STUDY ON CLINICAL PATTERNS AND RISK FACTORS IN POSTERIOR CIRCULATION STROKE

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DEPARTMENT OF MEDICINE

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI – 627011

TAMILNADU

CERTIFICATE

This is to certify that this dissertation entitled "**STUDY ON CLINICAL PATTERNS AND RISK FACTORS IN POSTERIOR CIRCULATION STROKE**" is a bonafide original work of Dr. KIRON V NAIR in partial fulfilment of the requirement for M.D. (Branch-I) General Medicine Examination of the Tamil Nadu Dr.M.G.R Medical University, Chennai to be held in April 2014.

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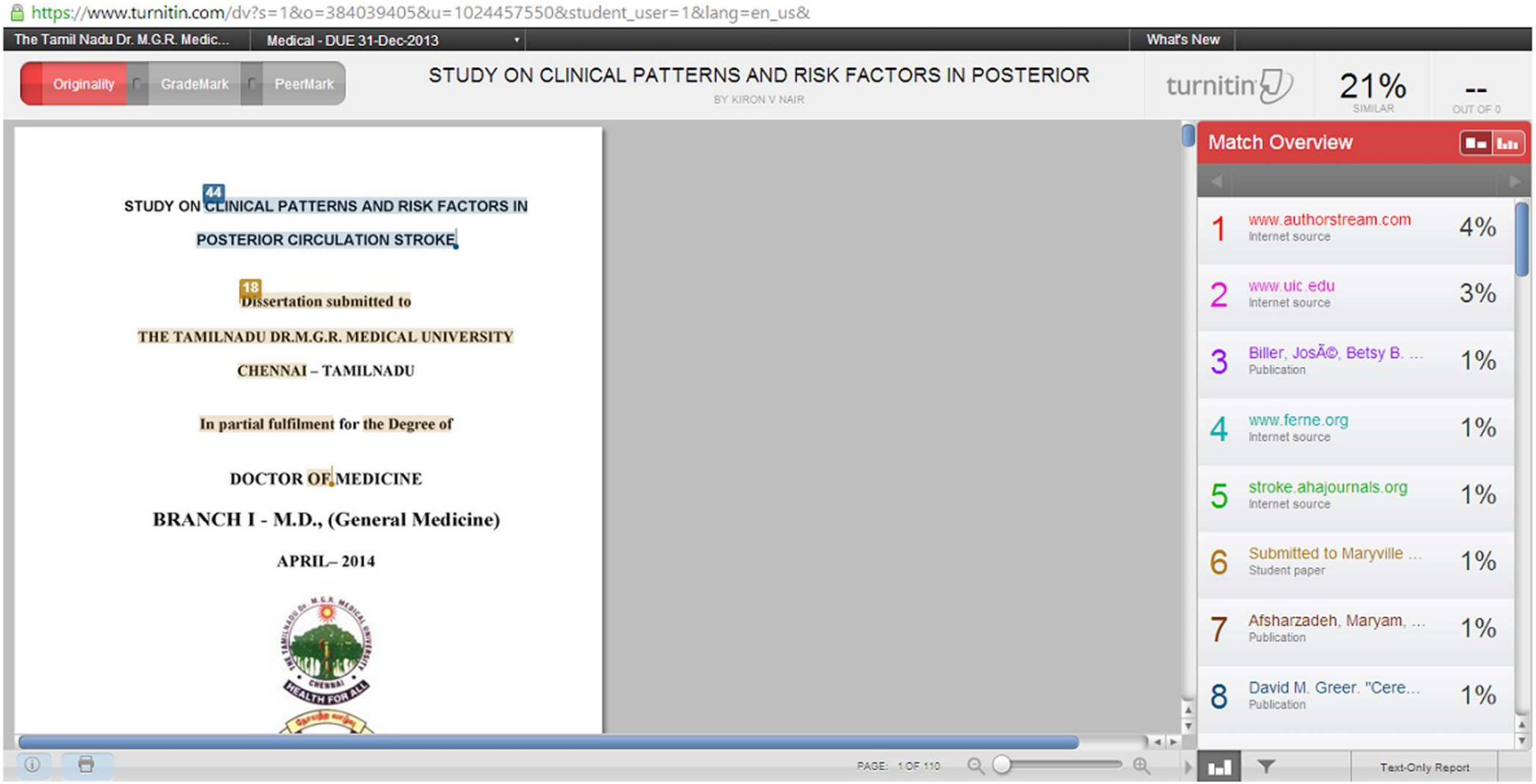
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DECLARATION

I **DR. KIRON V NAIR** declare that I carried out this work on **"STUDY ON CLINICAL PATTERNS AND RISK FACTORS IN POSTERIOR CIRCULATION STROKE"** at Department of General Medicine, Tirunelveli Medical College and Hospital during the period of July 2012 - October 2013. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree, diploma to any university, board either in India or abroad. This is submitted to the Tamilnadu Dr.M.G.R. Medical University, Chennai in partial fulfilment of the rules and regulation for the M.D. Degree examination in General Medicine.

Tirunelveli Medical College, **DR.KIRON V NAIR**

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CONTENTS

STUDY ON CLINICAL PATTERNS AND RISK FACTORS IN POSTERIOR CIRCULATION STROKE

ABSTRACT

AIM

1. To study the risk factors and clinical profile of patients admitted to Medical wards and intensive medical care unit of Tirunelveli Medical College Hospital with posterior circulation stroke.
2. To study the distribution of infarcts in different territories within the posterior circulation.
3. To compare the results with NEMC –Posterior circulation stroke registry and other available similar studies.

MATERIALS AND METHODS

This was a cross sectional observation study conducted in all patients admitted to the General Medicine wards and Intensive Medical care unit with clinical features suggestive of posterior circulation stroke. All patients with evidence of posterior circulation stroke in clinical features and/or imaging were taken up for the study based on strict inclusion and exclusion criteria.

Patients' details regarding various risk factors and clinical features were recorded on a well thought out and carefully prepared proforma.

The data was analysed and the results were compared with other available similar studies.

Results

- The average age of patients in this study was 60.2 years. Of the 50 patients studied, 74 % were males and 26 % were females. Most of the patients were above 50 years of age. Hypertension was found to be the most important risk factor (64 %) followed by smoking, hyperlipidemia and diabetes. Smoking was found to be more important than hypertension among males (75 %). The most common clinical symptoms were nausea and vomiting (54%), giddiness (50%), motor weakness (44%), visual field defects (36%) and ataxia (42%). Cranial nerve involvement was noticed in 28 % of patients. The territory of infarct commonly involved was the distal territory (48%), which includes midbrain, thalamus, occipital and temporal lobes and SCA supplied cerebellum. These results were compared with NEMC – PCSR and various other studies. The results were comparable between these studies.

Conclusions

All patients with brain ischemia deserve full evaluation of their brain for vascular lesions. Evaluation of risk factors and cardiac investigations are just as important as brain imaging, because a sizeable number of posterior circulation infarcts are cardio embolic, furthermore, brainstem infarcts, especially medullary infarcts can cause cardiac abnormalities. By

controlling these risk factors, we can reduce the incidence of posterior circulation stroke to a certain extent.

Key words: Posterior circulation stroke, clinical features, risk factors, NEMC –PCSR.

INTRODUCTION

Cerebrovascular accidents (Stroke) have been known since ancient times because of the characteristic clinical patterns they produce. Hippocrates (470 – 370 B.C.) described stroke as 'apoplexy' which means astonishment. [15]

The World Health Organization (WHO) defines stroke as, 'rapidly developing clinical signs of focal (or global) deficit of cerebral function, with symptoms lasting 24 hours or longer or leading to death, with no apparent cause other than vascular origin". [17]

Among neurological admissions to a hospital, more than 50% are strokes and in this, more than 85% is ischemic stroke. Of all Ischemic strokes, vertebrobasilar ischemic strokes account for 23% of first episodes of ischemic brain strokes, and 48% of these affect the brainstem.[4] Most ischemic brainstem strokes involve the pons (27%), followed by the medulla (14%), and the midbrain (7%).[5-8] 20-60 % patients have an unfavourable outcome. Basilar artery occlusion account for approximately 8 – 15% of posterior circulation strokes and carries mortality of more than 90%. [12]

It was Marburg in 1911, who first reviewed the subject matter of brain stem infarction and portrayed clinical cases of basilar territory syndromes. Later, in 1932, Pines and Gillinsky published an elaborate

report on serial sections of brainstem in a patient with basilar artery thrombosis. [16]

The posterior circulation is composed of the vertebral arteries which are paired, the basilar artery and the posterior cerebral arteries which are paired again, along with its branches.[3] It supplies approximately one-fifth of the total brain. Despite its comparatively smaller size, posterior circulation supplies the cerebellum, brainstem, subthalamus, thalamus, hippocampus, and medial temporal and occipital lobes. i.e., it supplies the strategically critical structures without which consciousness, motor activity, sensations and autonomic control over cardiovascular and respiratory systems cannot be preserved. Occlusion of each vessel in the posterior circulation produces its own distinctive syndrome. [3]

Posterior circulation Ischemia can present with clinical features ranging from subtle fluctuating brainstem symptoms attributable to intermittent insufficiency of the posterior circulation (VBI), to the more serious and debilitating syndromes like ‘locked in syndrome’ which is caused by either basilar artery or bilateral vertebral artery occlusion. [11]

Several factors are recognised to increase the burden of stroke. The most important of these include hypertension, atrial fibrillation, diabetes mellitus, cigarette smoking, and hyperlipidemia. Others, such as systemic diseases associated with hypercoagulable states, vasculitis due to

systemic diseases, use of birth control pills etc. contribute to a certain extent. [2]

The most common pathophysiologic mechanism of brainstem stroke includes embolism and lipo hyalinosis. An arterial dissection causes 20% to 30% of medullary strokes and approximately 5% of mesencephalic strokes but is rather rare in pontine strokes. [7, 9, 10]

Previously, it has been thought that posterior circulation stroke occurred largely due to local arterial atherosclerosis (large artery disease) and penetrating artery disease (lacunae). However, there is growing evidence that cardiogenic embolisation is more frequent than previously suspected and is found to be the cause in approximately 20-50% of strokes involving posterior circulation.[13, 14]

From literature, it was noted that there were only limited number of studies which evaluated posterior circulation stroke, especially in Indian population. Also, there are no clear hospital statistics available in India regarding the incidence of posterior circulation stroke.

The reason why this study on posterior circulation stroke was undertaken is that, it intends to shed some light into these grey areas by studying the associated risk factors and clinical patterns in our population and to compare the data with other available studies.

REVIEW OF LITERATURE

C.M. Fisher once remarked, house officers and students learn neurology "stroke by stroke",- as this cluster of diseases offered the most illuminating approach to localization in neurology. Among all the neurologic diseases of adult life, stroke ranks first in incidence and importance, i.e., no less than half of the neurologic disorders in a general hospital practice belongs to this group. [2]. In order to have a proper understanding of stroke and the area affected, we need to have a clear-cut idea of the anatomy and blood circulation of the central nervous system.

As we all know, Brain is nourished by two internal carotid and two vertebral arteries. These four arteries anastomose at the inferior surface of the brain to form the circulus arteriosus or circle of Willis.[18] The internal carotid artery and its branches constitute the anterior circulation of the brain and the paired vertebral arteries, the basilar artery, and the paired posterior cerebral arteries constitute the posterior circulation.[3]

Vascular Supply of the Cerebral Hemispheres

The blood supply of the brain is derived from branches of the aortic arch. Aortic arch gives rise to 3 major vessels: the brachiocephalic, the left common carotid, and the left subclavian arteries. The brachiocephalic, consecutively gives rise to the right subclavian and the

right common carotid arteries. The two common carotid arteries run upward just lateral to the trachea to level of the fourth cervical vertebra, where each bifurcates into the external carotid and internal carotid arteries.

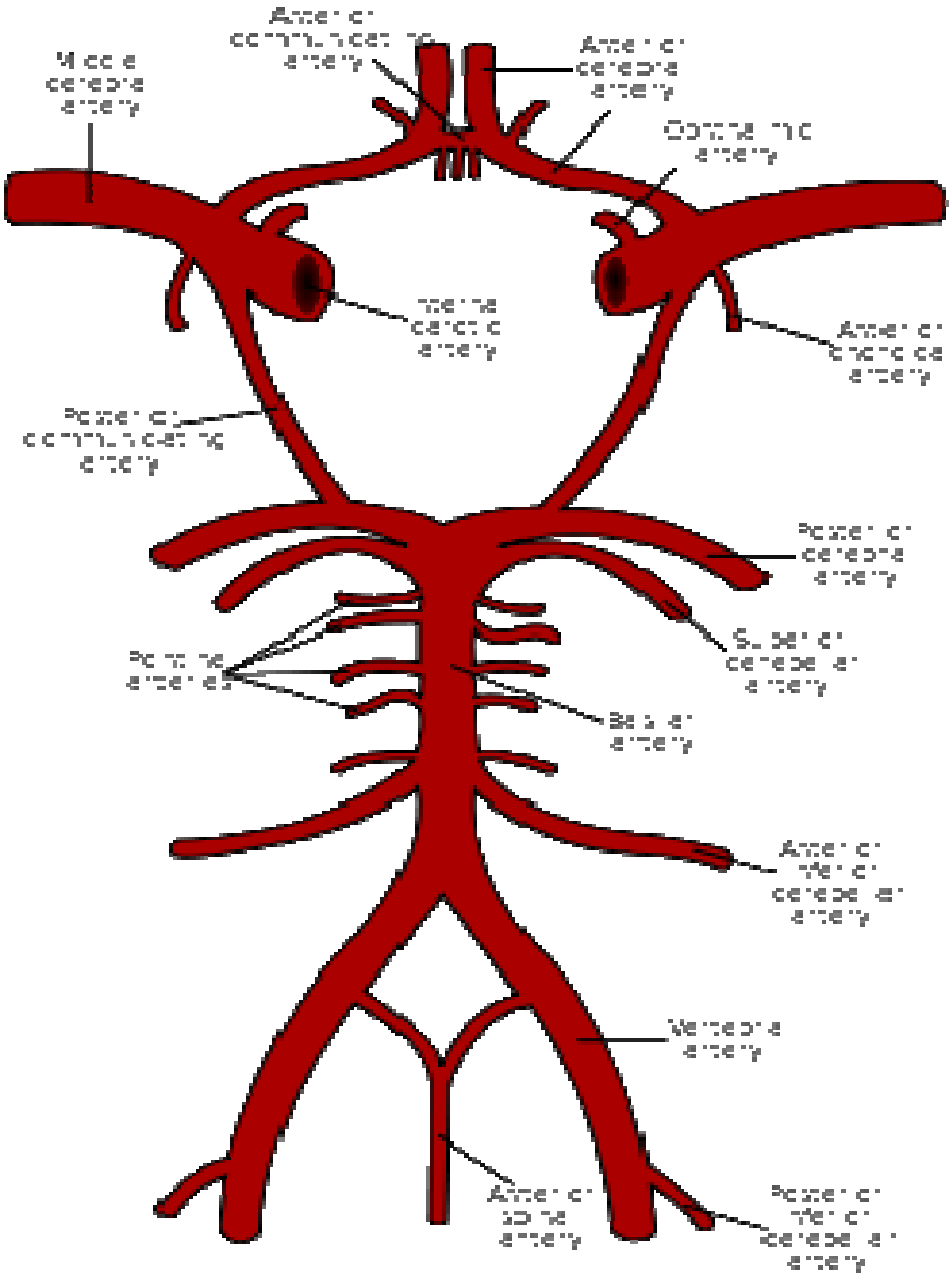

Fig: 1. Arterial supply of Brain

The posterior circulation is considered quite different from the anterior circulation and consists of vessels from each side which unite to form a Midline artery that supply the brainstem and spinal cord. There is a much higher incidence of asymmetric, hypoplastic arteries, variability in blood supply and persistence, of fetal circulatory patterns.

VERTEBRAL ARTERIES

The two vertebral arteries originate from their corresponding subclavian arteries just medial to the anterior scalene muscle and join together to form the basilar artery. After originating from the subclavian artery, it ascends up to the sixth cervical vertebra (1st part), go into the transverse foramen of C6 and run within the foramen transversaria upto C2 (2nd part). Then, it loops around the atlanto-occipital joint (3rd part), pierces the duramater to enter the cranial cavity through the foramen magnum and join the other vertebral artery at the medullo-pontine junction (4th part). Only the intra-cranial segment gives rise to branches that supply the brainstem and cerebellum. [1, 3, 18]

The intracranial portion of the vertebral arteries gives off posterior spinal arteries, anterior spinal arteries, penetrating medullary arteries and the large posterior inferior cerebellar arteries (PICAs). The posterior inferior cerebellar artery (PICA), in its proximal segment supplies the

lateral medulla and, in its distal part, the inferior surface of the cerebellum.

Atherosclerotic lesions have a predilection for the 1st and 4th segments of the vertebral artery. The first segment can be diseased at the origin of the vessel and may produce posterior circulation emboli. Presence of anastomotic channels between 2nd part of the artery and the arteries like ascending cervical, thyrocervical and occipital arteries prevent the occurrence of a low flow TIA or stroke in this case. But, in a setting, when one vertebral artery is atretic and an atherothrombotic lesion threatens the origin of the other, this collateral circulation, along with retrograde flow from the basilar artery, is often insufficient leading to the low flow TIAs in the form of vertigo, dizziness, syncope, alternating hemiplegia etc. In another scenario, when there is an occlusion of subclavian artery proximal to the origin of vertebral artery, there will be a reversal in the flow of blood in vertebral artery with exercise of the ipsilateral arm. This can lead to a posterior circulation TIA or “subclavian steal.”

Second and third segments of the vertebral artery are less prone for atherosclerosis. In turn, these are subjected more to dissection, fibromuscular dysplasia and rarely, encroachment by osteophytic spurs within the vertebral foramina leading to TIA.

Occlusion or thrombosis of the fourth segment causes ischemia of the lateral medulla leading to lateral medullary syndrome and occlusion of medullary penetrating branches can lead on to partial syndromes. Also, Paraplegia can occur in vertebral artery thrombosis if anterior spinal arteries are involved.

BASILAR ARTERY

It is formed by the union of both vertebral arteries at the ponto-medullary junction, ascends in the groove (sulcus basilaris) on the anterior surface of the pons and at the upper border of pons, it divides into two posterior cerebral arteries.

Branches from the basilar artery supply the base of the pons and superior cerebellum. They are grouped into four,

1. Paramedian branches: They are 7 to10 in number; perfuse a wedge shaped portion of pons on either side of the midline.
2. Short circumferential branches: These are 5–7 in number and perfuse the lateral two-thirds of the pons along with the middle and superior cerebellar peduncles.
3. Bilateral long circumferential branches (AICA and SCA): They take a path around the pons and nourish the cerebellar hemispheres.

4. Paramedian (interpeduncular) branches: arise from the bifurcation of the basilar artery and proximal posterior cerebral arteries: These supply the high midbrain and medial subthalamic regions.[2]

Atheromatous lesions are most frequent in the proximal basilar and distal vertebral segments, even though it can occur anywhere along the basilar trunk. Typically, lesions occlude either the proximal basilar along with one or both vertebral arteries. The clinical picture varies depending on the availability of retrograde collateral flow from the posterior communicating arteries. Eventhough atherothrombosis occludes the distal portion of the basilar artery, emboli arising from the heart or proximal vertebral or basilar segments are more commonly responsible for "top of the basilar" syndromes. [3]

As the brainstem contains a lot of structures in close apposition, brainstem ischemia can present with a diverse array of clinical syndromes, reflecting involvement of cranial nerve nuclei, the corticospinal, corticobulbar tracts and ascending sensory tracts.

POSTERIOR CEREBRAL ARTERY

The Posterior Cerebral Arteries arise from the rostral end of the basilar artery, within the interpeduncular cistern. It nourishes the occipital lobes and the inferior as well as medial portions of the temporal lobes, the mesencephalon, thalamus, and other structures. PCAs are the

terminal branches of the basilar artery in almost 75 % of cases. In about 20% of people, it originates from the ipsilateral ICA via the posterior communicating artery and in approximately 5 % individuals; both PCAs arise from the respective ipsilateral internal carotid arteries. In such cases, the precommunal or P1 segment of the true posterior cerebral artery is atretic.

PCA is divided into P1 segment, proximal to junction of PCA with posterior communicating artery and P2 segment distal to the junction.

The branches of the PCA have been divided into three groups [19]:

a) The penetrating arteries to the brainstem, thalamus, and other deep structures,
b) The dorsal callosal artery which anastomose with distal branches of the ACA, and
c) The cortical branches
    1. the anterior temporal,
    2. posterior temporal,
    3. parieto-occipital, and
    4. calcarine arteries.

The P1 portion of the PCA, giving rise to the interpeduncular branches (the largest of which is called Artery of Percheron), is also referred to as the mesencephalic artery or the basilar communicating artery. The arterial configuration of the paramedian mesencephalic

arteries varies considerably: in some cases, two small vessels arise symmetrically, one from each side; in others, a single artery arises from one posterior cerebral stem (proximal P1), which then bifurcates. In the latter case, one posterior cerebral stem supplies the medial thalamic territories on both sides, and an occlusion of this artery or one common paramedian trunk produces a bilateral butterfly-shaped lesion in the medial parts of the diencephalon. [20]

PCA syndromes usually result from atheroma formation or emboli that lodge at the top of the basilar artery; posterior circulation disease may also be caused by dissection of either of the vertebral artery or fibromuscular dysplasia.

COLLATERAL CIRCULATION IN BRAIN [1]

There are three main sources of collateral circulation to the brain that compensate in cases of carotid or basilar occlusion:

a) The circle of Willis, located on the ventral surface of the brain, which connects the anterior (internal carotid) and posterior (vertebrobasilar) circulation with each other,
b) Anastomoses between branches of the extracranial and intracranial arteries, and
c) Leptomeningeal anastomoses between the terminal branches of the major arteries of the cerebrum and cerebellum.

The most important intracranial anastomoses are those of the circle of Willis. Atypical configurations of the circle of Willis resulting from hypoplasia of one or more component stems are found in 79% of individuals. [1]

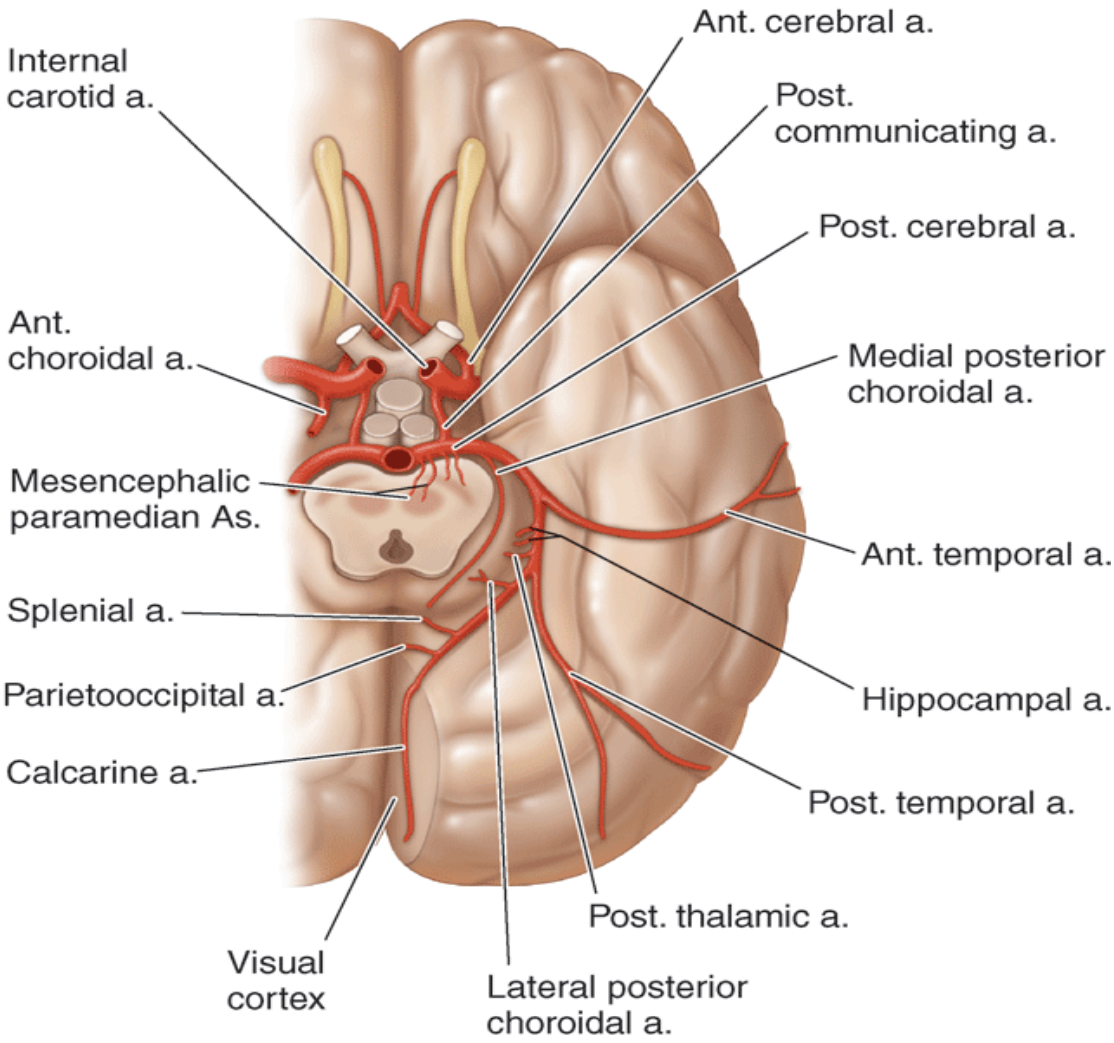

Fig -2, Inferior aspect of the brain with branches and distribution of PCA[3]

PATHOPHYSIOLOGY OF ISCHEMIC STROKE

The brain is a metabolically active organ. More than any other organ, the brain depends, moment to moment on an adequate supply of oxygenated blood. It uses glucose as its sole substrate for energy metabolism except in prolonged starvation, when other sources like amino acids are being used.[21] A constant supply of Adenosine Tri-Phosphate (ATP), which is derived from glucose metabolism is needed to maintain neuronal integrity and to keep the major extra- cellular cations Ca++ and Na+ outside the cells and the intracellular cation K+ within the cells.

The brain requires and uses approximately 50ml of oxygen and 75-100 mg of glucose each minute and a total of 125g of glucose each day. When the body is resting, human brain uses approximately 20% of cardiac output. Cerebral blood flow is approximately 50 ml for each 100 gm of brain tissue each minute and cerebral oxygen consumption is normally approximately 3.3 ml/100 g/min. [21, 22]

Energy consumption and blood flow in the brain depend on the degree of neuronal activity .PET and functional magnetic resonance imaging have proved unequivocally, that using the right hand increases, metabolism and cerebral blood flow in left motor cortex.

A critical occlusion of an intracranial vessel leads to a decline in blood flow to the brain territory it supplies. The extent of flow reduction

is a function of collateral blood flow and this depends on several factors which include the ones like the site of occlusion, individual vascular anatomy, and likely systemic blood pressure. A complete cessation of cerebral blood flow causes death of brain tissue within four to ten minutes; flow <16–18 mL/100 g of tissue per minute bring about infarction within an hour; and values <20 mL/100 g of tissue per minute leads to ischemia with no infarction unless prolonged for many hours or days. If blood flow is reinstated prior to a significant amount of cell death, the patient will experience only transient symptoms, and the clinical syndrome is called a TIA. [2, 3]

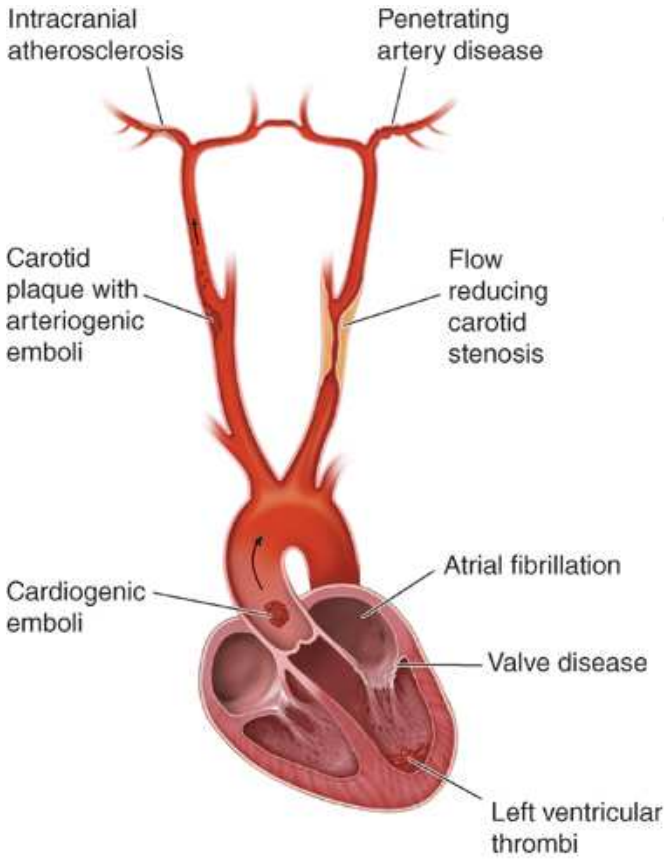

Fig.3. Diagram illustrating the major mechanisms that underlie ischemic stroke

As evidenced from the illustration, an ischemic stroke can be produced by a thrombotic, embolic or vasculitic phenomenon. Emboli can arise either from the heart or from an atheromatous plaque in large vessels. Also, ischemia can result from the narrowing and thrombus formation in a vessel or lipohyalinosis of penetrating vessels.

Once blood flow to a part of the brain is compromised, the survival of the tissue at risk depends on a number of modifying factors such as availability of collateral circulation, duration of ischemia, magnitude and rapidity of the reduction of flow and resistance of brain tissue ischemia.

Brain tissue encircling the core of infarction is ischemic but reversibly dysfunctional and is often referred to as the ischemic penumbra. The penumbra may be visualised by imag by using perfusion-diffusion imaging with MRI or CT. The ischemic penumbra wills eventually infarct if no change in flow occurs, and hence saving the ischemic penumbra is the goal of revascularization therapies. [2, 3]

The capacity of the cerebral circulation to maintain relatively constant level of cerebral blood flow despite changing blood pressure has traditionally been termed “auto regulation”. Cerebral blood flow remains constant when mean arterial blood pressures are between 50 and 150 mm of Hg. This accommodation eventually fails at the extremes of blood pressure, after which cerebral blood flow follows systemic pressure passively, either falling precipitously or rising to levels that damage the

walls of small vessels. The conditions in which the limits of autoregulation are exceeded are at the extremes of hypertensive encephalopathy at one end and circulatory failure at the other. [3, 2, 23]

Pathologically, cerebral infarction occurs by means of two clear-cut pathways: [3],

1. A necrotic pathway where there is cytoskeletal damage mainly due to energy failure of the cell; and
2. An apoptotic pathway where the cells become programmed to die.

Histopathologic changes that occur in an infarction are grouped into three categories. [23, 3,]

1. **Early changes:** These changes occur within 12 to 24 hours of the insult. Initially there will be microvacuolisation of the affected cell followed by eosinophila of the cytoplasm. Later, there will be nuclear pyknosis and karyorhexis giving rise to what is known as red neurons. Analogous acute changes soon follows in other cells like astrocytes and oligodendroglia. The cells which are most affected by a short duration ischemia, include Pyramidal cells of the Sommer sector (CA1) of the hippocampus, cerebellar Purkinje cells, and neocortical pyramidal cells. Normal reaction to this early tissue injury commence with infiltration by neutrophils.

2. **Subacute changes:** These changes occur within 24 hours to 2 weeks. During this period, the tissue will be necrosed, macrophages are recruited to the site, and there will be vascular proliferation, and reactive gliosis.
3. **Repair:** This phase occur approximately after 2 weeks. During this phase, all necrotic tissue will be removed and as a result of this along with the subsequent gliosis, a distortion of the normal organized CNS structure occurs. It is found that there will be a bumpy destruction of the the cerebral cortex with selective involvement of certain layers and preservation of others. This pattern of involvement is termed pseudolaminar necrosis.

The size, location, and shape of the infarct and the extent of tissue damage that results from focal cerebral ischemia brought about by occlusion of a blood vessel are determined by the modifying factors

DISTRIBUTION OF VASCULAR PATHOLOGY IN POSTERIOR CIRCULATION

Sites of predilection for atherosclerotic narrowing in the posterior circulation include the proximal origins of vertebral arteries and the subclavian arteries, the proximal and distal ends of intracranial vertebral arteries, the basilar artery and the origin of the posterior cerebral arteries. Atherosclerotic narrowing rarely affects the distal superficial branches like PICAs, AICAs and SCAs.

Lipohyalinosis and medial hypertrophy secondary to hypertension affect mainly the thalamogeniculate penetrators from the posterior cerebral arteries and paramedian perforating vessels to the pons, midbrain and thalamus from the basilar artery.

Atheroma formation or emboli that lodge at the top of the basilar artery or along the P1 segment may cause symptoms by occluding one or more of the small brainstem — penetrating branches. Occlusion in the posterior cerebral artery distal to the junction with the Posterior communicating artery (P2) may disrupt small circumferential branches.

Atherothrombotic lesions have a fondness for V1 and V4 segments of the vertebral artery. The first segment may become diseased at the origin of the vessel and may produce posterior circulation emboli. Collateral flow from the contra lateral vertebral arteries or the ascending cervical, thyrocervical or occipital arteries is usually adequate to prevent low flow transient ischemic attacks or stroke.

When one vertebral artery is atretic and an atherothrombotic lesion threatens the origin of the other, the collateral circulations, which may also include retrograde flow down the basilar artery is often insufficient. In this setting, low flow Transient ischemic attacks may occur, consisting of syncope, vertigo, and alternating hemiplegia. Disease of the distal fourth Segment (V4) of the vertebral artery can promote thrombus formation manifest as embolism or with propagation of basilar artery

thrombosis. Stenosis proximal to the origin of the posterior inferior cerebellar artery can threaten the lateral medulla and the posterior inferior surface of the cerebellum.

If the subclavian artery is obstructed proximal to the origin of the vertebral artery, there can be a retrograde flow in the ipsilateral vertebral artery. In such a condition, exercise of the ipsilateral arm will raise demand on vertebral flow further, producing posterior circulation TIA's 'subclavian steal syndrome.'

Although atheromatous disease rarely narrows the V2 and V3 segments of the vertebral artery, this region is subject to dissection, fibromuscular dysplasia and rarely encroachment by osteophytic spurs within the vertebral foramina.

Atheromatous lesions can crop up anywhere along the basilar trunk but are most frequent in the proximal basilar and distal vertebral segments.

Embolism from heart, proximal vertebral or basilar segments are more commonly responsible for 'Top of the basilar' syndromes.

Traumatic or spontaneous dissection usually involves the distal extra cranial carotid and vertebral arteries. Temporal arteritis affects the vertebral arteries just before they pierce the duramater to enter the cranial cavity.

Emboli can block any artery depending on the size and the nature of the embolic materials. In posterior circulation, emboli preferably block the intracranial vertebral artery, distal basilar artery and the Posterior cerebral arteries.

The extent and size of the infarct depend on the pace of occlusion, adequacy of collateral circulation, and resistance of brain structures to ischemia.

RISK FACTORS

The risk factors for posterior circulation strokes are the same as for other forms of cardiovascular disease. These are either modifiable or non-modifiable.

Table.1

Risk factors of Posterior circulation stroke.

Modifiable risk factors	Non modifiable risk factors
• High blood pressure • Cigarette smoking or exposure to second hand smoke. • Hyper cholesterolemia - total cholesterol level above 200mg/dL • Diabetes • Being overweight or obese • Physical inactivity • Obstructive sleep apnoea • Cardiovascular disease - heart failure, structural heart defects, infective endocarditis,arrhythmia. • Use of oral contraceptives or hormone replacement therapies that include estrogen. • Heavy or binge drinking. • Use of illicit drugs such as cocaine and methamphetamines.	• Age being 55 or above • Gender — Men have a higher risk of stroke than women. • Personal or family history of stroke, heart attack or TIA. • Race – African Americans • Heredity

Stroke risk increases considerably with each decade past age 55. [24] In women, stroke usually occurs at a later age and for the same reason, mortality is higher in them when compared with men.

It is estimated that about 15% of all strokes are preceded by a TIA. Several studies also reveal that, after a TIA, the short term risk for a stroke is about 10% at 2 days and approximately 17% at 90 days. In patients who have survived this initial high risk phase, the risk for a stroke during the next 10 years is roughly 19%. It is also interesting that, 12% of patients will succumb to death from a major cardiovascular event, either stroke or myocardial infarction within a period of one year of TIA. [24, 25, 26, 27]

It is very well established that, stroke producing potential of hypertension is very high for both ischemic stroke and hemorrhagic stroke. The treatment and lowering of BP among hypertensive patients was found to be associated with a significant reduction in stroke risk. [24]

As we all know, cigarette smoking is considered as one of the traditional and established modifiable risk factor for stroke. This includes both ischemic and hemorrhagic strokes, but the data which connects intracerebral hemorrhage with smoking is less consistent. When smokers who quit smoking for more than 10 years and non-smokers, were compared with current smokers, the incidence was significantly high in

current smokers (2-4 times). Across age, sex and race groups, stroke risk has been shown to come down with discontinuation of smoking. [24, 28]

Future risk of thromboembolic stroke was also found to be more likely in those with a low HDL cholesterol, high LDL cholesterol and raised triglycerides. [30]

Impaired glucose tolerance almost doubled the stroke risk when compared with patients having normal glucose levels and risk was three times high for patients with established DM. Ischemic stroke patients with DM are of relatively younger age and more likely to have hypertension, MI, and high cholesterol than nondiabetic patients.[24, 29]

Atrial fibrillation (AF) is a well established risk factor for stroke, all by itself increasing the risk approximately 5-fold throughout all ages and up to 18 fold if associated with rheumatic heart disease. [2, 24]

In Women, the risk of stroke was found to be increased in those who take estrogen containing oral contraceptive pills and those who undergo hormone replacement therapy.

Hereditary factors also play a role in the occurrence of stroke. In certain studies ,it was found that parental ischemic stroke by the age of 65 years was associated, in offspring, with a 3 times increase in ischemic stroke risk, even after adjustment for other stroke risk factors. [24]

Others like heavy or binge drinking, use of cocaine or methamphetamines are also associated with increased risk of stroke.

CLINICAL FEATURES

TIAs

TIAs are short-lived episodes of acute, focal, Nonconvulsive neurologic dysfunction presumably due to reversible ischemia to an area of the brain. To qualify as a TIA, the episode should be followed by complete recovery, and no neurologic residua should be detected after 24 hours.

Table - 2. Vertebrobasilar and Carotid TIA, a comparison.

Symptoms of Transient Ischemic Attacks		
Symptom	**Vertebrobasilar Artery Territory**	**Carotid Artery Territory**
Motor deficit	Bilateral or shifting weakness, clumsiness, or paralysis; ataxia, imbalance, or disequilibrium not associated with vertigo	Contralateral weakness, clumsiness, or paralysis
Sensory deficit	Bilateral or shifting numbness; paresthesias, including loss of sensation	Contralateral numbness; paresthesias, including loss of sensation
Speech disturbance	Dysarthria	Dysphasia, dysarthria
Visual disturbance	Diplopia, partial, or complete blindness in both homonymous visual fields	Ipsilateral monocular blindness (amaurosis fugax), contralateral homonymous hemianopia
Others	Combination of the above	Combination of the above

COMPLETED STROKE

A completed stroke refers to the temporal profile of the stroke syndrome in which the deficit is prolonged and often permanent, causing demonstrable parenchymatous changes. Most completed strokes reach the maximum of neurologic dysfunction within an hour of onset.

STOKE IN EVOLUTION

Stroke in evolution or progressive stroke, describes the temporal profile in which the neurologic deficit progresses in a stepwise fashion, culminating in a major deficit in the absence of treatment. If the site of ischemia is in the carotid arterial distribution, the evolution is usually complete within 24 hours. But, if the site of ischemia is the vertebrobasilar arterial system, the deficit may progress for up to 72 hours.

Progression may result from recurrent embolism, propagating intraluminal thrombus, inadequate collateral circulation, cerebral oedema, intracranial haemorrhage, or intercurrent medical complications.

As already mentioned, the posterior circulation irrigates the cerebellum, medulla, pons, midbrain, subthalamus, thalamus, hippocampus, and medial temporal and occipital lobes through its circumferential and penetrating branches. Occlusion of each vessel produces its own distinctive clinical syndrome.

Patients usually present with a wide array of symptoms of neurological dysfunction that includes motor weakness in the form of hemi or quadripareis, cerebellar involvement in the form of ataxia, cranial nerve involvement (mainly III-XII), autonomic disturbances , altered level of consciousness, vertigo etc. If brainstem is involved at multiple levels, multiple cranial nerves will be involved. Multiple cranial nerve signs indicate involvement of more than one brain stem level. Patients may also present with only hemi paresis, which may evolve rapidly to quadriparesis or a locked in syndrome. The onset of symptoms, most of the times will be gradual in contrast to the sudden onset in anterior circulation strokes.

As the posterior circulation provides nutrition to the cerebellum, brainstem, and occipital cortex, the symptoms often present, were dizziness, dysarthria, dystaxia, nausea and vomiting, headache, decreased alertness, diplopia, dysphagia and visual field disturbances.

Strokes involving brainstem is characterised by the typical crossed findings, which means cranial nerves are involved on one side and sensory as well as motor findings will be found on the other side of the lesion. Most of the times, the location of infarct can be traced with the help of these symptoms, if one have a decent knowledge of neuroanatomy.

POSTERIOR CEREBRAL ARTERY SYNDROMES

Posterior Cerebral Artery syndromes usually occur as a result of atheroma formation or emboli that get entrapped at the top of the basilar artery. Posterior circulation disease may also be caused by dissection of either vertebral artery or as a result of fibromuscular dysplasia. Occlusion of PCA leads to two distinct clinical syndromes.

1. P1 syndrome: *due* to the involvement of the proximal segment (P1 segment or the precommune segment) of the PCA or its penetrating branches which include thalamogeniculate, Percheron, and posterior choroidal arteries. This will lead to midbrain, thalamic and subthalamic signs.
2. P2 syndrome: due to blockage of the distal segment (P2 segment or postcommune segment) of the PCA which will lead to cortical temporal and occipital signs.

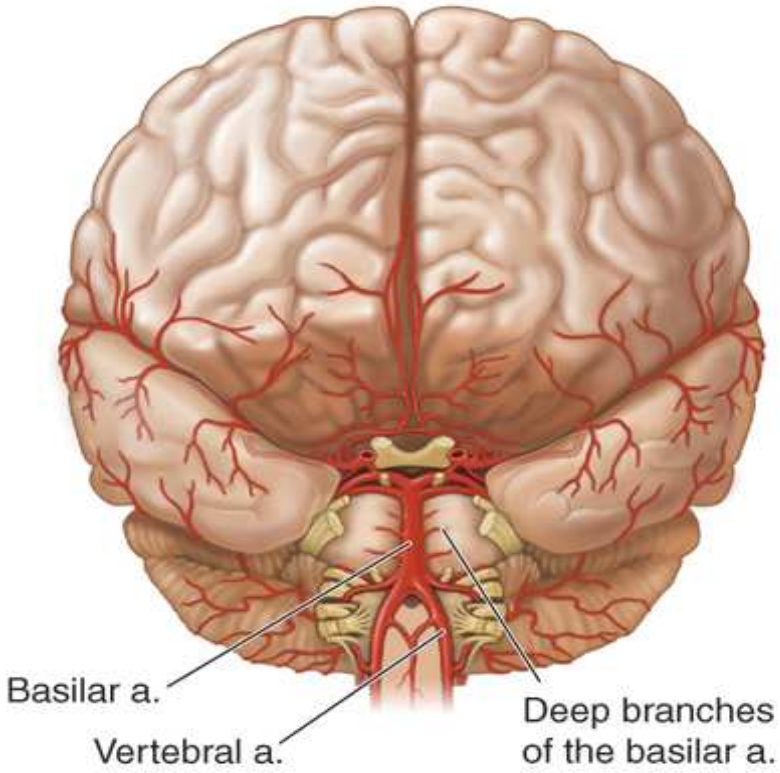

Fig.4. Deep penetrating arteries and peripheral branches of posterior circulation

P1 SYNDROMES:

Thalamic syndrome: all modalities of sensations are lost. Patient may have mild hemiparesis, spasms of the hand, choreoathetosis, intension tremors, dysesthesias and spontaneous pain .

Ventral posterolateral nucleus of thalamus in the territory of thalamogeniculate artery is primarily involved. Involvement of the adjacent subthalamic nucleus or its pallidal connections results in hemiballismus and choreoathetosis.

Thalamoperforate syndrome: Also known as central midbrain and subthalamic syndromes. This occur as a result of occlusion of interpeduncular and paramedian arteries. A superior crossed cerebellar ataxia or an inferior crossed cerebellar ataxia in which third cranial nerve also will be involved in which case, it is known as the **Claude's syndrome**. Dentatothalamic tract and third cranial nerve are the involved structures.

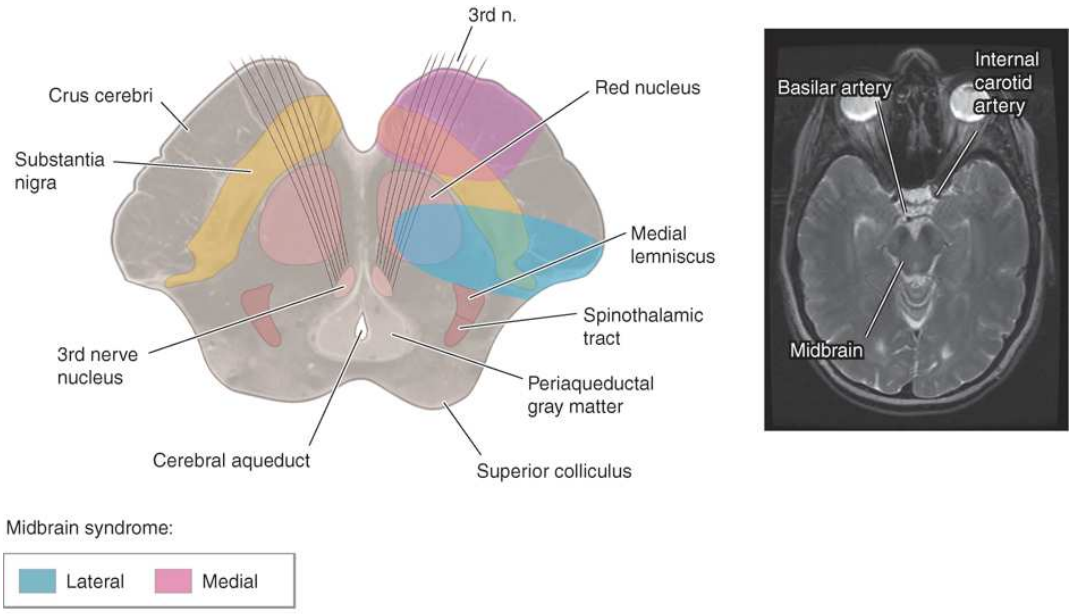

Fig.5. Midbrain Syndromes

Weber syndrome: Third-nerve palsy and contralateral hemiplegia as a result of involvement of third nerve and the cerebral peduncle.

Benedikt syndrome: Oculomotor palsy with contralateral cerebellar ataxia, tremor, and corticospinal signs as a result of infarct or hemorrhage in the tegmentum of midbrain and involve red nucleus, corticospinal tract, and brachium conjunctivum.

In **Nothnagel syndrome**, patient presents with ocular palsies, paralysis of gaze and cerebellar ataxia due to involvement of unilateral or bilateral 3rd nerve and superior cerebellar peduncles in the tectum of the midbrain. In **Perinaud syndrome**, dorsal midbrain is involved leading to paralysis of upward gaze and accommodation with fixed pupils. These two syndromes are seen more commonly with tumours and are rarely encountered in vascular lesions.

Also, patients can present with contralateral hemiplegia only, if cerebral peduncle alone is involved. They may also present with paralysis of vertical eye movement, skew deviation, sluggish pupillary responses to light, minor miosis and ptosis (retraction nystagmus and "tucked-in" eyelids), if supranuclear fibers to third nerve, high midbrain tegmentum ventral to superior colliculus are involved. Damage to motor tracts between red and vestibular nuclei will lead to decerebrate posturing.

Occlusion of the penetrating branches of thalamic and thalamogeniculate arteries produce less extensive thalamic and thalamocapsular lacunar syndromes.

The thalamic syndrome of Dejerine and Roussy results from obstruction in the thalamogeniculate branches, leading to infarction of the sensory relay nuclei in the thalamus. There is both a deep and cutaneous sensory loss, of the opposite side of the body, including the trunk and face, sometimes accompanied by a transitory hemiparesis. A

homonymous hemianopia may be also be present. The characteristic feature is always a sensory loss that includes the entire half of the body up to the midline. After an interval, sensation begins to return, and the patient may develop pain, paresthesia, and hyperpathia in the affected parts which may persist for years.

P 2 SYNDROMES

Occipital lobes and medial temporal lobes are involved if there is an obstruction in the distal segment of posterior circulation artery. Contralateral homonymous hemianopia with macular sparing is the usual presentation. Occasionally, only the upper quadrantanopia alone occurs instead of hemianopia. Macular, or central, vision is often spared as a result of collateral blood supply to the occipital pole from distal branches of the middle or anterior cerebral arteries.

Other features seen in a few instances are visual hallucinations in the blind parts of the visual fields or metamorphopsia and palinopsia. Occipital infarcts of the dominant hemisphere may cause alexia without agraphia, anomia, a variety of visual agnosias, and rarely some degree of impaired memory.

If hippocampus and medial part of the dominant temporal lobe is involved, patient may present with acute memory disturbances. The defect usually wanes off, because memory has representation on both sides. If the dominant hemisphere is affected and the infarct extends to

the splenium of corpus callosum, the patient may demonstrate alexia without agraphia. Occlusion of posterior cerebral arteries produce peduncular hallucinosis.

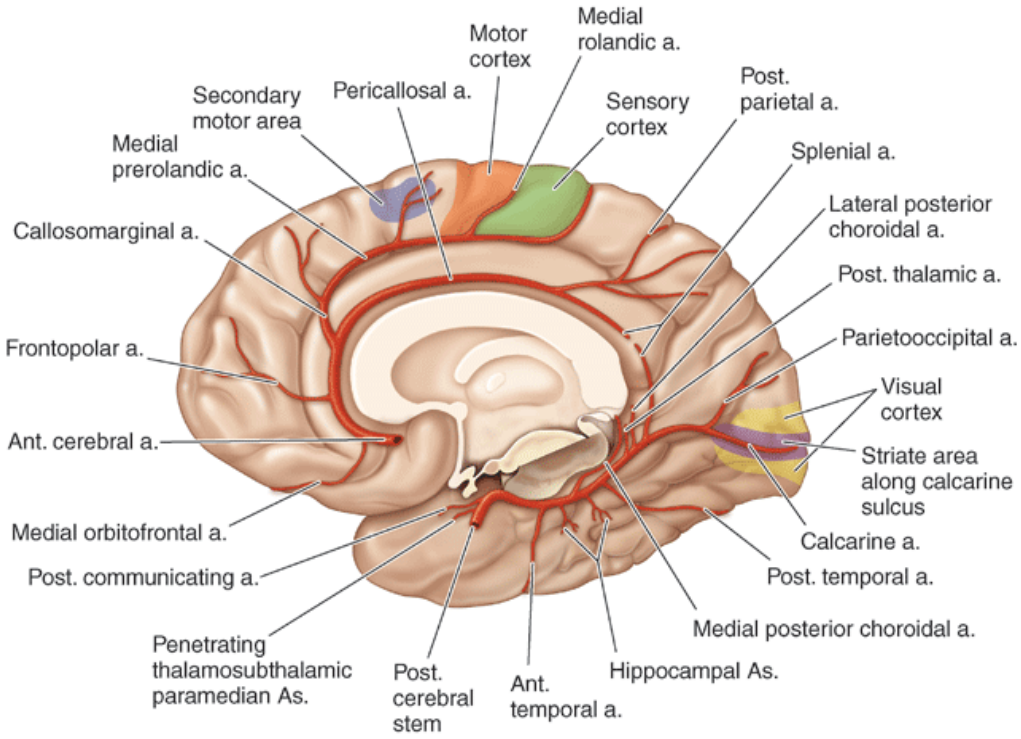

Fig.6. Cortical branches of Posterior cerebral artery

ANTON's SYNDROME.: This occurs in cortical blindness resulting from infarction in distal PCA territory involving both occipital lobes. Pupillary light reaction will be preserved in cortical blindness and the interesting feature is that the patient the patient will not be aware of his blindness, or may even deny it.

BALINT's SYNDROME: This refers to a constellation of symptoms which is seen with the involvement of visual association areas on both sides. This is described as a defect in the normal orderly visual scanning of the environment. In this condition, patients may encounter persistence

of a visual image for quite a few minutes despite gazing at another sight (palinopsia) or an inability to synthesize an image in totality.(asimultanagnosia).

With bilateral lesions that involve the inferomedial portions of the temporal lobes, including the hippocampi and their associated structures, the impairment of memory may be severe, causing the Korsakoff amnesic state.

VERTEBRAL AND POSTERIOR INFERIOR CEREBELLAR ARTERY SYNDROMES

The vertebral arteries are the chief arteries of the medulla; each supplies the lower three-fourths of the pyramid, the medial lemniscus, all or nearly all of the retroolivary (lateral medullary) region, the restiform body, and the posteroinferior part of the cerebellar hemisphere through the posterior inferior cerebellar arteries.

The occlusion of these arteries produce two distinct syndromes.

1. **Lateral medullary syndrome** : This occurs as a result of occlusion of vertebral, PICA, Inferior medullary artery, middle medullary artery or superior medullary artery

   There will be involvement of the nucleus and descending tract of trigeminal nerve leading to pain and dyesthesias over one half of the face. Cerebellum may be involved, leading to ataxia.Vestibular

nuclear involvement can lead to vertigo, nausea & vomiting, diplopia, nystagmus etc. Involvement of CN-IX and X will lead to hoarseness, dysphagia and depressed gag reflex.Horners syndrome may also be present. Facial weakness occurs due to involvement of VII N nucleus.These features are seen on the same side of lesion.

On the opposite side, there will be impairment of pain and temperature sensation over half of the body due to spinothalamic tract involvement.

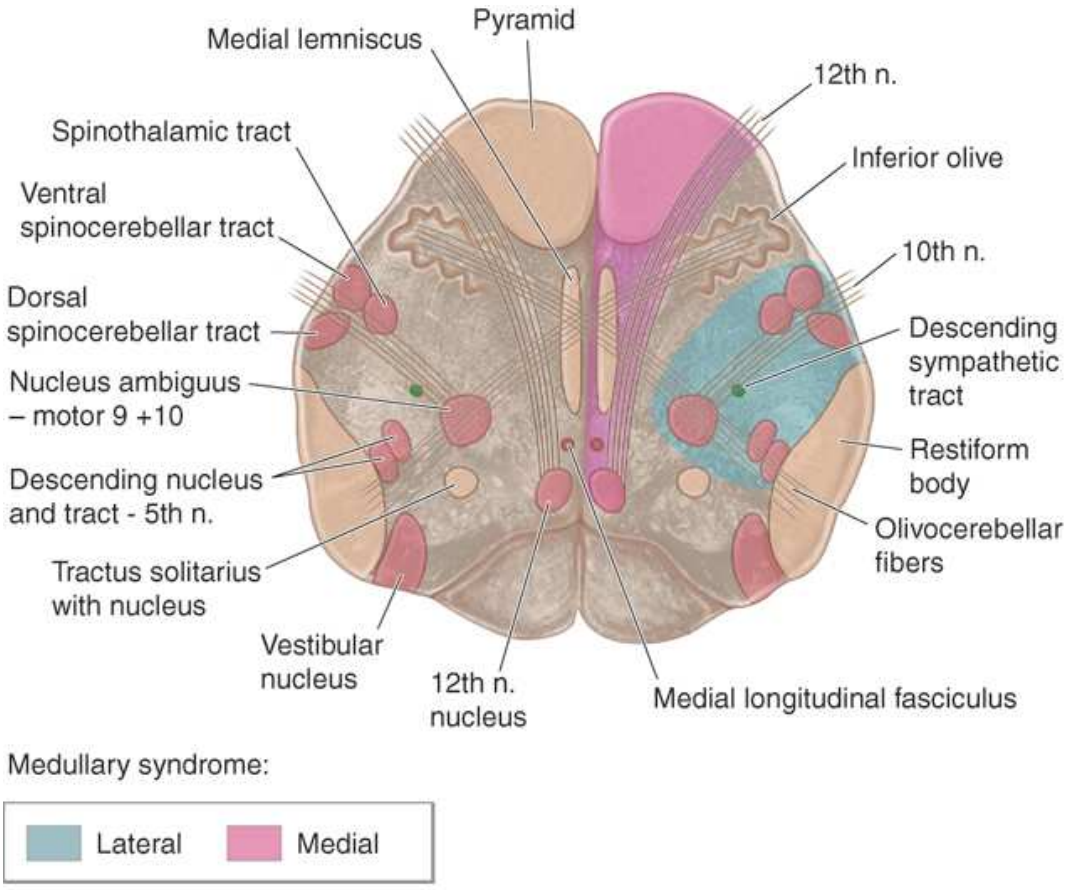

Fig.7 Medullary syndromes

2. **Medial Medullary Syndrome**: (occlusion of vertebral artery or of branch of vertebral or lower basilar artery)

   On side of lesion Paralysis with atrophy of one-half half the tongue due to Ipsilateral twelfth nerve.

   On side opposite lesion Paralysis of arm and leg, sparing face; impaired tactile and proprioceptive sense over one-half the body due to Contralateral pyramidal tract and medial lemniscus.

BASILAR ARTERY SYNDROMES

Basilar artery occlusion because of local thrombosis can arise in several ways:

1. Occlusion of the basilar artery itself, usually in the lower or middle third at the site of an atherosclerotic plaque;
2. Occlusion of both vertebral arteries, which produces the equivalent of basilar artery occlusion if the circle of Willis is inadequate; and
3. Occlusion of a single vertebral artery when it is the only one of adequate size.

Also, thrombosis may involve a branch of the basilar artery rather than the trunk (basilar branch occlusion).

The symptoms of transient ischemia or infarction in the territory of the basilar artery often do not indicate whether the basilar artery itself or

one of its branches is diseased. In general, symptoms of basilar branch TIAs affect one side of the brainstem, whereas symptoms of basilar artery TIAs usually affect both sides, although a "herald" hemiparesis can be an initial symptom of basilar occlusion. TIAs in the proximal basilar distribution may produce vertigo. Other symptoms that warn of basilar thrombosis include diplopia, dysarthria, facial or circumoral numbness, and hemisensory symptoms.

The syndrome of basilar artery occlusion, reflects the involvement of a large number of bilateral structures: corticospinal and corticobulbar tracts; cerebellum, middle and superior cerebellar peduncles; medial and lateral lemnisci; spinothalamic tracts; medial longitudinal fasciculi; pontine nuclei; vestibular and cochlear nuclei; descending hypothalamospinal sympathetic fibers; and the third through eighth cranial nerves.

The picture of complete basilar occlusion, however, is easy to recognize as a constellation of bilateral long tract signs (sensory and motor) with variable cerebellar, cranial nerve, and other segmental abnormalities of the brainstem. Or the patient is comatose because of infarction of the high midbrain reticular activating system. Others are mute and quadriplegic but conscious, reflecting interruption of descending motor pathways in the base of the pons with sparing the reticular activating system constituting the "locked-in" syndrome.

Midbasilar disease may also cause coma if the posterior communicating arteries are inadequate to perfuse the distal basilar artery territory but sparing of the reticular activating system.

Top of the basilar artery occlusion is most often embolic and characterized by transient loss of consciousness, oculomotor disturbances, hemianopia, and pupillary changes.

Occlusion of a branch of the basilar artery usually causes *unilateral* symptoms and signs involving motor, sensory, and cranial nerves. As long as symptoms remain unilateral, concern over pending basilar occlusion should be reduced. Various basilar branch syndromes are described below.

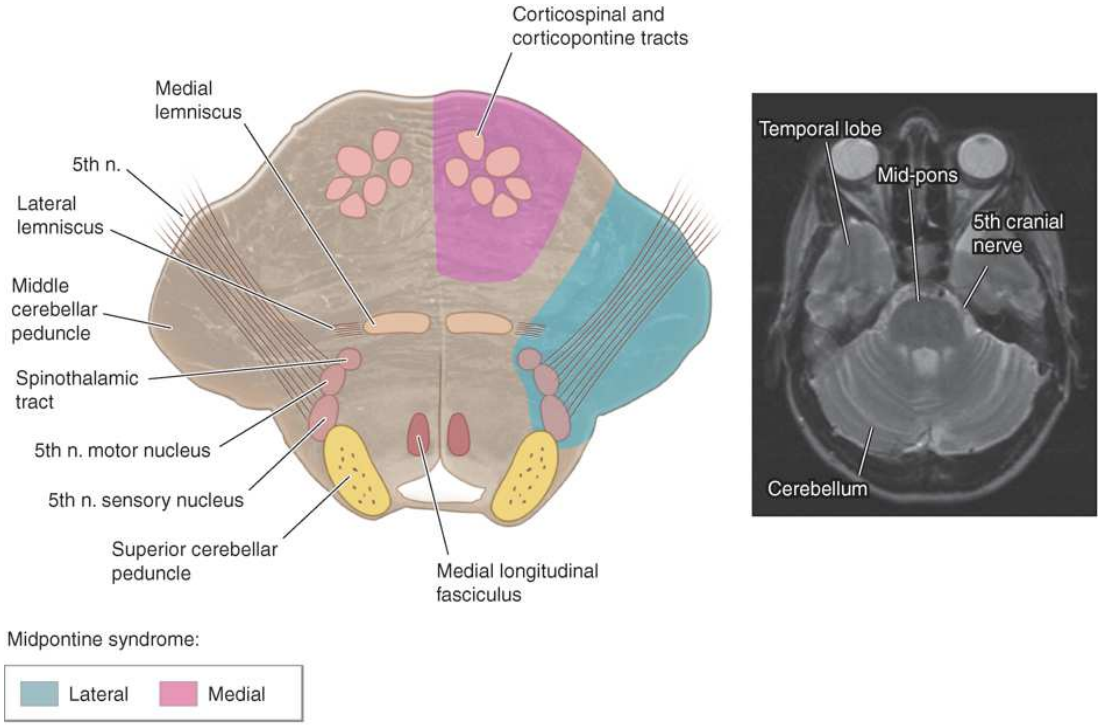

Fig.8. Mid pontine syndromes.

Signs and symptoms: Structures involved

1. **Medial midpontine syndrome** (paramedian branch of midbasilar artery) On side of lesionAtaxia of limbs and gait (more prominent in bilateral involvement): Pontine nuclei On side opposite lesion Paralysis of face, arm, and leg: Corticobulbar and corticospinal tract Variable impaired touch and proprioception when lesion extends posteriorly: Medial lemniscus.
2. **Lateral midpontine syndrome** (short circumferential artery) On side of lesion Ataxia of limbs: Middle cerebellar peduncle Paralysis of muscles of mastication: Motor fibers or nucleus of fifth nerve Impaired sensation over side of face: Sensory fibers or nucleus of fifth nerve On side opposite lesion Impaired pain and thermal sense on limbs and trunk: Spinothalamic tract
3. **Medial superior pontine syndrome** (paramedian branches of upper basilar artery) On side of lesion Cerebellar ataxia (probably): Superior and/or middle cerebellar peduncle Internuclear ophthalmoplegia: Medial longitudinal fasciculus Myoclonic syndrome, palate, pharynx, vocal cords, respiratory apparatus, face, oculomotor apparatus, etc.: Localization uncertain—central tegmental bundle, dentate projection, inferior olivary nucleus On side opposite lesion Paralysis of face, arm, and

leg: Corticobulbar and corticospinal tract Rarely touch, vibration, and position are affected: Medial lemniscus.

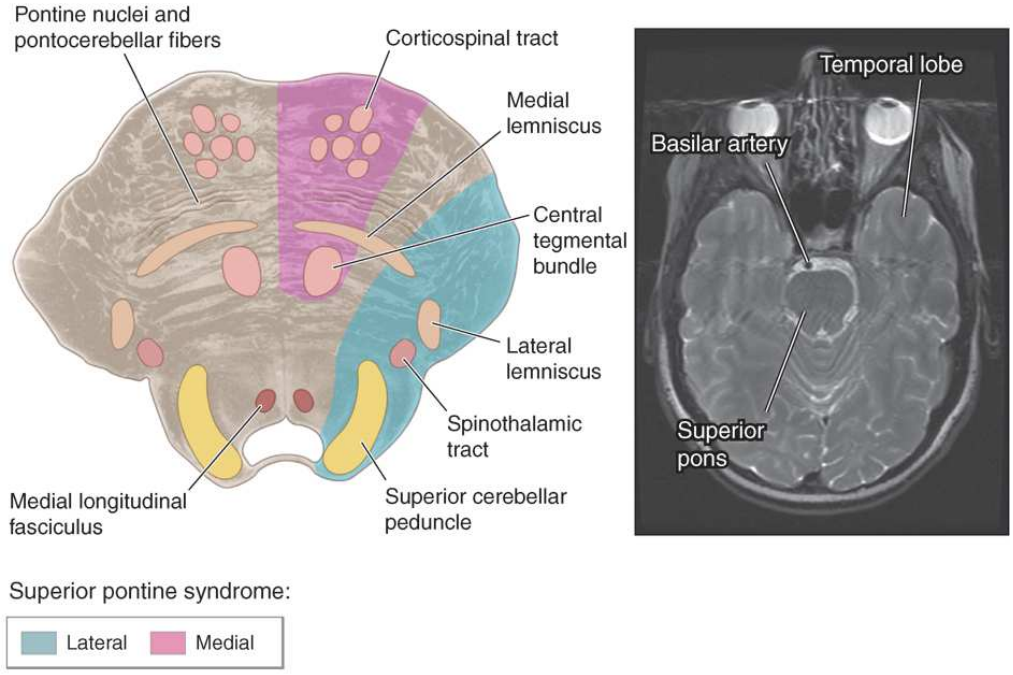

Fig.9. Superior Pontine syndromes.

4. **Lateral superior pontine syndrome / superior cerebellar artery syndrome :** On side of lesion Ataxia of limbs and gait, falling to side of lesion: Middle and superior cerebellar peduncles, superior surface of cerebellum, dentate nucleus Dizziness, nausea, vomiting; horizontal nystagmus: Vestibular nucleus Paresis of conjugate gaze (ipsilateral): Pontine contralateral gaze Skew deviation: Uncertain Miosis, ptosis, decreased sweating over face (Horner's syndrome): Descending sympathetic fibers Tremor: Localization unclear—dentate nucleus, superior cerebellar peduncle On side opposite lesion Impaired pain and thermal sense on face, limbs, and

trunk: Spinothalamic tract. Impaired touch, vibration, and position sense, more in leg than arm (there is a tendency to incongruity of pain and touch deficits): Medial lemniscus (lateral portion)

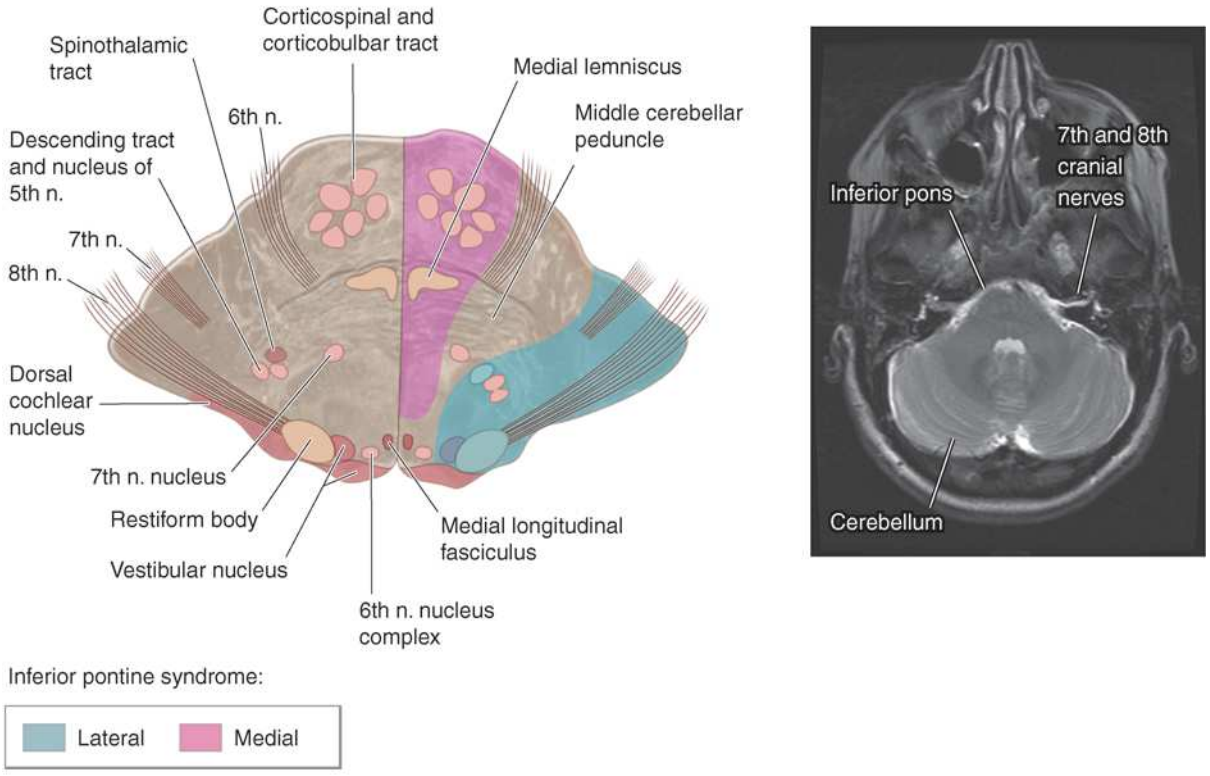

Fig.10. Inferior Pontine Syndromes

5. **Medial inferior pontine syndrome** (occlusion of paramedian branch of basilar artery) On side of lesion Paralysis of conjugate gaze to side of lesion (preservation of convergence): Center for conjugate lateral gaze Nystagmus: Vestibular nucleus Ataxia of limbs and gait: Likely middle cerebellar peduncle Diplopia on lateral gaze: Abducens nerve On side opposite lesion Paralysis of face, arm, and leg: Corticobulbar and corticospinal tract in lower

pons Impaired tactile and proprioceptive sense over one-half of the body: Medial lemniscus.

6. **Lateral inferior pontine syndrome** (occlusion of anterior inferior cerebellar artery) On side of lesion Horizontal and vertical ystagmus, vertigo, nausea, vomiting, oscillopsia: Vestibular nerve or nucleus Facial paralysis: Seventh nerve Paralysis of conjugate gaze to side of lesion: Center for conjugate lateral gaze Deafness, tinnitus: Auditory nerve or cochlear nucleus Ataxia: Middle cerebellar peduncle and cerebellar hemisphere Impaired sensation over face: Descending tract and nucleus fifth nerve On side opposite lesion Impaired pain and thermal sense over one-half the body (may include face): Spinothalamic tract

LACUNAR STROKES

Lacunes are small ischemic infarcts in the deep regions of the brain or brainstem that range in diameter from 0.5 to 15 mm as a result of occlusion of a single perforating vessel. [32] Lacunes usually occur in patients with lipohyalinosis of penetrating arteries or branches related to long-standing arterial hypertension. Diabetes mellitus and extracranial arterial and cardiac sources of embolism are found less frequently. The most frequent sites of lacunes are the putamen, basis pontis, thalamus,

posterior limb of the internal capsule, and caudate nucleus, in that order.[34, 35] Lacunar infarcts are responsible for approximately 20% of all strokes.

Eventhough more than 20 lacunar syndromes have been described, the four most important clinical syndromes are

1. **Pure motor stroke or pure motor hemiparesis**: occurs due to an internal capsule, corona radiata, or basis pontis lacune. It is characterized by a hemiparesis or hemiplegia involving the face, arm, and, to a lesser extent, the leg, accompanied by mild dysarthria, at the onset of stroke. Clinical findings usually do not distinguish between capsular or pontine pure motor hemiparesis, but the combination of dysarthria and a history of previous transient gait abnormality or vertigo favour a pontine location.[32]

2. ***Pure sensory stroke or pure hemisensory stroke***: occurs mainly due to a lacune involving the ventroposterolateral nucleus of the thalamus. Small ischemic strokes in the internal capsule/corona radiata, subthalamus, midbrain, or the parietal cortex and also, pontine lacunes localized to the medial lemniscus or paramedian dorsal pons can lead to a pure sensory stroke. It is often difficult to differentiate a brainstem pure sensory stroke from thalamic one. In pontine pure sensory stroke, vibration and position sense (medial

lemniscal modalities) are often reduced on the affected side, whereas sensation to pinprick and temperature (spinothalamic modalities) is preserved. Conversely, in cases of pure sensory stroke involving the thalamus, internal capsule, or corona radiata, both spinothalamic and medial lemniscal modalities are compromised. [37, 38, 39]

3. ***Ataxic hemiparesis***: occurs due to a lacune in the contralateral posterior limb of the internal capsule or the contralateral basis pontis. It is characterized by weakness of one side, predominantly the lower extremity, and a cerebellar type of incoordination of the arm and leg on the same side, but out of proportion to the weakness. [40]

4. ***Dysarthria and clumsy hand syndrome***: occur due to a lacune involving the depth of the basis pontis between its upper third and lower two-thirds. It is characterized by UMN facial weakness, deviation of the protruded tongue, dysarthria, dysphagia, loss of fine motor control of the hand, and an extensor plantar response.[40]

Vertebrobasilar insufficiency (VBI) is a term used to describe fluctuating brainstem symptoms, such as dizziness associated with cranial nerve symptoms or cerebellar dysfunction over a period of days to weeks. This

indicates insufficient flow through the posterior circulation and is essentially a brainstem TIA. Rarely VBI will present as vertigo alone.

The most dreaded posterior circulation infarction is the “Locked-in syndrome”, which is caused by basilar artery occlusion, resulting in bilateral findings due to midbrain infarction. The syndrome is characterized by a progression of symptoms leading to quadriplegia with paralysis of horizontal gaze and bilateral facial and oropharyngeal palsy. The patient is awake and is only able to move his or her eyes vertically. This is often preceded by brainstem TIAs occurring several times a day. Patients become stuporous or comatose as the reticular activating system becomes involved.

Table: 3. A bird's eye view of PCS syndromes [41]

Anatomic Region	Function	Vascular Supply	Clinical Features		
			Syndrome	Ipsilateral	Contralateral
Cerebellum	Regulation and control of muscle tone, coordination, control of posture and gait	Vertebral artery PICA	Anterior Vermis	Gait, trunk and leg dystaxia	
			Posterior Vermis	Truncal ataxia	
Midbrain	Modulation of sensation, movement and consciousness	Posterior cerebral artery	Weber	3rd nerve palsy	Hemiplegia- arm & leg
			Claude	3rd nerve palsy	Cerebellar ataxia, tremor
			Benedikt	3rd nerve palsy	Cerebellar ataxia, tremor and corticospinal signs
			Perinaud	Paralysis of upward gaze and accommodation, fixed pupils	

Pons	Modulation of sensation, movement and consciousness	Basilar artery	Locked-in syndrome	Bilateral hemiparesis- arm & leg, fcial weakness, lateral gaze weakness, dysarthria	
			Foville	Lateral gaze weakness, facial weakness	Hemiparesis- arm & leg
			Millard-Gubler	Lateral gaze weakness, facial weakness	
			Raymond	Lateral gaze weakness	Hemiparesis- arm & leg
		Basilar artery AICA	Marie-Foix	Arm and leg ataxia	Hemiparesis- arm & leg Hemisensory loss- pain & temperature
		Vertebral A PICA	Wallenberg (Lateral Medullary)	Nystagmus, Vertigo, Ataxia, Sensory loss in face, Hoarseness, Dysphagia, Horner syndrome	Hemisensory loss- pain and temperature

Medulla	Modulation of sensation, movement and consciousness	Anterior Spinal artery	Medial Medullary	Weakness and later hemiatrophy of tongue	Hemiparesis- arm & leg, Hemisensory loss- touch and proprioception
Cerebrum • Occipital lobe • Infero-medial temporal lobe	• Visual perception & recognition • Visual discrimination	Posterior cerebral artery	Balint	Bilateral loss of voluntary eye movements, poor visual-motor coordination, inability to understand visual objects	
		• Posterior cerebral artery • Top of Basilar	Anton	Bilateral loss of vision, unawareness or denial of blindness	
Thalamus	Integration of sensory and motor systems	Posterior cerebral artery	Dejerine-Roussy	Hemisensory loss- all modalities, hemi-body pain	

TIAs occur at least 2 weeks prior to posterior circulation stroke presentation in 50% of patients. In a study of 85 cases of angiographically proven basilar artery or bilateral vertebral artery occlusion, prodromal or progressive symptoms occurred in 75% of cases. The most common prodromal symptoms included vertigo, nausea and headache ranging from days to months before the stroke. Only 5% of patients had isolated vertigo as a prodromal symptom. In 63% of patients, the onset of symptoms was gradual and progressive. The most common presenting symptoms were vertigo, nausea, headache, dysarthria and cranial nerve palsies. Hemiparesis or Tetraparesis was present in 61% of the patients. Thirty-six percent presented awake, while twenty-three percent were somnolent and thirty percent were comatose. [42, 43]

INVESTIGATIONS

CT scan is the initial procedure of choice. But it is ineffective in showing brainstem and cerebellar infarct and is mainly used to exclude brainstem haemorrhages.

Scans obtained in the first several hours after an infarct generally show no abnormality, and the infarct may not be reliably seen for 24-48 hours. Even later CT scan may fail to show small ischemic strokes in the posterior fossa because of the bone artefact. Contrast enhanced CT scans adds specificity by showing contrast enhancement of subacute infarcts

and allows visualization of venous structures. Coupled with newer generation scanners, administration of intravenous contrast allows visualization of large cerebral arteries. Such ‘CT ANGIOGRAMS’ may be useful in acute stroke management to reveal the presence or absence of large vessel pathology. After an IV bolus of contrast, deficits in brain perfusion produced by vascular occlusion can also be demonstrated and used to predict the region of infarcted brain and the brain at risk of further infarction.

The advent of MRI, with its ability to provide better image of posterior fossa structures made investigations of vertebrobasilar territory infarcts more easy and accurate.

Magnetic Resonance Imaging reliably documents the extent and location of infarction in all areas of the brain, including the posterior fossa and cortical surface, if appropriate imaging sequences are obtained. It also identifies intracranial hemorrhages and other abnormalities. The higher, the field strength, the more reliable and precise the image will be. Diffusion-weighted imaging is more sensitive for early brain infarction than standard magnetic resonance sequences as is FLAIR (Fluid Attenuated Inversion Recovery) imaging. MR angiography is highly sensitive for extracranial internal carotid plaque as well as intracranial stenosis of large vessels with higher degrees of stenosis. MR angiography tends to overestimate the degree of stenosis when compared to

conventional X-Ray angiography. MRI with fat saturation is an imaging sequence used to visualize extracranial or intracranial arterial dissection. This sensitive technique images clotted blood within the dissected vessel wall and has revealed carotid or vertebral dissection as the cause of stroke in a sizable fraction of young patients (age < 45). Stroke with neck, jaw or retroauricular pain, with or without Horner's syndrome, should prompt this imaging modality or conventional X-Ray angiography.

MRI is less sensitive for acute blood products than CT and is more expensive and less readily available.

Transcranial Doppler ultrasound and MRA provided methods of studying the vertebral and basilar arteries safely and quickly. Extracranial ultrasound was more used to define lesions within the extracranial vertebral arteries and subclavian arteries and the carotid system.

Cerebral angiography.

Conventional X-ray cerebral angiography is the gold standard for identifying atherosclerotic stenosis of vertebral arteries and also to look for other pathologies including aneurysm, vasospasm, intraluminal thrombus, fibromuscular dysplasia, A-V fistula, vasculitis, and collateral channels of blood flow.

OTHER TECHNIQUES

Both xenon techniques (xenon-CT) and PET scans can quantify cerebral blood flow. These tools are generally used for research, but can be useful for determining the significance of arterial stenosis and planning for revascularisation surgery. Single photo emission tomography (SPECT)-perfusion, and MR-perfusion techniques report relative cerebral blood flow and currently are research tools.

Cardiac investigations also need to be done in patients with stroke to rule out a cardiac source of emboli. This includes an echocardiography in every patient as this picks up any cardiac as well as some lesions in the aorta.

It has now become possible to investigate the brain, cardiovascular system and the underlying stroke mechanism non invasively and quickly in patients with posterior circulation ischemia so that appropriate treatment could be instituted at the earliest.

MATERIALS AND METHODS

This study was an observational cross sectional conducted in Tirunelveli Medical College, Department of Medicine, during the period of July 2012 to October 2013 with the aim to study the clinical patterns and risk factors associated with posterior circulation stroke. Required clearances from the departments concerned and ethics committee was obtained prior to the start of the study.

All patients admitted to the General Medicine wards and Intensive Medical care unit with clinical features suggestive of stroke was considered for the study. All of them were subjected to a CT scan of the brain and patients with evidence of posterior circulation stroke in clinical features and/or imaging were taken up. Patients were included in the study based on strict inclusion and exclusion criteria.

All patients with clinical features suggestive of posterior circulation stroke and/or imaging showing infarcts within the posterior circulation territory were included in the study. Those with CT scan showing haemorrhage and those with evidence of infarcts in other areas like territory of anterior circulation, border zone infarction were excluded from the study.

An oral consent was taken from all patients for a detailed clinical examination and lab investigations. A detailed clinical history and a physical examination including complete neurological examination was

done on each patient using a carefully thought out and reviewed proforma. (Annexure -1)

Patients' details regarding age, sex, risk factors like Hypertension, Diabetes Mellitus, Atrial fibrillation, Ischemic heart disease, hypercholesterolemia, smoking, past history of TIA were recorded. The onset and chronology of symptoms and signs were also recorded.

All patients were subjected to a CT scan of the brain. MRI brain with MRA and MRV was done in patients who had clinical features suggestive of posterior circulation stroke, but with a normal CT scan or in cases where the clinical picture is confusing. Vertebral artery Doppler was done in patients with features suggestive of infarcts at multiple sites. All were subjected to routine lab investigations which include a complete blood count, Renal function tests, Routine blood sugar and those with elevated RBS was subjected to an FBS and a 2hr PPBS, serum electrolytes, Lipid profile and a urine analysis. They were also subjected to an ECG and Chest X-ray. Those with abnormalities in ECG and Chest X-ray were subjected to an echocardiogram.

After all the data was collected, the posterior circulation territory was subdivided into three regions, proximal, middle and distal similar to those done in the NEMC stroke registry study.

PROXIMAL TERRITORY

The intra-cranial vertebral arteries on both sides join at the medullo - pontine junction to form the basilar artery. The territory supplied by the intra – cranial vertebral arteries include the medulla and the parts of cerebellum supplied by the posterior inferior cerebellar artery(PICA) which inturn is a branch of the intra – cranial vertebral artery. This region is designated as the proximal intra – cranial posterior circulation territory.

MIDDLE TERRITORY

The Basilar artery bifurcates at the ponto – mesencephalic junction. The territory supplied by the basilar artery including the pons and the portion of the cerebellum supplied by Anterior Inferior Cerebellar Artery (AICA) branches is designated as middle intracranial posterior circulation territory.

DISTAL TERRITORY

The portion of the posterior circulation supplied by the distal basilar artery, the superior cerebellar artery (SCA), the posterior cerebral artery and their penetrating branches constitute the distal intra – cranial posterior circulation territory. The distal territory includes the midbrain, thalamus, SCA supplied cerebellum, occipital and temporal lobe regions.

The data was analysed and the clinical patterns associated with each territory and those in common were determined. Also the commonly associated risk factors were analysed. This data was compared with NEMC posterior circulation stroke registry.

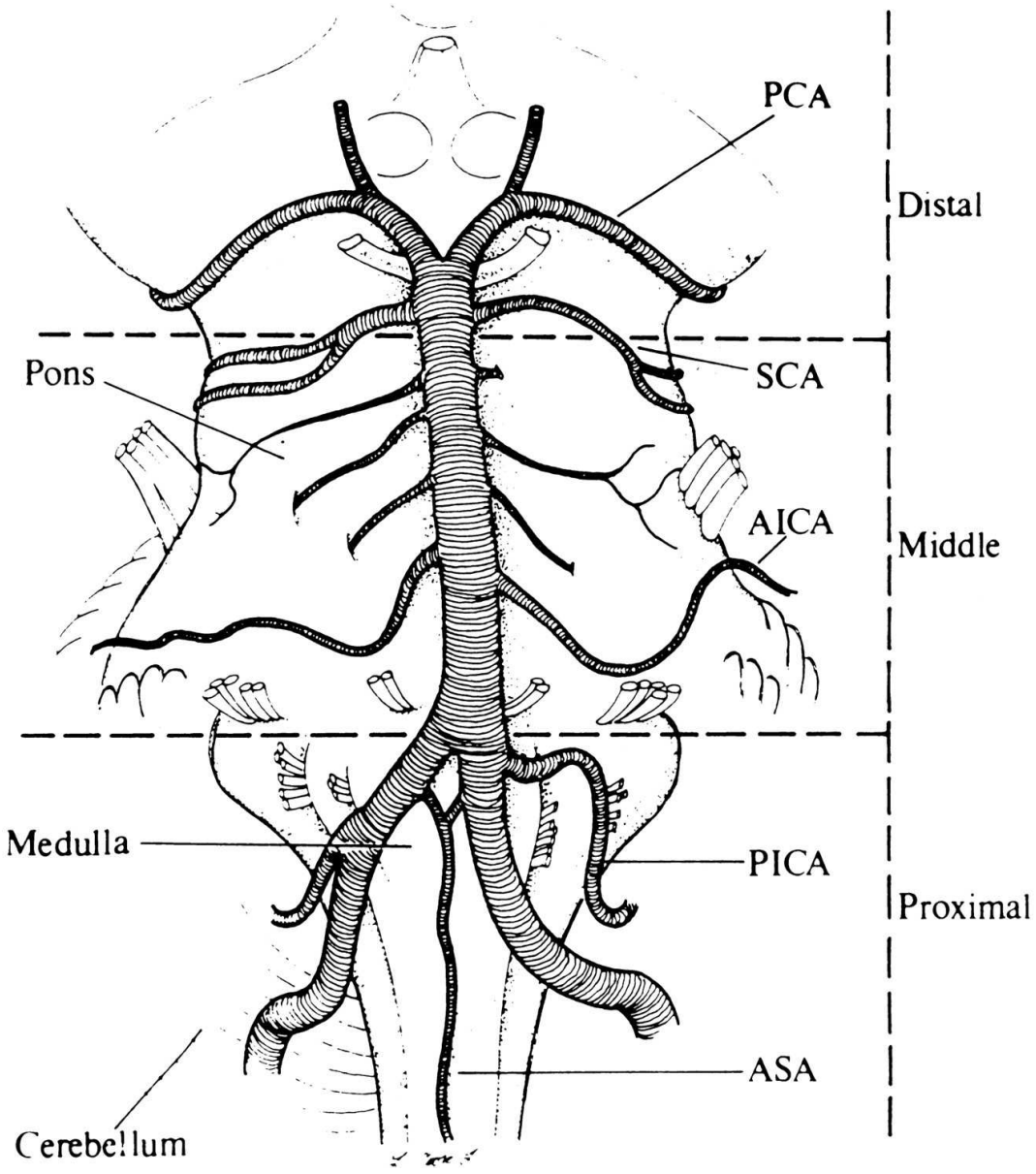

Fig. 11. Posterior circulation Territories

OBSERVATIONS AND RESULTS

50 patients admitted with clinical features suggestive of posterior circulation stroke were enrolled in this study.

Sex Distribution:

Among the 50 patients, 74% (37) were males and 26% (13) were females.

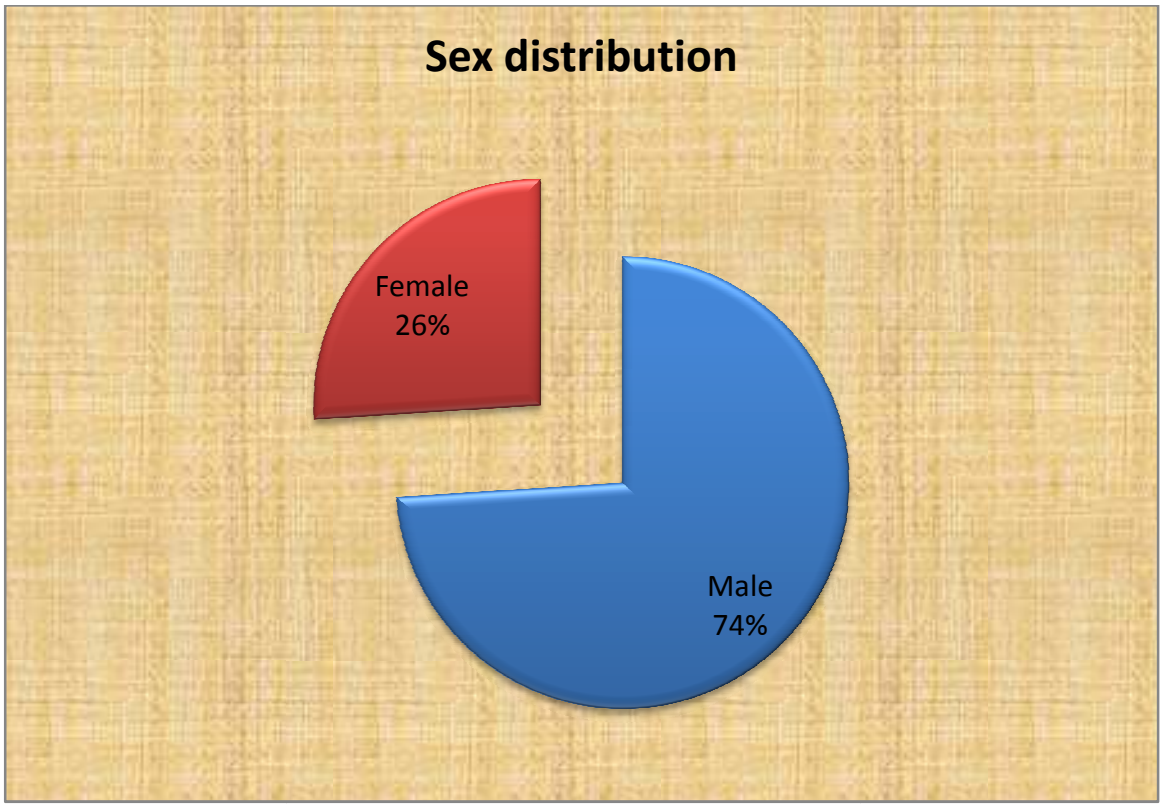

Fig: 12. Sex Distribution

AGE DISTRIBUTION:

The age distribution is summarised in figure: 13 and Table: 3.

In our study, the youngest patient was 39 years old, and the eldest one was 79 years. 82 % of the cases were above 50 years. Maximum number of cases was in the age bracket 61 -70 years (42 %). We had 2 patients younger than 40 years of which one was a lady. The age of occurrence of stroke was comparable in both sexes with both having a higher incidence in the 61-70 year old group. Also it was noted that, as age increases, the incidence of posterior circulation stroke also increased with the exception of >70 year old group.

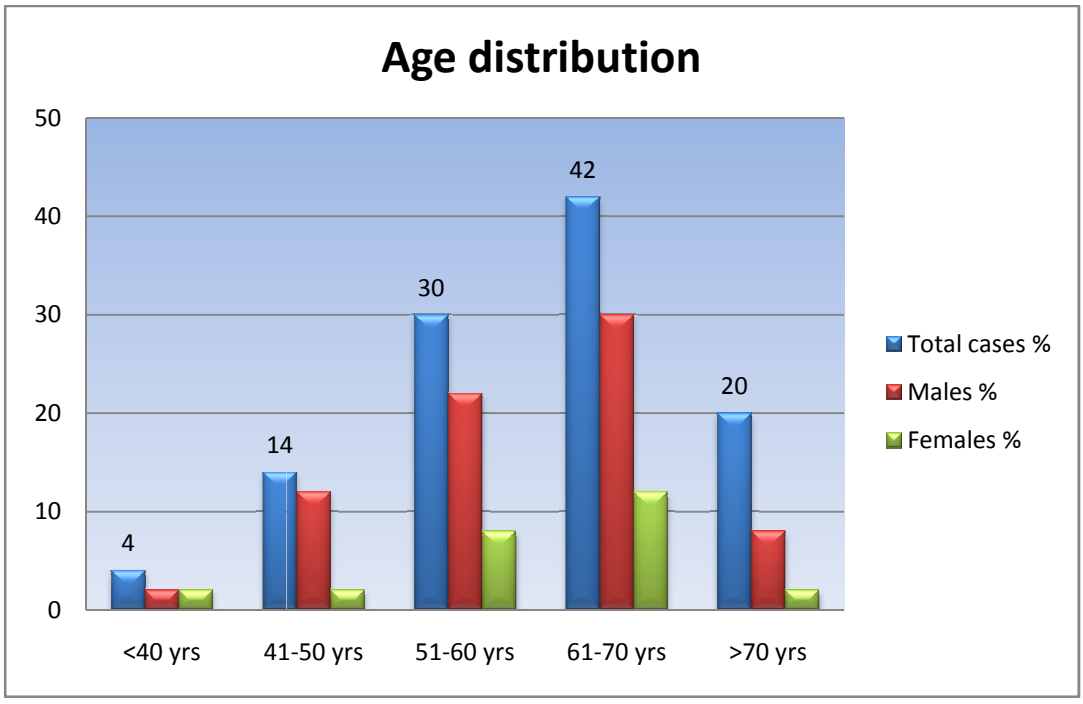

Fig.13. Age distribution

TABLE – 4

AGE DISTRIBUTION.

Age in years	Males	Females	Total No. of cases	Total Percentage
<40yrs	1	1	2	4 %
41-50	6	1	7	14 %
51 – 60	11	4	15	30 %
61 – 70	15	6	21	42 %
> 70	4	1	5	10 %
Total	37	13	50	100 %

Risk factors:

The possible risk factors were studied in all the 50 patients.

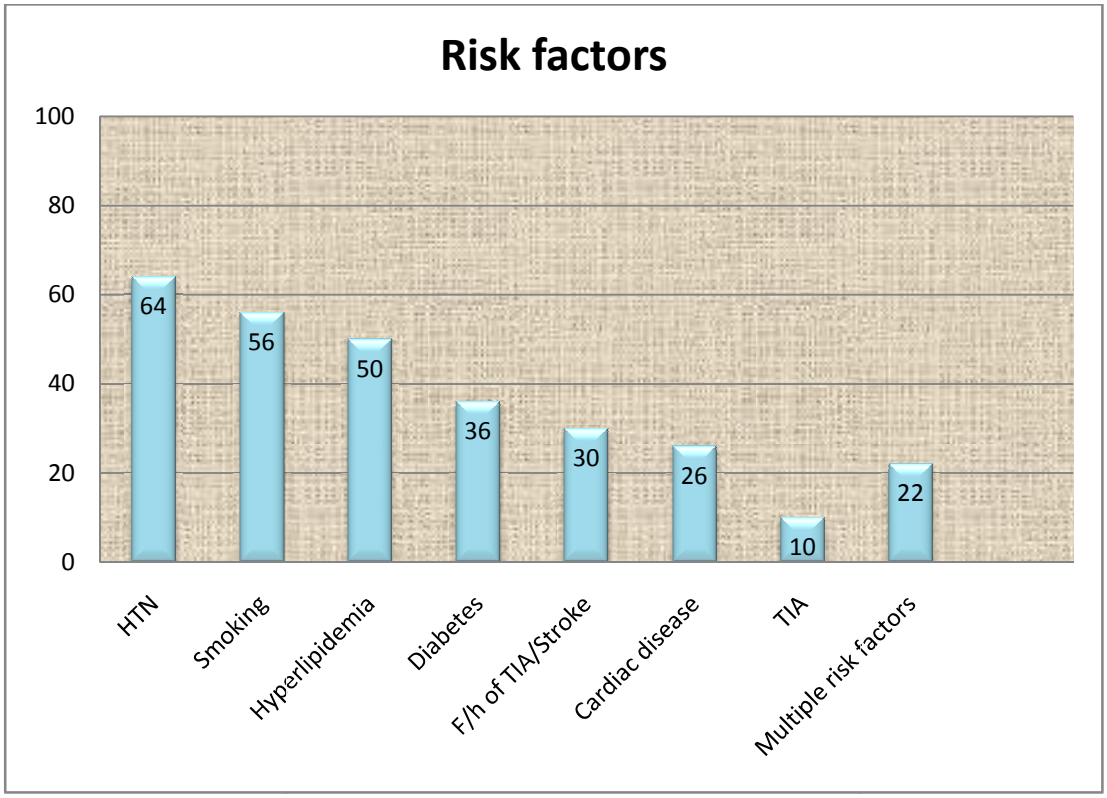

Fig.14. Risk factors associated with Posterior circulation stroke.

Sixty four percent of patients were Hypertensive. 36 percent were having Diabetes Mellitus. Twenty eight out of 37 (75.6%) males were smokers making it an important risk factor among males and 56% altogether. Hypercholesterolemia was also found to be an important risk factor to be present in 50 % of patients. 20 % patients had atleast one episode of TIA and 30 % patient had a family history TIA. 26 % of patient had cardiac illness in the form of CAD, Valvular heart disease, AF.

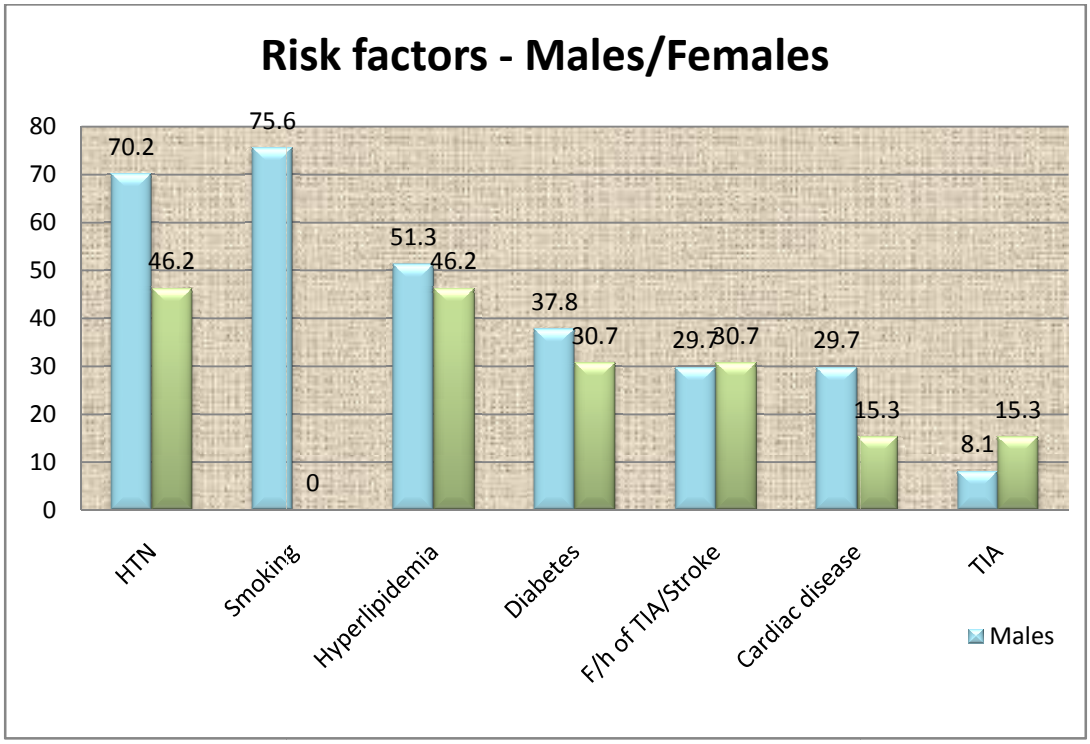

Fig.15. Risk factors according to sex.

When the risk factors when analysed according to sex, it was found that the most important risk factor in males was smoking followed by hypertension and hyperlipidemia. In females, hypertension and hyperlipidemia were found to be the most important risk factors followed by diabetes mellitus. Interestingly, incidence as well as family history of TIA was more among females.

Clinical features:

The clinical features at the onset of stroke were studied and the findings are summarised in Fig.16 and Fig.17.

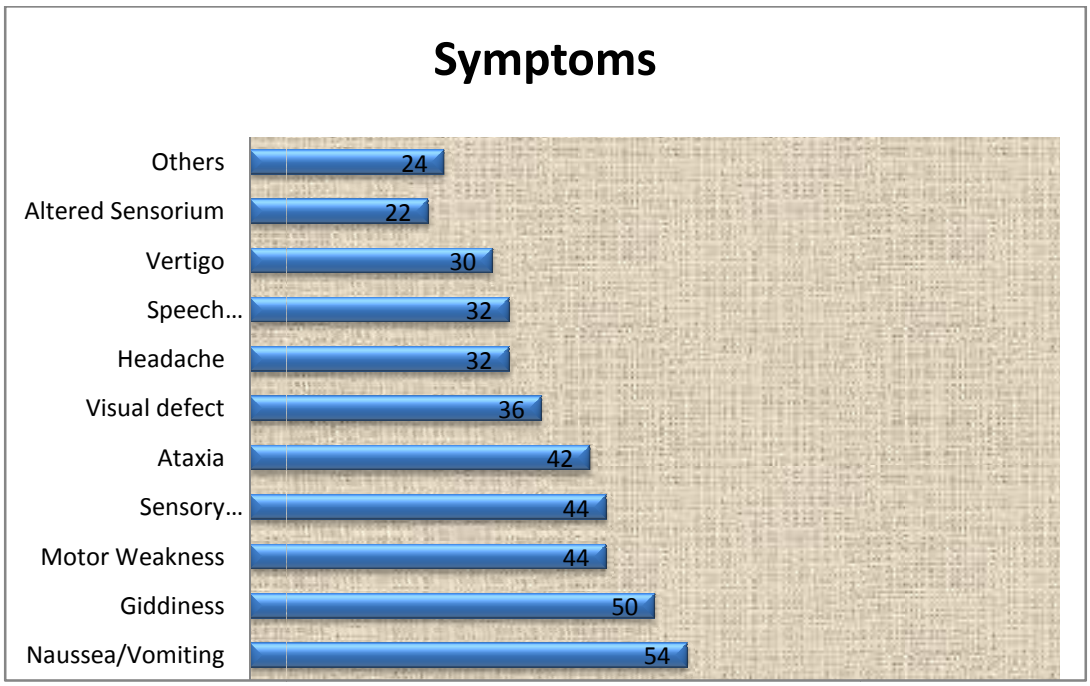

Fig. 16, Frequency of symptoms among the study subjects.

Most of our patients presented with vomiting and giddiness. 44% had weakness and sensory impairment mainly on one side. Ataxia was found in 42 % patients which included unilateral as well as truncal ataxia. Headache was found in 32 % patients and visual disturbances in 36 %. Visual disturbances included diplopia and field defects. 22 % patients presented with altered sensorium. 10 % of patients had seizures at the onset.

All patients were subjected to a detailed neurological examination at the time of admission. Clinical features that we found in most of the patients could be localised to a specific territory involved.

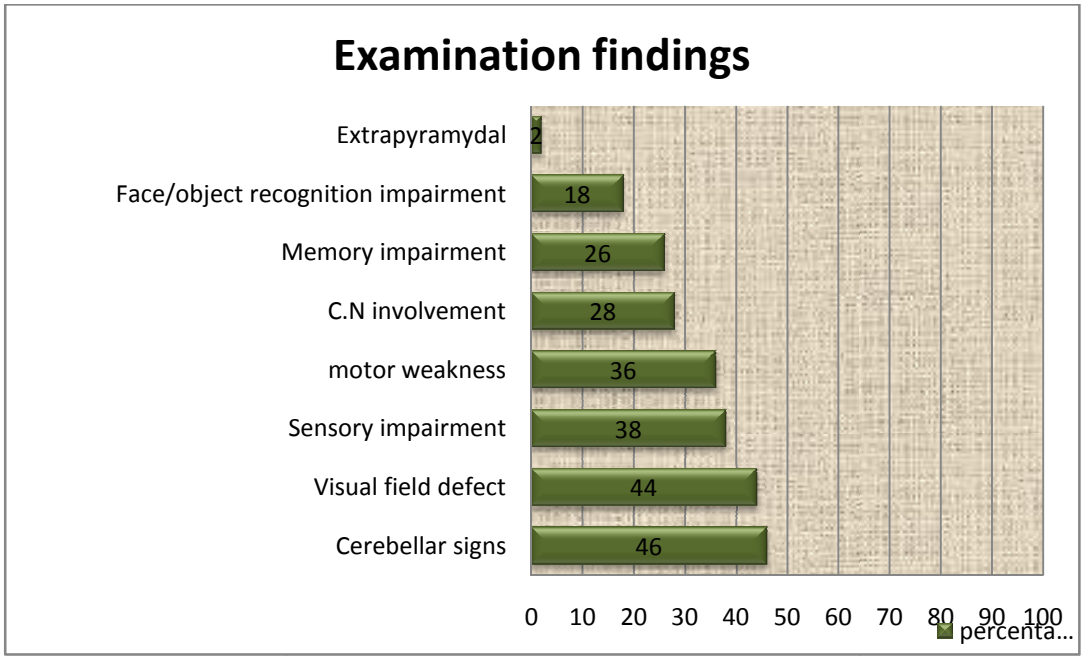

Fig.17.Clinical examination findings among study subjects.

These clinical features were mainly seen as a combination and not in isolation. These included, cerebellar signs (46 %), visual field defects (44 %), which included homonymous hemianopia and quadrantanopias, temporal lobe signs, weakness and sensory disturbances. Cranial nerve involvement was observed in 28 % of patients. We had patients with III, IV, V, VII, IX and X cranial nerves involved.

To describe the location of infarcts, we subdivided the posterior circulation into proximal, middle and distal intracranial arteries accordingly described by NEMC posterior circulation registry. The clinical features and neuroimaging are taken together to describe the location of infarct. For about 11 patients, CT was normal. In all of them, an MRI was taken and a lesion was made out.

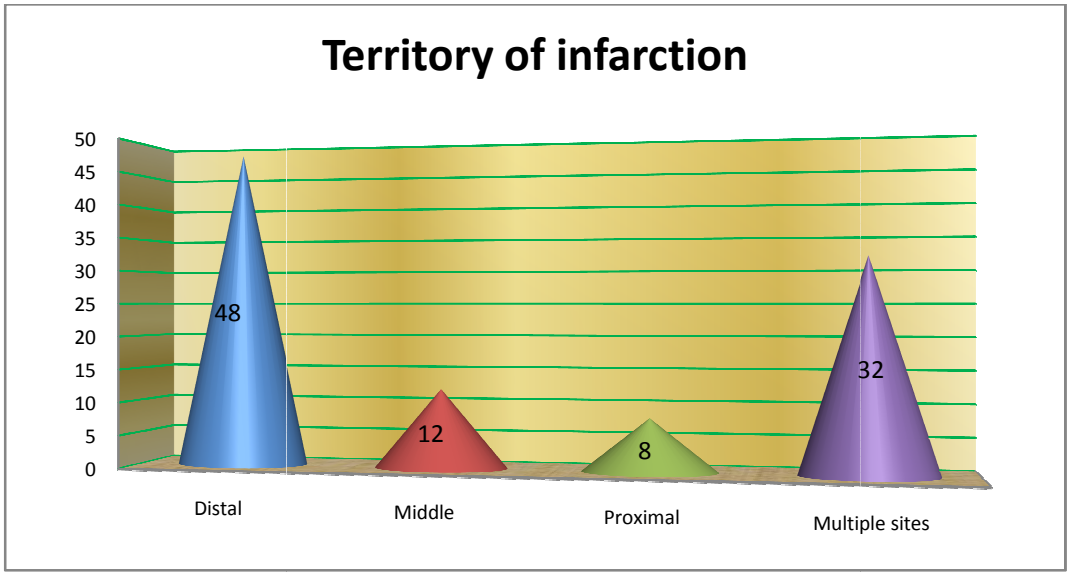

Fig.18. Frequencies of territories involved.

It was found that, in 48 % of the patients we studied, had an involvement of the distal territory. Isolated middle and proximal territory infarcts were less. In 12 % of patients, infarct was localised to middle territory and 8 % of patients had a proximal territory infarct. In the rest, which constituted 32 %, had infarcts involving more than one territory in varying combinations.

DISCUSSION

Posterior circulation stroke constitutes 15-20 % of all ischemic cerebrovascular accidents. It is considered to be more severe than anterior circulation strokes. It can range from a mild intermittent vertebrobasilar insufficiency to many other well established stroke syndromes like medial medullary syndrome, lateral medullary syndrome, locked-in syndrome and top of basilar artery syndrome. [44, 45]

Cerebrovascular disease is found to be more common in males than females but the severity is more in ladies. In our study, 74% of the subjects were male and 26% were females. In New England Medical Centre –Posterior circulation stroke registry (NEMC-PCSR), 63 % were men and 37% were women, substantiating male preponderance. Also, several other studies like R.B.Libman et al Ma.Cristina et al supports this fact. But, in certain studies like Smajloric et al, Khorasan Posterior Circulation Stroke Registry etc, the incidence was found to be similar. [45, 46, 47, 48, 49]

Cerebrovascular diseases are common in age groups above 50 years of age. Also, it is described in various studies and other literature that, as age increases, the stroke incidence also increases. In New England Medical Centre – Posterior circulation stroke registry, the mean age was 60.5 years. [50] In this study, we had a similar result with 82 % of patients above 50 years of age and a mean age of 60.2 years. It was also

found that, as age increases, the incidence of PCS also increased till the age group 61 – 70 years. We noticed a decreased incidence above 70 years of age. This could be due to the small sample size of our study or due to the fact that, in patients above 70 years of age, the severity of PCS may be high and most of them might have expired before reaching a referral centre like ours.

The risk factors in stroke are classified as modifiable and non modifiable. Male sex, older age, family history of stroke or TIA, history of previous TIA, race etc are non – modifiable risk factors. Hypertension, Diabetes mellitus, hypercholesterolemia, obesity, chronic smoking, physical inactivity, cardiac diseases like ischemic heart disease, atrial fibrillation, rheumatic heart disease etc are considered as modifiable risk factors.

Uma Sundar et al [51] found that 47.3 % of patients in their study were smokers and Huan et al found that 71 % patients in their study were hypertensives. A comparison of risk factors between these two studies and our study is shown in table-5.

Risk Factors	Present study (2013)	Uma et al (2007)	Huan et al (2002)
Hypertension	64%	35.5%	71%
Diabetes	36%	21%	22.6%
C/c smoking	56%	47.3%	38.7%
Hyperlipidemia	50%	44.4%	-
IHD	14%	17.1%	19.4%
RHD	6%	10.5%	-

Table. 5. Risk factors – A comparison of different studies.

In this study, hypertension (64 % of patients) was the most common risk factor. Chronic smoking was found in 56 % of the subjects. In males, smoking was found to be more important than hypertension (75.6 % Vs 70.2 %). Hyperlipidemia was found in 50 % of patients. 36 % of the subjects were diabetics. Embolism from heart was seen in 26 %. Clinical features which were more prominent at the onset of stroke in our study were Nausea/vomiting and giddiness which constituted 54 % and 50 % respectively. Other prominent symptoms included motor weakness, sensory impairment, ataxia, headache, speech disturbances, visual field

defects and altered sensorium. 10 % of patients in this study had seizures which included GTCS and focal seizures with secondary generalisation.

Timothy et al [52] had described vertigo without hearing loss as the commonest symptom in brainstem stroke syndromes. Vertigo occurs in both small vessel disease and large vessel disease. In the study by Manmohan et al [53], the incidence of vertigo was 56.25 % and the one by Patrick et al [54] showed the incidence of vertigo to be 30 %. In our study, the incidence of vertigo was 30 %. In NEMC-PCSR, the incidence was 36 %. A comparison of these studies is given in table. 6. In this study, vertigo was found more to be associated with middle and multiple territory involvement. All patients with middle territory infarct and 72 % of those with multiple territory involvement had vertigo at onset.

Vertigo in middle territory infarct could be explained by involvement of vestibular nucleus and its connections in the pontine region.

Altered sensorium was seen in 22 % of patients in this study and was more common with distal territory involvement.

Table.6. Clinical features – a comparison of different studies

Clinical feature	Present study (50)	NEMC-PCSR (407)	Manmohan et al (80)	Patrick et al (39)
Giddiness	50%	47%	-	-
Vertigo	30%	36%	56.25%	30%
Headache	32%	28%	25%	-
Nausea/Vomiting	54%	27%	42%	42%
Motor weakness	44%	41%	42.5%	43%
Ataxia	42%	31%	48.75%	36%
Visual defects	36%	24%	20%	13%
Dysarthria	32%	31%	-	28%

Seizures were seen in 10 % of patients in this study, out of which 80 % had GTCS and 20 % had focal seizure with secondary generalisation. Seizures were observed in 4 patients with distal territory infarct and 1 patient with middle territory infarct. Occurrence of seizures could probably be explained by involvement of temporal cortex due to PCA involvement, which also explains the occurrence of seizures in the

distal territory infarcts. The patient with middle territory infarct probably had emboli of cortical arteries and thrombosis of middle territory arteries causing seizure and infarct respectively.

In this study, 36 % had motor weakness and 46 % had cerebellar signs. Visual field defects were seen in 44 %. Cranial nerves were involved in 28 %. Memory impairment was seen in 26 % of patients. The comparison of this study with NEMC –PCR and other studies are summarised in table. 7.

Table.7. Clinical features – a comparison of different studies.

Clinical feature	**Present study (50)**	**NEMC-PCSR (407)**	**Manmohan et al (80)**	**Patrick et al (39)**
Cranial nerve involvement	28%	-	26.25%	64%
Cerebellar signs	46%	-	48.75%	29%
Memory impairment	26%	17%	-	-
Visual field defect	44%	57%	-	-

CT brain was done in all patients enrolled in this study. It was normal in 11 patients in our study. MRI with MRA was done in all these patients and an infarct was located in all these cases. In those with multiple site involvement, a vertebral artery Doppler was done.

Regarding the vascular territory involvement in this study, we have found that 48 % had isolated distal territory involvement. 32 % of patients had involvement of multiple territories. Other territories were comparatively less.

In the New England Medical Centre Posterior circulation registry, they also found that distal territory involvement was more common – 41 %. Also in the study conducted by Manmohan et al, distal territory was the one which was involved the most. The comparison between these studies is summarised in table.8

Table.8. Territories of posterior circulation commonly involved – a comparison of different studies.

Territory involved	Present study	NEMC-PCSR	Manmohan et al
Proximal only	8%	18%	30%
Middle only	12%	16%	3.75%
Distal only	48%	41%	66.25%
Multiple	32%	25%	-

The results of these studies were comparable, except for the fact that in Manmohan et al and NEMC-PCR proximal territory was involved more than middle territory.

Since in our study, MRI, MRA and transcranial Doppler was done only for limited number of patients, the correct distribution of thrombosis in extracranial / intracranial vertebral, basilar, posterior cerebral and superior cerebral arteries could not be established.

Embolism from cardiac origin could be identified in 26 % of the patient population, we studied. In the NEMC – PCSR, the embolism from cardiac origin were 24 %.

There are a number of lacunae in this study. This study may not reflect the actual picture of posterior circulation stroke in our setup. But it definitely will give us some idea on what to expect when we come across a case of PCS and also what to do to reduce its risk.

SUMMARY

In our study, we have found that,

- ❖ Males were affected more than females
- ❖ Age group commonly involved was, above 50 years.
- ❖ Most of the cases were in the age bracket 61-70 years.
- ❖ Hypertension and chronic smoking contributed the major risk factors in posterior circulation stroke followed by hyperlipidemia and diabetes.
- ❖ In males, chronic smoking was more important than hypertension.
- ❖ Nausea and vomiting was the commonest symptom at onset followed by giddiness, motor weakness and ataxia.
- ❖ Close to half of the patients had clinical features of either pyramidal signs and or cerebellar signs.
- ❖ The territory of infarct commonly involved was the distal territory, which includes midbrain, thalamus, occipital and temporal lobes and SCA supplied cerebellum.

CONCLUSION

All patients with brain ischemia deserve full evaluation of their brain for vascular lesions. With the advent of newer techniques like MRI with Diffusion Weighted Imaging, MRA, CT angiogram, extracranial and transcranial ultrasound, it has become extremely easy to evaluate a stroke patient noninvasively. Evaluation of risk factors and cardiac investigations are just as important as brain imaging, because a sizeable number of posterior circulation infarcts are cardio embolic, furthermore, brainstem infarcts, especially medullary infarcts can cause cardiac abnormalities.

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ANNEXURE -1

PROFORMA

POSTERIOR CIRCULATION STROKE

Name :

SR No:

Age/Sex:

DOA:

IP No:

DOD:

1.Time of Onset of Stroke

2.Progression of Stroke

- Progressive
- Peaks immediately/ Static
- Regression
- Regression/Progression

3. Chronology of associated symptoms.

1st symptom

2nd symptom

3rd symptom

4th symptom

Others

4. Sensorium at onset

5. Speech disturbance at onset

6.Past history of TIAs

- Time
- Motor
- Sensory
- Speech
- Visual
- Vestibular
- Memory
- Behavioural
- Others

7. Risk factor

	Since	on Treatment or not.
HTN		
DM		
CAD		
Valvular Heart Disease		
Atrial Fibrillation		
Smoking		
Hypercholesterolemia		
Others		

8. Family History of:

Stroke

TIA

CAD

DM

HTN

Clinical Examination

General Examination

Skin Xanthomas

Pulse:

Carotids

BP

BMI

Neurological examination

HMF:

Lobar functions.

- Occipital – vision

  Face/object recognition

- Temporal – Memory – Verbal

  Visual

Cranial Nerves

SMS

R

L

Power

Tone

DTR

BJ

TJ

SJ

KJ

AJ

Plantar

Sensory System

Cerebellar signs

Extrapyramidal signs

Bladder symptoms

Neck rigidity

Other system examination

CVS

RS

P/A

Musculoskeletal

INVESTIGATIONS

A. Blood Investigations

CBC

Sugar - Fasting

-PPBS

Urea

Creatinine

Lipid Profile

Total Cholesterol

VLDL

HDL

TGGL

B. ECG

C. Echo cardiography

D. CXR PA View

E. 1.CT Brain (Time when taken; How many hours after stroke))

2. MRI brain with MRA

3. Carotid and Vertebral Doppler studies.

ANNEXURE -2

MASTERCHART

S.I No:	Name	Age	Sex	IP no:	Symptoms										General examination				CNS Examination							Other systems			Risk Factors									Blood sugar		RFT		Territory of	CT Brain/Terr	MRI/MRA	Vertebral A Doppler	ECG	CXR	ECHO	
					Giddiness	Nausea/ vomiting	Vertigo	Ataxia	Headache	Weakness	Sensory imp.	Visual symptoms	Altered Sensoriu	Speech disturban	Others	Pulse	BP	Markers of	BMI	HMF - Lobar functions			Cranial Nerve	Spinomo tor	Sensory system	Cerebellar signs	Extrapyra mydal	Bowel/bla dderdysmp	CVS	RS	Abdomen	HTN	Diabetes	CAD	Valvular Heart	AF	Smoking	F/H of Stroke/TI	Hypercholest erolemi	TIA	FBS	PPBS	Urea	Creatinine							
																			Visior	Occipital Face/Obj recognitio	Temporal memory																															
1	Manickam	53	1	9544	1	1			1	1	1					88	150/90	1	22.3					1	1		NIL	N	N	N	N	1	1					1	1	138	152	28	0.8	1	5	1	N	N	N		
2	Sankaran	62	1	10287	1	1				1	1	1	1			92	160/90		24.6	1		1		1	1	1		N	N	N	1			1						98	116	31	1	1	1	6		LVH	CMLY	LVH	
3	Murisamy	69	1	10962	1	1	1	1	1			1	1	1		68	130/80	1	23.2	1						1		N	N	N		1	1			1		1	1	96	128	27	0.9	4	4	6	N	AWMI	N	RWMA	
4	Mariappar	54	1	11632		1			1	1	1				SEIZ	74	130/86	1	19	1				1	1			N	N	N	1							1		102	132	21	0.7	1	1	6		N	N		
5	Karuppaar	48	1	11847					1				1			93	140/90	1	27.2	1	1							N	N	N	1	1			1	1	1		1	167	213	46	1.4	1	1	6		AF	CMLY		
6	Abdul Han	72	1	13246				1	1			1			1 dysphagia	76	130/70		23.3				1		1	1		N	N	N	1							1		98	126	32	0.7	3	5	3	VA Thromb	N	N		
7	Natarajan	68	1	13984	1	1		1				1	1		1	84	120/80		31.2	1						1		N	N	N		1				1		1		110	136	30	1.1	4	4	6	D.FLOW	N	N		
8	Janaki	58	2	14428				1	1	1						86	170/100	1	22				1			1		N	N	N	1		1			1				97	138	37		2	2	6		EAWMI	CMLY	RWMA	
9	Sulochana	49	2	14562	1	1	1	1	1				1		1 ryst	82	160/100		23.5							1		N	N	N		1				1		1		152	186	24	1.2	1	1	6		N	N		
10	Manohara	66	1	14934									1	1		74	140/90	1	18.9	1	1	1						N	N	N	1	1						1		138	174	22	1	1	5	1		N	N		
11	Mohan Ku	54	1	16533	1	1				1	1		1			78	150/100	1	23.5					1	1			N	N	N	1					1			1	102	124	28	0.9	1	1	6		LVH	CMLY	LVH	
12	Manga	70	2	17417	1	1	1	1	1		1			1		92	130/80		24.2				1	1	1	1		MS/MR	N	N				1	1	1					114	164	32	0.7	4	4	6		LAE	CMLY	MS/MR	
13	Arunachal	72	1	17548				1							SEIZ	94	100/70	1	26.7			1				1		N	N	N	1	1					1	1		136	188	31	1.7	4	4	6	D.FLOW	N	N		
14	Loganatha	67	1	17892	1	1				1	1				SEIZ	68	150/100		28.2			1		1	1			N	N	N	1				1	1	1			90	120	37	0.9	4	4	6		N	N		
15	Petchiamm	78	2	18627					1	1						70	130/90	1	23	1			1	1				N	N	N	1	1						1	128	170	21	0.8	2	2	6		N	N		
16	Palpandi	62	1	18690	1	1	1	1	1					1		68	150/90		21.7							1							1			1		1		112	162	38	0.9	4	5	4		N	N		
17	Govindara	39	1	20145					1	1	1	1				72	120/80		24.3	1	1	1		1	1							1								112	138	21	1	1	1	6		N	N		
18	Nasariah	48	1	21638	1	1			1		1	1				76	130/80		21.6					1	1	1		N	N	N	1		1			1	1	1		92	127	29	1.5	4	4	6		AWMI	CMLY	RWMA	
19	Kayanchi	64	1	26783									1			78	140/90	1	23.4	1								N	N	N	1									92	119	38	0.8	1	1	6		N	N		
20	Karpagara	60	1	27341								1				80	150/100		25.8						1			N	N	N		1				1				132	187	36	1.3	1	5	1		LVH	N		
21	Mani	61	1	27619			1	1		1				1		84	120/80		21.7	1	1	1		1		1		N	N	N	1	1				1		1	1	178	238	52	1.3	4	4	6		N	N		
22	Radhakrist	73	1	29306	1	1	1							1		76	150/100	1	20.8									N	N	N	1				1					102	138	32	0.7	2	2	6	VBI	N	N		
23	Saraswath	57	2	30128						1	1	1			SEIZ	88	160/100		30.6	1				1	1			N	N	N						1		1	1	90	132	38	1	4	4	6	N	N	N		
24	Chandrase	55	1	34327	1	1		1	1							90	130/80	1	29.2							1		N	N	N	1				1					96	128	40	0.9	1	1	6		N	N		
25	Manickam	63	1	38182	1	1				1	1	1				82	130/80		21.4				1	1				N	N	N	1	1				1	1	1		187	226	42	1.2	1	5	1		N	N		
26	Pushpamm	57	2	38798			1	1	1		1			1		74	150/90	1	23.5							1		N	N	N						1				98	126	28	0.8	3	3	6		N	N		
27	Natarajan	47	1	40512	1	1	1	1			1	1				64	140/80		35.9	1				1		1		MS/MR/A	N	N	1				1					102	138	40	1	4	5	1		LVH/LAE	N	MS/MR/AS/AR	
28	Mahalinga	69	1	41209	1	1					1	1	1	1		66	140/90	1	21.4	1	1	1					1	N	N	N				1						100	120	40	0.7	1	1	6		N	N		
29	Ramachan	60	1	41903				1	1	1					Nyst	78	120/80		31.6							1		N	N	N		1				1		1	1	132	178	39	0.8	4	4	6		N	N		
30	Malliga	62	2	42368	1	1	1	1						1		62	130/80		28.2							1		N	N	N	1				1	1				92	121	38	0.7	1	1	6		N	N		
31	Karunakar	49	1	43890						1		1				78	150/80	1	20.1	1				1				N	N	N	1									111	122	34	0.9	4	4	6		N	N		
32	Seharani	58	2	43993							1		1			90	200/110		19.8			1		1				N	N	N	1	1					1	1		146	192	42	1	4	4	6		N	N		
33	Sarasha	65	2	45179					1			1				84	130/80	1	22.5	1	1				+			N	N	N					1					98	117	37	0.9	1	1	6		N	N		
34	Rajaram	60	1	45628		1	1	1						1	dysphagia	66	140/90		23.9				1		1	1			N	N	1		1		1		1				102	138	24	0.8	3	3	3		IWMI	CMLY	RWMA	
35	Thangaraj	48	1	46878						1			1			78	180/100		20.6						1			N	N	N	1					1	1		1	98	123	44	1.3	1	5	5		LVH	CMLY	LVH	
36	Shahul Ha	53	1	47201	1	1					1					86	170/110	1	24.8						1			N	N	N		1				1				188	232	48	1.7	1	1	6		LVH	CMLY	LVH	
37	Purushoth	64	1	47283					1	1					1	82	130/80		23.2	1				1				N	N	N	1						1			92	127	39	1.1	2	2	6		N	N		
38	Ummar	69	1	49776	1	1	1	1				1			1	94	140/80		22.8							1		N	N	N	1					1				102	118	40	1	1	1	6		N	N		
39	Lekshmi	63	2	49967					1	1						76	120/80		23.6	1		1						N	N	N	1	1				1	1			138	212	34	0.7	4	5	4		N	N		
40	Rajeswari	39	2	50105	1	1					1					72	150/100	1	18.9	1	1	1						N	N	N								1		98	116	38	0.7	1	1	6		N	N		
41	Delvasiban	54	1	50628	1	1	1	1				1			1 dysphagia	92	130/90		22.8			1	1		1	1		N	N	N					1					90	126	38	0.2	4	5	4		N	N		
42	Muthukris	48	1	51493						1				1		66	110/70		25.6					1	1			N	N	N	1		1			1	1			92	128	40	1	1	1	6		N	N		
43	Joseph	52	1	51984	1	1				1			1			76	150/90	1	21.4	1				1				N	N	N	1	1				1		1	1	92	121	21	1	4	4	6		N	N		
44	Venkatesa	68	1	52107					1					1	1	86	100/70		26.2			1				1		N	N	N	1									111	122	29	1.5	4	4	6		N	N		
45	Tharaman	67	1	52336	1				1				1			96	140/80	1	23.3	1	1	1						N	N	N					1					100	130	42	1.1	1	1	6		N	N		
46	Kalvarasu	61	1	53451	1	1	1	1							1 Nyst	68	120/80	1	21.4							1		n	n	n	1							1		102	138	21	1	3	5	1		N	N		
47	Naravanas	59	1	54187	1	1					1	1			SEIZ	76	210/110		22.5	1				1				N	N	N		1		1						188	246	29	1.5	4	4	6		LAE	CMLY	MS/MR	
48	Venkatiah	79	1	54128							1					88	130/90	1	20.3					1				N	N	N	1		1		1	1	1	1		138	179	40	0.9	1	1	6		IWMI	CMLY	RWMA	
49	Govindam	70	2	55106							1					78	130/80		19.9	1				1		1		N	N	N						1				92	127	39	1.1	2	2	6	VBI				
50	Vellathai	67	2	55797	1	1	1	1							1	96	140/90	1	24.2							1		N	N	N	1									92	110	40	0.8	1	1	6		N	N		

ANNEXURE -3

KEY TO MASTER CHART

Sex: 1- Male, 2 - Female

Symptoms

Giddiness to Speech disturbances: 1 –Present, 2- Absent

Others:1- Seizure, 2- Dysphagia, 3 – Nystagmus,

Markers of Atherosclerosis :

1 -Present, 2- Absent

BMI:

<18.5 – Underweight, 18.5 – 24.9 – Normal weight, 25 – 29.9 – Overweight, 30 and Above – Obese

CNS Examination

HMF- Lobar functions (Vision, Face/ Obj recognition, memory) : 1 – Present Cranial Nerves to bowel and bladder symptoms: 1- Present, 2- absent. Other systems: N- Normal.

Risk Factors: (HTN to Hypercholesterolemia): 1 – Present

TIA: 1 – Present

Territory involved clinically: 1- Distal, 2- Middle, 3-Proximal, 4 – Multiple.

CT Brain: - 1 - Distal, 2- Middle, 3-Proximal, 4 – Multiple, 5 – Normal study

MRI Brain: - 1 - Distal, 2- Middle, 3-Proximal, 4 – Multiple, 5 – Normal study, 6 – Not done.

ANNEXURE -4

ABBREVIATIONS

AF – Atrial Fibrillation

AICA – Anterior inferior cerebellar artery

CN – Cranial Nerve

CNS – Central Nervous system

CT – Computed Tomography

ICA – Internal carotid artery

MRI – Magnetic Resonance Imaging

NEMC – PCSR – New England Medical Centre - Posterior circulation Stroke Registry

PCA – Posterior Cerebral Artery

PICA – posterior inferior cerebellar artery

SCA – Superior cerebellar artery

TIA – Transient Ischemic Attack

VBI – Vertebrobasilar insufficiency.

WHO – World Health Organisation