“CLINICAL PROFILE OF PATIENTS WITH BRAINSTEM STROKE ADMITTED TO KLES DR PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE – A CROSS SECTIONAL STUDY”

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This is to certify that the dissertation entitled **"CLINICAL PROFILE OF PATIENTS WITH BRAINSTEM STROKE ADMITTED TO KLES DR PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE – A CROSS SECTIONAL STUDY"** is a bonafide research work done by **THE CANDIDATE REG NO. BG0108005** in the Department of General Medicine, Jawaharlal Nehru Medical College, Nehru Nagar, Belgaum – 590 010.

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LIST OF ABBREVIATIONS USED

AF	-	Atrial fibrillation
BP	-	Blood pressure
CAD	-	Coronary artery disease
CBC	-	Complete blood count
CCF	-	Congestive cardiac failure
CHD	-	Coronary heart disease
CSF	-	Cerebro spinal fluid
CT	-	Computed tomography
CVA	-	Cerebrovascular accident
DC	-	Differential count
DM	-	Diabetes mellitus
ECG	-	Electrocardiogram
FLAIR	-	Fluid attenuated inversion recovery
HDL	-	High density lipoprotein
HsCRP	-	High sensitive C-reactive protein
HTN	-	Hypertension
ICH	-	Intracranial hemorrhage
Involv	-	Involvement
IP No.	-	In patients number
LDL	-	Low density lipoprotein
MI	-	Myocardial infarction
MRI	-	Magnetic resonance imaging
n	-	Number of patients
No	-	Number
POCI	-	Posterior circulation infarct

PR	-	Pulse rate
RBS	-	Random blood sugar
RR	-	Respiratory rate
S. Creatinine	-	Serum creatinine
Sr.	-	Serum
T	-	Tesla
T1	-	Spin lattice relaxation time
T2	-	Spin spin relaxation time
TC	-	Total count
TIA	-	Transient ischemic attack
WHO	-	World Health Organization

ABSTRACT

Background and objectives

Globally there are approximately 5.8 million people who die from stroke each year and two-third of all stroke occur in people living in developing countries and there are globally 16.3 million new stroke events each year. The present study was undertaken to know various clinical presentation and correlation with anatomical site with neuroimaging.

Methodology

This one year cross-sectional study was conducted at KLES Dr Prabhakar Kore Hospital and Medical Research Centre Belgaum, during the period of January 2009 to December 2009 on 33 adult patients with brainstem stroke. Detailed history, clinical examination, blood investigations and cranial imaging were performed.

Results

Majority (36.3%) of patients were in the age group of 45 to 54 years with male predominance (63.50%) and presented with limb weakness (81.8%). On admission, 39.4% patients had GCS score < 8. The commonest cranial nerve palsy observed was combined 3^{rd}, 4^{th} and 6^{th} nerve palsy (36.3%). The ischemic brainstem stroke was more common (87.5%) than hemorrhagic stroke (12.2%). On neuroimaging, in patients with ischemic stroke commonest lesion was pontine infarct (39.4%) and midbrain and pontine bleed (9.3%) in hemorrhagic stroke. The most common associated risk factor was hypertension (60%). Mortality was high in patients of GCS score ≤ 8 (61.5%). Patients with hemorrhagic brainstem

stroke had higher mortality (75%) compared to ischemic brainstem stroke (17.2%).

Interpretation and conclusion

Nearly half of patients presented with classical well defined brainstem syndrome which was further confirmed on neuroimaging and it was pointing towards the involvement of the brainstem. Among known risk factors, hypertension was the commonest risk factors in both ischemic and hemorrhagic brainstem stroke. The mortality was higher in hemorrhagic stroke compared to ischemic brainstem stroke.

Keywords

Brainstem stroke; Medullary infarct; Midbrain infarct; Pontine infarct;

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INTRODUCTION

In vertebrate anatomy, the brainstem is the posterior part of brain, adjoining and structurally continues with the spinal cord. All information to and from our body passes through the brainstem on the way to or from the brain. Though small, this is an extremely important part of the brain as nerve connections of motor and sensory system from the main part of the brain to the rest of the body pass through the brainstem.

Globally there are approximately 5.8 million people who die from stroke each year and two-third of all stroke occur in people living in developing countries and there are globally 16.3 million new stroke events each year. Being leading cause of death worldwide, and also the leading cause of lasting disability, stroke is already a major burden for health system and population.[1]

Posterior circulation territory is of special interest, as it consists of the posterior pole of the brain, parts of the basal ganglia, cerebellum, brainstem and spinal cord. Posterior circulation stroke accounts for approximately 20% of all strokes. According to the National stroke Association 10% of total stroke are brainstem stroke.[2]

A stroke affecting the brainstem increases the risk for serious complications and is potentially life threatening since this area of brain controls function such as breathing and heart rate. Brainstem stroke is leading cause of death and disability worldwide.

Brain stem strokes are less common than hemispheric strokes but it is often difficult to localise them to their precise location, and the spectrum of clinical presentation is also very wide, it may initially present as a benign vertigo or headache, or they may rapidly deteriorate because of brainstem compression. However, there are some special clinical features associated with brainstem stroke of individual variety of syndrome which would help to distinguish one set of syndrome from other.

Recent clinical or clinico-radiological studies[3,4] of posterior circulation stroke have rapidly expanded our understanding and knowledge, but there is paucity of studies determining clinical features and outcome in brainstem stroke. In the present study brainstem strokes have been studied to correlate clinical profile. The present study will concentrate on clinico-anatomical correlation and clinical syndrome related to brainstem stroke.

With knowledge of their clinical presentation, the physician's ability to localise them to an anatomical site and to differentiate from hemispheric stroke may improve and one may think of early intervention in rapidly deteriorating patients as brainstem compression is very fatal. Hence the present study was undertaken to know various clinical presentation and correlation with anatomical site with neuroimaging.

OBJECTIVES

Objectives of the present study were;

1. To study the various clinical presentations of brainstem stroke.
2. To correlate anatomical site of lesion on neuroimaging.

REVIEW OF LITERATURE

HISTORICAL BACKGROUND

Hippocrates (460-370 BC), the father of medicine first recognized stroke over 2004 years ago. At this time, stroke was called 'apoplexy', which means 'struck down by violence' in Greek.[5]A few hundred years after Hippocrates, Galen (131-201AD) first described the anatomy of brain and its blood vessels.[6]Thomas Willis during the last half of the 17th century described a circle of anastomotic vessels at the base of brain which is well known by the name of 'circle of Willis'.[7]

R. L. K. Virchow (1821-1902) first described the phenomenology of arterial thrombosis and embolism and suggested three principal predisposing factors for venous thrombosis, which are known as Virchow's triad (Irregularity of lumen, impaired blood flow and increased coagulability).[8]

In the 18th and 19th centuries brinstem syndromes were described by authors such as Weber (1863), Benedikt (1889), Claude (1912), Millard and Gubler (1856), Foville (1858) and Wallenberg (1901). Another important contributor was Dejerine (1849-1917) who first described the clinical findings with various brainstem lesions.[9]

In 1946, Kubik and Adams' fully described the clinical pathology of brainstem infarction due to basilar artery disease.

During the 20th century, especially in the 1970 and 1980 there was an explosive growth of interest in and knowledge about stroke. Advances in technology like computed tomography (CT) and magnetic resonance imaging (MRI) allowed better visualization of the anatomy and functional aspects of the brain and vascular lesions.[10]

INTRODUCTION

Stroke is one of the top three causes of death worldwide; mortality over the first year after first stroke is approximately 20%.[11] Globally there are approximately 5.8 million people who die from stroke each year and two-third of all stroke death occur in people living in developing countries. Being the second leading cause of lasting disability in adults, stroke is already a major burden for health system and population.[1] The economic and social burdens of stroke, however, are not consequences of mortality; they are imposed by the large majority of stroke patients who survived but are physically and mentally disabled by stroke induced brain damage.[11]

Stroke is anything but a homogeneous entity. Stroke refers to any damage to the brain or the spinal cord caused by an abnormality of the blood supply. The term stroke is usually used when the symptoms begin abruptly, whereas cerebrovascular disease is a more general term that carries no connotation as to the tempo of brain injury.[10]

A stroke is a focal neurologic deficit caused by a local disturbance in cerebral circulation, predominantly either an obstruction of cerebral blood or a rupture to a vessel wall supplying blood to the brain.[12]

A stroke is rapidly developing clinical symptoms and/or signs of focal and at times global loss of brain function with symptoms lasting more than 24 hours or leading to death with no apparent cause other than that of vascular origin. This definition embraces stroke due to cerebral infarction, non-traumatic intracerebral hemorrhage, intra-ventricular hemorrhage and some cases of subarachnoid hemorrhage.[13]

EPIDEMIOLOGY

Understanding of the epidemiology of stroke has lagged that of coronary heart disease (CHD) partly because of lack of research findings for stroke and partly because stroke is a much more heterogeneous disorder.[14]

Mortality

The World Health Organization (WHO) estimates that worldwide a stroke accounts for 5.8 million deaths, approximately 10% of all deaths.[15] Stroke mortality varies widely between countries (27.8/100000 in Canada to 203.5/100000 in Russia). Stroke mortality has fallen over the last few decades in many countries; this may be due to fall in stroke incidence, lower case fatality rate or some artifacts of the collection of mortality data.[16] During the last decade stroke represented 1.2% of total deaths in India.[17]

Incidence and prevalence

According to the World Health Organization estimates, there are globally 16.3 million new stroke events each year. Of these 11.2 million occur in developing region, while 5.1 million are in subjects living in developed region.[18]

Age standardized stroke incidence rates per 100,000 varied from 101 to 285 in men and from 47 to 198 in women globally.[19]

Annual incidence rate of stroke in India was 13 to 120 per 100,000 in last decade. During the last decade, age adjusted prevalence rate of stroke was 250 to 350 per 100,000.[17]

The incidence of first-ever-in-a-lifetime posterior circulation infarction (POCI) in general population was 34/100,000 (95% confidence interval=24-43) and ranged from 8/100,000 in subjects aged 25-34 years to 514/100,000 in those with older than 85 years.[20]

In the Edge Stroke Registry there were 26.8% registered posterior circulation strokes. Of these, 21% of strokes were in the posterior cerebral artery territory, 7.8% of strokes were within the cerebellum, 48.3% in the brainstem, 9% in the thalamus and 13.4% in multiple posterior circulation locations.[21]

Epidemiological factors for increased risk of stroke

1. *Age* – Advancing age is the single most important forecaster of a high risk for stroke. The likelihood of stoke increases rapidly, after the age of 55 years.[22] Overall stroke incidence at age of 75 to 84 is about 25 times higher than at age of 45 to 54 years.[14] Atherosclerosis generally evolves slowly over a person's lifetime; thus, it is not surprising that it is the leading arterial cause of ischemic stroke among person older than 55.[22]

2. *Gender* – Gender differences in stroke incidence and mortality have been consistently demonstrated and indicate a greater risk of stroke among

men. The male to female ratio has been estimated to be 1.3 to 1.0 and differs by stroke subtype.[23] One exception to the slightly higher frequency of stroke among women between ages 15 to 30 which attributed to the potential for cerebrovascular complications of pregnancy or the puerperium.[24] The excess of vascular events in men has been attributed to differences in endogenous sex hormones, although there is little evidence to support this hypothesis.[14]

3. *Ethnicity and geography* – Various studies have found that blacks have a greater incidence and prevalence than whites of comparable age, gender and residence.[25] This pattern may be related in part to a higher prevalence of hypertension and diabetes in black population.[26]

   Both hemorrhagic and ischemic stroke affect persons in all ethnic groups in all parts of the world. Although cerebrovascular disease a worldwide scourge, rates vary greatly among countries and regions. Some of the disparity may be secondary to difference in diet or lifestyle. A diet in Eastern Asia differs markedly from that consumed in Mediterranean countries. The relative proportion of hemorrhagic stroke in Eastern Asia is higher than that in the other part of the world. Presumably, this difference reflects the high rate of hypertension in Asian population, which may be related to the high salt content of the diet or genetic predisposition.[22]

4. *Social and economic factors* – Reports from around the world demonstrate a trend for a higher rate of ischemic and hemorrhagic stroke among persons from lower socio-economic class than among more

affluent and educated populations. The high rate of stroke in poor may be explained by several factors like lack of education, health awareness and limited access to health information and facilities.[27]

5. *Environmental factors* – The incidence of both hemorrhagic and ischemic stroke has been associated with change in weather, season of the year, day of the week or the time of the day. In most studies, both stroke mortality and hospital admission rates are higher in winter than summer. This seasonal variation might be explained by the complications of the stroke being more likely to occur in the winter.[28] Ischemic stroke also peaks during the initial hours after awakening in the morning.[22] This time relationship might be secondary to hypertension or changes in coagulation factors that appear early in the morning.

PATHOPHYSIOLOGY

There are two major categories of brain damage in stroke patients:

1. *Ischemia* – A lack of blood flow depriving brain tissue of needed fuel and oxygen.
2. *Hemorrhage* – The release of blood into the brain and extravascular spaces within the cranium. Bleeding damages the brain by cutting off connection pathways and by causing localised or generalised pressure injury.[29]

Ischemia

- It can be further subdivided in to three different mechanisms: thrombosis, embolism and decreased systemic perfusion.
- By convention, thrombosis refers to an obstruction to blood flow due to a localized occlusive process. The most common type of vascular pathology is atherosclerosis.[30]
- In embolism material formed elsewhere within the vascular system lodges in a vessel and blocks the blood flow. The material arises proximally, most commonly from the heart. Cardiac sources of embolism include the heart valves, endocardium, and clots or tumors within the atrial or ventricular cavities.[10]
- In decreased systemic perfusion, diminished flow to brain tissue is caused by low systemic perfusion pressure. The most common causes are cardiac pump failure and systemic hypotension. Poor perfusion is most critical in border zone or so called watershed regions at the periphery of the major vascular supply territories.[31]
- All three mechanisms of ischemia lead to temporary or permanent tissue injury. Permanent injury is termed infarction. The extent of brain damage depends on the location and duration of the poor perfusion and the ability of collateral vessels to perfuse the tissue at risk.

Hemorrhage

- The term describes bleeding directly into the brain substance. The cause is most often hypertension, with leakage of blood from small intracerebral arterioles damaged by the elevated blood pressure.
- Bleeding diathesis especially from the iatrogenic prescription of anticoagulants, trauma, drugs, vascular malformations and vasculopathies can also cause bleeding into the brain. The degree of damage depends on the location, rapidity, volume and pressure of the bleeding.
- When bleeding dissects into the ventricles or on to the surface of the brain, blood is introduced into the cerebrospinal fluid (CSF). The blood clots and solidifies, causing swelling of adjacent brain tissues.
- In adverse situations, such as swelling or hemorrhage arising inside the bony skull, these structures may constitute prison, restricting and strangulating their enclosed contents and forcing herniation of tissue from one compartment to another.[10]

RISK FACTORS

Besides the reported differences in stroke occurrence by age, gender and race, numerous stroke risk factors have been identified that are potentially modifiable. Stroke morbidity and mortality are more likely to be significantly reduced by identification and control of environmental factors in the stroke-prone individuals. Modifiable stroke risk factors as determined by prospective cohort and case-control studies, include hypertension (HTN), diabetes mellitus (DM), cardiac diseases, hypercholesterolemia, cigarette use and alcohol use.[23]

Hypertension

- Hypertension is the principal risk factor for ischemic stroke as well as intracranial hemorrhage (ICH). Hypertension also predisposes to the cardiac conditions, notably myocardial infarction (MI) and atrial fibrillation (AF), promoting cerebral embolism. Thus hypertension serves the unique role of being a prime risk factor for stroke resulting from the most common mechanisms.[32]

- Hypertension, after age is the most powerful stroke risk factor. In the Framingham study, the age adjusted relative risk of stroke among those with definite hypertension was 3.1 for men and 2.9 for women. Even among borderline hypertensives, the relative risk was 1.5 compare to normotensives.[33]

- Hypertension plays a role in multiple mechanisms of stroke. Hypertension plays a role in the athero degenerative process in large blood vessels, resulting in occlusive and artery-to-artery embolic strokes. Also, the association between hypertension and cardiac disease is well known, making cardio-embolic brain infarction more likely. Hypertension also plays role in the rupture of cerebral aneurysms.[34]

Diabetes mellitus

- Diabetic persons are known to have greater susceptibility to coronary, femoral and cerebral artery atherosclerosis; up to 80% those with type 2

diabetes will demonstrate or die of macrovascular disease. Hypertension is common in diabetic persons, affecting approximately 60%.[35]

- Relative risks ranges from 1.5 to 3.0, depending on the type and severity of diabetes mellitus. In the Framingham study, the impact of diabetes was much clearer. The study found the stroke from diabetes in both sex did not diminish with age and was independent of hypertension.[33]
- Diabetes was associated with a two fold increased adjusted risk of thrombo-embolic stroke among Japanese men living in Hawaii and in men and women in Rancho Bernardo, California.[36]
- Diabetes is a risk factor for intracranial and extracranial large artery occlusive disease and penetrating artery disease. Intracranial branch artery atheromatous disease is particularly common among diabetic patients. Atheromatous branch disease affects predominantly the paramedian pontine penetrating arteries, anterior choroidal arteries, and anterior inferior cerebellar arteries.[37]

Cardiac disease

- Cardiac disease and impaired cardiac function are disease state or organ dysfunction that predispose to stroke.[32]

- Cardiac disease has been clearly associated with increasing the risk of ischemic stroke, particularly atrial fibrillation, valvular heart disease, myocardial infarction, coronary artery disease (CAD), congestive cardiac failure (CCF) and perhaps prolapsed mitral valve. Because certain stroke risk factors like hypertension may also be determinants of cardiac disease,

some cardiac condition may be viewed as intervening events in the causal chain for stroke.[23]

- Nonvalvular atrial fibrillation is also a potent predictor of stroke, increasing the relative risk of stroke nearly five fold.

- Coronary heart disease (CHD) predisposes to stroke by a variety of mechanisms - as a source for embolism from the heart; by virtue of shared risk factors; as untoward effects of medical and surgical treatment for coronary atherosclerotic disease; and less commonly, as consequences of pump failure. Stroke occurs most frequently within two weeks after acute MI, affecting between 0.7% and 4.7% of MI patients.[32]

Hypercholesterolemia

- Abnormalities of serum lipids (triglycerides, cholesterol, low density lipoprotein (LDL), high density lipoprotein (HDL) are clear risk factors for atherosclerotic disease, particularly coronary disease.

- Studies utilizing ultrasound technology are established that cholesterol or LDL cholesterol is directly associated and HDL cholesterol is inversely associated with extracranial carotid atherosclerosis and intima-media plaque formation.[38]

Smoking

- Cigarette smoking has been clearly established as a biologically-plausible, independent determinant of increased stroke risk. In the Framingham

study, cigarette smoking accounted for an adjusted relative risk of brain infarction of 1.7 after controlling for other cardio-vascular risk factors.[33]

- The mode of action of cigarette smoking in causing stroke is not entirely clear, but acceleration of atherosclerosis is one possibility. Cigarette smoking was found to be an independent determinant of carotid artery plaque thickness and the strongest predictor of severe extracranial carotid artery atherosclerosis.[23]

- Other potential biological mechanisms that can induce stroke include increased blood viscosity, hypercoagulability, elevated fibrinogen levels, enhanced platelets aggregation, and elevation of blood pressure.[39]

Alcohol use

- As in MI, the effects of alcohol consumption on stroke risk are related to the amount of alcohol consumed. Heavy alcohol use, either habitual or binge drinking, seems to be related to higher rates of cardiovascular disease. Light or moderate alcohol consumption, on the other hand, is inversely related to incidence of CHD.[40]

- In Northern Manhattan, however, J shaped relationship between alcohol and stroke was found, an elevated stroke for heavy alcohol consumption and a protective effect in light to moderate drinker when compared with nondrinker.[41]

- The various mechanisms through which alcohol may increase the risk of stroke include hypertension, hypercoagulable states, cardiac arrhythmias

and cerebral blood flow reduction. There is also evidence that light to moderate drinking can increase HDL cholesterol, reduce the risk of coronary artery disease, and increase endogenous tissue plasminogen activators. The combination of deleterious and beneficial effects of alcohol is consistent with the observation of dose-dependent relationship between alcohol and stroke.[12]

Blood homocysteine level

- Homocysteine is an intermediate in the metabolism of the amino acid, methionine. An elevated level of plasma total homocysteine increases the risk of vascular disease and is an independent risk factor for stroke.[42]

- In a number of cross-sectional studies, in case-control studies, and in meta-analysis, elevated values of plasma homocysteine were found to be associated with a higher incidence of stroke.

- A number of vascular and hematological abnormalities are associated with elevated serum homocysteine levels. Mild hyperhomocysteinemia is independent risk factor for carotid artery wall thickness and plaque formation. Other reported pathophysiological changes include endothelial cell injury, increased platelet aggregation and abnormalities of clotting cascade.[43]

CLINICAL MANIFESTATIONS

Considering the complexity of function of the brain, clinical symptomatology of stroke might be very complex. It is important to concentrate

on semiology and anatomico-pathological correlation of stroke. There are some pointers such as some special clinical features associated with strokes of individual varieties and the knowledge of brainstem anatomy which would help the physician to distinguish one set from the other. Knowledge of the vascular anatomy of the central nervous system is crucial for making decisions about evaluation and treatment of affected patients. Categorization of vascular events is often based on the involved vascular territory.

Clinical clues to localize brainstem infarction

- One of the first such cardinal symptoms is vertigo, which one must look for in the history, and if the patient has vertigo, it should immediately point towards a brainstem disorder.

- The second important feature is the presence of cranial nerve symptoms or signs in the history or on physical examination.

- The third important pointer is the presence of crossed signs, that is, presence of ipsilateral motor and sensory cranial nerve signs or symptoms and contralateral hemiplegia, hemianaesthesia, or both.

- The fourth important pointer is the presence of occulomotor signs. These features are some of the primary and common ones that the physician would need to pickup immediately and which suggest a brainstem disease.

- The fifth important pointer is bilateral simultaneous involvement of long tracts, either sensory or motor, either symmetric or asymmetric, either simultaneous or sequential. For example, if there is a patient of stroke

who has simultaneous bilateral hemiparesis or sequential hemiparesis, first on one side of the body and then after a while or the next day, on the other side, one should think of brainstem.[44]

Pontine stroke

Pontine stroke constitute approximately 15% of posterior circulation stroke. Multiple alternate syndromes of pontine infarction have been described.

1. Millard – Gubler and Foville syndrome
    - Contralateral hemiparesis
    - Ipsilateral paralysis of abducens nerve
    - Contralateral decrease of temperature and pain sensation
    - Ipsilateral peripheral paralysis of facial nerve.
2. Midpontine base syndrome:
    - Contralateral hemiparesis
    - Ipsilateral ataxia
    - Ipsilateral paralysis of masticators and decrease of all sensory modalities over the ipsilateral face.
3. Rostral pontine syndrome:
    - Ipsilateral decrease of temperature and pain sensation on the face.
    - Ipsilateral decrease of touch and discrimination sensation on the face.
    - Ipsilateral flaccid paralysis of masticators
    - Decrease of blink response
    - Contralateral decrease of temperature and pain sensation
    - Contralateral decrease of touch, position and vibration sensation

- Paralysis of facial nerve and lower cranial nerves
- Ipsilateral hemiataxia, dysdiadochokinesia

4. Caudal pontine syndrome
    - Ipsilateral conjugate gaze paralysis, nystagmus
    - Ipsilateral paralysis of eye abduction
    - Ipsilateral peripheral paralysis of facial nerve
    - Ipsilateral hemiataxia and dysdiadochokinesia
    - Contralateral decrease of pain and temperature sensation
    - Contralateral decease of touch, position and vibration sensation
    - Ipsilateral myorythmias of soft palate

Medullary stroke

Historically, the following classic alternating medulla oblongata syndromes are distinguished.

1. Dorsolateral medulla oblongata syndrome- Wallenberg's syndrome
    - Ipsilateral decrease of temperature and pain sensation on face
    - Horner's syndrome
    - Contralateral decrease of temperature and pain perception on the body
    - Ipsilateral ataxia and hypotonia
    - Ipsilateral paresis of soft palate and pharangeal muscle
    - Nystagmus, tachycardia or hiccup
2. Medial medullary syndrome- Dejerine's syndrome
    - Contralateral hemiparesis involving face, arm and leg

- Ipsilateral paralysis of hypoglossal nerve
- Myorythmias of pharynx
- Contralateral decrease of touch, position and vibration sensibility

3. Opalski syndrome
    - When ipsilateral hemiplegia is associated with symptoms of lateral medullary syndrome, correspond to submedullary syndrome of Opalski.

Midbrain stroke syndrome

Several clinical syndrome of midbrain stroke have been described including Weber, Cluade, Achard-levi and Benedict's syndrome.

1. Weber's syndrome

    - Ipsilateral occulomotor paresis
    - Contralateral hemiparesis with supranuclear paresis of facial and hypoglossal nerves
    - Contralateral ataxia
    - Contralateral rigidity

2. Claude's syndrome

    - Two clinical symptoms are typical for this syndrome
    - Ipsilateral occulomotor nerve palsy
    - Contralateral cerebellar dysfunction

3. Benedict's syndrome

    - Ipsilateral paralysis of occulomotor nerve
    - Contralateral decrease of touch, position and vibration
    - Contralateral rigidity
    - Contralateral chorea, athetosis

4. Eye movement disturbances in midbrain infarct

    - Conjugate vertical gaze palsy
    - Slowing smooth persuit
    - Torsional nystagmus
    - Pseudoabducens palsy
    - Convergence – retraction nystagmus
    - Vertical gaze palsy
    - Skew deviation

OUTCOME AND PROGNOSIS

As the second most common cause of death in the world and a major cause of long term disability, stroke continues to have a great effect on public health. Stroke accounts for the greatest number of hospitalization for neurological diseases.[45]

Stroke subtype and comorbid conditions influence outcome. Important outcome measures for stroke include mortality, recurrence, functional disability and quality of life.

Mortality

Death is the most important early outcome. Mortality is usually measured at 30 days and then annually. Two factors related to early mortality are stroke severity and stroke subtype.

Mortality following ischemic stroke

- The greatest risk of death for patients with cerebral infarction occurs in first 30 days, with case-fatality rates ranging from 8% to 20%.
- Longitudinal studies among patients with ischemic stroke have demonstrated that the risk of death at 5 years ranged from 40% to 60%, including early fatalities. The average annual mortality rate in the 30-day stroke survivors ranges from 8% to 9%, and a risk of death is two to three times higher for such patients then for the age-and sex-matched general population.
- Predictor of death after ischemic stroke is as summarized below:

Neurological status

- Decreased level of consciousness
- Major focal neurological impairment
- Stroke due to large artery occlusion
    - Internal carotid artery
    - Proximal middle cerebral artery
    - Basilar artery
- Recurrent strokes

- Cause of stroke
    - Large artery atherosclerosis
    - Cardioembolism
- Epidemiological features
    - Advanced age
- Comorbid disease
    - Diabetes mellitus or hyperglycemia
    - Heart disease – congestive heart failure
    - Lung disease
- Baseline brain imaging findings
    - Dense artery sign
    - Mass effect
    - Hemorrhagic transformation

Mortality after hemorrhagic stroke

- In comparison to patients with ischemic strokes the prognosis of patients with intracranial bleeding is much more guarded.
- Approximately 35% of patients with hemorrhagic stroke will die within 30 days of the event.
- Hemorrhagic stroke is second to cardiac ischemia and arrhythmias as a cause of sudden death.[22]
- Predictors of death after ICH included large size of the hemorrhage, enlargement of hemorrhage, high degree of impairment of consciousness

on admission, lower Glassgow Coma scale (GCS) score, presence of intraventricular hemorrhage, increased pulse pressure, old age, and infratentorial origin of ICH.[32]

Recurrence of stroke

Recurrence after ischemic stroke

- Recurrent stroke is a major cause of morbidity and mortality among stroke survivors. With improvements in survival after first ischemic stroke, stroke recurrence may account for a greater share of the future annual cost of stroke-related health care. [32]

- Stroke recurrence, particularly after ischemic stroke, varies between 1% and 4% in the first month. Recurrence at one year is about 5%-25%, and at 5 years it may be as great as 20% to 40%. Recurrence rate varies by subtype and comorbid condition.

- Prognostic factors for recurrent stroke are clinically important because they help to identify patients at high risk for recurrence and provide insights into ways to modify outcomes. Factors that have been associated with an increased risk of recurrent stroke in community-based and hospital-based series are as follows:

    - Increasing age
    - Male sex
    - Clinical stroke syndrome
    - History of transient ischemic attacks (TIA)

- Hypertension
- Cigarette smoking
- Alcohol abuse
- Diabetes mellitus
- History of coronary heart disease, atrial fibrillation, and other cardiac disease
- Dementia after stroke

Recurrence after hemorrhagic stroke

- Recurrence after ICH is usually low; however, the rate has not been well documented, probably because of the high early mortality.

- The recurrence rate for any stroke was 4.3% per patient-year. About three forth of recurrent strokes were ICHs, at a rate of 2.3% per patient-year; the recurrence rate for ischemic stroke was significantly lower, 1.1% per patient-year.

- Recurrence is uniformly associated with greater disability and a higher mortality rate.[46]

Functional disability after stroke

- Functional disability, the lack of ability to perform an activity or task in the range considered normal for an individual, is an important outcome after nonfatal stroke.

- The approximate proportion of stroke survivors who are independent at 6 months ranges from 40% to 65%, depending on the characteristics of the study population.

- The potential for recovery and the likelihood of long term survival, free of dependence on others are of concern for survivors of stroke, their families, and health care professionals.

- The major predictors of poor long term outcome in this group were a low level of activity before the stroke, subsequent recurrent stroke, older age, baseline disability defined by Barthel Index score, and severe stroke at onset.

METHODOLOGY

The present study was conducted at KLES Dr Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of January 2009 to December 2009.

Study design

This cross-sectional study was conducted on patients presenting with brainstem stroke.

Study Period

Present one year study was carried out during the period of January 2009 to December 2009.

Method of collection of data

Source of Data

Adult patients presenting with brainstem stroke to the KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum.

Sample size

This cross-sectional study was conducted on 33 adult patients.

Sampling procedure

All the patients with brainstem stroke admitted to KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum were included in the study.

Selection Criteria

Inclusion Criteria

- All the patients presenting with brainstem stroke.

Exclusion Criteria

- Traumatic brainstem hemorrhage
- Known case of cerebrovascular accident (CVA) and TIA
- Other neurological diseases

Procedure

The study was approved by the Ethical and Research Committee of Jawaharlal Nehru Medical College, Belgaum.

All patients presenting with brainstem stroke to the KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum were screened for the eligibility. After finding the suitability as per selection criteria they were selected for the study and briefed about the nature of the study and the procedures used and written informed consent was obtained (Annexure–I). The consented patients were enrolled in the present study.

The descriptive data of participants like name, age, sex, detailed history, were obtained by interviewing the participants and recorded on predesigned and pretested proforma (Annexure-II). Detailed history, clinical examination, blood investigations and cranial imaging were performed.

Investigations

- Complete blood count (CBC)
- Blood sugar
- Blood urea
- Serum creatinine
- Electrocardiogram (ECG)
- Computed tomography (CT) / Magnetic resonance imaging (MRI) brain
- Special investigations if required

Magnetic resonance imaging of brain

Magnetic resonance imaging was done in patients using Siemen's Symphony 1.5 Tesla (T) MRI machine. Brain plain imaging with or without contrast was done. Diffusion weighted imaging, T1, T2, FLAIR sequences were taken.

Computed tomography scan

- The patients were scanned using Siemen's single slice spiral CT machine.
- Brain plain with or without contrast scan was done.

Other investigations

Other investigations wherever indicated were performed.

- Biochemical tests.
    - Serum calcium.

- Serum magnesium.
- Serum cholesterol.
- Serum triglyceride.
- High sensitive C-reactive protein (HsCRP).
- Serum homocystein.

- Electrocardiogram, Chest X-Ray, Echocardiography.

Statistical analysis

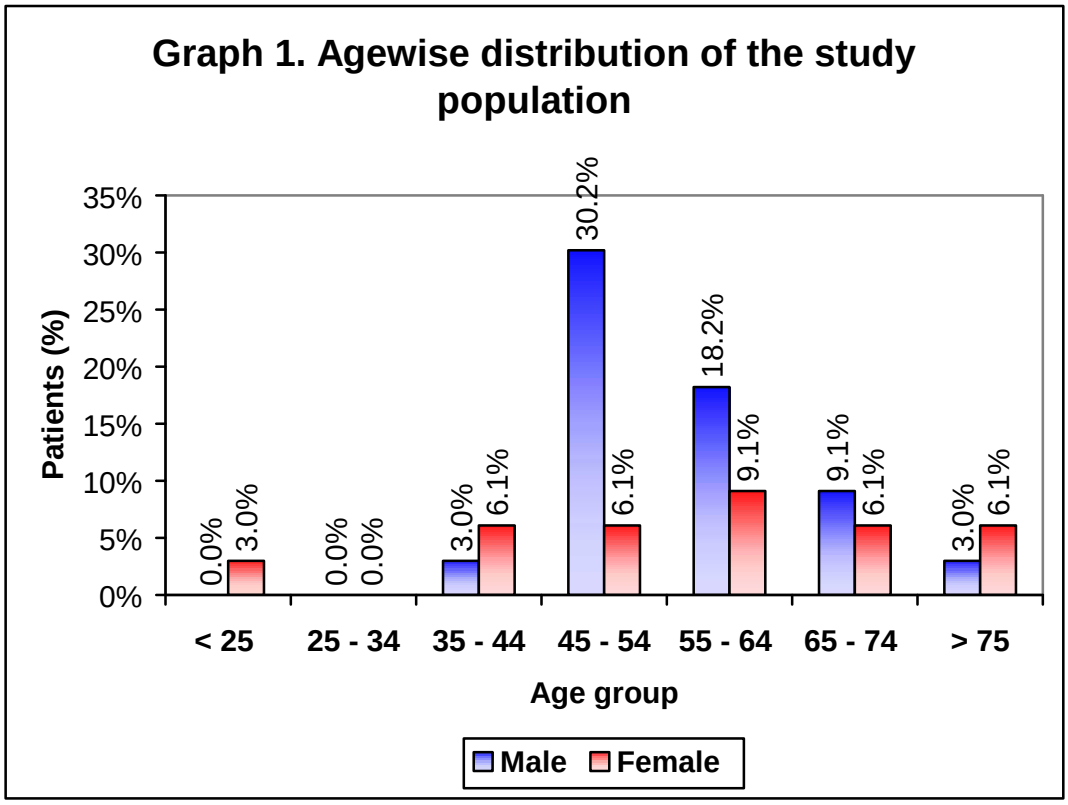
The data obtained was tabulated and analysed using rates, ratios and percentages for different clinical manifestations, cranial imaging findings, etiologies and diagnosis.

RESULTS

The present study was conducted on 33 patients of brainstem stroke admitted to KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. The results obtained are tabulated as below.

Table 1. Agewise distribution of the study population

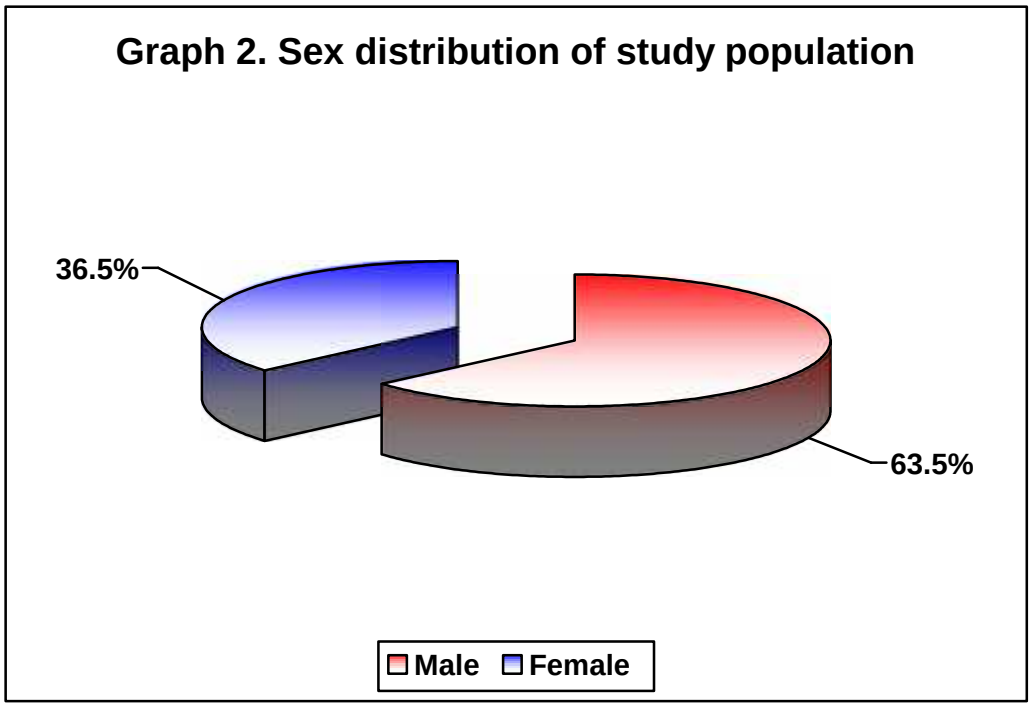
Age (Years)	Male		Female		Total	
	Number	**Percentage**	**Number**	**Percentage**	**Number**	**Percentage**
< 25	0	0.0	1	3.0	1	3.0
25 – 34	0	0.0	0	0.0	0	0.0
35 – 44	1	3.0	2	6.1	3	9.1
45 – 54	10	30.2	2	6.1	12	36.3
55 – 64	6	18.2	3	9.1	9	27.3
65 – 74	3	9.1	2	6.1	5	15.2
> 75	1	3.0	2	6.1	3	9.1
Total	**21**	**63.50**	**12**	**36.50**	**33.0**	**100.00**



The maximum numbers of patients were in the age group of 45 to 54 years (36.2%). Numbers of male patients were more than female patients. The youngest patient was 22 years old and the eldest was 90 years old.

Table 2. Sex distribution of study population

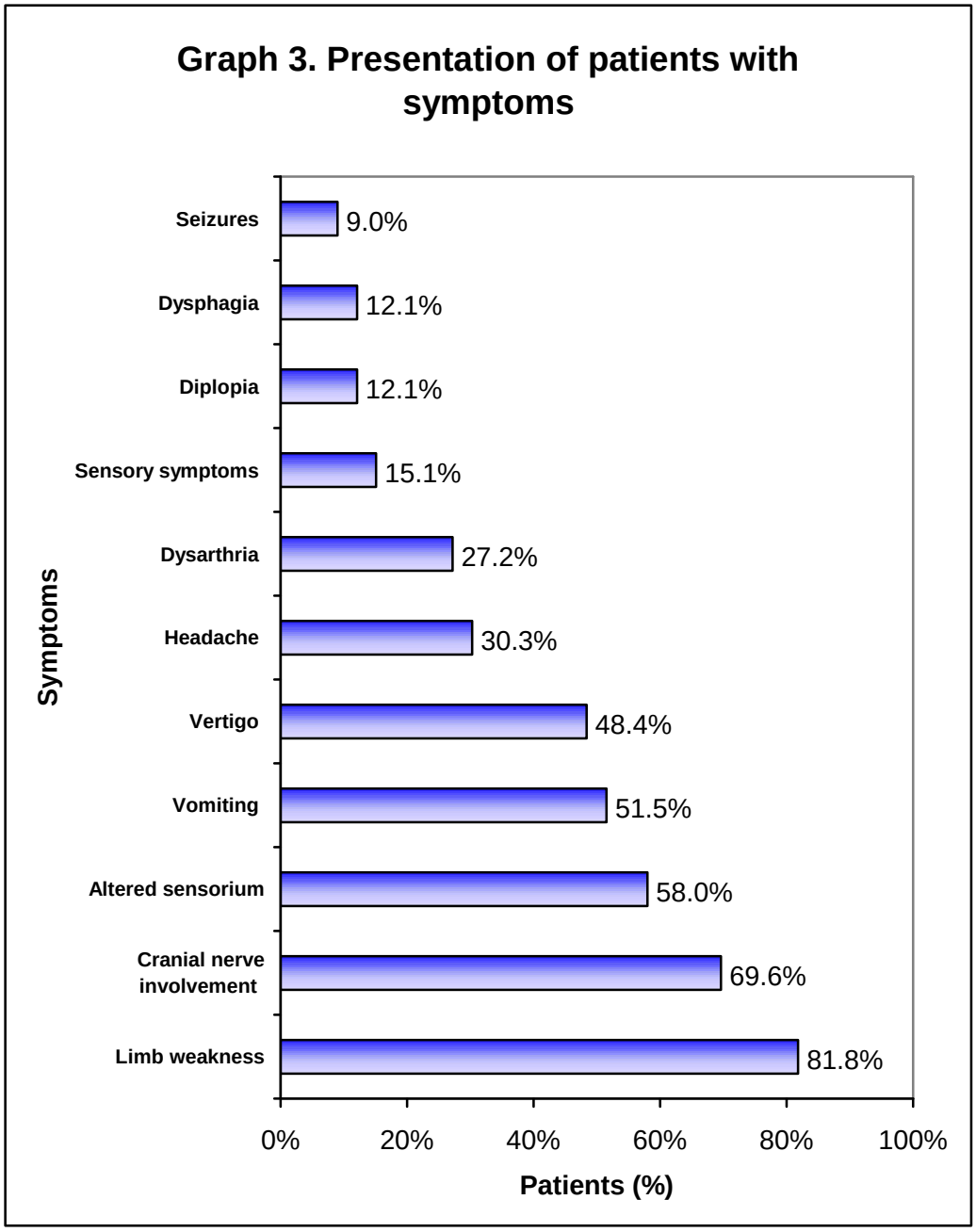
Gender	Patients	
	Number	**Percentage**
Male	21	63.5
Female	12	36.5
Total	**33**	**100**



In the present study population, out of 33 cases, 21 (63.5%) were males and 12 (36.5%) were females with Male to Female ratio of 1.75: 1.

Table 3. Presentation of patients with symptoms

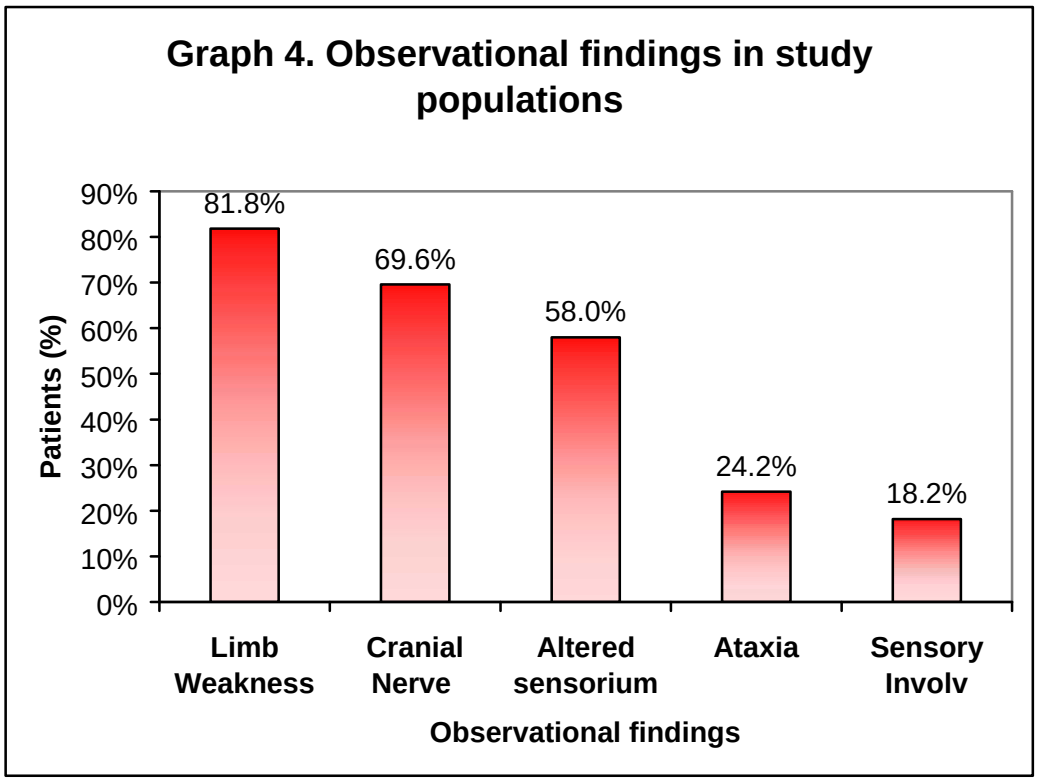
Clinical features	Patients	
	Number	**Percentage**
Limb weakness	27	81.8
Cranial nerve involvement	23	69.6
Altered sensorium	19	58.0
Vomiting	17	51.5
Vertigo	16	48.4
Headache	10	30.3
Dysarthria	9	27.2
Sensory symptoms	5	15.1
Diplopia	4	12.1
Dysphagia	4	12.1
Seizures	3	9.0



Majority (81.8%) of patients presented with limb weakness followed by cranial nerve involvement (69.6%), altered sensorium (58.0%), vomiting (51.5%), vertigo (48.4%), headache (30.3%) and dysarthria (27.0%). Other clinical features were as depicted in the table.

Table 4. Observational findings in study populations

Observational findings	Patients	
	Number	**Percentage**
Limb weakness	27	81.8
Cranial nerve involvement	23	69.6
Altered sensorium		
GCS ≤ 8	13	39.4
GCS 9 – 12	6	18.2
Total	19	58.0
Ataxia	8	24.2
Sensory involvement	6	18.2

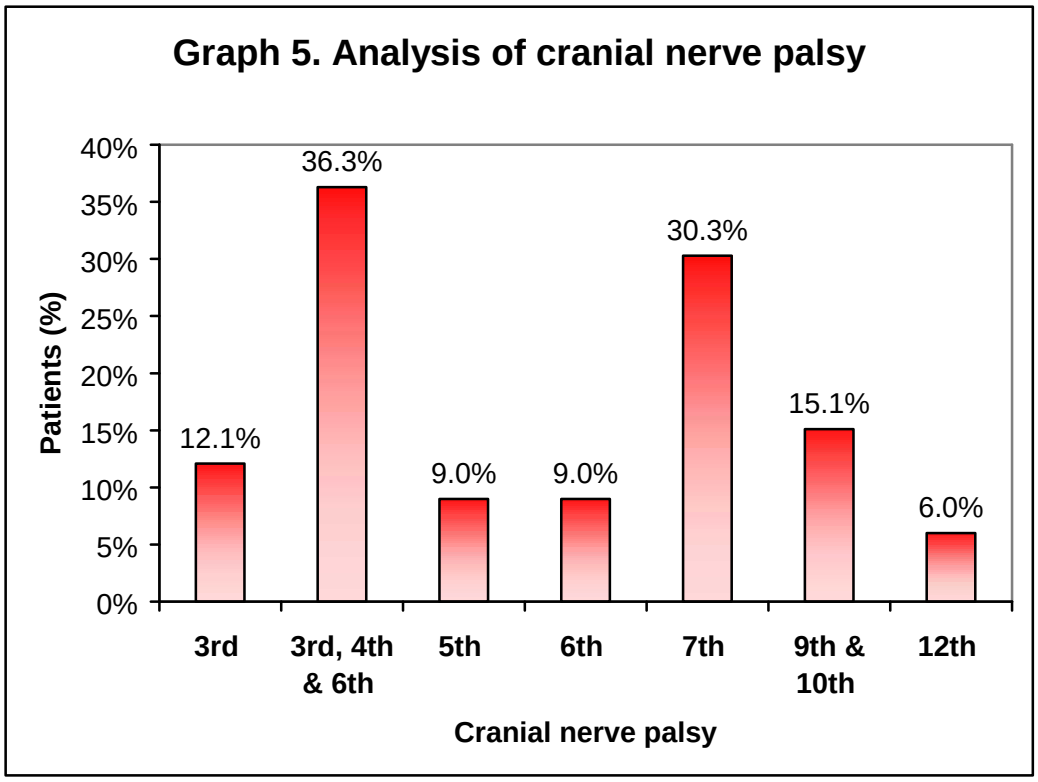


Majority of the patients presented with limb weakness (81.8%), cranial nerve involvement (69.6%) and altered sensorium (58.0%), while ataxia and sensory involvement were observed in minority of the patients.

On admission, out of 33 patients 39.4% patients had GCS score ≤ 8, 18.2% had between 9 to 12 and 42.4% had GCS score ≥ 13.

Table 5. Analysis of cranial nerve palsy

Cranial nerve involvement	Patients	
	Number	Percentage
3rd Nerve Palsy	4	12.1
3rd , 4th and 6th nerve palsy	12	36.3
5th Nerve palsy	3	9.0
6th Nerve palsy	3	9.0
7th Nerve palsy	10	30.3
9th and 10th Nerve palsy	5	15.1
12th Nerve palsy	2	6.0



Commonest cranial nerve palsy observed was combined 3rd, 4th and 6th nerve palsy (36.3%) followed by 7th nerve palsy (30.3%). Other cranial nerve involvement observed was as depicted in the table.

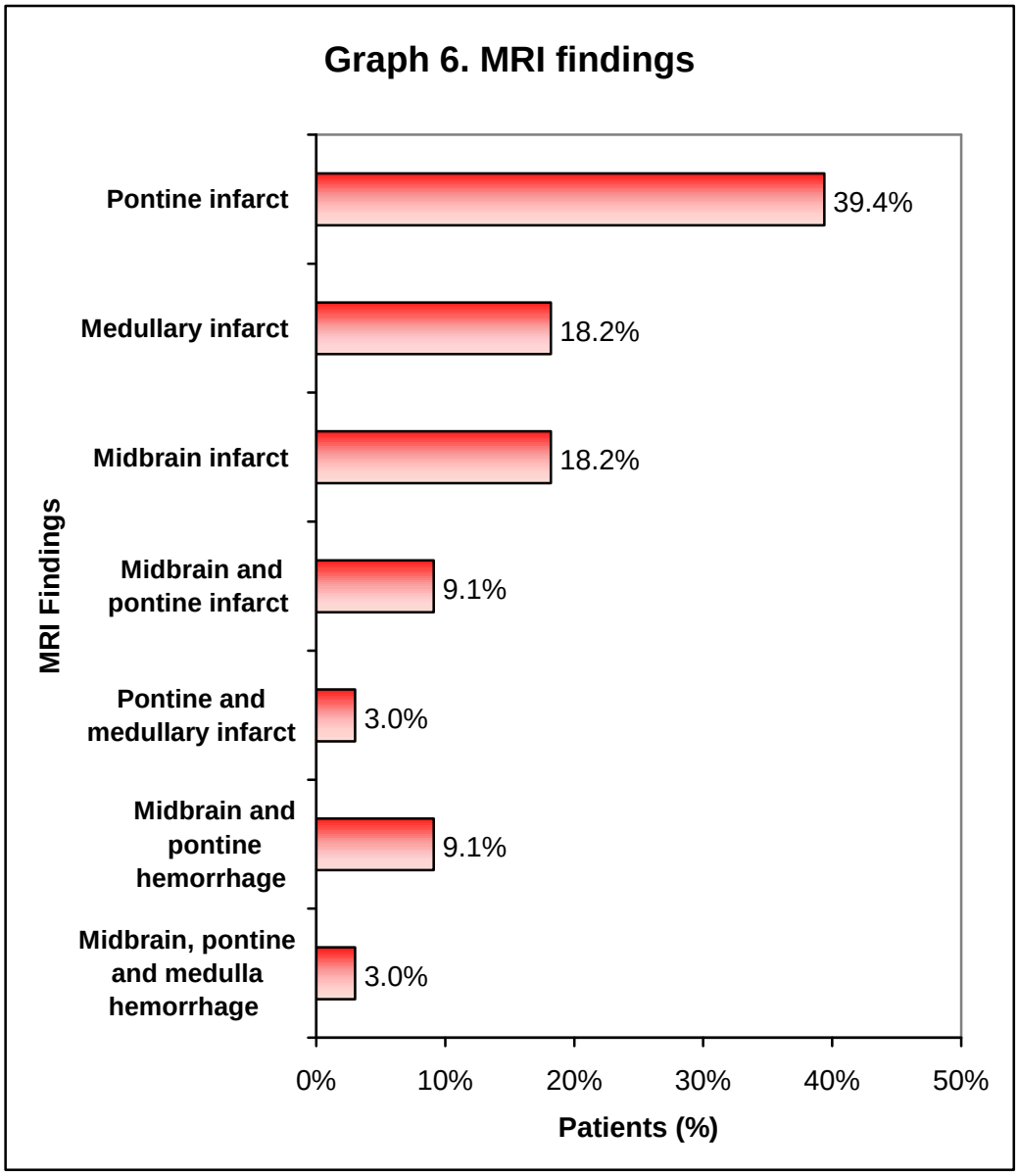
Table 6. Syndromes observed in 33 patients

Syndromes	Patients	
	Number	**Percentage**
Wallenberg syndrome	5	15.1
Weber's syndrome	3	9.0
Pure motor hemiparesis	1	3.0
Raymond syndrome	1	3.0
One and half syndrome	1	3.0
Millard-Gubler syndrome	1	3.0
Claude's syndrome	1	3.0
Dejerine's anterior bulbar syndrome	1	3.0
Top of basilar syndrome	1	3.0
Total	15	45.4

Wallenberg syndrome was the commonest (15.1%) observed syndrome followed by Weber's syndrome (9.0%) in the study population. Other syndromes observed were as depicted in table.

Table 7. MRI findings

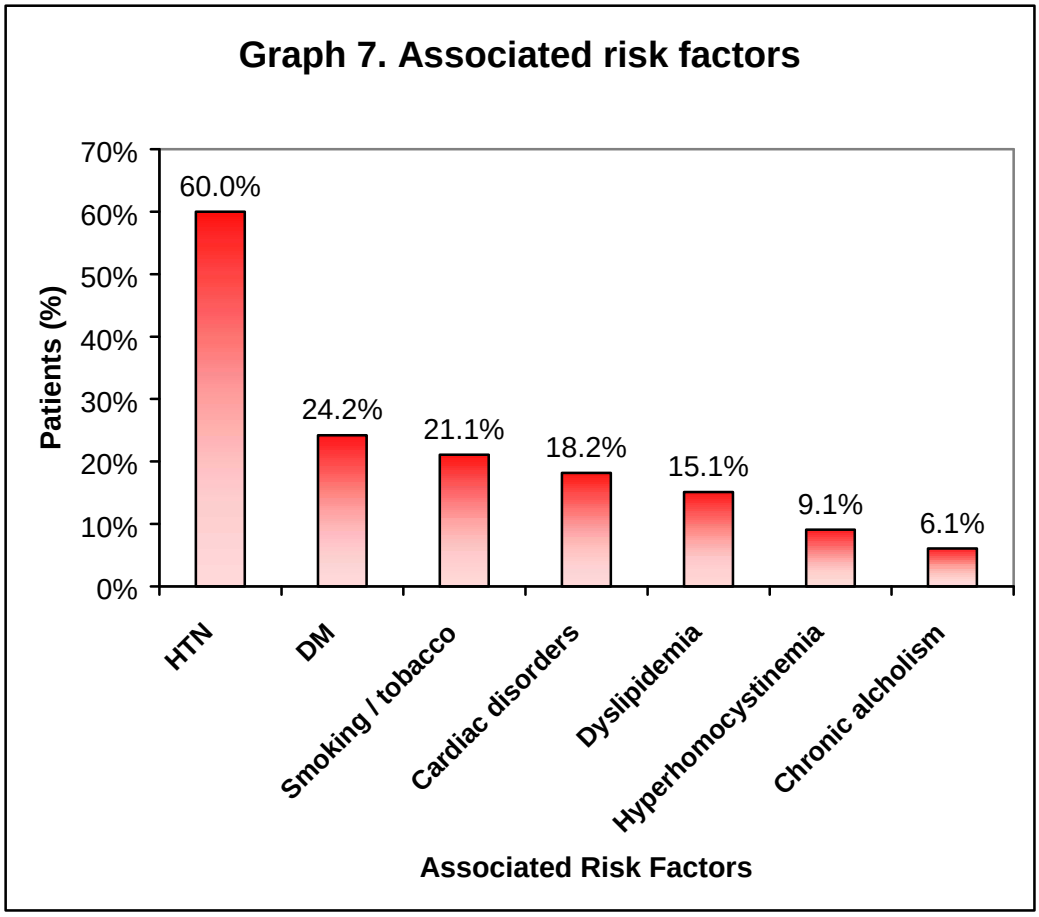
MRI findings		Patients	
		No	**%**
Brainstem ischemic stroke	Pontine infarct	13	39.4
	Medullary infarct	6	18.2
	Midbrain infarct	6	18.2
	Midbrain and pontine infarct	3	9.1
	Pontine and medullary infarct	1	3.0
	Total	29	87.8
Brainstem hemorrhagic stroke	Midbrain and pontine hemorrhage	3	9.1
	Midbrain, pontine and medulla hemorrhage	1	3.0
	Total	4	12.2



On neuroimaging in patients of ischemic stroke, commonest lesion was pontine infarct accounting for 39.4% followed by medullary and midbain infarct 18.2% each. Other ischemic strokes observed were as depicted in table. In hemorrhagic stroke commonest was midbrain and pontine bleed accounting for 9.1%.

Table 8. Associated risk factors

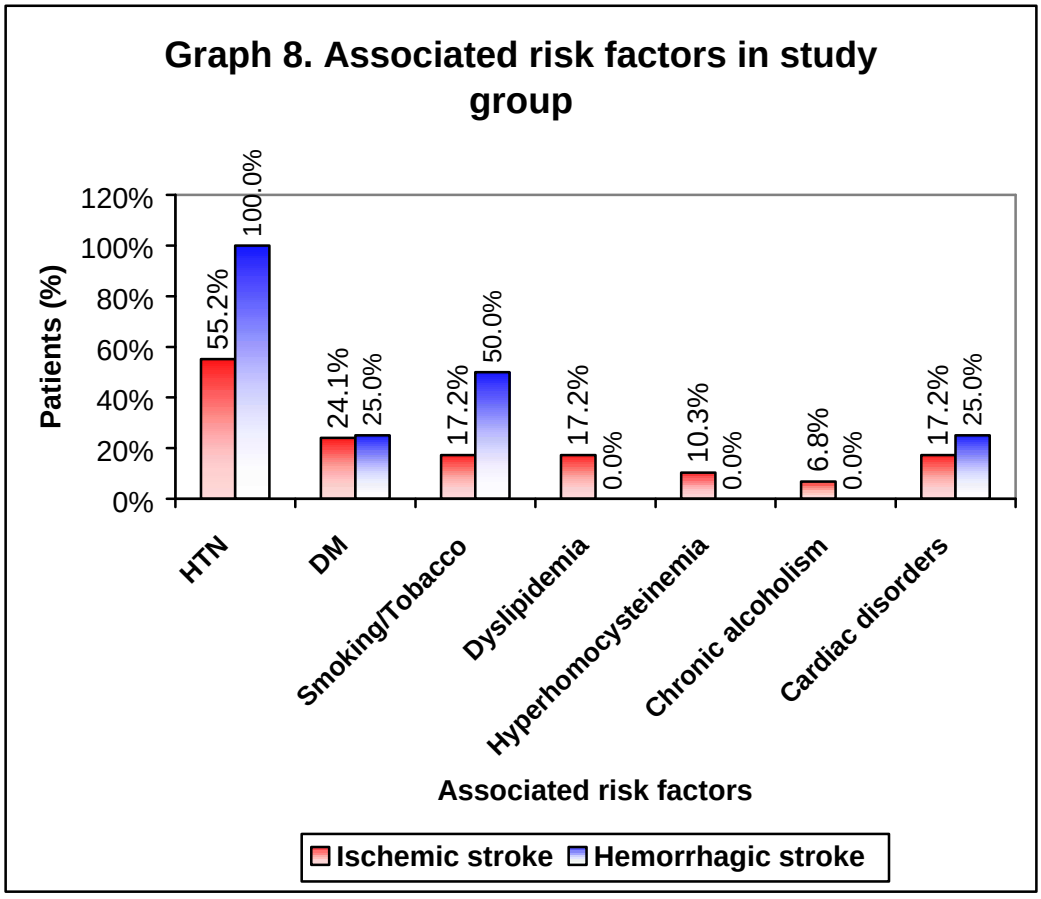
Associated risk factors	Patients	
	Number	**Percentage**
Hypertension	20	60.0%
Diabetes mellitus	08	24.2%
Smoking/Tobacco chewing	07	21.1%
Cardiac disorders	06	18.2
Dyslipidemia	05	15.1%
Hyperhomocysteinemia	03	9.1%
Chronic alcoholism	02	6.1%
No risk factors found	05	15.1%
Total	33	100%



Common associated risk factors observed were hypertension (60.0%), diabetes mellitus (24.2%), smoking / tobacco chewing (21.1%), cardiac disorders (18.2%) and dyslipidemia (15.1%). Other associated risk factors responsible for stroke are as shown in table.

Table 9. Associated risk factors in the study population

Associated risk factors	Ischemic stroke (n=29)		Hemorrhagic stroke (n=4)		Total (n=33)	
	No.	%	No.	%	No.	%
Hypertension	16	55.2	4	100.0	20	60.0
Diabetes mellitus	7	24.1	1	25.0	8	24.2
Smoking/Tobacco chewing	5	17.2	2	50.0	7	21.1
Cardiac disorders	5	17.2	1	25.0	6	18.2
Dyslipidemia	5	17.2	0	0.0	5	15.1
Hyperhomocysteinemia	3	10.3	0	0.0	3	9.1
Chronic alcoholism	2	6.8	0	0.0	2	6.1



In the patients of ischemic brain stem stroke (n=29) hypertension was the most commonest risk factor (55.2%) followed by diabetes (24.1%), smoking / tobacco chewing (17.2%), dyslipidemia (17.2%) and cardiac disorder (17.2%), and in the patients of hemorrhagic brainstem stroke hypertension was present in all (100%) the patients.

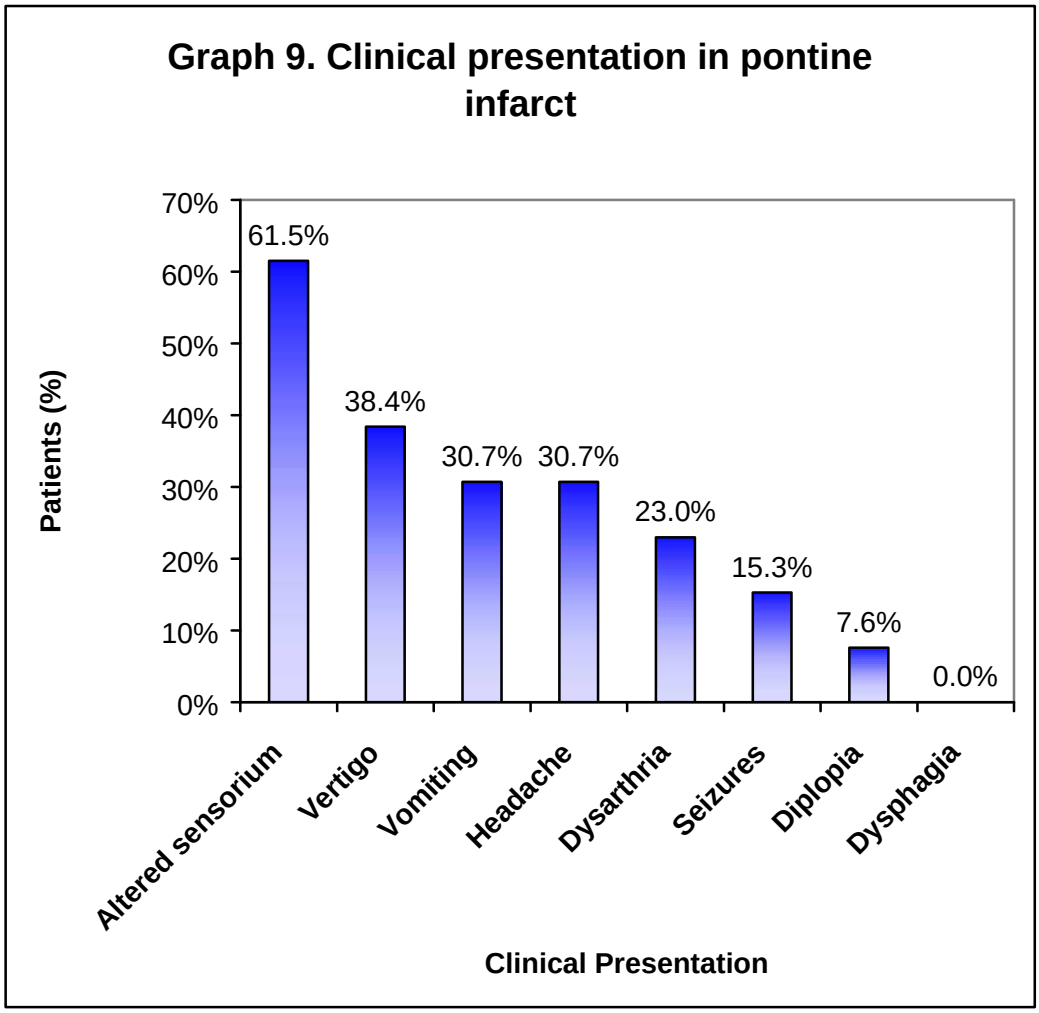
Patients with various risk factors, patient's presentations were mixed (ischemic / hemorrhagic), because of overlapping of the risk factors. Those patients who had only hypertension as risk factor, they had both presentations (Ischemic /hemorrhagic).

In patients of diabetes and hypertension as risk factors, majority had ischemic stroke, only one had hemorrhagic stroke. Out of seven patients with smoking / tobacco chewing, two had associated hypertension, both had hemorrhagic stroke.

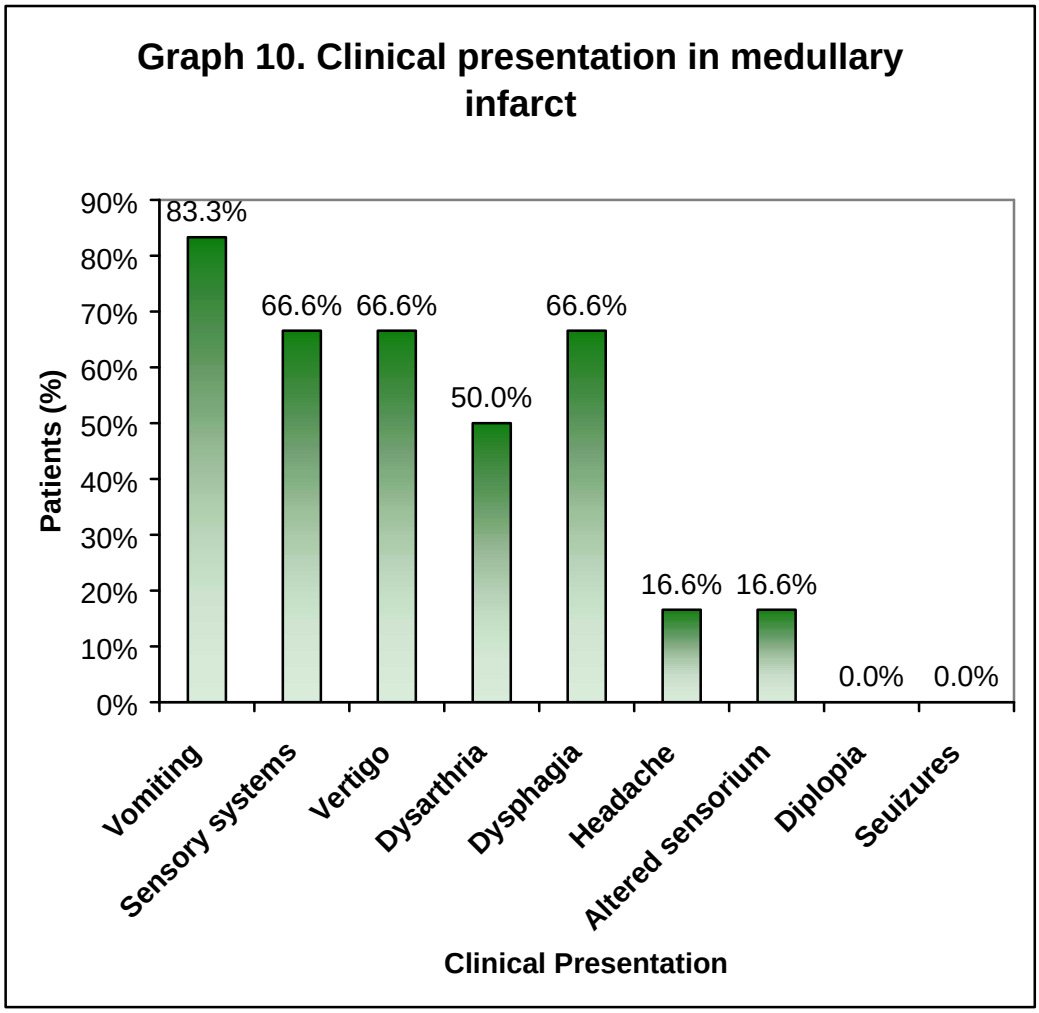
Table 10. Various clinical presentations

Clinical features	Involvement of one structure			More than one structure involved			
	Pontine infarct (n=13)	Medullary infarct (n=6)	Midbrain infarct (n=6)	Midbrain and pontine infarct (n=3)	Midbrain and pontine hemorrhage (n=3)	Miscellaneous (n=2)	Total (n=33)
Altered sensorium	8 (61.5%)	1 (16.6%)	2 (33.3%)	3 (100%)	3 (100%)	2 (100%)	19 (57.5%)
Vomiting	4 (30.7%)	5 (83.3%)	2 (33.3%)	1 (33.3%)	3 (100%)	2 (100%)	17 (51.5%)
Vertigo	5 (38.4%)	4 (66.6%)	3 (50.0%)	3 (100%)	0 (0%)	1 (50%)	16 (48.4%)
Headache	4 (30.7%)	1 (16.6%)	1 (16.6%)	0 (0%)	3 (100%)	1 (50%)	10 (30.3%)
Dysarthria	3 (23.0%)	3 (50.0%)	2 (33.3%)	0 (0%)	0 (0%)	1 (50%)	9 (27.2%)
Diplopia	1 (7.6%)	0 (0%)	3 (50.0%)	0 (0%)	0 (0%)	0 (0%)	4 (12.1%)
Sensory symptoms	0 (0%)	4 (66.6%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	4 (12.1%)
Seizures	2 (15.3%)	0 (0%)	1 (16.6%)	0 (0%)	0 (0%)	0 (0%)	3 (9.1%)
Dysphagia	0 (0%)	4 (66.6%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	4 (12.1%)

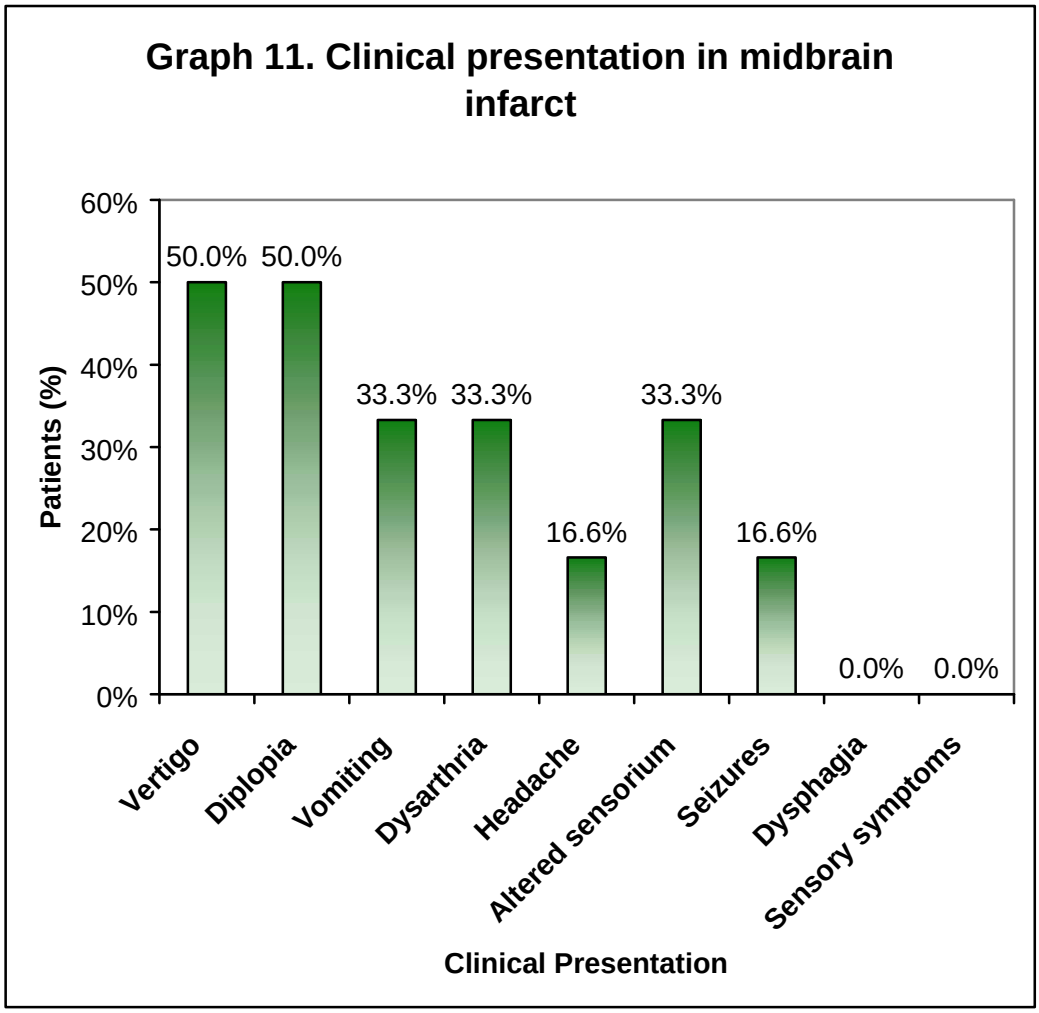
Depending on the lesion at different sites, various presentations of the patients are as shown in the table and attempt is made as well to depict independent lesion at different site on bar diagram.



Majority of the patients of pontine infarct presented with altered sensorium (61.5%), vertigo (38.4%), vomiting (30.7%) and headache (30.7%). Dysarthria (23%), seizures (15.3%) and diplopia (7.6%) were seen in minority of the patients.



Graph no. 10 shows that majority of the patients of medullary infarct presented with vomiting (83.3%), sensory symptoms (66.6%), vertigo (66.6%), dysphagia (66.6%) and dysarthria (50%).

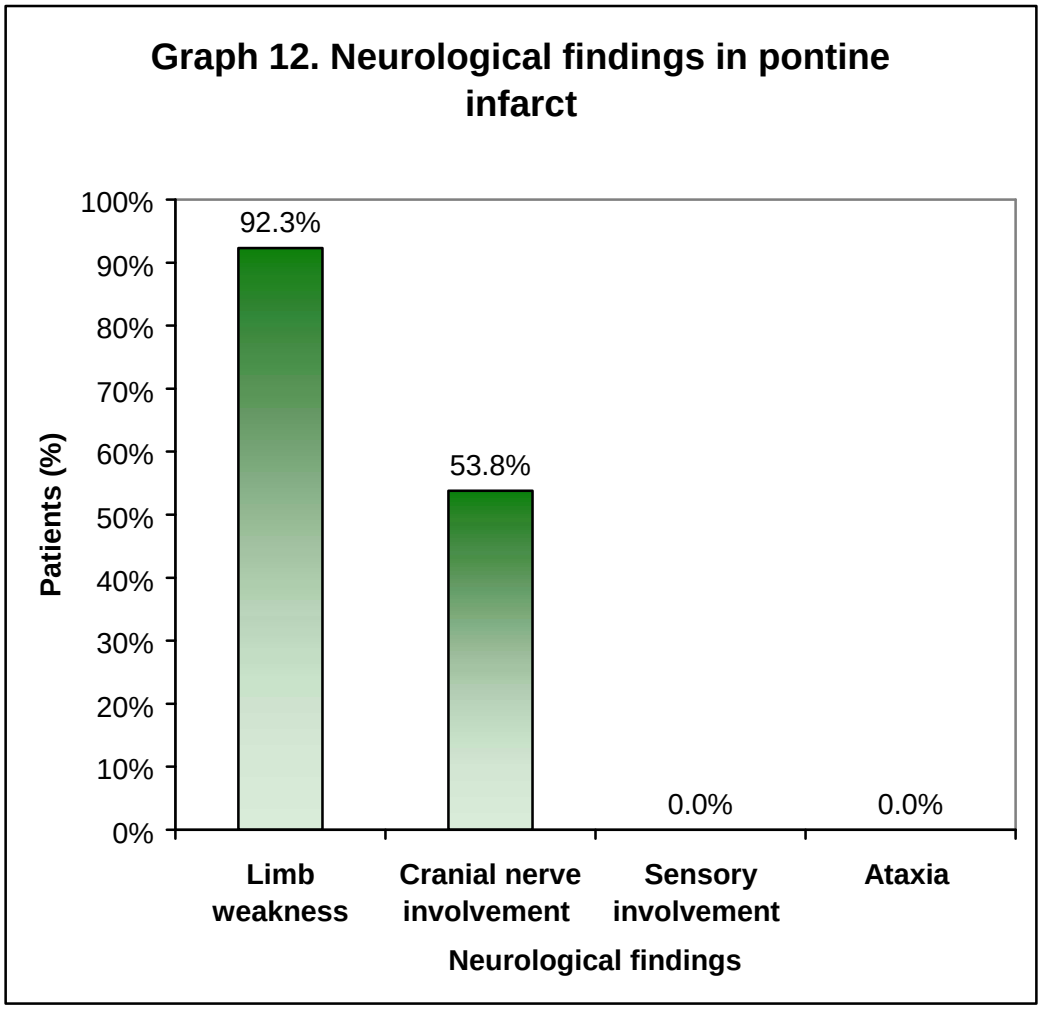


Vertigo (50%), eye movement disorders (50%), vomiting (33%) and speech disorders (33%) were the most common presenting clinical features. Altered sensorium and headache were seen in minority of the patients of midbrain infarct.

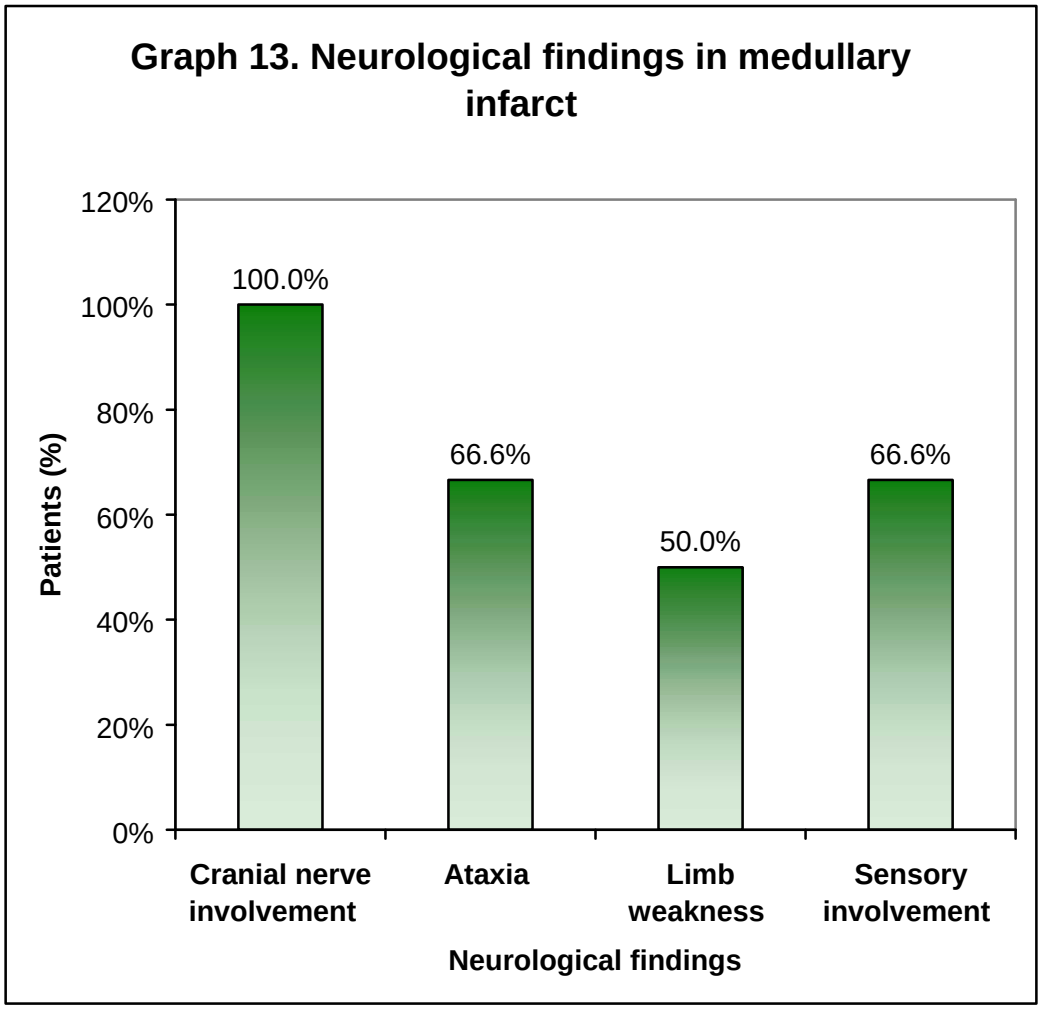
Table 11. Various clinical observations

Neurological findings	Involvement of one structure			More than one structure involved			
	Pontine infarct (n=13)	**Medullary infarct (n=6)**	**Midbrain infarct (n=6)**	**Midbrain and pontine infarct (n=3)**	**Midbrain and pontine hemorrhage (n=3)**	**Miscellaneous (n=2)**	**Total (n=33)**
Cranial nerve involvement	7 (53.8%)	6 (100%)	5 (83.3%)	3 (100%)	1 (33.3%)	1 (50%)	23 (69.6%)
Limb weakness	12 (92.3%)	3 (50%)	4 (66.6%)	3 (100%)	3 (100%)	2 (100%)	27 (81.8%)
Ataxia	0 (0%)	4 (66.6%)	3 (50%)	0 (0%)	0 (0%)	1 (50%)	8 (24.2%)
Sensory involvement	0 (0%)	4 (66.6%)	1 (16.6%)	0 (0%)	0 (0%)	1 (50%)	6 (18.2%)

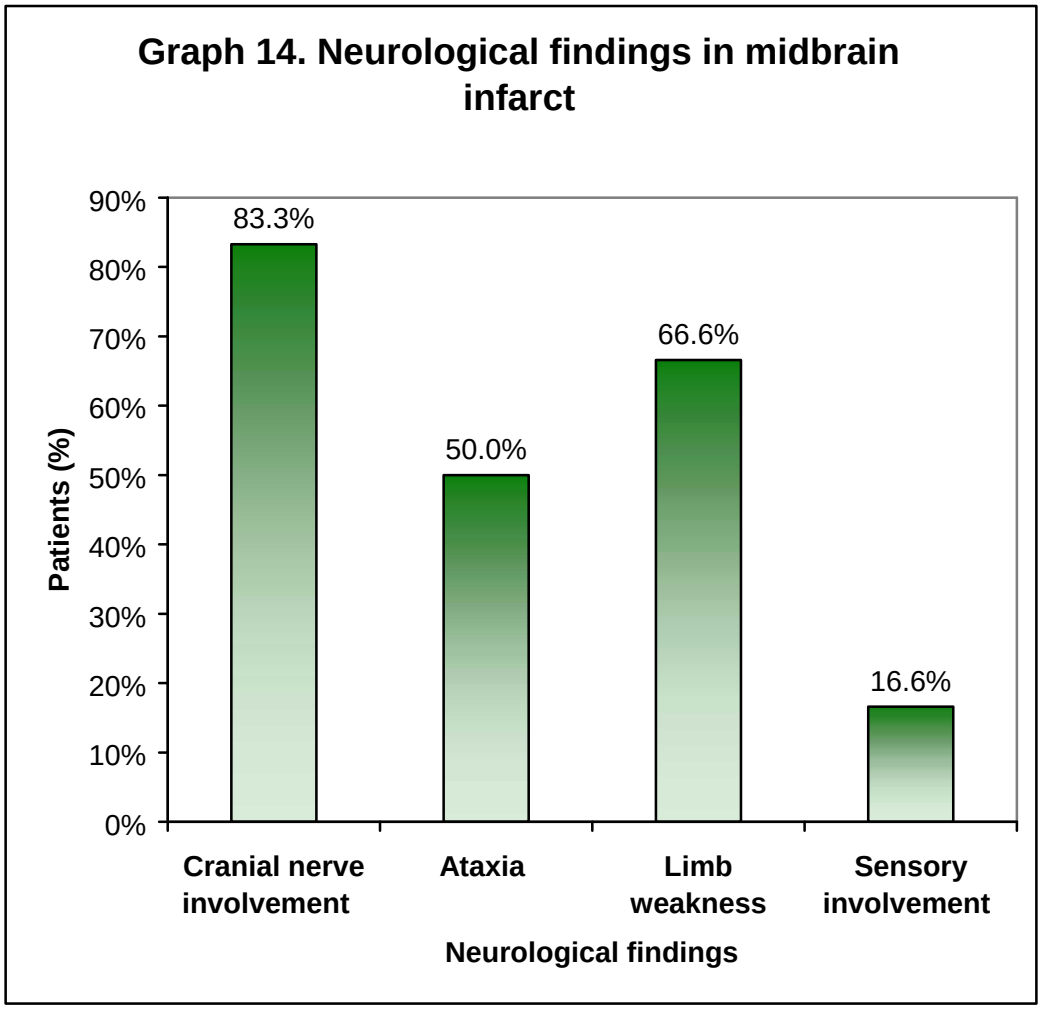
Various observations made are shown in above table with diagrammatic illustrations.



Limb weakness (92.3%) was the most common neurological findings in the patients of the pontine infarct followed by Cranial nerve involvement (53.8%).



Graph 13 shows that cranial nerve involvement (100%), limb weakness (50%), ataxia (66.6%) and sensory involvement (66.6%) were most commonly seen in the patients of medullary infarct.



Cranial nerve involvement (83.3%),limb weakness (66.6%), ataxia (50%) were the most common neurological findings in the patients of mid brain infarct, while sensory involvement (20%) was observed in minority of the patients.

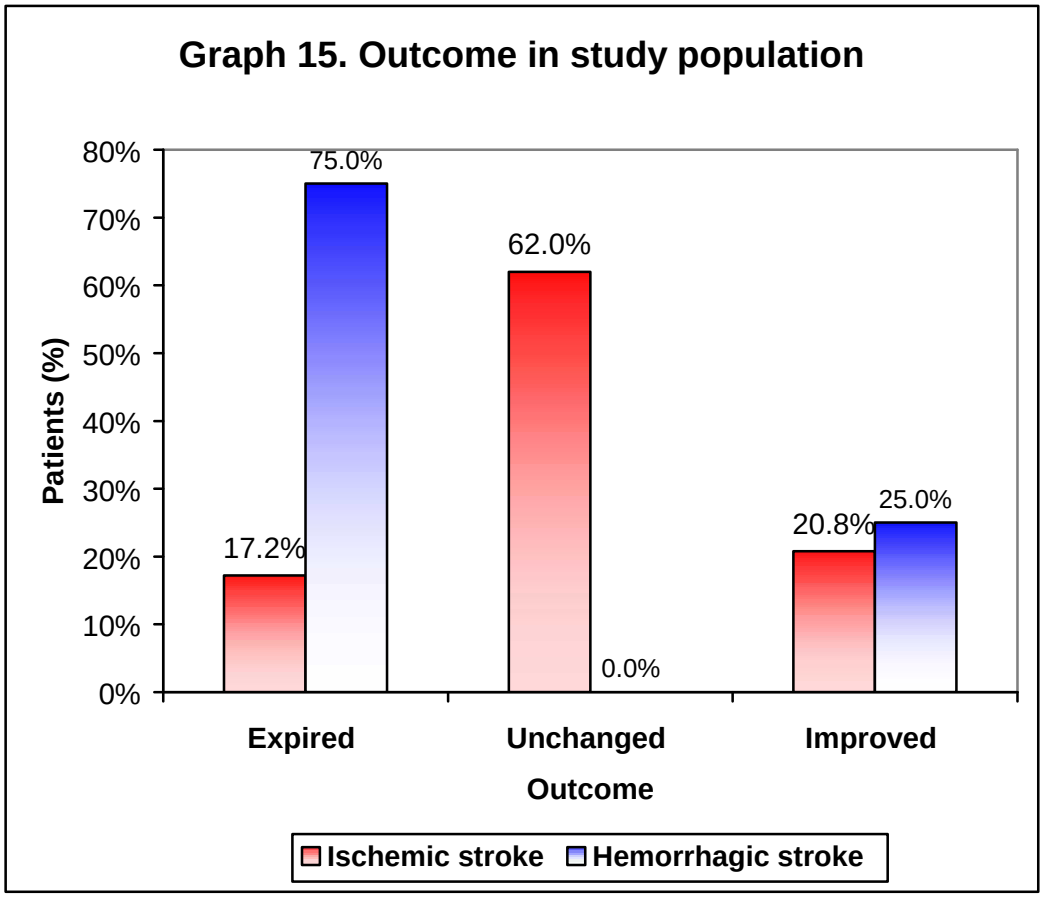
Table 12. Outcome in the study population in reference to GCS on admission

GCS	Total		Outcome					
			Improved		Unchanged		Expired	
	No.	%	No.	%	No.	%	No.	%
≤ 8	13	39.4	3	23.1	2	15.4	8	61.5
9 – 12	6	18.2	2	33.4	4	66.6	0	0.0
≥ 13	14	42.4	2	14.3	12	85.7	0	0.0
Total	33	100	7	21.2	18	54.5	8	24.2

Based on GCS score, we observed more mortality in patients of GCS score less than 8 as compared to GCS score more than 9. However, we had limitations to define morbidity because of majority patients belonged to GCS score of 13 to 15.

Table 13. Outcome in various study groups

Outcome	Outcome					
	Ischemic stroke (n=29)		Hemorrhagic stroke (n=4)		Total (n=33)	
	No.	%	No.	%	No.	%
Expired	5	17.2	3	75.0	8	24.5
Unchanged	18	62.0	0	0.0	18	54.5
Improved	6	20.8	1	25.0	7	21.2



Patients with hemorrhagic stroke as presentation mortality was more observed in comparison to ischemic stroke.

Table 14. Outcome based on lesions at various sites

Neurological findings	Involvement of one structure			More than one structure involved			
	Pontine infarct (n=13)	**Medullary infarct (n=6)**	**Midbrain infarct (n=6)**	**Midbrain and pontine infarct (n=3)**	**Midbrain and pontine hemorrhage (n=3)**	**Miscellaneous (n=2)**	**Total (n=33)**
Discharged	8 (61.6%)	6 (100%)	6 (100%)	3 (100%)	1 (33.4%)	1 (50%)	25 (75.5%)
Expired	5 (38.4%)	0 (0%)	0 (0%)	0 (0%)	2 (66.6%)	1 (50%)	8 (24.5%)
Total	13	6	6	3	3	2	33

Patients with pontine lesions alone had high mortality (38.4%) as compared to lesions in midbrain (0%) or medulla alone (0%). However combined lesions in midbrain and pons mortality were observed in two out of six patients, and one patient with lesion involving all three sites also died.

DISCUSSION

In the present study of 33 patients, it was observed that brainstem stroke was more common in the age group of 45 and above, that was between 45 to 54 (36.3%) and 55 to 64 (27.3%). This is in consistence with other studies who have the similar observations.[12,14,23,32]

With increasing age and associated risk factors, the risk of stroke would increase. This is similar to studies done by other workers.[12,14,23,32]

The proportion of strokes that are secondary to ischemia is high among elder persons. Atherosclerosis generally evolves over a person's lifetime; thus, it is the leading cause of ischemic stroke among elder persons.[22]

In the present study, it was observed that stroke was more common in males (63.5%) as compared to female (36.5%).

Overall, the annual age adjusted total initial completed stroke events rates were 5.89 per 1000 in men and 4.91 per 1000 in women, which also shows male preponderance.[32]

Gender differences in stroke incidence have been consistently demonstrated and indicate a greater risk of stroke among men.[23,47,48]

A study in Taiwan in 1992 and a study in Sweden in 1982 demonstrated that incidence of stroke was greater in men than women.[49,50]

It is well known that ischemic events occur with greater frequency in men than women. This pattern of stroke risk has been attributed to the presence of protective female sex hormones, although there is little evidence to support this hypothesis. Exogenous supplementation of estrogen to elderly men did not prevent them from risk of stroke; on the contrary they were with the increased risk of stroke. Same thing holds good with post menopausal women who were given hormone replacement therapy, did not benefit them from prevention of stroke.[51]

In the present study we have observed that the patients of brainstem stroke commonly present with clinical features like limb weakness (81.8%), cranial nerve involvement (69.6%), altered sensorium (58%), vomiting (51.5%), vertigo (48.4%), headache (30.3%), ataxia (24.2%) and dysarthria (27%).

A study in Switzerland (2002) had shown that the patients of basilar artery stenosis or occlusion presented with motor disorders (57%), consciousness disorders (52%), ataxia (52%), dysarthria (49%), vertigo (44%) and cranial nerve involvement(40%) , which is almost similar to our study.[47]

In another study, 39 patients with vertebrobasilar territory infarction the temporal profile revealed cranial nerve involvement (64%), altered sensorium (43.5%), weakness (38.4%), ataxia (28.2%), vertigo (28.2%), vomiting (28.2%) and dysarthria (25.6%).[48]

In a study in Switzerland (2003) had proved that basilar artery occlusion may give large spectrum of neurological features, of which the most frequent are

motor deficit (91.6%), dysarthria (62.5%), vertigo (54.1%), vomiting (54.1%) and altered sensorium (50%).[52]

Brainstem possesses several cranial nerve nuclei and serves as a conduit for important ascending and descending tracts. So involvement of brainstem will manifest as various clinical features depending on the structure involved.[53]

So as per our results cranial nerve involvement, altered sensorium, vomiting, vertigo, motor weakness and dysarthria were observed as manifestations of brainstem stroke.

The termporal profile of patients of our study was almost similar to the studies done by various other authors. This is because brainstem being a small structure contains important cranial nerve nuclei and ascending and descending tracts. Any smallest lesion would manifest with varied clinical manifestation.

In our study ischemic brainstem stroke was more common (87.8%) than hemorrhagic stroke (12.2%) which is in consistent with the incidence of intracerebral hemorrhagic stroke (10 to 20%).[32]

In a Korean study the frequency of primary intracerebral hemorrhage in the brainstem was 7.8%.[54]

In our study we observed 15 patients (45.45%) presented with classical well defined syndromes like Wallenberg syndrome (15.1%), Weber's syndrome (9.09%), Pure motor hemiparesis (3.03%) and other syndromes as shown in table 6.

These clinical syndromes in patients of brainstem stroke, when present with typical temporal profile leads the clinician to arrive at a clinical diagnosis easily. Later neuroimaging helps to confirm the clinical diagnosis and most of the times it correlates with the clinical observation.[44]

In our study we found the hypertension as the most common risk factor for the stroke accounting for 60.0%. This is almost similar to a study in a Kuwatian cohort of patients with vertebrobasilar territory infarct; hypertension was a commonest risk factor (55.0%).[55]

Another study in Australia on 116 patients of vertebrobasilar territory infarct, again hypertension was the commonest risk factor (62.0%).[56]

These suggest that hypertension is the most common well documented risk factor in the patients of brainstem stroke which is a modifiable risk factor and helps in prevention of stroke.[12]

Hypertension is associated with accelerated atherosclerosis, hypertensive heart disease and hypertensive cerebrovascular disease. This in turn leads to infarctions or hypertensive hemorrhage.[22]

Thus, hypertension scores the unique role of being prime risk factor for stroke.[32]

The other risk factors responsible for stroke observed were diabetes mellitus (24.2%), smoking (21.1%), Cardiac diseases (18.2%), dyslipidemia (15.1%), hyperhomocysteinemia (9.1%) and alcoholism (6.1%).

In a Kuwaitian study of cohorts of patients with vertebrobasilar territory infarct, diabetes mellitus was present in 35%, smoking in 30%, Cardiac diseases in 50%, dyslipidemia in 65% of patients.[55]

In other study of vertebrobasilar territory infarct, diabetes mellitus was present only in 11%, smoking in 41% and Cardiac diseases in 32% of patients.[56]

In an Indian study of 76 patients of posterior circulation stroke, diabetes mellitus was present in 21%, smoking in 35.5%, Cardiac diseases in 27.6%, alcoholism in 19.7% and dyslipidemia in 44.4% of patients.[4]

The risk of stroke with various other risk factors, slight difference was found in comparison with other workers. This may be because of varying sample size in the study population.

In the Framingham study, the impact of diabetes was much clearer. The study found that the stroke risk from diabetes in both men and women was independent of hypertension.[33]

Diabetes mellitus,[57] dyslipidemia[22] and tobacco use (chewing/smoking)[58] is associated with an increased risk of accelerated atherosclerosis, including coronary artery disease, and stroke. Similarly there is an association between the hyperhomocysteinemia and arterial/venous thrombosis, which is also associated with coronary artery disease and stroke.[59]

Cardiac disease may also independently increase the risk of stroke in patients of atrial fibrillation, rheumatic valvular heart disease, myocardial infarction and congestive cardiac failure.[12]

In our study six patients (18.2%) of various cardiac diseases had brain stem stroke.

The risk of stroke doubles with pre existing coronary artery disease and the risk may further increases with congestive heart failure.[60]

In our study hypertension was present as associated vascular risk factor in all the patients of hemorrhagic stroke (100%) while it was present in 55.2% of patients of ischemic stroke which is in consistent with other studies where hypertension was present in 76% of brainstem hemorrhage.[54]

We observed that patients presenting with combination of various symptoms were pointing the lesion to a particular site of brainstem.

In our study patients of pontine infarct (n=13) presented with major clinical features like limb weakness (92.3%), altered sensorium (61.5%), cranial nerve involvement (53.8%), vertigo (38.4%), headache (30.7%), vomiting (30.7%).

A study done in Japan (1985) on 26 patients of pontine infarct altered sensorium (76.0%), headache (38.4%), vomiting (38.4%), vertigo (26.9%) and weakness (34.6%) were common presenting clinical features.[61]

In this study, patients of medullary infarct (n=6) presented with vomiting (83.3%), vertigo (66.6%), sensory symptom (66.6%), ataxia (66.6%), dysphagia (66.6%) and limb weakness (50%).

In a South Korean study (2003) of 130 patients of pure lateral medullary infarction, vertigo (92%), sensory symptom (96%), ataxia (92%), dysphagia (65%) and Horner sign (88%) were common presenting symptoms/signs.[62]

Thus, clinical features such as vomiting, vertigo, sensory symptoms and ataxia were closely related and commonly found in patients of medullary infarct, suggesting that these are all caused by an involvement of the vestibular nuclei or vestibulo-cerebellar connection.[63]

We observed four patients of medullary infarct (66.6%) had well defined syndrome of Wallenberg, who had triad of Horner's sign, crossed pain, sensory loss and ataxia.

A study in Switzerland on 28 patients of lower brain stem infarct found features of Wallenberg syndrome in 86% of patients.[64]

It was our observation that patients of midbrain infarct (n=6, 18.2%) were presented with cranial nerve involvement (83.3%), limb weakness (66.6%), ataxia (50%), diplopia (50%), vertigo (50%), dysarthria (33.3%), sensory involvement (16.6%) and headache (16.6%).

This was similar to a study in England in 39 patients of midbrain infarct; observations were eye movement disorders (72%), limb weakness (62%), ataxia (56%) and sensory and visual field deficit (8%).[65]

In patients of midbrain infarct, the localizing neurological signs were involvement of 3rd nerve and its nucleus, red nucleus and fibers of cortico spinal tracts.[53]

In our observation, 66.6% of patients with midbrain infarction had 3rd nerve involvement, this helps in localizing lesion in patients of brainstem stroke.

In our study we made an attempt of knowing the outcome of patients with brainstem strokes and observations were 24.5% patients died, 54.5% remained status quo and 21.2% shown improvement.

This is almost similar to an Indian study, which also observed 18% mortality.[4]

In our study patients of hemorrhagic brainstem stroke were having higher mortality (75%) than the patients of ischemic brainstem stroke (17.2%), which is in consistent with the study done in Western Australia (1994) which had shown 100% mortality in brainstem hemorrhagic stroke.[66]

Higher mortality rate in hemorrhagic stroke may be because of inadequate methods of treatment and prevention of associated vascular risk factors.

We also made an attempt to prognosticate our patients based on GCS scoring system and observed that patients of GCS score ≤ 8 had poor outcome as compared to those who had score of ≥ 9.

The mortality was more observed in patients of pontine lesion as compared to medullary or midbrain lesions.

In an Indian study patients with basilar artery occlusion were associated with poor outcome (mortality / disability) and this was based on GCS scoring system.[4]

In Switzerland study, it was also shown that the presence of consciousness disorders alone was associated with poor outcome (mortality/severe disability) in 87% of the patients, which is also similar to our study.[47]

In patients of brainstem stroke, GCS scoring system helps to predict the outcome of patients. A low GCS score reflects poor outcome, which in turn reflects more severe neurological damage.

The present study made an attempt of determining the various presentations in patients of brainstem stroke with regards to various risk factors. Based on clinical observations and neuroimaging with application of GCS scoring system it was possible to predict the clinical outcome.

In the era of modern medicine with improved neuroimaging techniques and sound clinical knowledge of neuromedicine, it is possible to diagnose promptly and offer prompt treatment. However preventive measures are best to apply and if modifiable risk factors treated aggressively, it is possible to minimize the risk of stroke.

CONCLUSION

Among the patients presented with brainstem stroke 87.8% had ischemic brainstem stroke and 12.2% had hemorrhagic brainstem stroke.

Majority of patients of brainstem stroke (76%) had involvement of only one structure that is either midbrain, pons or medulla and the rest (24%) had involvement of more than one structure.

87.8% of patients had one or the other known risk factors for brainstem stroke. Amongst these, hypertension was the most common (60%) risk factor for the brainstem stroke.

Some patients had presentation with well defined syndromes depending on that it was easy to diagnose these strokes; further confirmed by neuroimaging technique.

Certain clinical features like vertigo, lower cranial nerve involvement and occulomotor nerve involvement were suggestive of brainstem involvement.

Patients with disturbed consciousness level and low GCS score on admission had poor outcome. Most of these patients belonged to hemorrhagic brainstem stroke.

SUMMARY

The present study was conducted to know the various clinical manifestations of brainstem stroke and an attempt was made to correlate anatomical site of lesion with neuroimaging. This study was conducted on 33 patients presenting with brainstem stroke to KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of Jan 09 to Dec 09.

Result of present study showed that patients presenting with brainstem stroke, 87.8% had ischemic brainstem stroke and 12.2% had hemorrhagic stroke.

Nearly half of patients presented with a classical well defined brainstem syndromes which was further confirmed on neuroimaging.

Among known risk factors hypertension was the commonest risk factor in both ischemic and hemorrhagic brainstem stroke.

The mortality was higher in hemorrhagic stroke compare to ischemic brainstem stroke. Patients with altered sensorium and lower GCS score on admission had poor outcome.

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ANNEXURE I - CONSENT FORM

CONSENT FOR PARTICIPATION IN RESEARCH

Mr./Mrs._____ we are requesting you to enroll yourself in study titled **"CLINICAL PROFILE OF PATIENTS WITH BRAINSTEM STROKE ADMITTED TO KLES DR PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE – A CROSS SECTIONAL STUDY"** conducted by **Dr **** *******, postgraduate student in MD GENERAL MEDICINE under the guidance of **Dr. **** ******* at J. N. Medical College, Belgaum.

You have been requested to participate in this research because your profile matches with the study group. All patients admitted with brainstem stroke can become participant of this study. During the study you will be asked some questions and you are supposed to answer to the best of your knowledge.

Your participation in the research is absolutely voluntary. Your decision to participate in the study or otherwise will not affect your relationship with Jawaharlal Nehru Medical College, Belgaum / KLES Dr Prabhakar Kore Hospital and Medical Research Centre, Belgaum. If you decide not to participate you are free to withdraw at any time.

The purpose of research is to find the various clinical presentations of brainstem stroke.

PROCEDURE INVOLVED

A detailed history taking, clinical examination, blood investigations and CT scan/MRI of brain will be done, which are not invasive procedures.

RISKS AND BENEFITS

There are no risks involved and benefits are many. The study helps to identify various clinical features of the disease and identify anatomical site. The results deduced at the end of study will help all similar patients admitted in the hospital.

ALTERNATIVES

Even if you decline to participate, there will not be any change in the line of your management or the relationship with your doctor. You will be told about all the new information that may affect your decision to participate in the study.

PRIVACY AND CONFIDENTIALITY

The only people to know that you are a research subject are the members of research team. No information about you or provided by you during the research will be disclosed to others without your written permission except:

1. In emergency to protect your rights and welfare.
2. If required by law.

FINANCIAL INCENTIVES FOR PARTICIPATION

You will not be paid any monetary benefits or free gifts for participation in the research. You will not be reimbursed for expenses.

AUTHORISATION TO PUBLISH RESULTS:

When the results of the research are published or discussed in a conference, no information will be displayed that would disclose your identity. Any information that is obtained in connection with this study and that can be identified with you will remain confidential.

CONSENT STATEMENT

I, the undersigned, have been explained in my own vernacular language about the study and my participation in the study is voluntary. If I want I can withdraw at any time. Also I have been given enough time to clear my doubts about the study and my rights as a study participant.

In case you have any questions related to the study you can contact Dr. ***** ****** (Phone No. ***** *****).

In case you have any questions about your rights as a study participant you can contact Dr. ***** *****.

Signature or the left thumb impression of the participant or legally authorized representative.

Participant's name_____ Signature_____

Witness name _____ Signature_____

Experimenter's name _____ Signature_____

Place _____ Date _____

ANNEXURE II – PROFOMA

CLINICAL PROFILE OF PATIENTS WITH BRAINSTEM STROKE ADMITTED TO KLES DR PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE – A CROSS SECTIONAL STUDY

Name of the patient

IP No.

Age

Sex

Date of Admission

Date of Discharge

Occupation

Address

HISTORY OF PRESENT ILLNESS

- Headache
- Seizure
- Vomiting
- Vertigo / Tinnitus / Syncope
- Altered Sensorium
- Loss of consciousness
- Speech disorder
- Visual disturbances
- Hearing loss

- Dysphagia / Nasal twang of voice / Nasal regurgitation.
- Weakness
- Involuntary movements
- Sensory Symptoms – tingling / numbness / pins & needles / Pain
- Bowel and bladder disturbances
- Fever

PAST HISTORY

- H/O STROKE
- HT
- DM
- Recent MI
- Any cardiac disease requiring warfarin therapy
- Bleeding / coagulation disorder

DRUG HISTORY

- H/O Thrombolysis
- H/O Anticoagulation therapy

FAMILY HISTORY

- H/O STROKE
- HT
- DM
- IHD

PERSONAL HISTORY

- Smoking
- Alcohol intake
- Tobacco chewing

GENERAL PHYSICAL EXAMINATION

Build & nourishment

Height

Weight

Scalp

Eyes

Nose

Oval Cavity & lips

Ears

Facies

Neck

Upper limbs

Chest & Abdomen

Lower limbs

Pigmentation

Lymphadenopathy

VITAL SIGNS

PR

BP

RR

Temperature

EXAMINATION OF CNS

HIGHER FUNCTIONS

1. Handedness

2. Level of Consciousness (GCS)
3. Orientation in time, place, person
4. Speech and language –
    - Comprehension
    - Fluency
    - Repetition
    - Naming
    - Reading & Writing
5. Memory –
    - Immediate
    - Recent
    - Remote
6. Intelligence
7. Delusions & hallucinations

CRANIAL NERVES Right Left

1st Olfactory N– Sense of smell

2nd optic N – Acquity of vision

Field of vision

Colour vision

Fundoscopy

3,4,6 Cranial Nerves – Diplopia

Squint

Nystagmus

Right Left

Ptosis

Eye movements

- Conjugate
- Individual eye ball movements

Pupils –

Size

Regularity

Reflexes

- Accommodation
- Direct light reflex
- Indirect light reflex

5th Trigeminal N

1) Sensory

   Ophthalmic

   Maxillary

   Mandibular

2) Motor

   Masseter

   Temporalis

   Pterygoids

Right Left

3) Reflexes

Corneal

Conjunctival

7th Facial N

A). Inspection

Forehead wrinkles

Closure of eyes

Bells sign

Epiphora

Exposure keratitis & conjunctivitis

Food sticking in vestibule of mouth

Flattening of nasolabial folds

B). Examination Proper

a) Frontal belly of occipito-frontalis

b) Procerious

c) Orbicularis oculi

d) Zygomaticus major, zygomaticus minor and levator angulioris

e) Orbicularis oris

f) Buccinator

g) Platysma

C). Taste Sensation - anterior 2/3rd of tongue

Right Left

8th – Auditory .N

1. Acuity of hearing (watch test)
2. Rinne's test
3. Weber's test

9th Glossopharyngeal .N

1. Taste on posterior 1/3rd of tongue
2. Pharyngeal reflex.

10th Vagues . N

Patate

Nasal regurgitation

Nasal twang of voice

Palatal elevation (Ah test)

Position of uvula

Larynx

Hoarseness of voice

11th Accesory N

- Sternocleidomastoid
- Trapezius

12th Hypoglossal .N

- Deviation of tongue on protrusion
- Power

MOTOR SYSTEM

	Right	Left

A). NUTRITION

1) Small muscles of hand

2) Forearm (above radial styloid process)

3) Upper arm (above medial epicondyle)

4) Leg. (above medial malleolus)

5) Thigh (above adductor tubercles)

B). TONE

1) Wrist

2) Elbow

3) Knee

4) Ankle

C). Power

Shoulder joint	Flexion	
	Extension	
	Adduction	
	Abduction	
Elbow joint	Flexors	
	Extensors	
Wrist joint	Flexors	
	Extensors	
Hand grip		

		Right	Left
Hip joint	Flexion		
	Extension		
	Adduction		
	Abduction		
Knee joint	Flexion		
	Extension		
Ankle joint	Plantar flexion		
	Dorsi flexion		

D). CO-ORDINATION

E). ABNORMAL MOVEMENTS

REFLEXES

Superficial

Corneal

Conjunctival

Palatal

Abdominal

Cremasteric

Plantar

Deep

Jawjerk

Biceps

Triceps

Supinator

	Right	Left
Knee		
Ankle		
Patellar clonus		
Ankle clonus		

Co-Ordination

Upper limb

- Finger nose test
- Dysdiadochokinesia
- Fine movements

Lower limb

- Heel knee test
- Tandem walking

GAIT

SENSORY SYSTEM

- Touch - Fine
  Crude
- Pain - Superficial
  Deep
- Temperature - Hot
  Cold
- Post. column Sensation -Vibration
  - Joint position sense
  - Rombergs Sign

	Right	Left

- Cortical sensations

  Tactile localisation

  2 point discrimination

  Stereognosis

  Graphesthesia

  Cerebral inattention

CEREBELLAR SIGNS

Nystagmus

Speech

Hypotonia

Dysmetria

Dysdiadochokinesa

Rebound phenomenon

Intentional tremor

Pendular knee jerk

Drunken gait

Miscellaneous

1) Skull & spine
2) Signs of meningeal irritation
    1. Neck rigidity
    2. Kernig's sign
    3. Brudzinski's neck sign
    4. Brudzinski's leg sign

3) Neurocutaneous markers

    1. Vascular naevi
    2. Neurofibromata
    3. Low hair line
    4. Short neck

4) Carotid artery pulsations

OTHER SYSTEMS

CARDIOVASCULAR SYSTEM

RESPIRATORY SYSTEM

PER ABDOMEN

INVESTIGATIONS

HB

TC

DC

RBS

Blood urea

S. creatinine

ECG

CT/MRI

PROVISIONAL DIAGNOSIS

ANATOMICAL

PATHOLOGICAL

ETIOLOGICAL

FINAL DIAGNOSIS

ANATOMICAL

PATHOLOGICAL

ETIOLOGICAL

MASTER CHART

				Presenting complaints										GCS Score		Associated Risk Factors				Clinical Observations								Vitals		
Sr. No.	IP. No.	Age (Years)	Sex	Headache	vomiting	Vertigo	Seizures	Visual disturbances	Speech disorders	Dysphagia	Sensory symptoms	Altered sensorium	On Admission	At discharge	Hypertension	Diabetes mellitus	Dyslipidemia	Others	Nystagmus	Cranial nerve involvement	weakness	Sensory involvement	Ataxia	Incoordination	Syndrome	Pulse rate (/min)	Temperature	Haemoblogin (gm%)
1	4E+05	75	F	N	Y	Y	N	N	N	N	N	Y	E1V1M1=3	-	Y	N	N	N	N	N	QP	N	N	N	N	NR	AB	12.7
2	4E+05	45	ML	N	Y	N	GTC	N	N	N	N	Y	E1V1M1=3	-	Y	N	Y	N	N	N	QP	N	N	N	N	NR	AB	16.9
3	3E+05	67	ML	N	N	N	N	N	DS	N	N	N	E4V2M6=12	12	N	N	N	N	N	N	MN RT UL	N	N	N	N	NR	AB	12.0
4	3E+05	65	ML	N	N	Y	N	N	N	N	N	Y	E3V4M6=13	14	N	N	N	N	N	RT7	HM RT	N	N	N	N	NR	AB	15.8
5	3E+05	35	ML	N	N	N	N	N	DS	N	N	Y	E2V1M3=6	10-10	N	N	N	RHD, MS	N	N	QP	N	N	N	N	NR	AB	13.8
6	3E+05	40	F	Y	N	Y	N	N	N	N	N	Y	E2V1M2=5	-	N	N	Y	N	N	N	QP	N	N	N	N	NR	AB	12.6
7	3E+05	48	ML	N	N	N	N	N	N	N	N	Y	E1V1M2=4	-	N	N	N	N	N	3,4,6	QP	N	N	N	N	NR	AB	13.2
8	3E+05	59	ML	Y	Y	Y	N	N	DS	N	N	N	E4V6M5=15	15	Y	N	N	AL, TB	N	RT6, RT7	HM LT	N	N	N	MGS	NR	AB	12.4
9	3E+05	52	ML	N	N	N	GTC	N	N	N	N	Y	E3V0M2=5	-	Y	Y	N	N	N	3,4,6	QP	N	N	N	N	NR	AB	14.4
10	3E+05	62	F	N	N	N	N	N	N	N	N	N	E4V5M6=15	15	Y	Y	N	N	N	RT7	HM RT	N	N	N	PMH	NR	AB	13.0
11	3E+05	52	F	Y	N	N	N	N	N	N	N	N	E4V5M6=15	15	N	N	N	N	N	LT6	HM RT	N	N	N	RAYS	NR	AB	10.4
12	4E+05	60	ML	N	N	N	N	DP	N	N	N	N	E4V5M6=15	15	N	Y	Y	N	N	3,4,6	N	N	N	N	OHS	NR	AB	9.0
13	3E+05	50	ML	Y	Y	Y	N	N	N	N	N	Y	E1V1M2=4	3-10	Y	N	N	SM	N	N	QP	N	N	N	N	NR	AB	15.5
14	4E+05	62	ML	N	N	N	N	N	N	N	N	Y	E2M5V2=9	13	N	N	N	AL, HYPHC	N	RT3	HM LT	N	N	N	WBS	NR	AB	12.1
15	3E+05	48	ML	N	Y	Y	N	DP	DS	N	N	N	E4V5M6=15	15	Y	Y	N	LVDYS	N	LT6	HM	LT T,PA,TP	LT	LT	TBA	NR	AB	10.6
16	3E+05	22	F	N	N	N	N	DP	N	N	N	N	E4V5M6=15	15	N	N	Y	HYPHC	N	LT3	N	N	LT	BL	CS	NR	AB	10.9
17	3E+05	52	ML	N	N	Y	N	DP	N	N	N	N	E2V5M6=13	15	Y	N	N	TB	N	LT3	N	N	Y	N	WBS	NR	AB	15.7
18	3E+05	55	ML	Y	Y	Y	N	N	DS	N	N	N	E4V5M6=15	15	Y	Y	N	TB	N	RT3	HM LT	N	N	N	WBS	NR	AB	14.5
19	3E+05	50	F	N	N	N	N	N	DS	Y	Y	N	E4V5M6=15	15	Y	N	N	N	N	RT HRN, RT5, RT7,RT9,RT10	N	LT PA,TP	RT	RT	RT LMS	NR	AB	13.4
20	3E+05	60	F	N	Y	Y	N	N	N	N	Y	N	E4V5M6=15	15	Y	y	Y	N	N	RT HRN, RT5, RT7,RT9,RT10	N	LT PA,TP	Y	N	RT LMS	NR	AB	10.4
21	3E+05	66	ML	N	Y	Y	N	N	DS	N	N	N	E4V5M6=15	15	Y	Y	N	SM, IHD	HZ, VT	LT12, LT7	HM LT	N	N	N	MMS	NR	AB	15.4
22	3E+05	50	ML	N	Y	Y	N	N	DS	Y	Y	N	E4V2M6=12	13	Y	N	N	AF	HZ	LT HRN, LT9,LT10,LT12	HM LT	RT PA,TP	Y	LT UL	LT LMS	NR	AB	17.6
23	3E+05	90	ML	Y	Y	N	N	N	N	N	N	Y	E3V2M4=9	-	Y	N	N	N	N	RT7	HM RT	N	N	N	N	NR	AB	15.7
24	3E+05	54	ML	N	Y	Y	N	N	N	N	Y	N	E4V5M6=15	15	Y	N	N	N	N	RT HRN, RT9,RT10	N	LT PA,TP	RT	RT	RT LMS	NR	AB	15.0
25	3E+05	35	F	N	Y	Y	N	N	N	N	N	Y	E1M2V1=4	6	N	N	N	MVP	N	3,4,6	QP	N	N	N	N	NR	AB	12.8
26	3E+05	45	ML	N	N	Y	N	N	N	N	N	Y	E2V2M4=8	12	N	N	N	HYPHC	N	BL7	QP	N	N	N	N	NR	AB	14.1
27	3E+05	55	ML	N	N	Y	N	N	N	N	N	Y	E2V4M5=11	10	N	N	N	N	N	3,4,6,BL7	HM LT	N	N	N	N	NR	AB	15.5
28	4E+05	80	F	Y	Y	N	N	N	N	N	N	Y	E1V1M1=3	10	Y	N	N	IHD	N	N	QP	N	N	N	N	NR	AB	15.1
29	3E+05	67	F	Y	Y	N	N	N	N	N	N	Y	E1V1M5=7	-	Y	Y	N	N	N	3,4,6	QP	N	N	N	N	NR	AB	13.0
30	3E+05	48	ML	Y	Y	N	N	N	N	N	N	Y	E1V1M1=3	-	Y	N	N	TB	N	N	QP	N	N	N	N	NR	AB	16.5
31	3E+05	64	F	N	Y	N	N	N	N	N	N	Y	E2V1M1=4	-	Y	N	N	N	N	N	QP	N	N	N	N	NR	AB	16.0
32	3E+05	65	F	Y	Y	Y	N	N	DS	N	N	Y	E4V5M6=15	15	y	N	N	SM	HZ	RT HRN, RT5,RT9,RT10	HM RT	LT PA,TP	Y	RT	RT LMS	NR	AB	13.9
33	3E+05	59	ML	N	N	N	GTC	N	N	N	N	Y	E3V4M4=11	11	N	N	N	N	N	N	QP	N	N	N	N	NR	AB	11.2

MASTER CHART

Sr. No.	IP. No.	Investigations																	Diagnosis	Outcome
		Total count (/mm3)	Differential count (%)	RBS (mg/dL)	Blood Urea (mg/dL)	Sr. Creat (mg/dL)	HsCRP	Sr. Na+ (meq/L)	Sr. K+ (meq/L)	Sr. Bicarb (meq/L)	Sr.Cl- (meq/L)	Sr. Mgn (mg/dL)	Sr. Ca. (mg/dL)	Sr. Chol. (mg/dL)	Homocyst (umol/L)	ECG	Echo	MRI		
1	4E+05	####	P84LO10EO1MO5	105	17	0.5	7.1	148	3.5	18	108	1.8	8.5	213	-	WNL	-	BL PTN INF	BL PTN INF with HTN	EX
2	4E+05	####	P86LO13EO0MO1	116	19	1.0	5.6	144	5.0	19	105	1.8	9.2	212	-	WNL	-	PTN INF	PTN INF with HTN with DSPD	EX
3	3E+05	8100	P71LO26EO0MO3	99	37	1.0	4.3	142	4.2	29	139	2.0	8.5	-	-	WNL	-	Lt PTN INF	PTN INF	DC
4	3E+05	####	P89LO6EO0MO5	106	43	1.0	-	143	3.7	26	102	2.0	8.9	-	-	WNL	-	Lt PTN INF	PTN INF	DC
5	3E+05	####	P88LO8EO0MO4	221	16	1.0	-	141	4.5	24	110	1.7	9.0	-	-	RBBB	Sev MS with PAH	PTN INF	PTN INF with RHD	DC
6	3E+05	####	P93LO6EO0MO1	157	16	0.9	-	146	4.0	25	116	2.0	9.0	219	-	WNL	-	PTN INF	PTN INF with DSPD	EX
7	3E+05	####	P92LO7EO0MO1	131	13	1.1	-	133	3.9	24	105	-	-	110	-	WNL	-	PTN INF	PTN INF	EX
8	3E+05	####	P56LO34EO2MO6	119	30	1.1	2.8	140	3.8	21	111	-	-	193	-	WNL	-	RT PTN INF	PTN INF with CHR AL	DC
9	3E+05	####	P77LO16EO0MO7	298	27	1.1	14.1	139	3.3	20	112	-	-	-	-	WNL	-	PTN INF	PTN INF with HTN with DM	EX
10	3E+05	####	P90LO9EO0MO1	287	22	0.8	3.0	125	4.7	25	91	1.9	9.3	224	-	RBBB	CONC LVH	Lt PTN INF	PTN INF with HTN with DM with CONC LVH	DC
11	3E+05	####	P87LO9EO0MO4	107	27	0.6	-	140	2.7	24	97	-	-	-	-	WNL	-	Lt PTN INF	PTN INF	DC
12	4E+05	7800	P62LO30EO0MO8	231	27	1.4	9.1	134	4.8	21	101	-	-	156	-	WNL	-	PTN INF	PTN INF with DSPD with DM	DC
13	3E+05	####	P85LO10EO0MO5	223	29	0.9	-	146	4.0	24	108	-	-	-	-	WNL	-	PTN INF	PTN INF with SM with CHR AL	DC
14	4E+05	7600	P50LO38EO0MO8	87	21	0.6	-	138	4.7	26	108	-	-	-	49.0	WNL	-	MD INF	MD INF with HYPHC with CHR AL	DC
15	3E+05	6200	P49LO43EO1MO7	259	14	1.2	5.6	142	4.7	24	106	-	-	116	-	WNL	Sev LV DYS	MD INF	MD INF with HTN with DM with cardiomyopathy	DC
16	3E+05	5900	P72LO23EO0MO5	102	12	0.6	-	140	3.7	27	110	-	7.9	120	33.8	WNL	-	MD INF	MD INF with HYPHC with DM	DC
17	3E+05	####	P86LO9EO0MO5	152	40	1.1	12.2	133	3.6	25	102	-	-	193	-	WNL	-	MD INF	MD INF with HTN	DC
18	3E+05	####	P66LO22EO2MO10	128	17	0.7	35.8	138	4.0	24	102	-	-	144	-	WNL	-	MD INF	MD INF with HTN with DM	DC
19	3E+05	7800	P66LO22EO2MO10	247	48	0.7	5.8	147	3.5	23	105	-	-	143	13.7	WNL	-	RT MED INF	MED INF with HTN	DC
20	3E+05	####	P82LO13EO0MO5	123	41	0.6	36.5	136	4.0	25	112	-	-	225	-	WNL	-	RT MED INF	MED INF with HTN with DM with DSPD	DC
21	3E+05	####	P85LO11EO0MO4	263	20	1.0	5.8	135	3.9	22	112	-	-	254	-	Old MI	Hypo Inf Wall	RT MED INF	MED INF HTN with DM with IHD	DC
22	3E+05	7900	P58LO30EO3MO9	140	17	0.9	-	140	4.1	26	104	-	-	175	-	VEN ECT	CONC LVH	LT MED INF	MED INF HTN with AF	DC
23	3E+05	####	P78LO18EO2MO2	164	56	1.0	-	144	3.4	24	106	-	-	-	-	WNL	-	RT MED INF	MED INF with HTN	DC
24	3E+05	####	P87LO7EO0MO6	129	21	1.0	-	133	4.2	25	93	-	-	204	-	WNL	-	RT MED INF	MED INF with HTN	DC
25	3E+05	####	P82LO13EO0MO5	101	26	0.8	-	144	4.2	21	113	-	-	-	9.0	WNL	MVP GR-2	MD+PTN INF	MD & PTN INF with MVP Gr-2	DC
26	3E+05	####	P69LO24EO0MO7	97	24	1.3	-	145	4.1	28	104	-	-	221	42.2	WNL	-	MD+PTN INF	MD & PTN INF with HYPHC	DC
27	3E+05	####	P86LO9EO0MO5	97	19	0.7	21.1	141	4.0	23	120	-	-	129	-	WNL	-	MD+PTN INF	MD & PTN INF	DC
28	4E+05	####	P88LO12EO0MO0	141	19	0.6	4.6	138	3.7	22	109	1.7	9.4	183	-	INV T	-	MD+PTN BLD	MD & PTN HMRG with HTN with IHD	DC
29	3E+05	####	P91LO6EO1MO2	123	21	0.7	4.6	140	4.3	21	110	-	-	133	-	WNL	-	MD+PTN BLD	MD & PTN HMRG with HTN with DM	EX
30	3E+05	####	P50LO38EO0MO8	108	53	3.7	-	143	3.1	27	131	-	-	-	-	WNL	-	MD+PTN BLD	MD & PTN HMRG with HTN	EX
31	3E+05	####	P89LO6EO0MO5	132	15	0.8	-	139	3.6	22	102	-	-	-	-	WNL	-	MD+PONS+MED BLD	MD & PTN & MED HMRG with HTN	EX
32	3E+05	####	P74LO18EO2MO6	81	15	0.5	-	135	4.3	22	117	-	-	168	-	WNL	-	PONTO-MED INF	PTN & MED INF with HTN	DC
33	3E+05	7800	P86LO6EO0MO8	200	18	0.5	-	140	4.4	25	103	-	-	-	-	WNL	-	MD INF	MD INF	DC

age				
	75	62	50	f
	45	48	60	F
	67	22	66	F
	65	52	50	F
	35	55	90	F
	40		54	F
	48			F
	59			F
	52			F
	62			F
	52			F
	60			F
	50			M
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35	80
45	67
55	48

64
65
59

ANNEXURE III - KEY TO MASTER CHART

%	-	Percentage
&	-	And
AB	-	Afebrile
AF	-	Atrial fibrillation
AL	-	Alcoholic
Bicarb	-	Bicarbonate
BL	-	Bilateral
BLD	-	Bleeding
Ca	-	Calcium
Chol	-	Cholesterol
CHR	-	Chronic
Cl^-	-	Chloride
Conc	-	Concentric
Creat	-	Creatinine
CS	-	Claude syndrome
DC	-	Discharged
dL	-	Deci litre
DM	-	Diabetes mellitus
DP	-	Diplopia
DS	-	Dysarthria
DSPD	-	Dyslipidemia
E	-	Eye

ECG	-	Electrocardiogram
Echo	-	Echocardiography
EO	-	Eosinophil
EX	-	Expired
F	-	Female
GCS	-	Glasgow Coma Scale
Gm	-	Gram
Gr	-	Grade
GTC	-	Generalized tonic clonic seizures
HM	-	Hemiparesis
HMRG	-	Hemorrhage
Homocyst	-	Homocysteine
HRN	-	Horner sign
HsCRP	-	High sensitive C- reactive p rotein
HTN	-	Hypertension
HYPHC	-	Hyperhomocysteinemia
Hypo inf	-	Hypokinesia of inferior
HZ	-	Horizontal
IHD	-	Ischemic heart disease
Inf	-	Infarction
Inv T	-	Inverted t waves
IP No	-	Indoor patient number
K^+	-	Potassium
L	-	Litre
LMS	-	Lateral medullary syndrome

LO	-	Lymphocyte
LT	-	Left
LVDYS	-	Left ventricular dysfunction
LVH	-	Left ventricular hypertrophy
M	-	Motor
MD	-	Midbrain
MED	-	Medullary
Meq	-	Milli equivalent
Mg	-	Milligram
Mgn	-	Magnesium
MGS	-	Millard Gubler syndrome
MI	-	Myocardial infarction
Min	-	Minute
ML	-	Male
mm^3	-	Cubic millimeter
MMS	-	Medial medullary syndrome
MN	-	Monoparesis
MO	-	Monocyte
MRI	-	Magnetic resonance imaging
MS	-	Mitral stenosis
MVP	-	Mitral valve prolapsed
N	-	Not present
Na^+	-	Sodium
NR	-	Normal
OHS	-	One and half syndrome
PA	-	Pain

PAH	-	Pulmonary artery hypertension
PMH	-	Pure motor hemiparesis
Po	-	Polymorhonuclear
PTN	-	Pontine
QP	-	Quadriparesis
RAYS	-	Raymond syndrome
RBBB	-	Right bundle branch block
RHD	-	Rheumatic heart disease
RT	-	Right
Sev	-	Severe
SM	-	Smoking
Sr. No.	-	Serial number
Sr.	-	Serum
T	-	Touch
TB	-	Tobacco
TBA	-	Top of basilar artery syndrome
TP	-	Temperature
UL	-	Upper limb
V	-	Verbal
VEN ECT	-	Ventricular ectopics
VT	-	Vertical
WBS	-	Weber’s syndrome
WNL	-	Within normal limits
Y	-	Present