DISSERTATION

ON

"A STUDY OF 150 CASES OF POSTERIOR CIRCULATION STROKE"

Submitted in partial fulfilment of

requirements for the degree of

D.M. NEUROLOGY (BRANCH - I)

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CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY OF 150 CASES OF POSTERIOR CIRCULATION STROKE"** submitted by **Dr. C.J.SELVAKUMAR** appearing for **D.M. Neurology** Degree (Branch - I) examination in **August 2010** is a bonafide record of work done by him under my direct guidance and supervision in partial fulfilment 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.

Dr. M .CHANDRA SEKARAN, M.D., D.M., **DEAN**

Professor and Head, Madurai Medical College,

Department of Neurology & Neurosurgery, Madurai - 625 020.

Madurai Medical College,

Madurai - 625 020.

DECLARATION

I solemnly declare that the dissertation titled **"A STUDY OF 150 CASES OF POSTERIOR CIRCULATION STROKE"** is done by me at Department of Neurology, Madurai Medical College & Govt. Rajaji Hospital, Madurai , during 2008-2009 under the guidance and supervision of **Prof. M.CHANDRA SEKARAN, M.D., D.M.**

This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfilment of requirements for the award of **D.M., degree in Neurology.**

Place: Madurai,

Date: 31-05-2010.

Dr.C.J.SELVAKUMAR,

Postgraduate Student in

D.M. (Neurology),

Department of Neurology,

Madurai Medical College,

Madurai - 625 020.

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CONTENTS

INTRODUCTION

Stroke is known to human race since antiquity. The seventh century great Indian physician, Charaka lucidly described the symptoms of stroke which he called `*Pakshaghat*` meaning hit one half of body. The other synonyms are *ardhang* or *lakwa*. According to Charaka, stroke affects either left or right side of body leading to impaired mobility and function of that half of body (hemiparesis) and difficulty in speaking which may be inability to talk at all (aphasia) or slurred speech (dysarthria). He had also identified head as the seat of vital organ, controlling the senses and nerve centers of the whole body. These meticulous observations of stroke symptoms are relevant even today.

Stroke is one of the major causes of death disability and dependency among all the neurological disorders. The World Health Organization (WHO) defines stroke as rapidly developing clinical symptoms and / or signs of focal, at times global loss of cerebral function, with symptoms lasting more than 24 hours or leading to death, with no apparent cause other than of vascular origin.

The global prevalence of stroke is estimated to be 5 to 8 /1000. Globally stroke incidence was also variable according to the ethnic differences in a common geographical location and ranged from 93 to 223/1,00,000 population. Epidemiology of stroke in India is difficult to study due to multiple factors.

Nevertheless, many investigators have addressed this question in various regions of India, which may be considered representative of the whole population. The crude prevalence rate was 220/1,00,000(range : 44-843/1,00,000).

The incidence rate of stroke in India was estimated to be 13/1,00,000 in a study done at Vellore on a population sample of 2,58,576 followed over two years, while another study conducted at Rohtak found the stroke incidence to be 33/1,00,000 (27/1,00,000 for first ever stroke). The stroke risk increases steeply as the age advances. In a study from Kashmir, prevalence rate of stroke was 41 per 1,00,000 population in the age range of 15-39 years, which increased to 1,075 per 1,00,000 for the age group of 50-59 years.

The term *stroke* is applied to a sudden focal neurologic syndrome[1], specifically the type caused by cerebrovascular disease. The term *cerebrovascular disease* designates any abnormality of the brain resulting from a pathologic process of the blood vessels, including occlusion of the lumen by embolus or thrombus, rupture of a vessel, an altered permeability of the vessel wall, or increased viscosity or other change in the quality of the blood flowing through the cerebral vessels. The vascular pathologic process may be considered not only in its grosser aspects—embolism, thrombosis, dissection, or rupture of a vessel—but also in terms of more basic or primary disorder, i.e., atherosclerosis, hypertensive arteriosclerotic change, arteritis, aneurysmal dilatation, and developmental malformation. Most

cerebrovascular diseases are manifest by the abrupt onset of a focal neurologic deficit, as if the patient was "struck by the hand of God"[2]. Stroke is one of the most common neurological disorders in clinical practice. An estimated 5.7 million patients died from stroke in 2005, with 87% of these deaths occurring in underdeveloped countries. Globally, stroke is the third leading cause of death and a major cause of adult disability. It poses serious medical, socioeconomic, and rehabilitation problems. With the prevalence of disability resulting from stroke is expected to rise as populations increase, this burden will increase greatly over the next 20 years, particularly in developing countries. Stroke physicians are faced with the challenge of providing effective stroke care and reducing the mortality, disability, and dependency of stroke survivors.

Stroke is the most important single cause of long term disability in a community setting as about 30 to 50% of stroke patients are left with residual deficits. The hospital based studies had shown that 2% of all, 4 to 5 % of medical and 20% of neurological admissions were due to stroke. Not only this, survivors of a transient ischemic attack (TIA) or stroke are at an increased risk of another stroke. The new American Heart Association (AHA) guidelines estimated that about 28% of the prevalent strokes comprised recurrent strokes. This reflects the magnitude of the problem posed by stroke globally.

The cost of stroke is difficult to calculate but the disability-adjusted life years(DALY) lost in India due to stroke in 1990 were 62,48,000 and estimates of deaths and DALYs lost due to stroke by 2020 are expected to be 5,98,000 and 52,23,000. Though research is ongoing to identify the distribution and determinants of stroke for a long time, a breakthrough still awaits. Stroke in India is then a major public health problem.

Strokes occur either in anterior circulation or posterior circulation. Posterior circulation supplies approximately one-fifth of the total brain. The area includes brainstem, cerebellum, temporal lobes,occipital lobes and thalamus and is supplied by 2 vertebral arteries, 1 basilar artery and 2 posterior cerebral arteries[3]. Posterior circulation strokes comprise 10-15% of all strokes, 80% of them being ischemic strokes. Despite its much small size the posterior circulation contains the brainstem, a midline strategically critical structure without which consciousness, movement and sensations cannot be preserved. Posterior circulation ischemia can range from fluctuating brainstem symptoms, caused by intermittent insufficiency of the posterior circulation (VBI), to the 'locked-in syndrome' which is caused by basilar artery or bilateral vertebral artery occlusion [4-7]. Stroke syndromes of the posterior circulation account for approximately 20% all strokes, with upto 20-60% of patients having an unfavourable outcome. Basilar artery occlusion (BAO)

represents 8-14% of all posterior circulation strokes and carries mortality of over 90%[8].

The etiology of posterior circulation strokes has been thought to be primarily due to local arterial atherosclerosis (large artery disease) and penetrating artery disease (lacunes). However there is increasing evidence that cardiogenic embolization is more common than previously suspected and is responsible for 20-50% of posterior circulation strokes.

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

The intracranial branches of the vertebral artery and basilar artery were minutely studied and a syndrome was described for each prompting a cynic to remark the neurologic equivalent of Hall of Fame is a brainstem eponym.

AIM OF THE STUDY

- To study the demographic profile and symptoms
- To study the risk factors
- To study the pattern of posterior circulation stroke
- To prognosticate the posterior circulation stroke based on clinical and radiological findings

REVIEW OF LITERATURE

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, and retention of fetal circulatory patterns.

The proximal portions of the posterior circulation on the two sides differ. On the right, the subclavian artery arises from the innominate artery, a common channel supplying the anterior and posterior circulation. On the left side, the subclavian artery usually arises directly from the aortic arch after the origin of the left common carotid artery.

The first branch of each subclavian artery is the vertebral artery (VA). The Vertebral arteries course upward and backward until they enter the transverse foramens of C6 or C5 and run with in the intravertebral foramen exiting to course behind the atlas before the piercing the duramater to enter the foramen magnum[10]. Their intracranial portions end at the ponto-medullary junction, where the two vertebral arteries join to form the basilar artery. The first portion of vertebral artery before entry into the bony vertebral column is V1, the portion within the vertebral columns is V2, the portion after exit from the vertebral column that arches behind

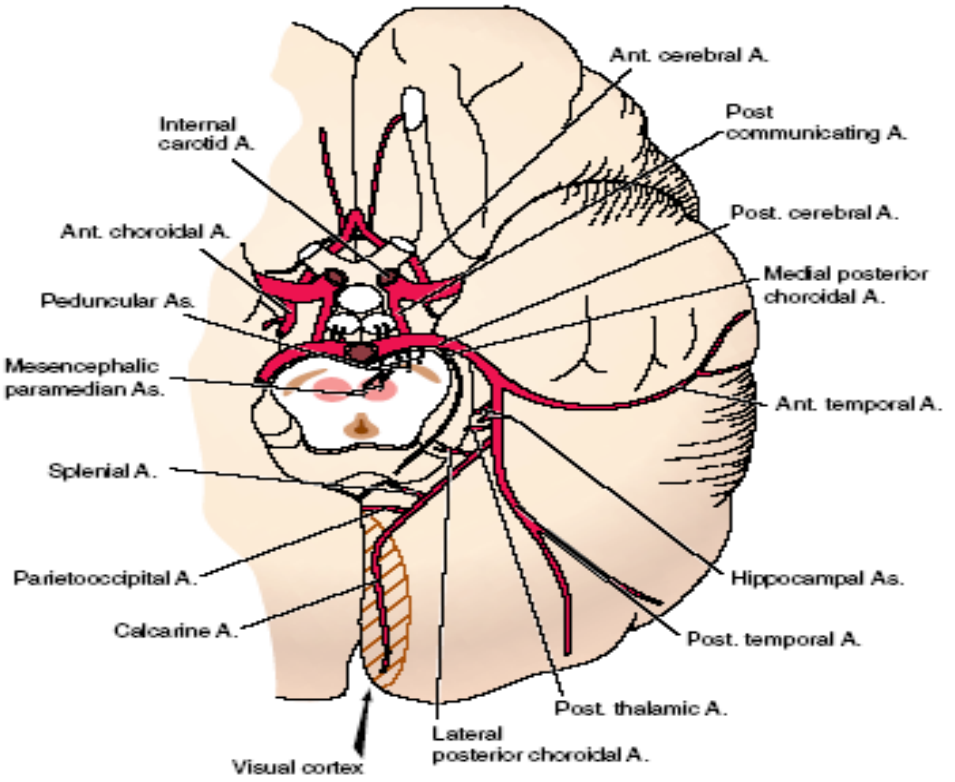
the atlas and before entry into the cranium is V3 and the intracranial portion is V4. In the neck vertebral arteries have many small muscular and spinal branches. The intracranial portion of the vertebral arteries gives off posterior and anterior spinal arteries, penetrating arteries to the medulla and the large posterior inferior cerebellar arteries (PICAs). Anastomotic channels exist among the ascending cervical arteries, the thyro cervical arteries, the occipital artery and the second segment of the vertebral artery.

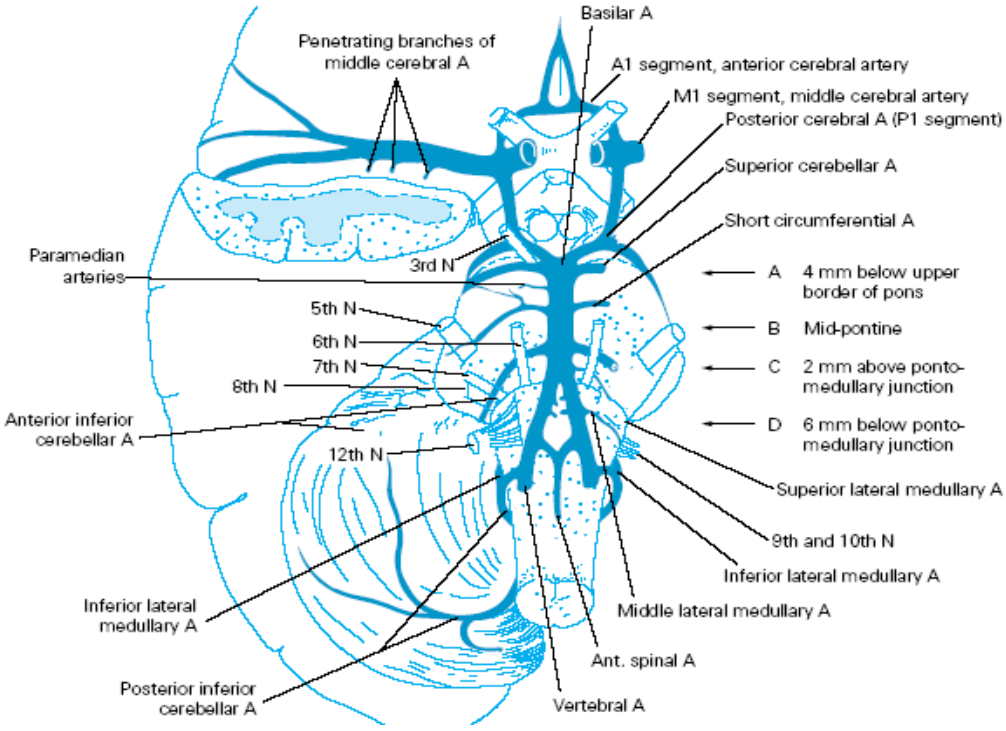
The Posterior Inferior Cerebellar Artery (PICA) is the largest branch of the vertebral artery, arising most commonly from its intradural segment. The PICA is divided into five segments according to Rhoton: an anterior medullary segment, beginning at the origin of the PICA and terminating at the level of the inferior olive, continues with the second or lateral medullary segment which ends at the level of the lower cranial nerves. The third, or tonsilomedullary, segment is closely related to the tonsils, forming a caudal loop. The fourth, or retrotonsilar, segment starts at the midlevel of the tonsil and ends where the artery exits to become hemispheric. The last segment, the hemispheric segment, supplies the occipital surface of the vermis and cerebellar hemisphere. The vascular territory of the PICA is highly variable and reflects the high degree of variability of this artery. It appears to be in balance with other major vessels in the posterior fossa. It supplies the lateral medullary area in 50% of cases. Its cerebellar territory includes the

globose and emboliform nuclei. The posterior inferior cerebellar artery (PICA) in its proximal segment supplies the lateral medulla and its distal branches supply the inferior surface of the cerebellum.

The basilar artery runs in the midline along the clivus, giving off bilateral anterior inferior cerebellar artery (AICA) and superior cerebellar artery (SCA) branches dividing at the pontomesencephalic junction into terminal posterior cerebral artery branches. The anterior inferior cerebellar artery (AICA) originates from the basilar artery at the level of the ponto-medullary sulcus and curves in a caudal direction around the pons towards the cerebellopontine angle. At this level it divides into superior and inferior trunks. The inferior trunk passes below the flocculus and vascularizes the inferior portion of the petrosal surface of the cerebellar hemisphere. The superior trunk has an upward curve and anastomoses with the superior cerebellar artery.

The superior cerebellar artery (SCA) originates from the superior segment of the basilar artery, usually a few millimeters before it divides into the posterior cerebral arteries. Duplication of the SCA is common and noted in 80% of cases. The course of the SCA is parallel to the posterior cerebral artery and is separated from the latter by the third nerve. Both arteries sweep around the brain stem towards the quadrigeminal plates. There, the SCA makes a sharp upward turn to reach the cerebellar hemisphere. It supplies the lower midbrain, upper pons, the dentate

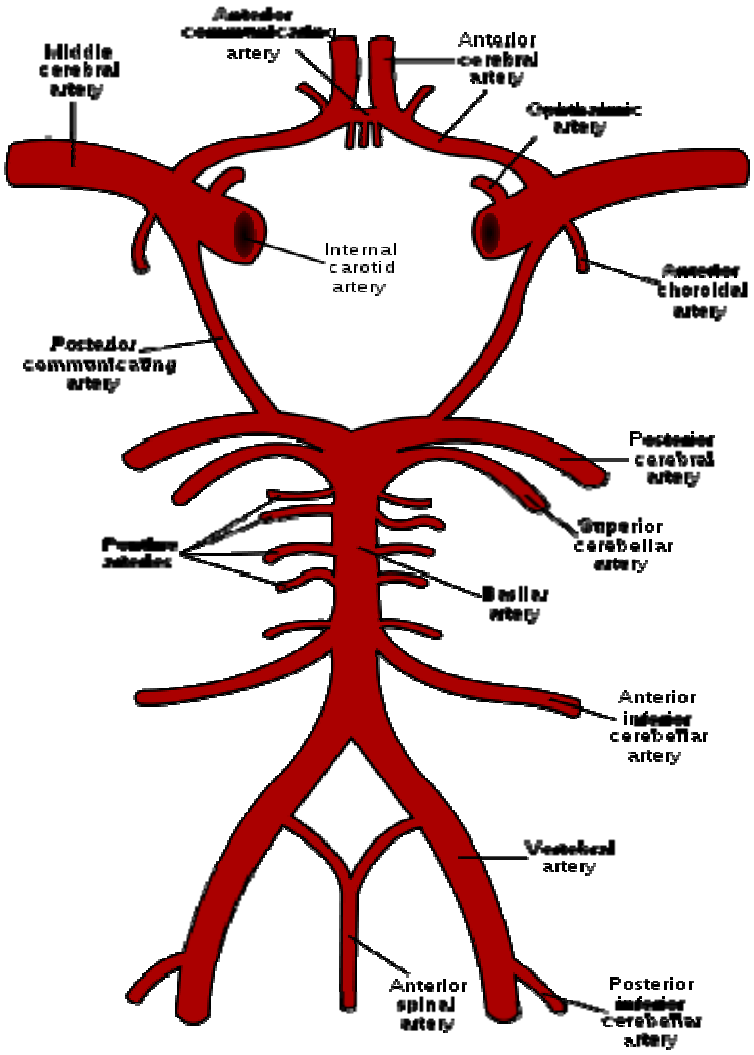




Posterior cerebral artery and its branches & Vertebrobasilar arterial system

nucleus and the tentorial surface of the cerebellar hemisphere.

Large paramedian arteries and smaller short circumferential arteries penetrate through the basal portion of the brain stem into the tegmentum. Long circumferential arteries course around the brainstem giving off branches to the lateral tegmentum.



Circle of Willis and Vertebrobasilar arterial system

In 75% of cases, both posterior cerebral arteries arises from the bifurcation of the basilar artery, in 20% one has its origin from the ipsilateral internal carotid artery (fetal origin of the PCA), in 5% of cases, both originate from the respective, ipsilateral internal carotid arteries. The precommunal, or P1 segment of the true posterior cerebral artery is atretic in such cases.

The peduncular or P1 segment of the true posterior cerebral artery gives rise to small brainstem penetrating branches that supply the medial part of cerebral peduncles, the substantia nigra, red nucleus, oculomotor nucleus, midbrain reticular formation, subthalamic nucleus, decussation of the superior cerebellar peduncles, medial longitudinal fasciculus and the medial lemniscus. The artery of Percheron arises from either the right or the left precommunal segment of the posterior cerebral arteries and it divides in the subthalamus to supply the inferomedial and anterior portions of the thalamus and subthalamus bilaterally.

The ambient or P2 segment of the posterior cerebral arteries gives off small circumferential branches that course around the midbrain to supply the lateral part of cerebral peduncles, medial lemniscus, tegmentum of the midbrain, superior colliculi, lateral geniculate body, posterolateral nucleus of the thalamus, choroid plexus and hippocampus. The thalamogeniculate branches, which originate from the P2 segment of the posterior cerebral artery, supply the thalamus, geniculate body, posterior limb of internal capsule and optic tract. The medial posterior

choroidal artery arising from proximal P2 supplies the colliculi, posterior thalamus, pineal gland and part of midbrain. The lateral posterior choroidal artery supplies the choroidal plexus of the third and fourth ventricles.

The quadrigeminal or P3 segment runs within the quadrigeminal cistern behind the brainstem. Its branches are the inferior temporal arteries that supply the inferior surface of the temporal lobe. The parieto-occipital artery travels in the parieto-occipital fissure and supplies most of the posterior one third of the brain's medial surface and a small area of the lateral surface. The calcarine artery runs in the calcarine fissure and supplies the visual cortex and the occipital pole. The splenial artery is where the posterior pericallosal artery anastomoses with the pericallosal artery from the ACA, runs adjacent to the corpus callosum at the splenium, and supplies the posterior part of the corpus callosum.

Physiology of blood flow in brain

The brain is metabolically active organ. The brain uses glucose as its sole substrate for energy metabolism. A constant supply of Adenosine Tri Phosphate (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 500 CC of oxygen and 75-100 mg of

glucose each minute and a total of 125g of glucose each day[11]. The brain uses approximately 20% of cardiac output when the body is resting. Cerebral blood flow is normally approximately 55ml for each 100 g of brain tissue per minute and cerebral oxygen consumption is normally approximately 3.5 cc / 100g / minute. Reduction of CBF below 10 to 12 mL/100 g/min causes infarction, almost regardless of its duration. The critical level of hypoperfusion that abolishes function and leads to tissue damage is therefore a CBF between 12 and 23 mL/100 g/min. At these levels of blood flow the electroencephalogram (EEG) is slowed, and below this level it becomes isoelectric. In the region of marginal perfusion, the K^+ level increases (as a result of efflux from injured depolarized cells) and adenosine triphosphate (ATP) and creatine phosphate are depleted. These biochemical abnormalities are reversible if the circulation is quickly restored to normal. Disturbance of calcium ion homeostasis and accumulation of free fatty acids interfere with full recovery of cells. A CBF of 6 to 8 mL/100 g/min causes marked ATP depletion, increase in extracellular K^+, increase in intracellular Ca, and cellular acidosis, invariably leading to histologic signs of necrosis. These changes do not become apparent for several hours. Free fatty acids (appearing as phospholipases) are activated and destroy the phospholipids of neuronal membranes. Prostaglandins, leukotrienes, and free radicals accumulate, and intracellular proteins and enzymes are denatured. Cells then swell, a process called

cellular, or *cytotoxic, edema*. Similar abnormalities affect mitochondria even before other cellular changes are evident.

Brain energy use and blood flow depend on the degree of neuronal activity. PET and functional magnetic resonance imaging show that using the right hand increases, metabolism and cerebral blood flow in motor cortex. The capacity of the cerebral circulation to maintain relatively constant level of cerebral blood flow despite changing blood pressure has traditionally been termed autoregulation. Cerebral blood flow remains constant when mean arterial blood pressures are between 50 and 150 mm of Hg[12].

The survival of the brain regions at risk depends on a number of factors[13]

1. Adequacy of collateral circulation
2. The state of systemic circulation
3. Serologic factors
4. Changes within the obstructing vascular lesions and
5. Resistance within the micro circulatory bed.

Distribution of Vascular pathology - Thrombosis

Normally, vertebral artery is about half the diameter of the internal carotid artery. One vertebral artery is frequently much smaller and the opposite vertebral artery is unusually large so that the total quantity of blood flowing through the basilar artery

is not altered. In some cases, when two vertebral arteries are of normal size they do not join in the usual fashion. One vertebral artery continues as basilar artery and the other as the posterior inferior cerebellar artery. The vertebral artery gives numerous branches as it ascends through the neck, it has a rich series of anastomoses with the thyrocervical trunk, the vertebral artery of the opposite side and the occipital branch of the external carotid artery. Any of these anastomoses may bypass segmental occlusion.

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[14-17]. Atherosclerotic narrowing rarely affects the distal superficial branches like posterior inferior cerebellar artery, anterior inferior cerebellar artery and superior cerebellar arteries.

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 predilections 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 contralateral vertebral arteries or the ascending cervical, thyrocervical or occipital arteries is usually sufficient to prevent low flow transient ischemic attacks or stroke. Whether infarction is single or multiple depends on the interplay of the atherosclerotic lesion in the collateral vessels, the leptomeningeal arteries and the posterior communicating artery that connects the carotid with the vertebrobasilar system.

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

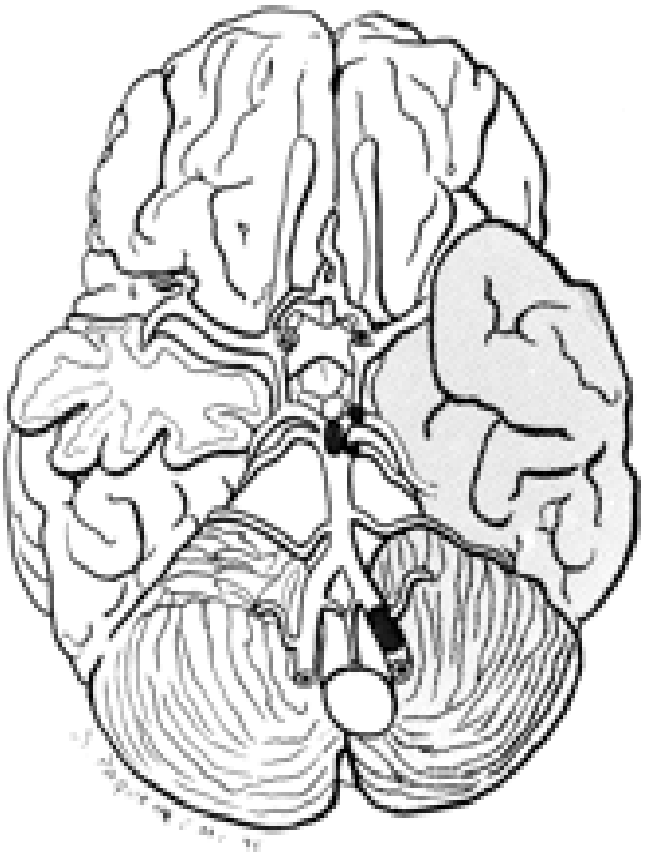
cerebellum.

If the subclavian artery is occluded proximal to the origin of the vertebral artery, there is reversal in the direction of blood flow in the ipsilateral vertebral artery. Exercise of the ipsilateral arm may increase demand on vertebral flow, 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[18], fibromuscular dysplasia and rarely encroachment by osteophytic spurs within the vertebral foramina.

Atheromatous lesions can occur anywhere along the basilar trunk but are most frequent in the proximal basilar and distal vertebral segments. Embolisms from heart or proximal vertebral or basilar segments are more commonly responsible for `Top of the basilar` syndromes. Temporal arteritis affects the vertebral arteries just before they pierce the duramater to enter the cranial cavity[19].

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 rate of occlusion, adequacy of collateral circulation, and resistance of brain structures to ischemia.



Intracranial posterior circulation recipient sites for embolism

Risk Factors

The risk factors for posterior circulation strokes are the same as for other forms of cardiovascular disease. These are either modifiable or nonmodifiable. The non-modifiable risk factors include age, gender, race, and family history of stroke or TIA. The modifiable risk factors include hypertension, heart disease such as atrial fibrillation, ischemic heart disease, rheumatic heart disease, hypercoagulable states, antiphospholipid antibody syndrome, homocystinemia etc.,. Stroke risk doubles with each decade past age 55.

Past history of stroke or a TIA increases the risk of having another stroke upto ten times. 35% patients of those who experience TIAs have a stroke within five years. Heart disease like atrial fibrillation increases the stroke risk upto six times. Smoking doubles stroke risk. The most common causes for vertebrobasilar occlusion are arteriosclerosis in the elderly and trauma in the younger population[20].

Trauma to the vertebral artery inflicted by hyperextension or sudden rotatory movements of the neck can produce disaster brainstem disease. The most common causes of such trauma seem to be hyperextension or whiplash injuries of the neck, over enthusiastic chiropractic manipulation, freek accidents occurring during football and swimming. A rarer cause is extension of the head on the neck for dental extraction or for intubation during general anaesthesia.

Puncture of the mid cervical portion for arteriography occasionally results in neural deficit. This procedure has been known to cause subintimal dissection leading to acute occlusion of the vessel. It can also initiate vasospasm which may result in signs and symptoms of cerebrovascular insufficiency. Osteoarthritis of cervical spine, congenital anomalies of cervical spine such as the Klippel-Feil syndrome, basilar impression, cervical rib and fibrous bands. Compression by slips of muscle from the insertion of the longus colli and scalenus anticus at C6 vertebra can also produce temporary arterial obstruction when the head is turned to one side.

Clinical features

Posterior circulation is not a homogenous entity. Many patients are severely disabled or die, where as others suffer only transient or minor disability. The prognosis varies and is dependent on multiple factors including

1. The nature, locus and severity of vascular lesions.
2. The presence of coexisting vascular lesions elsewhere.
3. Hemodynamic, circulatory and coagulation factors.
4. Congenital constitution of the individual vascular bed.

Patients usually present with a wide variety of symptoms of neurological dysfunction includes hemi or quadriparesis, cranial nerve defects (III – XII), respiratory difficulty, altered sensorium, vertigo and ataxia. Multiple cranial nerve signs indicate involvement of more than one brain stem level. Patients may present with only hemiparesis, which may progress rapidly to quadriparesis or a locked in syndrome. The onset of symptoms may not be as abrupt as with anterior circulation strokes[21]. As the posterior circulation supplies the brainstem, cerebellum, and occipital cortex, the symptoms frequently involve were dizziness, diplopia, dysarthria, dysphasia and dystaxia. The hallmark of posterior circulation stroke is crossed findings, with cranial nerve finding on the side of the lesion and

motor, sensory findings on the opposite side. The exact symptoms depend on the precise location of infarct.

THROMBOTIC STROKES

Posterior cerebral artery syndrome

Historically, the French neurologist Charles Foix[22] in 1923 first described the syndrome of infarction in the PCA territory as a thalamocapsular deficit. Embolic occlusion is the usual cause of stroke in this territory. Two clinical syndromes are commonly observed with occlusion of the PCA:

(1) *P1 syndrome*: midbrain, subthalamic, and thalamic signs, which are due to disease of the proximal P1 segment of the PCA or its penetrating branches (thalamogeniculate, Percheron, and posterior choroidal arteries)

(2) *P2 syndrome*: cortical temporal and occipital lobe signs, due to occlusion of the P2 segment distal to the junction of the PCA with the posterior communicating artery.

P1 syndrome

If the proximal P1 segment is occluded infarction usually occurs in the ipsilateral subthalamus and medial thalamus and in the ipsilateral cerebral peduncle and midbrain. A third nerve palsy with contralateral ataxia and tremor - Claude's

syndrome or with contralateral hemiplegia – Weber's syndrome may result. The ataxia indicates involvement of the red nucleus or dentatorubrothalamic tract and hemiplegia is localized to the cerebral peduncles. If the subthalamic nucleus is involved, contralateral hemiballismus may occur. Occlusion of the artery of Percheron causes paresis of upward gaze, drowsiness and often abulia.

Extensive infarction in the midbrain and subthalamus occurring with bilateral proximal posterior cerebral artery occlusion presents as coma, unreactive pupils, bilateral pyramidal signs and decerebrate rigidity.

The occlusion of penetrating branches of thalamic and thalamogeniculate arteries produce less extensive thalamic and thalamocapsular lacunar syndromes. The features of thalamic Dejerine – Roussy syndrome are contralateral hemisensory loss followed later by an agonizing tearing or burning pain in affected areas. Associated signs include hemiparesis, hemiballismus, choreoathetosis, intention tremor, inco-ordination and posturing of the hand and arm particularly while walking.

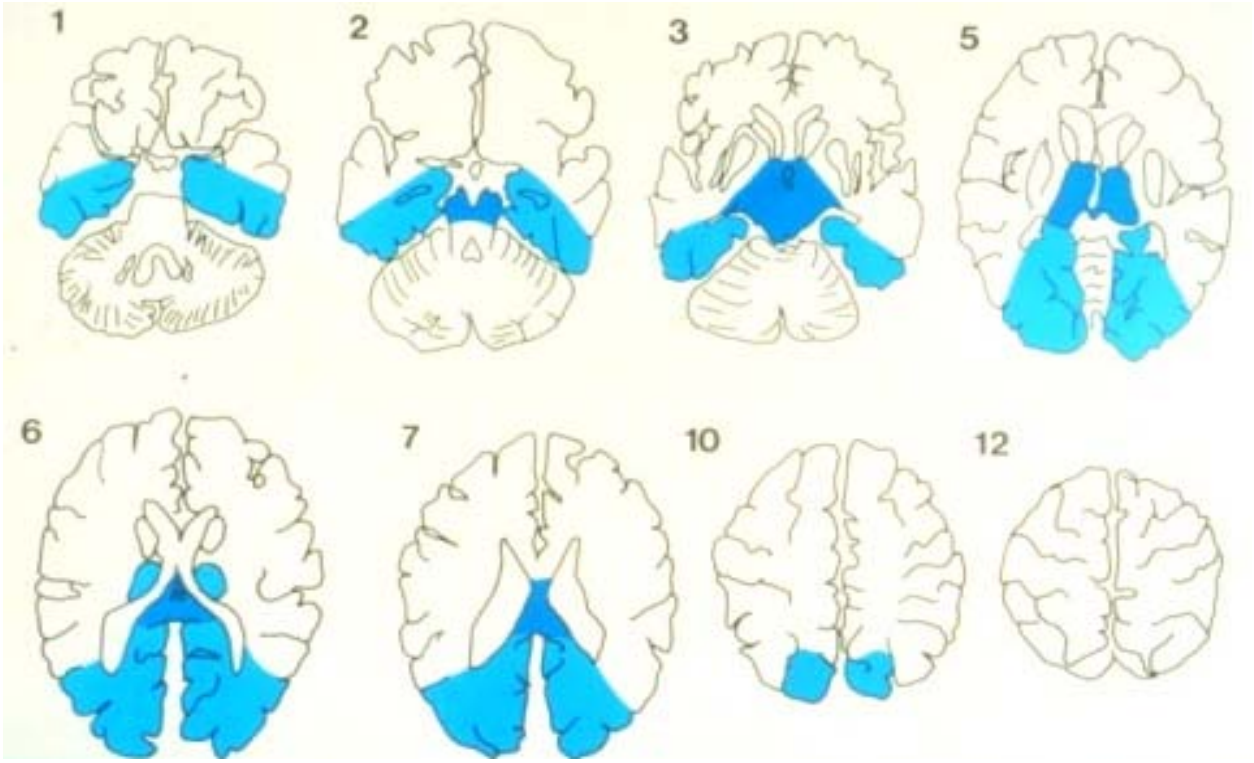
P2 syndrome

Occlusion of the distal PCA segments cause infarction of the medial temporal and occipital lobes. Contralateral homonymous hemianopia with macular sparing is

the usual presentation. Occasionally only the upper quadrant of the visual fields is involved.

Medial temporal lobe and hippocampal involvement may cause an acute disturbance in memory particularly if it occurs in the dominant hemisphere. The defect usually clears, because memory has bilateral representation.

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 produces peduncular hallucinosis.



Posterior cerebral artery – vascular territory

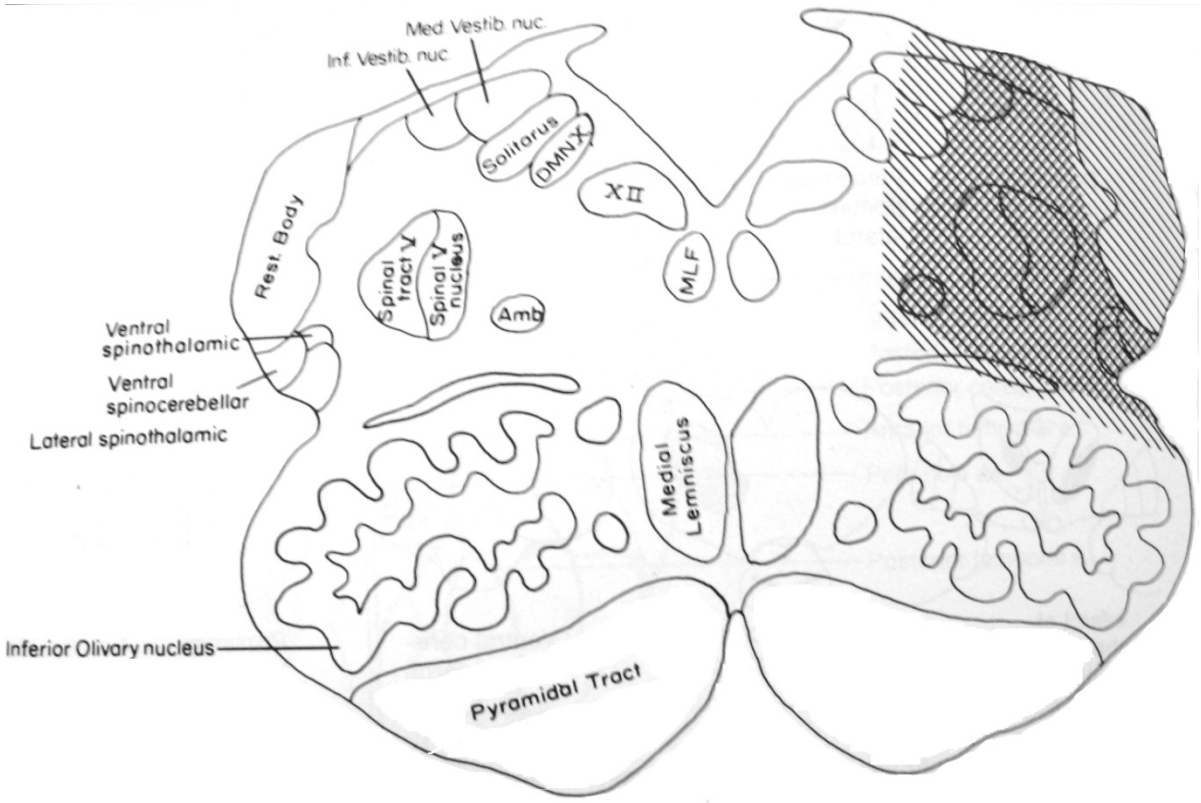
Bilateral infarction in the distal posterior cerebral arteries produces cortical blindness and the patient is often unaware of it, or may even deny it – Anton's syndrome. A constellation of symptoms termed Balint's syndrome can occur, usually with bilateral visual association area lesions. Bálint's syndrome involves deficits in the orderly visuomotor scanning of the environment (*oculomotor apraxia*) and in accurate manual reaching toward visual targets (*optic ataxia*). The third and most dramatic component of Bálint's syndrome is known as *simultanagnosia* and reflects an inability to integrate visual information in the center of gaze with more peripheral information. The patient gets stuck on the detail that falls in the center of gaze without attempting to scan the visual environment for additional information. The patient with simultanagnosia "misses the forest for the trees." Balint's syndrome occurs most often with infarctions secondary to low flow in the watershed between the distal posterior and middle cerebral artery territories. Patients may even experience persistence of a visual image for several minutes despite gazing at another scene (palinopia).

Vertebral and posterior inferior cerebellar arteries syndrome

The occlusion of these arteries produces two syndromes

1. Lateral medullary syndrome which presents with vertigo, numbness of the ipsilateral face and contralateral limbs, ipsilateral ataxia, diplopia,

hoarseness of voice, dysarthria, dysphagia and ipsilateral Horner's syndrome.



2. Rarely, a medial medullary syndrome occurs with infarction of the pyramid causing contralateral hemiparesis of the arm and leg, sparing the face. If the medial lemniscus and emerging hypoglossal nerve fibers are involved contralateral loss of joint position sense and ipsilateral tongue weakness occur.

Basilar artery syndrome

Basilar artery (BA) occlusive disease is a highly life threatening condition first described by Kubik and Adams (1946). Basilar artery occlusion secondary to

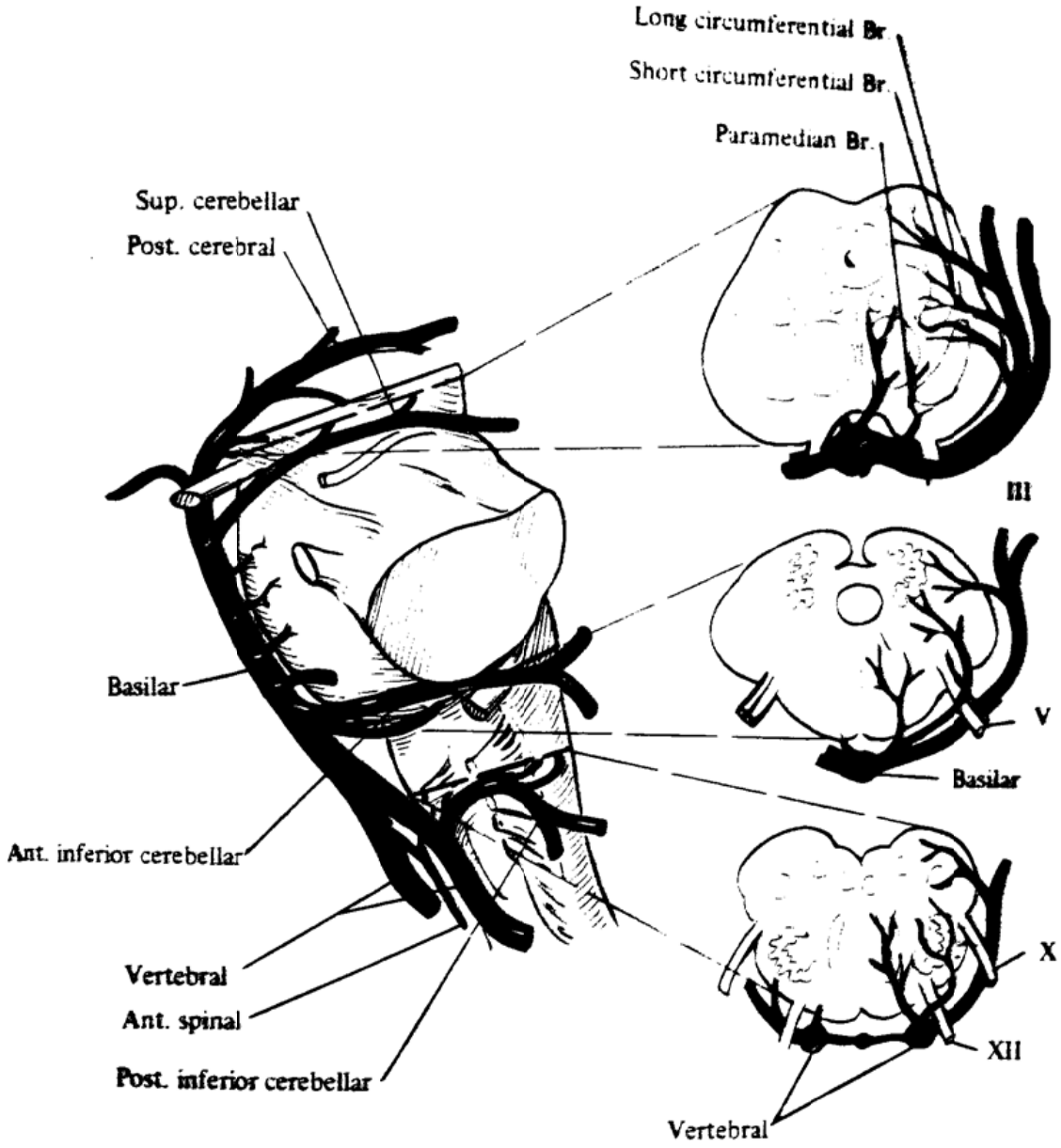
atherosclerosis is most prevalent in the sixth and seventh decades of life. Occlusion of the distal basilar artery is usually secondary to embolism and is most frequent in the fourth decade. The picture of basilar artery occlusion is easily recognized by a constellation of bilateral long tract signs (sensory and motor) with signs of cranial nerve and cerebellar dysfunction. Disturbance of eye movements occur because of infarction of the lateral gaze centers in the paramedian pontine tegmentum, e.g. the medial longitudinal fasciculus (internuclear ophthalmoplegia), the parapontine reticular formation (PPRF), which generates lateral gazes, or the combination of both, resulting in the so called "one and a half syndrome". Infarction of the medial pontine tegmentum will cause coma and is a poor prognostic sign. In most patients with BA thrombosis, obstruction is limited to the mid portion of the basilar artery. Embolic occlusion rather than thrombotic occlusion mainly blocks the distal part of the BA when it divides into the PCAs. The distal BA supplies the midbrain and the diencephalon by small perforating arteries. Occlusion of branches at the bifurcation (top) of the basilar artery results in a remarkable number of complex syndromes that include, in various combinations, somnolence or coma, memory defects, akinetic mutism, visual hallucinations, ptosis, disorders of ocular movement (convergence spasm, paralysis of vertical gaze, retraction nystagmus, pseudoabducens palsy, retraction of upper eyelids, skew deviation of the eyes), an agitated confusional state, and visual field defects The triad of hypersomnia,

supranuclear vertical-gaze defect, and amnesia (the so-called "paramedian diencephalic syndrome") is typically due to bilateral paramedian thalamic strokes in the territory of the anterior thalamic/subthalamic (or thalamoperforating) arteries ("*en ailesde papillon*")[22].

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 brief brainstem TIAs occurring several times a day. Patients become stuporous or comatose as the reticular activating system becomes involved.

Superior cerebellar artery syndrome

Occlusion of the superior cerebellar artery results in severe ipsilateral cerebellar ataxia, nausea and vomiting, dysarthria, and contralateral loss of pain and temperature sensation over the extremities, body, and face (spino and trigeminothalamic tract). Partial deafness, ataxic tremor of the ipsilateral upper extremity, horner's syndrome, and palatal myoclonus may occur rarely.



Vertebrobasilar arterial supply

Anterior Inferior cerebellar artery syndrome

Occlusion of the anterior inferior cerebellar artery produces variable degrees of infarction because the size of this artery and the territory it supplies vary inversely

with those of the PICA. The principal symptoms include: (1) ipsilateral deafness, facial weakness, vertigo, nausea and vomiting, nystagmus, tinnitus, cerebellar ataxia, horner's syndrome, and paresis of conjugate lateral gaze; and (2) contralateral loss of pain and temperature sensation. An occlusion close to the origin of the artery may cause corticospinal tract signs.

Lacunar stroke

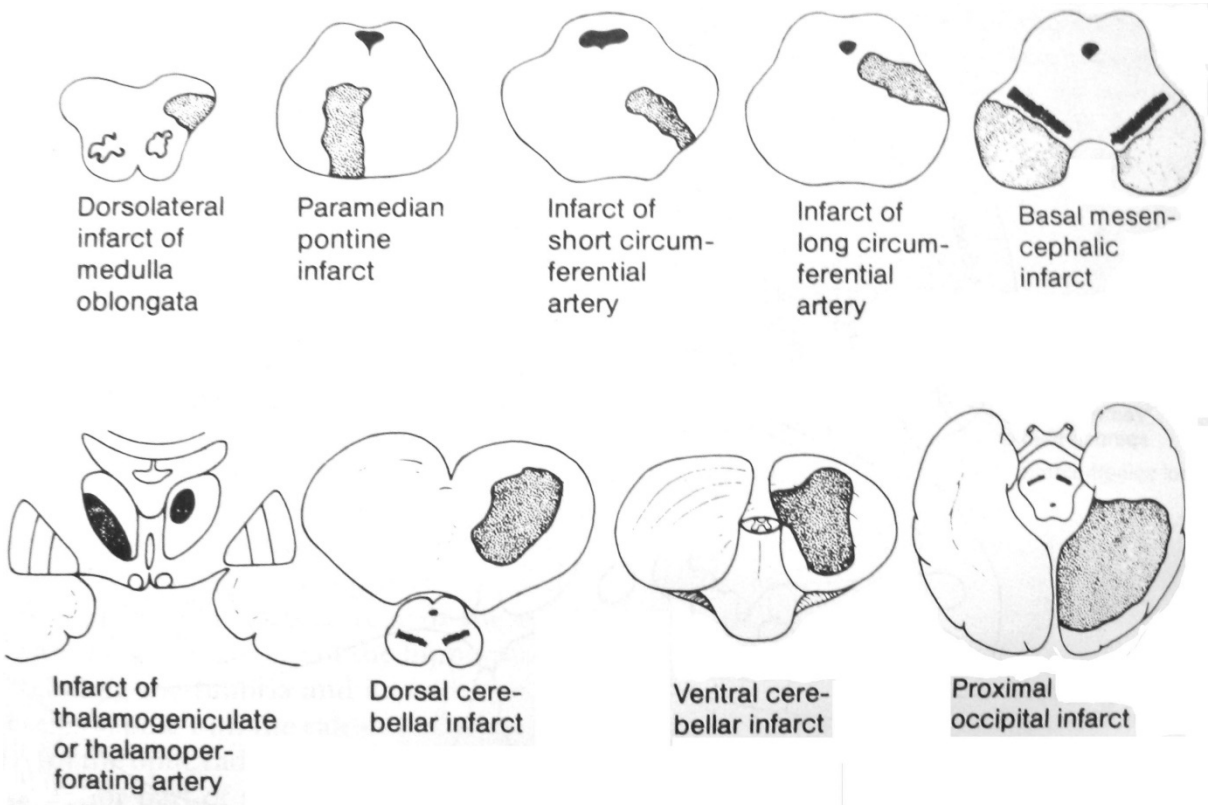
Refers to infarction following atherothrombotic or lipohyalinotic occlusion of one of the small vessels. They range in size from 3-4 mm to 1-2 cm. Hypertension and age are the principal risk factors. Lacunar infarcts cause approximately 20% of all strokes.

Brainstem lacunar infarcts produce a wide range of symptoms and signs.

1. Pure motor hemiparesis from an infarct in the posterior limb of internal capsule or basis pontis.
2. Pure sensory stroke from an infarct in ventrolateral thalamus.
3. Ataxic hemiparesis from an infarct in the base of the pons.
4. Dysarthria and a clumsy hand or arm due to infarction in the base of the pons or in the genu of internal capsule.

Syndromes resulting from occlusion of the penetrating arteries of the basilar artery include ipsilateral ataxia and contralateral crural paresis, hemiparesis with

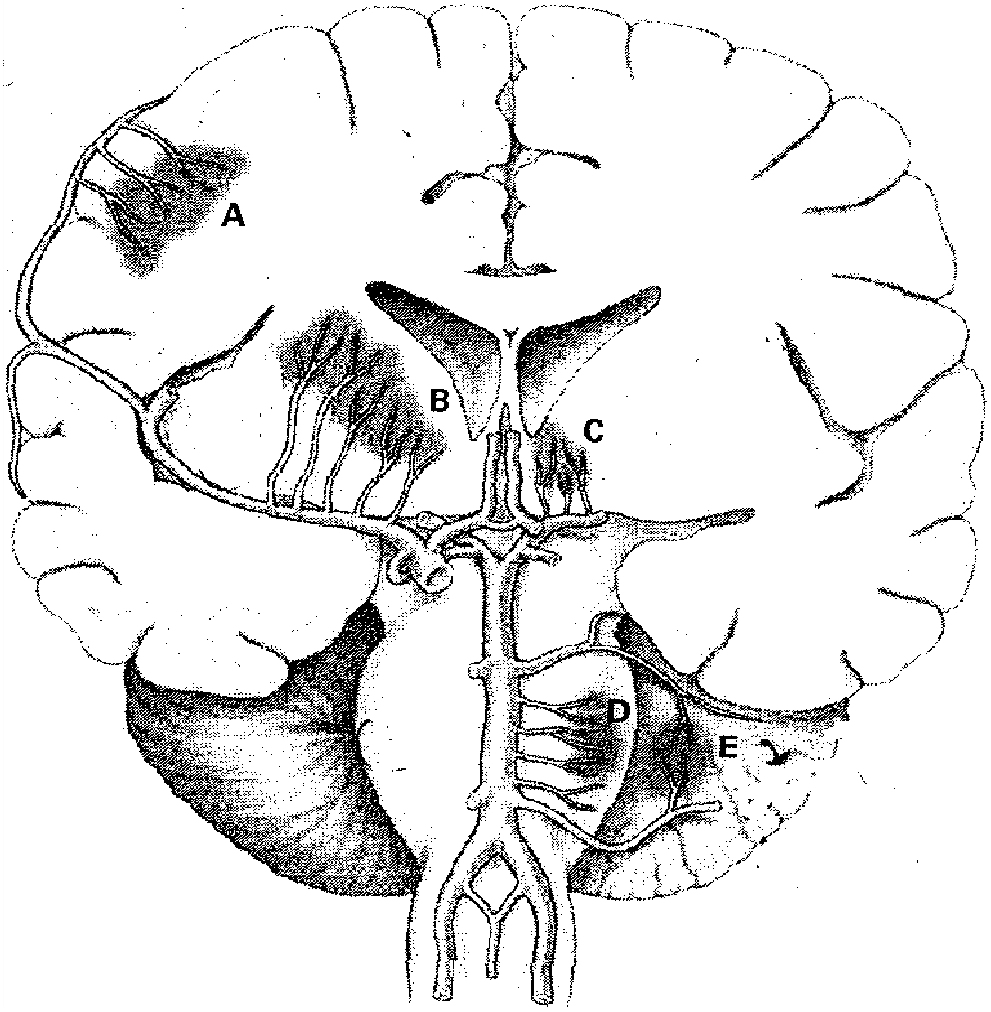
horizontal gaze palsy, and hemiparesis with crossed sixth nerve palsy. Lower basilar branch syndromes include internuclear ophthalmoplegia, horizontal gaze palsy and appendicular cerebellar ataxia.



Location of infarcts in posterior circulation

HAEMORRHAGIC STROKES

The incidence of spontaneous ICH may be higher among black persons than white persons, since a higher incidence of hypertension is seen in black persons younger than 45 years.



Location of haemorrhages in the brain

A-Lobar hge - penetrating. br of ACA , MCA , PCA , B-Basal ganglial hge - lenticulostriate br. of MCA, C-Thalamic hge- thalamogeniculate br. of PCA, D- Pontine hge- paramedian br. of Basilar artery, E-Cerebellar hge- penetrating br. of PICA , AICA , SCA.

Men have a 5-20% higher incidence of ICH than women. Of spontaneous intracerebral hemorrhage patients, 90% are older than 45 years.

The arteries in the brain damaged by exposure to chronic hypertension typically are the perforator arteries, which serve the basal ganglia, thalamus, and pons. Other

areas that also may be affected include the centrum semiovale and, occasionally, the cerebellum.

Thalamic hemorrhages also produce a contralateral hemiplegia or hemiparesis from pressure on, or dissection into, the adjacent internal capsule. A prominent sensory deficit involving all modalities is usually present. Aphasia, often with preserved verbal repetition, may occur after hemorrhage into the dominant thalamus, and constructional apraxia or mutism occurs in some cases of nondominant hemorrhage. There may also be a homonymous visual field defect. Thalamic hemorrhages cause several typical ocular disturbances by virtue of extension inferiorly into the upper midbrain. These include deviation of the eyes downward and inward so that they appear to be looking at the nose, unequal pupils with absence of light reaction, skew deviation with the eye opposite the hemorrhage displaced downward and medially, ipsilateral Horner's syndrome, absence of convergence, paralysis of vertical gaze, and retraction nystagmus. Patients may later develop a chronic, contralateral pain syndrome (Déjerine-Roussy syndrome).

In pontine hemorrhages, deep coma with quadriplegia usually occurs over a few minutes. There is often prominent decerebrate rigidity and "pin-point" (1 mm) pupils that react to light. There is impairment of reflex horizontal eye movements

evoked by head turning (doll's-head or oculocephalic maneuver) or by irrigation of the ears with ice water. Hyperpnea, severe hypertension, and hyperhidrosis are common. Death often occurs within a few hours, but small hemorrhages are compatible with survival.

Hypertensive hemorrhages in the cerebellum tend to occur adjacent to the dentate nucleus or in the deep white matter, depending on the perforating branches of the superior cerebellar or posterior inferior cerebellar arteries. Cerebellar hemorrhages usually develop over several hours and are characterized by occipital headache, repeated vomiting, and ataxia of gait. In mild cases there may be no other neurologic signs other than gait ataxia.

Dizziness or vertigo may be prominent. There is often paresis of conjugate lateral gaze toward the side of the hemorrhage, forced deviation of the eyes to the opposite side, or ipsilateral sixth nerve palsy. Less frequent ocular signs include blepharospasm, involuntary closure of one eye, ocular bobbing, and skew deviation. Dysarthria and dysphagia may occur. As the hours pass, the patient often becomes stuporous and then comatose from brainstem compression or obstructive hydrocephalus; immediate surgical evacuation before brainstem compression occurs may be lifesaving. Hydrocephalus from fourth ventricle compression can be relieved by external ventricular drainage, but definitive hematoma evacuation is

essential for survival. If the deep cerebellar nuclei are spared, full recovery is common. The major neurologic deficit with an occipital hemorrhage is hemianopia and with a left temporal hemorrhage, aphasia and delirium.

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.

TIAs occur for 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 syndrome. 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. 36% presented awake, while 23% were somnolent and 30% were comatose[23, 24].

Investigations

CT scan was the initial procedure of choice. But it is ineffective in showing brainstem and cerebellar infarcts. But it is used mainly to exclude brainstem hemorrhages. Scans obtained in the first several hours after an infarction generally show no abnormality, and the infarct may not be reliably seen for 24 to 48 hr. Even later CT may fail to show small ischemic strokes in the posterior fossa because of the bone artifact.

In patients presenting with a high pretest probability of posterior circulation stroke based on clinical symptoms, the presence of the HDBA sign (hyperdense basilar artery) on unenhanced CT is a strong predictor of basilar artery thrombosis, and both short- and long-term outcome[25]. 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.

The advent of MRI, with its ability to image posterior fossa structures clearly, made investigation of vertebrobasilar territory infarcts more feasible. 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 intra cranial haemorrhage and other abnormalities. The higher the field strength, the more reliable and precise the image.

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. Magnetic resonance angiography has a sensitivity of 97% and a specificity of 98.9%[26].

MRI is less sensitive for acute blood products than CT and is more expensive and less readily available. Claustrophobia also limits its application. Most acute stroke protocols use CT because of these limitations. However, MRI may be useful outside the acute period by more clearly defining the extent of tissue injury and discriminating new from old regions of brain infarction. MRI may have particular utility in patients with TIA: it is also more likely to identify new infarction, which is a strong predictor of subsequent stroke. The appearance of haemorrhages in MRI is given below.

Evolution of Intraparenchymal Hematoma

Phase	Time	Hemoglobin, Location	Appearance	
			T1-Weighted MRI	**T2-Weighted MRI**
Hyperacute	<24 h	Oxyhemoglobin, intracellular	Isointense or hypointense	Hyperintense
Acute	1-3 d	Deoxyhemoglobin, intracellular	Hypointense	Hypointense
Early subacute	>3 d	Methemoglobin, intracellular	Hyperintense	Hypointense
Late subacute	>7 d	Methemoglobin, extracellular	Hyperintense	Hyperintense
Chronic	>14 d	Ferritin and hemosiderin, extracellular	Hypointense	Hypointense

Diffusion-weighted imaging is more effective than T2-weighted imaging in patients with acute posterior-circulation strokes[27]. MRI with fat suppression is an imaging sequence used to visualize extra 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.

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 extra cranial subclavian arteries and the vertebral arteries.

Conventional X-ray cerebral angiography is the gold standard for identifying and quantifying 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 and positron emission tomography (PET)} 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 revascularization surgery. Single photo emission tomography (SPECT), CT – perfusion, and MR- perfusion techniques report relative cerebral blood flow and

currently are research tools. Cardiac investigations also improved with better echocardiography, which showed lesions in the aorta as well as the heart. Hence it becomes possible to investigate the brain and cardiovascular lesions, and stroke mechanism quickly and noninvasively in patients with posterior circulation ischemia.

Treatment

Blood pressure should be lowered if there is malignant hypertension or concomitant myocardial ischemia or if blood pressure is >185/110 mmHg and thrombolytic therapy is anticipated. In other patients blood pressure reduction is necessary when the BP>220/120mmHg in ischaemic stroke and BP>185/105 in haemorrhagic stroke.When faced with the competing demands of myocardium and brain, lowering the heart rate with a β_1-adrenergic blocker (such as esmolol) can be a first step to decrease cardiac work and maintain blood pressure. Fever is detrimental and should be treated with antipyretics and surface cooling. Serum glucose should be monitored and kept at <6.1 mmol/L (110 mg/dL) using an insulin infusion. Intravenous mannitol in doses of 1 g/kg, then 50 g every 2 or 3 h, may forestall further deterioration, but most of these patients, once comatose, are likely to die unless drastic measures are taken. In such instances, controlled hyperventilation may be useful as a temporizing maneuver. Corticosteroids are

probably of little value; several trials have failed to demonstrate their efficacy. In the past several years there has been renewed interest in hemicraniectomy as a means of reducing the mass effect and intracranial pressure in these extreme circumstances.

The National Institute of Neurological Disorders and Stroke (NINDS) recombinant tPA (rtPA) Stroke Study showed a clear benefit for IV rtPA in selected patients with acute stroke. The NINDS study used IV rtPA (0.9 mg/kg to a 90-mg max; 10% as a bolus, then the remainder over 60 min) vs. placebo in patients with ischemic stroke within 3 h of onset. A recent study extended the acceptable interval for drug administration to 4.5 h.

Warfarin and heparin had been used extensively to prevent TIAs and reduce the chances of an impending stroke. These anticoagulants may halt the advance of a progressive thrombotic stroke, but they are clearly not effective in all cases. The two situations in which the immediate administration of heparin has drawn the most support from our own clinical practice are in fluctuating basilar artery thrombosis and in impending carotid artery occlusion from thrombosis or dissection. Aspirin (325 mg daily) has proved to be perhaps the most consistently useful drug in the prevention of thrombotic and possibly in embolic strokes. In patients who cannot tolerate aspirin, the platelet aggregate inhibitor clopidogrel or

a similar drug (such as ticlopidine or dipyridamole) can be substituted. The European Stroke Prevention Study (ESPS) II showed efficacy of both 50 mg/d of aspirin and extended-release dipyridamole in preventing stroke, and a significantly better risk reduction when the two agents were combined. Control of blood pressure, diabetes mellitus and the administration of a lipid-lowering drug are advisable, even if lipid levels are normal. In the most comprehensive study of statins to date, the institution of high doses of drug reduced the incidence of subsequent stroke after a TIA or first stroke by 2 percent over 5 years (Stroke Prevention by Aggressive Reduction in Cholesterol Investigators [SPARCL trial]).

Endovascular mechanical thrombectomy has recently shown promise as an alternative treatment of acute stroke in patients who are ineligible for, or have contraindications to, thrombolytics or in those who have failed to have vascular recanalization with IV thrombolytics.

The MERCI (Mechanical Embolus Removal in Cerebral Ischemia) single-arm trial investigated the ability of a novel endovascular thrombectomy device to restore patency of occluded intracranial vessels within 8 h of ischemic stroke symptoms[28].

There is growing evidence that intraparenchymal hemorrhage may be exacerbated by acutely elevated blood pressure, and current recommendations are to lower mean arterial blood pressure to <130 mmHg. Blood pressure should be lowered

with nonvasodilating IV drugs such as nicardipine, labetalol, or esmolol. For cerebellar hemorrhages, a neurosurgeon should be consulted immediately to assist with the evaluation; most cerebellar hematomas >3 cm in diameter will require surgical evacuation. If the patient is alert without focal brainstem signs and if the hematoma is <1 cm in diameter, surgical removal is usually unnecessary. Patients with hematomas between 1 and 3 cm require careful observation for signs of impaired consciousness and precipitous respiratory failure. Patients with cerebellar hemorrhages or with depressed mental status and radiographic evidence of hydrocephalus should undergo urgent neurosurgical evaluation. Stuporous or comatose patients generally are treated presumptively for elevated ICP, with tracheal intubation and hyperventilation, mannitol administration, and elevation of the head of the bed while surgical consultation is obtained.

Patient care in comprehensive stroke units followed by rehabilitation services improves neurologic outcomes and reduces mortality. Use of clinical pathways and staff dedicated to the stroke patient can improve care. Proper rehabilitation of the stroke patient includes early physical, occupational, and speech therapy. It is directed toward educating the patient and family about the patient's neurologic deficit, preventing the complications of immobility (e.g., pneumonia, DVT and pulmonary embolism, pressure sores of the skin, and muscle contractures), and providing encouragement and instruction in overcoming the deficit. The goal of

rehabilitation is to return the patient to home and to maximize recovery by providing a safe, progressive regimen suited to the individual patient. Additionally, the use of restraint therapy (immobilizing the unaffected side) has been shown to improve hemiparesis following stroke, even years following the stroke, suggesting that physical therapy can recruit unused neural pathways. This finding suggests that the human nervous system is more adaptable than originally thought and has stimulated active research into physical and pharmacologic strategies that can enhance long-term neural recovery.

Outcome measures of stroke

Stroke scales enable the exact measurement of severity, disability and recovery[29,30] of patients over a given time period. During a follow up stroke study, it is essential to have prescribed assessment methods. Ideal stroke scale should be valid, reliable and simple to administer. One of the first and previously most frequently used scales, the Mathew stroke scale, was first reported in 1972 in an evaluation of glycerol therapy[31]. Subsequently other scales like Toronto stroke scale, Scandinavian stroke scale, Canadian Neurological scale, Orgongozo scale, European stroke scale were used. National Institutes of Health Stroke Scale (NIHSS) was presented in 1989[32], and is based on several previous scales – the Toronto scale, Oxbury scale, Cincinnati stroke scale, Edinburgh-2 coma scale and

the Boston diagnostic aphasia examination. Over recent years, the NIHSS has been the instrument most widely used in clinical trials. It is used in acute phase of ischemic stroke to assess severity of stroke, during follow up to note the recovery of patients, particularly after intervention strategies and to predict outcome. It contains 13 items which assess consciousness level, conjugate gaze, visual fields, motor function in extremities, sensory function, facial palsy, language assessment and neglect are included. Maximal score is 42 and minimum is zero. For practical purpose score of 0 to5 indicates mild deficit, 6 to 15 denotes moderate deficit and score of more than 15 is suggestive of severe neurologic deficit. National Institutes of Health Stroke Scale (NIHSS) score was lower (less severe) among Posterior circulation stroke than Anterior circulation stroke patients. Recently, a modified version of this scale (mNIHSS) has been presented, in which items with poor reliability or redundance were excluded and the choices for the sensory item collapsed. So this version contains 11 items[33]. Although simpler, this has to be tested in a prospective fashion.

Modified Rankin Scale (MRS) presented in 1957 is a 5-point global assessment categorization of a patient's function based on the ability to perform ADL's[34]. The Rankin Scale is more oriented towards handicap, in terms of dependence on others, irrespective of the nature of the disability. It measures independence of a stroke patient and the scale points are from zero to five with score of five corresponding

to severe disability requiring constant care. Grade 6 is given for patients who have expired.

Barthel index (BI) is a widely used measure of functional outcome[35,36] .The Barthel Index is more oriented towards assessing the activities of daily living, and concentrates on motor handicaps, ignoring communication skills. It measures performance of a stroke patient on ten activities of daily living. These activities involve self-care and mobility. The score range is zero to 100. A patient having score of 100 is completely independent and fully functioning.

MATERIALS AND METHODS

This study was conducted during the period of January 2008 To December 2009.

All patients admitted with clinical features suggestive of stroke were taken. All were subjected to CT scan brain and MRI Scan brain with MRA. Patients with evidence of posterior circulation stroke in clinical features & imaging were taken up for the study.

Inclusion criteria

1. All patients with clinical features suggestive of brainstem stroke.
2. Adult patients in the age range of 21-80 years.
3. Evaluated within 7 days from the onset of last symptoms.
4. All patients should have CT Brain, MRI brain with MRA done within 7 days of stroke.
5. MRI brain showing infarcts and haemorrhages within the posterior circulation territory.
6. Patients willing to come for regular follow up visits for clinical outcome assessment.

Exclusion criteria

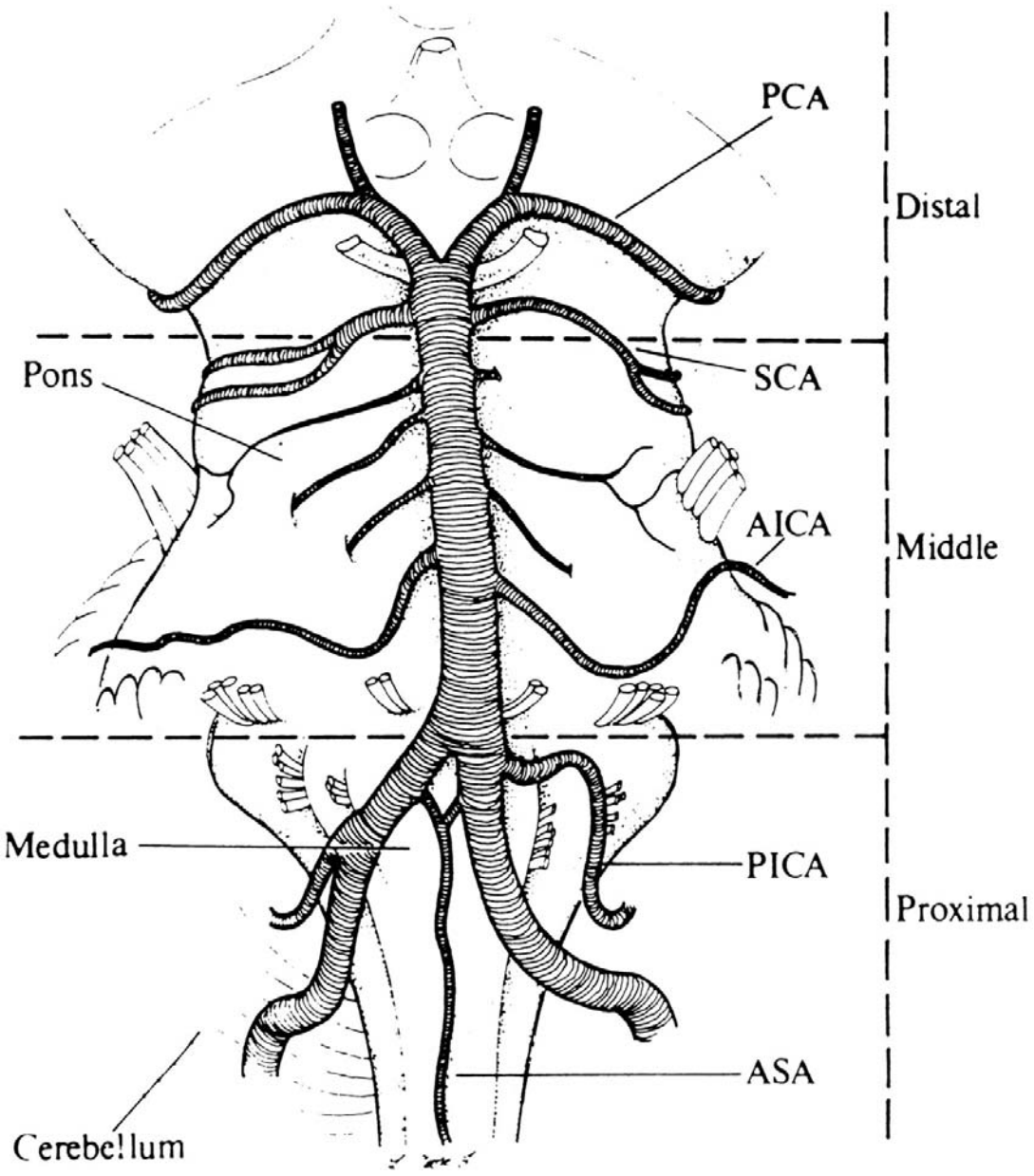
1. Patients of <21 or >80 years of age.
2. Patients seen after 7 days of onset of neurological symptoms.

3. Transient ischaemic attacks.
4. Patients with severe metabolic disturbances complicating the stroke.
5. Patients having evidence of infarcts in other areas i.e., territory of anterior circulation, border zone infarcts, venous infarcts, head injury, tumors..
6. Patients unable to come for follow up periodically at stipulated time.

Patient's details regarding age, sex, risk factors like hypertension, diabetes mellitus, atrial fibrillation, ischemic heart disease, hypercholesterolemia, smoking, past H/O TIA were recorded. The onset of symptoms and signs were recorded.

CT Brain, MRI brain with MRA and DWI was done in all patients.

We subdivided the posterior circulation territories into regions – proximal, middle and distal[9]. The intracranial vertebral artery joins at the ponto-medullary junction to form the basilar artery. The territory supplied by intra cranial vertebral arteries includes the medulla and cerebellum supplied by the posterior inferior cerebellar artery. This region is designated as proximal intracranial posterior circulation territory.The basilar artery bifurcates at the ponto- mesencephalic junction. The territory supplied by basilar artery including the pons and portion of the cerebellum supplied by AICA branches is designated as middle intracranial posterior circulation territory.



The portion of the posterior circulation supplied by the distal basilar artery, superior cerebellar artery, posterior cerebral artery and its penetrating branches is referred to as distal intracranial posterior circulation territory. The distal territory includes the mid brain, thalamus, SCA supplied cerebellum, occipital and temporal lobe regions.

OBSERVATION AND RESULTS

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

Age and sex distribution

Among these 150 patients there were 110 males (73.33%) and 40 females (26.67%).

The age group distributions were shown in table 1. The age group ranges from 21 years to 80 years. The maximum numbers of cases were in the age group of 51-60 years in both sexes.

Table 1

Age group in years	**Male n=110**	**Female n=40**
21 -30	3(2.73%)	-
31-40	11(10%)	2(5%)
41-50	21(19.09%)	5(12.5%)
51-60	31(28.18%)	14(35%)
61-70	28(25.45%)	10(25%)
71-80	16(14.55%)	9(22.5%)

Risk factors

The possible risk factors were studied in all 150 patients, about 72.67% had hypertension and around 41.33% gave history of chronic smoking. The risk factors identified is shown in Table 2.

Table 2

Risk factor	Number of patients (n=150)
1. Hypertension	109 (72.67%)
2. Smoking	62 (41.33%)
3. Diabetes mellitus	68 (45.33%)
4. Ischemic heart disease	22 (14.67%)
5. Hypercholestrolemia	75 (50%)
6. Rheumatic Heart Disease	2 (1.33%)
7. Chieropraxis	4 (2.67%)
8. Embolism from heart	23 (15.33%)

Clinical features

The clinical features at the onset of stroke were studied. Most of our patients presented with giddiness and vomiting. Headache was present in about 40 patients. 27 patients had altered sensorium at onset. 16 patients had seizures at

the onset, among which 10 had generalized tonic clonic seizures and 6 had partial seizures with secondary generalization. The clinical features at the onset were shown in table 3.

Table 3

S. No.	Clinical features	Number of patients
1.	Giddiness & Vomiting	110
2.	Headache	40
3.	Seizures	
a.	GTCS	10
b.	Partial seizures	6
4.	Altered sensorium	27

The other clinical features that were present in this study were according to the territory involved. This included homonymous hemianopia, temporal lobe signs, cerebellar signs, weakness, sensory disturbances, cranial nerve disturbances (III,

IV, V, VI, VII, IX and X). Most of these were combination of clinical features. This was shown in table 4.

Table 4

Clinical features	Number of patients(n=150)
Visual field defects	45 (30%)
Temporal Lobe Signs	16 (10.67%)
Cerebellar Signs	98 (65.33%)
Weakness	78 (52%)
Hemi sensory loss	40 (26.67%)
Cranial nerve involvement	45 (30%)
Combination of clinical features	93 (62%)

Past H/O TIA's were seen in only 42 patients, past H/O stroke is seen in 12 patients.

To describe the location of infarcts, we subdivided the posterior circulation in to proximal, middle and distal intra cranial arteries accordingly described by NEMC posterior circulation registry.

The clinical features and neuroimaging were taken together to describe the location of infarct.

Neuro Imaging

CT scan brain plain was done in all patients. 30 patients had haemorrhages and 82 patients had infarcts in CT Brain. CT brain was normal in 38 patients in our study. All the 38 patients had infarcts in MRI Brain.

MRI brain with MRA was done in 150 patients. 30 patients had haemorrhages and 120 patients had infarcts in MRI Brain.

In MRI Brain 52 patients had infarcts in thalamus, midbrain, temporal or occipital lobes, 14 patients had infarcts in pons, 9 patients had infarcts in cerebellum or medulla. 14 patients had infarcts in both temporal or occipital lobes and pons. 14 patients had infarcts in midbrain, thalamus and cerebellum. 5 patients had infarcts in pons and cerebellum. 12 patients had infarcts in cerebellum, occipital lobes, pons and thalamus.

In MRI with MRA isolated basilar artery thrombosis was seen in 7 patients and isolated posterior cerebral artery thrombosis was seen 15 patients. One patient had vertebral artery dissection. 4 patients had thrombosis in vertebral artery. 2 patients had thrombosis in vertebral, basilar and posterior cerebral arteries. 2 patients had thrombosis in vertebral and basilar arteries. 3 patients had basilar and posterior cerebral artery thrombosis. 5 patients had thrombosis in vertebral and posterior cerebral arteries. One patient had isolated PICA thrombosis.

In MRI Brain 9 patients had pontine haemorrhages, 4 patients had occipital lobe haemorrhages, 5 patients had thalamic haemorrhages, 3 patients had medullary haemorrhages, 2 patients had cerebellar haemorrhages, and 2 patients had haemorrhages in the midbrain. 2 patients had haemorrhages in the pons and occipital lobe. 2 patients had haemorrhages in the pons and cerebellum. 1 patient had haemorrhages in the medulla and cerebellum.

Among these 150 patients, we found that distal territory involvement was more common. Isolated middle and proximal territory infarcts were less in this study. In other patients we had varying combinations of proximal, middle and distal territory infarcts.

The location of territory was shown in table 5.

Table 5

No.	Location of Infarctions	Number of patients	
		Infarcts	**Haemorrhages**
1.	Distal only	52 (43.33%)	12 (40%)
2.	Proximal only	9 (7.5%)	4 (3.33%)
3.	Middle only	14 (11.67%)	9 (7.5%)
4.	Proximal + middle	5 (4.17%)	3 (2.5%)
5.	Proximal + distal	14 (11.67%)	-
6.	Middle + distal	14 (11.67%)	2 (1.67%)
7.	Proximal + middle +distal	12 (10%)	-

Prognosis was assessed using Modified Rankin Scale and Barthel Index. 20 patients with infarcts and 10 patients with haemorrhages died within one week. Modified rankin scale and barthel index was assessed at discharge and at one month. 60 patients with infarcts and 5 patients with haemorrhages were in MRS 0-2, 25 patients with infarcts and 5 patients with haemorrhages were in MRS 3-4, 15 patients with infarcts and 10 patients with haemorrhages were in MRS 5 at discharge.

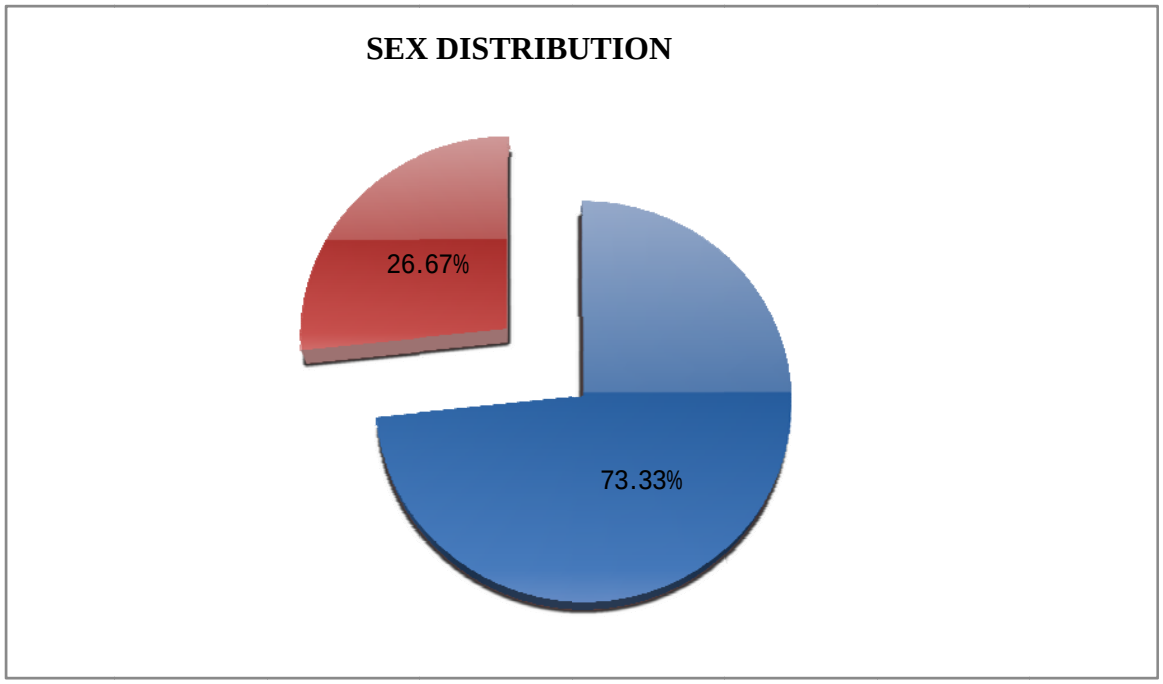
Table 6

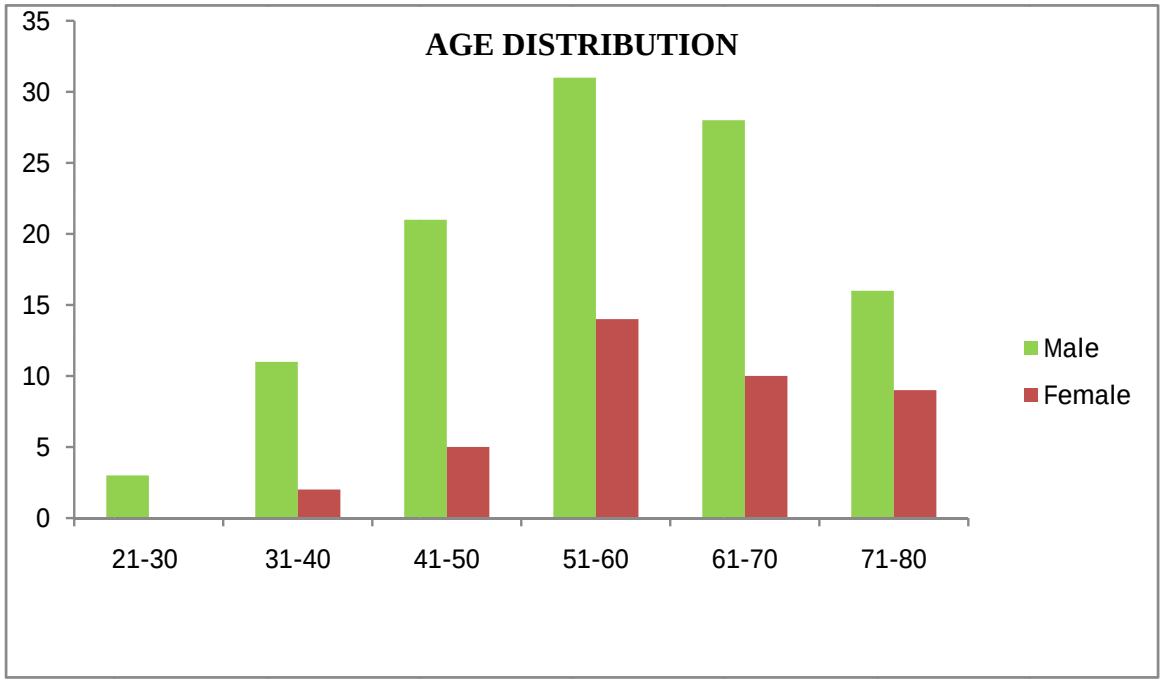
MRS	Infarcts		Haemorrhages	
	MRS at discharge	MRS at 1 month	MRS at discharge	MRS at 1 month
0-2	60	70	5	7
3-4	25	20	5	4
5	15	10	10	9

Table 7

BI	Infarcts		Haemorrhages	
	BI at discharge	BI at 1 month	BI at discharge	BI at 1 month
0-50	40	35	10	7
55-90	45	50	8	11
95-100	15	15	2	2

40 patients with infarcts and 10 patients with haemorrhages were in BI 0-50 (full dependency), 45 patients with infarcts and 8 patients with haemorrhages were in BI 55-90 (moderate dependency), 15 patients with infarcts and 2 patients with haemorrhages were in BI 95-100 (functional independance) at discharge.


SEX DISTRIBUTION
26.67%
73.33%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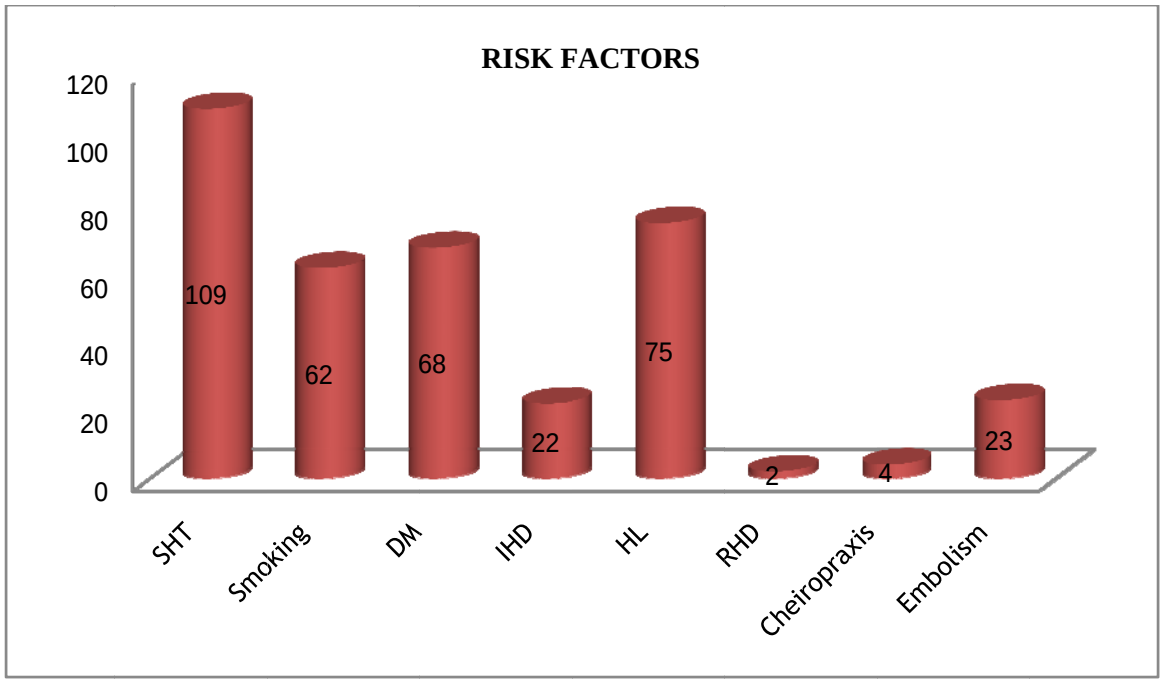
Female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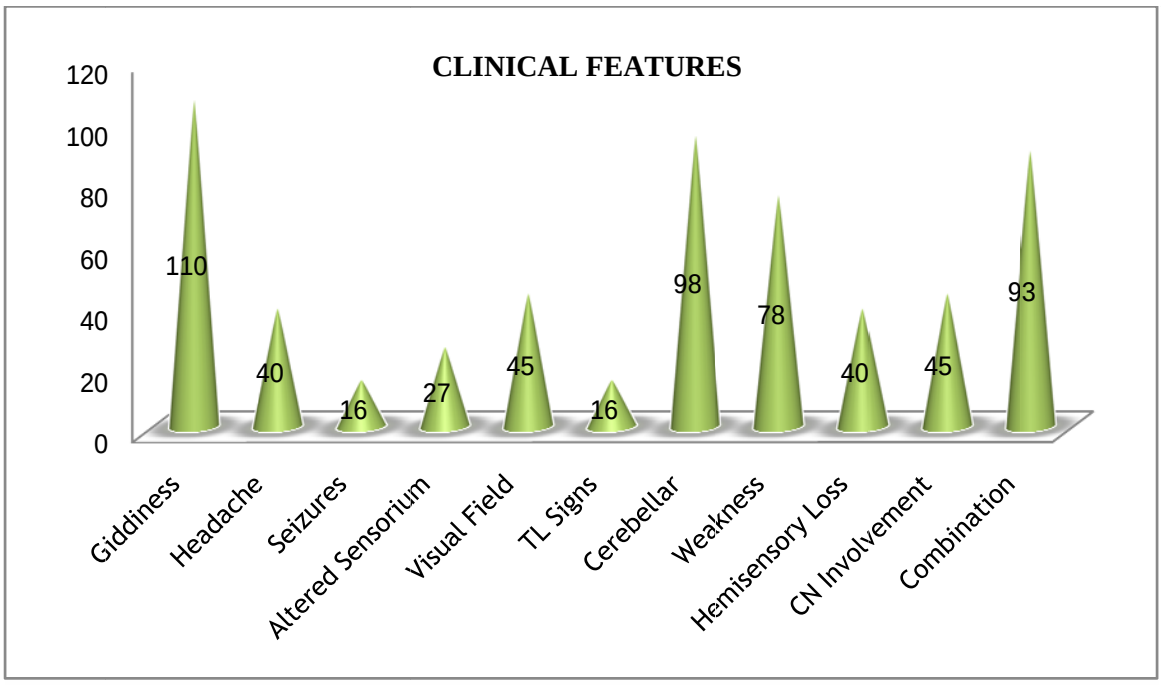
DM
IHD
HL
RHD
Cheiropraxis
Embolism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16
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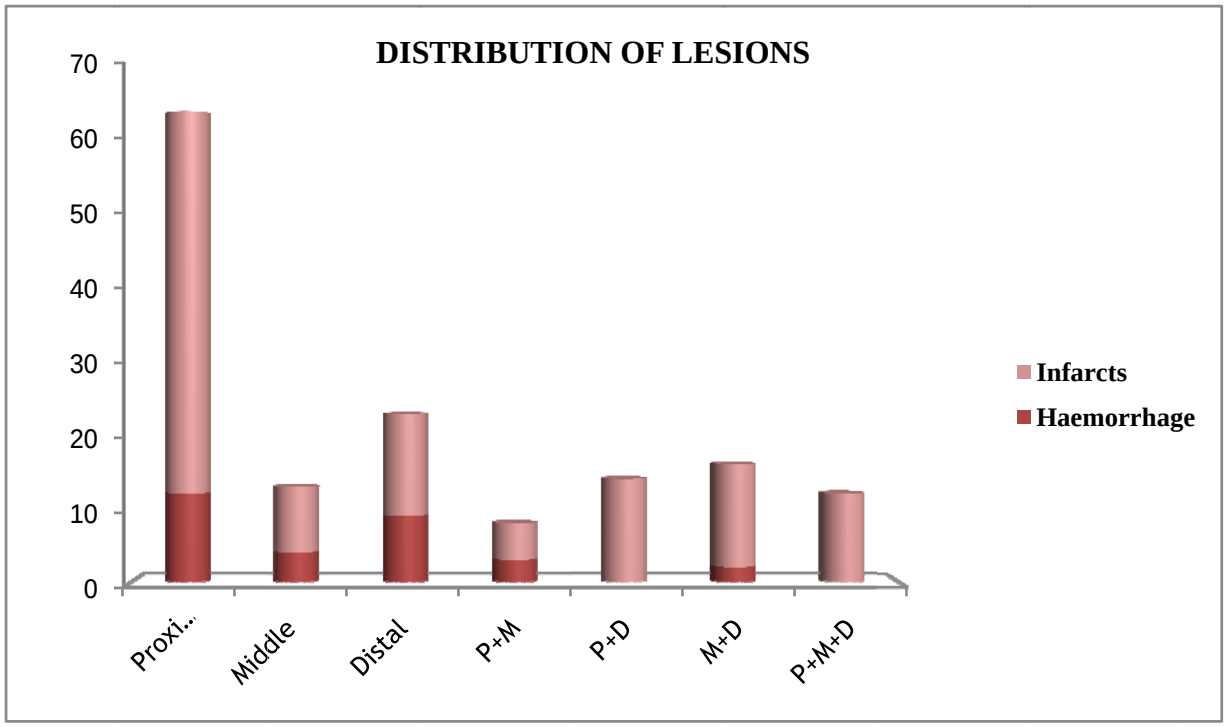
Headache
Seizures
Altered Sensorium
Visual Field
TL Signs
Cerebellar
Weakness
Hemisensory Loss
CN Involvement
Combination


DISTRIBUTION OF LESIONS
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60
50
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30
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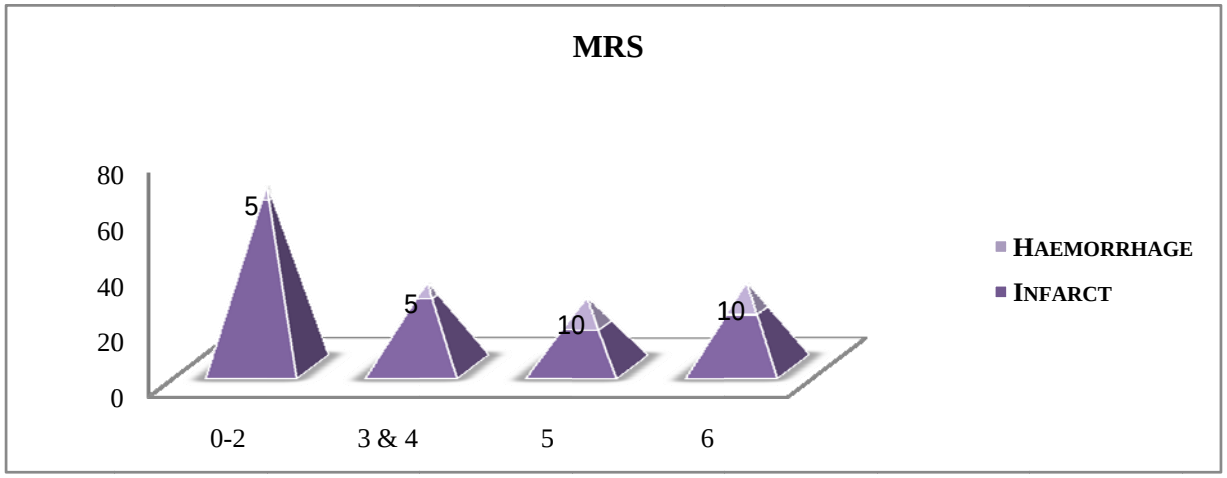
Middle
Distal
P+M
P+D
M+D
P+M+D
Infarcts
Haemorrhage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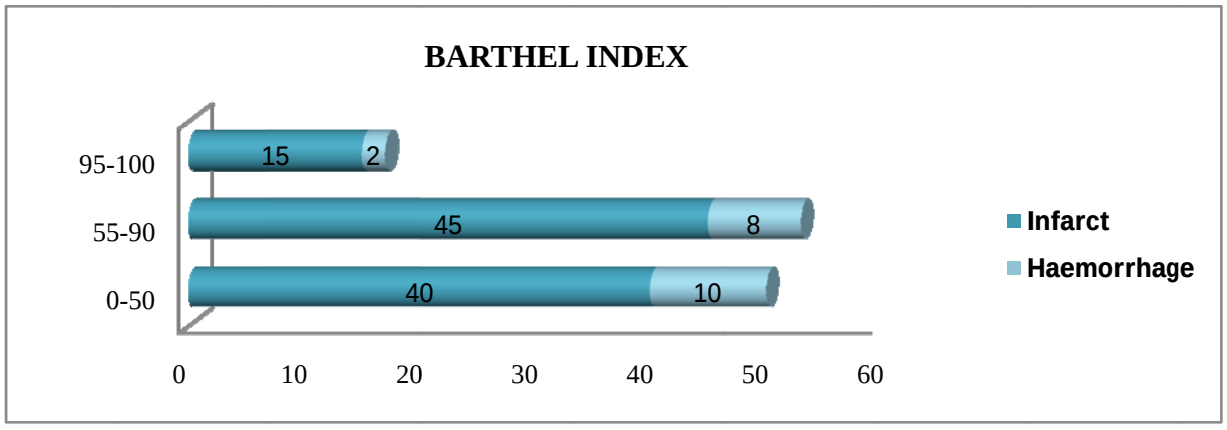
10
10
0-2
3 & 4
5
6
HAEMORRHAGE
INFARCT


BARTHEL INDEX
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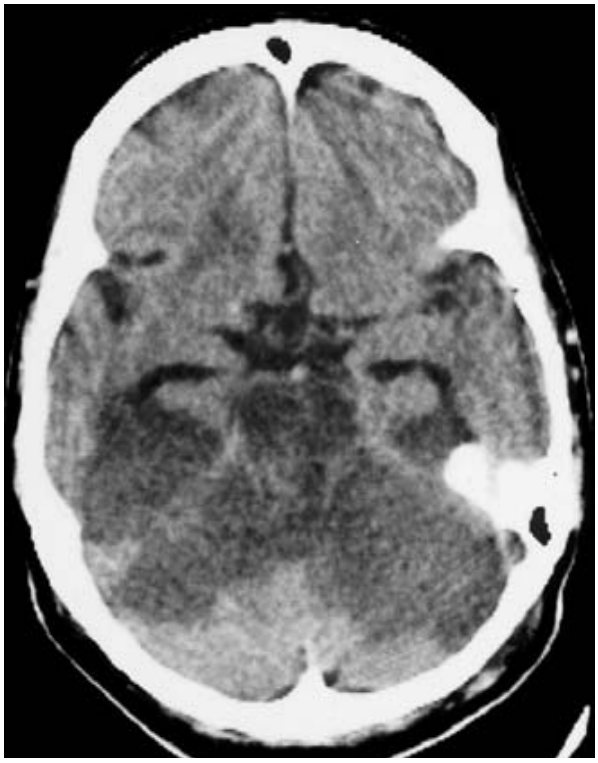
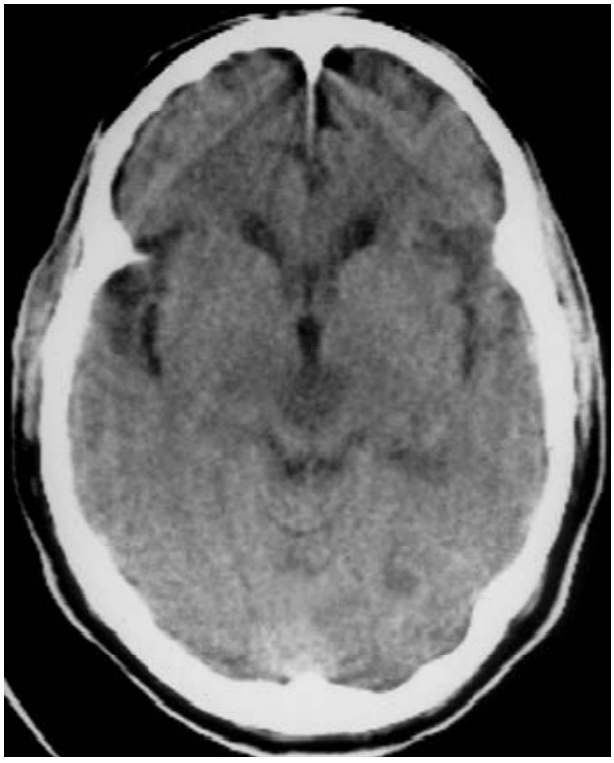
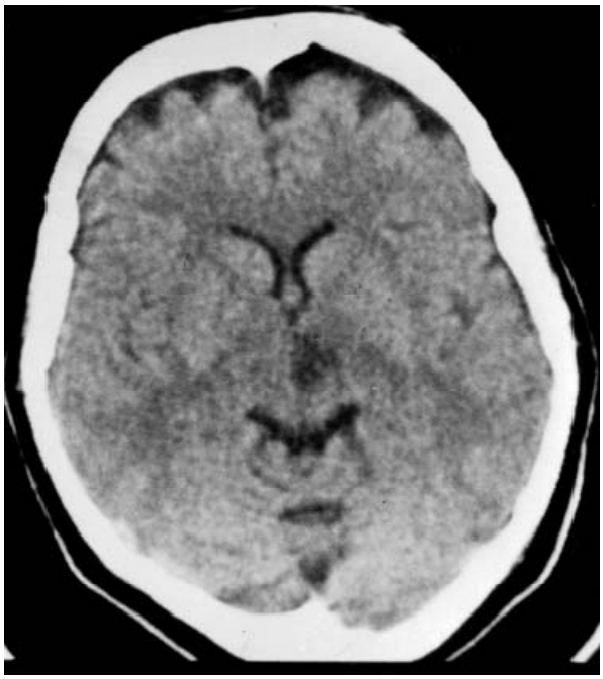
20
30
40
50
60
Infarct
Haemorrhage

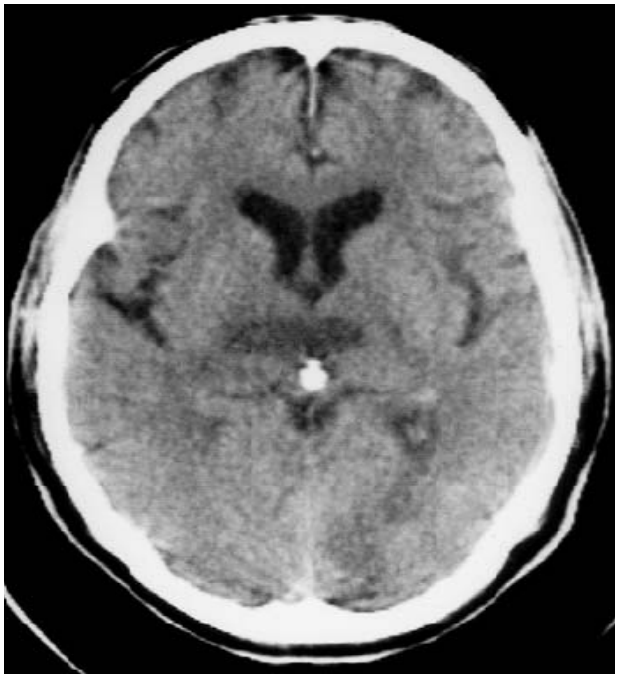


Infarct in brainstem, and temporal lobes with thrombosis of the basilar stem

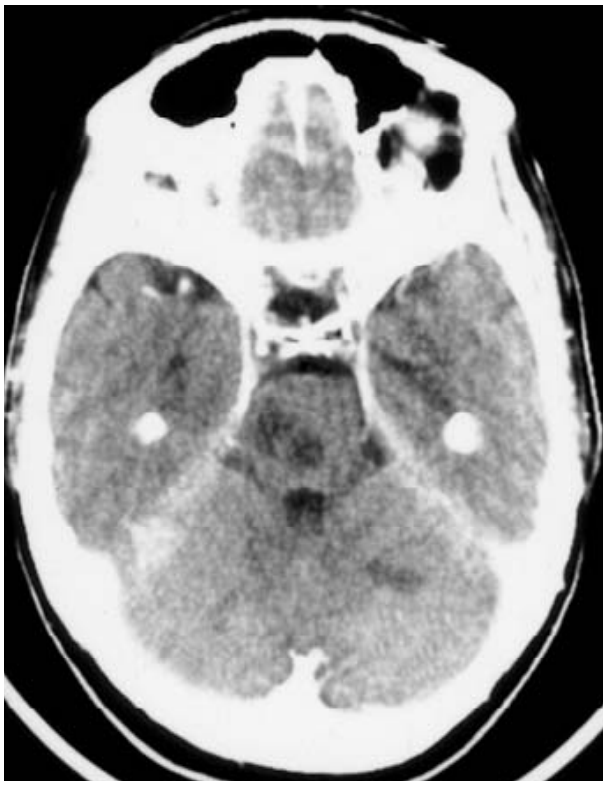


Midbrain infarct

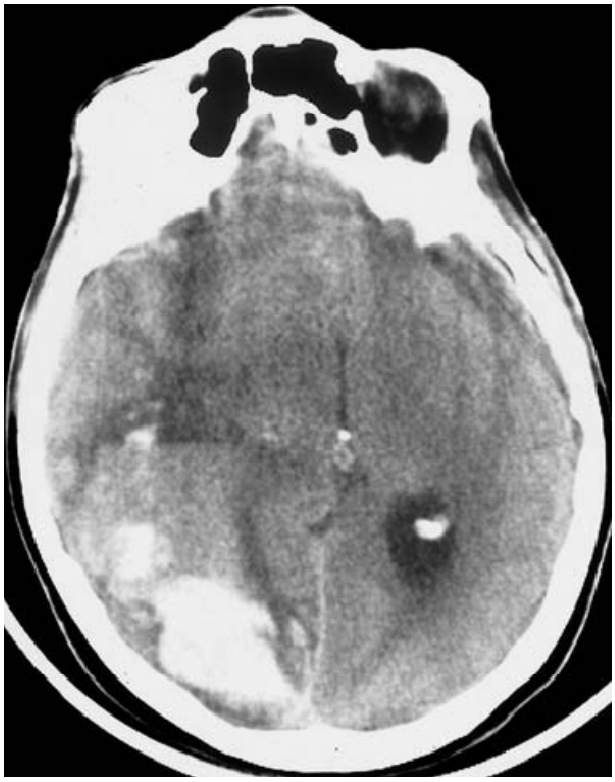




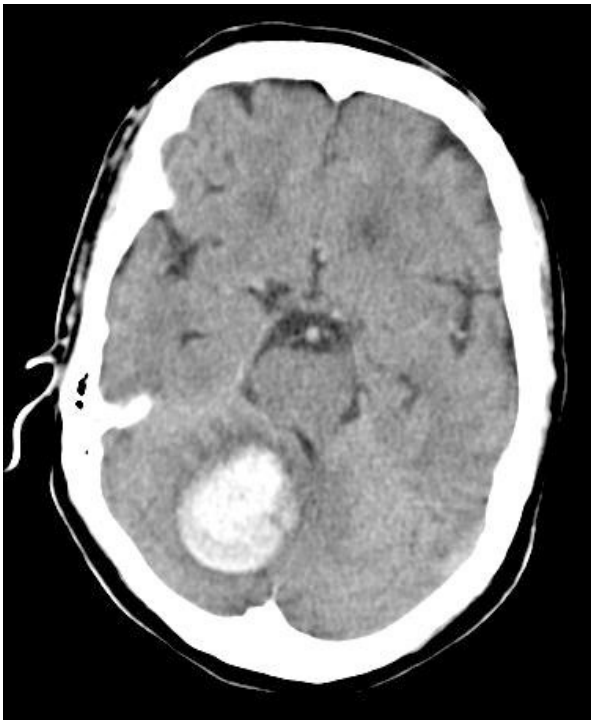
Thalamic infarcts: on the left and bilaterally



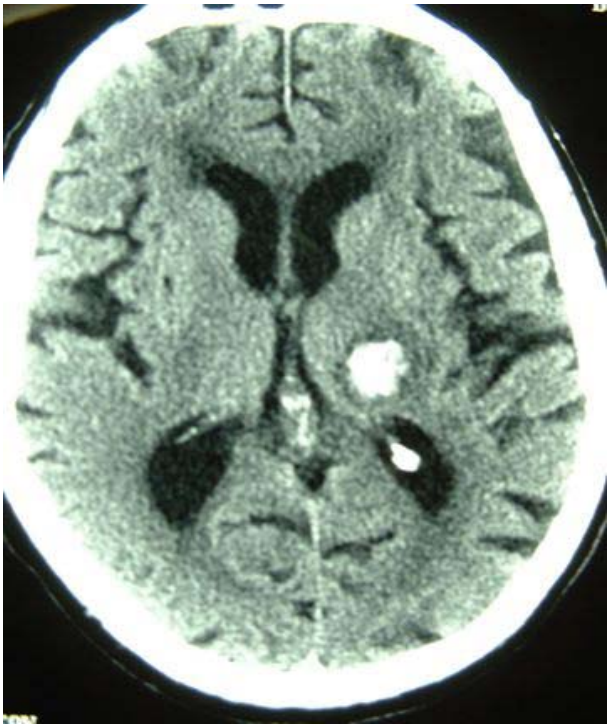
Infarcts in the pons & left cerebellar hemisphere



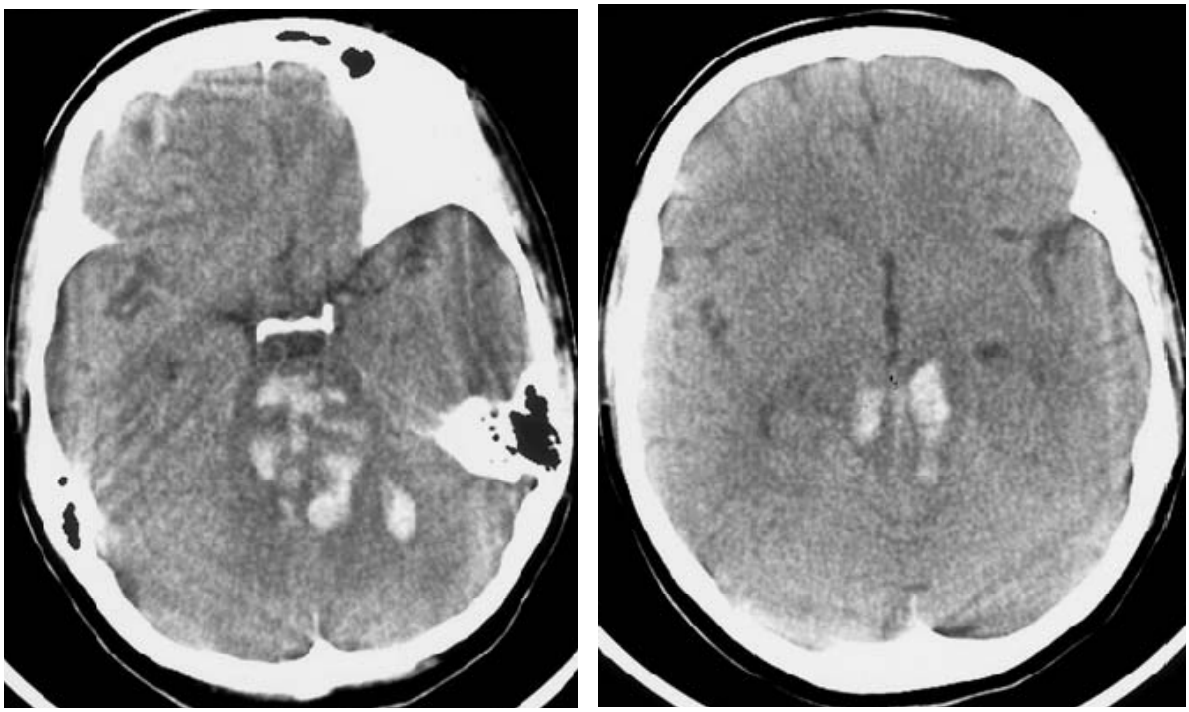
CT Brain showing left occipital lobe haemorrhage



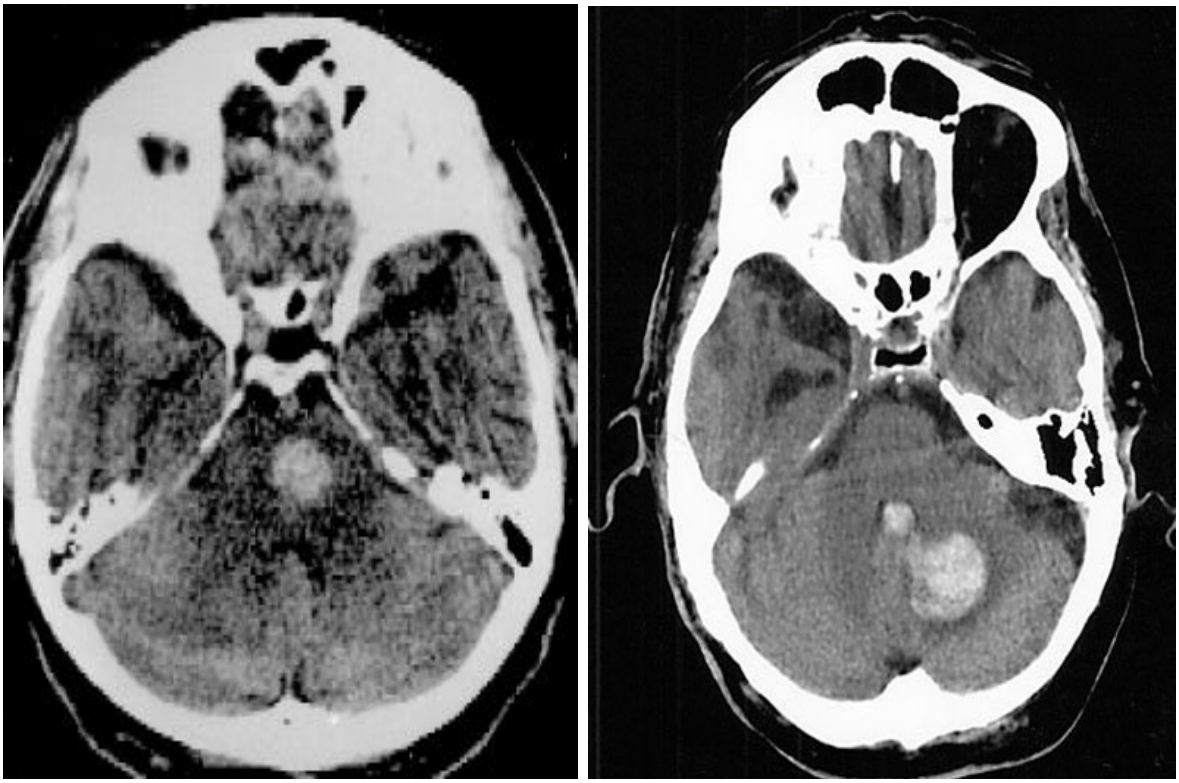
CT Brain showing right cerebellar haemorrhage



CT Brain showing right thalamic haemorrhage

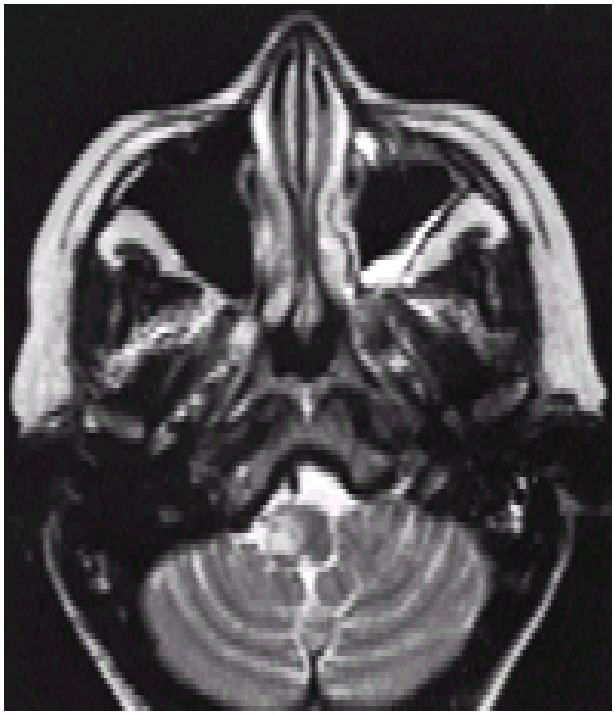


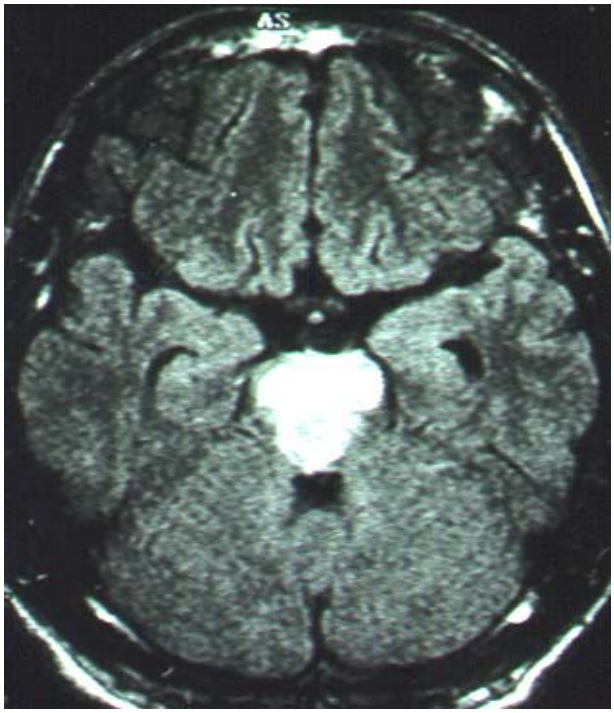
A large haematoma with a fragmented appearance massively occupies the brainstem and spreads upwards towards the thalamic nuclei



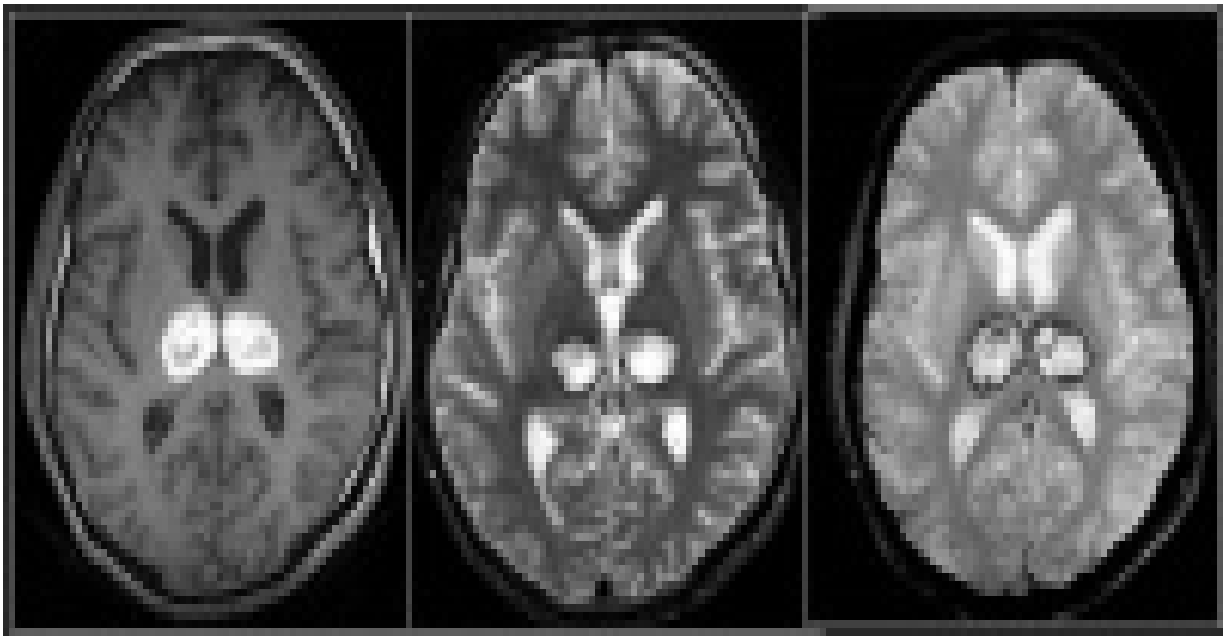
CT Brain showing pontine haemorrhage

CT Brain showing cerebellar haemorrhage

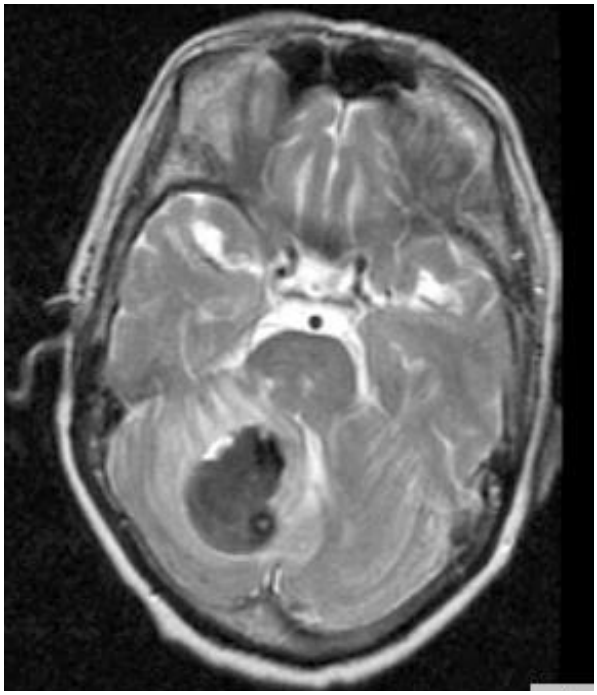




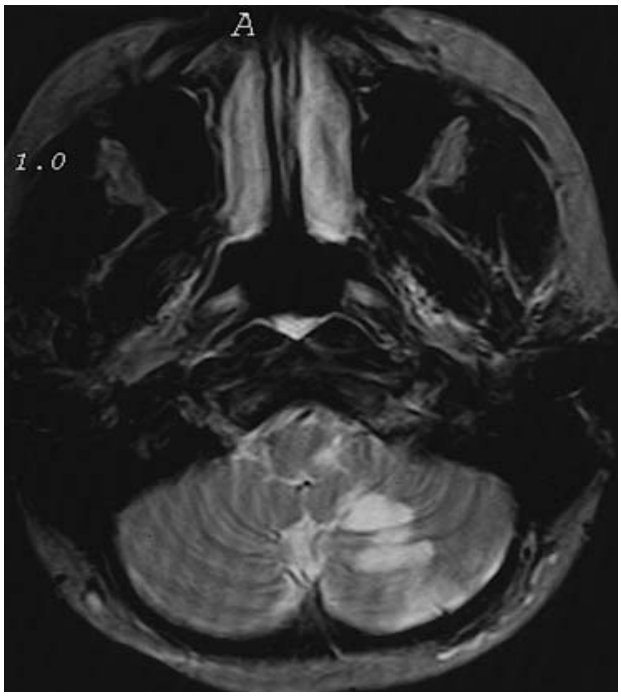
T2 images with right medullary infarct **FLAIR images showing pontine infarcts**



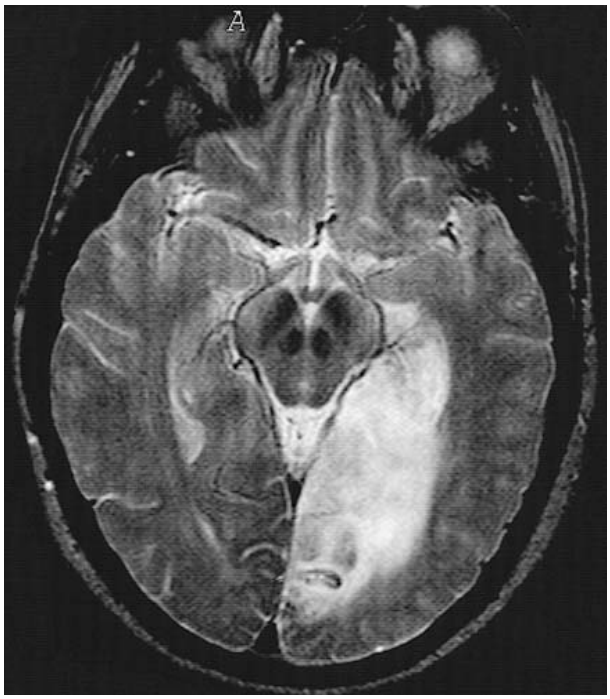
MR images show late subacute hemorrhage in both thalamic regions T1-weighted, T2-weighted, and gradient-echo (GRE) images all show a hyperintense hematoma.



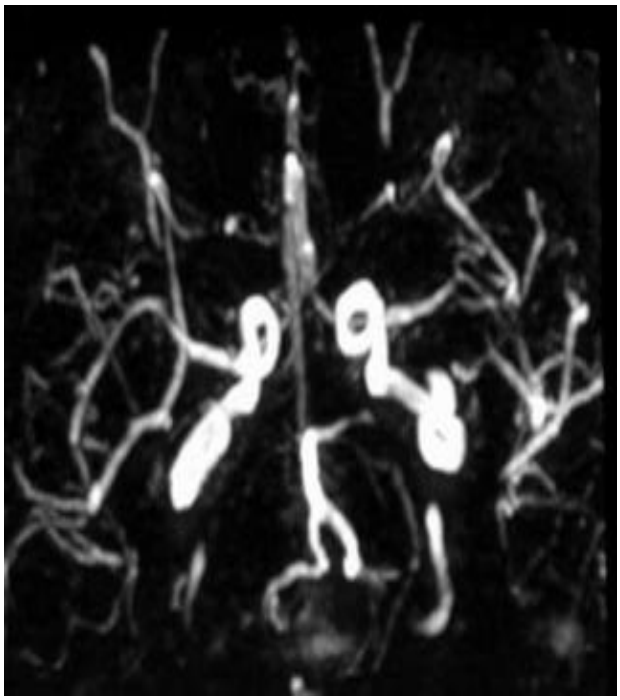
T2MR scan: right cerebellar haemorrhage



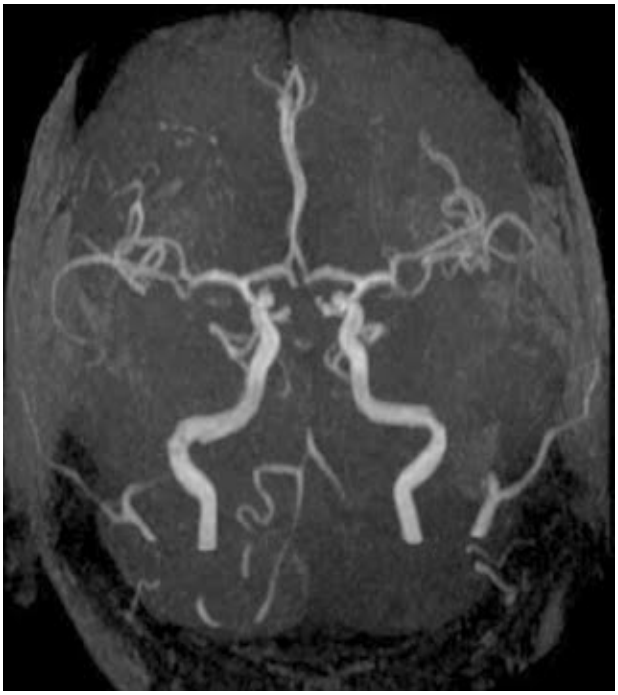
T2MR scan: left cerebellum ischaemic lesion

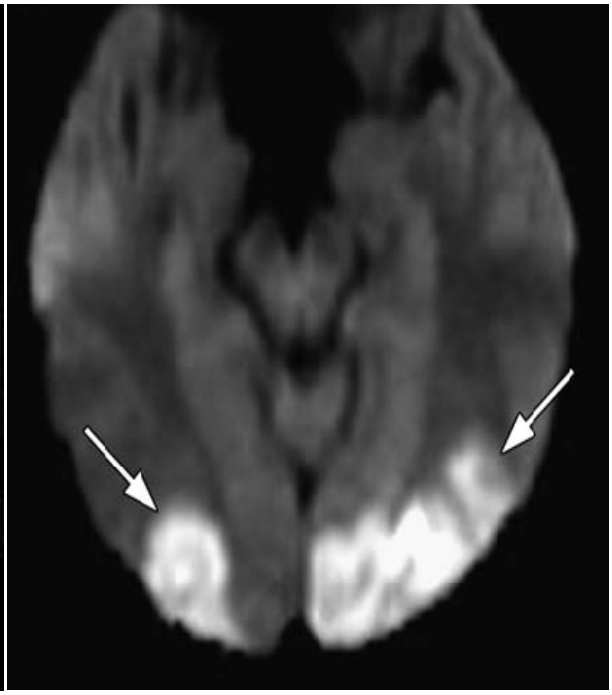


T2 MRI showing left temporooccipital ischaemia

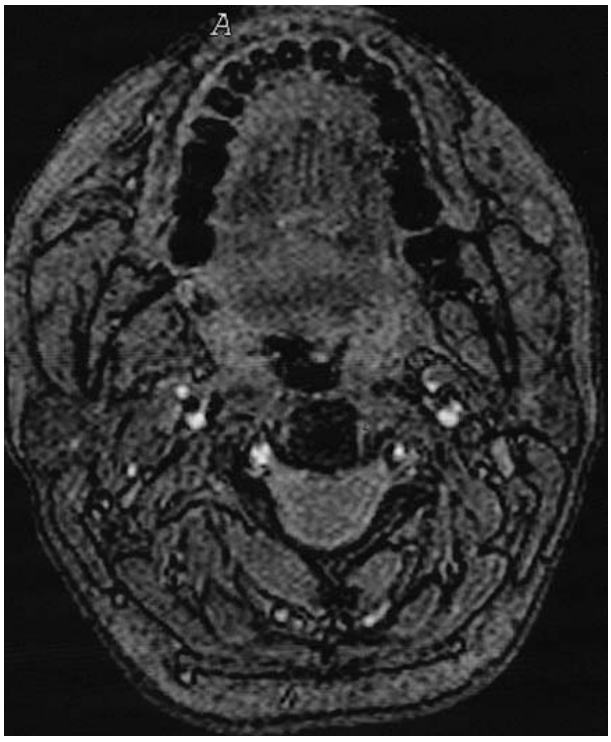


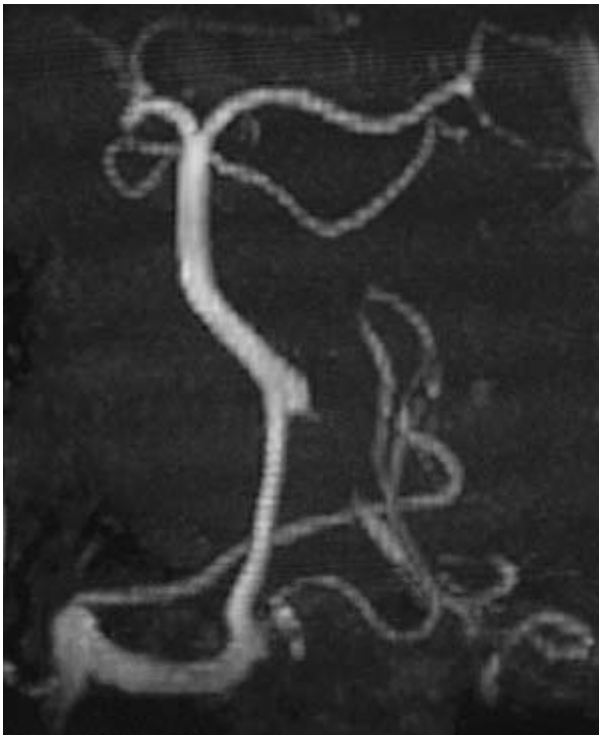
Right PCA thrombosis





MRA: Left VA, PICA & basilar artery thrombosis **DWI showing bilateral occipital hyperintensities**





Dissection of the left vertebral artery.with presence of hypointense parietal thrombus, in acute phase, at the vertebral artery;MRA: absence of signal in the last stretch of the left vertebral artery

DISCUSSION

Cerebrovascular disease was more common in men in the age group of 51-60 years. In `Tuffs Posterior Circulation Registry` 52% were male and 48% were females. Smajlonc D[37] studied ischemic insult in both anterior and posterior circulations and he found 18.2% had posterior circulation stroke and he also found females and males were equally affected (50%) in posterior circulation stroke. In this study, 73.33% were males and only 26.67% were females. Though the mentioned studies show sexual equality, our study showed a male preponderance.

Mean age of patients in our study was above 50 years, which is similar to other studies. As it is known that stroke risk doubles with each decade past age 55 years.

In the study by Uma Sundar et al[38], majority of patients in the PCS group were in the age group 40-55 years. This compares with previous studies from the Asian sub continent, but shows a distinctly younger group as compared to the NEMC registry. Tufts New England Medical Center posterior circulation stroke registry, the mean age of stroke was 61.5 years.

In our study 120 (80%) patients had ischaemic stroke and 30 (20%) patients had haemorrhagic stroke. In our study 23 (15.33%) patients had a cardioembolic source. In the study by Uma Sundar et al[38] among 76 serially recruited PCS, 77.6 %(59/76) were ischemic, 22% (17/76) being hemorrhagic. Among 77.6% of PCS

strokes a cardioembolic source was seen in 12/29 (42%) PCS cases and intraarterial cause was seen in 5/29 (17.2%).

The risk factors in stroke are classified as modifiable and non modifiable. Male sex & older age are non-modifiable risk factors. Hypertension, diabetes mellitus, hypercholesterolemia, obesity, chronic smoking, ischemic heart disease, atrial fibrillation, rheumatic heart diseases are other modifiable risk factors.

In this study, hypertension (72.67%) was the most common risk factor. Chronic smoking was found in 41.33% patients, diabetes mellitus in 45.33% and hypercholesterolemia in 50% of patients. Embolism from heart was seen in 15.33%. Huan et al [39] found that 71% of his patients had hypertension and chronic smoking was seen in 38% of patients. The comparison of risk factors between their study and our study is shown in table below.

Risk factors	**Present Study (2009)**	**Huan et al (2002)**
Hypertension	72.67%	71%
Chronic Smoking	41.33%	38.7%
Diabetes Mellitus	45.33%	22.6%
Ischemic Heart Disease	14.67%	19.4%

In the study by Subramaniam et al[40], on multivariable analysis, diabetes mellitus was associated with an increased odds of posterior circulation ischemic stroke.

Clinical features at the onset of stroke in our study were giddiness and vomiting in 110 patients. The other presentations in our study included headache in 26.67% of patients, altered sensorium in 34.67% of patients and seizures in 10.67% of patients. Seizures were of generalized tonic clonic type in 62.5% of patients and 37.5% of patients had partial motor seizures with secondary generalization.

Timothy et al[41] – had described vertigo without hearing loss at the commonest symptom in brainstem stroke syndromes. Vertigo occurs both in small vessel disease and large vessel disease. Huan et al[39] had found 30% of his patients had vertigo.

In this study, vertiginous onset was present in 73.33% patients with distal territory infarct and also in all patients with two or more territories involvement. Of the persons with multiple territory involvement if middle territory involvement is also there, vertiginous onset was more common. Only in 25% of patients with isolated proximal territory infarct vertigo at onset was present. We found that most of the patients who had middle territory infarcts (Pons & Anterior inferior cerebellar artery supplied cerebellum) had vertigo at onset. The vertigo in middle territory

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

Altered sensorium at the onset is seen in 18% of patients in this study. Huan et al[39] had found only 6% had altered sensorium. In this study among the 27 patients with altered sensorium at the onset, distal territory involvement was noted in 9 out of 27 (33.33%) patients, and middle territory involvement in 8 out of 27 (29.63%) patients. Patients who had proximal territory infarct did not have altered sensorium.

Seizures were seen in 10.67% of patients in this study. Out of which 62.5% (10) had generalized tonic clonic seizures and 37.5 % (6) had partial seizures with secondary generalization.

The seizures were observed in 10 patients with distal territory infarcts and in 4 patients with middle and distal territory infarcts and 2 patients with proximal and distal territory infarcts. Occurrence of seizures could probably be explained by involvement of temporal cortex due to PCA involvement.

In this study 52% of patients had pyramidal and 65.33% of patients had cerebellar signs. Visual field defects were seen in 30% of patients, 30% of patients had cranial nerves involvement. 26.67% of patients had hemisensory loss & 10.67% of

patients had temporal lobar signs. Huan et al[39] in his study had described pyramidal signs in 58% and cerebellar signs in 51%.

Past H/O TIA's were noted in 22 patients (14.67%). Whereas Huan et al[39] have found 50% had TIA preceding stroke. In this study the H/O TIA is less compared to Huans. Among 407 New England Medical Center Posterior Circulation registry[9] patients, 59% had strokes without transient ischemic attacks (TIAs), 24% had TIAs then strokes, and 16% had only TIAs.

CT brain was normal in 38 patients in our study. 30 patients had haemorrhages and 82 patients had infarcts in CT Brain.

MRI brain with MRA was done in all patients. 30 patients had haemorrhages and 120 patients had infarcts in MRI Brain.

Regarding the vascular territory involvement in this study we have found that 43.33% of patients with infarcts and 40% of patients with haemorrhages had isolated distal territory involvement. The other territory involvements and multiple territory involvement were less.

In New England Medical Center Posterior Circulation Registry[9], they also found that distal territory involvement was more common – 40.9%. Study by Uma Sundar et al[38] showed a predominance of distal intracranial (46%), followed by proximal intracranial (34%) vascular distribution in the PCS.

The comparison data of our study & NEMC posterior circulation registry is shown in table below.

No.	Location of Infarctions	Present Study	NEMC-Registry
1.	Distal only	42.67%	40.9%
2.	Proximal only	8.67%	18.2%
3.	Middle only	15.33%	16.1%
4.	Proximal + middle	5.33%	3.45%
5.	Proximal + distal	9.33%	8.93%
6.	Middle + distal	10.67%	2.59%
7.	Proximal + middle +distal	8%	9.79%

Thrombosis within the posterior circulation is seen in 39 patients among whom 7 had isolated thrombosis of basilar artery, 15 had isolated PCA artery thrombosis, 1 had PICA thrombosis and 2 had vertebral, basilar artery and PCA thrombosis and one had post traumatic vertebral artery dissection. (following neck manipulation).

Among 407 New England Medical Center Posterior Circulation registry[9] patients, isolated basilar artery stenosis was seen in 44.8%; 41.4% had basilar artery involvement as part of wide spread posterior circulation atherosclerosis and

embolism to the basilar artery in 13.8%. 62% patients had involvement of the midportion of basilar artery, 66% had H/O multiple TIA's and infarcts were common in middle territory. Severe occlusive lesions (>50% stenosis) involved more than one large artery in 148 patients; 134 had one artery site involved unilaterally or bilaterally. The commonest occlusive sites were: extracranial vertebral artery (52 patients, 15 bilateral) intracranial vertebral artery (40 patients, 12 bilateral), basilar artery (46 patients). In the study conducted by Bogousslavsky[42] et al, 27 patients (39%) had 50% stenosis or occlusion of the basilar artery and there were other large-artery lesions in 19 patients (27%), including vertebral (V_2-V_4) stenosis or occlusion (in seven).

15 patients who had PCA lesion in isolation had distal territory infarct.

One patient who had dissection of vertebral artery due to cheiropraxis[43], the proximal territory was involved.

Embolism from cardiac origin could be identified in 23 patients in our study (15.33%). In NEMC Posterior circulation registry the embolism from cardiac origin were 24%.

In our study 20(16.67%) patients with infarcts and 10(33.33%) patients with haemorrhages died within thirty days. Most of these patients had multiple territory involvement.

In NEMC Posterior circulation registry patients' thirty-day mortality was 3.6%. Embolic mechanism, distal territory location, and basilar artery occlusive disease carried the poorest prognosis.

In the study by Uma Sundar et al mortality was 14/76 (18%).Among 14 patients who died 6 patients had haemorrhages on imaging studies. The principal contributory factors to mortality in PCS were low Glasgow coma score at presentation, development of respiratory morbidity, and vascular lesions in 'middle plus distal' territory. In the study by Jones et al[44] mortality at the conclusion of the first week was 27% for the entire group.

Bougusslavsky's[45] study on unselected cases of PCS detected a mortality rate of 40%, whereas that of Hennessey, Pazdera et al[46] (from the multi-centric PCS registry), found a mortality rate of 3.6% at 1 month with an encouraging disability status (28% with no disability and 51% with minor disability on MRS), at 1 month poststroke.

None of our patients could be thrombolysed because of economic reasons and so the mortality percentage is slightly higher. In a study by Lindsberg et al[47] basilar artery occlusion was thrombolysed and recanalization was studied in 43 patients and verified in 26 (52%) of all patients. By 3 months, 20 patients (40%) had died while 11 had good outcomes (modified Rankin Scale score, 0-2); 12 (24%) reached

independence in activities of daily living (Barthel Index score, 95-100), and 6 (16%) were severely disabled (Barthel Index score, 0-50).

In our study 65 (43.33%) patients had MRS 0-2, 30 (20%) had MRS 3-4, 25 (16.67%) had MRS 5 and 30 (20%) had MRS 6 at discharge. In the study by Uma Sundar et al at discharge, 62% PCS patients were in group 2-3 of modified Rankin scale.

SUMMARY

In our study, we have found that

- Males were affected more than females.
- Age group commonly involved was 51-60 years.
- Hypertension, diabetes mellitus and chronic smoking are the major risk factors in posterior-circulation stroke.
- Giddiness was the commonest symptom at the onset.
- More than half of patients had clinical features of either pyramidal signs and/or cerebellar signs
- The territory of infarct commonly involved was distal territory which includes midbrain, thalamus, occipital & temporal lobes and SCA supplied cerebellum.
- Posterior cerebral artery was the commonest artery to get thrombosed.
- At discharge about 50% patients had slight disability as assessed by Modified Rankin Scale
- At discharge about 50% patients had moderate dependence as assessed by Barthel Index.

CONCLUSION

All patients with brain ischemia deserve full evaluation of their brain for vascular lesions. With the advent of newer techniques, MRI with DW imaging, MRA, extra cranial and transcranial doppler studies it is possible to investigate the brain and stroke mechanisms quickly and noninvasively.

Cardiac investigations are just as important in patients with posterior circulation ischemia, because a considerable number of posterior circulation infarcts are cardio embolic.

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