. A patient who has an attack of dizziness with diplopia and ataxic gait usually has a brain image but seldom has vascular or cardiac investigations. A diagnosis of "vertebrobasilar insufficiency" (VBI) is often made, and physicians then debate whether or not to treat with warfarin-type anticoagulants,and, if so, for how long and at what intensity. In contrast, a patient who has right-hand weakness and aphasia is usually evaluated and treated quite differently at the very same facilities. Brain imaging, cardiac investigations, noninvasive vascular tests of the carotid and intracranial anterior circulation with the use of extracranial and transcranial ultrasound and/or MR angiography (MRA) and CT angiography, and catheter angiography are often pursued, depending on the local technological capabilities and experience of the treating physicians. An effort is made to identify the etiology and mechanism of the ischemia. Treatment is then chosen among a variety of possibilities (including carotid artery surgery, angioplasty, anticoagulants, and antiplatelet aggregants) depending on the nature, location, and severity of the occlusive disease and the mechanism of ischemia. Why should anterior and posterior circulation ischemia be handled so differently? Does this schizophrenic approach make sense? After all, the internal carotid artery and its branches and the vertebral (VA) and basilar arteries (BA) and their branches are just a few inches apart; they are made of the same coats and look the same under the microscope except for size. These vessels carry the same blood under the same blood pressure. The diseases that affect the blood vessels in the 2 circulations are the same. Do stroke mechanisms really differ between the 2 circulations? How did this differing approach originate, and does it continue to make sense today?'

Louis caplan MD
The Thomas Willis Lecture
Posterior Circulation Ischemia: Then, Now, and Tomorrow [6]

Charles Foix and his colleagues dissected and described in detail the arteries of both the anterior and the posterior circulation. They described the arterial supply of the thalamus the posterior cerebral artery and its branches, and the blood supply of the pons and the medulla oblongata. Especially important was the description of the pattern of blood supply of the pons

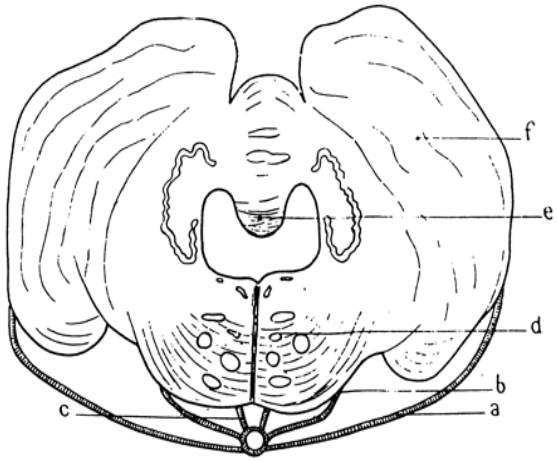


Figure 2 : Foix's schema of the blood supply of the brain stem. a, Long circumferential artery; b, short circumferential artery; c, larger paramedian artery; d, "protuberance" (pons); e, cerebellar vermis; and f, lateral lobe of the cerebellum

Vascular Pathology and the Mechanism of Brain Infarction

During the first half of the 19th century, the terms *encephalomalacia*, *softenings*, and *ramollissements* were in general use. These were all descriptive terms and did not indicate etiology Virchow introduced the terms *thrombus*, *thrombosis*, *embolus*, and *embolism* and deduced the general principles of thrombosis and embolism. Virchow's triad explained localized thrombus formation and

consisted of the following: (1) an abnormality of the intima and vascular wall, (2) an abnormality of blood flow, and (3) an abnormality of blood coagulability. Virchow's pathological studies revolutionized thinking about brain infarction, thrombosis, and embolism. The early studies of Charles Foix related strictly to the localization of ramollissements (brain softenings) and their vascular supply and accompanying clinical findings.Among 56 brains with infarcts, the artery supplying the infarcts was totally occluded in only 12 and subtotally in 14. In 30 patients the arteries were open. Foix and his colleagues speculated on possible explanations of the arterial patency: (1) arterial occlusion might follow softenings, (2) embolism with distal passage before necropsy, (3) insufficiency (*l'insuffisance arterielle*), that is, more proximally located circulatory failure, and (4) vasospasm (*spasme arterielle*) [6].

The next important contributor was Raymond Adams, a neuropathologist and clinical neurologist. described the clinical and necropsy findings in 18 patients who at necropsy had occlusion of the basilar artery. Eleven occlusions were thought to arise in situ, while 7 were considered embolic. They noted morphological distinctions between thrombosis and embolism, as follows:

Thrombosis of the basilar artery could usually be recognized at a glance. The thrombosed portion of the vessel was distended, firm, and rigid and the thrombus could not be displaced by pressure. . . . In embolism, the embolus was usually lodged in the distal portion of the basilar artery.

C. Miller Fisher is the individual probably most responsible for furthering information about stroke and stroke mechanisms during the 20th century Fisher's 1951 report[6] on occlusion of the internal carotid artery was a benchmark in the history of stroke.This article emphasized that occlusions commonly developed in the neck engrafted on atherosclerosis and that transient ischemic attacks (TIAs) often preceded and warned of the ensuing stroke. The carotid artery stenosis was possibly approachable surgically. Before this report, although in 1905 Chiari had described a patient with embolism arising in an occlusion of the internal carotid artery in the neck, anterior circulation infarcts were invariably attributed to middle cerebral artery disease. In 1954, Fisher reported subsequent observations on internal carotid artery disease. Later Fisher and his colleagues described the distribution of atherosclerotic lesions found at necropsy within the extracranial and intracranial anterior and posterior circulations. Within the posterior circulation, Fisher described occlusions of the vertebral artery in the neck ; with Kubik and Karnes he described the vascular pathology found at necropsy in patients with lateral medullary infarcts and emphasized that intra-arterial embolism ("local embolism") was an important mechanism of stroke in the posterior circulation as well as in the anterior circulation. In a series of meticulous analyses of serial sections from patients with small deep infarcts, many located in the brain stem, Fisher described the pathology in the penetrating arteries, lipohyalinosis and atheromatous branch occlusions, that caused penetrating artery territory infarcts.

Physiology of the Cerebral Circulation[7]

Brain Metabolism

Brain metabolism is steady and continuous with no rest periods, requiring a continuous supply of glucose and oxygen. It is highly depended upon oxygen and accounts for 20% of the body's oxygen usage. Consciousness may be lost in as little as 10 seconds once blood flow has ceased. Dependent upon oxygen, the brain does not switch to anaerobic metabolism, and a lapse of even a few minutes may cause irreversible damage. Sustained hypoglycemia may also damage brain tissue, since glucose is its major energy source. Neurons do not store glycogen, so energy depletion is rapid[1,7]

Autoregulation of Blood Flow[7]

The normal brain has the ability to regulate its own blood supply to maintain arterial pressure between 65 and 140 mm Hg.Factors that affect this can be viewed as extrinsic and intrinsic. The extrinsic factors affecting cerebral blood flow (CBF) are related primarily to the cardiovascular system and include blood pressure (BP), cardiovascular function, and blood viscosity. If systemic mean BP drops below 60 mm Hg, the brain's autoregulatory mechanism becomes less effective. The brain will initially attempt to compensate by extracting more oxygen from the available blood supply. If BP continues to drop, signs of cerebral ischemia will occur. The intrinsic factors are influenced by cerebral perfusion

pressure. This is the pressure difference between the cerebral arteries and veins. The goal is that cerebral perfusion pressure will remain constant despite changes in systemic BP. Cerebral vascular resistance increases or decreases in response to systemic BP to maintain a constant flow of blood to the brain.. Cerebral blood vessels are under the control of the sympathetic nervous system or sympatho-adrenal response. Sympathetic reflexes are believed to cause vasospasm in some types of brain damage. Intracranial pressure (ICP) is also an important regulatory mechanism. An increase in ICP will increase cerebrovascular resistance, reducing cerebral blood flow. ICP is affected by these three metabolic factors: 1.CO2 concentration, 2.H+ concentration, and3. O2 concentration. High CO2 tension is a strong vasodilator and can produce a marked increase in cerebral blood flow. A decreased pH (increased H+ levels) will increase blood flow. Low O2 tension is a powerful vasodilator, and high O2 Levels are a moderate vasoconstrictor.

Cerebral Perfusion Pressure[7]

Cerebral perfusion pressure (CPP) is the difference between mean arterial blood pressure (MAP) and the ICP. The normal range is 60 to 80 mm Hg.

MAP − ICP = CPP

The cranial cavity contains the brain, blood, and CSF. Of this, the brain takes up 80% with the blood volume and CSF each comprising 10%. Together, they maintain normal ICP of 50 to 200

mm H2O (4 to 15 mm Hg). Changes in one will cause a compensatory change in the other to maintain normal ICP. This is referred to as the Monro-Kellie hypothesis. The ICP will remain within normal range as long as any increase that occurs does not exceed the compensatory displacement of blood or CSF. If an over compensatory displacement occurs, the ICP will rise, resulting in cerebral hypoxia, or brain herniation .If the CPP falls below 50 mm Hg, cellular death occurs. When the pressure in the ICP exceeds MAP, tissue perfusion falls and demand exceeds supply, and cellular hypoxia occurs. Increases in ICP cause the CNS ischemic response. This is evidenced by a marked increase in MAP, reflexive slowing of heart rate, and widened pulse pressure. This triad of signs, Cushing reflex, is an important but a late indicator of increased ICP.

Strokes may either be haemorrhagic or ischaemic[8]. Eighty eight per cent of all strokes are ischaemic, 9% are due to intracerebral haemorrhage, and 3% are due to subarachnoid haemorrhage.

Haemorrhagic stroke

Intracranial haemorrhage may occur within the brain parenchyma (intracerebral haemorrhage) or within the surrounding meningeal spaces (including epidural haematoma, subdural haematoma, and subarachnoid haemorrhage).

In intracerebral haemorrhage, bleeding occurs directly into the brain parenchyma. In addition to the area of the brain injured

by the haemorrhage, the surrounding brain can be damaged by pressure produced by the mass effect of the haematoma. A general increase in intracranial pressure may occur.

Non-traumatic intracerebral haemorrhage is usually due to hypertensive damage to blood vessel walls. Chronic hypertension causes lipohyalinosis, fibrinoid necrosis, and the development of Charcot-Bouchard aneurysms in arteries throughout the brain, which may then rupture. Non-traumatic intracerebral haemorrhage may also be due to excessive cerebral blood flow (for example, haemorrhagic transformation of an ischaemic infarct); rupture of an aneurysm or an arteriovenous malformation; an arteriopathy; a coagulopathy; a vasculitis; haemorrhagic necrosis; or venous outflow obstruction.

Non-penetrating and penetrating cranial trauma are also common causes of intracerebral haemorrhage.

Strokes due to hypertension more commonly occur in sites such as the basal ganglia, thalamus, pons, cerebellum, and other brainstem sites, whereas those due to other causes more commonly occur in lobar regions (particularly the parietal and occipital lobes)[7].

Subarachnoid haemorrhage usually occurs after rupture of a berry aneurysm in the circle of Willis. Other uncommon causes include trauma, hypertensive haemorrhage, vasculitides, tumours, and coagulopathies. This results in blood accumulating in the basal cisterns and around the brainstem.

Ischaemic stroke

Ischemia occurs when there is a decrease in blood flow to less than 20 mL/100 g of brain tissue per minute. Reduction of blood flow to less than 16 mL/100 g of brain tissue per minute leads to tissue death within one hour. In the absence of blood flow, death of brain tissue occurs within 4 to 10 minutes[1]

An acute vascular occlusion results in ischaemia in the dependent area of the brain. About 80% of ischaemic strokes are due to thromboses and emboli. The most common sites of thrombotic occlusion are cerebral artery branch points, particularly in the distribution of the internal carotid artery. Arterial stenosis precipitated by turbulent blood flow, atherosclerosis, and platelet adherence cause blood clots to form. Less common causes of thromboses, particularly seen in younger stroke patients, include cervical artery dissection, essential thrombocythaemia, polycythemia, sickle cell anaemia, protein C deficiency, fibromuscular dysplasia of the cerebral arteries, and cocaine misuse.

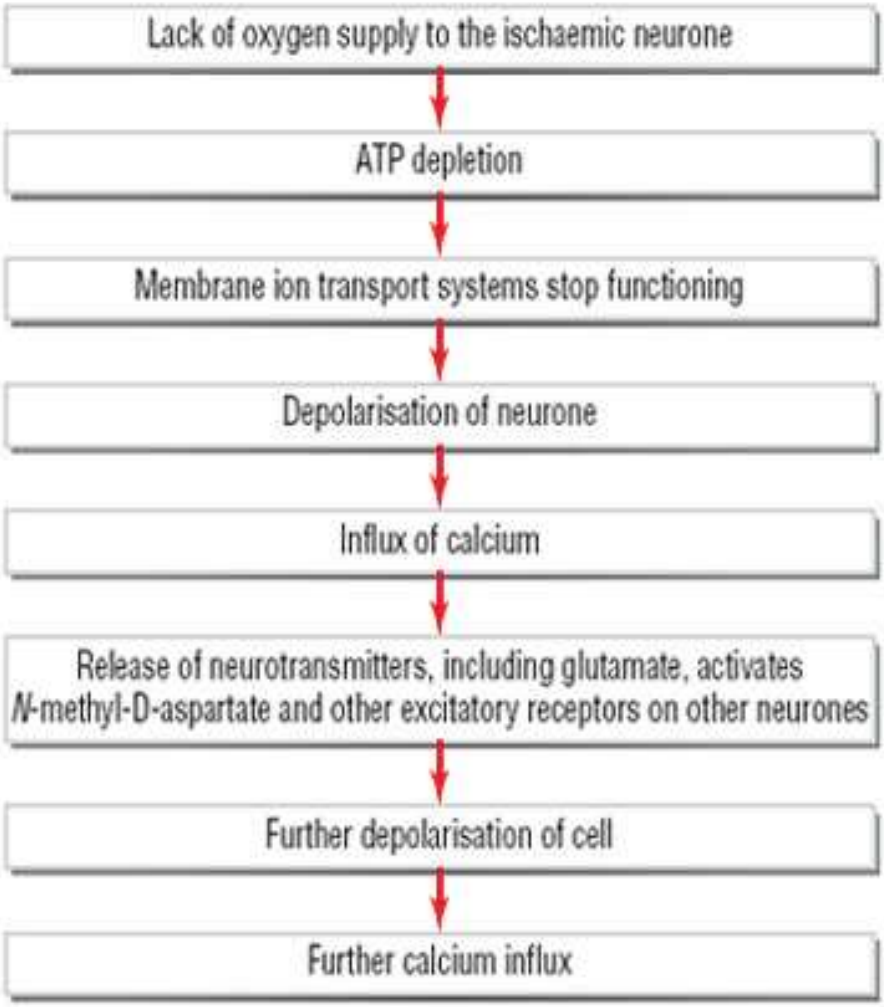
Emboli may arise from the heart, the extracranial arteries, or, rarely, the right sided circulation (paradoxical emboli), and can occlude the vasculature. Furthermore, rarely infective causes of emboli, such as subacute bacterial endocarditis, may cause occlusion, as may emboli due to iatrogenic causes, such as a cardiac prosthesis.

Small vessel disease within the brain causes a further 20% of ischaemic strokes. These are usually in patients with generalised small vessel disease - for example, hypertensive and diabetic patients. Multiple small emboli or an in situ process called lipohyalinosis (in which multiple microatheromata occlude the vessels) are thought to be responsible [10,11].

A system of categories of subtypes of ischaemic stroke mainly based on cause has been developed for the "Trial of Org" 10 172 in acute stroke treatment (TOAST) [12]. This classification denotes five subtypes of ischaemic stroke - large artery atherosclerosis, cardioembolism, small vessel occlusion, stroke of other determined cause, and stroke of undetermined cause.

In all cases, loss of perfusion to a part of the brain results in an "ischaemic cascade" [1] . Consequently the initial ischaemic insult is locally amplified.

The high intracellular calcium activates various enzymes that cause the destruction of the cell. Free radicals, arachidonic acid, and nitric oxide are generated by this process leading to further neuronal damage. Within hours to days of a stroke occurring, specific genes are activated that cause the formation of cytokines and other factors that in turn cause further inflammation and microcirculatory compromise. The area of damage thus spreads rapidly after the initial ischaemic event.



Risk factors [13]

Stroke has numerous risk factors, some of which (such as increasing age and systolic blood pressure) are risk factors for both ischaemic and haemorrhagic stroke, however, other factors are more specific for type of stroke. The table gives important risk factors and their relative risk. The risks factors for posterior ciculation and anterior circulation stroke are considered same.

Risk factors for stroke

Risk factor	Description	Relative risk*
Ischaemic stroke		
Age	For each successive 10 years after age 55	1.74
Family history	Paternal history of stroke or transient ischaemic attack	2.4
	Maternal history of stroke or transient ischaemic attack	1.4
	Parental history of coronary heart disease	3.33
Systolic blood pressure	For each 10 mm Hg increase	1.15
Atrial fibrillation	For successive decade of life above age 55, incidence doubles; non-valvular atrial fibrillation accounts for 1.5% strokes in people aged 50-59 years, rising to 23.5% in people aged 80-89 years	3-5
Myocardial disease	Coronary heart disease	2
	Electrocardiographic left ventricular hypertrophy	3
	Cardiac failure	3-4
Diabetes mellitus	People with diabetes mellitus have increased susceptibility to atherosclerosis and increased atherogenic risk factors, particularly hypertension, obesity and abnormal blood lipids; known diabetes mellitus	2.45
	Asymptomatic people who have a high average blood glucose (=225 mg/dl)	1.43
Cigarette smoking	-	1.9
Alcohol	Complex associated dependent on amount (J shaped association) and race	
Previous transient ischaemic attack	-	2.3
Carotid stenosis	-	2.03

Haemorrhagic stroke

Age	Incidence increases exponentially with increasing age; relative risk for 85 year olds compared with 70-74 year olds	2.5
Systolic blood pressure	110-139 mm Hg	1
	140-179 mm Hg	4
	=180 mm Hg	8
Anticoagulation (international normalised ratio)	<3.0	1
	3.5-3.9	4.6

*Confidence intervals omitted for clarity

- The major **PCA** stroke syndromes that have been divided into P 1 and P 2. syndromes. P 1 syndromes are due to disease of proximal segment or its penetrating branches (thalamogeniculate,Percheron and posterior choroidal). P 2 syndromes are due to disease of PCAA distal to junction of PCA with Posterior communicating artery, and produce cortical and temporal lobe signs [1].

- The major clinical features of PCA syndromes comprise of **Paramedian thalamic infarction [4,5]**

This syndrome, resulting from bilateral medial thalamic infarction, is part of the differential diagnosis of delirium and coma. Patients often are obtunded to comatose or agitated, and they may or may not have associated hemiplegia or hemisensory loss.

Occasionally, the cranial nerve III nucleus is involved, with resultant ophthalmoplegia. Patients may take days to weeks to recover and seem to be in a sleeplike state. Although alertness generally returns, prognosis for good functional recovery is poor because of severe memory dysfunction. The syndrome may result from a "top of the basilar" artery embolus. The artery of Percheron, referred to earlier, may be involved. This has been referred to as a posterior variant of the syndrome of akinetic mutism.

- **Pure hemisensory loss**

Infarction of the ventral posterolateral nucleus of the thalamus results in hemisensory loss. This is one of the well-described lacunar syndromes. Usually, the vessel involved is the thalamogeniculate branch. A related disorder is Dejerine-Roussy syndrome, in which quickly resolving hemiparesis and hemiataxia leave the patient with delayed, persistent, hemisensory disturbance with paroxysmal pain on the affected side.

- **Visual field loss**

A general rule of visual field examination is that the further posterior the lesion, the more congruous is the visual field loss. Bilateral infarctions of the occipital lobes produce varying degrees of cortical blindness depending upon the extent of the lesion. Patients often exhibit Anton syndrome, a state in which they fervently believe they can see when they cannot. Patients may describe objects that they have not seen previously in exquisite detail,

completely in error and oblivious to that error. Another intriguing phenomenon is blindsight—although cortically blind, patients can respond to movement or sudden lightening or darkening of environment. Unilateral infarction produces homonymous hemianopia. Sparing of the macula is encountered frequently in infarction of the occipital lobes due to PCA occlusion. Macular sparing is caused by collateral vascular supply to the occipital pole from posterior branches of the MCA and preservation of the optic radiations, though bilateral representation of macular vision also has been suspected. Infarction of the lateral geniculate nucleus may produce hemianopia, quadrantanopia, or sectoranopia. The vascular supply is dual; the anterior choroidal artery supplies the anterior hilum and anterolateral areas, and the posterior choroidal artery supplies the rest. Occlusion of the posterior choroidal artery may produce a distinct syndrome of hemianopia, hemidysesthesia, and memory disturbance due to infarction of the lateral geniculate, fornix, dorsomedial thalamic nucleus, and posterior pulvinar.

- **Visual agnosia**

Visual agnosia refers to a lack of recognition or understanding of visual objects or constructs. It is a disorder of higher cortical function. The strict diagnosis of visual agnosia requires intact visual acuity and language function. Most patients have bilateral lesions, sparing the visual cortex but disrupting or disconnecting visual information from reaching parts of the visual association cortex for reference to visual memories. The patient with visual agnosia can

recognize objects presented in another modality. True visual agnosia has been divided into apperceptive and associative subtypes. In apperceptive visual agnosia, patients cannot name objects presented to them, draw objects from memory, or identify or match objects. Yet, they can see and avoid obstacles when ambulating and detect subtle changes in light intensity. In associative agnosia, patients can draw objects to command and match them or point to them but cannot name them. They can see shapes and reproduce them in drawing, yet not recognize the identity of objects.

- **Balint syndrome**

Balint syndrome typically occurs in degenerative diseases but also may occur with bilateral parieto-occipital infarction, most often in the watershed between the PCA and MCA territories. It is a triad of visual simultanagnosia, optic ataxia, and apraxia of gaze. *Visual simultanagnosia* implies an inability to examine a scene and integrate its parts into a cohesive interpretation. A patient can identify specific parts of a scene but cannot describe the entire picture. *Optic ataxia* implies a loss of hand-eye coordination such that reaching or performing a motor task under visual guidance is clumsy and uncoordinated. Finally, *apraxia of gaze* is a misnomer describing a supranuclear deficit in the ability to initiate a saccade on command.

- **Disorders of face recognition**

Prosopagnosia refers to an inability to recognize faces. Typically, this deficit results from bilateral lesions of the lingual and fusiform gyri; however, cases of unilateral nondominant hemisphere lesions resulting in prosopagnosia have been reported. Usually, it does not occur in isolation and other object agnosias coexist. Autoprosopagnosia, or inability to recognize one's own face in a picture or mirror, may occur as a subset of this syndrome.

- **Palinopsia, micropsia, and macropsia**

These are illusory phenomena that are of uncertain pathophysiology. They may represent seizure activity and traditionally are treated with anticonvulsants. Palinopsia describes the persistence of a visual image for several seconds to days in a partially blind hemifield. Micropsia and macropsia describe situations where objects appear smaller or larger than expected.

- **Disorders of reading (alexia, dyslexia)**

Pure alexia may result from infarction of the dominant occipital cortex. Words are treated as if they were from a foreign language. Patients may retain the ability to formulate a word and its meaning if spelled out to them orally or if they trace the letters with their hand. Patients may then learn to read, albeit terribly slowly, in a letter-by-letter fashion, being unable to integrate multiple letter groups. Classic alexia without agraphia was described by Dejerine in

the late 19th century. In his case study, he emphasized a left occipital cortex lesion and also infarction of the splenium of the corpus callosum, which disconnected fibers from the right occipital lobe from reaching the angular gyrus. Rarely, the dominant-hemisphere, posterior temporal lobe is supplied by PCA. Damage to this area results in a Wernicke-type aphasia with associated dyslexia and right hemianopia due to concomitant left occipital infarction.

- **Disorders of color vision (achromatopsia, dyschromatopsia)**

Lesions of the lingual gyrus in the inferior occipital lobe may produce disorders of color perception. Testing with Ishihara plates reveals a deficit. Colors may be described as washed out or gray. This deficit usually occurs only in the contralateral visual field and is called "hemiachromatopsia."

A related problem is color anomia, also called color agnosia, in which patients can perceive and match colors but cannot associate them with the proper color names. This deficit also has been explained by a disconnection model.

- **Memory (amnesia)**

Infarction of the medial temporal lobe, fornices, or medial thalamic nuclei may result in permanent anterograde amnesia. Although traditionally bilateral infarction has been thought to be required for amnesia, memory functions may be lateralized such

that infarction of left-sided structures may have a more lasting impact on verbal function. Older patients frequently have lasting short-term memory impairment from unilateral PCA territory infarction. Recent imaging in patients with transient global amnesia has demonstrated diffusion-weighted lesions in unilateral temporal lobes resulting in temporary amnesia.

- **Motor dysfunction**

When the blood supply to the cerebral peduncles arises from perforators of P1 segment, infarction may occur, resulting in hemiplegia or hemiparesis. The clinical syndrome is no different from capsular infarction but often includes concomitant hemianopia because of occipital lobe involvement. The syndrome may mimic a large MCA infarct

Other clinical syndromes include , a third nerve palsy with contralateral ataxia (Claude's Syndrome) or with contralateral hemiplegia (Weber's Syndrome) may result. The ataxia indicates involvement of the red nucleus or dentatorubrothalmic tract. The hemiplegia is localized to the cerebral peduncle. If the subthalmic nucleus is involved. Contrlateral hemiballismus may occur. Occlusion of the posterior cerebral artery Can produce peduncular hallucinosis (Visual halluciations of brightly colored scenes and objects)

VERTEBROBASILAR ATRTERY DISEASE [6,7]

The most common heralding symptoms of vertebral and basilar artery disease include the following:

- Motor deficits, including facial paresis - 40-67% of case
- Dysarthria and speech impairment - 30-63% of cases
- Vertigo, nausea, and vomiting - 54-73% of cases
- Headache - 40-42% of cases
- Visual disturbances - 21-33% of cases
- Altered consciousness - 17-33% of cases

In a few cases, convulsion- like movements have been reported, and, along with hemiparesis, they are the only diagnostic clue.

Patients occasionally present with isolated vertigo or dizziness with no other neurological complaint [14,15,16]. Although no absolute model allows differentiation of patients with inner ear disorder from those with basilar artery occlusion, the presence of vascular risk factors, headache, and the inability to walk support the diagnosis of vertebrobasilar insufficiency (VBI). Any associated neurological signs of brainstem dysfunction (see above) also support the diagnosis of VBI. Tinnitus, unilateral hearing loss, and precipitation by movement support the diagnosis of peripheral vestibular dysfunction.

In the out patient department,differentiating, vertigo of central and peripheral origin is crucial in suspecting VBI. The following table depicts the difference between the two [17.18].

	Central Vertigo	**Peripheral Vertigo**
Intensity of Vertigo	Mild	Severe
Tinnitus	Rare	Frequent
Associated Cranial Nerve findings	Usual	None
Nystagmus:		
Visual fixation	No inhibition of nystagmus	Inhibits nystagmus
horizontorotary nystagmus	Rare	Common
Latency (time from head movement to onset of nystagmus)	None	3-40 seconds
Fatigue	None	Yes
Habituation	None	Yes
Reproducibility	Consistent	Variable

Physical Signs

An abnormal level of consciousness and motor signs, such as hemiparesis or quadriparesis (usually asymmetric), are seen in more than 70% of patients These findings are followed by pupillary abnormalities, oculomotor signs, and pseudobulbar manifestations (facial weakness, dysphonia, dysarthria, dysphagia) in more than 40% of patients

The signs described can be present in different combinations. The syndromes commonly associated with pontine ischemia are the locked-in syndrome and coma associated with oculomotor

abnormalities and quadriplegia. The syndrome associated with rostral brainstem ischemia is called top-of-the-basilar syndrome.

Top-of-the-basilar syndrome [18,19,20,21] is the manifestation of upper brainstem and diencephalic ischemia caused by occlusion of the rostral basilar artery, usually by an embolism. Patients present with changes in the level of consciousness that are associated with visual symptoms such as hallucinations and/or blindness. They can also demonstrate oculomotor abnormalities, including horizontal but most commonly vertical gaze abnormalities such as skew deviation. Third nerve palsy and pupillary abnormalities are also frequent. Motor abnormalities include abnormal movements or posturing.

The locked-in syndrome is caused by infarction of the basis pontis secondary to occlusive disease of the proximal and middle segments of the basilar artery, which leads to quadriplegia. Because the tegmentum of the pons is spared, the patient has a spared level of consciousness and preserved vertical eye movements and blinking.

Coma associated with oculomotor abnormalities and quadriplegia also indicates proximal and mid basilar occlusive disease with pontine ischemia.

Involvement of the abducens nucleus, the horizontal gaze center located in the paramedian reticular formation (PPRF) contiguous to the abducens nucleus, and/or the medial longitudinal fasciculus (MLF). Lesions to these structures result in the following:

Ipsilateral abducens palsy ,Ipsilateral conjugate gaze palsy,Internuclear ophthalmoplegia (INO) ,One-and-a-half syndrome caused by a lesion simultaneously affecting the PPRF and MLF, resulting in ipsilateral conjugate gaze palsy and INO .Ocular bobbing, which localizes the lesion to the pons (This is characterized by a brisk downward movement of the eyeball with a subsequent return to the primary position.)

Other reported signs of pontine ischemia include limb shaking, ataxia (usually associated with mild hemiparesis), facial weakness, dysarthria, and dysphagia.

The most frequent clinical manifestation of vertebral artery dissection is posterior headache or neck pain accompanied or followed by posterior circulation transient ischemic attack or stroke. Rarer clinical features include isolated headache or neck pain, cervical spinal cord ischemia and cervical root impairment. Asymptomatic vertebral artery dissections have been reported.

In the case of primary intracranial vertebral artery dissection or intracranial extension of an extracranial dissection, subarachnoid hemorrhage and rarely rostral cervical spinal cord ischemia or posterior fossa mass effect may occur.

Investigations in a stroke patient [3,9,22]

In the acute phase, routine blood tests are all that is needed. These include a complete blood count (with platelet count), prothrombin time (PT)/activated partial thromboplastin time

(aPTT)/international normalized ratio (INR), electrolytes, creatinine, serum creatine kinase (CK), and serum glucose. These tests are required to assess whether the patient is a candidate for thrombolysis and are a part of the stroke mechanism workup.

If the stroke mechanism is not evident on the basis of imaging studies, then special hematologic and serologic examinations should be ordered. A full coagulation workup typically might include assays for antiphospholipid antibodies and lupus anticoagulant. Hypercoagulable factors usually associated with venous infarction, such as protein C, protein S, factor V Leiden-activated protein C resistance, antithrombin III, and prothrombin gene polymorphism, may be appropriate if the stroke mechanism is thought to involve either venous thrombosis or paradoxical embolism. These tests are better done 2-3 weeks after the acute event. Blood smear examination, platelet aggregation studies, sucrose - lysis test for paroxysmal nocturnal hemoglobinuria, and Venereal Disease Research Laboratory test (VDRL) also should be considered. Abnormalities in any of these blood tests are rare causes of stroke. Nevertheless, particularly in a young patient, a full workup should be considered.

When the mechanism of stroke is atherosclerotic disease, follow-up blood investigations should be done to assess atherosclerotic risk factors. Diabetes should be considered and screened for. A fasting serum cholesterol profile may be artifactually low in the acute setting and should therefore be conducted 8-12

weeks after the stroke. High cholesterol is a definite risk factor for stroke and is amenable to treatment.

In selected patients, assessing fasting serum homocysteine levels is reasonable. High serum homocysteine level is an independent risk factor

Imaging Studies:

Neuroimaging: An emergent CT scan is required prior to considering thrombolysis. CT is less sensitive for the posterior fossa strokes because of bone artifact and decreased tissue detail. MRI is a the investigation of choice.

High-quality CT angiography can be used to delineate the extracranial and intracranial posterior circulation and is very helpful for evaluating patients with suspected basilar-artery occlusion

MRI defines multiple lesions and allows a much better examination of midbrain, subthalamic, and thalamic structures than CT scan. With the use of diffusion- and perfusion-weighted imaging, a much more complete assessment of the infarcted tissue and tissue at risk is available.

Other possible brain imaging procedures include single-photon emission computed tomography (SPECT) and positron emission tomography (PET).

SPECT is a nuclear medicine study using radioisotopes of technetium. It provides an analysis of relative blood flow by region, usually in the resting state. It is rarely useful in the clinical setting in acute stroke and can be considered a research tool.

PET can be used to analyze neurometabolism in vivo; it is at present a research tool.

Rarely, plain skull films demonstrate an unexpected tumor or calcification in an aneurysm.

Transcranial Doppler ultrasonography (TCD) is not yet widely used and remains largely a research tool; however, that it is a highly useful adjunct in the emergent evaluation of patients with stroke is becoming increasingly apparent. TCD is dependent upon the skill and experience of the operator. In skilled hands, both the distal basilar and P1 and P2 segments can be assessed. Much more information is available about the MCA than the PCA. However, TCD may detect acute clot in the PCA.

Carotid duplex ultrasonography is used widely and should not be overlooked in PCA stroke. When a fetal origin PCA is present, the cause of stroke still may be significant carotid artery atherosclerotic disease. The appropriate treatment for secondary prevention will then be carotid endarterectomy rather than medical therapy.

Selective catheter cerebral angiography remains the criterion standard to evaluate the vascular anatomy. However, it is invasive and does carry a small risk of procedure-related morbidity.

Increasingly, noninvasive methods of viewing the arterial anatomy are being developed—magnetic resonance angiography, CT angiography (CTA), and TCD. Currently these tests are reasonably good for assessment of the proximal circulation. When these are doubtful or more information is needed about the distal circulation, angiography is required. Angiography is needed to diagnose small aneurysms or vasculitis. In addition, angiography is required as a precursor to endovascular therapeutic techniques. Angioplasty and angioplasty with stenting are being adapted to the cerebral circulation.

Echocardiography: Standard transthoracic echocardiography (TTE) is used in investigation of possible cardiac sources of embolus. Transesophageal echocardiography (TEE) is used to examine the aortic arch as well as cardiac sources of emboli. It is about 3 times more sensitive than TTE in detecting possible sources of emboli. A recent study suggested that it is cost-effective in acute stroke, whereas the TTE is not, despite the greater cost of TEE.

Medical Care:

The treatment of stroke necestitates an understanding of the mechanism of stroke. Establishing a probable mechanism is at times straightforward; for example, the ECG demonstrates atrial fibrillation. At times, however, it is more difficult—for example, TEE with a bubble study demonstrates a patent foramen ovale. The medical treatment of stroke can be divided into acute, subacute and chronic phases.

- **Acute therapy** - Within 3 hours of stroke onset

The National Institutes of Neurological Diseases and Stroke (NINDS) [25] trial of recombinant tissue-type plasminogen activator tPA (rtPA), published in 1995, included all types of ischemic stroke . If a clear time of onset can be established, stroke in the PCA territory may be treated with intravenous rtPA. However, because hemianopia may not be recognized immediately, particularly left hemianopia, the risk that patients will mistake the time of stroke onset is significant.

Intra-arterial thrombolytic therapy has been used successfully for patients with suspected BAO. In a report by Hacke and Zeumer [26] , of 65 patients with vertebrobasilar occlusion, 43 received IA therapy with urokinase. Nineteen patients recanalized, with14 patients surviving and 10 patients having favorable outcomes. Those who did not recanalize died. Conventional antiplatelet and antithrombotic therapy was administered to 22 patients, with only 3 surviving. Zeumer [26] further reported on 28 patients with BAO and severe symptoms, who were treated with local intra-arterial therapy within 24 hours of symptom onset. Mortality was reduced to 50%, despite severe symptoms at presentation. Gonner [27] reported a similar experience with 10 BAO patients who had severe symptoms. Five had good recovery, associated with complete or partial recanalization, of whom 3 were treated beyond 6 hours of symptom onset. A series of small uncontrolled trials involving over 200

patients demonstrates a 50-100% recanalization rate resulting in a favorable outcome in 25-60% .

In nine additional reports, among 285 patients who were mostly given t-PA more than eight hours after the onset of stroke,45 62 percent had good recanalization and 28 percent of the overall population subsequently did well [28]. Brandt et al. found that among 51 patients who underwent thrombolysis for acute vertebrobasilar lesions, those with embolic occlusions that were short and involved the proximal basilar artery with good collateral arteries were most likely to have recanalization and a good outcome. Patients who were comatose or tetraplegic or who had chronic white-matter abnormalities had poor outcomes [29].

Subacute and chronic therapy - More than 3 hours from stroke onset

Even if thrombolytic therapy is contraindicated, the choice of acute or subacute therapy is dependent on the physician's understanding of the stroke mechanism.

Anticoagulation with heparin often is used, although no strong evidence exists among any stroke type that heparin is useful. The trial of ORG 10172 (danaparoid, a heparin like drug) in acute stroke treatment (ie, TOAST) [12,25] has suggested that anticoagulation may be beneficial in stroke due to large artery atherosclerotic disease with major stenosis, although the principal endpoints of the study did not confirm any benefit of anticoagulation. Although long-term

anticoagulation has been advocated for posterior circulation disease, the decision to use heparin in PCA stroke should be made on an individual basis.

Aspirin has been shown to be effective in reducing recurrence of acute stroke in the first 14 days and should be started at admission .

Although deep vein thrombosis (DVT) is unusual in patients with PCA stroke, any patient who is bedridden should receive prophylactic therapy for DVT.

Once the stroke mechanism has been determined, long-term secondary preventive treatment can be started. This would include anticoagulation for a cardioembolic source (eg, atrial fibrillation), intrinsic vertebrobasilar disease, or vertebral dissection. When the mechanism is cryptogenic, antiplatelet therapy generally is used.

Surgical Care:

Surgical and endovascular therapies aimed at the posterior circulation are largely experimental and are considered only as research protocols or in unusual circumstances. At present, little evidence exists to support medical therapy or surgical/endovascular therapy beyond case series and retrospective reports. Nevertheless, several procedures have been described and they include

- **Vertebral artery bypass and occlusion**

Extracranial (EC)-to-intracranial (IC) vertebral artery bypass may be undertaken by connection of the occipital artery to the vertebral, superior cerebellar, anterior ICA, or posterior ICA. The superficial temporal artery also has been used as a donor artery. Shunting to the PCA may be accomplished by using veins or synthetic grafts. In general, EC-to-IC circulation shunting has been relegated to use in extenuating circumstances since publication of the negative EC-IC bypass trial [30,31,32].

Atherosclerotic disease of the vertebral artery orifice has been treated with bypass (usually a common carotid to vertebral graft) and by subclavian artery reconstruction. Although endarterectomy is possible, it is done rarely. The proximal vertebral artery may be amenable to angioplasty and stenting [30 33,34].

Occlusion of the vertebral artery may be undertaken to exclude an aneurysm, dissecting aneurysm, or pseudoaneurysm from circulation. Rarely, dissection of the vertebral artery extends into the intracranial segment, ruptures, and causes subarachnoid hemorrhage. Similarly, vertebral artery dissection may fail to heal completely, leaving a proximal pseudoaneurysm as a source of future emboli. The traditional solution to these problems is to sacrifice the vertebral artery with proximal occlusion.

- Angioplasty and stenting

Angioplasty and stenting of the vertebrobasilar circulation are increasingly being reported. Nevertheless, where medical therapy has failed to control symptoms, accumulating reports of successful vertebral and even basilar angioplasty and stenting demonstrate the potential of this procedure.

Preliminary results of angioplasty or stenting of occlusive vertebral-arterylesions in the neck show that restenosis is more common than with carotid-artery stenting [33].The small diameter and angulation of the vertebralartery origin complicate endovascular treatment. Intracranial vertebral- and basilar-artery angioplasty and stenting have produced mixed results, with a relatively high rate of complications.[23]

MATERIALS AND METHODS

All patients who presented to the medical wards and out patient department of general medicine and neurology in GGH Chennai with symptoms and signs of posterior circulation stroke from the period of May 2005 to June 2006 were included in the study

CT SCAN OF BRAIN, ECG, CXR, ECHOCARDIOGRAM, 4 VESSEL DOPPLER AND FASTING LIPID PROFILE was performed in all these patients. MRI was done in affording patients.

INCLUSION CRITERIA

1. Patients who had signs and symptoms of posterior circulation stroke and those

2. Patients who had radiological evidence of posterior circulation stroke were included in the study.

EXCLUSION CRITERIA

1. Patients with clinical features and neuro - otological features of vestibular disorders and labyrinthine disorders

2. Patients with sub arachnoid hemorrhage, extra dural or subdural hemorrhage.

All patients were

- registered in the proforma regarding their age, sex, occupation and income
- questioned for the symptoms of posterior circulation stroke like dizziness, diplopia, dysphagia, nasal regurgitation, speech disturbances, headache, seizures, altered sensorium, unsteadiness, sensory disturbances, incoordination, motor weakness.
- a detailed history regarding the risk factor profile in the patients including systemic hypertension, smoking, alcoholism, dyslipdemia, diabetes mellitus, coronary artery disease, rheumatic heart disease, peripheral vascular disease, previous anterior circulation stroke, family history was documented

A detailed clinical examination on the neurological status of the patients and comprehensive examination of other systems including were recorded in the case sheets.

All patients were investigated for basic biochemical, hematological investigations. ECG, ECHO, CHEST X RAY were done in all patients. Patients with stroke in age less than 40 were also evaluated for hypercoagulable states (hyperhomocytienemia, protein C and S deficiencies, Antiphospholipid antibodies). MRI with MRA was done in selected patients.

Patients previous treatment history was analysed and classification of their premorbid hyperternsion according to JNC VII was made in those patients whose records were available.

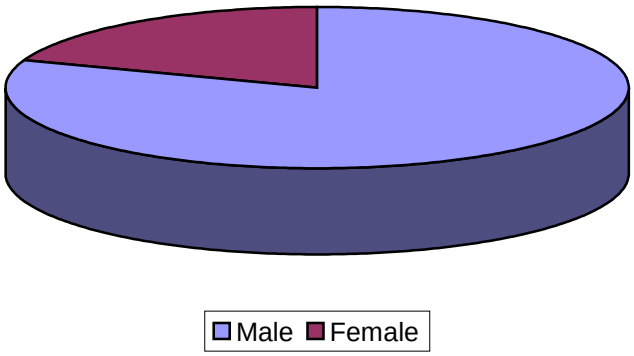
Carotid and vertebral artery Doppler was done in all patients and the degree of disease in the vessels were documented.

The nature of the study was observational and prospective one.

OBSERVATION

The study group included 41 patients. There were 33(80.5%) male and 8(19.5%) female patients. The ratio of male to female is 4:1. The age distribution ranged from as low as 13 years to upto 70 years, with the majority being males of elderly age group.

SEX DISTRIBUTION



The age distribution is shown in the Table 1

Age group In years	Males (n=33)	Females (n=8)
11-20	2	0
21-30	1	1
31-40	3	0
41-50	7	1
51-60	13	3
>61	7	3

RISK FACTOR PROFILE

The risk factor profile was analyzed in the 41 patients and the observation are shown in decreasing order of frequency in this Table 2

RISK FACTOR	**No (%) n=41**
Hypertension	26(63.4%)
Smoking	18(44%)
Alcoholism	17(42%)
Diabetes mellitus	16(39%)
Hyperlipidemia	12(29%)
Embolism from heart	9(21%)
Coronary artery disease	8(19%)
0ral anticoagulant use	6(14%)
Hyperhomocystienemia	2(05%)
Anti phospholipid antibody	1(02%)
Takayasu arteritis	1(02%)

CLINICAL PATTERNS

In the 41 patients 31(75%) patients had ischemic infarct , 10(25%) had hemorrhage and 2(5%) had hemorrhagic infarct. The symptoms analysis in this group revealed the following characteristics in decreasing order of frequency

Table 3

SYMPTOMS	NUMBER(%)
Giddiness and vomiting	23 (56%)
Incoordination	19 (46%)
Headache	13 (31%)
Seizures	9 (22%)
Motor weakness	7 (17%)
Sensory disturbance	7 (17%)
Altered sensorium	6 (15%)

The clinical examination revealed the following findings in decreasing order of frequency

Table 4

CLINICAL FINDINGS	NO OF PATIENTS (%)
Cerebellar signs	24(58%)
Visual field defects	22(53%)
Hemisensory loss	14(34%)
Motor weakness	13(31%)
Multiple cranial nerve palsy	6(15%)
Combination of clinical findings	27(66%)

The radiological evaluation revealed the following findings in decreasing order of frequency.

Table 5

RADIOLOGICAL FINDING	NO OF PATIENTS (%)
Cerebellar infarct	16(39%)
Medullary infarct	12(29%)
Cerebellar hemorrhage	4(10%)
Occipital hemorrhage	4(10%)
Brain stem infarcts	2 (05%)
Brain stem hemorrhage	2 (05%)
Combination of above	8 (10%)

The lipid profile abnormalities in patients with dyslipidemia is shown in this Table 6

LAB ABNORMALITY	NO OF PATIENTS (%)
HDL < 45 mg/dL	11(27%)
LDL > 100 mg/dL	9(22%)
Total Cholesterol >250 mg/dL	8(20%)
Triglyceride >200 mg/dL	4(10%)
Mixed abnormalities	6(14%)

The classification of hypertension among the patients showed the following features

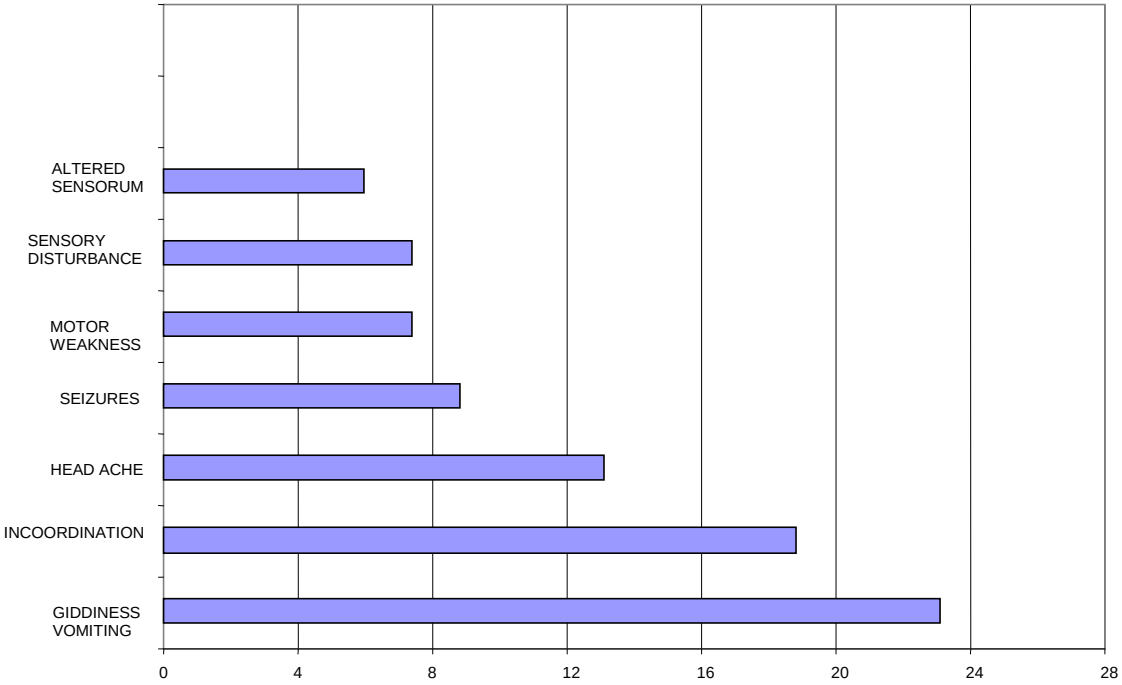
Table 7

JNC STAGE	**NUMBER(%)**
STAGE 1	8(20%)
STAGE 2	18(43%)

CHARTS AND GRAPHS

CHART 1

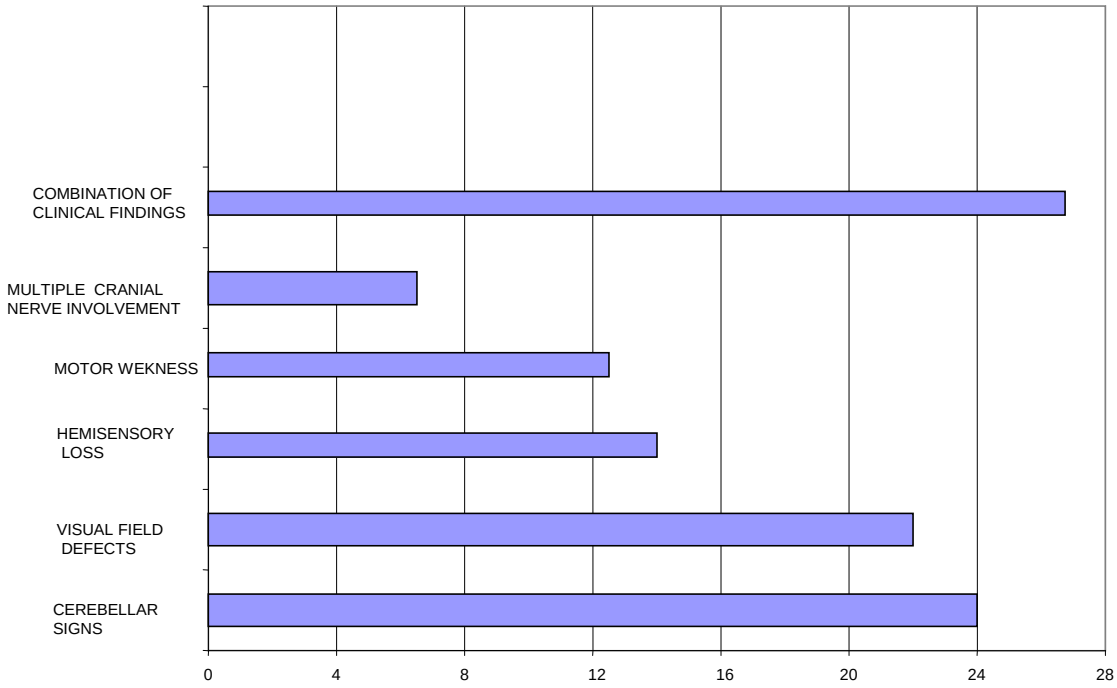
HISTORY

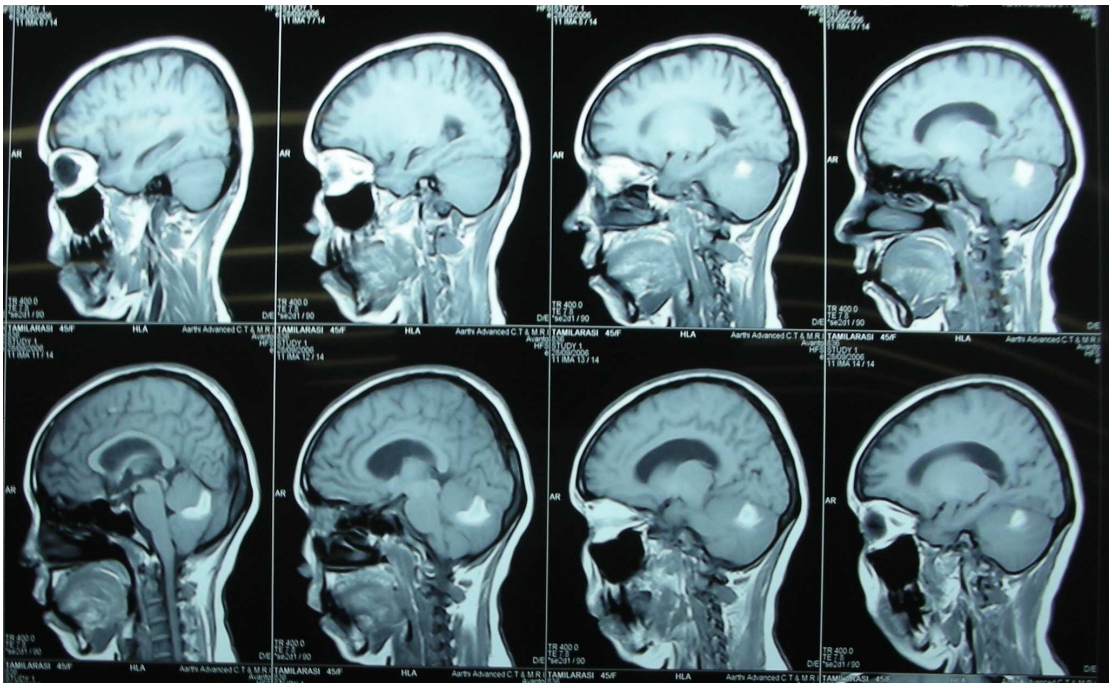


The clinical profile of the group of patients revealed

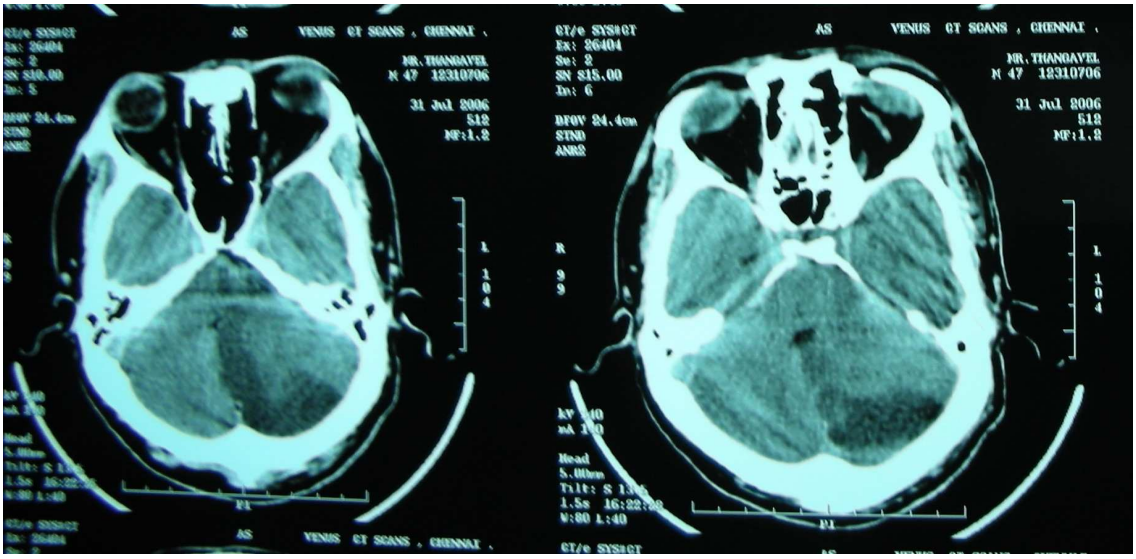
CHART 2

CLINICAL FINDINGS





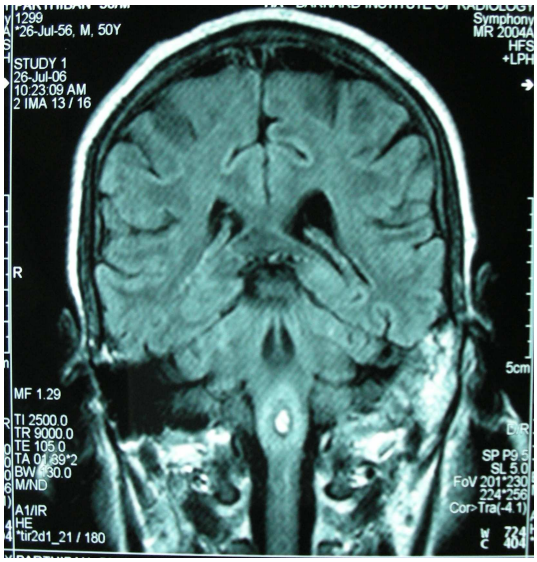
MRI OF CEREBELLAR HEMORRHAGE

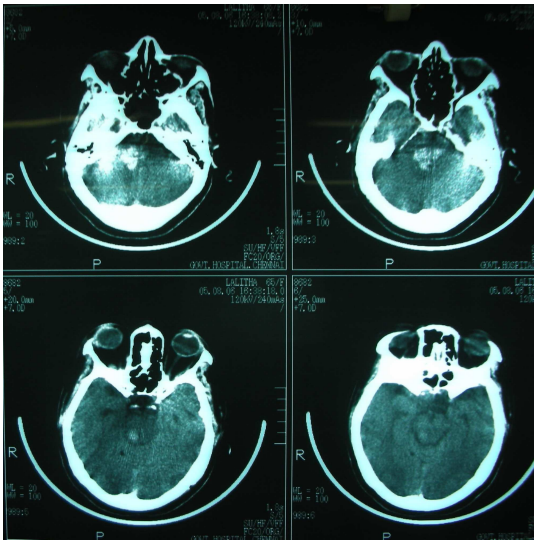


CT OF CEREBELLAR INFARCT

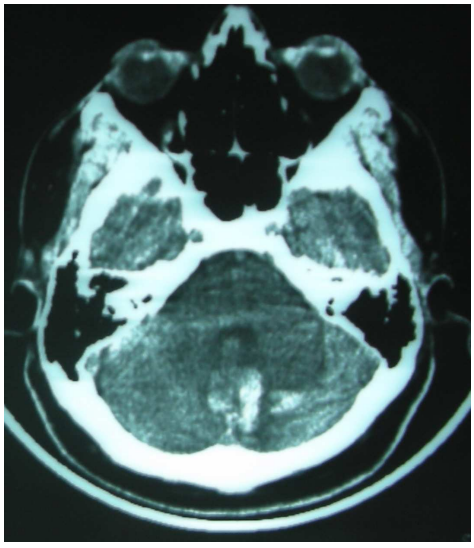
LEFT HORNER'S SYNDROME

HEMORRHAGIC INFARCT IN MEDULLA





CT OF BRAIN STEM HEMORRHAGE



CT OF CEREBELLAR HEMORRHAGE

CHART 3

MRI / CT Findings

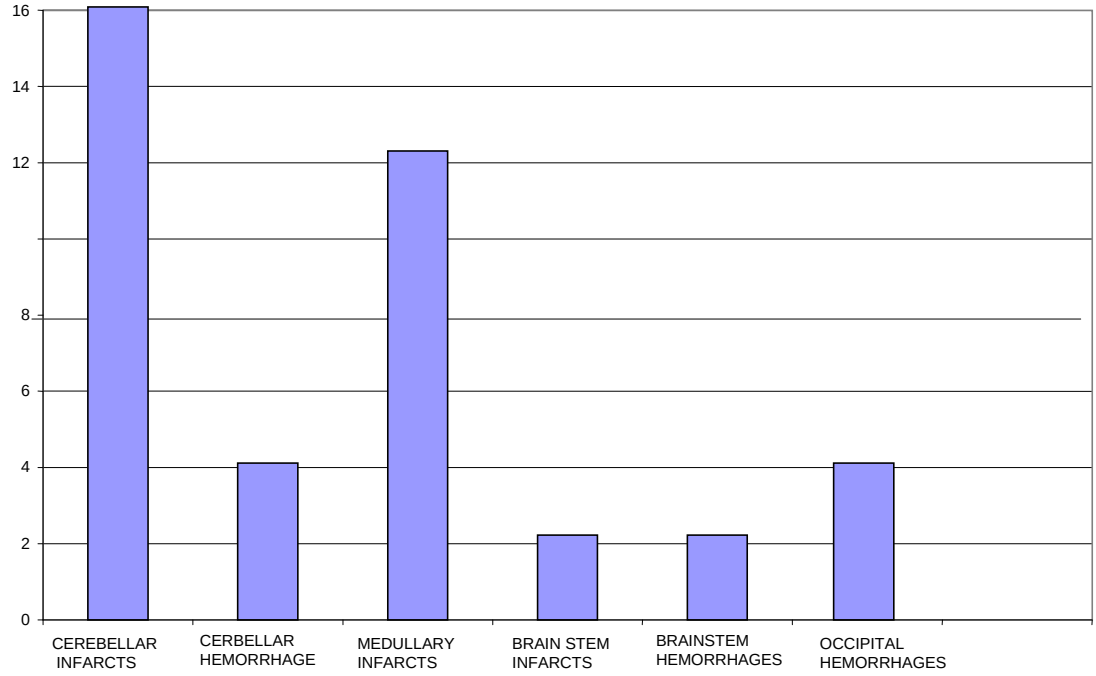


CHART 4

DOPPLER ABNORMALITIES IN THE STUDY GROUP

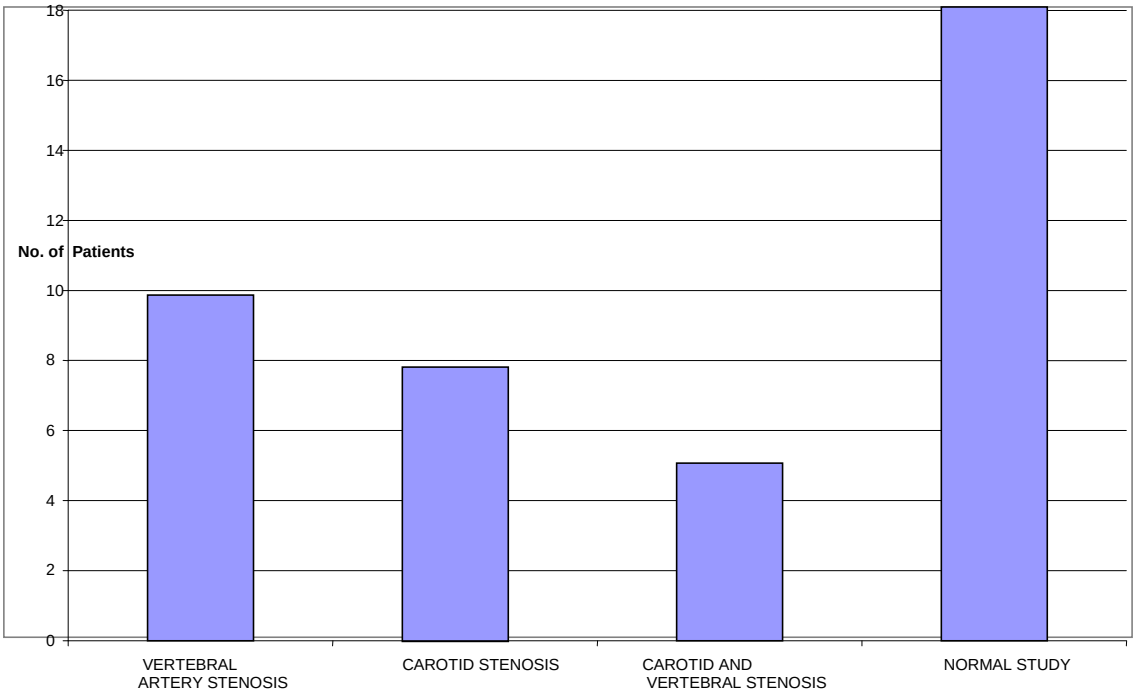


CHART 5

LIPID PROFILE ABNORMALITIES IN PATIENTS

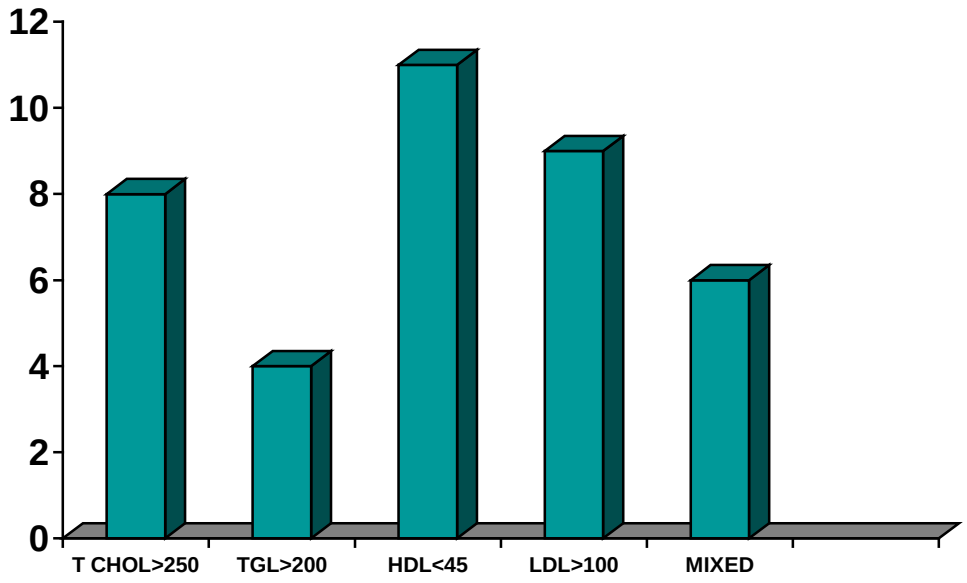
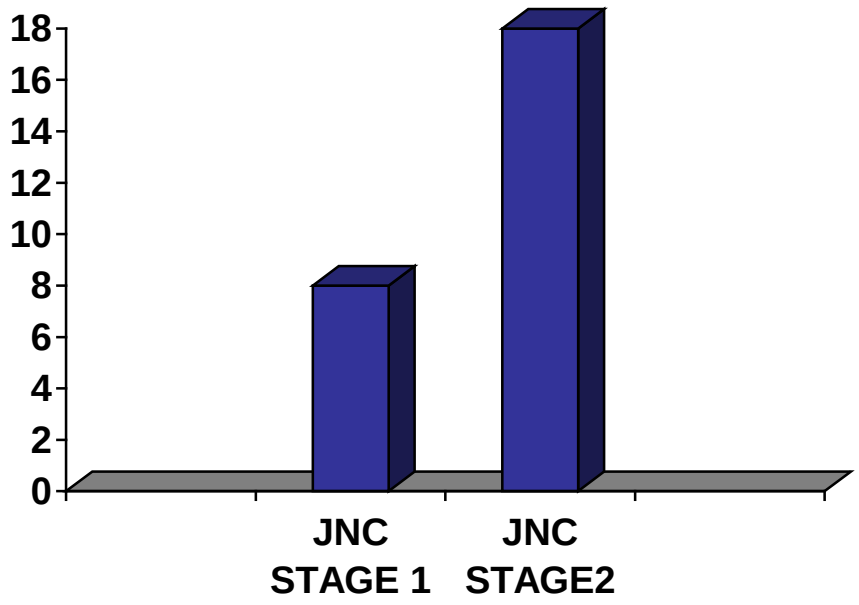


CHART 6

SYSTEMIC HYPERTENSION CLASSIFICATION ACCORDING TO JNC VII



DISCUSSION

In the study group of 41 patients, there was a male preponderance with a ratio of 4:1 as against in TUFTS posterior circulation registry[33] where there was 1:1 ratio. However another study by Corrado Argentino,et al[34] showed a male : female ratio of 4:1.

Posterior circulation stroke was common among individuals above 50 years . The mean age in the study group was 51 years.

The ratio between ischemic stroke and hemorrhagic stroke was 3:1 (31/10). This ratio was slightly higher when compared to anterior circulation stroke.

In females, out of the eight patients, six had hemorrhagic stroke (75%).as against in males where 29 out of 33 had ischemic infarcts predominantly (87%). In the females however there was history of oral anticoagulant use in three patients for known rheumatic heart disease induced atrial fibrillation.

As far as the the symptomatology was concerned , giddiness and vomiting , incoordination were the leading complaints during presentation occurring in almost half of the patients. Headache and seizures were present in 31 and 21 percent of patients and unilateral headache was striking feature in patients with cerebellar hemorrhage. In a study by R.B. Libman, et al[40] it was found that

headache was more common among posterior circulation strike patients,present in 15% of patients.

In seventeen percent of patients presented with motor weakness, it mimicked anterior circulation stroke clinically. This finding went hand in hand with the results of study by Corrado Argentino[34], et al which showed that shortly after onset the clinical discrimination between Anterior and atypical Posterior circulation infarcts is not reliable, which explains the frequent occurrence of this misdiagnosis. In such cases, emergency CT scan helps in the differential diagnosis only when it demonstrates an early focal hypodensity within the carotid territory. In the study by Huan et al[35], more than half of the patients had hemiparesis (58%).

Altered sensorium was present in fifteen percent of patients.

In a study by Timothy et al[14], vertigo without hearing loss was the commonest symptom in brain stem stroke syndromes. In this study also dizziness associated with vomiting was the commonest symptom.

A previous history of posterior circulation TIA was present in 15 percent and there were three patients who had previous anterior circulation stroke within a period of four years. This was in contrast to the study by Huan et al [35]which reported an incidence of TIA as 50%.

The cerebellar syndrome was the most common clinical presentation present in about 58 percent of individuals. Visual field defects including diplopia, hemianopia were present in 53 percent of patients. Hemisensory loss was present in about 34 percent (14 patients) in whom twelve had clinical features of lateral medullary syndrome. Multiple cranial nerve involvement was the presenting feature in fifteen percent of patients. The common cranial nerves affected include III,IV,V, VII, IX,X.

Two patients presented with complex oculomotor involvement as the sole presentation and MRI revealed brain stem infarcts in those patients. In the NEMC posterior circulation registry less than one percent of the 407 patients had only one complaint as presenting symptom [6].

One patient had simulataneous involvement of anterior and posterior circulations.

Hypertension topped the risk factor with 64 percent followed by smoking, alcoholism and diabetes mellitus in 44, 42 and 39 percent of patients respectively. Hyperlipidemia was present in 29 percent of patients .

Embolism from heart and coronary artery disease were present in about 21 and 19 percent respectively. This was comparable to NEMC Posterior circulation registry data where embolism from heart was demonstrated in 24% [6,33].

In the study of the nine patients who had embolism from the heart, seven had pre existing rheumatic heart disease and two had systemic hypertension and atrial fibrillation. Of the nine patients, six were on oral anticoagulant use.

In the study,among eight patients who had coronary artery disease as risk factor, three had extensive anterior wall myocardial infarction and five had inferior wall myocardial infarction pattern ECG wise which was confirmed by regional wall motion abnormalities in ECHO.

Comparison of risk factors in present study with study by Huan et al [35]

Table 8

RISK FACTORS	PRESENTSTUDY(n=41)	HUAN et al(n=31)
Hypertension	64%	71%
Smoking	44%	39%
Diabetes mellitus	39%	23%
CAD	19%	19%
Atrial fibrillation	17%	10%

Of the two patient who had posterior circulation infarcts within 20 years, one presented with diabetic ketoacidosis and he had bilateral posterior cerebral artery infarcts , whereas the other had bilateral cerebellar infarct and he had primary antiphospholipid antibody syndrome. The latter patient had bilateral hemorrhagic infarcts, which is quite rare presentation.

Another patient who had both anterior and bilateral posterior circulation infarcts proved to have Takayasu's arteritis.

One male patient presented with multiple infarcts in brainstem and cerebellum had no routine risk factors had elevated fasting homocystiene levels

Bilaterality of involvement was seen among patients who had hematological and metabolic disorders.

Among twelve(30%) patients who had dyslipidemia, low HDL (27%) was the most common abnormality followed by elevated LDL(22%), elevated total cholesterol(20%), elevated triglycerides(10%).a mixed pattern of the above abnormalities were found in 14% patients.

Alcoholism was strikingly prevalent among male patients who had cerebellar infarcts (fourteen of those sixteen had a strong history of alcoholism) . Among females hemorrhages involving the cerebellum accounted for almost half of all strokes.

Patients who had massive cerebellar infarcts and hemorrhages reported headache and had signs suggesting increased intracranial pressure. One patients with cerebellar bleed was successfully operated and other three conservatively managed.

Among 26 patients who had systemic hypertension before the stroke , analysis of the treatment records showed that 8 patients were in JNC stage I and 18 patients were in JNC stage II.

In the study population 24 patients had clinical features suggesting cerebellar involvement and the radiological investigations demonstrated isolated cerebellar involvement in twenty four patients (16 had infarcts and four had hemorrhage).

Of the fourteen patients suspected to have lateral medullary syndrome clinically, twelve had medullary infarcts and the other two had medullary and pontine infarcts.

Four patients suspected to have brainstem involvement had brainstem infarcts in two and hemorrhage in two patients.

One patients who had takayasu's arteritis had both MCA and PCA territory involvement.

In this study the observed one month mortality rate was 5%.(2/41). In general The rate of death immediately after posterior-circulation stroke is approximately 3 to 4 percent.38,39

In the NEMC-PCR, 3.6 percent of patients died, and 18 percent of patients had a major disability [36].

It was obvious that an intensive search for factors other than routine risk factors for stroke should be carried out , especially in individuals less than forty years.

SUMMARY

Out of the presented study following conclusions were derived

1. Male sex was the predominant population affected by posterior circulation stroke

2. Most commonly, people above 50 years were affected.

3. Uncontrolled hypertension, smoking and alcoholism were the major risk factors for posterior circulation stroke followed by diabetes mellitus.

4. Alcoholism was a major risk factor for cerebellar infarcts.

5. Low HDL was the commonest lipid abnormality among posterior circulation stroke patients.

6. Elderly females had a predilection to cerebellar hemorrhages for unknowm reasons.

7. Giddiness , vomiting and incoordination were the major presenting symptom of posterior circulation stroke.

8. Clinical features suggesting anterior circulation stroke can occur with posterior circulation stroke and careful visual field testing helps in clinical differentiation of both

9. Aggressive search for treatable risk factors should be made in younger patients

CONCLUSION

All patients with brain ischemia whether anterior or posterior circulation need extensive evaluation of risk factors especially modifiable. With the advent of newer investigations like MRI with DIFFUSION WEIGHTED IMAGING, MRA, CT ANGIO,EXTRA CRANIAL AND INTRA CRANIAL ULTRASOUND, the stroke mechanism could be precisely diagnosed.

Extensive cardiac work up and evaluation for hypercoagulable states proves to be useful in identifying correctable causes for stroke and hence secondary prevention.

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PROFORMA FOR STUDY OF POSTERIOR CIRCULATION STROKE

Name
Age
Sex
Occupation
Income
MRD No:
D.O.A
D.O.D

HISTORY (NEUROLOGICAL SYMPTOMS)

SYMPTOMS, PRESENT/ABSENT, DURATION

Dizziness

Headache

Unsteadiness/in-coordination

Visual Disturbances / Diplopia

Dysphagia/Nasal Regurgitation,

Weakness

Vomiting

Altered Sensorium

Convulsions
No.Of.Episodes
Type

Speech Disturbances

Head Trauma

Neuro psychiatric Manifestations

Chronology Of symptoms (Short history)

Progression of illness till interview

Static

Progressive

Regressive

Fluctuating

NON-NEUROLOGICAL SYMPTOMS

Neck Pain
Radiating arm Pain
Ear Discharge
Diarrhea
Bladder/Bowel Symptoms
Rheumatological/Musculo Skeletal complaints
Arthralgia
Oral ulcers
Alopecia
Skin rash
Photo Sensitivity
Raynauds Phenomenon
Others (Specify)

PAST HISTORY

Diabetes Mellitus

Hyper tension

CAD

RHD

CKD

Dyslipidemia

Smoking

Exposure History

Peripheral vascular disease

Previous CVA

Previous TIA

Family History

DRUG HISTORY

MENSTURAL HISTORY

OBSTETRIC HISTORY

TEMPORAL HISTORY

Duration from onset of symptoms to admission

Duration from onset of symptoms to CT /MRI evaluation

Duration from onset of symptoms to anto coagulation

EXAMINATION,

Febrile
Anemia
Clubbing
Edema
Jaundice
Temperature
Cyanosis
Lymphadenopathy
Ht
Wt
BMI
PR
BP

CNS EXAMINATION
Higher function
Consciousness
Drowsy
Normal
Stupor
Coma
GCS
Speech
Memory
Others(Specify)

CRANIAL NERVES,	**RIGHT**	**LEFT**	**NORMAL/PALSY**
I. Olfaction			
II.Visual acuity			
Field			
Color vision			
III, IV, VI			
Pupil size (Horners Syndrome)			
Ocular movts			
Palsy			
III/IV			
VI			
V			
VII - Facial lag			
UMN/LMN			
VIII			
IX			
X			
XI			
XII			

Spino motor system

	RIGHT	LEFT
UL Tone LL		
UL Power LL		
Reflexes		
Superficial 1. Corneal 2. Conjuctivial 3. abdomen 4. plantar		
UL Deep tendon reflexes LL		
Co-ordination		

Sensory System:

Touch		
Pain		
Vibration		
Joint Position sense		

GAIT

FUNDUS

Papilledema HT retinopathy DM retinopathy Retinal Hemorrahge		

EXTRA PYRAMMIDAL INVOLVMENT

BLADDER/AUTONOMIC INVOLVMENT

MENINGEAL SIGNS

CERBELLAR SYSTEM EXAMINATION

RIGHT LEFT

SPEECH (TYPE OF DYSARTHRIA)

TONE

INCOORDINATION

Heel shin test

Finger Nose incoordination

Disdidachokinesia

Tandem walking

RHOMBERG'S SIGN

TREMORS

MACROGRAPHIA

REBOUND PHENOMENON

NYSTAGMUS

PENDULAR KNEE JERK

DYSMETRIA

OTHER SYSTEMS EXAMINATION

CVS

S1

S2

MURMUR ABNORMAL FINDINGS

RS

ABDOMEN

CLINICALDIAGNOSIS

INVESTINATIONS

1.HEMOGRAM
Hb
TC
DC
ESR
PLATELETS

2.BLOOD SUGAR
BLOOD UREA
SERUM CREATININE
SODIUM,
POTASSIUM

3.LIPID PROFILE
TOTAL CHOLESTEROL
LDL
HDL
TRIGLYCERIDES

BT
CT
a PTT
PT
VDRL
HIV
X RAY CHEST
ECG
ECHO

DOPPLER 4 VESSEL

CT BRAIN

MRI BRAIN

OTHERS(SPECIFY)

FOLLOW UP

MASTER CHART

S NO	NAME	SEX	AGE	HT	DM	IHD	SMOKE	ALCOHOL	LIPIDS	CLINICAL FEATURES	CT/MRI	ECHO	DOPPLER	OTHERS
1	UDHUMAN	M	51	Y	N	N	Y	Y	N	giddiness vomiting	BRAIN	LVH	VERTEBRAL	
										hemisensory loss	STEM	DIAS.	DISEASE	
										visual field defect	INFARCT	DYSF		
										motor weakness		SCL		
										cranial nerve palsy				
2	IYALAMUDHAN	M	54	Y	N	N	Y	Y	N	giddiness vomiting	CEREBELLAR	LVH	VERTEBRAL	
										visual field defect	INFARCT RT		DISEASE	
										cerebellar signs RT				
3	NAGARAJ	M	41	N	N	N	Y	Y	N	giddiness vomiting	CEREBELLAR	NORMAL	NORMAL	
										incoordination	INFARCTLT			
										cerebellar signs LT				
4	SHANTHI	F	23	N	N	N	N	N	N	giddiness vomiting	MEDULLA	RHD	NORMAL	AF
										incoordination	INFARCT	MS		WARFARIN
										cranial nerve palsy		MR		
										seizures		PHT		
5	BABU	M	31	N	N	N	N	N	N	hemisensory loss	MEDULLA	NORMAL	NORMAL	HYPERHOMO CYSTIENEMIA
										visual field defect	INFARCT			
										incoordination	BRAINSTEM			
										cereebellar signs	INFARCT			
6	SOMIAH	M	49	Y	N	N	Y	Y	N	giddiness vomiting	CEREBELLAR	NORMAL	VERTEBRAL	
										incoordination	INFARCT LT		DISEASE	
										cerebellar signs LT				
										visual field defect				
										motor weakness LT				
7	HARIHARAN	M	34	Y	N	N	N	N	N	head ache	OCCIPITAL	LVH	VERTEBRAL	RENAL
										visual field defect	HEMORRGAGE	DIAS DYS	DISEASE	ARTERY
										motor weakness LT	RT			STENOSIS

8	GIREESH	M	13	N	N	N	N	N	N	giddiness vomiting	CEREBELLAR	NORMAL	NORMAL	PRIMARY
										head ache	INFARCT			APLA
										visual field defect	BILATERAL			
										bilateral cerebellar signs				
										altered sensorium				
9	SARVEEN KUMAR	M	14	N	Y	N	N	N	N	incoordination	MEDULLA	NORMAL	NORMAL	DKA
										hemisensory loss	INFARCT			
										visual field defect	BILATERAL			
										cranial nerve palsy	PCA INFARCT			
10	LALITHA	F	42	N	Y	N	N	N	Y	head ache	BRAIN	RHD	WARFARIN	AF
										motor weakness	STEM	MS		WARFARIN
										seizures	HEMORRHAGE	MR		
										altered sensorium		AF		
11	MUTHUKATHEEJA	F	53	Y	Y	N	N	N	Y	incoordination	BRAIN	DCM	CAROTID	
										hemisensory loss	STEM	LV DYS	AND	
										visual field defect	INFARCT	SEVERE	VERTEBRAL	
										motor weakness				
										cranial nerve V,IX,X				
12	SUBBURAJ	M	61	Y	Y	N	Y	Y	Y	giddiness vomiting	MEDULLA	LVH	CAROTID	
										cerebellar signs	INFARCT	MOD	DISEASE	
										hemisensory loss		LV DYS		
13	MAIMUNBEE	F	57	Y	N	N	N	N	N	head ache	OCCIPITAL	NORMAL	CAROTID	
										visual field defect	HEMORRGAGE		AND	
										motor weakness RT	RT		VERTEBRAL	
										seizures			DISEASE	
14	SHANMUGAM	M	37	N	N	N	N	N	N	giddiness vomiting	CEREBELLAR	NORMAL	NORMAL	HYPER
										incoordination	INFARCT RT			HOMO
										alteread sensorium				CYSTIENEMIA

15	DILLI	M	23	N	N	N	N	N	N	giddiness vomiting	BIL MCA	CONC	BILATERAL	TAKAYASU
										cerebellar signs	INFARCT	LVH	CAROTID	ARTERITIS
										hemisensory loss	PCA INFARCT		SUBCLAVIAN	
										visual field defect			VERTEBRAL	
										lt weakneaa			STENOSIS	
16	LOGANATHAN	M	59	N	Y	N	N	N	N	visual field defect	OCCIPITAL	NORMAL	NORMAL	
										seiizures	HEMORRGAGE			
										headache	RT			
										lt monoparesis				
17	JANAKIRAMAN	M	55	Y	Y	Y	Y	N	Y	incoordination	MEDULLA	IWMI	VERTEBRAL	
										cerebellar signs	INFARCT	DCM	DISEASE	
										hemisensory loss		SEVERE		
										visual field defect		LV DYS		
18	KANNIRAJ	M	66	Y	Y	Y	Y	Y	Y	giddiness vomiting	CEREBELLAR	AWMI	CAROTID	
										incoordination	INFARCT	NORMAL	DISEASE	
										cerebellar signs LT	LT	LV FUN		
19	SURESH	M	57	Y	N	N	N	N	N	head ache	BRAIN	NORMAL	NOT DONE	
										motor weakness	STEM			
										unconsciousness	HEMORRHAGE			
										seizures				
20	YESURAJA	M	59	Y	Y	N	Y	Y	N	giddiness vomiting	CEREBELLAR	LVH	CAROTID	
										incoordination	INFARCT RT		DISEASE	
										head ache				
										seizure				
21	CHINNAYAN	M	47	Y	Y	N	N	N	N	visual field defect	MEDULLA	RHD	VERTEBRAL	
										cerebellar signs	INFARCT	MS	DISEASE	
										hemisensory loss		PHT		

22	VISWANATHAN	M	58	Y	N	N	N	Y	N	giddiness vomiting	CEREBELLAR	NORMAL	CAROTID	
										cerebellar signs RT	INFARCT RT		AND	
										motor weakness			VERTEBRAL	
													DISEASE	
23	CHRISTOPHER	M	46	Y	Y	N	N	N	N	hemisensory loss	MEDULLA	NORMAL	NORMAL	
										cranial nerve palsy	INFARCT			
										horners syndrome LT				
24	MUSTAFA	M	63	Y	Y	N	Y	Y	N	giddiness vomiting	CEREBELLAR	LVH	NORMAL	
										incoordination	INFARCT LT	MILD		
										cerebellar signs LT		LV DYS		
										visual field defect				
25	VELRAJAN	M	45	N	N	N	N	N	N	hemisensory loss RT	MEDULLA	RHD	CAROTID	WARAFARIN
										visual field defect	INFARCT	MS TR	DISEASE	AF
										horners syndrome RT				
26	KALIAPPAN	M	57	Y	N	N	N	Y	N	giddiness vomiting	CEREBELLAR	NORMAL	VERTEBRAL	
										incoordination	INFARCTLT		DISEASE	
										head ache				
										cerebellar signs LT				
27	MALAYAPPAN	M	43	Y	N	N	N	Y	N	giddiness vomiting	CEREBELLAR	NORMAL	NORMAL	
										incoordination	INFARCT RT			
										cerebellar signs RT				
										motor weakness RT				
28	BALACHANDER	M	56	N	N	N	N	Y	N	giddiness vomiting	CEREBELLAR	RHD	CAROTID	AF
										incoordination	INFARCT LT	MS	DISEASE	WARFARIN
										cerebellar signs LT	LACUNAR	PHT		
											INFARCTS			

29	RAMALINGAM	M	51	N	N	N	Y	Y	N	cerebellar signs RT	MEDULLA	NORMAL	CAROTID	
										hemisensory loss LT	INFARCT RT		DISEASE	
										horners syndrome RT				
30	SRINIVASAN	M	67	N	Y	N	Y	N	Y	head ache	CEREBELLAR	IWMI	VERTEBRAL	
										cerebellar signs RT	HEMORRHAGE		DISEASE	
										visual field defect	RT			
31	NOORJAHAN	F	70	Y	Y	N	N	N	Y	giddiness vomiting	CEREBELLAR	IWMI	NORMAL	AF
										incoordination	HEMORRHAGE	LVH		WARFARIN
										head ache	RT	DIAS		
										cerebellar signs RT		DYSFN		
32	AKBAR	M	55	Y	Y	Y	Y	N	Y	hemisensory loss LT	MEDULLA	AWMI	CAROTID	
										cranial nerve palsy IX.X	INFARCT		DISEASE	
										visual field defect				
33	VELAN	M	42	Y	N	N	Y	N	N	giddiness vomiting	OCCIPITAL	NORMAL	NORMAL	
										LT Hemiaopia	HEMORRGAGE			
										head ache	RT			
										motor weakness				
34	NATARAJARATHINAM	M	67	Y	Y	Y	Y	Y	Y	cerebellar signs RT	CEREBELLAR	AWMI	VERTEBRAL	
										incoordination	INFARCT RT		DISEASE	
										seizures				
35	MAYILAPPAN	M	54	Y	N	N	N	N	N	incoordination	MEDULLA	NORMAL	CAROTID	
										hemisensory loss RT	INFARCT		DISEASE	
										cranial nerve palsy IX,X				
36	CHITHIRAI	M	55	N	N	N	Y	Y	Y	giddiness vomiting	CEREBELLAR	NORMAL	NORMAL	
										cerebellar signs RT	INFARCT RT			
										seizures				

37	IRULAYEE	F	70	N	N	N	N	N	N	giddiness vomiting	CEREBELLAR	NORMAL	CAROTID	
										head ache	HEMORRHAGE		AND	
										cerebellar signs BL	BILATERAL		VERTEBRAL	
										visual field defect			DISEASE	
38	ARUMUGAM	M	62	N	N	N	Y	N	N	visual field defect	BRAIN	NORMAL	CAROTID	
										cranial nerve palsy III.VI	STEM		AND	
										altered sensorium	INFARCT		VERTEBRAL	
													DISEASE	
39	TAMILARASI	F	67	Y	N	Y	N	N	Y	giddiness vomiting	CEREBELLAR	IWMI	NORMAL	WARFARIN
										altered sensorium	HEMORRHAGE	LA		
										head ache	LT	thrombus		
										cerebellar signs LT				
										visual field defect				
40	JAMES	M	55	Y	N	N	Y	Y	N	giddiness vomiting	CEREBELLAR	LVH	NORMAL	
										cerebellar signs RT	INFARCT RT			
										motor weakness				
41	SIVANADAN	M	61	Y	Y	Y	Y	Y	N	giddiness vomiting	CEREBELLAR	IWMI	NORMAL	
										cerebellar signs LT	INFARCT LT	LVH		
										Incoordination		DD		
										seizures				