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Characteristics of CT Findings in Patients of Acute Stroke and Their Relationship to Mortality

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University of Rajshahi

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CHARACTERISTICS OF CT FINDINGS IN PATIENTS OF ACUTE STROKE AND THEIR RELATIONSHIP TO MORTALITY

THESIS SUBMITTED FOR THE DEGREE
OF
DOCTOR OF PHILOSOPHY
IN THE
INSTITUTE OF BIOLOGICAL SCIENCES
UNIVERSITY OF RAJSHAHI, RAJSHAHI-6205
BANGLADESH

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BANGLADESH

TITLE

CHARACTERISTICS OF CT FINDINGS IN PATIENTS OF ACUTE STROKE AND THEIR RELATIONSHIP TO MORTALITY

DECLARATION

I, hereby, declare that, the research work as a thesis entitled,

"Characteristics of CT Findings in Patients of Acute Stroke and Their Relationship to Mortality" submitted in the Institute of Biological Sciences, University of Rajshahi, Bangladesh for the degree of **Doctor of Philosophy (PhD)** is the result of original research work carried out under the supervision of Professor Dr. Niranjan Kumar Sana, Department of Biochemistry and Molecular Biology, University of Rajshahi, Rajshahi, Bangladesh and Professor Dr. Quamruddin Ahmad, Professor and Head, Department of Neurology, Rajshahi Medical College, Rajshahi, Bangladesh.

I, further, declare that the thesis or part of the thesis has not been concurrently submitted elsewhere for any degree of diploma and the thesis contains no materials previously published of written by another person, except where due references are made in the text.

(Md. Durrul Huda)

CERTIFICATE

This is to certify that **Md. Durrul Huda** is the author of the thesis entitled **"Characteristics of CT Findings in Patients of Acute Stroke and Their Relationship to Mortality"** submitted to the Institute of Biological Sciences, University of Rajshahi, Bangladesh for the degree of **Doctor of Philosophy (PhD).** He worked under our supervision.

To the best of our knowledge, the thesis has not been previously submitted elsewhere for any degree or diploma.

We are forwarding this thesis for the examination/evaluation for the degree of Doctor of Philosophy awarded by the University of Rajshahi, Bangladesh.

Md. Durrul Huda has fulfilled all the requirements according rules of the University for the submission of a thesis for PhD degree.

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DEDICATED TO

My parents without their inspiration
I couldn't reach at this level and my patients
without their co-operation I couldn't have
completed this research work.

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The Author

ABSTRACT

Stroke is one of the leading causes of death after heart disease and cancer in the developed countries and one of the leading causes of death in Bangladesh.

The aim of this study was to assess the CT findings of acute stroke patients and their relationship with mortality. This study also evaluates the risk factors of stroke and thus help in awareness building in reducing stroke. This study will provide a basis for statistical studies on the prognostic factors of intracerebral haemorrhage / infarct for future studies.

In the present study, a total number of 321 CT diagnosis stroke patients were selected from different clinics of Rajshahi and the department of Radiology and Imaging, Rajshahi Medical College, Rajshahi, Bangladesh in collaboration with department of Neuromedicine of Rajshahi Medical College, Rajshahi from January 2012 to December 2015. Diverse clinical presentation of stroke, presence of various risk factors, CT findings, type of stroke and correlation between Computed Tomography Scan findings with mortality within 7 days & 28 days were studied. The main objective of this study was to correlate the CT findings of acute stroke and their correlation with mortality. The result of this study was compared with the results of similar kind of studies carried out both at home and abroad.

CT provides an excellent imaging modality for early detection of infarct which helps to improve patient management. In the present study, an analysis of 321 patients with spontaneous intracranial hemorrhage and ischaemic stroke was done. This study concluded the following aspects:

Incidence of non traumatic spontaneous intracranial hemorrhage and ischaemic stroke was highest around 61 to 70 years of age.

Sex incidence of stroke was high in male and male to female ratio was 60.12:39.88.

Hypertension was the major causative factor for stroke and it was in 225(70.09%) patients.

Intraparenchymal hemorrhage was the commonest variety of non traumatic spontaneous intracranial hemorrhage i.e. more than five times of subarachnoid hemorrhage.

Basal ganglia were the commonest location of intraparenchymal hemorrhage.

Among the patients with intraparenchymal hemorrhage, 44.44% patient died within 28 days.

The predominant neurological defect was hemiparesis/hemiplegia (91.3%) which was found among 66(91.7%) of CT diagnosed ischemic cases and 28(90.3%) haemorrhagic cases. Headache and neck rigidity were more commonly associated with haemorrhagic stroke.

Primary intracerebral haemorrhage was detected in 99(88.39%) cases and subarachnoid haemorrhage in 13 (11.61%) cases.

The commonest site 62(29.67%) of infraction was basal ganglia and paraventricular region followed by parietal region 55(26.32%).

Middle cerebral artery was involved in 159(76.08%) of cases of infarct.

CT scan detected infarcts < 2 cm in 78(37.32%), 2-5 cm in 106(50.72%) and >5 cm in 25(11.96%) patients.

Mass effect was found more in haemorrhagic stroke than in ischaemic stroke.

The significant predictors of 28 days mortality were intraventricular hemorrhage, midline shift, hydrocephalus, subarachnoid extension of bleed and volume of hematoma 61 ml and above and infarct diameter.

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LIST OF ABBREVIATION

ACA	Anterior cerebral artery.
ADC	Apparent diffusion co-efficient.
AIS	Acute ischemic stroke.
ASVD	Atherosclerosis vascular disease.
BBB	Blood brain barrier.
BIRDEM	Bangladesh Institute of Research and Rehabilitation of Diabetes, Endocrine and Metabolic disorder.
CAT	Computerized axial tomography.
CSF	Cerebro spinal fluid.
CT	Computed tomography.
DM	Diabetes Mellitus.
DIC	Disseminated intravascular coagulation.
DMRT	Duncan's multiple range test.
EEG	Electroencephalogram.
FLAIR	Fluid-attenuated inversion recovery.
GCS	Glasgow coma scale.
GRE	Gradient recalled echo.
HTN	Hypertension.
HU	Hounsfield unit.
ICH	Intracerebral haemorrhage.
IHD	Ischaemic heart disease.
IPGMR	Institute of Post Graduate Medicine and Research.
IPH	Intraparenchymal haemorrhage.
IVH	Intraventricular haemorrhage.
MCA	Middle cerebral artery.
MMCH	Mymensingh Medical College Hospital.
MRI	Magnetic resonance imaging.
MR	Magnetic resonance.

NCCT	Noncontrast computed tomography.
OR	Odds ratio
PCA	Posterior cerebral artery.
PET	Positron emission tomography.
PICH	Primary intracerebral haemorrhage.
RMCH	Rajshahi Medical College Hospital.
ROI	Region of interest.
SAH	Subarachnoid haemorrhage.
SICH	Spontaneous intracerebral haemorrhage.
SPECT	Single photon emission computed tomography.
TIA	Transient ischaemic attack.
tPA	Tissue plasminogen activator.
VBA	Vertebrobasilar artery.
WHO	World health organization.

CHAPTER-1

INTRODUCTION

1.1. INTRODUCTION

Stroke is a clinical entity characterized by an acute focal, irreversible neurologic deficit that is not caused by direct physical trauma. It is defined as "A neurological deficit of sudden onset; with focal rather than global neurological dysfunction, with symptoms lasting more than 24 hours or resulting in death before 24 hours; and in which, after adequate investigation, symptoms are presumed to be a non traumatic vascular origin." (Bamford 1992).

Cerebrovascular accident or stroke is defined as an acute loss of focal and at times global (applied to patients in deep coma and those with subarachnoid haemorrhage) cerebral function, the symptoms lasting more than 24 hours or leading to death with no apparent cause other than that of vascular origin (Hatano 1976).

Stroke syndromes have significant clinical and pathological heterogeneity that is reflected in their underlying gross pathologic and imaging appearance. Arterial ischemia/infarction is by far the most common cause of stroke, accounting for 80% of all cases. The remaining 20% are mostly hemorrhagic, divided between primary spontaneous intracranial hemorrhage (sICH), nontraumatic subarachnoid hemorrhage (SAH), and venous occlusions (Osborn 2013).

The main types of stroke occur for the first time in the community in the following proportions: Cerebral infarction- 85%, Primary intracerebral haemorrhage- 10% and subarachnoid haemorrhage- 5% (Brown 1992).

Cerebrovascular accidents are one of the leading causes of death after heart disease and cancer in the developed countries and one of the leading causes of death in Bangladesh. The exact prevalence rate of this disease in the

population is not known. The incidence rate and the death rate from stroke increases dramatically with age. About 15 to 30% of patients die with each episode of cerebral infarction and 16 to 80% with cerebral haemorrhage. Those who survive are usually left with permanent disability. Thus, stroke becomes a great medical and social problem (Osborn 2013).

Stroke is the third leading cause of death after ischaemic heart disease and cancer all over the world specially in the developed countries and is responsible for much of the physical and mental disability in the elderly (Warlow 1996). In another study, it is the second commonest causes of death (9%) and a major cause of disability world wide. The age adjusted annual death rate from stroke in UK is about 200 per 100,000 (12% of all death) (Kumer *et al.* 2009). Annually 16.3 million people suffer from stroke world wide, among which 11.2 million events occur in developing countries like ours and about 5.8 million people die of stroke each year, the two third of which occurs in developing nations (Truelsen *et al.* 2008). The loss of these patients from the work force and the extended hospitalization they require during recovery make the economic impact of the disease one of the most devastating in medicine. In developed countries, epidemiological studies have lately addressed the causes and natural history of the disease and the need for hospital and community services.

An analysis of representative U.S. population indicates that the stroke incidence is approximately 0.5 to 1 per 1000 population. Incidence rates in most European countries are slightly higher 1.5 per 1000, but several Eastern European countries and Japan has rates of 3 per 1000. Importantly there is a strikingly higher incidence (20-30 per 1000) for those over age of 75 (Pulsinelli *et al.* 1992).

Stroke is also the principal cause of death and chronic disability in all developed countries. Stroke causes about 10 deaths per 100,000 population

per annum in Europe and USA in those aged over 40 years, rising to 1000 per 100,000 per annum at 75 years (Clark 1990).

Apart from increasing age, hypertension is the most important risk factor. The incidence of stroke is increased with lower socio-economic class; this probably reflects increasing prevalence of hypertension and smoking (Brown 1992). Diabetes Mellitus, hyperlipidaemia, heart disease, oral contraceptive and excess intake of alcohol are also considered as risk factors for developing stroke (Cull & Will 2014).

The gravity of the situation in Bangladesh can be assessed by the high rate of admission of stroke patients in general hospitals. In a study in Institute of Post Graduate Medicine and Research (IPGMR) in 1986, it was found that stroke patients comprised 16.96% of all the patients attending Neuromedicine outpatient department. Of them 70% was due to thromboembolism, 22% cerebral haemorrhage and the rest 8% due to undetermined lesion (Haque *et al.* 1986).

The rational management of patient presenting with an acute stoke should be based on knowledge of its pathological types and locating the sites of lesion. Before the introduction of Computed Tomography (CT) Scan, diagnosis of stroke was based on clinical findings, cerebral angiography and lumbar puncture with CSF examination. But the distinction between haemorrhage and infraction is not always apparent clinically. To enhance the diagnostic accuracy, various clinical scoring systems like Glasgow coma scale, have been introduced but those are also not simple to follow.

The advent of CT in early 1970s greatly facilitated the diagnosis and management of stroke and added significantly to our understanding of pathophysiological brain alterations in case of humans. With CT it is now possible for the first time to noninvasively and reliably diagnose and

distinguish between stroke due to cerebral infarction and stroke due to hemorrhage. In addition, other brain lesions, at times, may clinically present as stroke like syndromes such as primary or metastatic brain tumor or subdural hematoma that can usually be clearly differentiated by CT examination (Sandercock *et al.*1985).

Computed Tomography is one of the most accurate methods available for identifying and localizing an infraction within the brain. Ischemic infarction, haemorrhagic infarction and intracerebral hematoma are usually differentiated. CT also permits identification of the acute and chronic sequence that may develop after a sequence of infarction. These include, in acute phase, brain swelling and conversion of a bland infarct into haemorrhagic infarct and in chronic phase, cystic parenchymal change, cortical atrophy and focal ventricular dilation (Osborn 2013).

Remarkable advances in the experimental and clinical evaluation of stroke have been made during the past 20 years. There is now greater understanding of the biochemical mechanism of ischemic cell death. A host of tools are now available for imaging the cerebrovascular trees and the brain parenchyma at the time ischemic symptoms including ultrasound, computed tomography, CT angiography and Magnatic Resonance (MR) Imaging. MR angiography and MR imaging with diffusion and perfusion assessment has shown great promise as an early diagnostic tool in the evaluation of the inpatient suffering from acute ischemic episode. This technique however, is yet not in widespread use (Marks 1998). Magnetic Resonance imaging is unquestionably superior to CT to demonstrate infraction early and lesions in posterior fossa but it is more often negative in the acute stage sub-arachnoid haemorrhage or non-specific in acute intracerebral haemorrhage (ICH). Moreover, MRI has got disadvantages, which include high cost and poor availability in developing countries like Bangladesh, long scan time and

incompatibility with the metallic objects like pacemakers and prostheses. Again similar sensitivities were demonstrated for MR (85.5%) and CT angiography (88.5%) for circle of Willis (Katz *et al.* 1995). Other imaging studies as Xenon CT, single photon emission CT (SPECT) and positron emission tomography (PET) are occasionally but not generally used (Bryan 1990).

The introduction of computed tomography to clinical practice has a great impact on our knowledge of cerebrovascular disorders, and cerebral CT has become the most commonly used primary radiologic investigation for stroke. Cerebral CT has shown that prognosis of intracerebral haemorrhage (ICH) is not as poor as was supposed when small haemorrhage were often undiagnosed or misdiagnosed as ischemic events, and it has changed the order of diagnostic procedures for stroke. Furthermore, in differentiating ischemic infarcts from hemorrhagic lesions, cerebral CT has proved to be of crucial importance for therapeutic consideration, particularly anticoagulant treatment (Sotaniemi *et al*. 1990).

CT scan of brain revolutionized the method of diagnosis of stroke and has become the confirmatory radiological diagnostic tool for stroke. There are several possible reasons for performing CT scan of the head in patients with acute stroke-namely, to establish the diagnosis; to identify types of stroke amenable to surgery; to exclude ICH; and in spontaneous subarachnoid hemorrhage (SAH) (Sandercock *et al.* 1985).

Krassen Nedeltchev *et al.* (2010) reported 13% died within 30 days of their first-ever stroke in ischaemic patient. In a study done by Collins *et al.* (2003) from the Department of Veterans Affairs (VA), 34 866 patients with firstever ischaemic stroke were retrospectively identified and the authors reported 8.2% mortality at 30 days after the stroke. De Jong *et al.* (2003) observed similar rates (10% mortality at 30 days) in 998 patients with first-ever

cerebral infarction. At the same time, Rothwell *et al.* (2004) reported a 17.2% case fatality due to initial ischaemic stroke in the Oxford Community Stroke Project (OCSP, 1981 – 1984) and 17.8% in the Oxford Vascular Study (OXVASC, 2002-2004).

CT evaluation of stroke is very important in diagnosis. Prediction of stroke can be done by the findings of CT. It will be a cost effective procedure in Bangladesh. There is a good correlation between progressions of a stroke with CT findings. In this study attempt was taken to find out relationship between mortality of stroke patients with their CT findings. We also extended our research by seeing the risk factors, location of insults, arterial territories involved in infarct to enrich our study.

To the best of my knowledge in Bangladesh there is no adequate data on incidence and mortality from stroke. Mortality from haemorrhagic and ischaemic stroke varies. It needs to find out mortality from both type of stroke. Keeping in mind the above facts, the present study has been undertaken with the asscess the characteristics CT findings of acute stroke and their relationship to mortality.

CHAPTER-2

RATIONALE, HYPOTHESIS, AIMS AND OBJECTIVES

2.1. RATIONALE OF THE STUDY

Stroke is a common problem in Bangladesh where a large number of people suffering from this disease because of ignorance and poor knowledge of risk factors of the disease. It is one of the common causes of mortality and morbidity. Moreover it possesses a serious economic and health burden for the family and the state.

Pullicino *et al.* (1997) stated that haemorrhage and infarct volume, pineal displacement on a scan performed within 48 hours can identify a group of patients with a high risk of early death, is of practical importance because it can facilitate early management decisions in these patients. It should allow the physicians to target a group of patients at high risk for early death for detailed discussion of management option, including surgical decompression prior to the onset of neurologic deterioration.

Because of the rapid and severe devastation associated with intracerebral hemorrhage/infarct, innovative treatments need to developed and evaluated. Bedside estimation of volume of stroke severity can be a powerful tool for selection and stratification of patients in these future treatment studies.
Rajshahi Medical College Hospital is a leading Government owned tertiary level hospital. A good number of stroke patients from different social, cultural educational and economical levels from in and surroundings areas of Rajshahi city attend the respective OPDs or are admitted to this hospital to receive treatment for different disease including stroke.

So far the knowledge goes; no work has been done in this field in Bangladesh. The present study, therefore, was undertaken. The information emerging from the study will hopefully be useful in the prediction stroke and therefore, will be a milestone for further study of stroke predictor.

2.2. HYPOTHESIS

CT findings are well correlated with mortality of stroke and can be a good predictor of mortality of stroke.

2.3. AIMS AND OBJECTIVES

2.3.1. GENERAL OBJECTIVES

To study the CT findings of acute stroke patients and their relationship with mortality within 7 days and 28 days.

2.3.2. SPECIFIC OBJECTIVES

1. Study of CT findings of acute first ever stroke.
2. Measurement of volume of haemorrhage and size of infarct on the day of scan.
3. Determination of subarachnoid haemorrhage.
4. Determination of extension of PICH into ventricles.
5. Determination of hydrocephalus vs mortality.
6. Measurement of mass effect by midline and pineal displacement and effacement of cerebral sulci and fissure.
7. Identification of vascular territory of infarct.
8. Mortality follows up at 7^{th} and 28^{th} day.

CHAPTER-3

REVIEW OF LITERATURE

3.1. RELATED STUDIES

Stroke is a heterogeneous collection of different disorders that affect the vasculature of the brain. It is the third leading cause of death after ischaemic heart disease and cancer, and an important cause of hospital admission and long-term disability in most industrialized countries (Bonita 1992).

The main categories of stroke are cerebral infarction, primary cerebral haemorrhage and subarachnoid haemorrhage. As a rough estimate about 80% of strokes are due to cerebral infarction, 10% each due to SAH and intracerebral haemorrhage (Warlow 1996).

In many clinical studies, the percentage of embolic stroke was 15-20% (Hart 1992). Stroke affects more males than females, except in the very young and very old (Cull *et al.* 2014).

There is much less difference in the incidence of cerebral infraction between males and females (Warlow 1996). But mortality is higher in women about 16% of all women are likely to die of a stroke where as 8% of men die due to stroke (Bonita 1992).

Hypertension is the most important predisposing factor for both ICH and cerebral infraction and the risk of infarction increases with increasing levels of either systolic or diastolic pressure. It is said to be contributing in about 70% of the strokes. Diabetes is associated with a three-fold increase in the risk of stroke (Pulsinelli *et al.* 1992). The results from the Framingham study indicate that smokers had more than three times the risk of cerebral infraction (Yano *et al.* 1986). It is difficult to assess whether stroke is due specifically to hyperlipidaemia or a combination of conditions that include hyperlipidaemia. Abnormalities in the blood leading to a hypercoaguable state may be important in causing thrombosis (Clark 1990). The effects of racial and

genetic factors are not fully known. Hypoglycaemia may cause cerebral infraction.

In the CT evaluation of 300 stroke patients performed in the department of Radiology and Imaging, BIRDEM, Dhaka, during the period of April'91 to April'92, where Bashar *et al.* (1992) found that 219 (73%) cases of intracerebral infarcts and 68 (27%) haemorrhagic lesions. Majority of the infarct i.e. 75% were found in the middle cerebral artery territory and 12% were in the anterior cerebral artery territory.

In Bangladesh, there are no adequate data on the incidence and mortality from stroke in the community. In one study conducted at Dhaka Medical College Hospital (DMCH), stroke was found to be second most common cause of emergency admission in the medicine wards and constituted about 10-12% of the total patients in these wards. About 3% of the admitted patients in Chitagong Medical College Hospital (CMCH) were suffering from stroke and 5.87% of the same in Diabetic Hospital (Latif *et al.* 1990).

The usefulness of CT was assessed in 325 consecutive patients with a 'clinically definite first stroke''from a community stroke registrar. CT was useful in excluding intracranial haemorrhage as the cause of first stroke in four patients receiving anticoagulants and seven patients receiving anti-platelet treatment; it also showed intracranial haemorrhage in one patients taking aspirin. The CT scan provides very useful information in a majority of patients who can be selected on quite simple criteria: (a) doubt (usually because of an inadequate history) where the patient had stroke or a treatable intracranial lesion; (b) the possibility of cerebellar haemorrhage or infarction; (c) the exclusion of intracranial haemorrhage who either are alredy taking or likely to need anti-haemostatic drugs or are being considered for carotid endarterectomy; (d) if the patiet deteriorates in a condition atypical of stroke (Sandercock *et al.* 1985).

3.2. ANATOMY OF THE BRAIN

3.2.1. Gross anatomy

The brain lies in the cranial cavity and is continuous with the spinal cord through the foramen magnum. It is surrounded by three meninges: the dura mater, the arachnoid mater and the pia mater. The brain is conventionally divided into three major divisions. These are the forebrain, the midbrain and the hindbrain. The hindbrain may be subdivided into medulla oblongata, the pos and the cerebellum. The forebrain is also subdivided into diencephalons and the cerebrum. The brain stem is a collective term for the medulla oblongata, pons and midbrain.

Cerebrum (Fore brain)

The cerebrum is the largest part of the brain. It is composed of two cerebral hemispheres, which are connected by a mass of white mater called corpus callosum. Each hemisphere extends form the frontal to the occipital bones superior to the anterior and middle crania fossa; posteriorly the cerebrum lies longitudinal fissure. The surface layer of each hemisphere, the cortex is composed of gray mater. The cerebral cortex is thrown into folds or gyri separated by fissures or sulci. A number of large sulci are conveniently used to subdivide the surface of each hemisphere into lobes. The lobes are named form the bones of the cranium under which they lie.

The frontal lobe is situated anterior to the central sulcus and superior to the lateral sulcus.The parietal lobe is situated posterior to the central sulcus and superior to the lateral sulcus. The occipital lobe lies inferior to parieto-occipital sulcus. Inferior to lateral sulcus is situated the temporal lobe (fig. 1). The extreme ends of each hemisphere are called the frontal, occipital and temporal poles (Snell 2006).

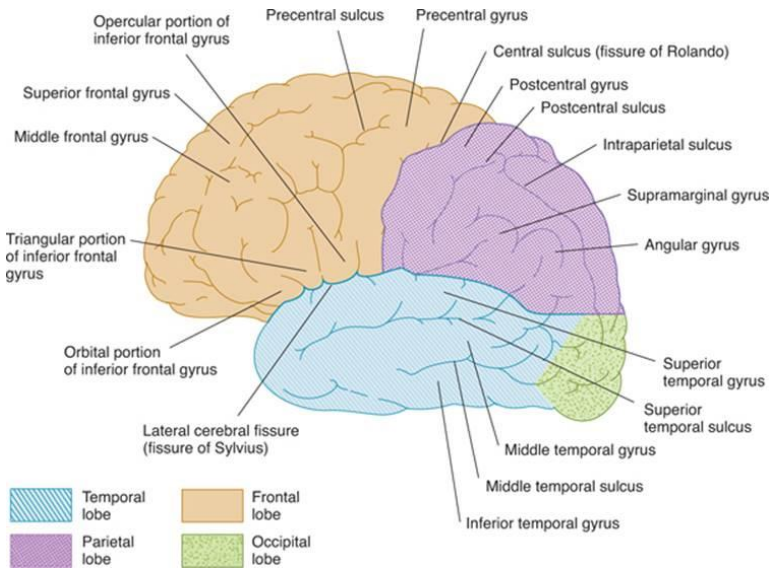

Fig. 1. Lateral view of cerebrum.
(Source: https://doctorlib.info/anatomy/clinical-neuroanatomy/10.html)

White matter of cerebrum

The white matter of cerebrum consists chiefly of myelinated fibres that connect various parts of the cortex to one another and also to the other parts of the CNS. The fibres are:

Association fibres

These are fibres, which connect different cortical areas of the same hemisphere to one another.

Projection fibres

These fibres connect the cerebral cortex to other parts of the CNS e.g. the brainstem and the spinal cord. Many important tracts of the e.g. corticospinal and corticopontine are made up to projection fibres.

Commissural fibres

These are the fibres which connect corresponding parts of the two hemispheres.

Corpus callosum

The corpus callosum is the largest commissure to the brain. It connects the two cerebral hemispheres. Parts of corpus callosum -

i) The genu is the anterior end.

ii) The rostrum.

iii) The trunk.

iv) The splenium.

Internal capsule

Internal capsule is a large band of fibres situated in the infero-medial part of each cerebral hemisphere. In horizontal section of brain, it appears V shaped with its concavity directed laterally. The concavity is occupied by the lentiform nucleus. The internal capsule contains fibres going to and coming from the cerebral cortex. It can be compared to a narrow gate where the fibres are densely crowed. The internal capsule is divided into the following parts-

i) The anterior limb.

ii) The posterior limb.

iii) The genu.

iv) The retrolentiform part.

v) The sublentiform part.

Basal nuclei

The basal nuclei are subcortical, intracerebral masses of gray matter forming important parts to the extra pyramidal systems. They include:-

i) The corpus striatum which is partially divided by internal capsule into two nuclei-

a) The caudate nucleus,

b) The lentiform nucleus which consists of lateral putamen and medial globus pallidus.

ii) The amygdaloid body forms part of the limbic system.

iii) Claustrum.

The Diencephalon or Interbrain

The diencephalon is almost entirely hidden from view by the cerebral hemispheres. It consists of a dorsal thalamus and a ventral hypothalamus.

The mid brain

The mid brain is also called the mesencephalon. It connects the hind brain with the fore brain. Its cavity is known as the cerebral aqueduct. It connects the third ventricle with the fourth ventricle. The mid brain passes through the tentorial notch and is related on each side to the parahippocampal gyri, the optic tracts, the posterior cerebral artery, the basal vein, the trochlear nerve and the geniculate bodies, Anteriorly it is related to the interpeduncular structures and posteriorly to the splenium of the corpus callosum, the great cerebral vein, the pineal body and the posterior end of the right and left thalami.

The hind brain

The medulla is the lowest part of the brainstem; extending from the lower part of the pons to a plane just above the first cervical nerve where it is continuous with the spinal cord. Anteriorly it is related to the clivus and meninges and posteriorly to the valecula of the cerebellum. Along with other parts of the hindbrain, the medulla occupies the infra-tentorial space.

The pons

The pons is the middle part of the brainstem, connecting the mid brain with the medulla. Literally, the ward pons means ‘Bridge’.

The cerebellum

The cerebellum is the largest part of the hindbrain. It is situated in the posterior cranial fossa behind the pons and medulla. It is an infratentorial structure that coordinates the voluntary movements of the body.

The ventricular systems of the brain

There are four ventricles (fig. 2.) within the brain that contain choroid plexuses producing CSF. The two lateral ventricles join to the third ventricle via the midline foramen of Monroe. The lateral ventricles are open C-shaped cavities extending from front to back within deep brain tissue of cerebral hemispheres.

They are covered by a layer of ependymal cells forming inner walls. The anterior extensions into the frontal lobes are called frontal horns. The frontal horns are separated from each other by septum pellucidum. The frontal horns are outlined laterally by the head of caudate nuclei. Anteriorly the frontal horns are bounded by the genu of corpus callosum. The body of the lateral ventricles extends posteriorly over the corpus callosum and is outlined by the body of caudate nuclei. The posterior extension of the body of the lateral ventricles forms the occipital horns. Medially the occipital horns are bounded by the splenium of corpus callosum. The temporal horns are extension of lateral ventricles into the temporal lobes. The place where the temporal and occipital horn and the body of the lateral ventricle meet is called the atrium and is the most expanded part of the lateral ventricle. The main portion of the choroid plexus is located in the atrium. The third ventricle is a midline structure situated between the two thalami. Anteriorly the floor of the third ventricle is formed by the tuber cinereum and posteriorly it is bounded by the cerebral peduncles. The roof of the third ventricle is formed by the velum interpositum. The roof of the third ventricle is formed by the velum interpositum. The fourth ventricle is located in the posterior fossa between the pons and medulla oblongata anteriorly and cerebellum posteriorly. The fourth ventricle is connected to the third ventricle via the cerebral aqueduct. CSF which is excreted by the choroid flows from the lateral ventricles to the third ventricle and from there to fourth ventricles, exiting the ventricle via

foramen of Magendi and foramen of Luschka to drain into cisterna magna (Nahaeedy 1998).

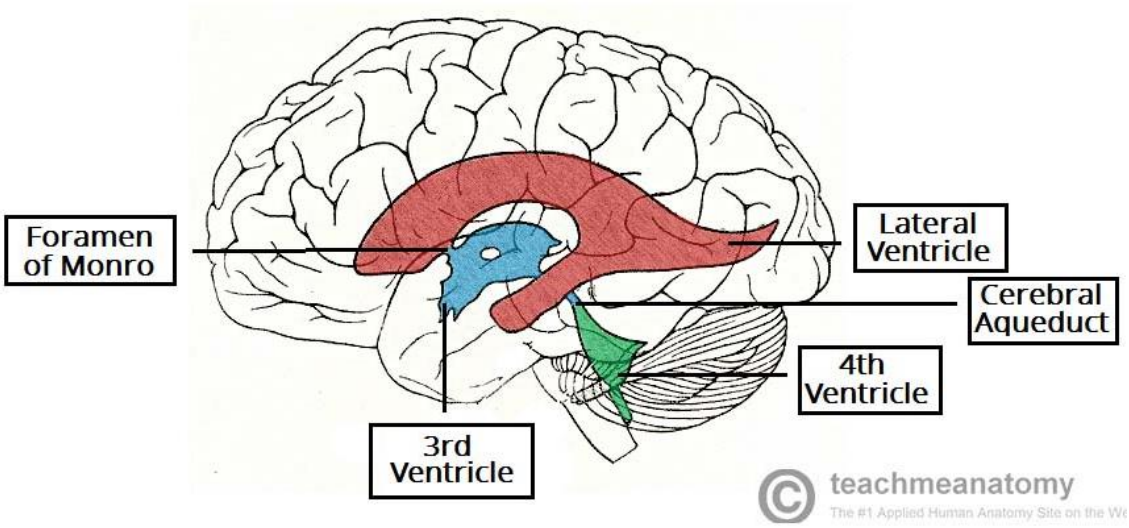

Fig. 2. Ventricular system of brain.
(Source: http://teachmeanatomy.info/neuro/vessels/ventricles/)

3.2.2. Blood supply of the brain

Arteries of the brain

Brain is supplied by the two internal carotid and the two vertebral arteries. The four arteries lie within the subarachnoid space and their branches anastomose on the inferior surface of the brain in the interpeduncular fossa to form the circle of Willis (fig. 3.). It is formed by the anastomosis between two internal carotid arteries and the tow vertebral arteries. The anterior communicating, anterior cerebra, internal carotid, posterior communicating posterior cerebral and basilar arteries all contribute to the circle (Snell 2006).

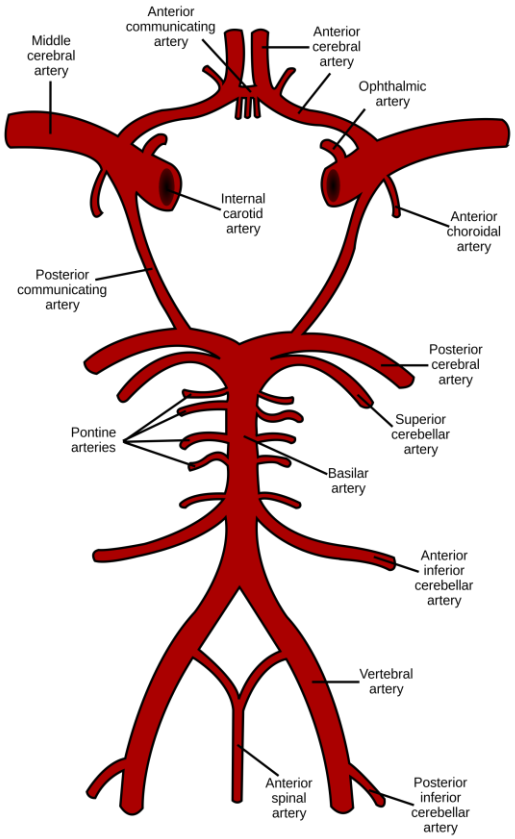

Fig. 3. Blood supply of the brain (Circle of Willis).

(After Wikipedia, https://en.wikipedia.org/wiki/Circle_of_Willis)

Blood supply of cerebral hemisphere

The anterior, middle and posterior cerebral arteries give rise two sets of branches - a) cortical and b) central. The cortical branches ramify over the surface of the cerebral hemisphere and supply the cortex. Central branches pass deep into the substances of the cerebral hemisphere to supply.

Arterial supply of the cerebral cortex

Cortical branches of the anterior, middle and posterior cerebral arteries supply the cerebral cortex. The middle cerebral artery supplies the greater part of the superolateral surface. The main artery supplying the medial surface is the anterior cerebral. The posterior cerebral artery supplies the area of this surface belongings the occipital lobe. The middle cerebral artery supplies the lateral part of the orbital surface and the anterior cerebral artery

supplies the medial part of this surface. The posterior cerebral artery supplies the tentorial surface. The temporal pole is however, supplied by the middle cerebral artery.

Arterial supply to the specific areas of the brain

Internal capsule

It is supplied by the central branches of the middle cerebral artery, the anterior cerebral artery, the posterior communicating artery and the anterior choroidal artery.

Corpus striatum

It is supplied by central branches of middle cerebral artery and anterior cerebral and communicating arteries.

The choroid plexus

The anterior choroidal artery branches of internal carotid and the posterior choroidal artery branch of posterior cerebral artery supply the choroid plexuses of the lateral and third ventricles.

Thalamus

It is supplied by central branches of the posterior cerebral artery, posterior communicating and basilar arteries.

Midbrain

The midbrain is supplied by the posterior cerebral, superior cerebellar and basilar arteries.

Pons

The pons is supplied by the basilar and the antero-inferior and superior cerebellar arteries.

Medulla oblongata

The medulla oblongata is supplied vertebral, anterior and posterior spinal, posterior inferior cerebellar and basilar arteries.

Cerebellum

The cerebellum is supplied by the superior cerebellar, anterior-interior cerebellar and posterior inferior cerebellar arteries (Snell 2006).

Vascular territories of the brain on axial section (fig. 4a, 4b)

On axial CT-Scans, the vascular territory is readily delineated by drawing a line along the anterior and posterior course of the lateral border of the lateral ventricles. The area between the anterior portion of the interhemispheric fissure and line along the anterior lateral wall of the lateral ventricle is the anterior cerebral territory. The area between the posterior portion of the interhemispheric fissure and a line along the posterior lateral wall of the lateral ventricle is the posterior cerebral territory. The area lateral to the line along the lateral ventricle is the middle cerebral territory. In high convexity slices above the ventricular level, almost all parasagittal areas of frontal and parietal lobes are supplied by branches of anterior cerebral artery with the small portion in the posterior medial area supplied by the branches of posterior cerebral artery. The ill-defined anterior area between the anterior cerebral artery and middle cerebral artery is called anterior water shed region. The posterior water shed area lies between the territory of middle cerebral and posterior cerebral arteries (Naheedy 2017).

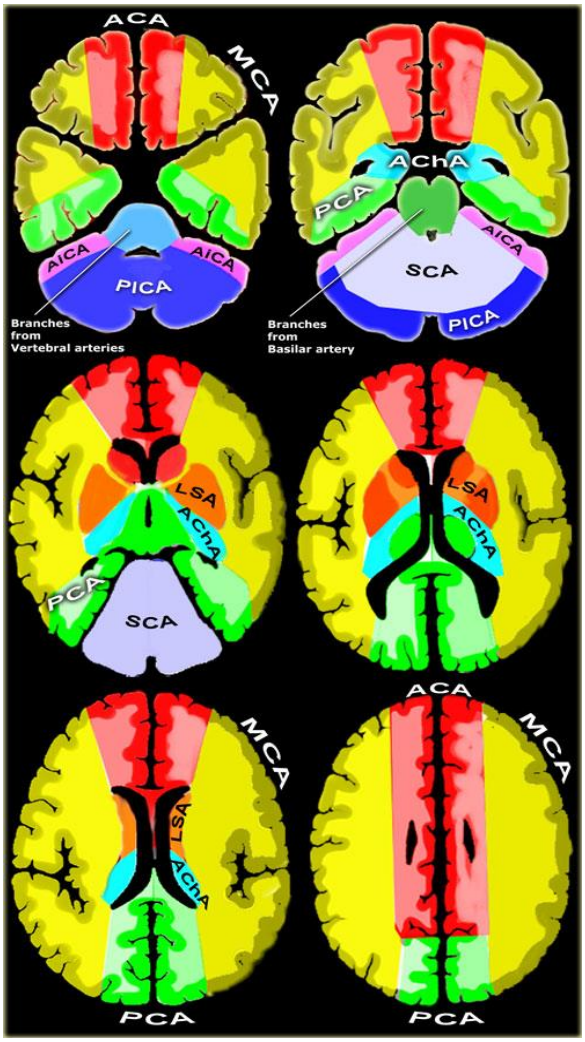

Fig. 4a. Vascular territories of brain in axial section.
(Source:http://www.radiologyassistant.nl/en/p484b8328cb6b2/brain-ischemia-vascular-territories.html)

Venous drainage

Veins of the cerebrum

External cerebral veins

The superior cerebral veins pass upward and empty into the superior sagital sinus. The superficial middle cerebral veins drain the lateral surface of the cerebral hemisphere and empties into the cavernous sinus. The deep middle cerebral veins drain the insula and are joined by the anterior cerebral and striate veins to form basal vein. The basal vein joins the great cerebral vein which in turn drains into the straight sinus.

Internal cerebral vein

There are two internal cerebral veins and they are formed by union of the thalamostriate vein and the choroid vein at the interventricular foramen. They unite to form the great cerebral vein which empties into straight sinus.

Veins of the specific brain areas

The midbrain is drained by veins that open into basal or great cerebral veins. The pons is drained by veins that open into the basal vein, cerebellar veins or neighboring venous sinuses. The medulla oblongata is drained by veins that open into the spinal veins and neighboring venous sinuses. The cerebellum is drained by veins that empty into the great cerebral vein or adjacent venous sinuses (Snell 2006).

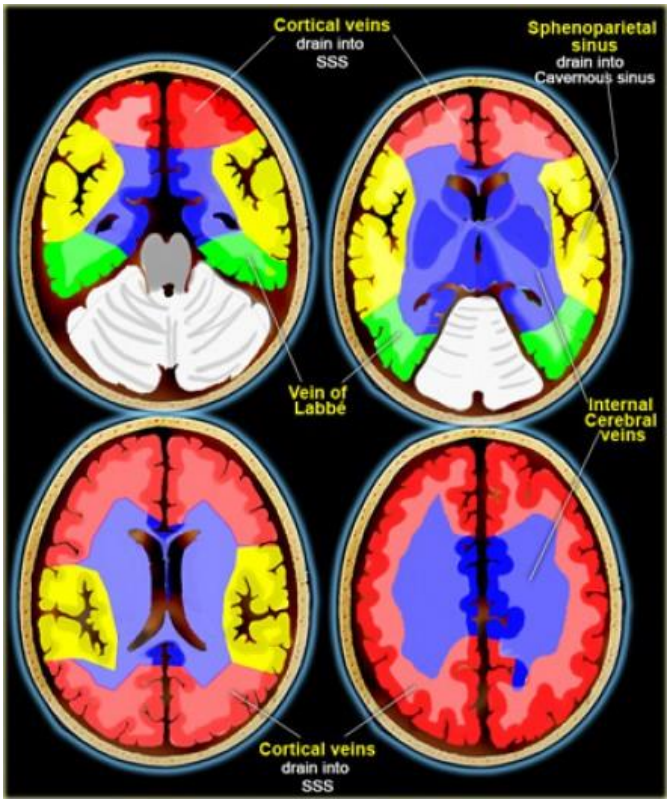

Fig. 4b. Venous drainage of brain.
(Source:http://www.radiologyassistant.nl/en/p484b8328cb6b2/brain-ischemia-vascular-territories.html)

3.3. Stroke

3.3.1. Definition

The term stroke can be clinically defined as: "A neurological deficit of sudden onset; with focal rather than global neurological dysfunction, with symptoms lasting more than 24 hours or resulting in death before 24 hours; and in which, after adequate investigation, symptoms are presumed to be a non traumatic vascular origin" (Bamford 1992). A stroke results either from ischaemic infarction of part of the brain or from intracerebral or subarachnoid haemorrhage. Any lesion of the vessel wall like occlusion of the lumen by a thrombus or embolus, rupture of a vessel, altered permeability of the vascular wall and increased viscosity or other changes in the quality of blood may cause stroke. Arterial disease may be due to atherosclerosis, hypertensive

atherosclerotic change, arteritis, aneurysmal dilatation or development malformation.

3.3.2. PATHOPHYSIOLOGY

Cerebral ischemia and infraction

Pathophysiology (fig. 5.)

Cerebral ischemia is significantly diminished blood flow to all parts (global ischemia) or selected areas (regional or focal ischemia) of the brain. Stroke is a dynamic process in which the location and degree of cerebral ischemia and resultant infraction change overtime. Ischemia manifestations are predicated by the clinically important parameters of flow decrement, location, duration, and tissue volume involved (Terpening 1992, Price 1986).

Physiology of cerebral ischemia and infraction

Stroke progresses in stage from ischemia to acute infraction. This process involves many simultaneous, as well as sequential events. In the most common situation, i.e., ischemia due to middle cerebral artery occlusion, there is a densely ischemia central focus and a less densely ischemia "penumbra" cells within the densely ischemic area are usually damaged irretrievable unless reperfusion is quickly established whereas cells within the penumbra may remain viable but at risk for several hours. Current salvage therapies attempt to rescue these "at risk" cells (Bryan 1990, Siesj 1992, Marshall *et al.* 1993).

Pathology and Pathogenesis

The principal cause of cerebral infarction is atherosclerosis and its sequelae. In industrialization nations, atherosclerosis is the underlying basis for cerebral thromboembolism in over 90% of all cases. Atherosclerosis is also the most common cause of craniocerebral vascular stenosis in adults (Okazaki 1989).

Pathology

Atherosclerotic plaques are eccentric focal fibrofatty intimal thickening. Atherosclerosis affects large, medium and small arteries and arterioles. Craniocerebral ASVD occurs commonly and most severely at the internal carotid artery origin and the distal basilar artery (Okazaki 1989, Yasake *et al*. 1993).

Etiology

The pathogenesis of atherosclerosis remains controversial. There is probably no signal cause, no signal initiating event, and no exclusive pathogenitic mechanism. Two major theories are that ASVD is a reaction to injury or a cellular proliferation disorder with underlying mechanisms similar to neoplastic transformation. Aspects of both may be involved in the complex events associated with ASVD (Davies 1990).

Atherogenesis is probably initiated by focal endothelial change of subtle intimal injury that leads to platelet aggregation. The flow reversal that is normally seen in the posterior carotid bulb may also be a contributing factor to platelet adhesion and initial plaque formation. Endothelial injury permits increased permeability to macromolecules such as low density lipoproteins. Monocyte-derived macrophages and smooth muscle cells are recruited to the intima where they proliferate and accumulate fatty esters, becoming lipid-filled foam cells. As these cells die, their detritus produces the extracellular cholesterol deposits that from the atherosclerotic plaque (Davies 1990).

Intimal fatty streaks are the earliest macroscopically visible lesions in atherosclerosis. As the disease progressive a fibrotic cap is formed that covers a core of foam cells, necrotic debris, and cholesterol crystals. Underlying secondary inflammatory changes ensue with formation of granulation tissue and neovascularity. Eventually, intraplaque subintimal haemorrhage and necrosis occur. As the plaque ruptures, endothelial integrity is lost. The

ulcerated intimal plaque may serve as a nidus for thrombi that embolize distally (Vinitski *et al*. 1991).

Brain cells demand a continuous supply of oxygen and glucose from the blood. Gray matter requires blood flow of 60 ml to 80 ml/100g of tissue/min, whereas white matter needs only 20 to 25 ml/100g/min (Pulsinelli 2000). Cellular dysfunction can be observed when blood flow drops to 50% of these levels. At 15 to 18 ml/100g/min, electroencephalogram (EEG) detectable electrical activity ceases within seconds. With this so-aclled "electric failure" ATP levels are lowered, but intracellular ion concentrations are well maintained (Pulsinelli 2000). Importantly, if adequate blood flow is restored, this state is reversed. If blood flow falls below 10 ml/100g/min for several minutes, the cells lose their ability to maintain intracellular ion concentrations. This so-called state of "energy failure" is irreversible; the cells will die even if blood flow is restored. Between "electric failure" and "energy failure" the cells exist in a state of functional silence but potential reversibility, known as the ischaemic penumbra. Although "energy failure" will cause cell death within minutes, the penumbral state can persist for several hours. Complete reversal of electric failure can occur if blood flow is restored. The existence of a penumbral region surrounding the completely ischaemic core of an infarct has been demonstrated in experimental and clinical models. The endothelial lining of the vasculature of the brain is unique. The tight junctions between the cells of the vessel walls prevent the passage of many substances that readily permeate vessels elsewhere in the body. Ischaemic injury to the vascular endothelium damages the BBB, allowing molecules and blood cells to leave the vascular space and enter the brain parenchyma. The breakdown in the BBB will generally resolve within 6 weeks.

Edema

The edema associated with brain infarction is a combination of cytotoxic and vasogenic edema. In the first few hours, cytotoxic edema develops as a result of loss of cellular control over water influx. Vasogenic edema develops hours to days after onset of ischaemia as damage to micro vessel endothelium progresses. Vasogenic edema is caused by gradual leakage through the BBB. The extent of edema usually peaks at about 72 hours. Rapidly subsiding thereafter. Brain swelling can lead to herniation and death. In theory, absence of perfusion should prevent edema development. However, trace amounts of residual and collateral flow are almost always available to provide substrate for edema production (Bahn *et al.* 1996).

Lacunar infarcts

Occlusions of deep cerebral vessels, such as the lenticulostriate branches of the middle and anterior cerebral arteries, the anterior choroidal artery, the thalamo perforating branches of the basilar artery, are more probably caused by thrombus. These small arteries usually have no anstomoses, and many are single, unbranched vessels, "end-arteries." A small infarct, "lacunar infarct" or "lacune" develops which becomes cystic and is less than 15 mm in diameter. Lacunae commonly occur in chronic hypertensive patients that have developed arteriolar lipohyalonosis and have a tendency to occur at multiple locations in the same patient. The most frequent location, for lacunar infarcts is the basal ganglia followed by the internal capsule, thalamus, and brainstem (Grossman 1996).

Haemorrhage

In addition to allowing vasogenic edema to develop, breakdown of the blood-brain barrier may also allow red blood cells to cross into the extravascular space, hemorrhagic infarctions cover a spectrum form a few scattered parenchymal lesions to frank haematomas. The presence of hemorrhage in

the ischaemic or infarcted tissue may have important therapeutic ramification. If the ischaemic tissue is reperfused at some critical time, the infarct which until now remained nonhaemorrhagic (pale), may become haemorrhagic (red). Reperfusion of ischaemic tissue may occur after clot lysis, development of collateral flow, or reestablishment of normal blood pressure. Small punctate or petechial hemorrhage is present in many cases of ischaemic infarct. These are generally confined to the gray matter and do not exert mass effect. It is important to emphasize that the development of petechia hemorrhage does not necessarily imply serious clinical deterioration or risk. Its frequency does not increase with the use of anticoagulants such as heparin, although anecdotal reports suggest that the extent of hemorrhage may be worsened.

The parenchymal haematoma is larger confluent collection of blood that exerts mass effect and may contain a fluid level. They often involve white matter structures and dissect widely, frequently into the ventricular system. These are associated with abrupt clinical deterioration during the first few days and may possibly cause death. Parenchymal hemorrhages are less common than petechial hemorrhage: clinical worsening (associated with parenchymal haematomas) is seen in only 10% of patients with haemorrhagic infarction.

Vascular Territories

Cortical infarcts secondary to the occlusion of large cerebral vessel lead to characteristic clinical and anatomic patterns of brain injury. The search for a lesion that matches a predicted vascular territory is fundamental to the anatomic analysis of stroke patients. Most infarcts are seen in the distribution of the middle cerebral artery (MCA). The posterior cerebral artery (PCA) is the next most frequent followed by the anterior cerebral artery (ACA) (Bahn *et al*.1996).

Pathophysiology of ICH (fig. 5.)

Origin of bleeding

Intracerebral haemorrhage (ICH) is commonly arterial in origin, arising not only from the primary arterial site but also from smaller vessels around the expanding heamatoma margin. Cortical veins and dural sinuses are less common sources of ICH (Hayman *et al*. 1989).

Thrombosis, Clot Formation, and Haemorrhage Evolution

Thrombosis and clot formation are complex dynamic processes in which gross structure and macroscopic composition of thrombi change with time. Physiologic processes such as clot retraction, cellular infiltration, and fibrinolysis plus red blood cell morphology, haemoglobin denaturation, and the development of blood degradation products interact to affect the MR appearance of ICH (Blackmore *et al*. 1990).

Immediate effects of haemorrhage

An intracerebral haematoma is initially liquid, composed of 95% to 98% oxygen-saturated haemoglobin. Within seconds after loss of vessel integrity, platelet thrombi from and erythrocyte aggregation in the extravasated blood begins. An unrestricted fibrin mass is formed first as plasma clotting factors converting soluble proteins unto a gel matrix. This creates a complex inhomogeneous mass that contains erythrocytes, white blood cells, and small platelet clumps interspersed with proteins-rich serum (Williams *et al.* 1989).

Hyperacute haemorrhage

Over the next 4 to 6 hours, peripheral edema begins to develop and hemoconcentration ensures as the protein clot retracts, packing the red blood cell (RBCs) to haematocrit of approximately 70% to 90%. During the heperacute stage the haematoma still contains intact biconcave RBCs with oxygenate haemoglobin. Glucose depletion in the haematoma center occurs

over the next several hours. As their energy source in diminished, the extravasated RBCs gradually lose their biconcave shape and become spherical. Significant changes in protein concentration also occur during this stage as molecular cross-linking proceeds and free water within the clot diminishes (Hayman *et al.* 1991, Kirkatrick *et al*. 1992).

Acute haemorrhage

By 12 to 48 hours after clot formation begins, the RBCs become significantly dehydrated. As they shrink and lose their spherical shapes, trapped RBCs acquire irregular speculated projections and from "echinocytes." Haemoglobin desaturation also occurs. By 24 to 72 hours, most intracerebral haematomas contain shrunken but intact erythrocytes with high concentration of deoxygenate intracellular haemoglobin. Edema surrounding the clot is pronounced at this stage (Chon *et al*. 1991, Clark *et al*. 1990).

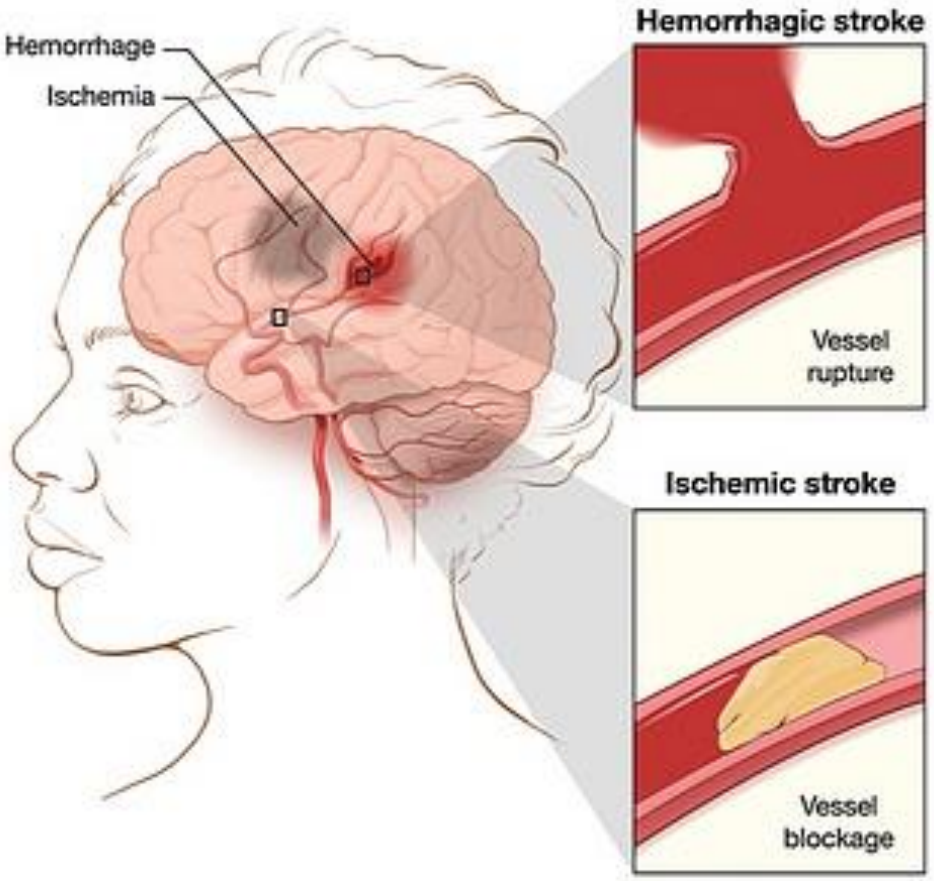

Fig. 5. Pathophysiology of haemorrhage and infarct of brain

3.3.3. Classification of stroke

A. According to clinical manifestations:

1) Completed stroke : a) Major and b) Minor

2) Evolving stroke

3) Progressive diffuse loss of cerebral function

B. According to mechanism:

1. Primary ischaemic (80%)

a) Cerebral thrombosis (50%): i) Large artery and ii) Small artery (lacunar)

b) Cerebral embolism (20%)

c) Uncommon causes (5%)

2. Primary haemorrhage (20%)

a) Intracerebral Haemorrhage

b) Subarachnoid haemorrhage

3.3.4. Risk factors

The risk factors which are likely to be associated with stroke are shown below in table 1.

Table 1. Risk factors associated with stroke.

Major risk factors	Other risk factors
Advanced age	High alcohol intake
Arterial hypertension	Positive family history
Cigarette smoking	Oral contraceptives
Diabetes Mellitus	Heart disease
Hyperlipidaemia	Collagen vascular diseases
Polycythaemia/Thrombocythaemia	Past history of cerebrovascular disease

(After Cull *et al.* 2014)

3.3.5. Causes of stroke (Grossman, 1996)

A. Focal (Arterial Territory) Brain Infarct

1. Atherosclerosis
   i. Carotid artery origin (embolic)
   ii. Cardiac origin (embolic)
   iii. Primary intracranial artery occlusion
   iv. Vascular dementia (Binswanger's)
2. Hypertension: lacunar infarct
3. Emboli of nonatherosclerotic origin
   i) Bacterial endocarditis
   ii) Cardiac myxoma
   iii) Aneurysm
4. Arteritis
   i) Lupus erythematosis
   ii) Polyarteritis nodosa
5. Meningitis
   i) Granulamatous and fungal (basal)
   ii) Suppurative (convexity)
6. Vasospasm: Ruptured aneurysm
7. Arterial compression: Incisural herniation
8. Intracranial nonatherosclerotic occlusive disease
   i) Moyamoya
   ii) Neurofibromatosis
9. Hematological disorders
   i) Sickle cell disease
   ii) Polycythemia
10. Coagulation disorders: Disseminated intravascular coagulation (DIC)
11. Drug induced
   i) Oral contraceptives

ii) Illicit drug abuse

12. Dissection of intracranial arteries

B. Haemorrhage: The causes of ICH and SAH are mentioned in the next section (Shown in Cerebral haemorrhage below).

3.3.6. Mechanism of stroke

Cerebral infarction

Occlusion of major cerebral artery usually leads to infarction unless, a collateral circulation is well developed as in some young people. Thrombosis at the site of atheromatous degeneration in a major cerebral vessel probably the commonest mechanism but embolism of thrombotic or atheromatous material from the heart or an extra-cranial artery is also frequent. Thromboembolism of cardiac origin may arise from mural thrombus after myocardial infarction and are often associated with atrial fibrillation, especially when it is secondary to vascular disease. Cardiac emboli tent to be large and cause occlusion of one of the principal cerebral arteries or a major branch, thereby causing usually major strokes. When cerebral tissue is deprived of blood supply, it undergoes infarction within a few minutes and releases excitatory amino acids. Released excitatory amino acids may exacerbate the neuronal damage by promoting calcium influx. The damaged neurons and glia becomes oedematous after some hours causing more damage by further impairing cerebral blood flow.

Cerebral haemorrhage

About half the strokes caused by intracranial haemorrhage are due to sub arachnoid bleeding from rupture of an aneurysm at the circle of Willis or less commonly from an arterio-venous malformation. In other patients, haemorrhage is mainly into the cerebral substance and is due to rupture of small perforating arteries or arterioles weakened by hypertension or

atheromatous degeneration. Intra-cerebral haemorrhage of this type tends to occur in three distinct sites:

a) The internal capsule - from lenticulo-stricate arteries.
b) The pons - from perforating branches of the basilar artery.
c) The cerebellum.

SAH may induce secondary arterial spasm and thereby cerebral infarction. Although large cerebral haemorrhage cause severe disability, small bleeds in the deep cerebral white matter may cause severe disability, small bleeds in the deep cerebral white matter may cause only mild and transient defects. Cerebral haemorrhage can be fatal if secondary compression of the brain stem occurs. There can be blood in both subarachnoid space and brain tissue after intracranial haemorrhage from any cause. Systemic hypertension, most important pre-disposing factor for stroke is associated with hyalinization and disorganization of the walls of small arteries within the brain. This produces irregular vessel lumina and leads to the development of micro-aneurysms (Charcot-Bouchard aneurysms). Hypertensive ICH tends to occur in the basal ganglia, brainstem or cerebellum (Brown 1992).

3.3.7. Clinical stages and presentations of stroke

A. Clinical Stages

The clinical course after a stroke may be divided into several stages. However, there are no strict guidelines for the terminology regarding the stages of stroke. The authors Bahn, Osser and Cross in their review article "CT and MRI of Stroke" (Bahn *et al.* 1996) adopted terminology that reflects the relevant pathophysiology of ischaemic stroke. The stages of stroke are as follows.

a) Hyperacute stage

The first 6 postictal hours, corresponding with the window of potential therapeutic reversibility as well as the estimated time to the onset of blood

brain barrier breakdown. The actual time frame associated with this stage may depend on the region of the brain involved, the state of collateral circulation and the use of cytoprotective agents.

b) Acute stage

It extends form 6 to 24 hours and corresponds to the defined separation between a transient ischaemic attack (TIA) and the stroke. By 24 hours after the onset of clinical symptoms, the area of ischaemia will be irreversibly injured and will not likely be amenable to reperfusion rescue.

c) Subacute stage

Between 24 hours to 6 weeks, the patient is in the subacute stage. We commonly divide this stage into- i) Early (24 hours to 1 week) and ii) Late (1 to 6 weeks). Most of the post infarct complications will occur in the early stage. The late stage is predominantly marked by resolving edema and, in general much fewer stroke related neurologic complications.

d) Chronic stage

Beyond 6 weeks, the patient is considered to be in the chromic stage of stroke.

B. Clinical presentations

Depending on temporal profile of stages of stroke, stroke may present in the following ways:

a) Completed stroke i.e. rapid onset of focal cerebral dysfunction with symptoms persisting more than 24 hours. It is of two types:

i) Major stroke - where significant disability lasts more than one week.

ii) Minor stroke- where recovery takes place without significant disability within short period, usually within one week.

b) Evolving stroke i.e. the symptoms worsen gradually or in a step-wise fashion over a matter of hours of days. Cerebral tumour or subdural haematoma may produce similar clinical picture.

c) Progressive diffuse loss of cerebral function.

There are no clinical features that reliably distinguish primary intracerebral haemorrhage form cerebral infarction. Headache, coma at onset and vomiting are more common in intracranial haemorrhage but are not diagnostic (Brown 1992).

Imaging of stroke

Neuroimaging of cerebral haemorrhage

Until recently, non-contrast CT was the gold standard for the diagnosis of intracerebral haemorrhage, and at many centres it is still the imaging modality of choice for the assessment of intracerebral haemorrhage, owing to its widespread availability and rapid acquistion time. Conventional T1-weighted and T2- weighted MRI pulse sequences are not sensitive to blood in the hyperacute stage; however, several recent studies have now shown that Gradient recalled echo (GRE). MRI sequences are as accurate as CT for the detection of intraparenchymal haemorrhage and far superior to CT for the detection of chronic haemorrhage (Chalela *et al.* 2007).

In some cases, MRI might actually detect haemorrhages that are missed on CT (Packard *et al.* 2003) and MRI is better than CT at identifying underlying structural lesions and for quantifying perihaematomal oedema. However, up to 20% of patients with acute stroke are unsuitable for MRI (e.g., they have pacemakers or metal implants or are unable to lie flat or still for the duration of the scan) (Singer *et al.* 2004). For both MRI and CT, baseline and serial studies can be used to identify patients who might benefit from acute interventions (e.g., hydrocephalus that requires cerebrospinal fluid drainage or large or expanding cerebellar haematomas that require posterior fossa decompression). Both modalities can include post contrast studies and vessel angiography to rule out underlying structural lesions (e.g., arteriovenous malformation, aneurysm or tumour). In general, contrast studies and catheter

angiography are indicated in patients without a clear underlying aetiology or in patients with haemorrhages in unusual locations (Broderick *et al*. 2007). One recent study suggested a high yield for angiography, even in patients with putaminal haemorrhage, unless the patient is older than 55 years and has hypertension (Park *et al*. 2007).

Neuroimaging provides considerable information with regard to prognosis. Early CT studies in patients with intracerebral haemorrhage have shown that more than a third of patients have substantial (>33%) haematoma growth when imaged within 3 hours of onset. Haematoma expansion is associated with larger baseline haematoma volumes (Broderick *et al*. 2007). In turn, haematoma expansion and the presence of intraventricular haemorrhage are predictors of poor outcome. Cortical intracerebral haemorrhage can be associated with better functional outcome; however, the rate of long-term recurrence is increased by a factor of 3·8 (Castellanos *et al*. 2005). Several recent studies have reported that contrast extravasation seen on CT angiography might also be an early predictor of haematoma expansion and poor outcome. Therefore, CT angiography could have important applications for the selection of patients for acute therapies (e.g., blood pressure management or haemostatic therapy) (Wada *et al*. 2007).

T2* weighted gradient-echo images depict an acute intracranial haemorrhage as an area of abnormal blooming. Susceptibility weighted imaging is a recently developed technique that uses both magnitude and phase images from a high-resolution, three-dimensional, fully velocity compensated gradient-echo sequence. Compared with CT and other MR imaging methods, this technique may be a powerful new approach for detecting a cerebral hemorrhage in a patient with acute stroke. With the use of techniques such as T2* weighted MR imaging, very small cerebral haemorrhages are increasingly detectable in patients with acute stroke.

However, the risk of thrombolytic therapy in patients with MR imaging–depicted microhaemorrhages is unclear, since the present criteria for thrombolysis are based on CT evidence of haemorrhage (Rovira *et al.* 2004, Wycliffe *et al*. 2004, Viswanathan *et al.* 2006).

Imaging techniques also provide insights into the underlying pathophsyiology of intracerebral haemorrhage. Interest in these imaging approaches is driven by the belief that poor outcome in primary intracerebral haemorrhage results, in part, from ongoing secondary neuronal injury in the perihaematomal region. Some of the studies that use PET, single photon emission computed tomography (SPECT), and, more recently, perfusion MR and perfusion CT have shown perihaematomal regions of hypoperfusion and bioenergetic compromise. The results of MRI studies have suggested that approximately a third of patients who are imaged in the acute phase will have reduced perihaematomal apparent diffusion coefficient abnormalities on diffusion-weighted imaging and further studies have characterised the time course for early perihaematomal changes. Apparent diffusion coeffcient values were low during the first day and then gradually increased, which probably matches the development of perihaematomal oedema (Herweh *et al*. 2007, Pascual *et al*. 2007).

Neuroimaging of infarct

Unenhanced CT

Unenhanced CT is widely available, can be performed quickly, and does not involve the administration of intravenous contrast material. It not only can help identify a hemorrhage (a contraindication to thrombolytic therapy), but it also can help detect early-stage acute ischemia by depicting features such as the hyperdense vessel sign, the insular ribbon sign, and obscuration of the lentiform nucleus. The last two features are caused by a loss of contrast between gray matter and white matter on CT

attenuation. This feature is referred to as the hyperdense vessel sign (or, in cases of middle cerebral artery (MCA) involvement, hyperdense MCA sign (Tomandl *et al*. 2003).

Although this sign is highly specific, its sensitivity is poor. A hyperdense MCA sign also may be seen in the presence of a high hematocrit level or MCA calcification, but in such cases the hyperattenuation is usually bilateral (10-13). Rarely, fat emboli appear hypoattenuated when compared with attenuation in the contralateral vessel. Acute ischemia of the lenticulostriate territory may result in obscuration of the lentiform nucleus, which appears hypoattenuated because of cytotoxic edema. This feature may be seen on CT images within 2 hours after the onset of a stroke. Cytotoxic edema of the insular cortex, which is susceptible to early and irreversible ischemic damage, also causes local hypoattenuation, which results in the so-called insular ribbon sign (Lee *et al*. 2005, Tomura *et al*. 1988, Truwit *et al*. 1990).

Early CT signs of brain ischemia include focal or hemispherical hypoattenuation (hypodensity), insular ribbon sign, and obscuration of the lentiform nucleus, sulcal effacement and hyperdense arteries. These signs are related to consequences of cellular hypoperfusion and cytotoxic edema and the last one point on thrombosis of specific blood vessel (Wardlaw *et al.* 2005, Radhiana et *al.* 2013).

Focal or hemispherical hypodensity sign is presented with hypoattenuation of the brain parenchyma, due to the cytotoxic edema. It is found in 20% to 60% of acute stroke cases. It can be present in the cases of watershed ischemia in the area of bordering arteries. The identification of this sign during early stroke is difficult but it can be

width" and "centerlevel" rates. If this sign appears earlier, the prognosis is worse (Somford 2002, Gacs *et al.* 1983, Leiva-Salinas *et al.* 2010).

Loss of insular ribbon sign is defined as lessening precision in delineation of gray-white matter interface at lateral margin of insula. It is prevalent in infarction of the MCA (or internal carotid artery) territory and reported to be present in 75-100% of the cases. The insular segment of the MCA and its claustral branches supply the insular ribbon and because of a lack of collaterals (watershed zone), it is especially sensitive on the occlusion of specific MCA part. Insular ribbon sign rarely appeared alone and more than 50% of patient with it also had obscuration of basal ganglia and effacement of the hemispherical sulcus. The concomitant presence of these three signs point on internal carotid artery (ICA) occlusion and bad prognosis (Koga *et al.* 2003, Wardlaw *et al.* 2005).

The obscuration of the basal ganglia (lentiform nucleus) is one of the earliest sign in patients with AIS, in some patients happens within first hour, while 73-92% of patients had it within 6 hours of stroke onset. When ischemic, an obscured outline or partial disappearance of the lentiform nucleus can be seen on NCCT. This sign is described as decreased attenuation involving the basal ganglia which was explained with the vulnerability of basal ganglia due to the lack of collaterals. Because of that, this sign appears with ICA or M1 segment of MCA occlusion .However, if the embolic occlusions had been in more distal part of the MCA or in other arteries, CT may not show abnormality in the basal ganglia at all (Ambrose 1974, Truwit *et al.* 1990).

Hemispherical (cortical) sulcal effacement sign is defined as decreased contrast, loss of precise delineation of the gray white interface in the margins of cortical sulci thanks to the edema in ischemic tissue. It

cortical infarction which has better prognosis (Furlan *et al.* 2006, Koga *et al.* 2003).

Hyperdense artery sign presents a thrombotic event or better still say, linear or eventually dot change in the arterial lumen. The incidence of this sign varies between 5%- 41%. Hyperdensity is usually seen in MCA lumen but it is also described in other arteries (ICA, posterior cerebral artery and anterior cerebral artery) (Lev *et al.* 2001, Jensen *et al.* 2010, Gacs *et al.* 1983).

The presence of an acute thrombus in the proximal part of MCA creates a linear hyper-attenuation on NCCT, and in distal parts of MCA it is described as dot sign. The first one is connected with severe neurological deficit and bad prognosis, while the other one has lighter form. In the interpretation of hiperdensity sign, caution is needed, because the high density can be a result of calcified arteriosclerosis and high hematocrit values (Leary *et al.* 2003, Hacke 2004, Jha *et al*. 2009, Pikija *et al.* 2016).

The recognition of acute ischemic stroke and it early signs is crucial for physicians and patients. The role of the early CT signs is great but the proper interpretation of the image and consideration of patient's clinical image are necessary as well as physician's experience in this field. The mean sensitivity and specificity of physician's reliability detection of early CT signs were 55% (range 20-87%) and 87% (range 56-100%) respectively. Also, there are disagreements in identification of these changes (Wardlaw *et al.* 2013, Radhiana *et al.* 2013).

Importance of Window Settings

Lev *et al.* (1999) showed sensitivity and specificity of 57% and 100%, respectively, for acute ischemic stroke detection at unenhanced CT with the use of standard window settings (width, 80 HU; center, 20 HU). Sensitivity increased to 71% with a change of window width and center

specificity. Hence, detection of acute ischemic stroke on unenhanced CT images may be improved by using variable window width and center level settings to accentuate the contrast between normal and edematous tissue.

CT Angiography

CT angiography is a widely available technique for assessment of both the intracranial and extracranial circulation. Its utility in acute stroke lies in its capabilities for demonstrating thrombi within intracranial vessels and for evaluating the carotid and vertebral arteries in the neck. CT angiography typically involves a volumetric helical acquisition that extends from the aortic arch to the circle of Willis. The examination is performed by using a time-optimized bolus of contrast material for vessel enhancement. Postprocessing is performed at a three-dimensional display workstation to generate multiplanar reformatted images and maximum intensity projection images. Intraarterial thrombolysis may be more efficacious than intravenous therapy in patients with acute stroke and a significant thrombus burden.

Therefore, the CT angiographic demonstration of a significant thrombus burden can guide appropriate therapy in the form of intraarterial or mechanical thrombolysis. Furthermore, the identification of carotid artery disease and visualization of the aortic arch may provide clues to

the cause of the ischemic event and guidance for the interventional neuroradiologist. Thus, CT angiography is useful for evaluating the intracranial and extracranial vessels and guiding appropriate therapy.

CT Perfusion Imaging

CT perfusion imaging can be used to measure the following perfusion parameters: cerebral blood volume (i.e., the volume of blood per unit of brain tissue; normal range, 4-5 ml/100 gm); cerebral blood flow (i.e., the volume of blood flow per unit of brain tissue per minute; normal range in

time difference between the arterial inflow and venous outflow; and time to peak enhancement, which represents the time from the beginning of contrast material injection to the maximum concentration of contrast material within a region of interest (ROI). Compared with MR imaging, xenon-enhanced CT, positron emission tomography, and single photon emission CT, CT perfusion imaging is more widely available and can be performed quickly on any standard helical CT scanner immediately after unenhanced CT. CT perfusion maps then can be generated in a short time at an appropriate workstation. CT perfusion imaging also allows quantitative and qualitative evaluation of the cerebral blood volume, cerebral blood flow, and mean transit time (Tomandl *et al.* 2003, Koenig *et al.* 1998).

The clinical application of CT perfusion imaging in acute stroke is based on the hypothesis that the penumbra shows either *(a)* increased mean transit time with moderately decreased cerebral blood flow (60%) and normal or increased cerebral blood volume (80%-100% or higher) secondary to autoregulatory mechanisms or *(b)* increased mean transit time with markedly reduced cerebral blood flow (30%) and moderately

reduced cerebral blood volume (60%), whereas infarcted tissue shows severely decreased cerebral blood flow (30%) and cerebral blood volume (40%) with increased mean transit time (Nabavi *et al*. 1999, Wintermark *et al*. 2001).

Conventional MR Imaging

Conventional spin-echo MR imaging is more sensitive and more specific than CT for the detection of acute cerebral ischemia within the first few hours after the onset of stroke. It has the additional benefit of depicting the pathologic entity (stroke and its mimics) in multiple planes. The MR sequences typically used in the evaluation of acute stroke include T1 weighted spin-echo, T2 weighted fast spin-echo, fluid-attenuated

gadolinium-enhanced T1 weig in-echo sequences. Typical MR imaging findings in patients with hyperacute cerebral ischemia include hyperintense signal in white matter on T2 weighted images and fluid-attenuated inversion recovery images, with a resultant loss of gray matter- white matter differentiation analogous to the loss at CT; sulcal effacement and mass effect; loss of the arterial flow voids seen on T2 weighted images; and stasis of contrast material within vessels in the affected territories (Provenzale et *al.* 2003, Elster et *al.* 1990).

Like the hyperattenuated vessel sign seen at CT, a low-signal-intensity or high-signal-intensity vessel sign due to intravascular thrombus can be seen on MR images obtained with a T2* weighted gradient-echo or fluid-attenuated inversion recovery sequence, respectively (Schellinger *et al*. 2005, Rovira *et al*. 2004).

Conventional MR imaging is less sensitive than diffusion-weighted MR imaging in the first few hours after a stroke (hyperacute phase) and may

result in false-negative findings. Since the advent of diffusion MR imaging, conventional MR imaging sequences play only a relatively minor role in acute stroke imaging, whereas diffusion-weighted sequences may be appropriately included in any MR imaging protocol for evaluation of acute stroke.

MR Angiography

Like CT angiography, MR angiography is useful for detecting intravascular occlusion due to a thrombus and for evaluating the carotid bifurcation in patients with acute stroke. Time-of-flight MR angiography and contrast-enhanced MR angiography are commonly used to evaluate the intracranial and extracranial circulation.

Diffusion-weighted MR Imaging

Although diffusion-weighted MI 46 g for acute stroke evaluation was introduced in the clinical setting in the mid-1990s, its widespread application was delayed because of the requirement for high-strength magnetic field gradients. Diffusion-weighted imaging sequences now are incorporated into most MR imaging protocols and are essential components of an acute stroke evaluation (Schaefer 2000).

Underlying Principles

The normal motion of water molecules within living tissues is random (Brownian motion). In acute stroke, there is an alteration of homeostasis, which normally maintains steady-state proportions of intracellular and extracellular water. Acute stroke causes excess intracellular water accumulation, or cytotoxic edema, with an overall decreased rate of water molecular diffusion within the affected tissue. Measurement of net water molecular motion was first attempted by Stejskal *et al.* (1965),

who used a T2-weighted spin-echo MR imaging sequence with two extra gradient pulses that were equal in magnitude and opposite in direction. For various reasons, this technique results in a loss of signal in tissue. Tissues with a higher rate of diffusion undergo a greater loss of signal in a given period of time than do tissues with a lower diffusion rate. Therefore, areas of cytotoxic edema, in which the motion of water molecules is restricted, appear brighter on diffusion-weighted images because of lesser signal losses. On diffusion- weighted images from patients with hyperacute stroke, ischemic tissue appears bright in comparison with normal brain tissue (Schaefer 2000).

Current diffusion-weighted MR imaging techniques employ echo planar sequences that are highly resistant to patient motion. Image acquisition can be performed in a few seconds to 2 minutes and has increased sensitivity to signal changes that are due to molecular motion. Other

shot spin echo, and single sh pin echo techniques; line scan diffusion weighted imaging; and spiral diffusion weighted MR imaging. The actual diffusion coefficient cannot be measured by using diffusion weighted MR imaging, for a number of reasons (including the inability of diffusion weighted imaging to depict the difference between molecular motion due to concentration gradients and molecular motion due to thermal or pressure gradients or ionic interactions) (Schaefer 2000). Hence, the diffusion coefficient obtained from orthogonal diffusionweighted MR images in all three planes is called the apparent diffusion coefficient (ADC).

Perfusion weighted MR Imaging

While diffusion-weighted MR imaging is most useful for detecting irreversibly infarcted tissue, perfusion-weighted imaging may be used to identify areas of reversible ischemia as well. Perfusion weighted MR imaging techniques rely either on an exogenous method of achieving perfusion contrast (i.e. the administration of an MR contrast agent) or on an endogenous method (i.e. the labeling of hydrogen-1 protons in water, also known as arterial spin labeling). Exogenous techniques are typically susceptibility based and depend on T2* effects, but they may be T1 weighted instead. Dynamic susceptibility weighted (T2* weighted) sequences probably are most commonly used in acute stroke evaluation, while the other MR perfusion imaging techniques are more commonly used in tumor evaluation or other applications (Schaefer 2000).

3.4. CT-Scan of brain

3.4.1. Aim of CT

The aim of early NCCT evaluation is to make a distinction between ischemic stroke and cerebral hemorrhage. Proven beneficial therapy for

order to be applied patients must be carefully evaluated (Schellinger *et al.* 2004, Radhiana et *al.* 2013, D[48] *l.*2012).

Thrombolysis is limited to the first 3 to 4.5 hours after symptom onset. Disregard of this rule usually leads to new and probably fatal hemorrhage in the area of infarction. This is why, rapid diagnosis must be brought. NCCT diagnosis of infarction which takes more than one third of middle cerebral artery (MCA) area and/or intracranial hemorrhage should not be given thrombolytic therapy. It is strongly recommended by current guidelines that patients with suspected TIA undergo CT, or MRI if urgently available. In addition to the diagnostic

modalities, interventional neuroradiology procedures have crucial place in therapeutic approach of stroke patients during appropriate “window frame” (Qureshi *et al.* 2017, Hacke et *al.* 1998, Pavabvash et *al.* 2017).

3.4.2. Background of CT-scan

A new method of forming images from X-ray was developed and introduced into clinical use by the British physicist Godfrey Hounsfield in 1972 and is referred to as Computed Transmission Tomography, Computed Tomography (CT) or Computerized axial Tomography (CAT). The Basic and revolutionary assumption was that measurements taken of X-rays transmitted through the body contained information on all the constituents of the body in the path of the beam. By using multidirectional scanning of the object multiple data are collected. In Computed Tomography, X-ray output is collimated to very narrow beam. While passing through the patient, it is partially absorbed, and the remaining photons of the X-ray beam fall on radiation detectors instead of X-ray film. When they strike the detector the X-ray photons are converted to scintillations. These can be quantified and record digitally. The information fed into a computer and presented in analog

image is usually in the form of a gray scale in which whiteness is proportional to X-ray attenuation nt of the tissue at each point of the scan. Thus radio-opaque tissue appear white and radiolucent tissue black. The Hounsfield scale is an arbitrary one with air at- 1000 units and water at 0 units as fixed points (fig. 6.). The first machine had only two detectors and sharply collimated beams of X-rays. The modern machines use a fan beam of X-rays and multiple detectors (cited by Sutton *et al.* 2007).

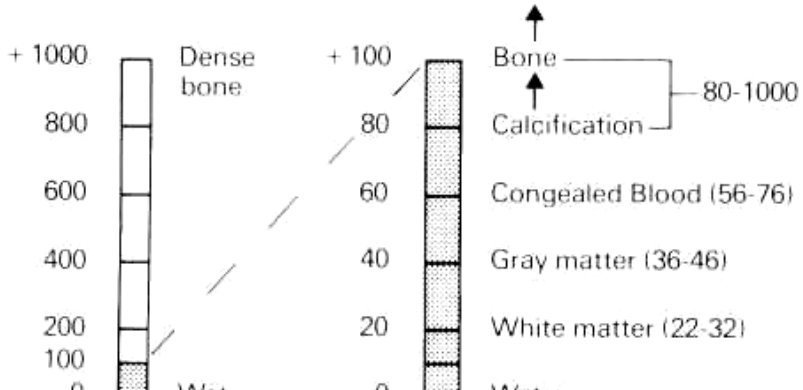

Fig.6. The Hounsfield CT scale. The full scale on the left extends over 2000 units. The expanded scale on the right extends over 200 units and includes all body tissues. Head scans are usually done routinely at a window
Level (L) of 34-40 and a window *(W)* covering 0-75.

3.4.3. Positioning of patient in CT-Scan of brain

The axial plane is the routine projection but direct coronal scans can be made in particular cases. A routine examination of the brain involves making 9 to 10 axial sections with interslice gap of 10 mm and 5 mm (for posterior fossa).

scans are usually done routinely at a window level (L) of 52 to 58 and a window width (W) of 0 to 80.

3.4.4. Cardinal CT signs of abnormality

a) Abnormal tissue density - abnormal tissue may be of higher or lower density than the normal surrounding brain. It is important to outline the location and extent of such variation in density. b) Mass effect-the lateral ventricles should be examined to see if they are displaced or compressed. Shift of midline structures e.g. the septum pellucidum, the third ventricle or the pineal is a common finding with masses. Ventricular dilatation will occur if the mass obstructs the flow of CSF. The need for radiologic evaluation of the stroke patient arises from the desire to confirm the diagnosis, exclude

other conditions that can mimic stroke clinically, and exclude haemorrhagic complications before anticoagulant or thrombolytic therapy. The neuroimaging findings on CT and MR vary with the severity and location of the infarct, the coexistence of other cerebrovascular or systemic disorders, and, of course, the age of the process (Armstrong & wastie 2009).

3.4.5. CT-Scan findings in the stages of stroke

A. Infarction

Hyperacute

Although changes at the cellular level occur within minutes of the loss of adequate blood supply, grossly visible changes in the brain are not identifiable by CT for at least several hours and often not for 1 to 2 days. In this hyperacute stage (< 6 hrs postictus), a normal CT is the rule. Occasionally, subtle focal hypoattenuation and swelling can be indentified in the affected area, with the loss of definition of the lentiform nucleus or insular cortex. The larger the area of infarction, the earlier it will be detected by CT. Other causes of focal neurological deficits, such as migraines, TIAs

patients with underlying processes such as abscess, tumour, or SAH or parenchymal haemorrhage often demonstrate specific abnormalities. CT is also very sensitive to the presence of hyperacute haemorrhage.

Table 2. CT findings of infarction (according to different stages)

Stage of stroke	CT scan findings
Hyperacute	➢ Normal parenchyma (except in occasional large infarct showing subtle swelling and hypoattenuation ➢ Hyperdense middle cerebral artery sign
Acute (6-24 hours)	➢ Progressive swelling and hypoattenation ➢ Loss of gray-white

	differentiation (insula, basal ganglia)
Early subacute 1 day - 1 week)	➢ Progressive swelling/mass effect/herniation ➢ Secondary ischaemic infarcts ➢ onset of enhancement ➢ + Haemorrhagic transformation
Late subacute (1-6 weeks)	➢ Resolving swelling ➢ Fogging ➢ Contrast enhancement (peaks) ➢ + Haemorrhagic transformation
Chronic (> 6 weeks)	➢ Resolved mass effect ➢ Hypodensity ➢ Volume loss ➢ Resolved enhancement (eventually)

(source: Bahn *et al.* 1996)

Acute

Most large infarcts will become visible on CT by the end of the acute stage. All of the findings described above for the hyperacute stage become more apparent and are seen in more cases. Loss of normal gray- white distinction becomes apparent. These are coupled with slight hypodensity in defined

cisternal spaces. The presence of [illegible]hage (or findings associated with non-ischaemic disease processes) [illegible]ficant for treatment planning and prognosis.

Subacute

In the early subacute period, from 1 day to 7 days postictus, there is progressive mass effect and hypodensity corresponding to the peak of edema between days 3 and 5. Potential complications at this stage include brain swelling and herniation. Secondary vascular compression can cause further ischaemia and swelling. Clinical deterioration from brain swelling must be distinguished from postinfarction haemorrhagic complications. During the late subacute stage, from approximately 7 days through 6 weeks, capillary

growth and macrophage invasion, possibly coupled with small petechial haemorrhages, coincide with resolving edema and swelling. The edema subsides, the density of the infarcted area by CT scan increase toward isodensity as result of the reparative tissue growth. An unenhanced infarct examined by CT 2 to 3 weeks after the insult may be inapparent or appears smaller than its actual size although this is less problematic with modern high-resolution CT scanners.

Chronic

As the infarct enters the chronic stage; progressive hypodensity redevelops and progresses as the necrotic tissue is resorbed. The infarction becomes well marginated. The surrounding subarachnoid and ventricular spaces enlarge with the parenchymal loss. Wallerian degeneration of the descending white matter tracts such as the corticospinal tracts is often seen. Diffrent stages of infarcts are shown in table 2.

Contrast enhancement

The lack of an intact blood brain barrier underlies the physiology of

material administration usually becomes visible within several days to week. Common sites of enhancement in 53 cortical gray matter and the basal ganglia. Enhancement of a cortical infarct often appears as a "gyriform" pattern. Because enhancement corresponds to the presence of new "leaky" capillaries, which grow in from the periphery, a ring enhancement pattern can also be seen. Contrast enhancement typically peaks between 14 and 21 days and then gradually diminishes. However it might persist for a month or more. Contrast enhancement present for longer 6 to 8 weeks after infarction should raise the suspicion of underlying process such as neoplasm.

Haemorrhagic infarct

The identification of heamorrhage within an infarct or as the primary cause of the patient's symptoms is an important role of CT in the evaluation of stroke. As many as 40% of patients in autopsy series will show evidence of haemorrhagic transformation, although the percentages among embolic stroke patients are much higher. Also larger infarcts are more likely to heamorrhage. The CT detection rate for haemorrhage in stroke has been reported to be 20% -30%. It is higher in embolic strokes. Petechial haemorrhage is commonly seen by day 4 or 5, but it may be detected as early 24 hours. The CT appearance consists of hyperdense bands in the grey matter or at the periphery of the infarct. Depending on the extent of the petechiae, the hyperdensity will range from patchy to confluent. The distinction from parenchymal haematoma may be difficult at times (Hart 1992, Easton *et al.* 2017). In general, a primary haematoma in haemorrhagic infarct appears more homogenous, sharply defined, and hyperdense than petechial haemorrhage.

B. Haemorrhages

Intracerebral haematoma

Because of the clear distinction between high attenuation of extravasated blood and that of the surrounding brain, CT scanning is the most accurate radiological method for demonstrating intracerebral haematoma on CT, the haemorrhage shows an area of increased attenuation from 50 - 90 Hounsfield units and is surrounded by a thin low attenuation ring that probably results from clot retraction and damage to the blood-brain barrier.

Haemorrhage can dissect through the parenchyma into the subarachnoid space or ventricles. In subarachnoid extension the normal low attenuation

CSF appears isodense with the brain or has areas of increased attenuation. The mass effect depends on the size of the bleed but is frequently less than with tumours of comparable size. Congealed blood appears as an area of high attenuation. Attenuation values increase with progressing haemoconcentration. The high attenuation of ICH is seen immediately from the time of haemorrhage. It decreases slowly over the subsequent weeks, until eventually a low-density cystic area remains. Resolution of haematoma density takes place from the periphery. Resolution at CT does not necessarily mean that the haematoma is absorbed but merely that it has become isodense. Enhancement may occur around a clot due to damage to the blood-brain barrier and neovascularity. At the stage where the clot is isodense or of low density there is clear danger of confusing the CT appearances with those of an abscess or tumour post-enhancement. Differentiation will then depend on history.

Subarachnoid Haemorrhage

Subarachnoid haemorrhage from any cause may be associated with a number

- Subarachnoid blood may be identified anywhere in the subarachnoid space from its high attenuation. It is usually confined to the basal cisterns.
- Intracerebral and/or intraventri 55 od.
- Areas of infarction or ischaemic low density areas.
- Hydrocephalus, as dilatation of ventricles.
- Demonstration of the causative lesions (aneurysm, angioma or tumour).

Pitfalls of CT scan

1. The scan may be normal in the first 24 hours after an ischaemic stroke before there is a visible decrease in attenuation.
2. Although haemorrhage can be seen within a few minutes as an area of increase attenuation, after a few weeks the lesion becomes cystic and of

low attenuation. Thus if CT scanning is not performed within 2 weeks, it may not be possible to distinguish between infarction and primary intracerebral haemorrhage. Here MRI is more sensitive.

3. Some patients with definite ischaemic stroke, particularly those with small deep infarcts or ischaemia in the posterior fossa, have normal CT scans, even after the first 24 hours (Brown 1992).

CHAPTER -4

MATERIALS AND METHODS

4. MATERIALS AND METHODS

4.1. Study type

Cross sectional observational stud

4.2. Study time

04 years (January, 2012 to December, 2015). It includes article reviews, development of thesis protocol, reporting and data collection, data analyses and paper writing.

4.3. Study place

Department of Neurology and Radiology, Rajshahi Medical Collage Hospital, Rajshahi, Bangladesh.

4.4. Sample size: 321

4.5. Study population

Stroke patient of both male and female attending RMCH constitute the study population.

4.6. Sample procedure

Positive sampling technique was followed. After proper diagnosis of the patient of stroke by CT they were given a serial number who justified the criteria to be selected as sample. Then the patient was selected for the study. If the patient's information was not available on follow up on 7^{th} day or 28^{th} day he or she was dropped from the study.

4.7. Selection criteria

4.7.1. Inclusion criteria

Acute stroke patients who underwent CT scan of brain and CT findings were conclusive of acute stroke.

4.7.2. Exclusion criteria

1. Patients of brain trauma, meningitis, encephalitis, TIA, Tumor,

2. Recurrent stroke.
3. Spontaneous epidural and subd 58 ıtoma.
4. Poor quality film of CT.
5. Patient who did not give the consent to participate in the study.
6. Patient who dropped from the study.
7. Lacunar infarct and multiple insults.

4.8. Categorization of the patient

The subjects of the present study were categorized into two groups. One group was haemorrhagic stroke another was ischaemic stroke. The haemorrhagic stroke was again divided into intraparenchymal haemorrhage and subarachnoid haemorrhage.

4.9. Instruments of the research

The present study utilized two main instruments,

a) CT machine and its accessories.

b) Detailed socio-demographic data and CT findings (Appendix-i).

4.10. Ethical consideration

The research/study was approved by the University of Rajshahi, Bangladesh. The aim and objectives of the study along with its procedure and benefits of the study were explained to the respondents (patients or guardians of the patients) in easy understandable native language and consent was taken from each patient/guardian (Appendix-ii).

4.11. Procedure of data collection

This study was carried out on 321 admitted stroke patients presented with symptoms of stroke and subsequently proved by CT findings. The patients/guardian was interviewed by the researcher himself by using a questionnaire (Appendix-i) and by face to face interview and follow up by

written consent was obtained for participation. Basic demographic information including age, resider l or Urban), educational attainment, occupation, family income was noted. Detailed history was taken about dietary habit (vegetarian or non vegetarian), smoking and alcohol consumption, family history of stroke, past history of hypertension, heart disease, diabetes mellitus.

4.12. CT scan of the patients

All the 321 patients who were included in the study underwent CT scanning of brain with CT scanners in the Radiology and Imaging department of Rajshahi Medical College Hospital and private clinics of Rajshahi. CT films were interpreted carefully. CT scanning of the brain of the patients was taken

by 1cm consecutive cuts with 15^0 to 20^0 angulations to canthomeatal line for the supratentorial part and for the posterior fossa 0.5 cm cut was taken.

4.13. Criteria used for clinical diagnosis of different type of stroke

Infarction

- Focal neurologic deficits, relatively less rapid progression.
- Occurring during awakened/asleep.
- Conscious/drowsy.
- History of TIA.
- Carotid bruit.

Intracerebral haemorrhage

- Focal neurologic deficits, more rapid progression.
- Headache with vomiting.
- Coma.
- Occurring during awakened particularly at stress.
- Convulsion/or neck rigidity.

Subarachnoid haemorrhage

- Intense headache.
- Neck rigidity.

4.14. CT criteria for diagnosis of different lesions in stroke patients

Infarction

The cardinal signs of CT scan of an infarct were:

i) An area of decreased attenuation within the cerebral substance.

ii) Dense middle cerebral artery.

iii) Obscuration of lentiform nucleus. Insular ribbon sign hypodensity of insular cortex.

iv) May or may not be accompanied by mass effect.

Intracerebral haemorrhage (ICH)

The CT scan signs of cerebral haemorrhage were:

i) An area of increased attenuation ranging from 50-90 Hounsfield units in brain substance.

ii) The above area surrounded by a thin low attenuation ring.

iii) May or may not be accompanied by mass effect.

Intraventricular haemorrhage

Increased density in the ventricles.

Subarachnoid haemorrhage (SAH)

The CT findings of SAH were:

Increased density in subarachnoid space. Acute SAH is typically 50-60 Hounsfield units (HU).

4.15. Measurement of outcome variables

The variables which were observed during the study were:

1. Sociodemographic:

Age, sex, education, income, food habit, smoking.

i) Signs/symptoms of stroke.

ii) Risk factors of stroke.

3. CT findings:

i) Infarcts/ICH/SAH.

ii) Sites and vascular territory of infarcts.

iii) Sites of ICH and volume.

iv) Mass effect.

v) Measurement of pineal gland and midline shift.

4.16. Data recording and analysis

All relevant information from history, clinical findings and investigations were recorded in a predesigned questionnaire which was approved by the

supervisor. After proper verification, data were coded and entered into the computer and were analyzed according to the objectives of the study by using SPSS 16. The difference were considered significant at a $p < 0.05$.

4.17. Operational Definition

Hypertension

Hypertension was considered to be present if the patients were taking antihypertensive drugs or if the systolic blood pressure > 140 mm of Hg of and diastolic blood pressure > 90 mm of Hg on three separate readings, according to JNC-8 guideline(Paul *et al*. 2014).

Diabetes

Diabetes was confirmed in patient's clinical record taking oral hypoglycemic drugs or insulin or if the fasting blood glucose was greater than7.0 mmol/L (WHO diagnostic criteria).

Smoking

A smoker was considered as one who smoked at least 1 cigarette or biri per day and continued for at least 6 months within the last 10 years from the time

considered who never smoked or smoked occasionally, not more than 1 cigarette or biri per day, less tha 62 nths or quite smoking for last 10 years (Agarwal *et a1*. 2012). Smoking was assessed as a part of questionnaire. Smoking habit was recorded as packed year which is essentially the magnitude of primary smoke exposure. Pack year was calculated by multiplying the number of packs of cigarettes smoked per day by the number of years the person has smoked.

Economic status categorization

Economical status was categorized according to per month income of each patient. According to Bangladesh Bureau of Statistics (2011), patient was considered as poor who earned upto 60000 taka/year, middle class: 60,000-

180000 taka/year and was considered as rich who earned more than 180,000 taka per year.

Cardiovascular causes

Patients were considered to have a cardiac abnormality, when they had a self reported history of myocardial infarction, coronary artery bypass grafting, angina or percutaneous transluminal angioplasty, evidence of left hyperventricular hypertrophy and atrial fibrillation if any were documented.

Family history

A positive family history was too considered if a patient had first-degree relative (parent or sibling) who had a stroke.

Dietary habits

Patients were considered as vegetarian, nonvegetarian.

Brain edema

Represents moderate amount of bulk water within acute hematomas/infarct.

Computed Tomography

Images a section or slice of the patient. This is accomplished by obtaining a

conventional tomography and radiography, X-rays do not pass through neighboring anatomy, only through the section of interest.

Estimated hematoma volume

It is computed by getting the product of L × W × AP × 0.523 where L is the vertical diameter showing hematoma on CT scan, W is the greatest transverse diameter of the bleed, AP is the greatest antero-posterior diameter of the hematoma perpendicular to W and 0.523 is a constant factor (fig. 7.).

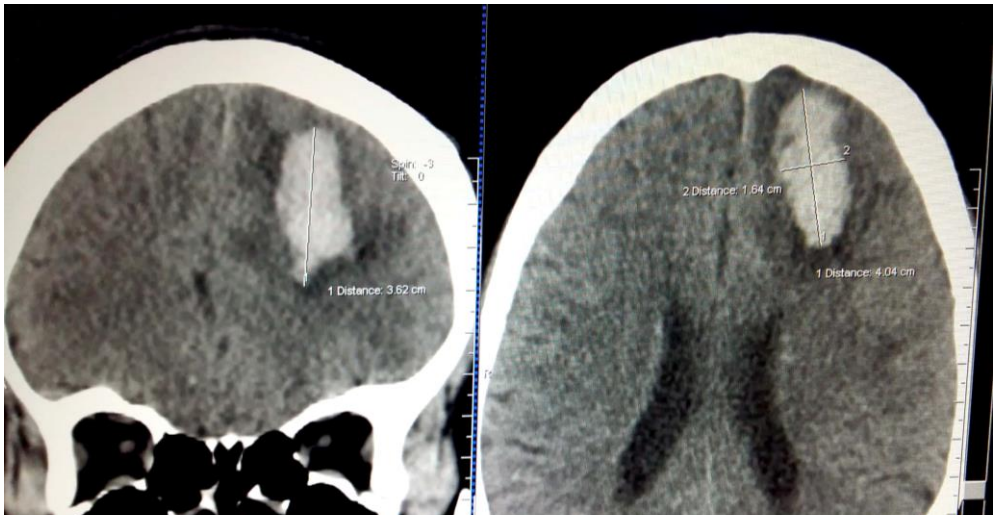

Fig. 7. Measurement of volume of intracerebral haemorrhage.

Hydrocephalus

Refers to ventricular dilatation secondary to obstructed CSF flow, decreased CSF absorption, or a combination of both.

Infarct

Refers to subtle loss of gray-white differentiation and/or sulcal effacement on CT (seen in hyperacute stage) which eventually progresses to become a hypodense area (acute/subacute stage) which is limited to the major vascular

border zone. Measurement of infarct was taken as the largest diameter (fig. 8.).

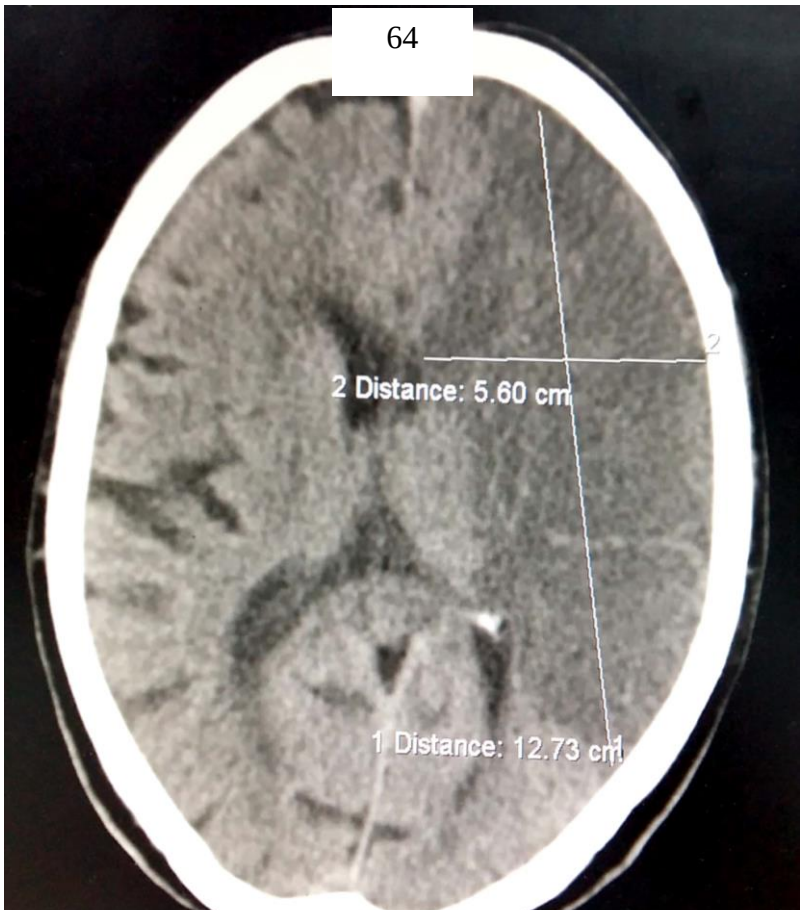

Fig. 8. Measurement of cerebral infarct.

Intraventricular hemorrhage

Presence of blood within the ventricular system which can be a complication of hypertensive hemorrhage.

Midline shift

Midline shift is the largest perpendicular distance between distance between an imaginary reference line joining the frontal crest and internal occipital protuberance, and the most shifted point of the septum pellucidum.

Assessment of midline Shift

(A) At the level of foramen of M e intracranial length ("a") and the distance from the skull to the septum pellucidum ("b") are measured. The midline shift can then be calculated as: (a/2) – b (fig. 9.) and in the same way measurement of pineal gland displacement was taken at the level of pineal gland where "a" was the intracranial length and "b" was the distance from the skull to the pineal gland (fig. 10.).

(B) By using a fine-pointed colored pencil (black) and a ruler, midline shift is measured by determining the largest perpendicular distance between an imaginary reference line joining the frontal crest and internal occipital protuberance, and the most shifted point of the septum pellucidum. By using a piece of paper with a straight edge, measurement is taken and placed

parallel to a caliper beside the chosen scanogram and result is recorded. We used the first method for measurement of midline shift.

Since manual measurement was done (without the benefit of being exact), the researcher did two trials and average of the measurements was computed and was recorded in millimeter (mm). All data collected were placed in a master list and eventually used in statistical analysis.

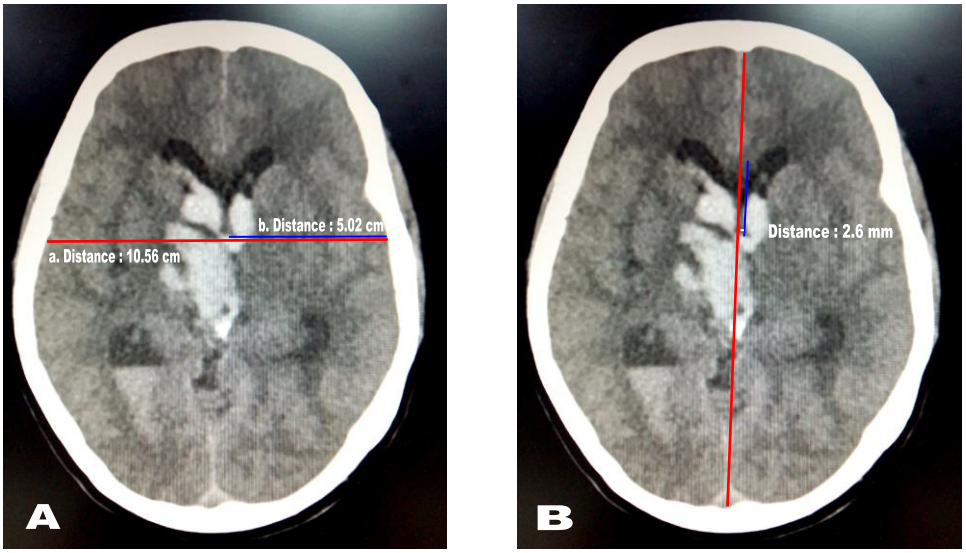

Fig. 9. Measurement of displacement of septum pellucidum.

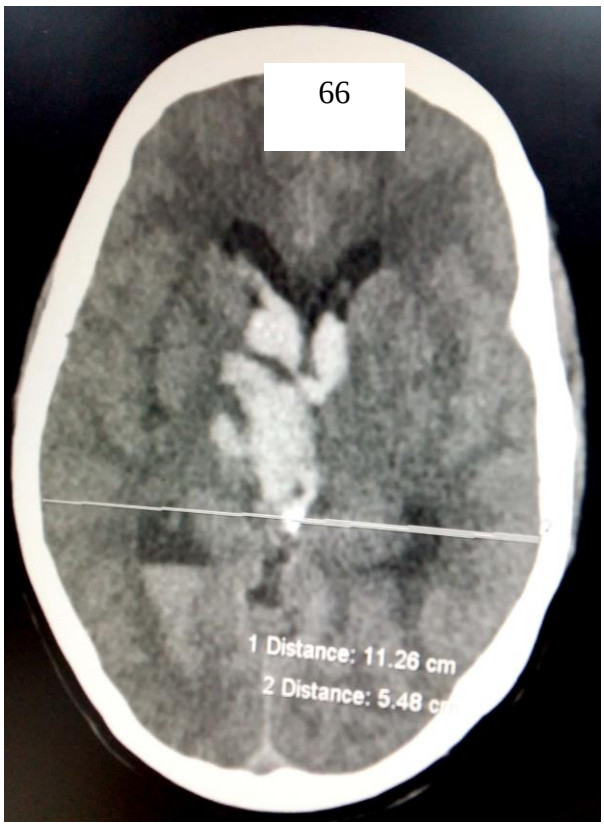

Fig. 10. Measurement of pineal gland displacement.
(1 = Intracranial diameter, 2 = Distance from the skull to pineal gland).

Subarachnoid hemorrhage

Refers to extravasation of blood into the subarachnoid space, 80% of which is caused by ruptured aneurysms.

CHAPTER -5

RESULTS AND OBSERVATIONS

5.1. Results and observations

This cross sectional observational ıs performed in different clinics of Rajshahi and the department of Radiology & Imaging, Rajshahi Medical College, Rajshahi in collaboration with the department of Neuromedicine of Rajshahi Medical College, Rajshahi from January 2012 to December 2015. The study population was comprised of total 321 patients who had definite evidence of first ever stroke and diagnosed by computed tomography (CT). Among them haemorrhage were 112 (34.89%) & infarct were 209(65.11%).

The demographic characteristics, the clinical pattern, risk factors, type of stroke and its size, volume, site, arterial territory, mass effect as determined by midline displacement as evidenced by septum pellucidum and pineal displacement with effacement of cerebral sulci and fissures were our observations. Ventricular extension of the lesion was also ascertained.

5.2. Demographic profile of stroke

No of respondent-321

Age: range 31 to 95 years

Mean age 64 ± 9.75 years

Male: Female: 193(60.12):128(39.88)

Residence: Urban 64(19.94%) & rural 257(79.06%)

Economic status

Low income group - 135(45.16%)

Middle income group -116(48.61%)

High income group - 6(06.23%)

Occupation

Farmer - 83(25.85%)

Govt. and non govt. service holder & retired-65(20.26%)

Businessman -68(21.18%)

Laborer-24(7.48%)

Others -16(4.97%)

Literacy level

Illiterate-65(20.25%)

Below primary-48(14.95%)

Primary & or high school pass-129(40.19%)

HSC pass-15(4.68%)

Graduate-64(19.93%)

5.3. Age and sex distribution

Age and sex distribution of the study population have been shown in table 3. The mean age of the study population was 64 ±9.75 years with a range of 34 to 95 years and divided into five different age groups; viz, 31- 40 years, 41-50 years, 51-60 years, 61-70 years and above 70 completed years in this

study. It was found that the highest number of the patients 118(36.76%) belongs to 7th decade. The next highest group belongs to 6th decade 103(32.09%). It was found that 54(16.82%) was above 8th decade, 39(12.15%) in 5th decade and 7(2.18%) were in 4th decade.

Among the patients, 61-70 age group range showed highest frequency for male and female. The mean age of the study population was 64 ± 9.75 years.

Table 3. Distribution of the study patients by age (years) (n=321)

Age (years)	Male		Female		Total No.
	No. of patients	%	No. of patients	%	
31-40	4	1.25	3	0.93	7(2.18%)
41-50	23	7.17	16	4.98	39(12.15%)
51-60	63	19.63	40	12.46	103(32.09%)
61-70	72	22.43	46	14.33	118(36.76%)
> 70	31	9.66	23	7.17	54(16.82%)

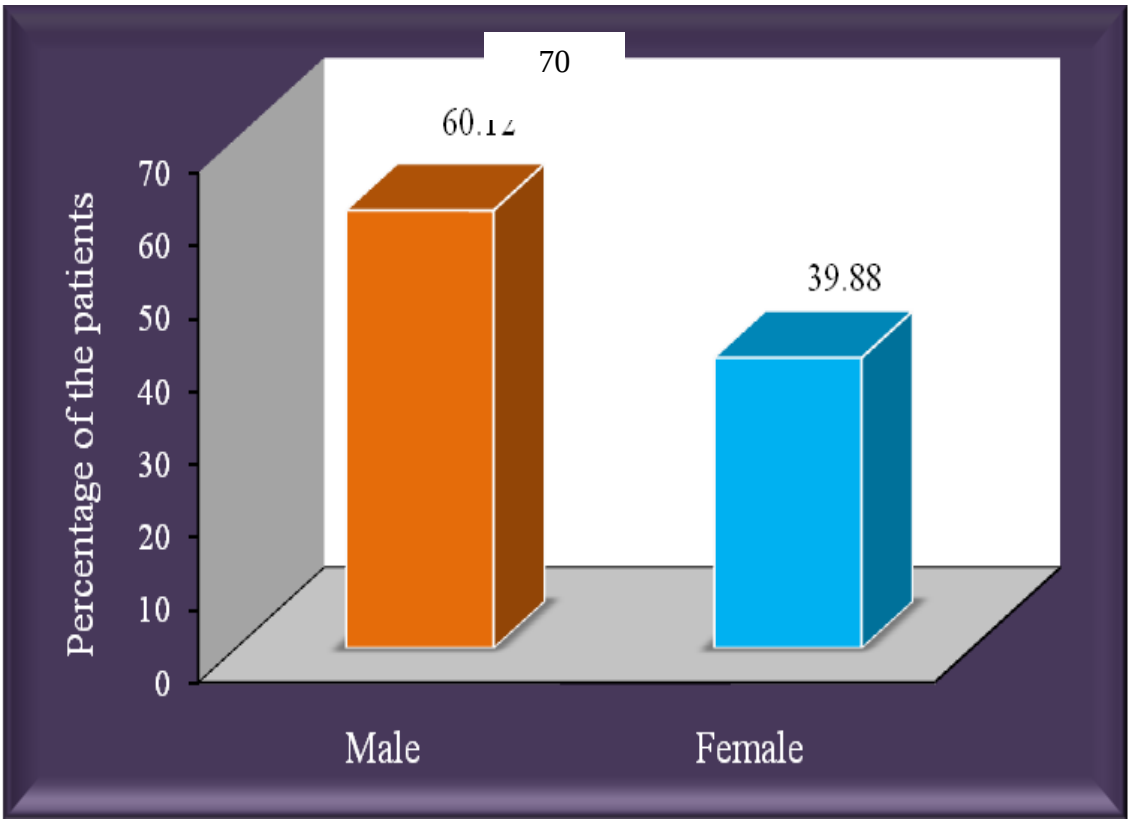

Fig.11. Figure of percentage distribution of male and female patients.

In this study, we found 193(60.12%) patients male and 128 (39.88%) female which show male preponderance and male female ratio 60.12: 39.88. Among both the male and female patients highest percentage of stroke was in 61-70 age group range (Table 3, fig. 11.).

5.4. Occupation wise distribution of the patient

There were 257 (80.06%) patients from rural area and 64 (19.94%) were from urban area. Among the respondents 83(25.85%) were farmer, 65(20.26%) were government or non-government service holders and retired persons from service. 68(21.18%) were businessmen, 65(20.26%) were housewives, 24(07.48%) were laborer and others were 16(4.97%). Among the cases highest percentage was the farmers followed by businessmen (Table 4,fig. 12.).

Table 4. Distribution of the study patients by occupational status (n=321)

Occupation	Rural		Urban		Total No.
	No. of patients	%	No. of patients	%	
Farmer	78	24.29	5	1.56	83(25.85)
Service holder & related	37	11.53	28	8.73	65(20.26)
Businessman	56	17.44	12	3.74	68(21.18)
House wife	56	17.45	9	2.81	65(20.26)
Laborer	18	5.61	6	1.87	24(7.48)
Others	12	3.73	4	1.24	16(4.97)
Total	**257**	**80.06**	**64**	**19.94**	**321**(100.00)

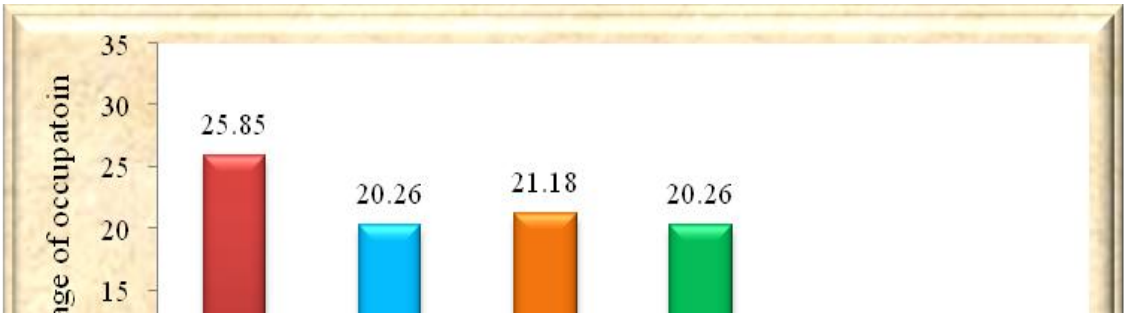

Fig. 12. Occupational status of the patient.

5.5. Economic status of the studied patient (n=321)

Economic status was assessed and 145(45.16%) patients were from low income group (upto 60000 TK per anum), 156(48.61%) were from middle

from high income group. Highest percentage of stroke was in middle class group followed by lower income g 72 ble 5, fig. 13.).

Table 5. Distribution of the study patients by economical status (n=321)

Income (yearly)	Rural (n=257)		Urban (n=64)		Total No.
	No. of patients	%	No. of patients	%	
< 60000 (Low)	135	52.53	10	15.63	145(45.16%)
60000-180000 (Middle)	116	45.14	40	62.50	156(48.61%)
>180000 (High)	6	2.33	14	21.87	20(06.23%)

n: number of patients, this income status classification was according to Bangladesh Bureau of Statistics, 2011.

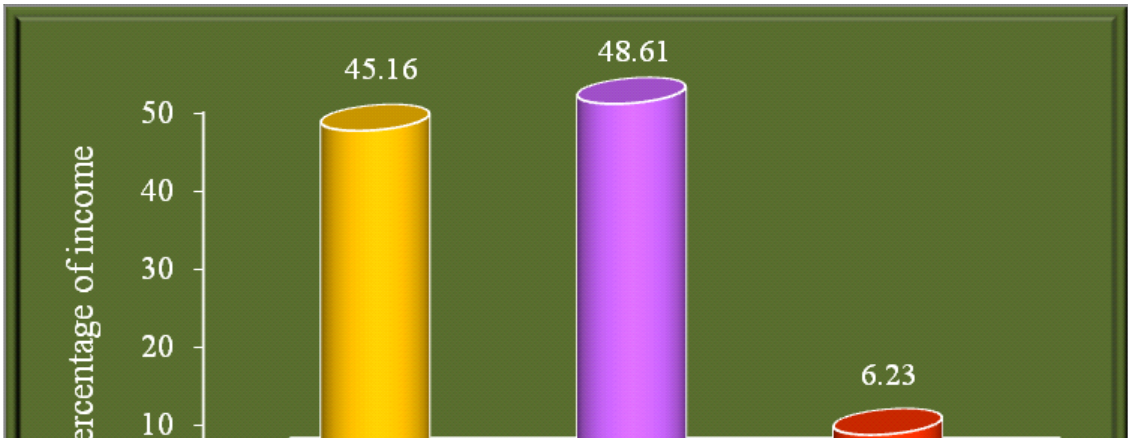

Fig. 13. Economic status of the patient showed highest percentage is middle class group.

5.6. Literacy level

The figure shows 65(20.25%) were illiterate, below primary were 48(14.95%), primary & SSC pass 129(40.19%), HSC pass 15(4.68%), graduates were 64(19.93%). Highest percentage of education was upto primary & SSC pass 129(40.19%) followed by illiterate 65(20.25%). Data

37(57.81%) among the urban people and 27(10.50%) among the rural people (Table 6, fig. 14).

Table 6. Distribution of the study patients by literacy level (n=321)

Literacy level	Rural (n=257)		Urban (n=64)		Total No.
	No. of patients	%	No. of patients	%	
Illiterate	58	22.57	7	10.94	65(20.25%)
Below primary pass	42	16.34	6	9.37	48(14.95%),
Primary and SSC pass	125	48.64	4	6.25	129(40.19%),
HSC pass	5	1.95	10	15.63	15(4.68%),
Graduation	27	10.50	37	57.81	64(19.93%).

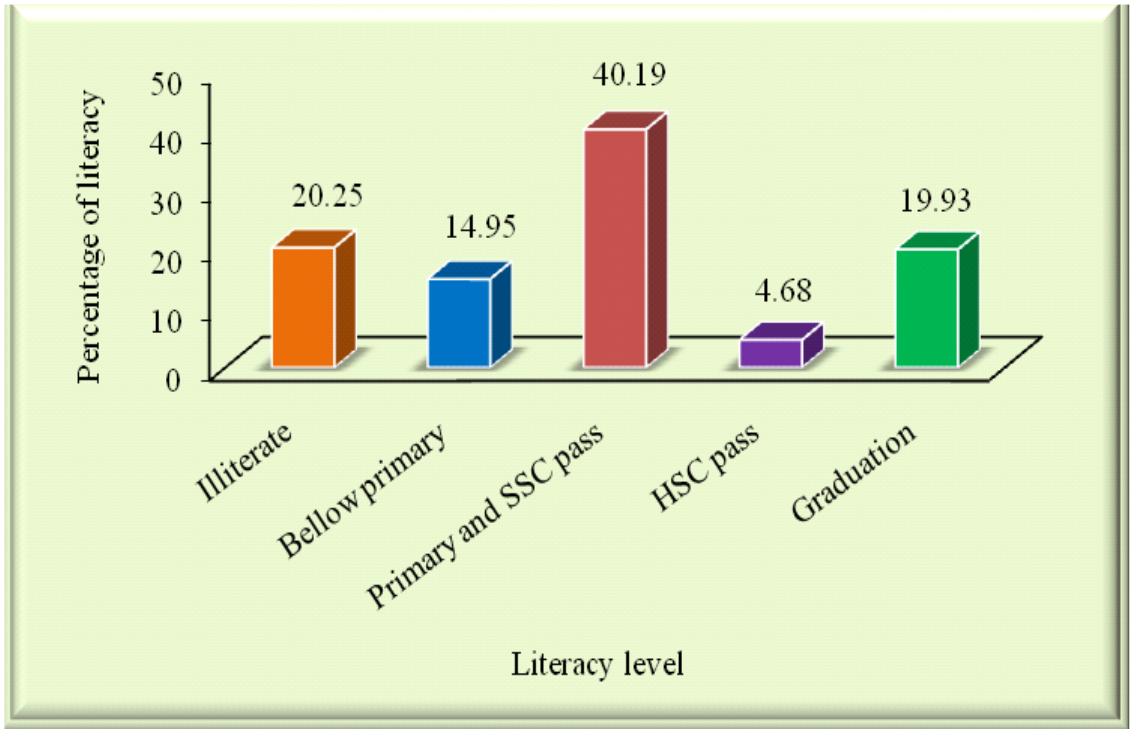

Fig. 14. Literacy level of the respondents.

5.7. Dietary habitat and alcohol ɪtion

None of the patients was foun rian. All the patients were non-vegetarian. Only 1(0.31%) patient gave the history of regular alcohol consumption. 3(0.93%) gave the history of occasional alcohol consumption which was not significant. Rest of the patients 317(98.75%) never drunk any form of alcohol in their life (Table 7 and 8).

Table 7. Distribution of the study patients by dietary habits (n=321)

Dietary habitat	No. of patients	% of patients	P value
Vegetarian	0	0.0	0.001
Non-vegetarian	321	100.0	

P value reached from chi square test. The difference was significant ($p<0.05$).

Table 8. Distribution of the study patients by alcohol consumption (n=321)

Alcohol consumption	No. of patients	% of patients
Regular	1	0.31b
Occasional	3	0.93b
Never drunk	317	98.76a

The value followed by the same letter in a column is not significantly different at P <0.01 according to Duncan's multiple range test (DMRT).

5.8. Smoking habit status of the studied subjects

It was found that 113(35.20%) of the sample population were smoker and 208(64.80%) were non smoker. Among the smokers haemorrhagic stroke were 45(39.82%) and ischaemic stroke were about 68(60.18%). It was observed that infarct was more common among the smokers (Table 9 and 10).

Table 9. Distribution of the study patients by smoking (n=321)

Smoking	N ients (n=321)	% of patients
Smoker	113	(35.20)
Non smoker	208	(64.80)

Table 10. Stroke for the smoker (n=113)

Stroke	No. of patients (n=113)	% of patients	P value
Hemorrhagic	45	39.82	0.29
Infarcts	68	60.18	

P value reached from unpaired t-test. The difference was insignificant (p>0.05) between two groups.

5.9. Family history and stroke

We found that 23(7.17%) patients had family history of stroke and 298(92.83%) had no family history of stroke (Table 11).

Table 11. Distribution of the study patients by family history of stroke (n=321)

History of stroke	No. of patients (n=321)	% of patients
Family stroke	23	7.17
No family stroke	298	92.83

5.10. Hypertension

Among 321 patients, 225 (70.09%) were hypertensive and 96(29.91%) patients were normotensive. Among the hypertensive patients 56.44% had hemorrhagic stroke and 43.56% had ischemic stroke (Table 12 and 13).

Table 12. Distribution of the study patients by blood pressure (n=321)

Blood pressure	atients (n=321)	% of patients
Hypertension	225	70.09
Normal	96	29.91

Table 13. Stroke type for the hypertension (n=225)

Hypertension patients	No. of stroke patients	% of patients	P value
Hemorrhagic (n=112)	92	82.14	0.43
Infarcts (n=209)	133	63.64	

P value reached from unpaired t-test. The difference was not significant (p>0.05) between two groups.

5.11. Diabetic Mellitus and stroke

Among the 321 stroke patients, 20.25% patients (male 56.92% & female 43.08%) were diabetic and 79.75% were non diabetic. Among the 20.25% of the diabetic patients 61.54% were ischemic stroke and 38.46% were hemorrhagic stroke (Table 14 and 15).

Table 14. Distribution of the study patients by diabetic mellitus (n=321)

Diabetic mellitus	Male (193)	Female (128)	Total No. (%)
Diabetic	40 (12.46)	25 (7.79)	65 (20.25)
Non diabetic	153 (47.66)	103 (32.09)	256 (79.75)

Table 15. Stroke type for the diabetic mellitus (n=65)

Stroke	No. of pa 77 (n=6	% of patients	P value
Infarcts	40	61.54	0.72
Hemorrhagic	25	38.46	

P value reached from unpaired t-test. The difference was insignificant (p>0.05) between two groups.

5.12. Risk factors

In the present study hypertension was found to be the most common risk factor affecting 225(70.09%) out of 321 patients. It was found 129(57.33%) were male and 96(42.67%) were female. Among 193 male patients HTN were 66.84% and among the female it was 75.00%. Diabetes mellitus and heart disease were found respectively in 65(20.25%) and 63(19.63%) among

the study patients. Among the male patients DM were 19.17% and in female it was 21.86%. Among the heart disease patients male were about 61.90% and female were 38.10%. Among 193 male patients heart disease was 20.21% and among 128 female it was 18.75%. The habit of smoking was present in 113(35.20%), family history of stroke was about 23(7.17%) and H/O oral pill 20(15.63 %.) among female (Table 16).

Table 16. Distribution of the study patients by risk factors (n=321)

Risk factors	Male(193)		Female(128)		Total No. (%) N=321
	No. of patients	(%)	No. of patients	(%)	
Hypertension	129	57.33/66.84	96	42.67/75.00	225 (100.0)/(70.09)
Smoking	111	98.23/57.51	2	1.77/1.56	113 (35.20)
Diabetes mellitus	37	56.92/19.17	28	43.08/21.86	65 (20.25)
Heart disease	39	61.90/20.21	24	38.10/18.75	63 (19.63)
H/O oral pill	-	-	20	15.63	20 (6.23)
Family					

5.13. Clinical presentation

In most of the patients of c[illegible] the presenting complaint was hemiplegia/hemiparesis 295(91.9[illegible]nich was found 194(92.82%) in ischemic stroke and 101(90.18%) in haemorrhagic stroke. Impaired consciousness was about 105(50.24%) in infarct & 90(80.36%) in haemorrhagic stroke. About 187(58.26%) had headache. Among the infarct patient 103(49.28%) had headache and among haemorrhagic stroke 84(75.00%) had headache. About 136(42.37%) had cranial nerve palsy. It was found about 119(56.94%) in infarct & 17(15.18%) in haemorrhagic stroke. Speech disturbance was 154(47.98%). It was 99(47.37%) in infarction & 55(49.11%) in haemorrhagic cases. Vertigo was 59(28.23%) in infarct & 56(50.00%) in haemorrhagic patient. Hypertension was 225(70.09%) in our study. Among the ischaemic stroke (infarct) hypertension was 133(63.64%) in haemorrhagic stroke it was 92(82.14%). Neck rigidity was 14.35% in

infarct and 62.5% in haemorrhagic stroke. Vomiting was 9.09% in infarct, 36.61% in haemorrhagic stroke (Table 17).

Table 17. Distribution of the study patients by clinical presentation (n=321)

Sign symptoms	Infarcts (n=209)		Hemorrhage (n=112)		Total No. (%) (n=321)
	No. of patients	%	No. of patients	%	
Hemiplegia	194	92.82	101	90.18	295 (91.90)
Impaired consciousness	105	50.24	90	80.36	195 (60.75)
Headache	103	49.28	84	75.00	187 (58.26)
Vomiting	19	9.09	41	36.61	60 (18.69)
Cranial nerve palsy	119	56.94	17	15.18	136 (42.37)
Speech disturbance	99	47.37	55	49.11	154 (47.98)
Vertigo	59	28.23	56	50.0	115 (35.83)

Hypertension	133	63.64	92	82.14	225 (70.09)
Neck rigidity	30	14.35	70	62.5	100 (31.15)

5.14. Type of stroke

Among 321 patients, 209(65.11%) had ischemic stroke, 99(30.84%) had parenchymal haemorrhage and 13(4.05%) had subarachnoid haemorrhage. Total amount of haemorrhagic stroke was 112(34.89%). Among the male patients 121 (62.69%) were ischaemic and 72(37.31%) were haemorrhagic stroke. Among the female patients 88(68.75%) were ischaemic & 40(31.25%) were haemorrhagic stroke (Table 18a, 18b and 18c).

Table 18a. Type of stroke of the study patients (n=321)

Type of stroke	No. of patients	% of patients
Infarcts	209	65.11
Haemorrhagic	112	34.89

Table 18b. Type of stroke (Infarcts, parenchymal and subarachnoid) of the study patients (n=321)

Type of stroke		No. of patients	% of patients
Infarcts		209	65.11
Haemorrhagic	Parenchymal	99	30.84
	Subarachnoid	13	4.05

Table 18c. Distribution of the study patients by type of stroke (n=321)

Stroke	Male (n=193)	Female (n=128)	P value

	No. of patients	%	No. of patients	%	
Infarcts	121	62.69	88	68.75	0.265
Haemorrhagic	72	37.31	40	31.25	

P value reached from chi square test. The difference was not significant (p>0.05).

5.15. Site of infarcts

Site of infarcts was assessed. Out of 209 patients of infarction the commonest site of infarction was basal ganglia and paraventricular region 62(29.67%) followed by parietal region 55(26.32%) followed by internal capsule 23(11.00%). The next common location was frontoparietal region 19(9.09%),

lobe 5(2.39%) and brainstem and temporo-parietal lobe 4(1.91%) each (Table 19).

Table 19. Sites of infarct lesion 81

Site	Number (N=209)	Percentage (%)
Parietal region	55	26.32
Frontal lobe	17	8.13
Occipital lobe	5	2.39
Basal ganglia and Paraventricular	62	29.67
Fronto-parietal	19	9.09
Temporo-parietal	4	1.91
Internal capsule	23	11.00
Thalamus	8	3.83
Brainstem	4	1.91

Cerebellum	12	5.74
Total	**209**	**100**

5.16. Arterial territory and infarcts

Majority of the ischemic stroke occurred in the territory of middle cerebral artery (MCA) and was 76.08%. Involvements of anterior and posterior cerebral artery were found in 10.05% & 5.74% subjects respectively. It was about 8.13% in vertebrobasillar circulation (Table 20, fig.15.).

Table 20. Arterial territory and infarcts (n=209)

Territory	No. of patients	% of Patients
MCA	159	76.08
ACA	21	10.05
PCA	12	5.74
VBA	17	8.13

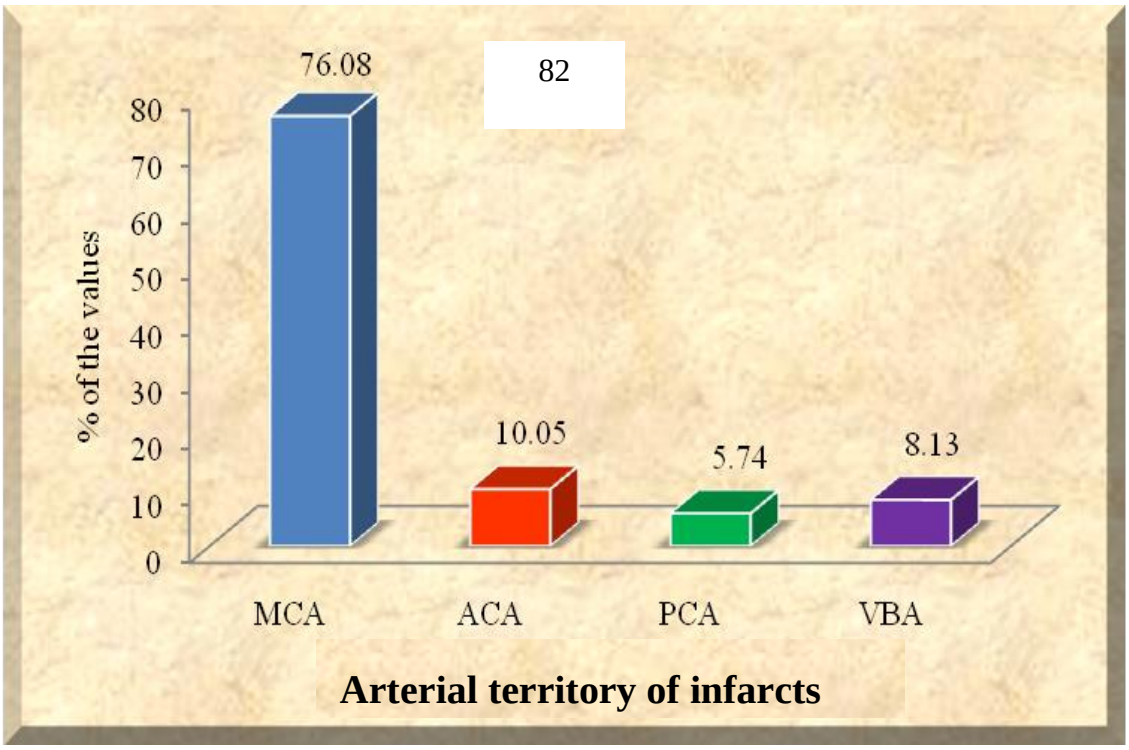

Fig. 15. Arterial distribution of infarct.

5.17. Diameter of infarct and their relationship to mortality

CT detected infarct was <2 cm in 78(37.32%) patients and mortality was 1(1.28%) within 7 & 28 days in each period. Infarct size between 2- 5 cm was 106(50.72%) and death was 23(21.70%) & 13(12.26%) within 7 & 28 days respectively and total death was 36(33.96%) within 28 days. Infarct size >5 cm was 25(11.96%) and death was 18(72.00%) & 5(20.00%) within 7 & 28 days respectively and total death was 92.00% within 28 days and it was statistically significant (p<0.05). This indicates that with increasing size of infarct, mortality will increase and it has an impact on the prevalence of death (Table 21, fig. 16.).

Table 21. Diameter and mortality of infarct (n=209)

Diameter (cm)	No. of patient (%)	Within 7	ᵗʰ to 28th days	Total death (Within 28 days)	P value
		No. of death (%)	No. of death (%)		
<2	78 (37.32)	1 (1.28)	1 (1.28)	2 (2.56)	0.001
2-5	106 (50.72)	23* (21.70)	13 (12.26)	36 (33.96)	
>5	25 (11.96)	18 (72.0)	5 (20.0)	23 (92.0)	
Total	**209 (100)**	**42 (20.10)**	**19 (9.09)**	**61 (29.19)**	

P value 0.001 means significantly higher at (p<0.05).

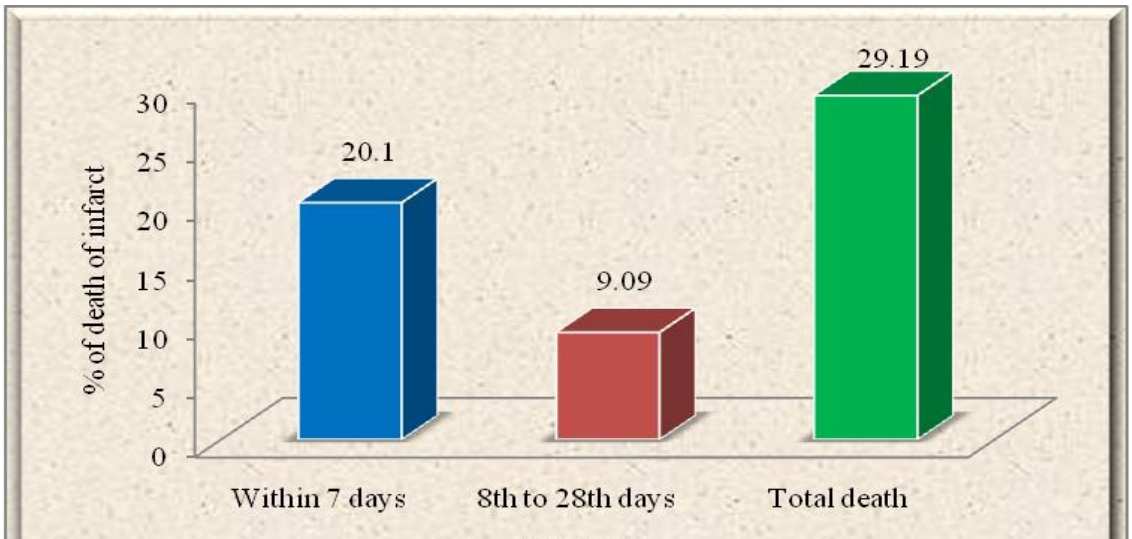

Fig. 16. Infarct diameter vs mortality.

5.18. Mass effect of infarct--mortalitv by pineal gland and septum pellucidum displacement

5.18a. Pineal gland displacement

Mass effect was determined by displacement of midline as evidenced by pineal gland & septum pellucidum displacement, effacement of cerebral sulci & fissure. But mortality was influenced by pineal gland & septum pellucidum displacement.

CT detected pineal gland without displacement or displacement upto 3 mm was 167(79.90%) & death was 14(8.38%) & 10(5.99%) within 7 & 28 days respectively and total death was 24(14.37%) within 28 days. Pineal gland displacement >3 mm was found in 42(20.10%) & death was 28(66.67%) & 9(21.43%) within 7 & 28 days respectively and total death was 37(88.09%) within 28 days and it was statistically significant ($p<0.037$). The highest number of death was from pineal gland displacement > 3 mm and within 7 days. (Table 22, fig.17.).

Table 22. Mass effect of infarct mortality by pineal gland displacement (n=209)

Pineal gland displacement	No. of patient 209(%)	Within 7 days (n=42)	8th to 28th days (n=19	Total death (Within 28 days)	P value
		No. of death (%)	No. of death (%)		
Without displacement or upto 3 mm	167 (79.90)	14 (8.38)	10 (5.99)	24(14.37%)	0.037
> 3 mm	42 (20.10)	28 (66.67)	9 (21.43)	37(88.09%)	

P value reached from chi square test. The difference was significant ($p<0.05$) between two groups.

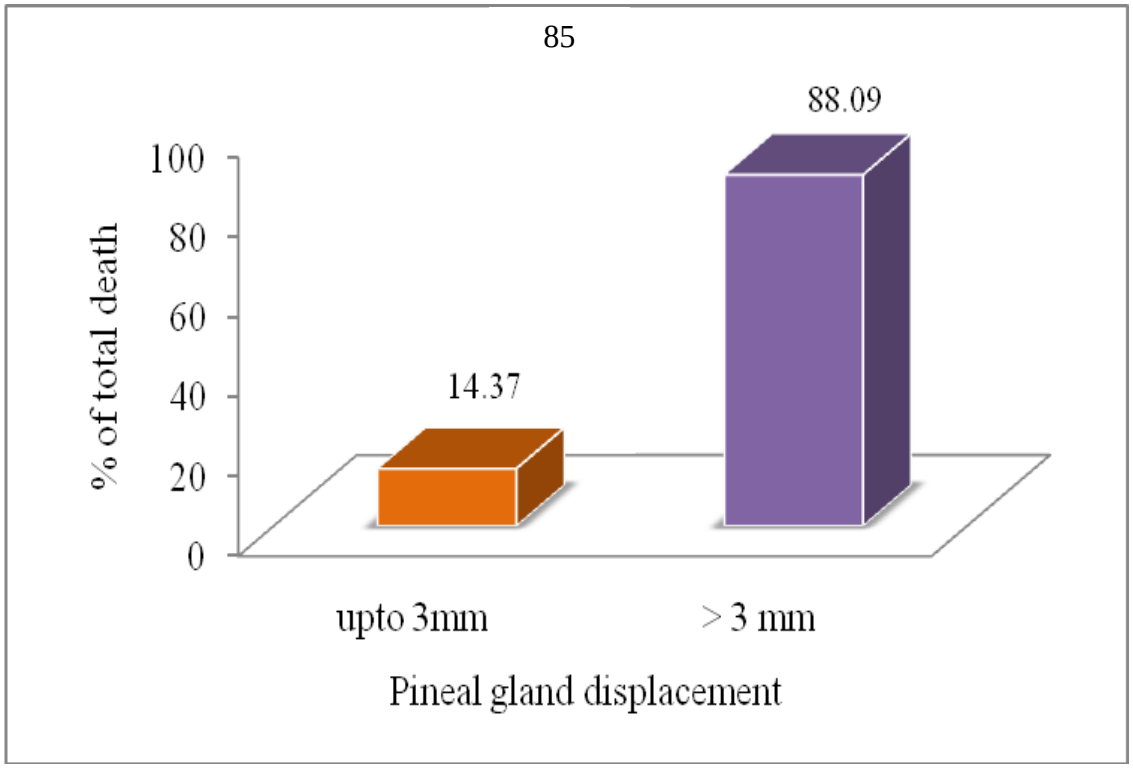

Fig. 17. Infarct pineal gland displacement in infarct vs mortality.

5.18b. Septum pellucidum displacement

CT detected septum pellucidum without displacement or displacement upto 5 mm was 173(82.76%) and death was 12(6.94%) & 14(8.09%) within 7 & 28 days respectively and total death was 26(15.03%) within 28 days. Septum pellucidum displacement > 5 mm was found in 36(17.22%) and death was 30(83.33%) & 5(13.89%) within 7 & 28 days respectively and total death was 35(97.22%) within 28 days and it was statistically significant (p=0.014). The highest mortality was from septum pellucidum displacement > 5 mm and within 7 days (Table 23, fig. 18.).

Table 23. Mass effect of infarct mortality by septum pellucidum displacement (n=209)

Septum pellucidum displacement	No. of patient (%)	Within 86 (n=	th to 28th days (n=19)	Total death (Within 28 days)	P value
		No. of death (%)	No. of death (%)		
Without displacement or upto 5 mm	173 (82.76)	12 (6.94)	14 (8.09)	26(15.03%)	0.014
> 5 mm	36 (17.22)	30 (83.33)	5 (13.89)	35(97.22%)	

P value reached from chi square test. The difference was significant (p<0.05) between two groups.

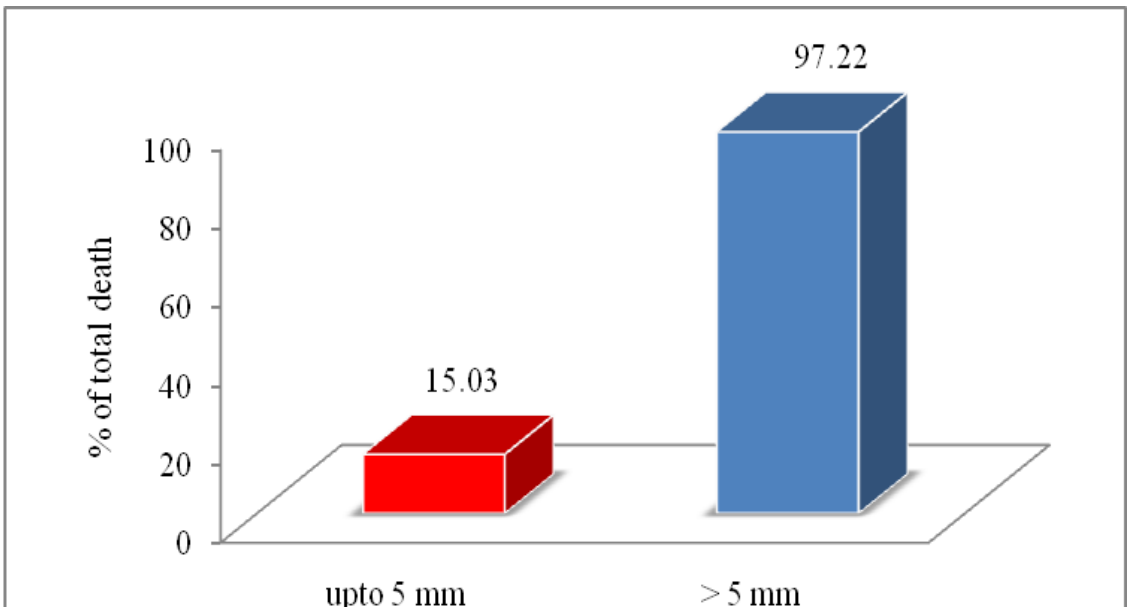

Fig. 18. Septum pellucidum displacement in infarct vs mortality.

5.19. Site of haemorrhage and mortality

The site of ICH is more common in capsulogangloinic region 61(61.62%) followed by lobar 17(17.17%). Next common site is thalamus 10(10.11%), cerebellum 7(7.07%), brainstem 4(4.04%). In capsuloganglionic region death was 21(34.42%) & 10 (16.39%) in 7 and 28 days respectively and total death

total death was 2(11.76%) in lobar haemorrhage. In thalamic region death was 5(50.00%0 & 2(20.00%) in 7 days respectively and total death was 7(70.00%). In brainstem haemorrhage death was 50.00% and 25.00% in 7 days & 28 days respectively and total death was 75.00%. In cerebellar haemorrhage no death was found in 7 days but 14.29% death found in 28 days. Total death was 44(44.44%) in 28 days and most common site for death was brainstem, thalamus and capsuloganglionic region in order of frequency. P value was significantly higher and it was 0.018 (Table 24, fig. 19.).

Table 24. Mortality by site (n=99)

Site	No. of patient (%)	Within 7 days	8th to 28th days	Total death (Within 28 days)	P value
		No. of death (%)	No. of death (%)		
Capsulogangloinic	61 (61.62)	21 (34.42)	10 (16.39)	31 (50.81)	0.018
Lobar	17 (17.18)	1 (5.88)	1 (5.58)	2 (11.76)	
Thalamus	10 (10.11)	5 (50.0)	2 (20.0)		
Cerebellum	7 (7.07)	0 (0.0)	1 (14.29)	1 (14.29)	

Brainstem	4 (3.57)	2 (50.0)	1 (25.0)	3 (75.0*)	
Total	**99 (100)**	**29 (29.30)**	**15 (15.15)**	**44 (44.44)**	

P value 0.018 means significantly higher at ($p<0.05$).

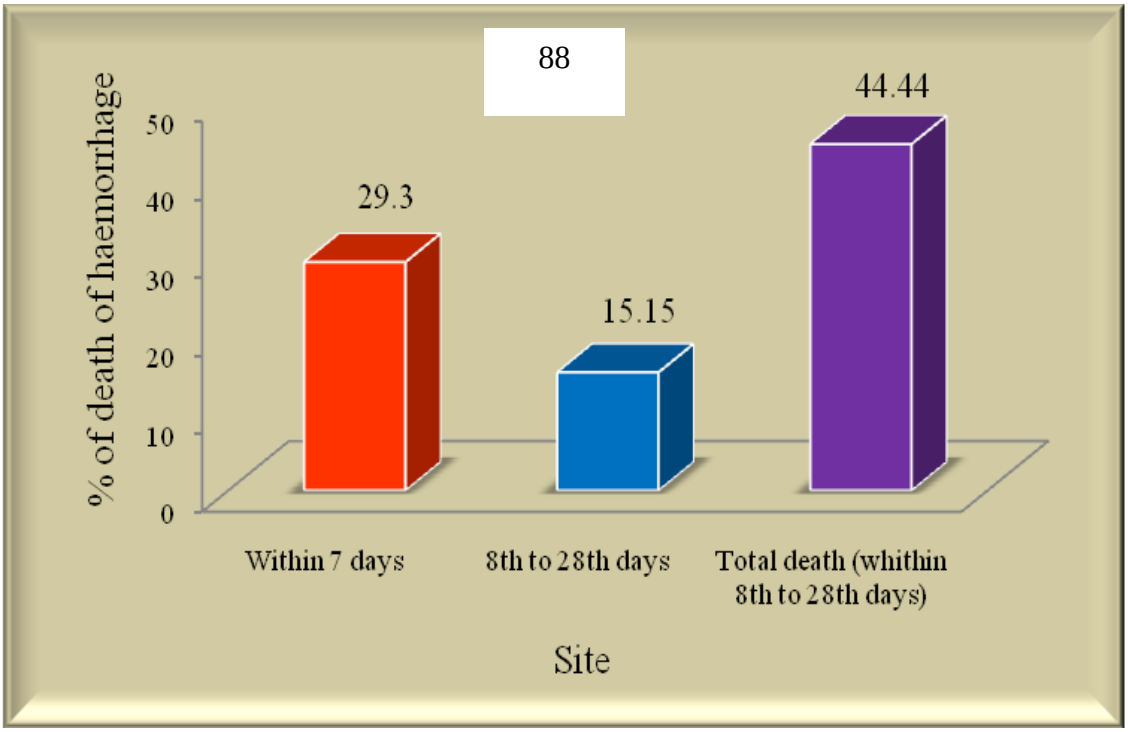

Fig. 19. Relation of haemorrhage site vs mortality.

5.20. Volume of haemorrhage and mortality by volume of ICH

CT detected haemorrhage upto 40 ml was 45(45.45%) patients and mortality was 3(6.67%) & 2(4.44%) within 7 & 28 days period respectively and total

death was 5(11.11%). Amount of haemorrhage 41 to 60 ml was 23(23.23%) patients and mortality was 6(26.09%) & 5(21.74%) within 7 & 28 days period respectively and total death was 11(47.83%). 61-80 ml hematoma was found in 19(19.19%) and mortality was 10(52.63%) & 7(36.84%) within 7 & 28 days period respectively and total death was 17(89.47%). Haematoma volume >80 ml was in 12(12.12%) patients and mortality was 10(83.33%) & 1(9.67%) within 7 & 28 days respectively and total death was 11(91.67%) (Table 25).

Table 25. Volume of haemorrhage and mortality of ICH (n=99)

Volume (ml)	No. of patient 99(%)	Within 7 day (n=29)	th days (n=15)	Total death (Within 28 days)	P value
		No. of death (%)	No. of death (%)		
Upto 40	45 (45.45)	3 (6.67)	2 (4.44)	5 (11.11)	0.011
41-60	23 (23.23)	6 (26.09)	5 (21.74)	11 (47.83)	
61-80	19 (19.19)	10 (52.63)	7 (36.84)	17 (89.47)	
>80	12 (12.12)	10* (83.33)	1 (8.33)	11 (91.67)	

P value 0.011 means significantly higher at ($p<0.05$).

5.21. Mass effect of haemorrhage-mortality by pineal gland and septum pellucidum displacement.

5.21a. Pineal gland displacement in haemorrhage

Mass effect was determined by pineal gland and septum pellucidum displacement, effacement of ventricles and cerebral subarachnoid spaces.

Mortality was influenced by pineal gland and septum pellucidum displacement and intraventricular extension of haemorrhage.

CT detected pineal gland without displacement or displacement upto 3 mm was 60(60.61%) and death was 7(11.67%) & 6(10.00%) within 7 & 28 days respectively and total death was 13(21.67%) within 28 days. Pineal gland displacement > 3 mm was found in 39(39.39%) and death was 22(56.41%) & 9(23.08%) within 7 & 28 days respectively and total death was 31(79.49%) within 28 days and it was statistically significant (p value 0.028). The highest number of death was from pineal gland displacement > 3 mm and within 7 days (Table 26, fig. 20.).

Table 26. Mass effect of haemorrhage mortality by pineal gland displacement (n=99 90

Pineal gland displacement	No. of patient 99(%)	Within 7 days (n=29)	8th to 28th days (n=15)	Total death (Within 28 days)	P value
		No. of death (%)	No. of death (%)		
upto 3 mm	60 (60.61)	7 (11.67)	6 (10.00)	13(21.67%)	0.028
> 3 mm	39 (39.39)	22 (56.41)	9 (23.08)	31(79.49%)	

P value reached from chi square test. The difference was significant ($p<0.05$) between two groups.

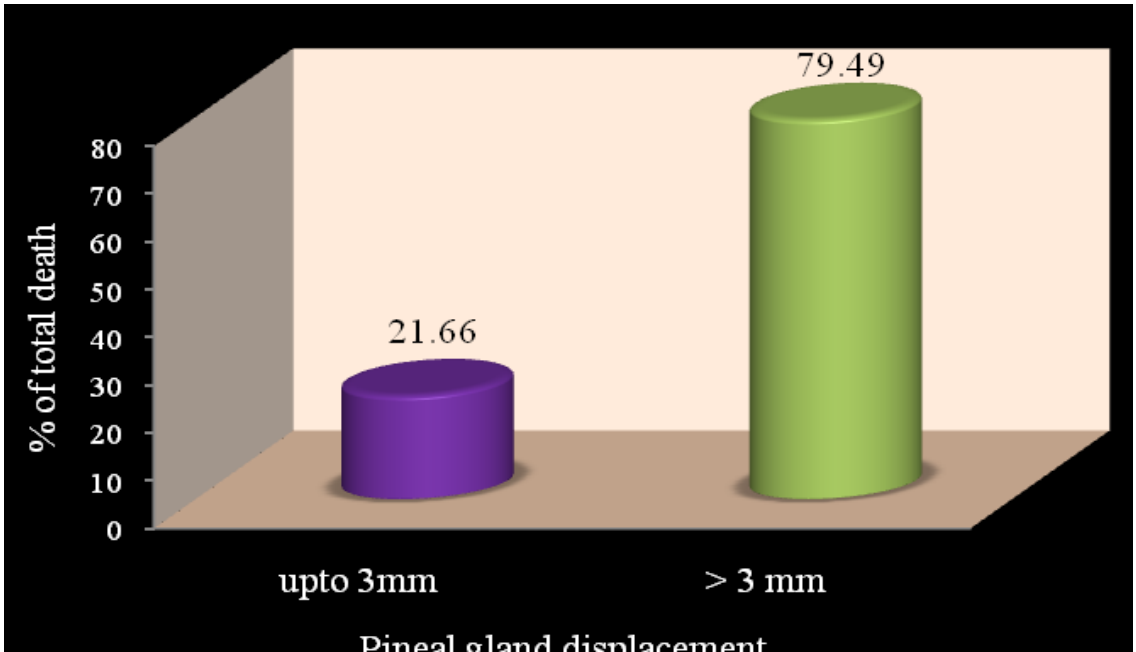

Fig. 20. Relation of pineal gland displacement vs mortality in haemorrhage.

5.21b. Septum pellucidum displacement in haemorrhage

Septum pellucidum displacement upto 5 mm or without displacement was 70 (70.54%) patients and death was 15(21.43 %) & 8(11.43%) within 7 & 28 days respectively and total death was 23 (32.86%) within 28 days. Septum pellucidum displacement > 5 mm was found in 29(29.29%) and death was 14(48.28%) & 7(24.14%) within 7 & 28 days respectively and total death was 21(72.42%) within 28 days and it was statistically significant (p value 0.033)

Table 27. Mass effect of haemorrhage mortality by septum pellucidum displacement (n=99)

Septum pellucidum displacement	No. of patient (%)	Within 7 days (n=29)	8th to 28th days (n=15)	Total death (Within 28 days)	P value
		No. of death (%)	No. of death (%)		
upto 5 mm	70 (70.71)	15 (21.43)	8 (11.43)	23 (32.86%)	0.033
> 5 mm	29 (29.29)	14 (48.28)	7 (24.14)	21(72.42%)	

P value reached from chi square test. The difference was significant (p<0.05) between two groups.

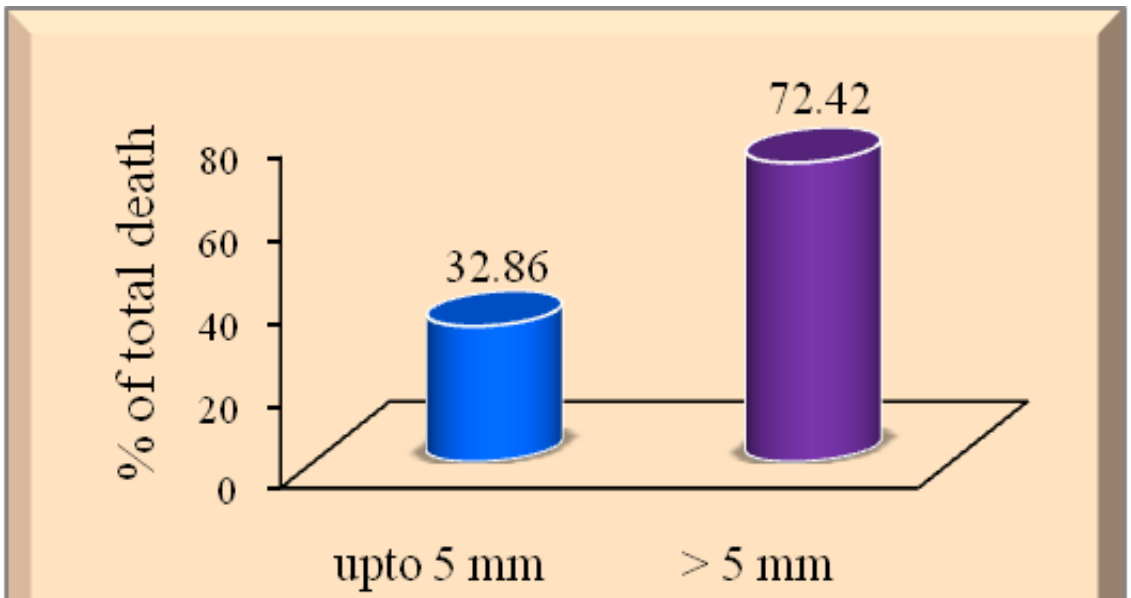

Fig. 21. Relation of septum pellucidum displacement vs mortality in haemorrhage.

5.22. Mortality by intraventricular extension of ICH

Intraventricular extension of haemorrhage was 35(35.35%) and death was 22(62.86%) & 9(25.71%) within 7 & 28 days respectively and total death was 31(88.57%) within 28 days. There was no extension of haemorrhage in

respectively and total death was 13(25.71%) within 28 days. It was statistically significant (p value 0. 92 ole 28, fig. 22.).

Table 28. Ventricular extension of the haemorrhage (n=99)

Ventricular extension	No. of patient (%)	Within 7 days (n=31)	8th to 28th days (n=18)	Total death (Within 28 days)	P value
		No. of death (%)	No. of death (%)		
No	64 (64.65)	7 (10.94)	6 (9.38)	13(25.71%)	0.017
Yes	35 (35.35)	22 (62.86)	9 (25.71)	31(88.57%)	

P value reached from chi square test. The difference was significant ($p<0.05$) between two groups.

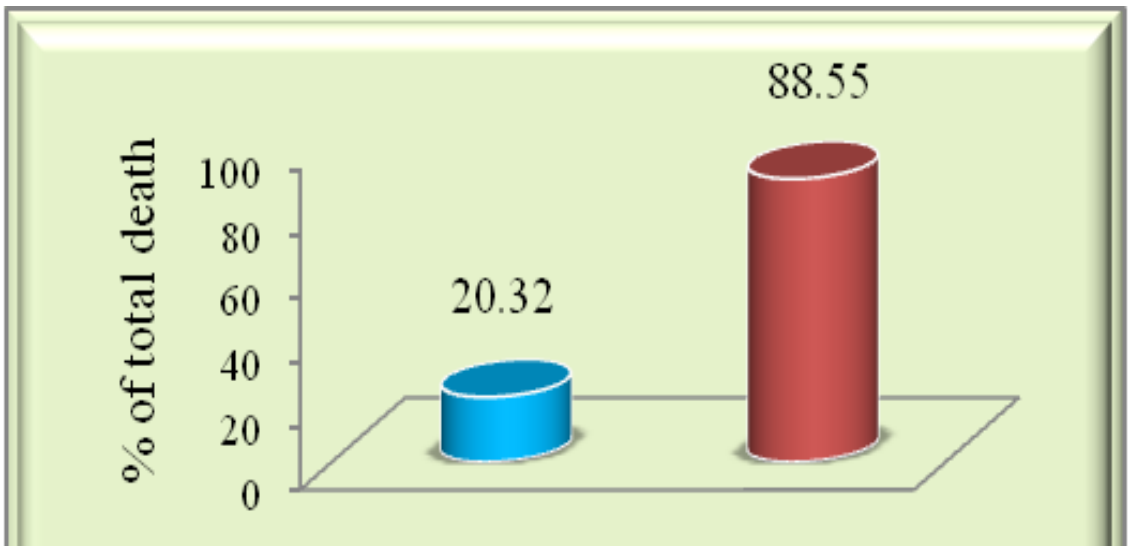

Fig. 22. Relation of intraventricular extension of haemorrhage vs mortality in haemorrhagic stroke.

5.23. Mortality by hydrocephalus in haemorrhagic stroke

Hydrocephalus present in haemo 93 stroke was 43(43.43%) and death was 21(48.84%) & 11(25.58%) within 7 & 28 days respectively and total death was 32(74.42%) within 28 days. There was no hydrocephalus in 56(56.57%) and death was 8 (14.29%) and 4(7.14%) within 7 & 28 days respectively and total death was 12 (21.43%) within 28 days. P value was 0.018 and it was statistically significant (Table 29).

Table 29. Correlation of hydrocephalus and mortality in haemorrhage stroke (n=99)

Hydrocephalus	No. of patient 99(%)	Within 7 days (n=29)	8th to 28th days (n=15)	Total death (Within 28 days)	P value
		No. of death (%)	No. of death (%)		
No	56 (56.57)	8 (14.29)	4 (7.14)	12 (21.43)	0.018
Yes	43 (43.43)	21 (48.84)	11 (25.55)	33 (76.74)	

P value reached from chi square test. The difference was significant ($p<0.05$) between two groups.

5.24. Mortality in subarachnoid haemorrhage

Haemorrhagic stroke comprised 112(34.89%) patients of which 99(88.39%) were intracerebral haemorrhage and 13(11.61%) were subarachnoid haemorrhage. Among the patients of this group death was 7(53.85%) & 2(15.38%) within 7 & 28 days respectively and total death was 9(69.23%) within 28 days. P value was 0.011 and it was significant (Table 30).

Table 30. Correlation of subarachnoid haemorrhage and mortality (n=13)

SAH	Within 7 days	8th to 28th days	Total death (Within 28 days)	P value
	No. of death (%)	No. of death (%)		
13	7 (53.85)	2 (15.38)	9 (69.23)	0.011

P value reached from ungrouped t-test. The difference was significant ($p<0.05$).

CHAPTER -6

DISCUSSION

6.1. Discussion

In the present study, a total numb :T diagnosis stroke patients were selected from different clinics of Rajshahi and the department of Radiology & Imaging, Rajshahi Medical College, Rajshahi in collaboration with department of Neuromedicine of Rajshahi Medical College, Rajshahi from January 2012 to December 2015. Diverse clinical presentation of stroke, presence of various risk factors, CT findings, type of stroke and correlation between Computed Tomography Scan findings with mortality within 7 days & 28 days were studied. Different risk factors were recorded and analyzed for their association with stroke. Among the non-modifiable risk factors, age and sex were studied. Elderly people are the most vulnerable group for developing stroke.

Stroke is an important public health problem and a burden to community and health care providers. It is the third most common causes of death after ischemic heart disease and cancers not only in the developed countries but

also worldwide. Stroke occurs predominantly in middle and late years of life. Several line of evidence suggests that hypertension, diabetes mellitus, hyperlipidemia, ischemic heart disease, atrial fibrillation, smoking and carotid stenosis are contributing factors for stroke. The prevalence of risk factors varies in different countries. Despite numerous prior studies of stroke risk factors, many remains unknown and several inconsistencies continue to exist. However, the minor differences in the prevalence of stroke risk factors in different communities are probably due to differences in culture, disease patterns, living habits and distribution of various ethnic groups.

In the present study, the male preponderance was noted. It was 60.12% in male & 39.88% in female. In a study Zafar and Khan (2008) found 62% male & 38 % female in hypertensive stroke patients. Frank *et al.* (1984) found

found male to female ration of 1.9:1. In another study, the male female ratio was found to be 1.6:1 (Davies *et* [96]). Kumar *et al.* (2016) in study of ischemic stroke found male and female ratio was 97:23 (N=120, male: 97, female: 23). Hossain *et al.* (2011) reported most of the patients suffering from stroke were male (74%) and male to female ratio was 2.8:1. Chowdhury *et al.* (1990) also reported male female ratio was 2.6:1. In case of hemorrhagic stroke, incidence was more in male (15.75%) with male female ratio 3:1. In case of ischemic stroke, incidence in male was 57.5% (46) with male female ratio 1.35:1(Siddique *et al*. 2009). Bhowmik *et al.* (2014) reported 68% were male and the rest 32% were female.

The present study differs with a previous study of Alamgir *et al.* (1995) which showed male: female ratio was 4:1.

The higher preponderance of males in this study may be due to cultural attitude of our society that the females are less frequently brought to the

hospital than the males and male are more affected than female. This needs to be corroborated with studies conducted in other service institutes.

The higher male preponderance in this study may also be due to the fact that women are neglected part of the society and they are not brought to hospital if not otherwise seriously ill.

Total studied subjects were 321. Among the studied subjects majority (n=118, 36.76%) were 61-70 years of age, next were 51-60 years (n=103, 54, 32.09%) and then >70 years (n=54, 16.83%). The mean age of the study population was 64 ± 9.75 year.

Frequency of stroke rises exponentially with increasing age. Stroke is less common below the age of 40 years (Clark, 1990) which is also reflected in the present study where about 275(85.66%) affected patients were over 50

findings are also consistent with that of another study (Bashar *et al.* 1992). The mean age in both male and : 97 as the same 71 years (Frank *et al.* 1984) and the mean age of the patients was 63.3 ± 11.1 years with the range of 32- 75 years (Haque *et al*. 2013). Singh *et al.* (2006) reported the age of the haemorrhagic stroke ranged from 25 to 85 years with a mean age of 58.6 ± 12 years. Male-female ratio was 2.6:1. Majority of the cases belonged to the age group of 51 to 60 years (31%). The number of cases between 41 to 70 years represented 78% of all cases. Hossain *et al.* (2011) found majority of the study subject (94%) were above the age of 40 years and the peak incidence was between 51 to 70 years (69%).

In a study done by Bhowmik *et al.* (2014) the mean age was 61.45 years with standard deviation of mean (SD) ± 11.65 years and their age ranged from 35 to 84 years. Majority (35%) of the respondents were found in the age group of 50-59. About 8% subjects were found in 30-39 years of age group. Twenty

four (24%) subjects had age between 60-69 years. Ten (10%) subjects belonged to 80 years and above age groups.

In an ischemic stroke study, Kumar *et al.* (2016) found in age group 33 to 45 years, there were 12 male and 2 female. Total percentage was 14 (11.66 %). In age group ≥46 years, male was 80 and female was 20. And total percentage was 100(83.33%). Aho *et al.* (1980) found the peak incidence was at or above the age of 85 years. This is contradicted with our study. This discrepancy with the present time is that a small portion of the population of our country survives upto that age.

Socioeconomic basis of stroke was studied. We found middle class group was most affected in stroke. It is 156(48.60%) in the middle class group. Economic status in higher group is 145(45.17%) and lower group is 20(6.23%). Kumar *et al.* (2016) showed that middle class people were

the most sufferer(n = 66, 55%). This study correlates with that of chapman (1966). From this study we get [98] ession that poor and rich are less sufferer from stroke. But this may not be true in case of poor as many of the patients are not brought to hospital. So the actual trend of incidence might be different.

Hossain *et al.* (2011) reported the low-income group (monthly income TK <5000) comprised the majority (47%). Hart *et al.* (2000) concluded that poor socioeconomic circumstances were associated with greater risk of stroke, which was also found in other studies (Casper *et al.* 1991, Shaper 1991).

In this study, literate group comprised 256(79.75%) and illiterate was 65(20.25%). Of the literate group, 48(14.95%) patients received primary & 129(40.19%) secondary schooling, 15(4.68%) patients received college education and only 64(19.93%) went to university or similar institutions.

In a study done by Hossain *et al.* (2011) literate group comprised 63%. Of the literate group, 31% patients received schooling, 19% patients received college education and only 13% went to university or similar status institutions.

In occupational category, farmers 83(25.85%), service holders 65(20.26%), businessmen 68(21.18%), laborer 24(7.48%) and house wives 65(20.26%) were affected by this disease. Our study correlates with the study done by Hossain *et al.* (2011) who reported businessmen (17%) in male population and house wives (16%) in female population were affected by this disease.

The present study showed that the study subjects were from both urban and rural areas with rural dominance (80.06%). This indicates that incidence of stroke is common in rural population which was contradicted by the study of Bashar *et al.* (1992) who showed mainly urban preponderance and in another study done by Hossain *et al*. (2011). It is showed that the study subjects were

reason might be that, the study was done in the hospitals of Dhaka and Mymensing Medical College Ho 99 MCH) respectively where mostly the urban population could avail the hospital facilities due to economic condition and large city areas. The present study was conducted in RMCH, Rajshahi which covers a wide range of mainly rural areas of Rajshahi and neighbouring districts.

The influence of different risk factors in stroke has shown considerable variation in different studies. In the present study 225 (70.09%) cases were hypertensive. Known hypertension was 188(58.56%), and 37(11.53%) more patients showed persistent high blood pressure during admission. It was found 129(57.33%) in male and 96(42.67%) in female among the total 321 population but male was 129(66.84%) hypertensive and female was 96(75.00%) hypertensive among the male and female patients respectively.

Alamgir and Mannan (1975) showed 58% of the patients of stroke were hypertensive. Kumar *et al.* (2016) reported out of 120 ischemic patients, 95(79.17%) patients were risk factor hypertension. Bashar *et al*. (1992) found 36% stroke patients were hypertensive. Hayee *et al.* (1999) found that 52.11% were hypertensive in stroke patients.

Singh *et al.* (2006) stated hypertension was found to be the commonest risk factor (78% of the cases) in hemorrhagic stroke. Similar observation was reported by Weisberg (1979) in 81%, by Douglas *et al.* (1982) in 80% and 75% of ICH by Scott *et al*. (1985). Rathore *et al.* (2011) reported 75.8% patients were hypertensive.

In this study 65(20.56%) of the patients were diabetic. Among the diabetic patients 37(56.92%) were male and 28(43.08%) were female. Among the male patients 37(19.18%) were diabetic and among the female patients 28(21.86%) were diabetic. Zafar and Khan (2008) found diabetes mellitus

diabetic. About 23% stroke cases were found to be diabetic by Bashar *et al.* (1992). Out of 120 patients, [illegible] patients had risk factor diabetes mellitus (Kumar *et al.*, 2016). Hossain *et al.* (2011) showed that 21% of the stroke patients were diabetic. Similar percentage also found with a study in India done by Dhamija *et al.* (1998). In BIRDEM a study on 165 cases of diabetic patients, all of them developed stroke in less than 10 years duration (Latif, 1990). It is difficult to assess which of the risk factors has predominant role on stroke. But when all these risk factors are present, the relative risk of suffering from stroke is greater (Alam, 1999).

Diabetes mellitus has long been recognized as a risk factor for vascular disease as well. It was diabetes mellitus (15% and 26.25%) in haemorrhagic and ischemic stroke respectively (Siddique *et al.* 2009). Rathore *et al.* (2011)

reported 16.1% patients were diabetic. The present study also shows diabetes as a significant risk factor.

Evidence of heart disease was found in 63(19.63%) stroke patients in the present study. Among the heart disease patients 39(61.90%) were male and 24(38.10%) were female. Among the male patients 39(20.21%) had heart disease and among the female patients 24(18.75%) had heart disease. It is a known fact that cardio-embolism causes 15% of all strokes (Easton *et al.*, 2017). Zafar and Khan (2008) found ischaemic heart disease in 26% patients in haemorrhagic stroke. Anderson *et al.* (1994) reported ischaemic heart disease was 7% in PICH. Kumar *et al.* (2016) noticed 15% patients had the history of coronary heart disease and 7(5.83%) patients had atrial fibrillation in ischaemic stroke. In a study done by Hayee *et al.* (1999), it was found that 29.66% of the patients were suffering from different heart diseases. Hossain *et al.* (2011) found 24% heart disease patients and among the heart disease patients, 16.66% had myocardial infarction, 45.83% had ischaemic heart

fibrillation. A study in Britain, Macfarlane *et al.* (1991) found that men with definite evidence of previous my 101 nfarction had four fold higher risk of stroke compared to men with no pre exısting IHD. Budlie (1991) revealed that 24% of the stroke patient had evidence of recurrent myocardial infarction. Anderson (1991) found that non-valvular atrial fibrillation is responsible for about half of the cases of cardio embolic stroke. Rathore *et al.* (2011) reported 44.9% patients had coronary artery disease and atrial fibrillation in 15 cases (12%).

We found smoking appears as an important risk factor for stroke and it was 113(35.20%). It was 111(98.23%) in male patients and 2(1.37%) in female patients among the smokers. Among the smokers, in this study 45 patients (39.85%) had hemorrhagic stroke and 68 patients (60.18%) had ischemic

stroke. Kumar *et al.* (2016) reported 41.67% were smoker among the ischaemic patient. Siddique *et al.* (2009) found smoking appears as an important risk factor in both hemorrhagic and ischemic stroke. This study correlates with Donnan *et al.* (1989) who found smoking as a strong risk factor for cerebral infarction. Singh *et al.* (2006) cited cigarette smoking was associated with ICH in 24% of cases. Comparable observations were made by Shinton and Beevers (1989) in 27%, and by Tatu *et al.* (2000) in 18% of ICH cases. Rathore *et al*. (2011) found 57% smoker in stroke patients in their study. Saha *et al.* (2016) found history of smoking was present in 54% cases.

Saha *et al.* (2016) in their study showed that 6% had heart disease (4% valvular heart disease and 2% ischaemic heart disease. They found among the risk factors hypertension was present in 60% cases. Hossain *et al.* (2011) found hypertension was in 63% cases and Siddique *et al.* (2013) found in 69% cases.

Saha *et al.* (2016) found diabetes mellitus was present in 17% cases. The

(20%) were suffering from diabetes mellitus (Boysen 1988). Hossain *et al.* (2011) found DM in 21% and Sid *ıl.* (2013) found in 11%.

Frequency of different features in ischaemic and haemorrhagic stroke were recorded.In the present study, the predominant clinical feature with which the stroke patients presented was hemiplegia and hemiparesis 295(91.90%). It was 194(92.82%) in ischaemic stroke and 101(90.18%) in haemorrhagic stroke. Quaraishi (1987) in his study on clinical presentation of acute cerebrovascular disease in 50 stroke patients also found that 94% of the patients had the same feature. Zafar and Khan (2008) found hemiparesis or hemiplegia (78%) was the commonest presenting feature followed by speech dysfunction (60%). Siddique *et al.* (2009) in his study on clinical presentation

and epidemiology of stroke - a study of 100 cases found commonest neurological deficit was hemiparesis in both hemorrhagic (85%) and ischemic (80%) stroke patients respectively. Saha *et al.* (2016) reported the commonest neurological deficit was hemiplegia (61%) and impaired consciousness was found in 66% cases.

Bhowmik *et al.* (2014) reported among the study subjects, majority (89%) of the subjects presented with hemiplegia after an attack of acute stroke. Other common clinical presentation were aphasia (71%), headache (39%), convulsion (23%), vomiting (18%) and cranial nerve palsy (17%).

Impaired consciousness was found in 195(60.75%) patients. From the present study it is evident that 90 patients presented with impaired consciousness which was conspicuously high (80.36%) among patients with haemorrhagic stroke. It was 105(50.24%) in ischaemic stroke. It has been observed that unconsciousness is a predominant feature of ICH (Easton *et al.* 2017). Siddique *et al.* (2009) reported in case of ischemic stroke majority (52.5%)

hemorrhagic stroke most common (50%) presentation was on grade 3 level of unconsciousness.

Other clinical findings were headache, vomiting, cranial nerve palsy, speech disturbance, vertigo, hypertension and neck rigidity.

About 187(58.26%) had headache. Among the infarct patient 103(49.28%) had headache and among haemorrhagic stroke 84(75.00%) had headache. About 136(42.37%) had cranial nerve palsy. It was found about 119(56.94%) in infarct & 17(15.18%) in haemorrhagic stroke. Speech disturbance was 154 (47.98%). It was 99(47.37%) in infarction & 55(49.11%) in haemorrhagic cases. Vertigo was 59(28.23%) in infarct & 56(50.00%) in haemorrhagic patient. Hypertension was in 225(70.09%), 133(63.64%) in ischaemic stroke & 92(82.14%) in haemorrhagic stroke. Neck rigidity was 30(14.35%) in

infarct and 70(62.5%) in haemorrhagic stroke. Vomiting was 19(9.09%) in infarct, 41(36.61%) in haemorrhage.

It was observed that cranial nerve palsy was found more frequently in infarcted cases. Headache and neck rigidity were significantly correlated with haemorrhagic stroke. Significance of headache and neck rigidity is in keeping with literature (Clark, 1990) and with study findings of Quaraishi (1987).

Siddique *et al.* (2009) found among the associated features of stroke, headache and vomiting (headache in 60% cases and vomiting in 75% eases) are more marked in cases of hemorrhagic stroke. Headache in most cases preceded the onset of stroke and sometimes followed the incident. It is apparent from this study that headache and vomiting have got greater association with hemorrhagic stroke. This study also correlates well with that of Scott and Miller (2000), showing marked association of headache and vomiting with hemorrhagic stroke.

Headache is more frequently associated with haemorrhagic stroke (34-60 per

commonly associated with transient ischaemic attacks (TIAs) than ischaemic stroke, with incidence rates of 2 104 :ent reported by Edmeads (1979). Fisher and Pearlman (1967) reported ischaemic stroke in the basilar distribution area, especially the posterior cerebral circulation, is more often associated with headache than stroke in the carotid distribution area. Portney *et al.* (1984) reported headache is more frequently associated with:

- Haemorrhagic stroke than infarcts
- Posterior than anterior circulation infarcts
- Thrombotic than embolic ischaemic events
- Cortical than deep white matter infarcts
- Venous than arterial infarction.

Tentschert *et al.* (2005) reported in study of 2196 patients with ischaemic stroke, found that:

• Patients less than 40 years old had a four-fold increased risk of headache at stroke onset compared with patients aged 80 years and older. The probability of developing headache decreased steadily with increasing age.
• Women were more likely to develop headache at stroke onset than men.
• Previous history of migraine especially with aura was strongly associated with headache at stroke onset, as shown in various other studies.
• For reasons unknown, patients with right hemispheric ischaemic stroke had a higher prevalence of headache than left hemispheric lesions.

Frequency of speech disturbance in ischaemic and haemorrhagic stroke were recorded. It was 47.37% and 49.11% in ischaemic and haemorrhagic stroke respectively. Siddique *et al.* (2009) reported motor dysphasia in 12 (66.25%) cases of hemorrhagic and 47 (58.75%) cases of ischemic stroke.

The rational management of a patient presenting with an acute stroke should be based on knowledge of its pathological type. The diagnosis of the type of stroke by analysis of the patient's clinical features has often been shown to be

features of ischaemic and haemorrhagic stroke. Neurological examinations are subject to observer variation [105] ıical history taking varies with the memory and powers of communication of the patient and his family. There are also diverse opinions regarding the usefulness of diagnostic criteria proposed by relevant authorities in various parts of the word for differential diagnosis of stroke.

In the present study, the type of stroke was diagnosed by CT findings. It was found that 209(65.11%) cases were cerebral infraction and 112(34.89%) cases were haemorrhagic stroke. Out of 112 cases of haemorrhagic stroke, (99)88.39% were ICH and (13)11.61% were due to subarachnoid haemorrhages. Including every patient admitted with acute stroke, without prejudice of age and clinical condition, there were 31% of 88 strokes in

Chinese population was the result of PICH (Louis Kreel, 1991). Their findings correlate with our study. This agrees closely with the figure of 30.6% reported by Huang et al (1990) in Hong Kong Chinese population. Hossain *et al.* (2011) in study found that 61% had ischaemic stroke while 39% had haemorrhagic stroke.

But our findings differ from that of Quareshi (1987) who found 84% ischaemic stroke and 16% haemorrhagic stroke and 12-14% were haemorrhagic stroke in Caucasians (Fieschi *et al.* 1988, Rowe *et al.* 1988). Our study also differs from the study of Hayee *et al.* (1999) which was done in Dhaka where the incidence of ischaemic stroke was (83.89%).

Higher rate of haemorrhagic stroke has also been reported in number of hospitals in Asian countries such as Singapore (33%), Malaysia (33%), Thailand (30%), Korea (31%) and Taiwan (31%) (Poungvarin 1998, Wong 1998). One of the causes of high incidence of haemorrhagic stroke in this hospital based present study is more related to haemorrhagic stroke.

These findings are more or less similar to the studies done at home and abroad. Bashar *et al.* (1992) found 73% cases of cerebral infracts and 27% haemorrhagic lesions. In their stu [106] ercock *et al.* (1985) found cerebral infraction in 74.5%, intracerebral haemorrhage in 14.1% and non-stroke lesion in 1.5% cases and pathological type of stroke could not be detected in 9.8% cases. Sotaniemi *et al.* (1990) found CT detected ischemic lesions in 66.2%, haemorrhagic in 33.8% and non-stroke abnormality in 3.2% cases. Cerebellar stroke is relatively uncommon as a cause of first stroke (3.1% of all cases) (Sandercock *et al.* 1985).

Site of the infraction may give clues to the pathogenesis. For example, infraction in the watershed zones suggests a haemodynamic origin but multiple cortical infarcts in different territories suggest a cardiac source of emboli (Brown 1992).

In our study the commonest site of infarction was basal ganglia and paraventricular region 62(29.67%) followed by parietal region 55(26.32%) followed by internal capsule 23(11%). The next common location was frontoparietal region 19(9.09%), frontal lobe 17(8.13%), cerebellum 12(5.74%), thalamus 8(3.83%), occipital lobe 5(2.39) and brainstem 4(1.91%).

Bhowmik *et al.* (2014) reported ischaemic stroke as in location parietal lobe of brain (57%) was mostly affected. Basal ganglia (45%), internal capsule (56%), brain stem (6%), thalamus (6%) and cerebellum (8%) were the other common sites of involvement. Ischemic infarcts were also found in paraventricular location (18%), in frontal (6%) and temporal (7%) lobes.

In our study majority of the ischemic stroke occurred in the territory of middle cerebral artery (MCA) and was 159(76.08%). Involvements of anterior and posterior cerebral artery were found in 21(10.05%) & 12(5.74%)

al. (2016) found among infarction majority had infarct in middle cerebral artery territory (68.9%). Kundu (2001) found arterial territorial involvement in ischaemic cases 72.5% in the MCA territory followed far behind by ACA (14.5%) territory and PCA (8.7%) territory. Bashar *et al.* (1992) found that MCA was responsible for 79% of the arterial territorial involvement, 12% were ACA territory and 9% were PCA territory. Middle cerebral artery infract is the most frequent and is almost always caused by ischaemia (Grossman 1996).

Bhowmik *et al.* (2014) reported majority of the subjects (76%) middle cerebral arterial territory was affected. Involvements of anterior and posterior cerebral arteries were found in 7% and 5% subjects respectively. In 6% cases, involvement of vertibro-basilar arterial system was observed. Four percent

subjects had involvement of both middle and posterior arteries. Both anterior and posterior arterial territory infarcts were found in 2% cases.

Mahalakshmi *et al.* (2015) reported middle cerebral artery was most commonly involved in 76.5% of patient's posterior cerebral artery was involved in 10.3% cases and anterior cerebral artery in 7.35% cases. Multiple infarcts were present in 5.9% patients.

Kertesz *et al.* (1985) observed that about 88% subjects had single MCA territory involvement and 7% subjects had both middle and posterior cerebral arteries involvement. Stolz *et al.* (2008) reported similar result where it was seen that about 82% of all stroke patients had involvement of MCA territory. Involvements of anterior and posterior cerebral arteries were found in 7% and 5% subjects respectively. Kang *et al.* (2008) found that ischemic stroke affected mostly ACA territory in their study population (consisting diabetic and non- diabetic). In a study conducted by Biller *et al.* (1988) revealed that

In this study of 112 patients of spontaneous ICH, 99 patients had intraparenchymal haemorrhage (88.39%) and 13 patients had SAH (11.61%). In this study out of patients with 108 enchymal haemorrhage 84 patients (84.85%) were hypertensive and 15(15.15%) were from non hypertension. Out of 13 patients with subarachnoid haemorrhage 8(61.54%) were hypertensive and 5(38.46%) were non hypertensive.

The capsulo-basal ganglia region (61.61.2%) was the commonest site of haemorrhage in the present study followed by lobar (18.75%), thalamus (9.82%) and cerebellar (6.25%) regions. These findings are almost consistent with that of Bashar *et al.* (1992), where basal ganglion and internal capsule region were the site of involvement of 52% of the haemorrhagic lesions, 37% in other cortical areas, 6% in the brain stem and 5% in the cerebellum and

Kundo *et al. (2001)* where the basal ganglia paraventricular region (65.2%) was the commonest site of haemorrhage in his study.

About 90% intracerebral haemorrhages are supratentorial and the basal ganglion and thalamus are the most common sites. Basal ganglionic may rupture into adjacent ventricles (Grossman 1996, Easton *et al.*2017). Basal ganglia (55%) was the commonest site of bleed followed by thalamus (26%), cerebral hemispheres (11%), brain stem (8%) and cerebellum (7%) (Zafar *et al.* 2008). Singh *et al.* (2006) noted the sites of lesion in intracerebral haemorrhage determined by CT scan in order of frequency in their study were (i) putamen/lentiform nucleus of basal ganglia (65%), (ii) lobar (17%), (iii) thalamus (13%), (iv) pons (3%) and (v) cerebellum (2%).

Feldmann (1991) reported the sites of involvement by ICH in order of putamen (35%), lobar (30%), cerebellum (15%), thalamus (10%) and pons (5%). Tatu *et al* (2000) found ICH to be the most prevalent in lobar (36.5%), followed by lentiform area (32%), thalamic (15.7%), cerebellar (8.8%), midbrain and pons (2%), intraventricular haemorrhage (9.2%), caudate (1%)

Scott *et al*. (1985) in their study found that putaminal bleeding (35%) was the commonest followed by lobar (

alamus (10%), cerebellum (15%), pons (5%) and caudate (5%).

The finding in the present study is comparable with Scott *et al.* (1985) except for cerebellum which is the least common site in the present study. These differences in frequency of ICH locations could be due to differences in geographical and genetic factors and multiple different risk factors.

In this study, supratentorial location of the bleed 88(88.8%) was more common than infratentorial location 11(11.11%). It was also shown that non lobar hemorrhage 82(82.83%) was more common than lobar hemorrhage 17(17.17%). In our study, out of 99 patients with intraparenchymal

hemorrhage, 61 patients had capsuloganglionic bleed (61.62%) followed by 17 cases of lobar bleed, (17.17%), 10 cases of thalamic bleed (10.10%), 07 patients had cerebellar bleed (7.07%) and 4 had brainstem bleed (4.04%). According to Justine Elliott *et al.* (2010), fifty percent of hypertension-related hemorrhage occurs in deep structures (basal ganglia and thalamus) and 30% in superficial (lobar) areas. According to Zafar *et al.* (2008) basal ganglia (55%) was the commonest site of bleed followed by thalamus. In another study by Kase *et al.* (1982) showed that the location of hypertensive intracerebral hemorrhage was putaminal 33%, lobar 23%, thalamic 20%, cerebellar 8%, pontine 7% and miscellaneous 9%, which is matching with our study. According to Yousuf *et al.* (2012) the commonest location of ICH was lobar (43.8%) followed by basal ganglia/internal capsule (28.1%) and multilobar (13.1%). According to Swamy (2007) majority (45%) of the clots were lobar followed by basal ganglia bleed (35%).

In a study done by Mini *et al.* (2017) found in their study out of 100 patients of spontaneous ICH, 76 patients had intraparenchymal hemorrhage (76%)

hemorrhage 60 patients (79%) were hypertensive and 16(21%) were non hypertension. Out of 24 patient 110 ıbarachnoid hemorrhage 18(75%) were hypertensive and 6 (25%) were non hypertensive and supratentorial location of the bleed (82%) was more common than infratentorial location (18%). It was also shown that non lobar hemorrhage (76.3%) was more common than lobar hemorrhage (23.7%). 7 cases of thalamic bleed (9.2%), 5 patients had thalamo ganglia bleed (6.5%), 10 patients had cerebellar bleed (13%) and 4 had brainstem bleed (5%).

This study correlated with many of the above mentioned studies which showed that commonest location of intracerebral hemorrhage was basal ganglia.

It was evident from our study that 209(65.11%) were infarcts. Among them CT detected infarct was <2 cm in 78(37.32%) patients and mortality was 1(1.28%) within 7 & 28 days in both period. Infarct size between 2- 5 cm was 106(50.72%) and death was 23(21.70%) & 13(12.26%) within 7 & 28 days respectively and total death was 36(33.96%) within 28 days. Infarct size >5 cm was 25(11.96%) and death was 18(72.00%) & 5(20.00%) within 7 & 28 days respectively and total death was 92.00% within 28 days and it was statistically significant ($p<0.05$). This indicates that with the increasing size of infarct, mortality will increase and it has an impact on the prevalent death.

The result is almost consistent with Bryan *et al.* (1991) where the authors found 42.85% infarcts of <2cms, 38.1% 2-5 cm and rest 19.0% was large infarcts of ≥5cm in 21 CT positive infarcts within 24 hours.
Gustavo Saposnik *et al.* (2008) reported seven-day case fatality was 6.9% (249/3631), 30-day case fatality was 12.6% (457/3631).

CT detected pineal gland without displacement or displacement upto 3 mm was 167(79.90%) and death was 14(8.38%) & 10(5.99%) within 7 & 28 days

displacement >3 mm was found in 42(20.10%) and death was 28(66.67%) & 9(21.43%) within 7 & 28 days re 111 y and total death was 37(88.09%) within 28 days and it was statistically significant ($p<0.037$). The highest number of death was from pineal gland displacement > 3 mm and within 7 days.

CT detected septum pellucidum without displacement or displacement upto 5 mm was 173(82.76%) and death was 12(6.94%) & 14(8.09%) within 7 & 28 days respectively and total death was 26(15.03%) within 28 days. Septum pellucidum displacement > 5 mm was found in 36(17.22%) and death was 30(83.33%) & 5(13.89%) within 7 & 28 days respectively and total death was

35(97.22%) within 28 days and it was statistically significant (p=0.014). The highest mortality was from septum pellucidum displacement > 5 mm and within 7 days.

More than half 61(61.62%) of ICH produced mass effect in comparison to only 64(30.62%) of infarcted cases. It is common findings in case of haemorrhagic lesions which was also evident in a study conducted by Scott *et al.* (1974) where all 23 intracerebral haemorrhages were all 23 intracerebral on CT by compression and displacement of ventricular structures and pineal and choroid glomus dislocation.

In this study among the parenchymal haemorrhagic stroke out of 99 patients, 29(29.29%) patients died within 7 days, 15(15.15%) patient died within next 21 days and the total dead patient is 44(44.44%). So mortality rate was 44.44% within 28 days in this study. In the present study of haemorrhagic stroke over all 28 days mortality rate was found to be 44.44% and 29.29% within first 7 days. Douglas and Haerer (1982) reported a mortality rate of 40% and Dixon *et al* in 1985 reported 33% mortality. According to Yousuf *et*

mortality rate during the first month following ICH as 31% .The mortality rates in the above mentioned stud[illegible] mpatible with our study.

The over all mortality rate of 52% [illegible]ys was reported by Bamford *et al.* (1990) with 56% of the death occurring in the first 3 days of onset. In other studies, 30 days ICH mortality rate was found to be 30% by Fieschi (1988) and 35% by Anderson *et al.* (1994). Tatu *et al.* (2000) reported over all mortality of 24.2% at 30 days and death in the first 3 days constituted 48% of all deaths.

Singh *et al.* (2006) stated in their study over all 30 days mortality rate was found to be 43% with first 3 days and mortality of 58% of total death which could be comparable to above studies. Similar 30 days mortality rate was

found in the study by Frank (1992). However, Silver *et al.* (1984) reported 80% mortality within 72 hours in their study. These differences in the mortality may be due to variations in population, risk factors and facilities availability.

We have classified the patients with intraparenchymal hemorrhage according to location i.e. whether infratentorial or supratentorial. Out of 11 patients with infratentorial bleed 4(36.36%) died within 28 days. There was no significant relation between location of bleed and the outcome. No definite association noted with location i.e. infratentorial location of bleed. But out of 11 infratentorial bleeds 4 were brainstem bleed. Death rate was 75% in brainstem bleed in this study irrespective of volume or other parameters. This showed that instead of infratentorial location, brainstem probably might be an important predictor for 30 days mortality.

Anderson (1994) reported 28 days case fatality rate among the ICH locations as 100% in brain stem, 30% in cerebellum, 22% in basal ganglia and thalamus, and 21% in lobar haemorrhage. Similar patterns of case fatality were also observed in the present study other than cerebellar ICH.

We have classified the patients with intraparenchymal hemorrhage according to the volume of haemorrhage. [113] udy 45(45.45%) patients had the volume of hematoma upto 40 ml and death was 11.11% within 28 days, 41-60 ml was in 23(23.23) and death was 47.83%, 61-80 ml was in 19(19.19) and death was 89.47% and above 80 ml was in 12(12.12) and death was 91.67% within 28 days. P value was 0.011 (significant). That means the majority of patients with larger bleed died early.

Joseph *et al.* (1993) reported that, the 30-day mortality for the 188 cases of intracerebral hemorrhage was 44%, with half of deaths occurring within the first 2 days of onset. Volume of intracerebral hemorrhage was the strongest

predictor of 30 days mortality for all locations of intracerebral hemorrhage. Using three categories of parenchymal haemorrhage volume (0 to 29 cm^3, 30 to 60 cm^3, and 61 cm^3 or more), calculated by a quick and easy-to-use ellipsoid method, and two categories of the Glasgow Coma Scale (9 or more and 8 or less), 30 days mortality was predicted correctly with a sensitivity of 96% and a specificity of 98%. Patients with a parenchymal haemorrhage volume of 60 cm^3 or more on their initial computed tomogram and a Glasgow Coma Scale score of 8 or less had a predicted 30 days mortality of 91%. Patients with a volume of less than 30 cm^3 and a Glasgow Coma Scale score of 9 or more had a predicted 30days mortality of 19%. Only one of the 71 patients with a volume of parenchymal haemorrhage of 30 cm^3 or more could function independently at 30 days.

Size of the hemorrhage is definitely related to prognosis, in agreement with Drury *et al* (1984) and Kanaya *et al. (*1980). Cesare Fieschi *et al.* (1988) found that age was related to prognosis; however, no relation between age and prognosis was found by Steiner *et al.* (1985) whereas Douglas and Haerer (1982) found higher mortality in patients <50 years old than in

Singh *et al.* (2006) found the mean volume of ICH in their study was 46.6 ml for all patients and among the de[114]n volume was 65.6 ml. They also found mortality was as high as 90.9% when the volume of ICH was more than 80 ml.

Tatu *et al.* (2000) found the mean volume of 34.1 ml for all the patients and 76.2 ml among the worst outcome comprising death in 92%. These differences in the mean volume of haematoma could be due to various associated risk factors among different population and the nature of patient recruitment.

Lampel *et al.* (1995), Massaro (1992) and Garde (1983) quoted that critical lethal outcome was associated with 50 ml or 80 ml in lobar haemorrhage. Kase (1982) found lobar ICH with volume larger than 50 ml who were comatose on admission have mortality close to 100%. Singh *et al.* (2006) found in their study statistical significant findings of 85.2% and 90.9% mortality among the ICH volume greater than 60 ml and 80 ml respectively. Similar pattern of higher mortality among the patients having larger haematoma volume was also noted in the present study with statistical significant findings of 89.47% and 91.67% mortality among the ICH volume greater than 60 ml and 80 ml respectively. Mukherjee and Hazra (1998) observed 67.3% mortality among ICH volume greater than 40 ml.

According to Yousuf *et al.* (2012) the overall mortality rate was 32.5%. The significant independent predictors of acute in hospital mortality were Glasgow Coma Scale (GCS) on admission, posterior fossa bleed, hematoma volume > 60ml, midline shift and intraventricular extension of hemorrhage. According to Helweg-Larsen *et al.* (1984) the acute mortality of ICH was 27%. Determinant for the immediate prognosis was the level of consciousness and the volume of the hematoma (crucial size was 50 ml). Intraventricular hemorrhage was a bad prognostic sign only in the ganglionic-

Michel Lelo Tshikwela and Ben ngo-Mbenza (2012) reported that ICH volume is a significant and independent predictor of mortality. ICH volume ≥ 25 ml was the most important and independent predictor of mortality as reported by their studies. This may be explained by the elevated intracranial pressure and cerebral edema associated with the hemorrhagic volume. Similar findings were found by Obajimi *et al.* (2002), Longstreth (2001) and Roquer et *al.* (2005).

ICH volume ≥30 cm^3 together with Glasgow Coma Scale score, presence of intraventricular blood, and age ≥80 years have been included as independent variables for 30 days mortality in the ICH score developed by Hemphill *et al*. (2001). Tatu *et al*. (2000) found that outcome was closely associated with initial haematoma volume. In their report, Rankin 1 – 3 was associated with a mean volume of 13.1 ml, Rankin 4 - 5 with 32.9 ml and death with 78.8 ml in 95% of cases. Singh *et al*. (2006) in a study showed Rankin score 1-3 with initial mean ICH volume of 21.3 ml, Rankin 4 and 5 with 45.4 ml and death with 80.0 ml in 90.9%.

Mass effect in the form of midline shift noted and was evident by pineal gland and septum pellucidum displacement. CT detected pineal gland displacement upto 3 mm or without displacement was 60 patients (60.61%) and death was 11.67 % & 10.00% within 7 & 28 days respectively and total death was 21.67% within 28 days in haemorrhagic stroke. Pineal gland displacement > 3 mm was found in 39 patients (39.39%) and death was 22 (56.41 %) & 9 (23.08%) within 7 & 28 days respectively and total death was 31 (79.49%) within 28 days and it was statistically significant (p value 0.028). The highest number of death was from pineal gland displacement >3 mm & within 7 days. That means midline shift as evident by pineal gland

Septum pellucidum displacement upto 5 mm or without displacement was 70 (70.71%) patients and death was 15(21.43 %) & 8(11.43%) within 7 & 28 days respectively and total death 116 (32.86%) within 28 days. Septum pellucidum displacement >5 mm was found in 29(29.29%) and deaths were 14(48.28 %) & 7(24.14%) within 7 & 28 days respectively and total deaths were 21(72.41%) within 28 days and it was statistically significant (p value 0.033).

Wiggins *et al.* (1984) reported that ICH with hypertension in 62% of cases and midline shift or pineal gland displacement >3mm showed mortality rate of 40%. Singh *et al.* (2006) in their study, ICH with hypertension in 80% cases and pineal gland displacement >3 mm shows (70%) mortality rate.

In this study of 99 patients of intraparenchymal hemorrhagic stroke, 35 patients (35.35%) had intraventricular extension. Out of this 35 patients with intraventricular extension, 22 (62.86%) patients died within 7 days and 9(25.71%) died within next 21 days. So the total patients died due to intraventricular extension of haemorrhage were 31(88.57%). The p-value was 0.017(significant). That means there was significant association of intraventricular extension of IPH with high mortality.

Mini *et al.* (2017) reported out of 76 patients of intraparenchymal hemorrhage, 27 patients (36%) had intraventricular extension. Out of this 27 patients with intraventricular extension, 20(74%) patients died and 7(26%) were alive. The 7 patients who were alive are in category 3 (severely disabled) of Glasgow Outcome Scale. The p-value was 0.000(significant). That means there was significant association of intraventricular extension of IPH with high mortality and morbidity. Our study correlates with his study.

Intracerebral haemorrhage with intraventricular extension influenced the

Weisberg (1979) and Fieschi (1988) respectively. Singh *et al.* (2006) in their study of ICH with intraventricula [117] on influenced the mortality rate of 74% than without intraventricular extension of 29% mortality. They also found mortality was significantly higher among patients of ICH with pineal gland displacement of more than 3 mm and intraventricular extension.

Chiranjib Nag *et al.* (2012) stated in a study that IVH has not statistically become significant for a poor outcome but intraventricular extension of bleed

from other anatomical location of hemorrhage is an independent poor prognostic factor (OR = 7.846, 95 % CI of 2.766-22.254, *P*< 0.0001).Ventricular compression by hematoma either alone or in combination with midline shift becomes statistically significant (OR = 2.700, *P* = 0.002 and OR = 2.124, *P* = 0.025, respectively). Hallevi *et al.* (2008) found that patients with IVH were twice as likely to have a poor outcome (OR 2.25, *P* = 0.001) when compared to patients without IVH. In a study on Japanese stroke patients, Hosomi *et al.* (2009) found that IVH along with hemorrhage size and ICH severity on admission is related to high mortality.

According to Daverat *et al.* (1991) the mortality rate during the first month following ICH was 31%. The significant independent predictors of early (30 days) mortality were hemorrhage size, midline shift, and intraventricular spread of the hemorrhage. Broderick *et al.* (1993), found that the mortality rate at one month was best predicted by determining the initial score on the Glasgow Coma Scale (less <9) and the initial volume of the hematoma (>60). According to Anderson *et al.* (1994), the overall 28 days case fatality was 35%, but this varied from 100% for hemorrhages in the brainstem to 22% for those in the basal ganglionic or thalamic region. Other predictors of early death were intraventricular extension of blood, volume of hematoma, mass effect, and coma and severe paresis at onset. According to Michel Lelo

35%. ICH volume >25 ml, presence of coma, left hemispheric site of ICH were identified as significant [118] ependent predictors of 30 days mortality. Midline shift >7 mm, a consequence of ICH volume, was also a significant predictor of mortality.

This study demonstrates the impact of hydrocephalus on outcome from ICH. Hydrocephalus was associated with a considerably higher mortality and fewer patients being discharged to home. We found Hydrocephalus present in

haemorrhagic stroke was 43(43.43%) and death was 21(48.84%) & 11(25.58%) within 7 & 28 days respectively and total death was 32(74.42%) within 28 days. There was no hydrocephalus in 56(56.57%) and death was 8 (14.29%) and 4(7.14%) within 7 & 28 days respectively and total death was 12 (21.43%) within 28 days. P value was 0.018 and it was statistically significant.

Michael Diringer (1998) demonstrated those with hydrocephalus were younger (57 ± 15 versus 67 ± 15 years), had lower GCS scores (8.2 ± 4.2 versus 1162.9), were more likely to have ganglionic or thalamic hemorrhages, and were intubated more frequently (70% versus 27%). Hospital mortality was higher in patients with hydrocephalus (51% versus 2%), and fewer patients went home (21% versus 35%). Those who died had higher hydrocephalus scores (9.67 ± 7.1 versus 5.75 ± 4.5).

The relationship between hydrocephalus and outcome may vary with different hemorrhage locations. In hematomas that occur close to the ventricles, IVH and thus hydrocephalus are common (Chung *et al.* 1996). Small thalamic haemorrhages can easily cause hydrocephalus by compressing the cerebral aqueduct, whereas small ganglionic haemorrhages rarely have any impact on ventricular size.

Thanh *et al.* (2000) reported hydrocephalus was present in 40 of the 100

76% of those who died. Kumral *et al.* (1995) concluded that hydrocephalus was a predictor of mortality in t[119] emorrhage. Radberg *et al.* (1991) analyzed lobar, basal ganglia, and brain stem hemorrhage and found that dilatation of the contra lateral ventricle was associated with a mortality of 67%. We have obtained a similar percentage of patients with hydrocephalus.

Intra ventricular extension of bleed, hematoma size is significant predictors of mortality in all above mentioned studies. In this study also there was good

correlation between these factors and 28 days mortality. Midline shift was mentioned as significant predictor of mortality in some of above studies. This study also showed good correlation between midline shift and 28 days mortality. The significant predictors of 28 days mortality in our study were intraventricular hemorrhage, midline shift, subarachnoid extension of bleed, hydrocephalus, and volume of hematoma 60 ml and above.

Haemorrhagic stroke comprised of 112 (34.89%) patients of which 99 (88.39%) were intracerebral haemorrhage and 13(11.61%) were subarachnoid haemorrhage. Among the patients of this group death was 7 (53.85%) & 2 (15.38%) within 7 & 28 days respectively and total death was 9 (69.23%) within 28 days, p value was 0.011 and it was significant. In this study, sub arachnoid bleeding was found as a good predictor of mortality. According to Buensuceso (2007), the only strong predictor of mortality on CT scan findings is the presence of subarachnoid hemorrhage, having three times higher risk of dying compared to those patients without SAH. No significant correlation was found between infratentorial location of bleed and mortality in this study, but mortality was 75% in brainstem haemorrhage. According to Anderson *et al.* (1994) also there was good correlation between brainstem haemorrhage and 30 days mortality.

It is estimated that death from SAH occurs before hospital admission in 12%

In population-based studies of Latin American and the Caribbean 2-5% of all strokes was subarachnoid haemo[120] avados *et al.* 2007). In a study of 2418 consecutive patients from 19 hospitals in the city of Fortaleza, Brazil, the frequency of admissions of subarachnoid haemorrhage patients was 6.0% (Carvalho *et al.* 2011).

The case fatality of SAH seems to remain high worldwide. Mortality rates vary widely across published epidemiological studies, ranging from 8% to 67% with a median mortality rate in the United States of 32% versus 43% in Europe and 27% in Japan (Nieuwkamp *et al.* 2011).

CHAPTER -7

CONCLUSION

7.1. CONCLUSION

Stroke is one of the foremos of morbidity, mortality and a socioeconomic challenge. This is particularly true for developing countries like Bangladesh, where health support system including the rehabilitation

system is not within the reach of ordinary people. It is crystal clear that, this devastating condition not only affects the patient but also their family. The objective of this hospital-based study was to identify the important risk factors for stroke prevalent in our society both among the urban and rural population and mortality of the patient from both ischaemic and haemorrhagic stroke. This study may not have reflected the exact situation but gives an approximate picture of the disease. There are many risk factors for stroke, some are modifiable and some are not. In this study a number of modifiable risk factors were identified, of which hypertension remains the most important factor. Next were smoking, diabetes mellitus and ischaemic heart disease. Stroke is more preventable than curable. In an under developing country like ours the best policy for combating stroke is primary prevention. This study reveals that the major risk factor hypertension needs maximum attention for the prevention of stroke. By controlling hypertension we can significantly reduce the incidence of stroke. For this we need increased awareness among people regarding hypertension and its complications.

Again, the present cross sectional observational study of 321 cases of stroke of hospital based patients may not reflect the exact situation of the disease in the community. But its nearness to be reality cannot be underestimated. This is reflected in the statistical data of the present study, which correlates well with the other studies carried out at home and abroad. The results of the study demonstrate that there is good correlation between CT findings and mortality.

Nevertheless the present study showed that death and alive on the 7th & 28th day were well correlated with the initial ICH volume, site, midline displacement, intraventricular e hydrocephalus which could be regarded as a good indicator for mortality. Like this infarct diameter, mass effect, location is good indicator of 7th & 28th day mortality. Such results

should provide a basis for statistical studies on the prognostic factors of intracerebral haemorrhage / infarct for future studies.

7.2. Study Limitation

Certain limitations relevant to the interpretation of this study were noteworthy. The limitation of the present study were-

1. The patient series was totally hospital based and therefore does not represent a random epidemiological sample.
2. CT findings may be negative in early ischaemic stroke, so many of the ischaemic strokes were not included in this study.
3. Because of the small number of the sample, there was more or less a likelihood of an error to actual occurrence of stroke and their correlation to CT findings with mortality.

7.3. Future Research

Science is dynamic and there is always a scope of improvement and change in time to come ahead. With progressive aim to move ahead, we aspire to achieve highly accurate and reliable results. Thus, every study leaves back scopes for other researcher to do something more advanced and varied in order to touch the height of perfection. This study examined only 321 subjects (193 male and 128 female), future researchers can expand the study by including more number of subjects so as to make generalization of the results and practice, further studies with a larger sample size and in multiple centers and a total community area are required. Thus, it could be applied to real life situation.

CHAPTER - 8

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8.1. REFERENCES

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CHAPTER -9

APPENDICES

APPENDIX - I

Characteristics of CT findings in pat ute stroke and their relationship to mortality

QUESTIONNAIRE:

SI No.: **Date:**

A. Sociodemographic characteristics of Patient:

1. **Name:** . . . . . .
2. **Age** (in completed years):
3. **Sex:** Male = 1, Female = 2. ☐
4. **Address:** **Mobile No:**
5. **Marital status:** Married = 1, Bachelor = 2. ☐
6. **Occupation :** 1. Service 2. Business 3. Farmer ☐
   4. Laborer 5. Housewife 6. Others
7. **Economic condition:**

   1. Poor (Upto < 60000 tk/year)
   2. Middle class (60000 - 180000 tk/year)
   3. Rich (>180000 tk/year)

   ☐
8. **Alcohol consumtion:** No = 1 Yes = 2. ☐
9. **Dietary habit:** Vegetarian = 1, Non-vegetarian = 2. ☐

B. Risk factor:

1. **Hypertension:** No = 1, Yes = 2. ☐
2. **Smoking:** No = 1, Yes = 2. ☐
3. **Diabetes mellitus:** No = 1, Yes = 2. ☐
4. **Heart disease:** No = 1, Yes = 2. ☐
5. **Family history of stroke:** No = 1, Yes = 2. ☐
6. **Oral pill (Female):** No = 1, Yes = 2. ☐

C. Clinical presentation:

1. **Hemiplegia/ Hemiparesis:** No = 1, Yes = 2. ☐
2. **Impaired consciousness:** No = 1, Yes = 2. ☐
3. **Headache:** No = 1, Yes = 2. ☐
4. **Vomiting:** No = 1, Yes = 2. ☐
5. **Cranial nerve palsy:** No = 1, Yes = 2. ☐

7. **Vertigo:** No = 1, Yes = 2. ☐
8. **Hypertension:** N Yes = 2. ☐
9. **Neck rigity:** No = 1, Yes = 2. ☐

D. Investigation:

1. **CT Scan** ☐

E. CT Findings: Parenchymal haemorrhage

1. **Volume - ml**
2. **Site -**

a.	**Capsuloganglionic**	No = 1,	Yes = 2.	☐
b.	**Lobar**	No = 1,	Yes = 2.	☐
c.	**Thalamus**	No = 1,	Yes = 2.	☐
d.	**Cerebellar**	No = 1,	Yes = 2.	☐
e.	**Brain stem**	No = 1,	Yes = 2.	☐
f.	**Intraventricular extension**	No = 1,	Yes = 2.	☐
g.	**Hydrocephalus**	No = 1,	Yes = 2.	☐

F. Subarachnoid haemorrhage: No = 1, Yes = 2. ☐

G. Mass effect:

a.	**Midline (Septum pellucidum / Pineal displacement)**	No = 1,	Yes = 2.	☐
b.	**Effacement of ventricles / Sulci**	No = 1,	Yes = 2.	☐

H. Infracts:

1. **Size – cm**
2. **Site**

a.	**Basal ganglia and para ventricular**	No = 1,	Yes = 2.	☐
b.	**Frontal lobe**	No = 1,	Yes = 2.	☐
c.	**Occipital lobe**	No = 1,	Yes = 2.	☐
d.	**Fronto parietal**	No = 1,	Yes = 2.	☐
e.	**Fronto temporal**	No = 1,	Yes = 2.	☐
f.	**Internal capsule**	No = 1,	Yes = 2.	☐

h.	**Brain stem**	No = 1,	Yes = 2.	☐
i.	**Cerebellum**	No = 1,	Yes = 2.	☐

3. Mass effect:

a.	**Midline (Septum pellucidum / Pineal displacement)**	No = 1,	Yes = 2.	☐

b. **Effacement of ventricles / Sulci** No = 1, Yes = 2. ☐

4. Arterial territories:

ACA = 1 MCA = 2 PCA= 3 ☐

F. Out comes:

a. **On 7 days:** 1. Alive 2. Died ☐

b. **On 28 days:** 1. Alive 2. Died ☐

APPENDIX-II

PARENTS' CONS 144 RM (ENGLISH)

(Consent for participation in the research work entitled ***"Characteristics of CT findings in patients of acute stroke and their relationship to mortality"***

I, hereby declare that me / father / mother / guardian of the patient

_____Aged _____ (D/M/Y) _____

Address _____

and I have participated in the above mentioned research work being conducted by **Md. Durrul Huda** (PhD Fellow, Institute of Biological Sciences, University of Rajshahi, Bangladesh). I have been explained clearly about the purpose and benefits of the study. I have been informed that through this research, besides the present treatment there may have the possibility of new advancement in the field of Medical Science particularly CT findings in patients of acute stroke and their relationship to mortality.

Freedom has been given to me for the participation in the study or to discontinue participation at any time without any prior notice. Moreover, I have been assured that the information will be kept confidential.

I also declare that I will not demand any financial support for taking part in this research work. All the above been explained in detail to me clearly in my own language. I have given my consent voluntary for inclusion in the study as a subject.

Signature/Thumb impression Date.....

APPENDIX – III

PHOTOGRAPH

Photo-1: Patient’s position in CT machine.

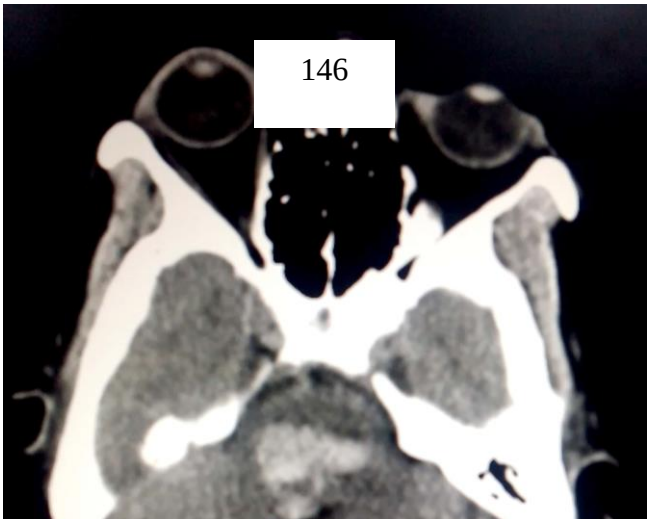

Photo-3: CT scan of the brain showing acute brain stem haemorrhage.

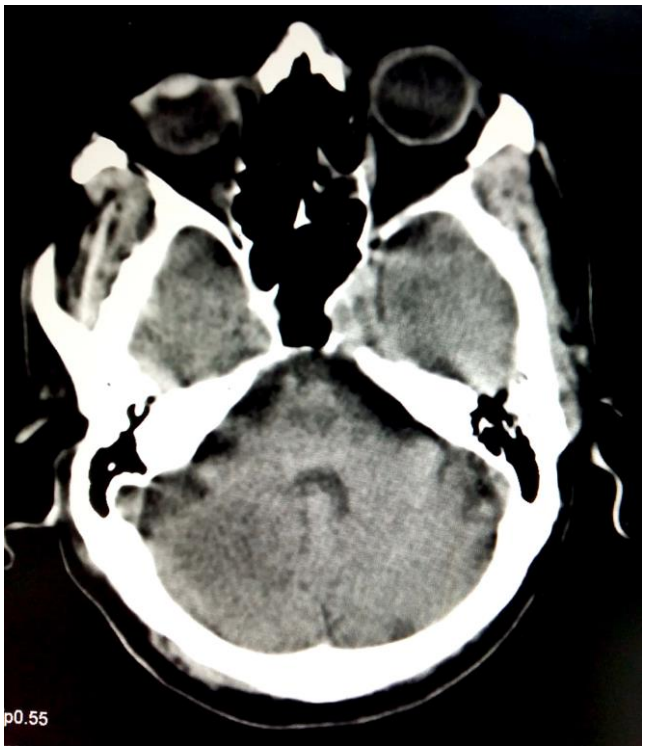

Photo-4 : CT scan of the brain showing cerebellar infarct.

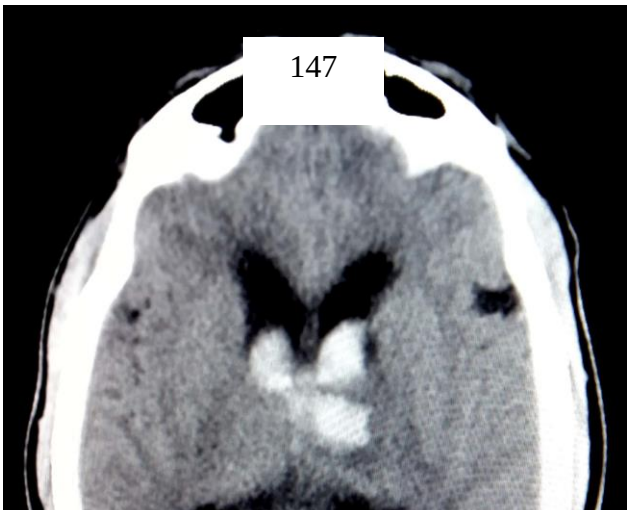

Photo-5 : CT scan of the brain showing acute thalamic haemorrhage with ventricular extension.

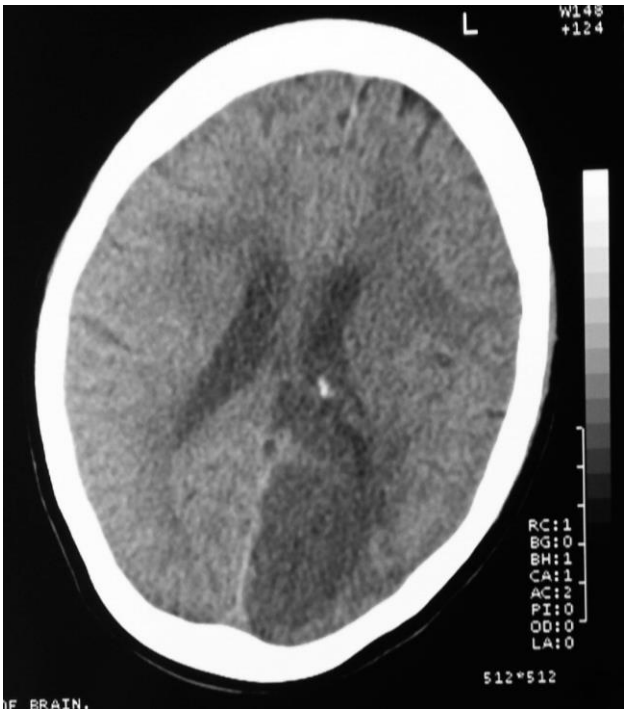

Photo-6 : CT scan of the brain showing PCA infarct.

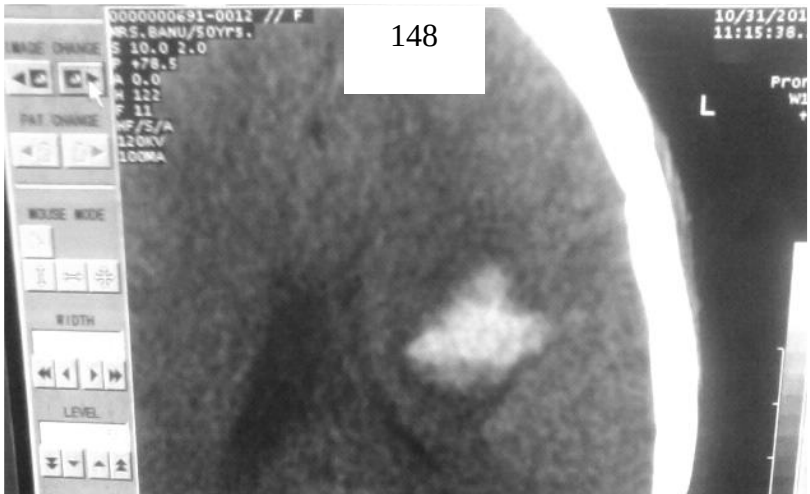

Photo-7 : CT scan of the brain showing parietal haemorrhage.

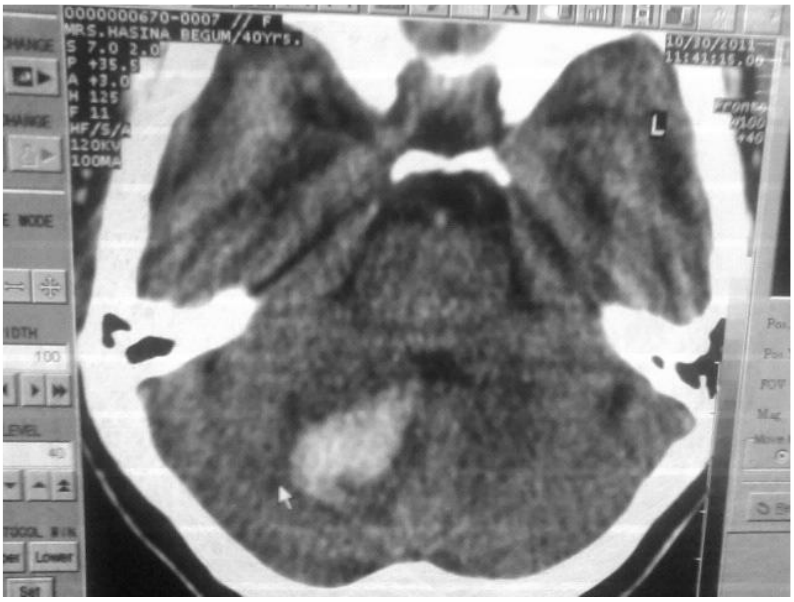

Photo-8 : CT scan of the brain showing cerebellar haemorrhage.

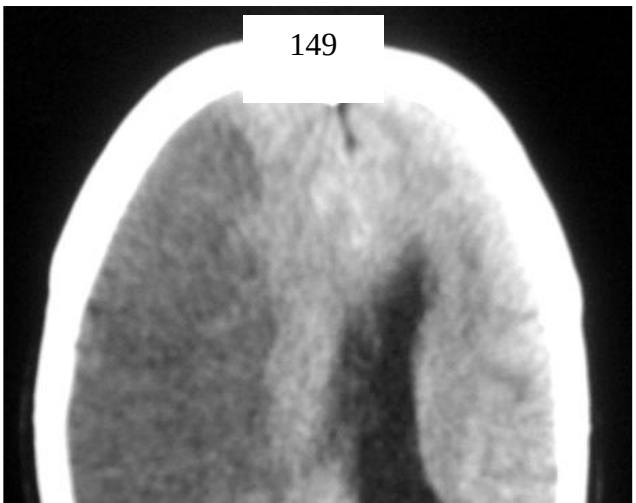

Photo-9 : CT scan of the brain showing fronto-parietal infract.

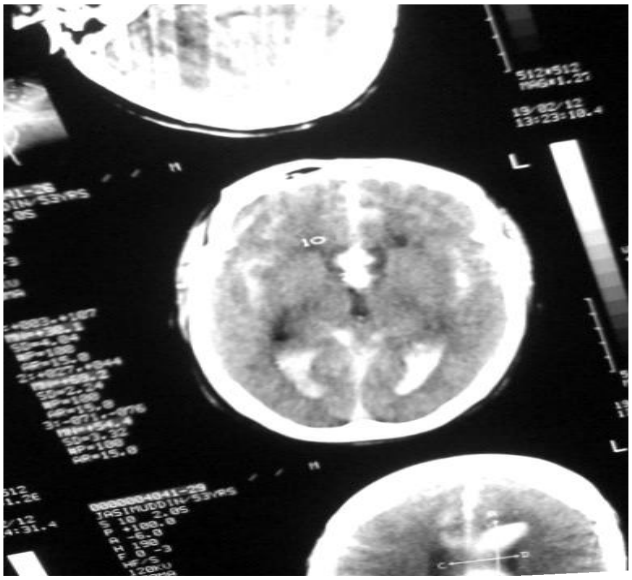

Photo-10 : CT scan of the brain showing acute subarachnoid haemorrhage.