A STUDY OF CLINICAL PROFILE OF HAEMORRHAGIC STROKE IN HYPERTENSIVE PATIENTS

DISSERTATION SUBMITTED

In partial fulfillment of the requirement for the degree of

(Branch-I) M. D. (GENERAL MEDICINE)

of

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CHENNAI- 600032

DEPARTMENT OF GENERAL MEDICINE

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI- 11

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BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"A STUDY OF CLINICAL PROFILE OF HAEMORRHAGIC STROKE IN HYPERTENSIVE PATIENTS"** submitted by **Dr. V.GOPINATH** to the Tamilnadu Dr. M.G.R Medical University, Chennai, in partial fulfillment of the requirement for the award of M.D. Degree Branch – I (General Medicine) is a bonafide research work carried out under direct supervision & guidance.

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CERTIFICATE

This is to certify that the Dissertation **"A STUDY OF CLINICAL PROFILE OF HAEMORRHAGIC STROKE IN HYPERTENSIVE PATIENTS"** presented by **Dr. V.GOPINATH** is an original work done in the Department of General Medicine, Tirunelveli Medical College Hospital, Tirunelveli for the award of Degree of M.D. (Branch I) General Medicine. Under my guidance and supervision during the academic period of 2016 -2019.

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DECLARATION

I solemnly declare that the dissertation titled **"A STUDY OF CLINICAL PROFILE OF HAEMORRHAGIC STROKE IN HYPERTENSIVE PATIENTS"** is done by me at Tirunelveli Medical College Hospital, Tirunelveli Under the guidance and supervision of Prof.Dr.S.Alagesan M.D,D.M (Neuro), the dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University towards the partial fulfilment of requirements for the award of M.D. Degree (Branch I) in General Medicine.

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PROTOCOL TITLE: A STUDY OFCLINICAL PROFILE OF HAEMORRHAGIC STROKE IN HYPERTENSIVE PATIENTS
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DESIGNATION OF PRINCIPAL INVESTIGATOR: PG STUDENT
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Dear, Dr.V.GOPINATH, MBBS., Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during the IEC meeting held on 10.03.2017.

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1. TIREC Application Form ☐
2. Study Protocol ☐
3. Department Research Committee Approval ☐
4. Patient Information Document and Consent Form in English and Vernacular Language ☐
5. Investigator's Brochure ☐
6. Proposed Methods for Patient Accrual Proposed ☐
7. Curriculum Vitae of the Principal Investigator ☐
8. Insurance /Compensation Policy ☐
9. Investigator's Agreement with Sponsor ☐
10. Investigator's Undertaking ☐
11. DCGI/DGFT approval ☐
12. Clinical Trial Agreement (CTA) ☐
13. Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA) ☐
14. Clinical Trials Registry-India (CTRI) Registration ☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of the validity
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CERTIFICATE – II

This is to certify that this dissertation work title **"A STUDY OF CLINICAL PROFILE OF HAEMORRHAGIC STROKE IN HYPERTENSIVE PATIENTS"** of the candidate **Dr.V.GOPINATH** with registration Number **201611354** for the award of **M.D.** Degree in the branch of **GENERAL MEDICINE (I).** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion page and result shows **10 percentage** of plagiarism in the dissertation.

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INTRODUCTION

Cerebrovascular accidents is an important cause of morbidity and mortality all around the world. Stroke due to Intracerebral haemorrhage (ICH) is common second only to the strokes caused due to thrombosis and embolism which constitutes ischemic stroke. The most important risk factor for stroke is hypertension. The incidence of ICH is about 15% of all strokes in the western world and 20 to 30% in the Asian continent. Mortality rate is around 30-50% following a major haemorrhage within a month. The hospital mortality rate was also high which was more than 60% in a comatose or ventilated patient after the incidence of haemorrhagic stroke.

Hypertension is an increasingly important medical and public health issue. The prevalence of hypertension increases with advancing age to the point where more than half of the people in 60-69 years of age and more than three- fourth of people more than 70 years of age are affected. Data from observational studies involving more than 1 million individuals indicated that for every 20mmhg increase in systolic BP and 10mmhg increase in diastolic BP there is doubling of mortality from both Ischaemic heart disease and stroke. Hypertension is second only to diabetes for the cause of alarming increase in end stage renal disease. So it is an important public concern to be addressed seriously.

With the increased incidence of non traumatic intracerebral bleeds with hypertension, it becomes imperative to study about the clinical profile of patients admitting with hypertensive ICH .It helps in identifying to what extent hypertension remains the cause of intracranial haemorrhage, the role of CKD as an add on risk factor in hypertensive bleed, the role of good drug compliance for hypertension in preventing ICH, the correlation between location of bleed and hypertension being the cause. This prospective study reports would be helpful in educating patients and creating awareness about the importance of strict drug compliance for hypertension in preventing the disastrous complications it would bring on for the patients.

AIM AND OBJECTIVES OF THE STUDY

1. To assess the drug compliance in known cases of hypertension who are all presenting with haemorrhagic stroke
2. To assess the location of bleed in patients presenting with haemorrhagic stroke.
3. To assess the other risk factors contributing to the poor outcome of haemorrhagic stroke.

REVIEW OF LITERATURE

HYPERTENSION

Hypertension is one of the leading cause of global burden of the disease. It has to be given prime importance since most of the cases are diagnosed only at the time of development of complications. Hypertension is present in all populations worldwide except for a small number of individuals living in developing countries. In industrialized societies, blood pressure increases steadily during the first two decades of life. Hypertension continues to be the major risk factor for premature cardiovascular disease (CVD) worldwide .Treatment of hypertension has made a major impact in reducing the incidence of major diseases like CORONARY ARTERY DISEASE, CONGESTIVE HEART FAILURE, PERIPHERAL VASCULAR DISEASE, STROKE. The prevalence of hypertension is increasing worldwide, In developed countries, because of increasing longevity with its burden of systolic hypertension and in developing countries because of increasing obesity, diabetes, and dyslipidemia related to urbanization. Labelling the patient as a hypertensive is complicated, because so many factors like subjective error, instrumental error and observer error may alter the BP recording.

EPIDEMIOLOGY

Hypertension is a worldwide epidemic, Globally, an estimated 26% of the world's population (972 million people) has hypertension, and the prevalence is expected to increase to 29% by 2025, driven largely by increases in economically developing nations. The high prevalence of hypertension causes a tremendous public health burden. As a primary contributor to heart disease and stroke, the first and third leading causes of death worldwide, respectively, high blood pressure was the top modifiable risk factor for disability adjusted life-years lost worldwide in 2013. There are no well coordinated national surveys for the prevalence of hypertension available from the Indian subcontinent. Several regional small surveys reported in last two decades have reported a prevalence which varies from 6.15%-36.36% in men and 2%-39.4% in women in urban area and from 3%-36% in men, 5.8%-35.8% in women in rural areas.

CAUSES OF HYPERTENSION:

Primary hypertension is the most prevalent form of hypertension accounting upto 90% of all cases of hypertension where exact cause is unknown.

Secondary HTN where some other disease or abnormality such as tumours of adrenal gland, Renal causes include chronic pyelonephritis, chronic glomerulonephritis, Chronic kidney disease ,Urinary tract

obstruction, Renin-producing tumor, Liddle syndrome, renovascular conditions.

Endocrine causes account for 1-2% and include exogenous or endogenous hormonal imbalances. Exogenous causes include administration of steroids, oral contraceptives like estrogen.

Endogenous hormonal causes like Primary hyperaldosteronism, Cushing syndrome, Pheochromocytoma, Congenital adrenal hyperplasia

Collagen vascular disease, vasculitis, pregnancy, coarctation of aorta are also involved in causation

Drugs and toxins that Cause hypertension include the following:

Alcohol, Cocaine, Cyclosporine, Tacrolimus, NSAIDs, Erythropoietin, Adrenergic medications, Decongestants containing ephedrine, Herbal remedies containing licorice (including licorice root) or ephedrine (and ephedra), Nicotine

Other conditions like Hyperthyroidism and hypothyroidism, Hypercalcemia, Hyperparathyroidism, Acromegaly, Obstructive sleep apnea Pregnancy-induced hypertension can result in hypertension.

DIAGNOSIS

The documentation of hypertension, which is confirmed after an elevated blood pressure (BP) on at least 3 separate occasions (based on the average of 2 or more readings taken at each of ≥2 follow-up visits after initial screening). An accurate measurement of blood pressure is the key to diagnosis. Several determinations should be made over a period of several weeks. At any given visit, an average of 3 blood pressure readings taken 2 minutes apart using a mercury manometer is preferable.On the first visit, blood pressure should be checked in both arms and in one leg to avoid missing the diagnosis of coarctation of aorta or subclavian artery stenosis. The patient should rest quietly for at least 5 minutes before the measurement. Blood pressure should be measured in both the supine and sitting positions, auscultating with the bell of the stethoscope. As the improper cuff size may influence blood pressure measurement, a wider cuff is preferable, particularly if the patient's arm circumference exceeds 30 cm. Although somewhat controversial, the common practice is to document phase V (a disappearance of all sounds) of Korotkoff sounds as the diastolic pressure. A detailed history should extract the necessary information. The evaluation of hypertension involves accurately measuring the patient's blood pressure, performing a focused medical history and physical examination, and obtaining results of routine laboratory studies. A

12-lead electrocardiogram should also be obtained. In all patients following factors should be assessed.

- Possible causes of hypertension
- Presence of end-organ damage
- Cardiovascular risk factors

Baseline values for judging biochemical effects of therapy

Based on recommendations of the Eight Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 8), the classification of BP for adults aged 18 years or older has been as follows.

BP Category	SBP		DBP
Normal	<120 mm Hg	and	<80 mm Hg
Elevated	120–129 mm Hg	and	<80 mm Hg
Hypertension			
Stage 1	130–139 mm Hg	*or*	80–89 mm Hg
Stage 2	≥140 mm Hg	*or*	≥90 mm Hg

In individuals with suspected secondary hypertension and/or evidence of target-organ disease, more detailed evaluation such as CBC, chest radiograph, uric acid, and urine microalbumin may be needed.

Ambulatory or home blood pressure monitoring provides a more accurate prediction of cardiovascular risk than do office blood pressure readings.

LIFESTYLE MODIFICATION:

Weight loss (range of approximate systolic BP reduction [SBP], 5-20 mm Hg per 10 kg)

Limit alcohol intake to no more than 1 oz (30 mL) of ethanol per day for men or 0.5 oz (15 mL) of ethanol per day for women and people of lighter weight (range of approximate SBP reduction, 2-4 mm Hg)

Reduce sodium intake to no more than 100 mmol/day (2.4 g sodium or 6 g sodium chloride; range of approximate SBP reduction, 2-8 mm Hg)

Maintain adequate intake of dietary potassium (approximately 90 mmol/day)

Maintain adequate intake of dietary calcium and magnesium for general health .Stop smoking and reduce intake of dietary saturated fat and cholesterol for overall cardiovascular health Engage in aerobic exercise at least 30 minutes daily for most days (range of approximate SBP reduction, 4-9 mm Hg)

PHARMACOLOGICAL THERAPY:

If lifestyle modifications are insufficient to achieve the goal BP, there are several drug options for managing hypertension. Thiazide diuretics, Angiotensin-converting enzyme inhibitor (ACEI) / Angiotensin receptor blocker (ARB), or Calcium channel blocker (CCB) are the preferred agents in non black populations, whereas CCBs or thiazide diuretics are favored in black hypertensive populations. These recommendations do not exclude the use of ACE inhibitors or ARBs in treatment of black patients, or CCBs or diuretics in non-black persons. Often, patients require several antihypertensive agents to achieve adequate BP control.

Hypertension is the most important modifiable risk factor for coronary heart disease the leading cause of death , stroke ,congestive heart failure, end-stage renal disease, and peripheral vascular disease. Therefore, health care professionals must not only identify and treat patients with hypertension but also promote a healthy lifestyle and preventive strategies to decrease the prevalence of hypertension in the general population.

PATHOPHYSIOLOGY OF HYPERTENSION IN CAUSING INTRACERBRAL BLEED

A very high blood pressure leads to damage to end organs, tissue dysfunction and ultimately death. So that’s why, it is important to know

the pathophysiology of hypertension and its complications. Both systolic and diastolic blood pressure help in diagnosing the risk. Almost 95% of hypertension is idiopathic or otherwise known as essential hypertension. The incidence and prevalence of complications related to hypertension increases in propensity with advancing age. A very tiny percentage of patients with hypertension show rapidly rising BP, which if untreated may result in death within 1 to 2 years and is known as malignant hypertension. Hypertension increases arteriosclerosis and causes degenerative changes in the walls of arteries which can lead to cerebrovascular accident and Aortic dissections. Hypertension causes 2 forms of blood vessel pathologies namely hyaline arteriosclerosis and hyperplastic arteriosclerosis.

In hyaline arteriosclerosis vessels show homogenous hyaline pinkish thickening. This is due to leakage of plasma proteins through injured cells of endothelium as well as increased smooth muscle proliferations. It is a generalised pathology.

In hyperplastic arteriosclerosis, patients with severe hypertension, vessels shows concentric, laminated, thickening of vessels showing onion skin appearance. The laminations are made of thickened smooth muscle cells, basement membrane with fibrinoid deposits and vessel wall necrosis. This can be mostly seen in kidney.

The role of hypertension in intracerebral haemorrhage is very important to understand the need and significance of treatment of hypertension. ICH due to hypertension results due to spontaneous rupture of small penetrating artery deep in brain matter. The commonest sites of intracerebral haemorrhage in brain are putamen, basal ganglia, thalamus, cerebellum and pons. The reason for the haemorrhage is because the small arteries in these areas are prone to vascular injury in hypertensive patients i.e., Lipohyalinosis. Haemorrhage in other areas of brain suggest other causes of bleeds like neoplasms, haemorrhagic disorders, vascular malformation and cerebral amyloid angiopathy.

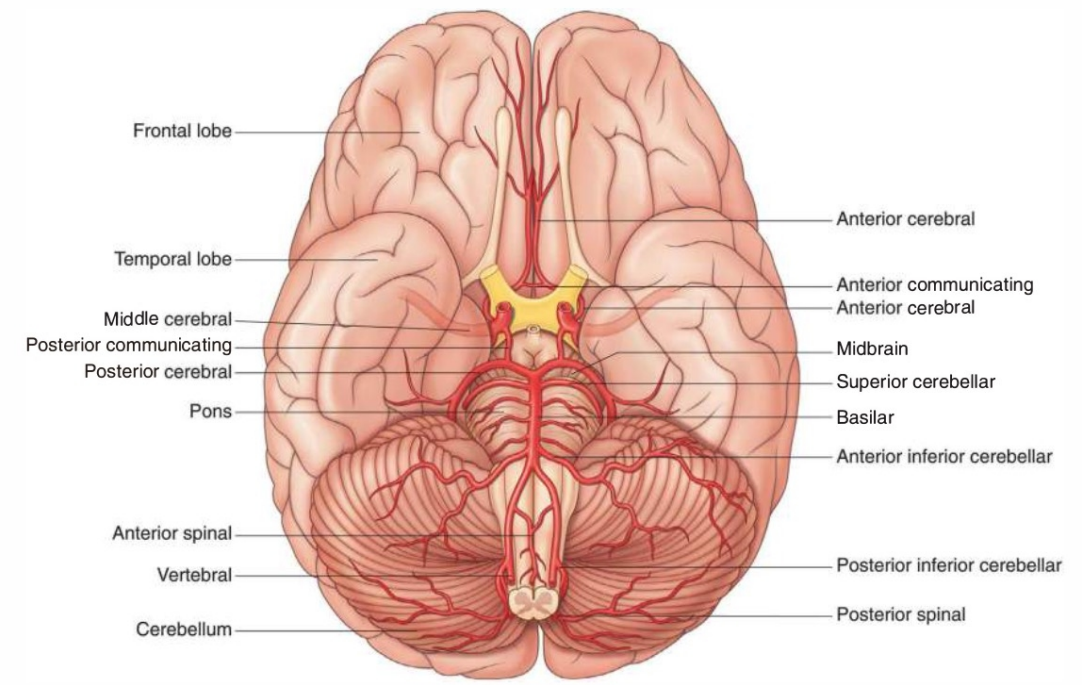
The intracerebral haemorrhage may results in clot which may be small or large which will compress the brain tissue and result in herniation of brain matter and ultimately death. Dissection of blood into ventricles is a poor predictor of morbidity which results in hydrocephalus.

Most of the intracerebral haemorrhages progress over 30- 90 mins and those associated with therapy of anticoagulants progress till 48 hours. Now a days, it is believed that even in patients without coagulopathy expansion of clot happens on first day. The macrophages start to phagocytize the clot at the surface within the first 48 hours. The next 6 hours the clot will resolve gradually to a cavity which is slit like and covered with glial tissue scar and macrophages.

BLOOD SUPPLY OF BRAIN

Cerebral arteries

The blood supply of the Brain is mainly from 2 Arteries 1) Vertebral artery 2) Internal carotid artery. Vertebral artery supplies inferior half of brain i.e., Brainstem, Midbrain, Occipital lobe, most of the Thalamus, Inferior Temporal lobe. Internal carotid arteries supplies frontal lobe, parietal lobe, lateral part of temporal lobe, Internal capsule and basal ganglia.



Carotid Artery:

The internal carotid artery (ICA) is one of the Terminal branches of common carotid artery. The cervical portion of ICA has no branches. It enters the skull in petrous part of temporal bone through carotid canal. ICA

has petrous segment which pierces the petrous bone and intracranial segment which passes through the cavernous sinus. The ‘S’ shaped course within the cavernous sinus can be called as ‘carotid siphon’. Adjacent structures to the ICA within the sinus are Occulomotor Nerve, Trochlear nerve, Ophthalmic and maxillary division of Trigeminal nerve and Abducent Nerve. ICA was also surrounded by sympathetic plexus within the cavernous sinus. The juxtasellar portion of ICA renders meningohypophyseal trunk, supplies the posterior pituitary lobe and meninges adjacent to it. ICA gives ophthalmic artery, posterior communicating antery and anterior choroidal artery just after exiting the cavernous sinus. Left and right ICA & their communicating branches & basilar artery forms the circle of Willis at the brain base. There may be many anomalies in the Circle of Willis like Duplication, persistent embryonic remnants or absent vessels. Normal circle of willes can be found only in 50 % of the population without any congenital abnormalities. Around 80% of population with neural dysfunction have abnormality in the circle of willis.

Ophthalmic artery gives orbital, extra orbital, central retinal, anterior ciliary, long and short posterior ciliary branches. The posterior communicating artery, which communicates ICA and posterior cerebral artery also supplies anterior medial thalamus and walls of 3rd ventricle.

The anterior choroidal artery goes along the optic tract and around cerebral peduncle enters inferior horn of lateral ventricle and supplies choroid plexus of lateral ventricle. Anterior choroidal artery supplies optic tract, Hippocampus, tail of caudate nucleus, medial and intermediate globus pallidus, middle third of cerebral peduncles, outer lateral geniculate body along its course. It also supplies posterior 2/3rd of posterior limb of internal capsule, Retro lenticular and sub- lenticular parts of internal capsule.

ICA terminates by dividing into anterior cerebral artery and middle cerebral artery near the medial side temporal pole and lower Sylvian fissure.

Anterior cerebral artery (ACA):

Anterior cerebral artery is the smallest terminal branch of ICA runs forward before optic chiasma and medially to the medial longitudinal fissure.

Anterior communicating artery communicates the Left and Right anterior cerebral artery.

Angioraphically ACA can be divided into three segments.

A1- Horizontal or Pre communicating

A2 – Vertical or Post communicating

A3- Distal or cortical branches

Then ACA courses superiorly and anteriorly within the inter hemispheric fissure, medial to hemisphere close to the corpus callosum.

Four primary cortical branches

1. Orbitofrontal artery
2. Frontopolar artery
3. Callosomarginal artery
4. Pericallosal artery

Orbitofrontal artery supplies olfactory lobe, gyrus rectus, medial and inferior parts of orbital gyri.

Frontopolar artery supplies medial surface of the prefrontal region.

Callosomarginal artery travels backward in the cingulate sulcus and gives three branches anterior, middle and posterior internal frontal branch and then continues backward to the posterior parietal region.

Finally ACA continues as pericallosal artery along the body and posterior part of corpus collosum & ends by anastamosing with branches of posterior communicating artery. The cortical branches of ACA supplies

Medial & orbital surface of frontal lobe.

Medial surface of parietal lobe

Cingulate gyrus

Genu of corpus callosum

Anterior 4/5 th of corpus callosum

Corpus callosum was supplied by multiple small branches of ACA and pericallosal arteries.

The Recurrent Artery of Heubner (Medial striate artery) largest deep branch of ACA, recurrent in its course joins with deep branches of middle cerebral artery after passing through anterior perforated space. Heubner's artery supplies lower part of head of caudate nucleus, lower part of anterior putamen, anterior pole of Globus pallidus, frontal half of anterior limb of internal capsule, Anterior external capsule and lateral ventricle.

Middle cerebral Artery (MCA)

Middle cerebral artery is the largest cerebral artery. Angiographically it is divided into four segments.

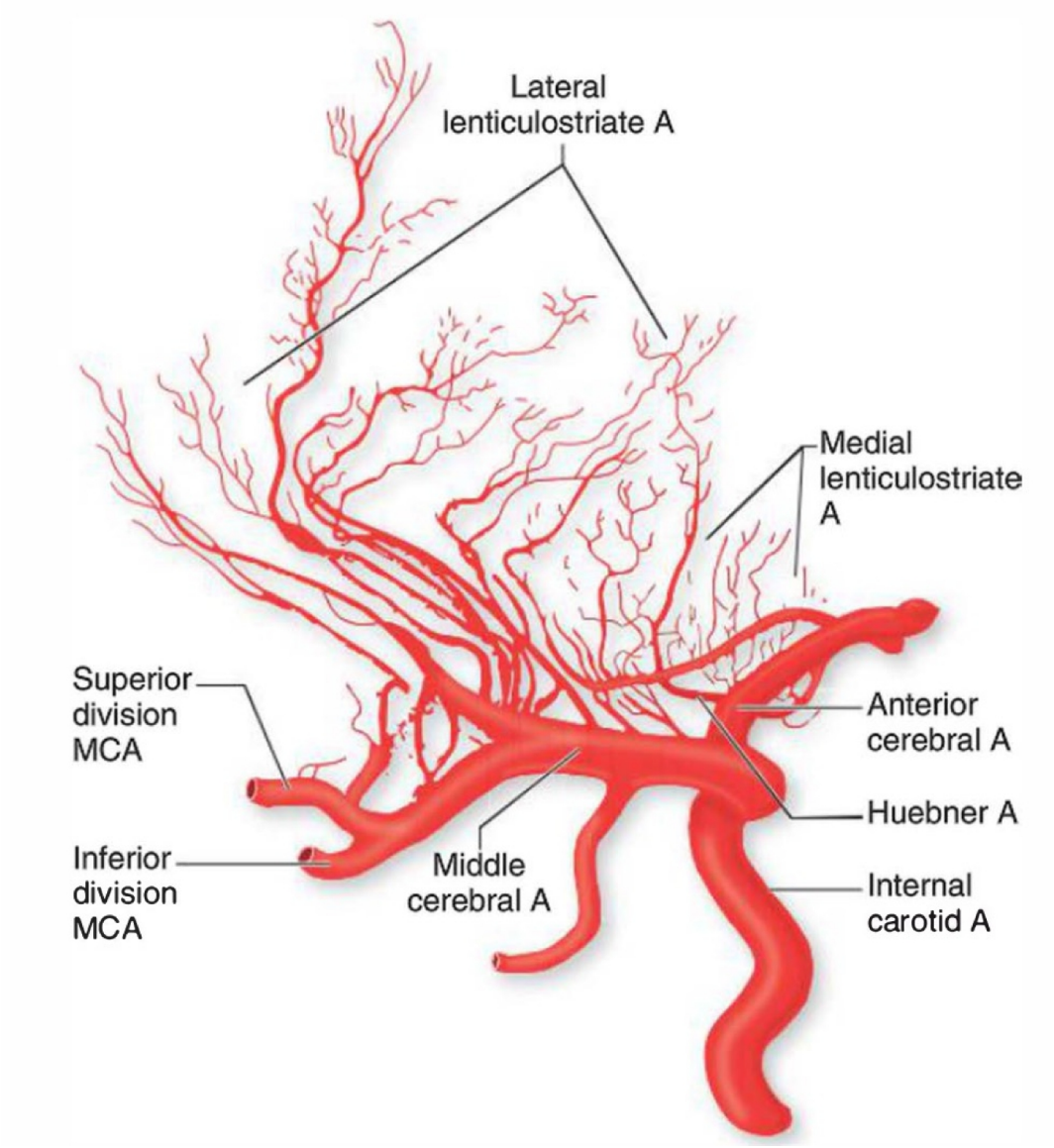
1. M1 (horizontal)
2. M2 (Insular)
3. M3 (Opercular)
4. M4 (cortical branches)

Middle cerebral artery turns laterally (after giving off posterior communicating artery) i.e. M1 segment gives lenticulostriate arteries.

Lenticulostriate arteries supplies all portion of putamen except anterior pole, superior portion of head of caudate nucleus & all of its body, globus pallidus lateral portion, posterior part of anterior limb of internal capsule, Genu & Anterior 1/3rd of posterior limb of internal capsule.

Lenticulostriate arteries are the most common involved vessels in hypertension induced fibrinoid necrosis. This will leads to lacunar strokes or haemorrhgic stroke. Charcot termed these arteries as ‘artery of cerebral haemorrhage’ because of the frequent occurrence of stroke in this region.

MCA enters the Sylvian fissure laterally, then travels along the depths of the fissure between the frontal and temporal lobes. Many cortical branches of MCA supplies lateral surface of brain.



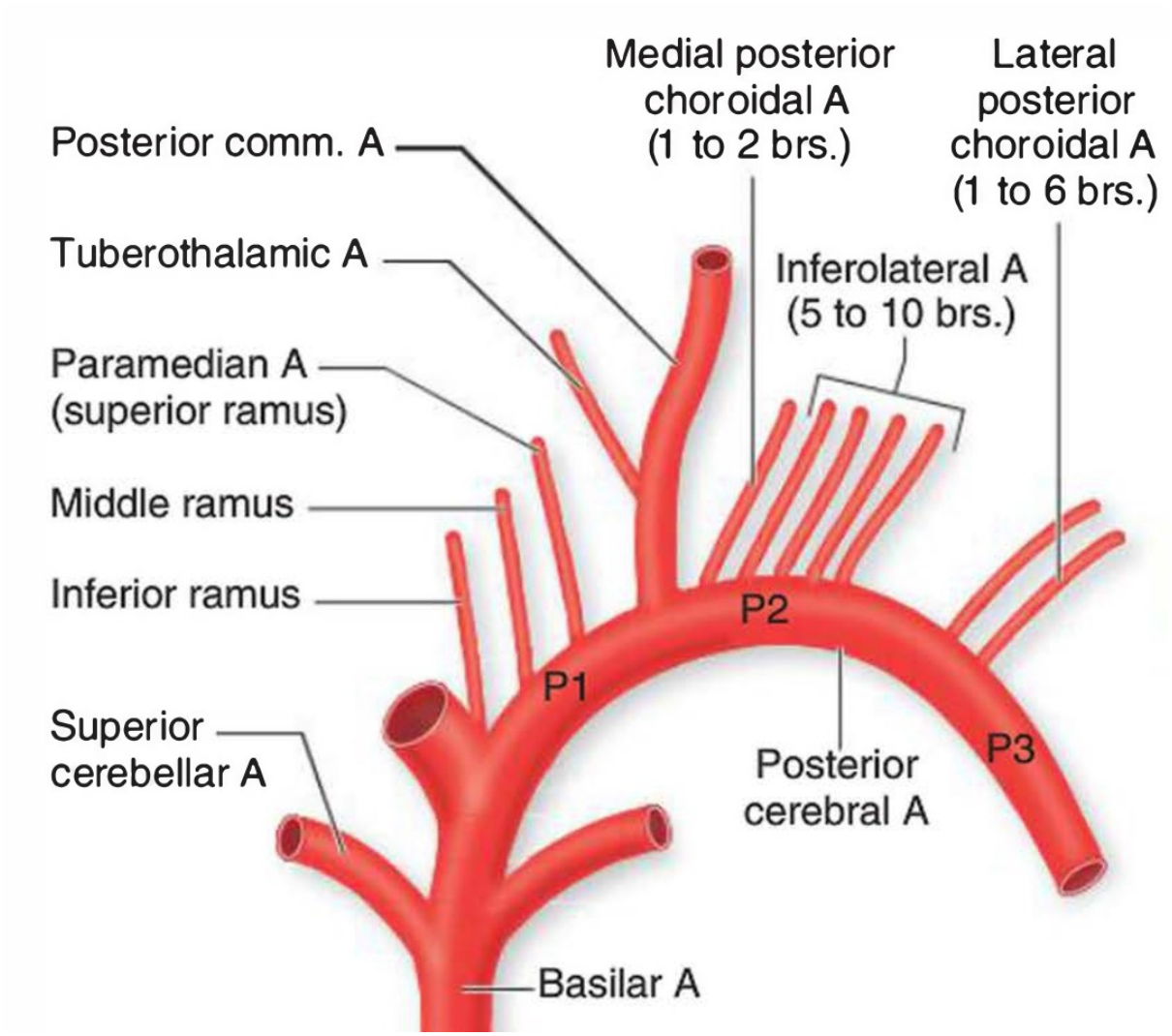
- Anterior temporal artery courses out of the Sylvain fissure runs towards temporal lobe and supplies temporal pole, Anterior 1/3 of superior & middle temporal gyri.
- Orbitofrontal artery supplies blood to orbital surface of frontal lobe, Lateral orbital gyrus, lateral portion of inferior frontal convolution.

- Pre Rolandic artery gives branches to anterior precentral gyrus posterior portions of middle & inferior frontal convolutions.
- Rolandic artery gives branches to posterior precentral gyrus and anterior postcentral gyrus.
- Anterior parietal artery supplies posterior postcentral gyrus and anterior parietal convolution.
- Posterior temporal artery supplies supramarginal gyrus and inferior parietal lobule.
- Angular artery, terminal branch of MCA, supplying angular gyrus and parietal lobe adjoining area
- Terminal cortical branches of MCA anastomose with branches of ACA and PCA. These create a collateral blood supply, which prevents the extent of infarction if a particular vessels is occluded.

Vertebrobasilar system :

Bifurcation of the basilar artery leads to the formation of posterior cerebral arteries (PCA), angiographically PCA is devided in 4 segment

1. P1 (Mesencephalic)
2. P2 (ambient)
3. P3 (quadrigeminal)
4. P4 (Calcrine)



PCA travels backward and around the cerebral peduncle laterally, close to upper pole & parallel to superior cerebellar artery. After the communication with posterior commutating artery, PCA travels medial surface of cerebral hemispheres, beneath the splenium and reaches medial & inferior surface of temporal lobe & occipital lobe. There it gives four cortical branches.

1. Anterior temporal artery
2. Posterior temporal artery

3. Calcrine artery
4. Parietoocipital or posterior occipital artery

Cortical branches of PCA supplies medial portion of occipital lobe along with entire visual cortex, inferior temporal lobe and splenium of corpus callosum. Then it winds around lateral surface of occipital and temporal lobe and anastomose with terminal branches of MCA and ACA.

Brainstems receives its blood supply from two main arteries – vertebral and basilar arteries. The paired vertebral arteries enters skull through foremen magnum – Angiographically vertebral artery divides into

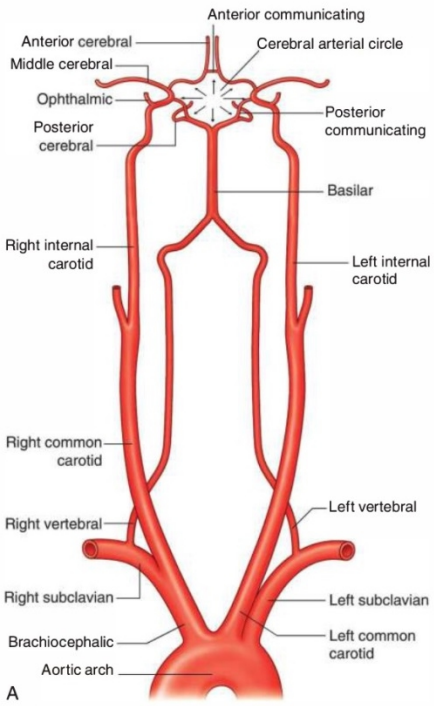
1. V1 (extra osseous)
2. V2 (foraminal)
3. V3 (extra spinal)
4. V4 (intradural)

Right vertebral artery arises from right subclavian artery and left is the direct branch of arch of aorta, proximal to the left subclavian artery origin. Among the two vertebral arteries, Left is usually dominant, sometimes right vertebral artery may be absent completely. The two vertebral arteries travels through foramen of transverse process of cervical vertebra C6-C2. At the craniovertebral junction these two arteries deviates from the

foremen and loops before traversing foramen magnum. Then they unite to form basilar artery at the level of pontomadullary junction. At the mid portion of each vertebral artery the posterior inferior cerebellar artery (P1CA) arises and supplies medulla and cerebellum.

Just prior to the formation of basilar artery, vertebral artery gives anterior and posterior spinal artery. Paired anterior spinal arteries unite at mid medulla travels downwards and supplies lower medulla, cervico medullary junction& upper spinal cord. Posterior spinal artery supplies the dorsal spine and remains separate.

Basilar artery, a short and thick artery terminates and bifurcates at the upper border of pons.



Three types of vessels arise from basilar artery.

1. Paramedian perforators
2. Short circumferential arteries
3. Long circumferential arteries

- Para median perforators - small vessels originating from basilar main truck supplying the midlines structures.
- Short circumferential vessels –larger vessels supplies lateral structures.
- Long circumferential vessels – posterior inferior cerebellar artery (PICA), anterior inferior cerebellar artery (AICA) superior cerebellar artery (SCA). These partial long circumferential vessels supplies lateral brainstem and the cerebellum.

Basilar artery terminates by dividing into two posterior cerebral arteries at the level of pontomesecephalic junction. Interpeduncular branches gives blood supply to deep midline superior brainstem, medial cerebral peduncle, III CN, IV CN and adjutant subthalamic region. Superior and inferior colliculus supplied by quadrigeminal branches. Vessels arising from the top of the basilar artery and adjacent proximal PCA supplies medial and lateral posterior choroidal, thalamoperforators & thalmogeniculates.

Posterior choroidal arteries supplies mid brain, choroid plexus of 3rd ventricle, superior medial thalamus.

Posteromedial arteries from posterior communicating artery supplies hypophysis, IIIrd ventricle, medial and anteromedial thalamus, infundibulum, Tuberal and mammillary region of hypothalamus, red nucleus, subthalamic structures, medial cerebral peduncle. Posterolateral (or) thalamogeniculate arteries supplies Inferior half of thalamus, Posterior internal capsule, geniculate bodies, superior colliculus and superior cerebellar peduncle.

INTRACEREBRAL HAEMORRHAGE

ICH causes high degree of morbidity and mortality. It account for 10 to 15% of all strokes with an estimated 80 day mortality ranging from 30 to 52%. There are various etiologies for ICH which includes trauma, long standing arterial hypertension, intracranial aneurysms, intracranial vascular malformations, bleeding diathesis, cerebral amyloid angiopathy, primary or metastatic brain tumour, antiplatelet therapy, anticoagulant therapy, illicit drug usage, thrombolytic therapy.

Supratentorial ICH (80%) are more common than infratentorial ICH (20%) due to hypertension.

Hypertensive intra cranial bleed commonly involve the territories of lenticulostriate branch of MCA, thalamoperforator, superior cerebellar and paramedian branches of basilar artery.

Clinical features of intra cranial bleed are similar to that caused by the ischemic stroke. The most common presenting signs are altered level of consciousness, severe headache, vomiting, seizures, hemiplegia. The clinical course is characterised by gradual deterioration over a period of time. The enlargement of haemorrhage is maximum during the first few hours after the onset of symptoms and hence associated with poor outcome. The 'spot sign' within the brain haemorrhage caused due to the active extravasation of contrast in contrast enhanced CT brain is indicative of active bleed within the haemorrhage.

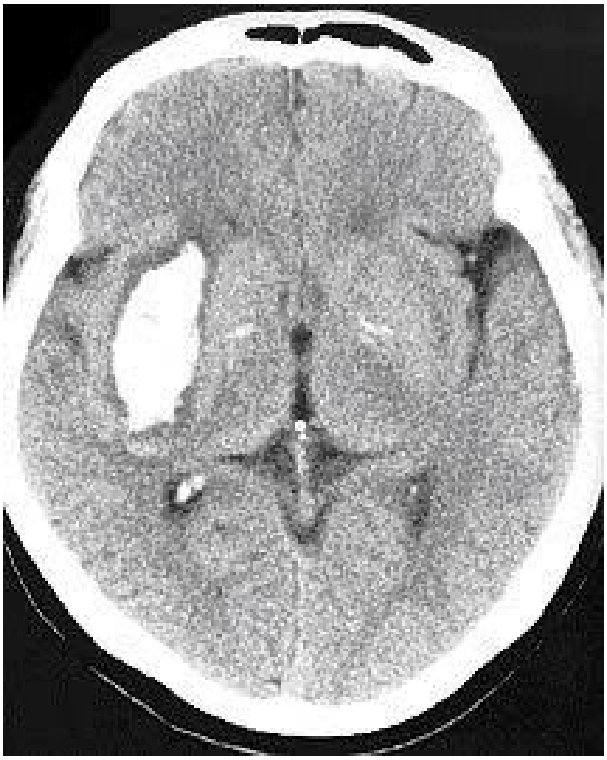
The clinical finding depends on the location, size, compressive effect caused by the haematoma on the adjacent structures. Seizures are common if the bleed is likely to cause cortical irritation. If the bleed spreads on the sub arachnoid space, signs of meningial irritation may be present. Fundus examination may reveal retinal haemorrhages. The most common areas of hypertensive ICH are

- Putamen
- Subcortical white matter
- Thalamus

- Pons
- Cerebellum
- Ventricles

Location specific signs

1. **Putaminal haemorrhage**



It is the commonest site of hypertensive ICH. The bleed may be confined to the putamen or extend to involve internal capsule, centrum semiovale, corona radiata, external capsule, extreme capsule or into the ventricular system.

The most common site in putamen is lenticular nucleus either isolated or along with insula. So the presentation would be contralateral

hemiplegia / hemiparesis and gaze preference towards the side of lesion. Pupils are not affected. Contralateral sensory loss may be present. Aphasia may present if there is haemorrhage in the dominant putamen. Non dominant putaminal haemorrhage may cause, apractagnosia, constructional apraxia, Hemineglect in visual field. The peculiar presentation on non-dominant putamen haemorrhage is 'alloestheisa' ie, painful stimulus in the area of hemisensory loss will be perceived on the corresponding opposite side of the body after about 30 seconds. It is commonly noted in trunk and proximal limbs, rarely in face and distal limbs. Hydrocephalus, large haematoma volume, poor consciousness level at the time of presentation are predictors of poor outcome.

Chung et al, divided striato-capsular haemorrhages into 6 types. The region 'striato-capsular' includes lenticular nucleus, caudate nucleus, internal capsule, external capsule, sub insular area.

The 6 types are

- Anterior type (11%)
- Middle type (7%)
- Postero medial type (4%)
- Posterolateral type (33%)
- Lateral type (21%)
- Massive 24%)

Anterior type

These are small haematoma in the territory of artery of Heubner which involve head and body of the caudate nucleus. The haemorrhage commonly extends into anterior horn of lateral ventricle. The clinical presentation is usually severe headache, neck stiffness, contralateral weakness of upper limb and lower limb, confusion, abulia, perseveration. prognosis is usually good.

Middle type

It involves the territory of lenticulostriate branch of middle cerebral artery. The areas involved are Globus pallidus and medial putamen. Intraventricular extension is rare. The clinical presentation is contralateral sensory and motor deficits and gaze paresis towards the side of lesion. The prognosis is good.

Posteromedial type

It is very small haematoma in the territory of Anterior choroidal artery which involves anterior half of the posterior limb of internal capsule. The presentation is dysarthria, hemiplegia, hemisensory loss. The prognosis is excellent.

Posterolateral type

These are large size haemorrhage in the territory of posteromedial branch of lateral lenticular striate arteries, which involve posterior half of putamen and posterior limb of internal capsule. The clinical presentation includes altered sensorium, contralateral motor and sensory deficit, hemineglect or anosognosia prognosis is poor in 75% of cases and good in 25%.

Lateral type

These are large elliptical shaped haemorrhages in the territory of lateral branch of lateral lenticutostriate arteries which affects the area between external capsule and insular cortex. The clinical features include altered sensorium, gaze paresis initially. Later on, there may be motor defects, aphasia, anosognosia. Prognosis is good except if the hematoma is large.

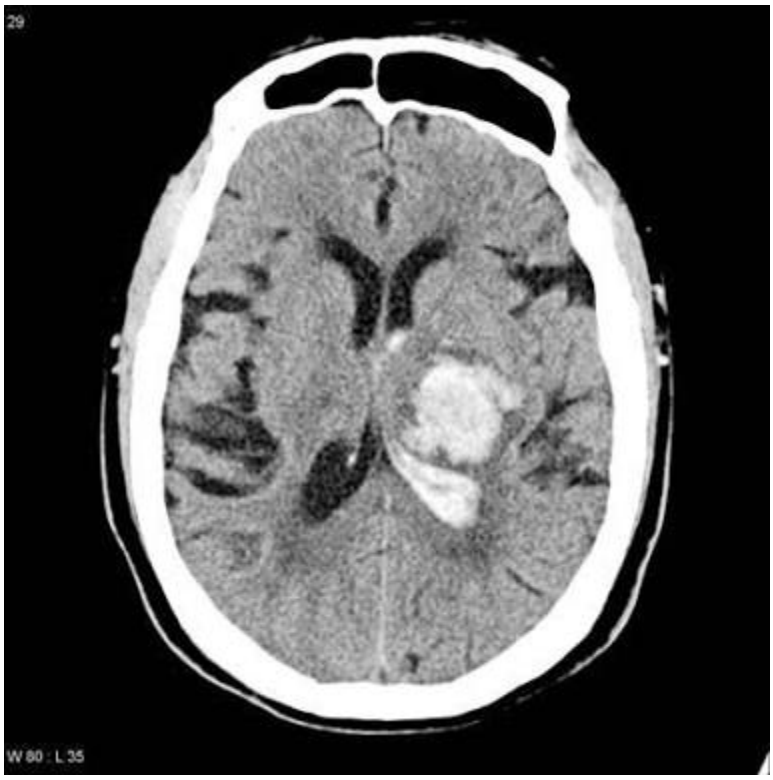
Massive type

It is a very large haemorrhage covering the entire striatocapsular area. The caudate nucleus and anterior limb of internal capsule may be spared occasionally. These are usually associated with intraventricular expansion and obstructive hydrocephalus. The GCS is usually poor with

gaze preference opposite to the side of lesion. Signs of subfalcine or transtentorial herniation may be present.

2. Thalamic haemorrhage

Hypertension is the most common cause of haemorrhage in the thalamus. The haemorrhage may extend to internal capsule, subthalamus, mid brain and third ventricle. Extension to the ventricle has good prognosis.



The clinical features are contralateral hemisensory loss, vertical gaze palsy. If the haemorrhage extends to internal capsule, there is contralateral hemiplegia. Thalamic haemorrhage on dominant side causes aphasia whereas the haemorrhage on non-dominant side causes visuospatial

abnormalities, anosognosia and arm levitation. If the ascending reticular activation system is affected the symptoms may vary from hyper somnolence to altered level of consciousness. Other manifestations being vertical gaze palsy, convergence –retraction nystagmus, pupillary light-near dissociation, disconjugate with abduction restriction of one or both eyes (PSEUDO-SIXTH NERVE PALSY). The eyes may be tonically deviated opposite to site of haemorrhage (WRONG WAY EYES). The clinical features does not reliably differentiate between infarct and haemorrhage.

A study of 50 patients with thalamic bleeds was done and classified

Small bleeds (<8mm) (7 patients) – Transient hemiparesis, numbness or headache with papilloedema. All of them recovered completely.

Medium sized bleeds (9=30mm) (24 patients) – had hemiparesis and hemiplegia. No intraventricular extension.

Large bleeds (>30mm) (19 patients) – intraventricular extension was present. They had impaired consciousness, hemiparesis, headache, pinpoint puil, vertical gaze palsy was present. All the patients died.

The small thalamic bleeds are further classified according to the site of location of bleed.

Anterolateral type

Mild 'prefrontal' signs such as impaired verbal memory and inattention would be present. Motor and sensory deficits are mild.

Posterolateral type

Severe motor and sensory deficits would be present along with eye signs such as miosis, loss of light reflex and upward gaze palsy. This type has the poorest prognosis among the small thalamic bleeds. The patients would have persistent residual motor and sensory deficits.

Medial type

These patients initially present with impaired consciousness followed by prefrontal signs such as memory impairment or inattention of longer duration.

Dorsal type

They have predominant 'parieto occipital signs' for example aphaisa in dominant sided lesions. Topographic memory disturbance in non-dominant sided lesions.

Posterior thalamic haemorrhage presents as an syndrome and has the following components.

- Contralateral saccadic hypometria
- Broken pursuit on the ipsilateral side.
- Ipsilateral ptosis and miosis (due to involvement of hypothalamus)
- Unilateral sensory ‘thalamic neglect’.
- Contralateral hemiplegia and hemianaesthesia.
- Gaze preference towards the side of the lesion.

In another study, thalamic haemorrhages was classified into 5 other different types.

Anterior type (7%)

It involves territory of tuberothalamic arteries. There is often intraventricular extension into the anterior horn of lateral ventricle. Behavioural disturbance is the major presentation. Prognosis is good.

Posteromedial type (14%)

It involves the territory of thalamo-subthalamic paramedian arteries. It usually accompanies intraventricular extension into third ventricles causing obstructive hydrocephalus compressing the midbrain. It has poor prognosis.

Posterolateral type (44%)

It involves territory of thalamogeniculate arteries. The haemorrhage is usually large with intraventricular extension into posterior limb of

internal capsule. The patients have hemiplegia, hemianaesthesia, hemineglect and language disturbances. It has very poor prognosis.

Dorsal type (18%)

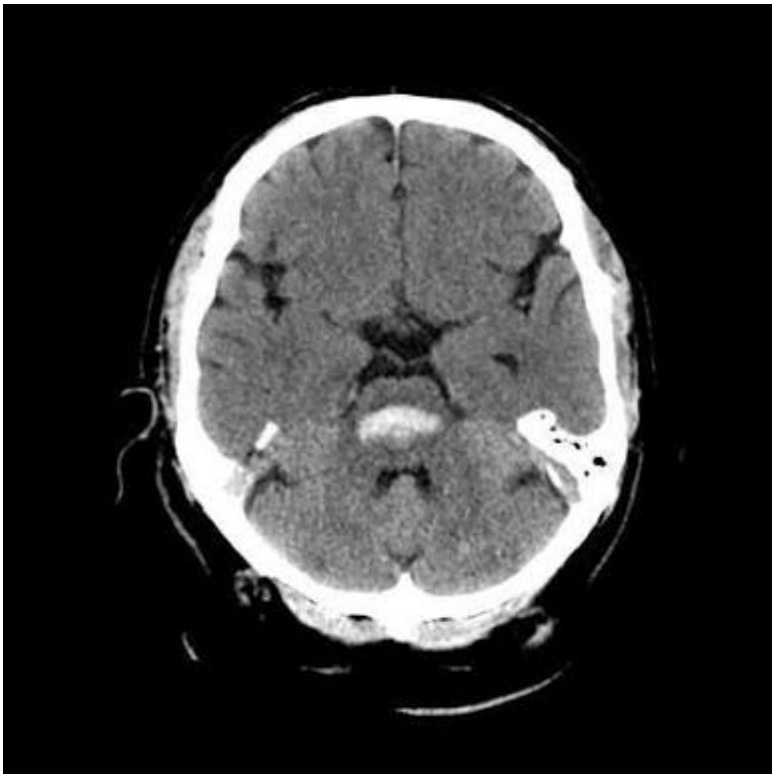
It involves territory of posterior choroidal arteries. Sensory and motor deficits are mild. It has very favourable prognosis.

Global type (18%)

This type includes haematomas involving entire thalamus.

It resembles posterolateral type. It is associated with hydrocephalus mass effect.Patients often exhibit decerebrate posture, comatosed with upward gaze palsy and pinpoint pupil.Mortality is around 80%.

3. **Pontine haemorrhage**



It is usually caused due to hypertensive ICH. It is caused by ruptured paramedian arterioles. The usual site is in the mid pontine level at the junction of basis points and tegmentum. Pons is a small area in the brain stem, in which the haemorrhage into it causes intraventricular extension into the fourth ventricle causing obstructive hydrocephalus with mass effect into the surrounding structures. The presenting features are coma with decerebrate rigidity, quadriparesis, tachycardia, hyperthermia, absent corneal and conjuncitival reflexes, pinpoint pupils with preserved light reflex, impaired horizontal eye moments. Some cases show occular bobbing.

A peculiar group of patients presents with ‘locked in syndrome’. It is a condition in which the patient is mute and lacks total motor activity. But he is awake, alert capable of perceiving all sensory stimuli. Horizontal moments of eye are abolished due to the PPRF involvement. The only possible movements of the patient are movement of eyelid and vertical eye movement. EEG ensures the wakefulness state of the patient. It is usually due to ventral pontine haemorrhage. If the haemorrhage extends into midbrain the vertical eye movements may also be lost accompanied with ptosis.

Pontine haemorrhage rarely cause bilateral deafness because of the lesion in the ventral acoustic striae decussating in trapezoid body.

Clinically, pontine haemorrhage is of 3 types

Classic type (60%)

Complete pontine destruction with quadriplegia, coma hyperthermia and tachycardia. High risk of mortality is there.

Hemipontine syndrome (20%)

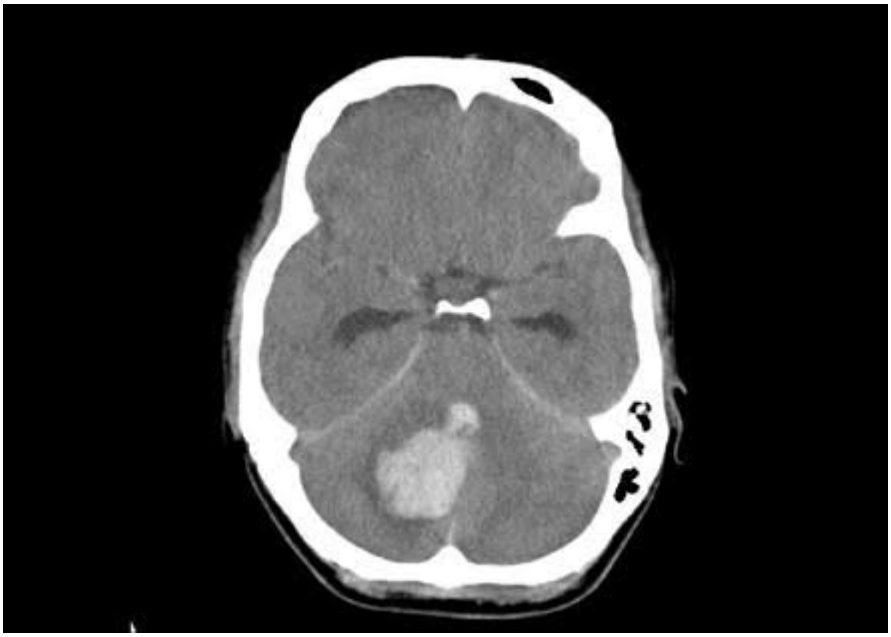
It involves basis points and tegmentum unilaterally. Clinically, it present as hemiparesis, preserved sensorium, ipsilateral impaired corneal and conjunctival reflex, skew deviation, dysarthria, ipsilateral LMN facial paly, sensory loss in ipsilateral face and contralateral extremity. It has better prognosis.

Dorsolateral tegmental syndrome (20%)

They present with same clinical features as hemipontine syndrome along with gaze palsy or unilateral or bilateral 6^{th} nerve palsy.

4. Cerebellar haemorrhage

It accounts for 10% of nontraumatic ICH. These haemorrhages have varied clinical presentation depending on the location, size of the bleed, associated brainstem compression and extension into the fourth ventricle. A massive bleed may cause tonsillar herniation, local brainstem compression or upward tentorial herniation.



Hypertensive cerebellar bleed is usually at the site of Dentate nucleus due to rupture of distal branch of superior cerebellar artery. Partly the bleed may involve the vermis but it is usually due to vascular malformation.

Clinically it present as acute onset of occipital or frontal headache, giddiness, nausea, vomiting, slurring of speech, inability to stand or walk. The signs are truncal or limb ataxia, ipsilateral gaze palsy, small reactive pupils. LMN facial palsy and horizontal gaze palsy denotes pontine compression, hemiparesis of variable degrees may be present. Other signs include occular bobbing, skew deviation, decreased corneal reflex, Bradycardia, hypertension. Small (<3cm) cerebellar bleeds may present with isolated vomiting with no other signs.

5. Caudate haemorrhage

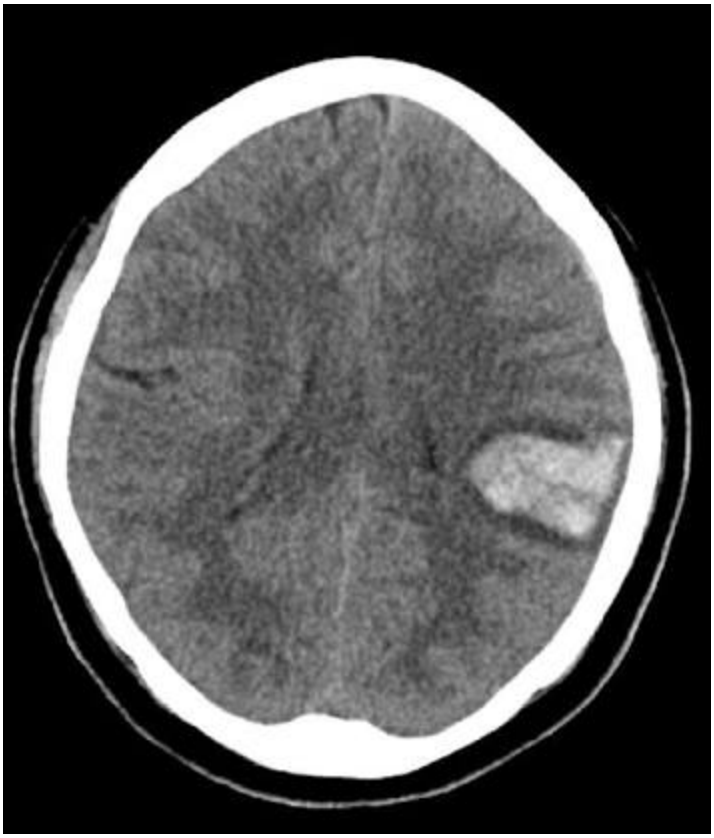
It is least common site of hypertensive ICH. It causes headache, vomiting, neck stiffness, behavioural and memory disturbances. It is due to rupture of artery of Heubner of medial lenticulostriate arteries. Clinically contralateral sensory and motor deficits and gaze palsies may be present. Rarely, ipsilateral horner syndrome may be present, if the bleed extends into the hypothalamus.

6. Lobar haemorrhage

It accounts for 30% of nontraumatic bleed. It usually arise from the gray white matter junction and extend into the underlying white matter involving mainly one lobe.

Frontal lobe bleeds causes headache, vomiting, contralateral motor deficits, abulia, gaze preference towards the site of lesion.

Parietal bleed causes, pain in the temple, hemisensory loss in the opposite side, hemineglect, variable homonymous hemianopia in the opposite side, variable degree of hemiparesis and anosognosia. A partial bleed in the dominant side cause Gerstmann syndrome which encompasses, dyscalculia, finger agnosia, right left disorientation, dysgraphia.



Temporal lobe bleeds in dominant side cause variable types and degrees of aphasia. It also cause, superior quadrantanopia in the contralteral side and rarely agitated delirium.

Oribital lobe bleeds causes sudden homonymous hemianopia in the opposite side.

Lobar bleeds sometimes present as seizures and headache.

7. Midbrain haemorrhage

Patients presents with altered sensorium, headache, vomiting, anisocoria, loss of light reflex. It may cause ‘parinaud’s syndrome’ also called as partial dorsal midbrain syndrome caused due to rostral tectal

bleeds. It can also cause bilateral horners syndrome due to unilateral superior colliculus bleed, and bilateral trochlear nerve palsy.

8. Medullary haemorrhage

It is extremely rare. It may cause headache, vertigo, dysphagia, nystagmus, hypoglossal plasy, ataxia, motor deficits.

9. Internal capsule haemorrhage

Small haemorrhages within the internal capsule may cause pure motor or sensory deficits. It is usually due to extension of bleed form the adjacent structures prone to hypertensive haemorrhages. Very rarely, paraparesis may result from bilateral haemorrhages in posterior limb of internal capsule.

10.Interventricular haemorrhage

It is usually due to the extension of primary parenchymal haemorrhage. Clinical feature are mainly due to the mass effect caused by the obstructive hydrocephalus secondary to the haemorrhage. Feature include nausea, headache, vomiting, altered sensorium, memory deficit, neck stiffness.

INVESTIGATIONS:

CT IMAGING :

This is the prime modality of investigation. The haemorrhage appears as hyperdense lesion in non contrast CT brain. It can be detected in CT even after few seconds of haemorrhage. Supratentorial lesions are detected reliably.

After about 2 weeks, the lesion becomes Isodense with that of the surrounding area,since the X ray attenuation values of the clotted blood diminishes significantly.

However the mass effect and edema may be seen even after 2 weeks

Complications of CT

1. Radiation exposure

   It is between 2-15 MSV

2. Contrast nephropathy (No contrast is need to rule out ICH)

   It is a self limiting condition in which the serum creatinine usually returns to normal with in 1-2 weeks.

MAGNETIC RESONANCE IMAGING:

It helps in picking up posterior fossa lesions

It can also detect AV malformation.

Contraindictations of MRI :

Cardiac pacemakers

Magnetic venacaval filters,

Coils, stunt, prosthesis in cochlea.

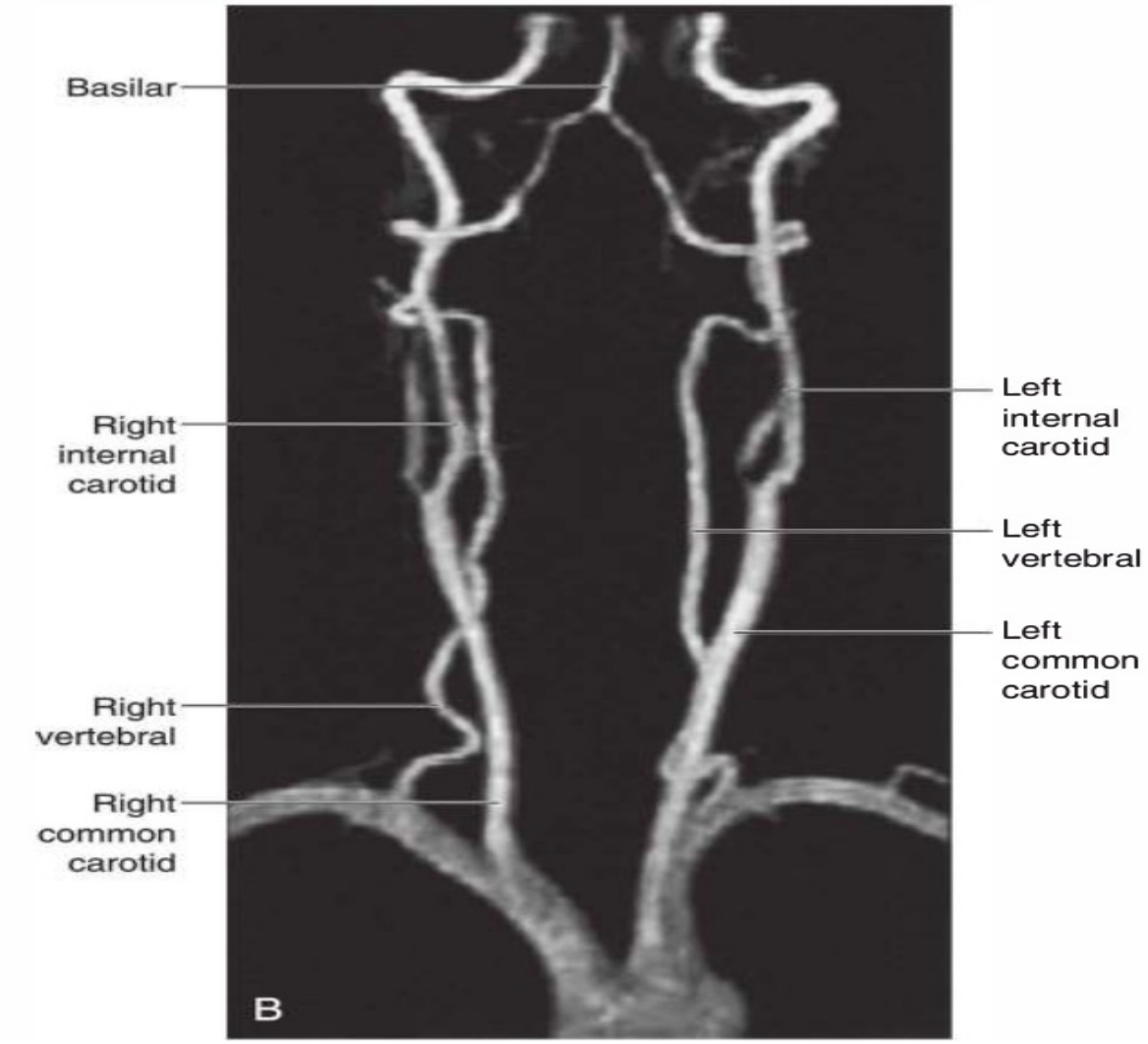
Internal defibrillator devices.

Swanz ganz catheter

Magnetic dental implants

ANGIOGRAPHY:

It is used for evaluation of Small vessel pathology in the brain, aneurysms, assessing vascular malformations.



CT Angiography of ICA

Complications :

Stroke – this is due to thrombus formation in the catheter tip or the catheter may dislodge an atherosclerotic plague.

Risk factors include

- ❖ Reduced cardiac output
- ❖ Advanced age
- ❖ Atherosclerosis

OTHER INVESTIGATIONS

- **MRI brain sequence which is sensitive** for hemosiderin ie,iron sensitive imaging – used for detection of micro bleeds which is helpful in the diagnosis of cerebral amyloid angiopathy.
- The demonstration of amyloid in the vessels of CNS by congo red staining is the definitive diagnosis for cerebral amyloid angiopathy.
- **Toxicology screening** for cocaine and methamphetamine which causes sympathetic hyperactivity there by leading to ICH by increasing the blood pressure
- Routine basic investigations including complete blood hemogram, blood urea, serum creatinine.
- Platelet and coagulation profile gain special attention for patients of suspected coagulopathy.

Treatment of ICH:

1. General supportive care.
2. Arrest / reduce bleeding from the site of bleeding
3. Treatment of raised ICT and reduced cerebral perfusion.
4. Evacuation of blood from ventricles & parenchyma

GENERAL CARE:

1. Secure an iv line.
2. Maintain airway. Breathing and circulation
3. Ryles rube feeding

   Early institution of ryles tube feeding has better prognosis when compared with prolonged iv fluid support. The ryles tube also serves in preventing aspiration pneumonitis which is the most common complication in causing death in the first week of ICH.
4. Continuous bladder drainage: The second most common cause of death in case of ICH is UTI progressing to urosepsis. So rather than catheterising the bladder, condom collection of urine can be done.
5. Eye care: in case of weakness of facial muscle (facial N palsy) there might be lagophthalmos, so appropriate antibiotic eye drops and lubricant eye drops can be given.

6. Hyperthermia / hypothermia is to be managed appropriately to avoid extension of haemarrhage. In case of fever the septic foci is to be identified and then appropriate antibiotic and antipyretics is to be given.
7. Frequent change in posturing of the patients, pneumatic compression can be done to avoid complications like DVT/ PE
8. Physiotherapy.

Treatment of raised ICT:

Raised ICT means ICP> 20 mmHg for more than 55 mins. Patients with altered sensorium along with CVA and who have a history of vomiting with seizure activity before or after onset of haemorrhage are ideal candidates of reducing ICT. It is modest to maintain the cerebral perfusion pressure of >70mmHg to maintain cerebral autoregulation.

i) Head end elevation to 30^0

ii) Iv mannitol – removes fluid from the oedematous area surrounding ICH. It also reduces blood viscosity and promotes vasoconstriction thereby producing a fall in cerebral blood volume. Mannitol can be given not exceeding than 5 days to avoid renal complications. Concurrent diuresis with furosemide is to be given. Maintain serum osmolarity in the range of 300-310 mosm / L with osmotherapy

iii) 3% nacl can be given in cases not responding to mannitol.

iv) Though neuromuscular blockade is not routinely tried, it can be done when all above measures go in vain.

Last resorted method include hyperventilation, barbiturate come and VP shunts etc.

Others:

i) Maintaining blood glucose values <140 mg/dl in the first 24 hours provides good prognosis

ii) Fluids: maintaining euvolemia is appropriate

The pH and bicarbonate levels is to be maintained with frequent ABG analysis

iii) Seizure in an ICH patient can further deteriorate the prognosis by producing neuronal injury. So it is better to give antiepileptic coverage for the patients from Day 1 and then gradually taper it.

Maintenance of BP in a case of ICH:

It is not necessary to reduce blood pressure in all patients in first 24 hours. 15% reduction in Blood Pressure in patient with ICH whose MAP exceeds 130 mmHg is indicated. An appropriate antihypertensive should not produce a sudden fall in BP and it must be cerebroprotective and have shorter t1/2. Vasodilators when given worsen ICH by increasing ICT and increase the blood volume in cerebral blood vessels.

The novel antihypertensive that are used in practice in the decreasing order of frequency are

Labetalol > Ca^{2+} channel blockers (nimodipine)> Hydralazine > ACE inhibitors.

Haemostatic therapy in ICH

Hematoma volume, intraventricular Haemorrhage, altered sensorium are the predictors of poor outcome in case of ICH. Of these, the most important being hematoma volume, to assess the 30^{th} day mortality rate. The haemorrhage can extend till 48 hrs even after the onset of hemiplegia. So repeat CT after 48 hours is essential in case of ICH. So haemostatic therapy can be tried. The agents commonly used are tranexamic acid, aminocaproic acid. Sometimes FFP and r factor VII a complex, prothrombin concentrate complex can be used. All these agents prevent the risk of rebleeding in case of SAH. However, there is no antidote for oral dabigatran. So patients who are on dabigatran presenting with ICH, prothrombin complex concentrate can be tried. In ICH patients with patient <50,000 / mm^3, platelet transfusion can be carried out.

Surgery in ICH:

Indication i) Supratentorial haemorrhage

ii) Hematoma over the vermis

iii) Hematoma size >3cm

iv) altered sensorium with intact brainstem reflex

v) hydrocephalus form brainstem compression

ICH score

Age	**Point**
<80 yrs	0
>80 yrs	1
Hematoma Volume	
<30 cc	0
> 30 cc	1
IVH	
Absent	0
present	1
Infratentorial origin of haemorrhage	
No	0
Yes	1
Glassgow Coma Score	
13-15	0
5-12	1
3-4	2

ICH Score	% mortality @ 30 Day (95%- CI)	% walk in depend @ 12 months
0	0 (0-13)	70 (53-84)
1	13 (4-29)	60 (47-73)
2	26 (11-46)	33(21-48)
3	72 (53- 86)	13 (5-25)
4	97 (82- 100)	3 (0-16)
5	100 (54-100)	8 (0-38)

PREVENTION

- Hypertension is the most important cause for primary ICH.
- Avoid alcoholism and smoking
- Avoiding the use of the illicit drug like cocaine & amphetamine
- Salt restricted diet <2g / Day
- Regular exercise for atleast for 40 mins once in 3 days / week to reduce obesity
- Jogging / Running for half an hour to reduce weight.
- Adequate hydration
- Balanced diet.

MATERIALS AND METHODS

SOURCE OF DATA:

Cases admitted in our hospital with haemorrhagic stroke will be closely monitored from the day of admissions to the day of discharge.

METHOD OF COLLECTION OF DATA:

STUDY TYPE:

Prospective study

AREA OF STUDY:

Medicine IMCU and follow up of cases in medicine ward

SAMPLE SIZE:

All the haemorrhagic stroke cases admitted in TVMCH during the study period of one year (March 1st 2017 to February 31st 2018).

STUDY DESIGN AND SAMPLING:

All the cases of haemorrhagic stroke admitted in our hospital with respect to inclusion and exclusion Criteria.

INCLUSION CRITERIA:

All the patients admitted with haemorrhagic stroke

1. With History of Hypertension on Regular Treatment
2. With History of Hypertension on irregular treatment
3. Patients presented with elevated BP on admission which on further follow- up for 2 weeks found to be a case of Hypertension

EXCLUSION CRITERIA:

- Head trauma
- Haemorrhagic transformation of infarct
- Subdural or epidural haemotoma
- Primary intraventricular haemorrhage
- Intracranial neoplasm
- Cerebral Amyloid Angiopathy
- AV malformation or aneurysm
- Patients on anticoagulation therapy
- Known haemophiliacs
- Known cases of chronic liver disease, connective tissue disorders
- Patients who presented with elevated BP on admission and found to be Normotensive on further follow – up.

METHODLOGY:

This study was carried out among 100 acute haemorrhagic stroke patients (clinically and radiologically confirmed) with respect to the inclusion and exclusion criteria, who were admitted in IMCU of TVMCH. These patients has been subjected to plain CT scan brain on the day of admission itself for radiological confirmation and to localize the lesion in brain. Then patient was taken a detailed history and critically assessed clinically along with special emphasis on calculation of GCS and fundus

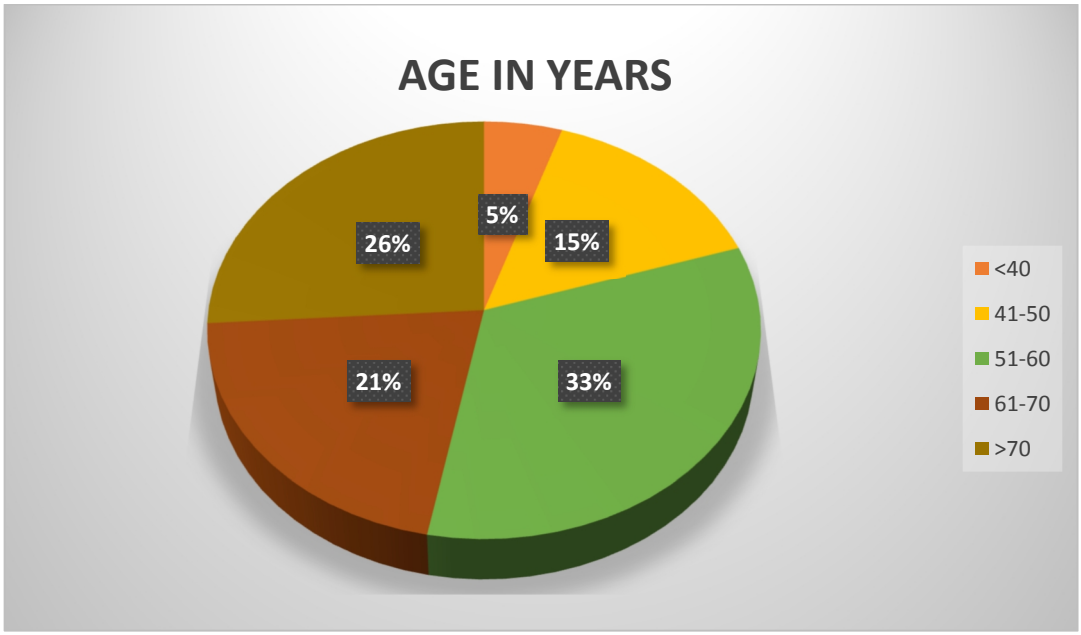
examination for the presence of retinopathy. Then routine basic investigations were taken such as urea, creatinine, urine routine, ECG.

RESULTS

TABLE NO 1: AGE DISTRIBUTION

AGE IN YEARS	NO OF PATIENTS	PERCENTAGE
<40	5	5%
41-50	15	15%
51-60	33	33%
61-70	21	21%
>70	26	26%

FIG.NO 1: AGE DISTRIBUTION

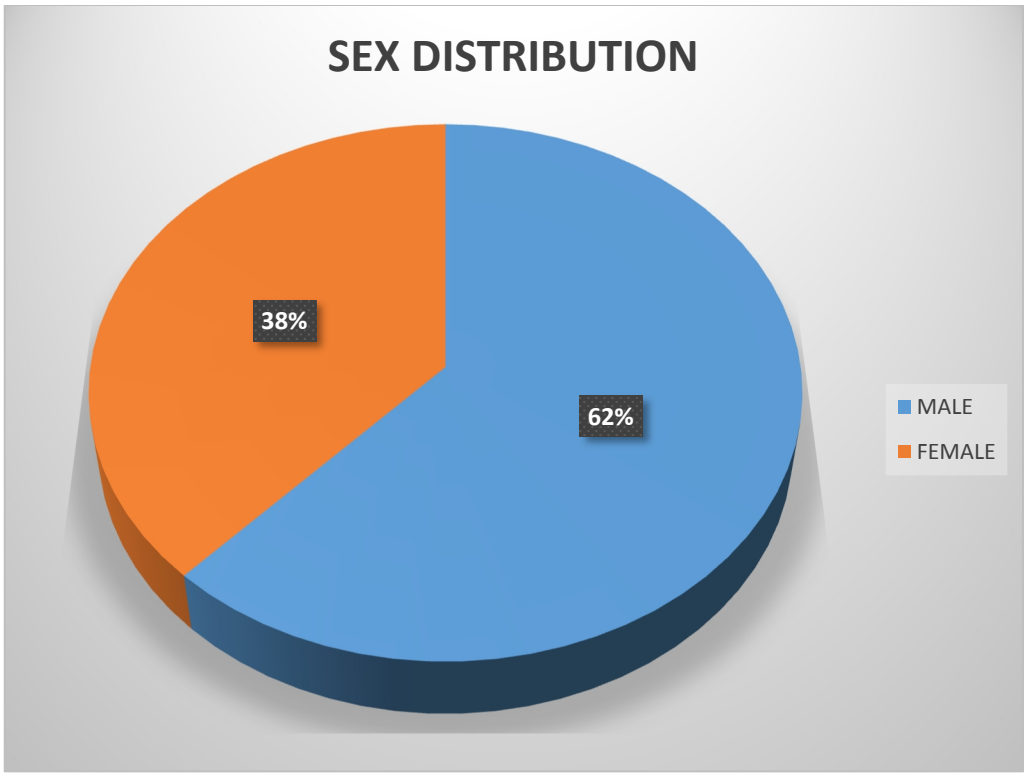


In the study conducted among 100 ICH patients 5 patients were below 40 years of age, 15 patients were between 41 to 50 years of age , 33 patients were between 51 to 60 years of age, 21 patients were between 61 to 70 years of age and 26 patients were more than 70 years of age.

TABLE NO 2: SEX DISTRIBUTION

SEX	NO OF PATIENTS	PERCENTAGE
MALE	62	62%
FEMALE	38	38%

FIG.NO 2: SEX DISTRIBUTION

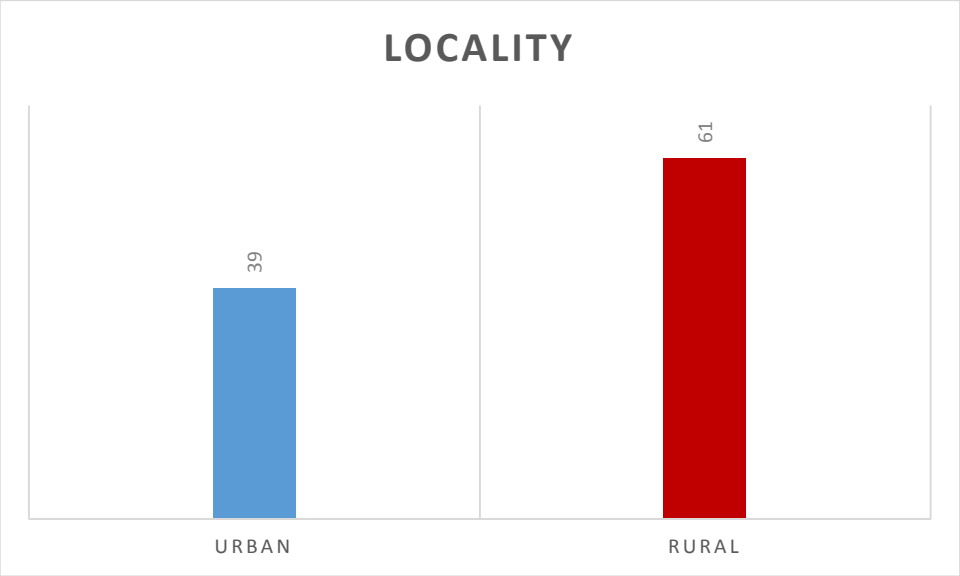


Among the 100 patients studied 62 patients were male and 38 patients were female.

TABLE NO 3: LOCALITY

LOCALITY	NO OF PATIENTS	PERCENTAGE
URBAN	39	39%
RURAL	61	61%

FIG.NO 3: LOCALITY

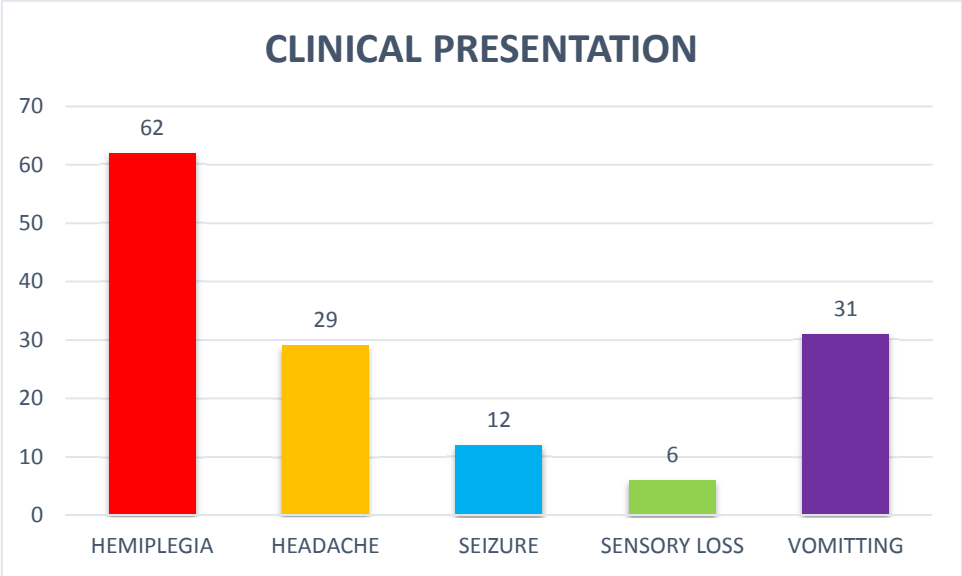


In this study, among the 100 patients studied 39 patients were from urban population and 61 persons were from rural population.

TABLE NO 4: CLINICAL PRESENTATION

CLINICAL PRESENTATION	PRESENT	PERCENTAGE
HEMIPLEGIA	62	62%
HEADACHE	29	29%
SEIZURE	12	12%
SENSORY LOSS	6	6%
VOMITTING	31	31%

FIG NO 4: CLINICAL PRESENTATION

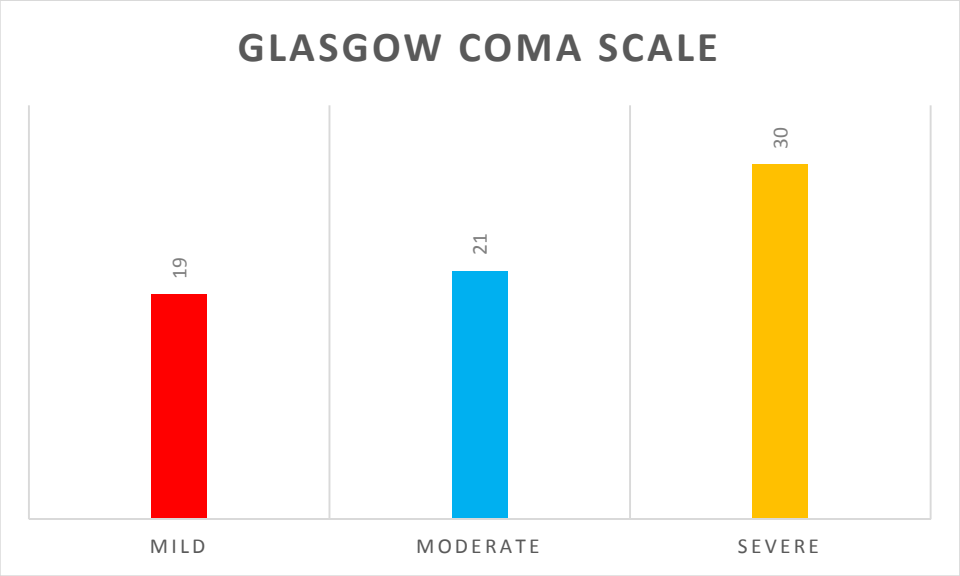


In 100 patients studied in our hospital, 62 patients had hemiplegia, 29 patients had headache, 12 patients had seizures, 5 patients had sensory loss and 31 patients had vomiting initially at the time of presentation.

TABLE NO 5: GLASGOW COMA SCALE

GCS	NO OF PATIENTS	PERCENTAGE
12-15 (Mild)	19	19%
9-11(Moderate)	21	21%
< or = 8 (Severe)	60	60%

FIG.NO 5: GLASGOW COMA SCALE



Among the 100 patients studied 19 patients had GCS more than 12,21 patients had GCS between 8 and 12 , 30 patients had GCS less than or equal to 8.

TABLE NO 6: SYSTOLIC BP ON ADMISSION

SYSTOLIC BP	NO OF PATIENTS	PERCENTAGE
>180	91	9%
<180	9	9%

FIG.NO 6: SYSTOLIC BP ON ADMISSION

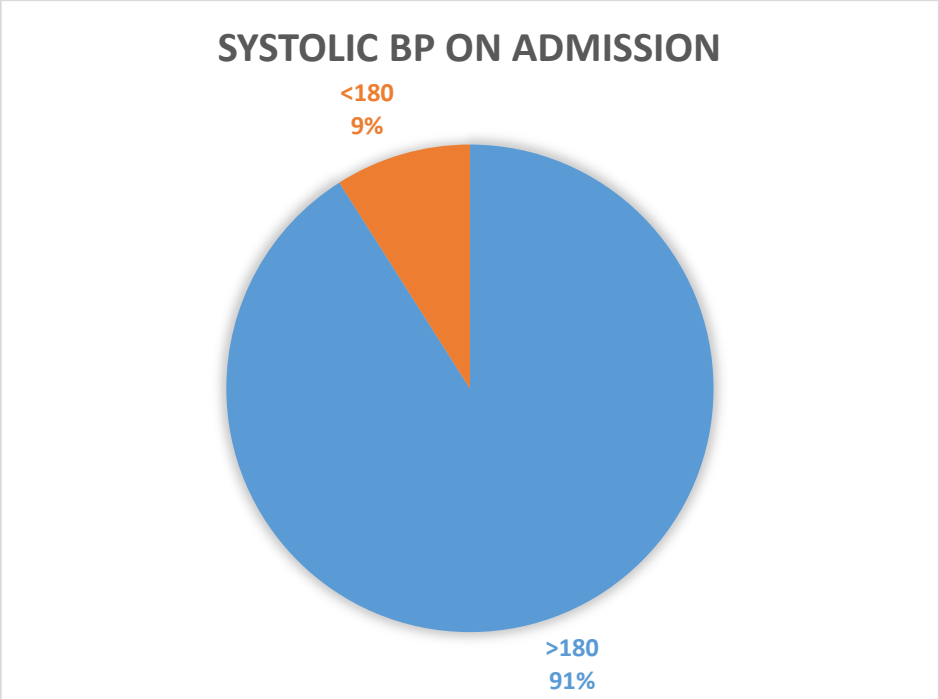
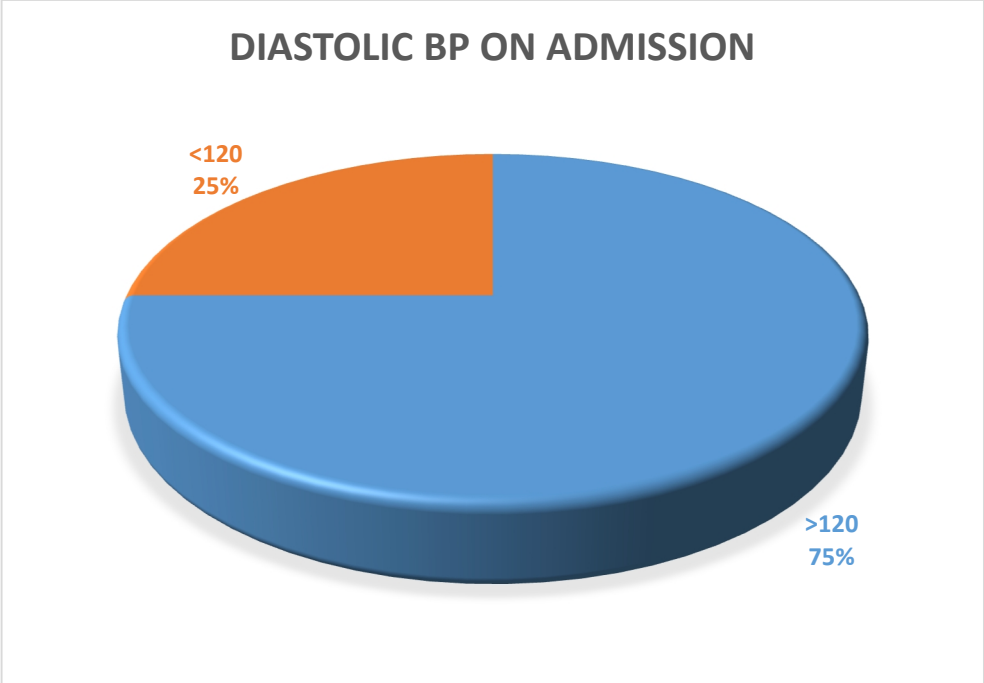


TABLE NO 7: DIASTOLIC BP ON ADMISSION

DIASTOLIC BP	NO OF PATIENTS	PERCENTAGE
>120	75	75%
<120	25	25%

FIG.NO 7: DIASTOLIC BP ON ADMISSION

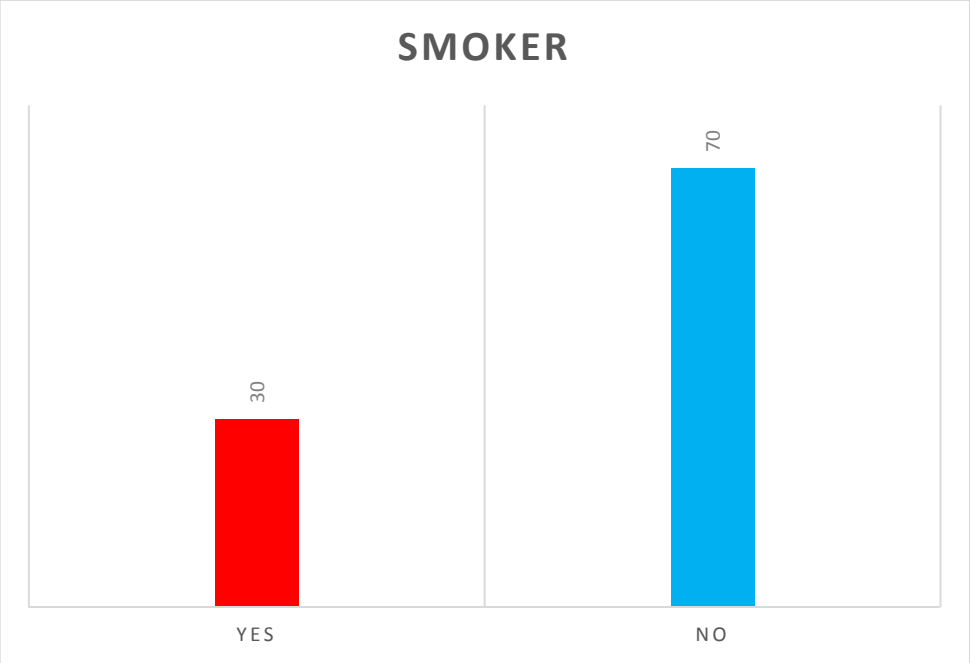


In this study of 100 patients of ICH 91 patients had systolic BP of more than 180 and 75 patients had diastolic BP of more than 120 at the time of presentation.

TABLE NO 8: SMOKER

SMOKER	NO OF PATIENTS	PERCENTAGE
YES	30	30%
NO	70	70%

FIG.NO 8: SMOKER

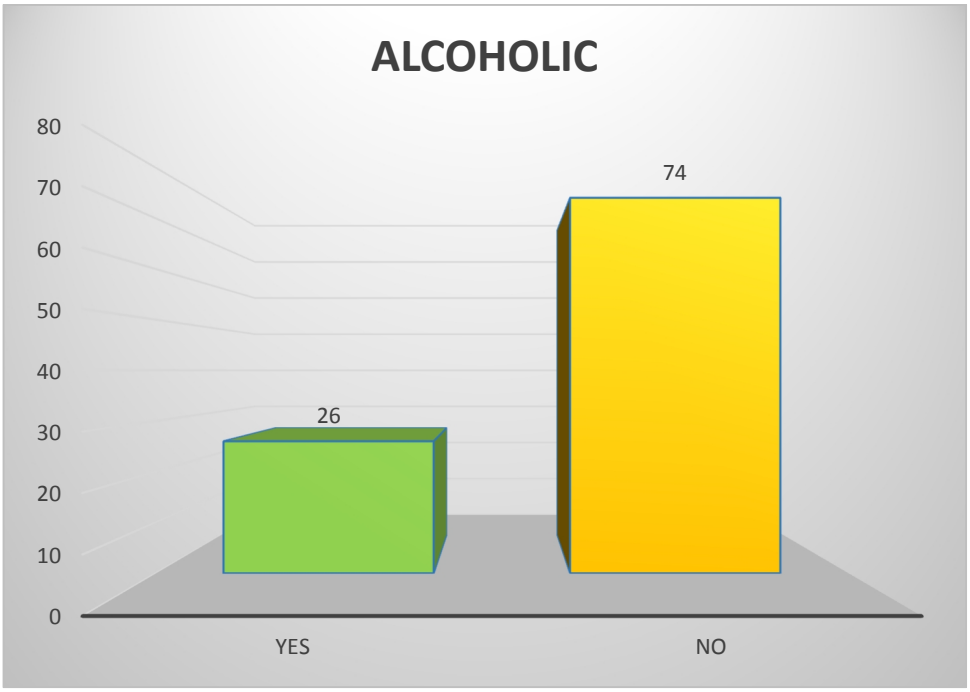


Among the 100 patients studied 30 persons were smoker and 70 persons were non-smokers.

TABLE NO 9: ALCOHOLIC

ALCOHOLIC	NO OF PATIENTS	PERCENTAGE
YES	26	26%
NO	74	74%

FIG.NO 9: ALCOHOLIC

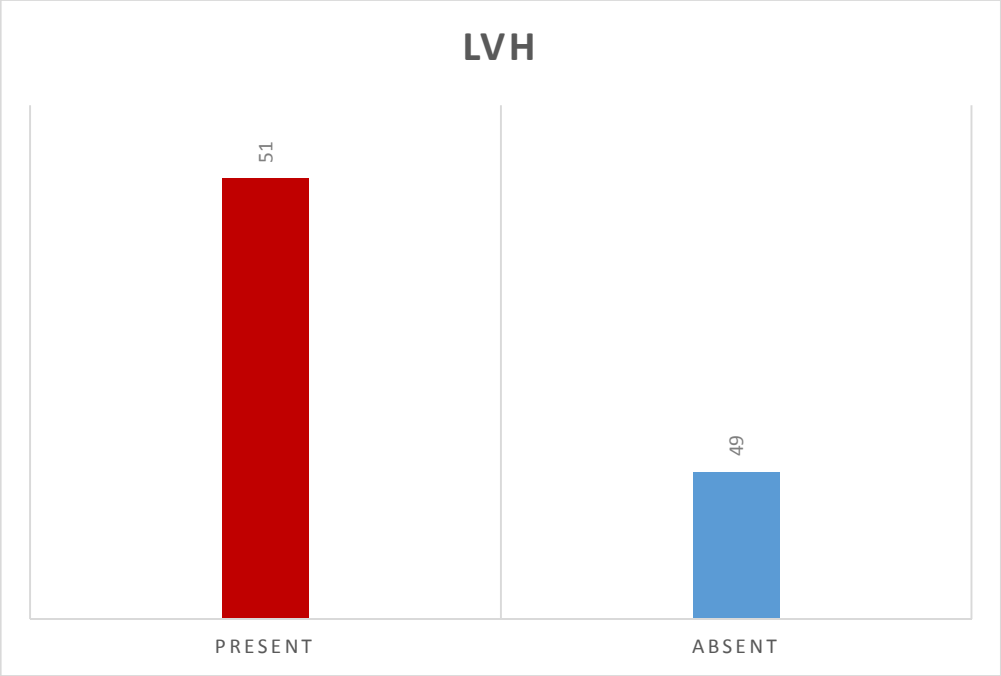


In this study of 100 patients 26 persons were alcoholics,74 patients were non alcoholics.

TABLE NO 10: LVH

LVH	NO OF PATIENTS	PERCENTAGE
PRESENT	51	51%
ABSENT	49	49%

FIG NO 10: LVH

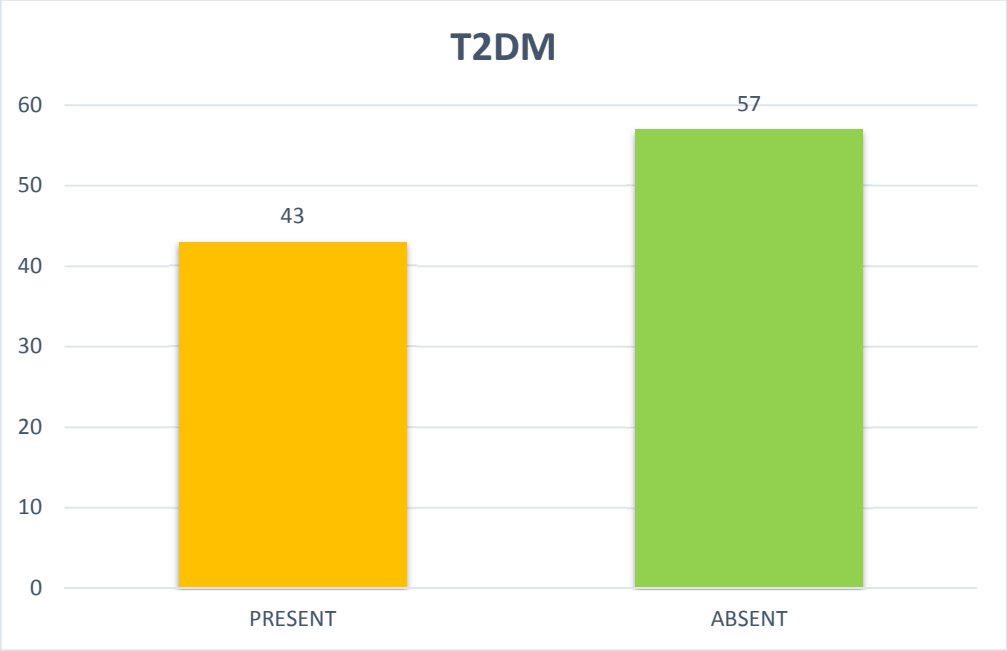


In this study of 100 patients, 51 people had left ventricular hypertrophy as evidenced by ECG at the time of presentation.

TABLE NO 11: T2DM

T2DM	NO OF PATIENTS	PERCENTAGE
PRESENT	43	43%
ABSENT	57	57%

FIG.NO 11: T2DM

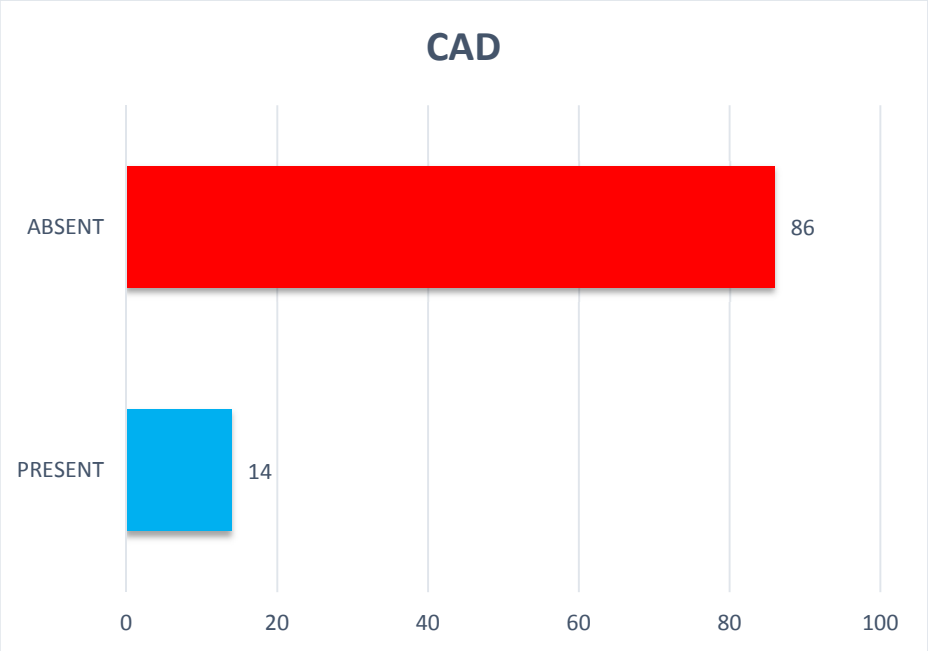


Among the 100 patients studied 43 patients were type 2 diabetic and 57 patients were non diabetic.

TABLE NO 12: CAD

CAD	NO OF PATIENTS	PERCENTAGE
PRESENT	14	14%
ABSENT	86	86%

FIG.NO 12: CAD



In this study of 100 patients 14 patients had coronary artery disease.

TABLE NO 13: CHRONIC KIDNEY DISEASE

CHRONIC KIDNEY DISEASE	NO OF PATIENTS	PERCENTAGE
PRESENT	34	34%
ABSENT	66	66%

FIG.NO 13: CHRONIC KIDNEY DISEASE

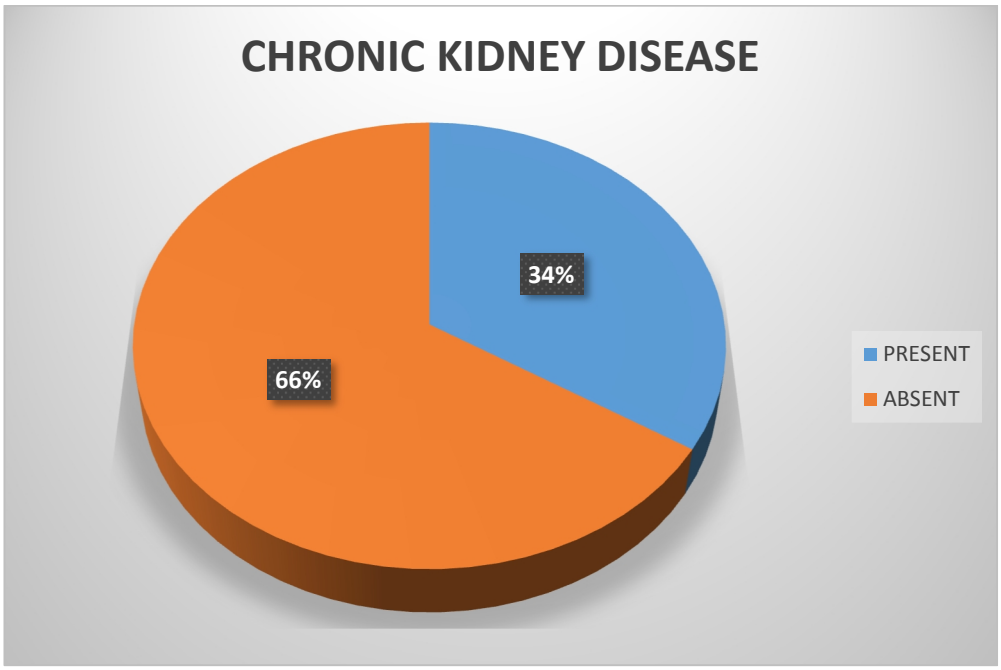
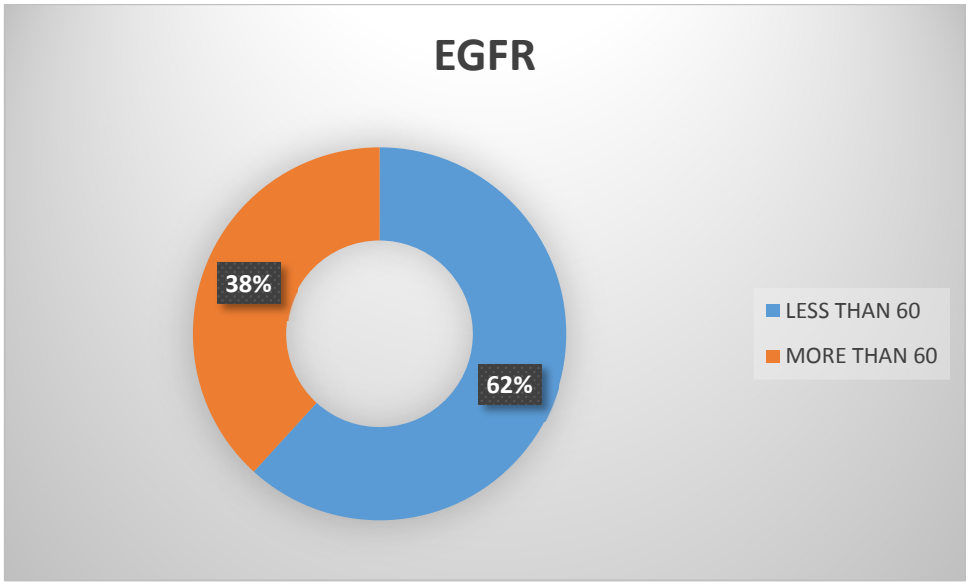


TABLE NO 14: EGFR

EGFR	NO OF PATIENTS	PERCENTAGE
LESS THAN 60	21	62%
MORE THAN 60	13	38%

FIG.NO 14: EGFR

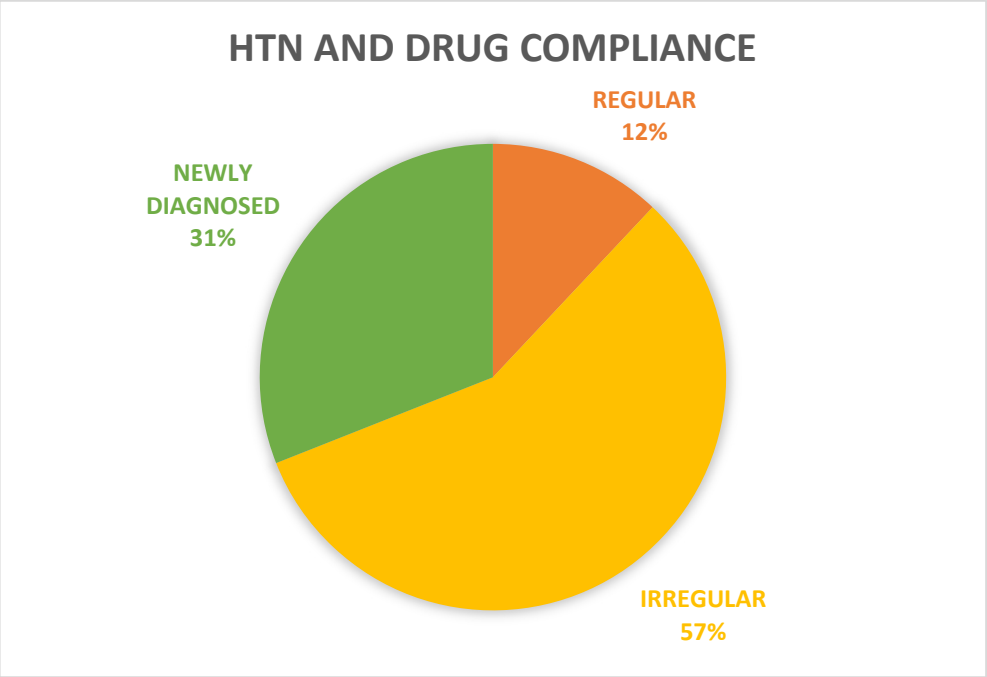


Among the 100 patients studied 34 patients had chronic kidney disease on which 62 patients had egfr less than 60 and 38 patients had egfr more than 60.

TABLE NO 15: HTN AND DRUG COMPLIANCE

HT MANAGEMENT	NO OF PATIENTS	PERCENTAGE
REGULAR	12	12%
IRREGULAR	57	57%
NEWLY DIAGNOSED	31	31%

FIG.NO 15: HTN AND DRUG COMPLIANCE



Among the 100 patients studied 31% were newly diagnosed hypertension, 57% were known hypertensives on irregular treatment, 12% were known hypertensives on regular treatment.

TABLE NO 16: HYPERTENSIVE RETINOPATHY

HYPERTENSIVE RETINOPATHY	NO OF PATIENTS	PERCENTAGE
PRESENT	33	33%
ABSENT	67	67%

FIG.NO 16: HYPERTENSIVE RETINOPATHY

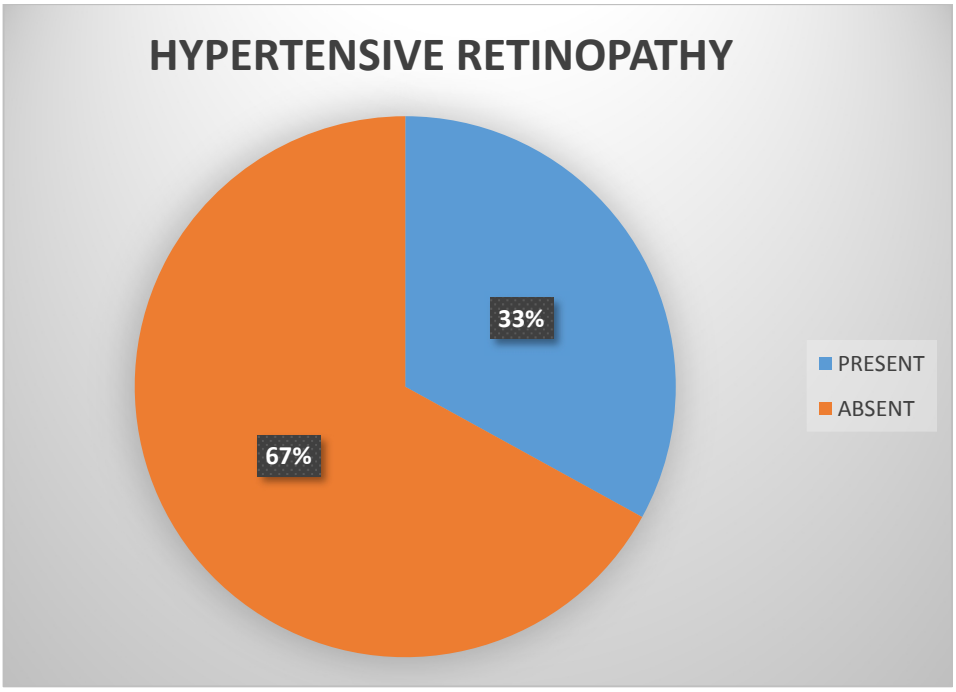
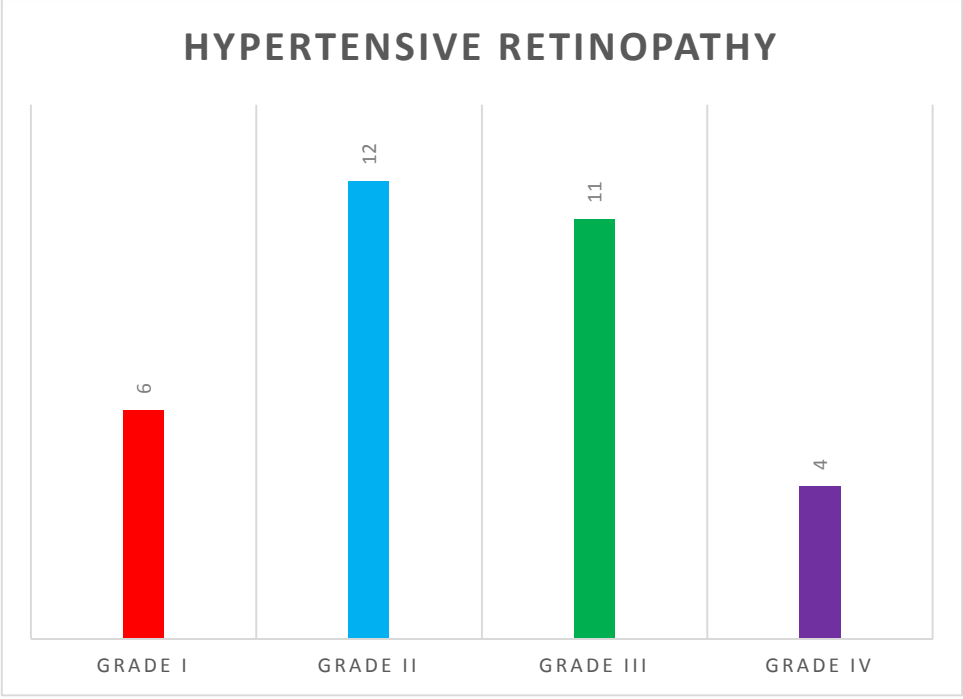


TABLE NO 17: HYPERTENSIVE RETINOPATHY

HYPERTENSIVE RETINOPATHY	NO OF PATIENTS	PERCENTAGE
GRADE I	6	18%
GRADE II	12	37%
GRADE III	11	33%
GRADE IV	4	12%

FIG NO 17: HYPERTENSIVE RETINOPATHY



In this prospective study of 100 patients 67 patients had hypertensive retinopathy at the time of presentation. Among those 67 patients, 6 persons had GRADE 1, 37 persons had GRADE II, 11 persons had GRADE III and 4 persons had GRADE IV hypertensive retinopathy.

TABLE NO 18: PROTEINURIA

PROTEINURIA	NO OF PATIENTS	PERCENTAGE
PRESENT	42	42%
ABSENT	58	58%

FIG NO 18: PROTEINURIA

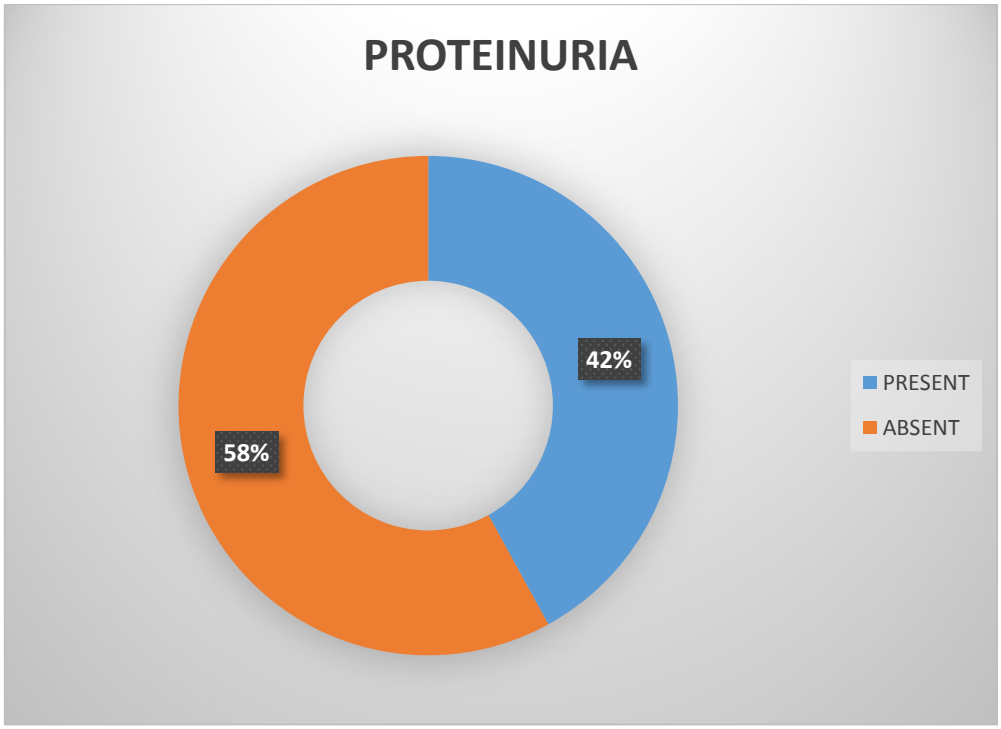
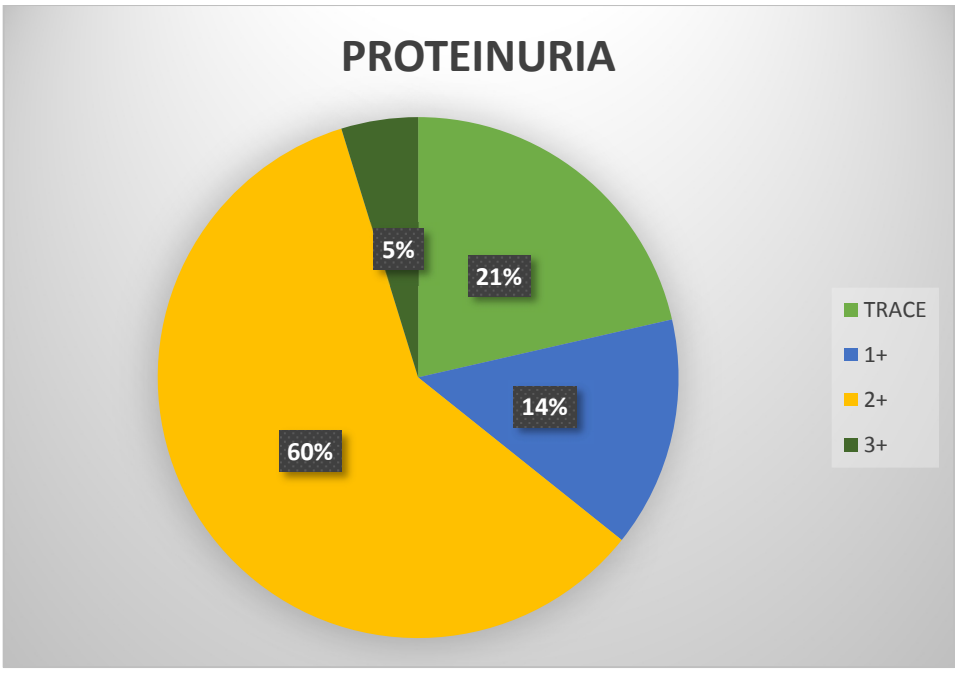


TABLE NO 19: PROTEINURIA

PROTEINURIA	NO OF PATIENTS	PERCENTAGE
TRACE	9	13%
1+	6	16%
2+	25	60%
3+	2	5%

FIG NO 19: PROTEINURIA

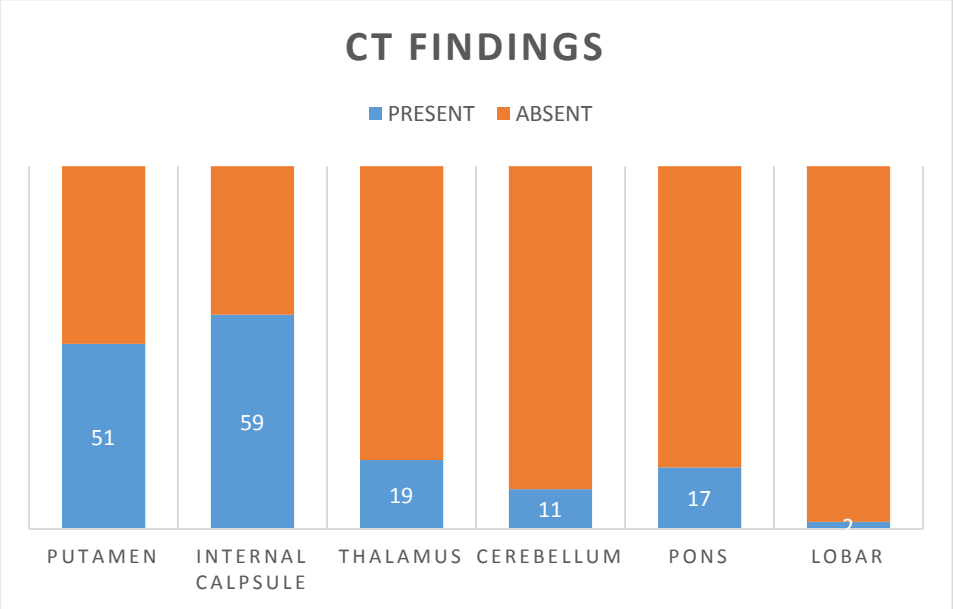


Among the 100 patients studied 42 patients had proteinuria at the time of presentation.Among the 42patients, 9 patients had trace proteinuria ,6 patientss had 1+ proteinuria ,25 patients 2+ proteinuria and 2 patients had 3+ proteinuria at the time of presentation.

TABLE NO 20: CT FINDINGS

SITE OF BLEED	NUMBER OF PATIENTS
PUTAMEN	51
INTERNAL CAPSULE	59
THALAMUS	19
CEREBELLUM	11
PONS	17
LOBAR	2

FIG NO 20: CT FINDINGS

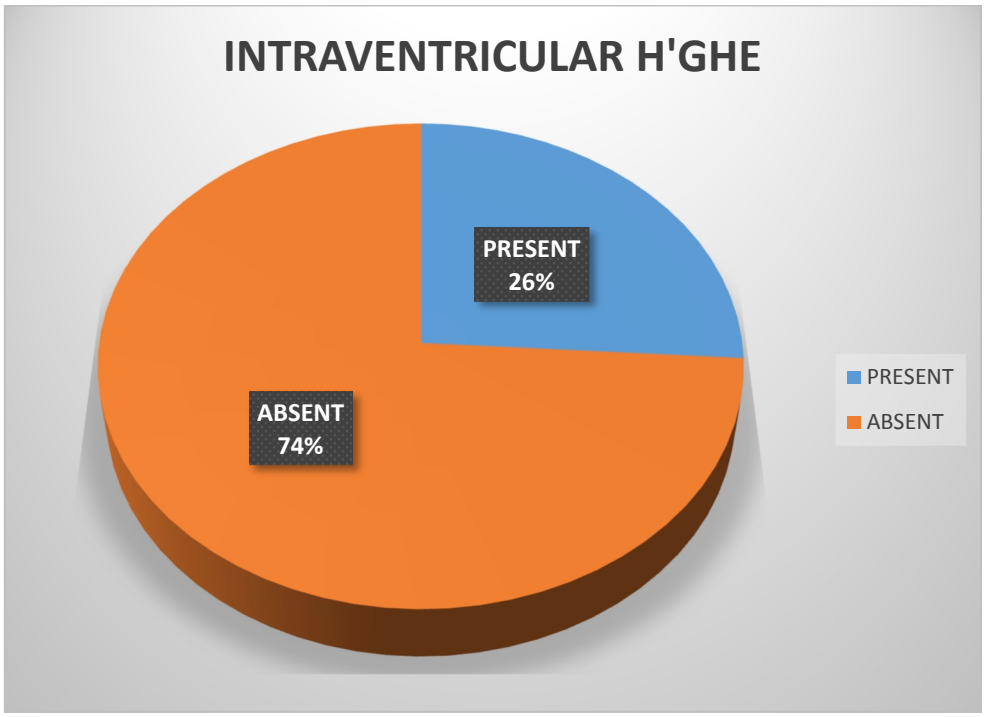


In this study of 100 patients in our hospital, 51 patients had bleed in Putamen, 19 patients had bleed in thalamus, 11 patients had bleeed in cerebellum,17 patients had bleed in pons and 2 patients had lobar bleed, 59 patients had bleed in internal capsule and associated area.

TABLE NO 21: INTRAVENTRICULAR H'GHE

CT FINDINGS -IVH	NO OF PATIENTS	PERCENTAGE
PRESENT	26	26%
ABSENT	74	74%

FIG NO 21: INTRAVENTRICULAR H'GHE

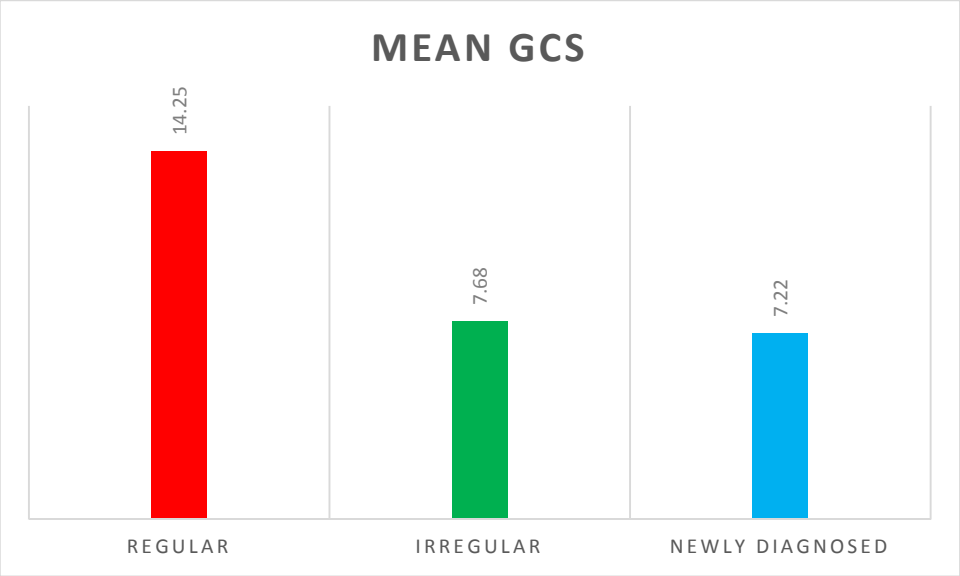


In the 100 patients studied, 26 patients had intraventricular haemorrhage as a part of extension from primary parenchymal bleed.

TABLE NO 22: MEAN GCS

HT MANAGEMENT	GLASGOW COMA SCALE	
	MEAN	SD
REGULAR	14.25	1.42
IRREGULAR	7.68	3.88
NEWLY DIAGNOSED	7.22	3.28
P VALUE - 0.001		
SIGNIFICANT		
ANOVA		

FIG NO 22: MEAN GCS

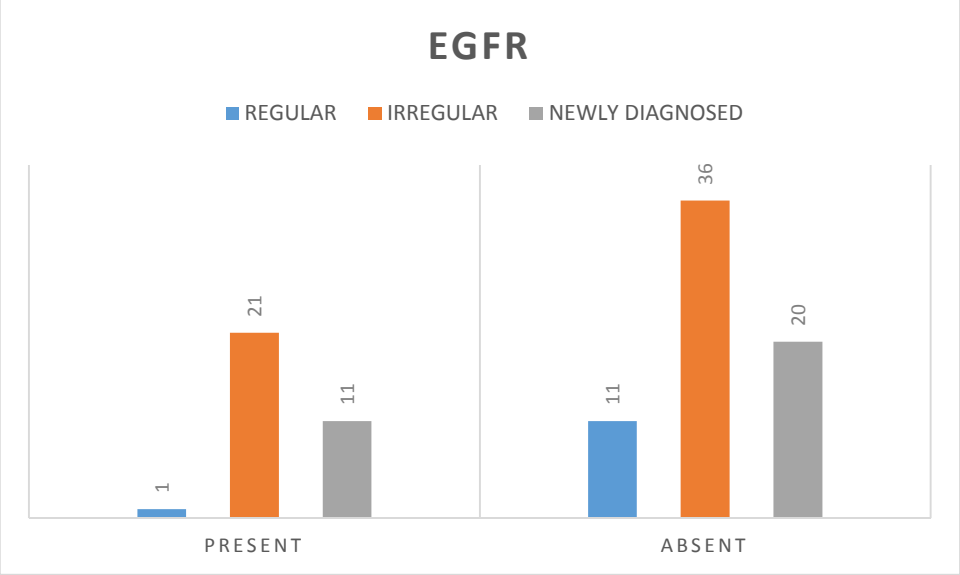


On statistical analysis of the study of 100 patients there was a statically significant correlation between the hypertension treatment and the GCS at the time of presentation. Those who were on regular treatment had a better GCS at presentation than those were on irregular treatment.

TABLE NO 23: EGFR

HT MANAGEMENT	CHRONIC KIDNEY DISEASE	
	PRESENT	ABSENT
REGULAR	1	11
IRREGULAR	21	36
NEWLY DIAGNOSED	11	20
P VALUE - 0.152		
NON SIGNIFICANT		
KRUSKAL WALLIS TEST		

FIG. NO 23: EGFR

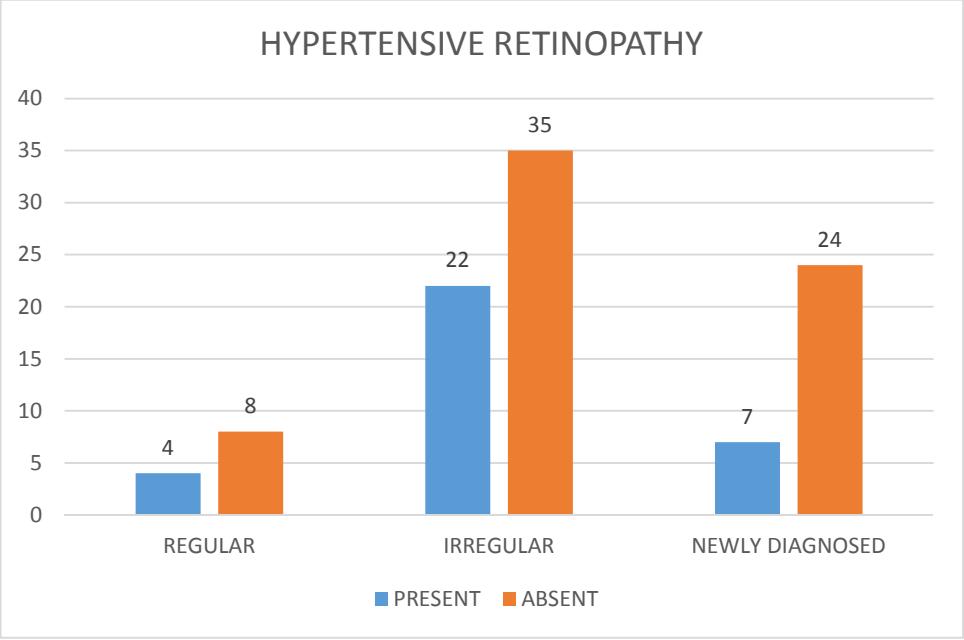


In the 100 patients studied, there was no statistical significance between hypertension management and occurance of CKD in those patients.

TABLE NO 24: HYPERTENSIVE RETINOPATHY

HT MANAGEMENT	HYPERTENSIVE RETINOPATHY	
	PRESENT	ABSENT
REGULAR	4	8
IRREGULAR	22	35
NEWLY DIAGNOSED	7	24
P VALUE - 0.031		
SIGNIFICANT		
KRUSKAL WALLIS TEST		

FIG. NO 24: HYPERTENSIVE RETINOPATHY

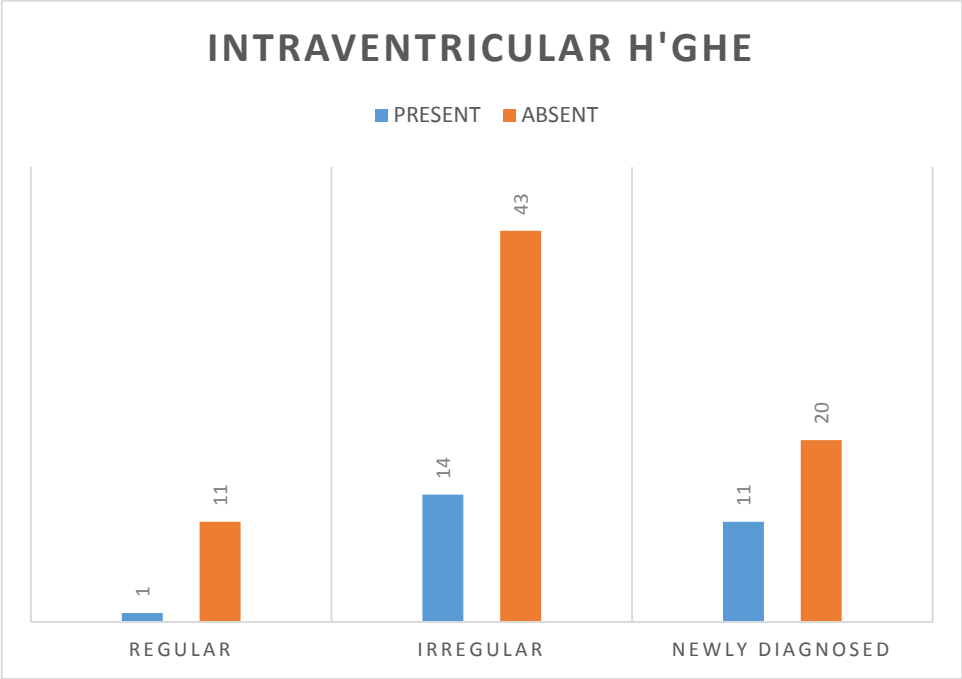


There was strong statistical correlation between irregulary treated hypertension patients and presence of hypertensive retinopathy

TABLE NO 25: INTRAVENTRICULAR H'GHE

HT MANAGEMENT	INTRAVENTRICULAR HGHE	
	PRESENT	ABSENT
REGULAR	1	11
IRREGULAR	14	43
NEWLY DIAGNOSED	11	20
P VALUE - 0.017		
SIGNIFICANT		
KRUSKAL WALLIS TEST		

FIG. NO 25: INTRAVENTRICULAR H'GHE

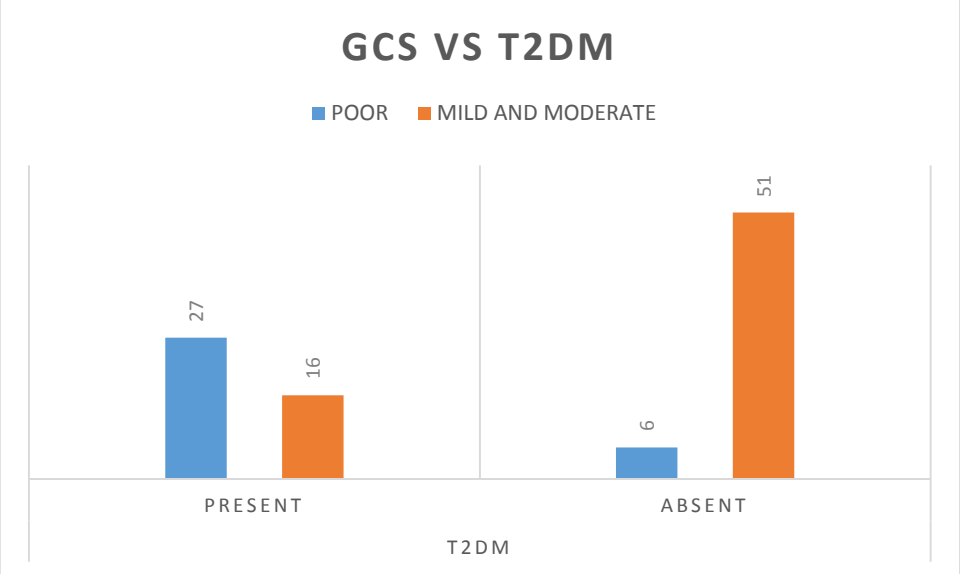


There was a strong correlation proved statistically between irregularly treated hypertensive patients and presence of intraventricular haemorrhage.

TABLE NO 26: GCS VS T2DM

GCS	T2DM	
	PRESENT	ABSENT
POOR	27	6
MILD AND MODERATE	16	51
P VALUE - 0.001		
SIGNIFICANT		
CHI SQUARE TEST		

FIG. NO 26: GCS VS T2DM

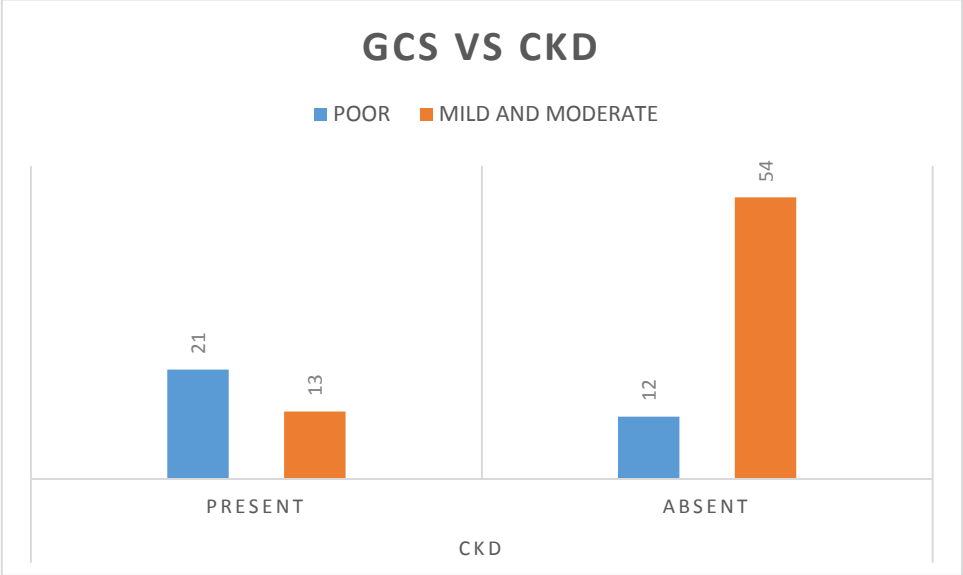


There was significant correlation proved between presence of T2DM and presence of poor GCS in the ICH patients studied.

TABLE NO 27: GCS VS CKD

GCS	CKD	
	PRESENT	ABSENT
POOR	21	12
MILD AND MODERATE	13	54
P VALUE - 0.001		
SIGNIFICANT		
CHI SQUARE TEST		

FIG. NO 27: GCS VS CKD

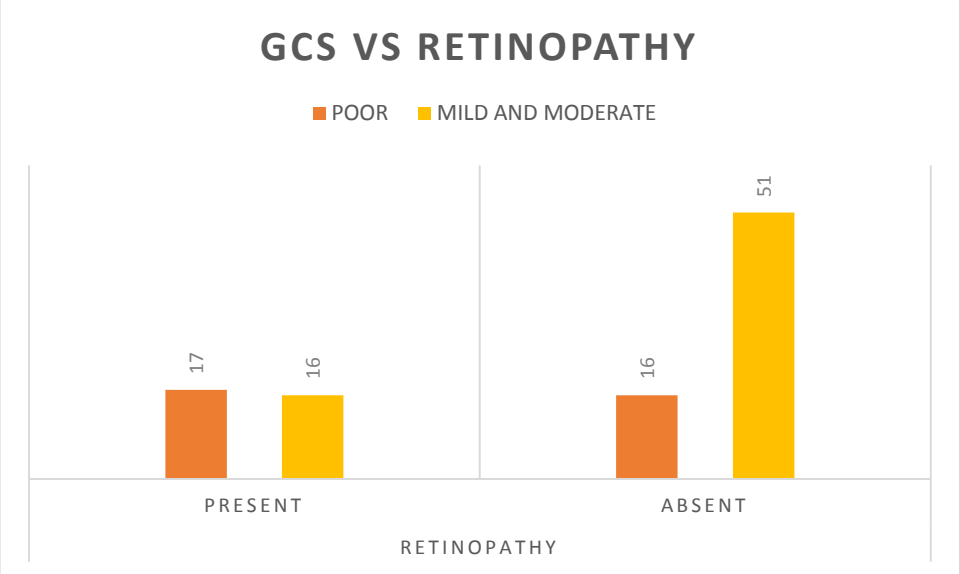


A very strong correlation was statistically found between the presence of CKD and poor GCS at the time of presentation.

TABLE NO 28: GCS VS RETINOPATHY

GCS	RETINOPATHY	
	PRESENT	ABSENT
POOR	17	16
MILD AND MODERATE	16	51
P VALUE - 0.213		
NON SIGNIFICANT		
CHI SQUARE TEST		

FIG.NO 28: GCS VS RETINOPATHY

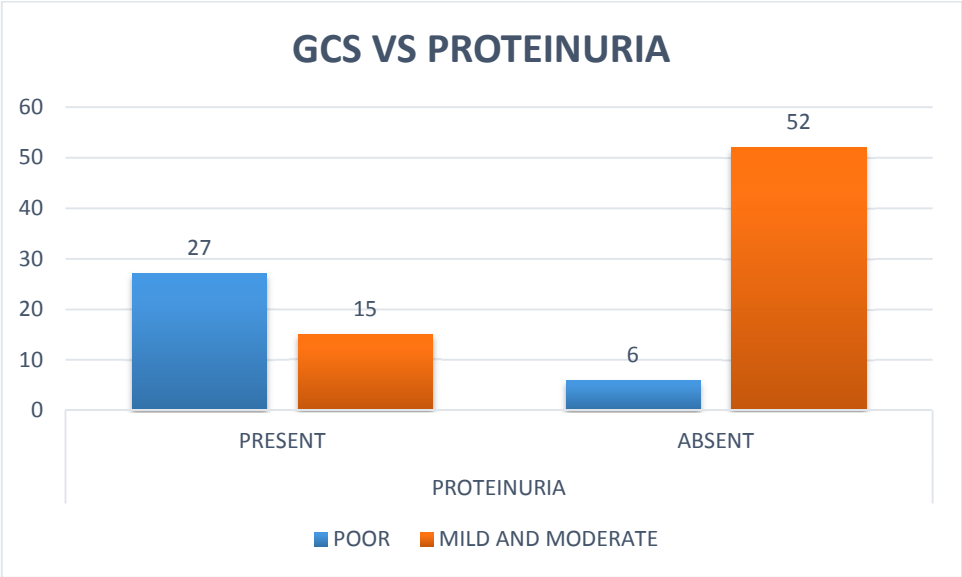


No correlation was noted between patients with presence of retinopathy and poor GCS in this study.

TABLE NO 29: GCS VS PROTEINURIA

GCS	PROTEINURIA	
	PRESENT	ABSENT
POOR	27	6
MILD AND MODERATE	15	52
P VALUE - 0.001		
SIGNIFICANT		
CHI SQUARE TEST		

FIG NO 29: GCS VS PROTEINURIA

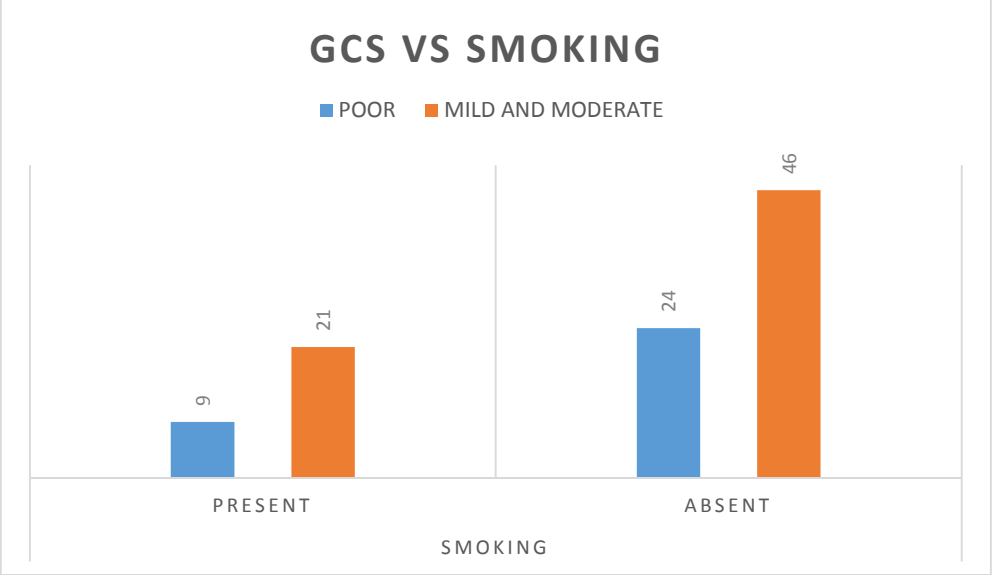


There was significant correlation between poor GCS and the presence of proteinuria.

TABLE NO 30: GCS VS SMOKING

GCS	SMOKING	
	PRESENT	**ABSENT**
POOR	9	24
MILD AND MODERATE	21	46
P VALUE - 0.676		
NON SIGNIFICANT		
CHI SQUARE TEST		

FIG NO 30: GCS VS SMOKING

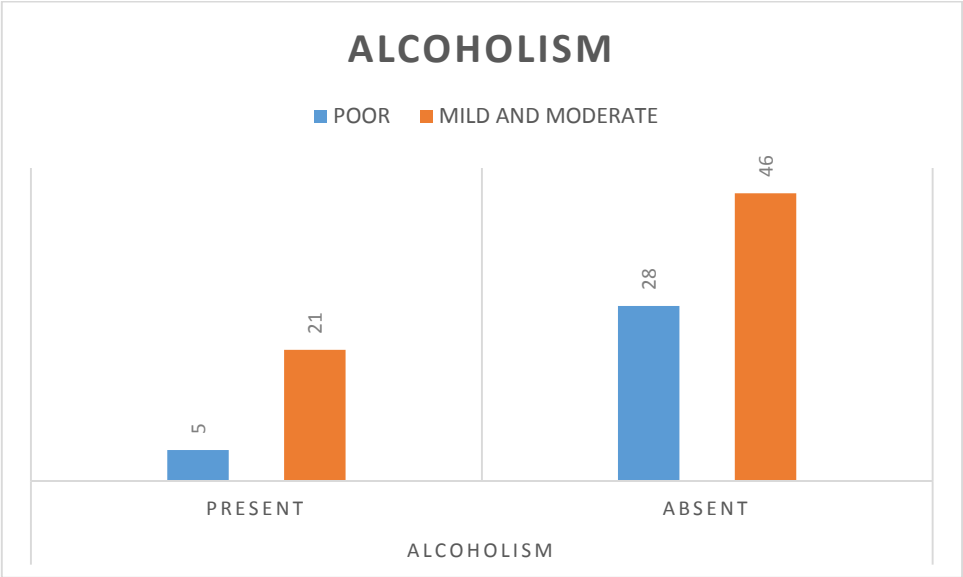


No correlation was found between smoking history and presence of poor GCS at the time of presentation,.

TABLE NO 31: ALCOHOLISM

GCS	ALCOHOLISM	
	PRESENT	ABSENT
POOR	5	28
MILD AND MODERATE	21	46
P VALUE - 0.083		
NON SIGNIFICANT		
CHI SQUARE TEST		

FIG. NO 31: ALCOHOLISM



No correlation was found between alcoholic history and presence of poor GCS at the time of presentation,

DISCUSSION

Haemorrhagic strokes are common and constitutes about 20-30% of all strokes in India and in Asian population Prognosis of patients admitted with Haemorrhagic stroke are usually poor when compared to Ischaemic strokes. Hypertension is the commonest risk factor and constitutes about 80% of Non-traumatic ICH either alone or in association with other causative factors. Increased Blood pressure during admission and inadequate BP control after admission has been associated with Haematoma expansion and Increased mass effect, leading on to deterioration of clinical status of the patient. Therefore, rapid reduction of BP is recommended if the patient is presenting with Systolic BP >160mmHg by using IV Labetalol or Nicardipine. Both of this drugs are equally effective. BP should be maintained at <130/80 mmHg in ICH survivors to prevent the risk of recurrent ICH.

In this study, 100 patients of Non-traumatic ICH were taken after excluding others with the Exclusion criteria. Among 100 patients presented with ICH, 33% of patients were in the age group of 51-60 years, 26% of patients were >70 years of age, only 5% patients were in <40 years of age, that too most of the patients less than 40 years are having Chronic kidney disease. In 2013, a study conducted by Nileshkumar et al. shows that age group of 55-64 years has the highest incidence of ICH. This shows that the

prevalence of ICH due to Hypertension is more common with 50-60 years of age group. Age has been found to be an Independent predictors in studies of Hemphill et al., While it has been less predictor in studies conducted by Juvela et al., Juarez et al., Lisk DR et al. and Qureshi et al.

The sex preponderance for male continues to be higher when compared to female population as already proved by many studies. In this study 62% of ICH were present in Males, 38% in Females among 100 study population. In this study, rural population having more preponderance than Urban population due to lack of awareness of Non-Communicable disease prevention. In our study, 68% of ICH patients from the Rural areas and 39% from the Urban.

In this study, Among 100 study population, the presenting clinical feature of 62% of patients were Hemiplegia, 29% were having headache, 12% of patients presented with seizures, 6% with sensory loss and 31% with vomiting. These presenting features are either Isolated feature or associated with other clinical features. In this study, 60% of patients presented with Glassgow coma score (GCS) less than or equal to 8, 21% are having GCS 9-12 and 19% patients have GCS of 13-15 at the time of admission. In 2012, a small clinical study conducted by Adria Arboix et al. Concluded that 66.7% of patients presented with Motor deficit , 16.7%

with Nausea and vomiting. Our study also reveals that Motor deficit i.e, Hemiplegia is the most common presentation in ICH irrespective of GCS.

In this study, 31% of patients were not a known case of Hypertension. In this patients, ICH may be the first clinical manifestation. These patients are found to have long standing undiagnosed Hypertension. Patients with history of hypertension and are not on Irregular treatment were more commonly end up in Hypertensive crisis (such as Haemorrhagic stoke). In this study, among 100 patients, 69 patients were known hypertensives, in that 57 patients were not on regular treatment, 12 patients were on regular treatment. This reveals that ICH is more common in patients with poor BP control i.e, irregular treatment. This can be supported by a study conducted by Daniel Woo, Mary Harenbusch (stroke 2004; 35:1703-1708) concludes that 'Untreated hypertension is highly prevalent and important risk factor for Haemorrhagic strokes.

In this study, with the sample of 100 patients, 51 presented with putamen bleed, 59 patients presented with Internal capsule bleed, 19 patients presented with Thalamic bleed, 11 patients presented with Cerebellar haemorrhage, 17 patients with pontine bleed and only 2 patients with lobar haemorrhage. 59 patients presented with Intraventricular extension of bleed. Intraventricular extension is more commonly associated with Capsuloganglionic, Thalamic bleed, pontine Haemorrhage.

These findings were conformed by taking Noncontrast CT Brain at the time of admission. A study conducted by Virendra C.patil et al. in 2015 published that 79.31% of ICH is more common in Basal ganglia, 10.34% in thalamus, 3.44% in pons. Our study reveals that Putamen and Internal capsule is the most common site of ICH.

A study conducted by David Tanne et al. (Cerebrovasc Dis 2011; 31:271-277) also concluded that moderate / severe CKD patients presenting with ICH have poor Prognosis. In this study, there is a statistical significant correlation between poor GCS (less than or equal to 8) and the presence of T2DM, chronic kidney disease and proteinuria which is evidenced by the P value.

SUMMARY

1) ICH is more common among people above 50 years of age
2) Most of the patients had poor GCS at the time of presentation
3) Most of the patients with ICH had irregularly treated hypertension
4) Males were affected more than females
5) Rural population was affected more than urban population
6) Most of the patients had systolic BP > 180 and diastolic >120 at the time of presentation
7) 51% of patients had LVH at the time of presentation
8) Most common site of bleed is Putamen and Internal capsule
9) Most common presentation is hemiplegia
10) There was a significant correlation between irregularly treated hypertension and poor GCS at the time of presentation
11) Statistical significance was found between presence of T2DM and poor GCS
12) Significant correlation was found between presence of CKD and poor GCS
13) Significant correlation was found between presence of proteinuria and poor GCS.

CONCLUSION

- Haemorrhagic stroke is more prevalent among known hypertensine patients with poor drug compliance.
- Putamen & Internal Capsule is the Most Common site of Haemorrhagic stroke.
- Presence of Chronic Kidney Disease and Diabetes Mellitus Contributes to poor outcome in ICH patients.

LIMITATIONS OF THE STUDY

- The study populations is small
- The haemorrhage cannot be solely attributed to hypertension as there might be some other confounding factors attributing to it.

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PROFORMA

Name:

Age:

Sex: D.O.A:

Occupation: D.O.D:

Address:

Clinical presentation :altered sensorium/motor disturbances/speech disturbances/headache/convulsions/sensory disturbances/vertigo/vomiting.

Clinical history:

h/o traumatic head injury YES/NO

h/o previous stroke YES/NO

h/o cancer YES/NO

h/o CAD/ any valvular heart disease YES/NO

h/o haemorrhagic disorders YES/NO

h/o any oral anticoagulant/anti-platelet intake YES/NO

h/o chronic kidney disease YES/NO

Personal history :

Smoker : YES/NO duration Alcoholic: YES/NO duration:

Treatment history :

Systemic hypertension : YES/NO Duration : Regular/irregular treatment/newly diagnosed(may be the first presentation with haemorrhagic stroke)

Diabetes YES/NO Duration: Regular/irregular treatment

Previous dialysis(HD/PD) YES/NO

General examination :

Pallor/icterus/cyanosis/clubbing/lymphadenopathy/pedal edema

BP: (on admission) PR:

CNS: CVS:

RS:

PA:

INVESTIGATIONS:

ECG:

Blood urea: S. creatinine: eGFR: RBS:

Lipid profile:

Coagulation profile:

Na+: K+:

Localization of haemorrhage in NCCT brain:

Left/right hemispheres

B/L hemispheres

Basal ganglia unilateral/bilateral

Ventricular extension

Lobar(frontal/parietal/occipital)

Internal capsule

Cerebellum

Thalamus

Pons

Paraventricular region

SL No.	NAME	AGE	SEX	Residence	CLINICAL PRESENTATION						HYPERTENSION			T2DM	CKD		CAD	SMOKER	ALCOHOLIC	BP ON ADMISS		HYPERTENSIVE RETINOPATHY	PROTEINURIA	LVH	LOCALISATION OF BLEED IN CT						
					GCS Score	Hemiplegia	Headache	seizures	sensory loss	Vomiting	REGULAR Rx	IRREGULAR Rx	NEWLY DIAGNOSED		egfr >60	egfr <= 60				SBP> 180	DBP >120				PUTAMEN	INTERNAL CAPSULE	THALAMUS	CEREBELLUM	PONS	LOBAR	with IVH
1	ASHRAF	56	M	U	4		p	p		p		*		p	p			yes		p	p	GRADE III	2+	p	p	P					P
2	MARIAPPAN	36	M	R	3							*		p		p		yes	yes	p	p	GRADE IV	3+	p	p	P					
3	SEENISAMY	46	M	R	9	P							*					yes	yes	p			TRACE	p	p	P					P
4	MAHALAKSHMI	41	F	R	8	P	p			p			*			p	p			p	p		TRACE	p	P	P					
5	RUKMANI	63	F	U	3							*		p						P	P	GRADE II	TRACE						P		
6	MUNIYASAMY	56	M	R	10	P							*						yes	p				P	p	P					P
7	PADMAVATHI	59	F	U	7	p						*		p											P	P					
8	ABARANAM	52	M	R	15	P			p		*							yes	yes	p	p	GRADE I					P				
9	SAMY DOSS	71	M	R	5							*		p				yes		p	p				p	P					P
10	PATHIRAKALI	66	F	U	12	p						*															P				
11	MOHAMMED ISMAIL	59	M	R	15		p			p	*							yes	yes	p	p		TRACE					P			
12	ESAKKIAMMAL	62	F	U	15	P				p		*		p			p					GRADE I			P	P					
13	LAKSHMANAN	49	M	R	11	P					*								yes					p	P	P					
14	SUBBIAH	38	M	U	12	p							*	p	P			yes		p	p		2+	p	p	P					P
15	THANGAMUTHU	44	M	R	8	P		p				*												p	P	P					
16	KANIAMMAL	51	F	R	5								*			P				p	p	GRADE III	2+	p					P		
17	MARIAPPAN	67	M	R	12	P						*							yes	p	p				p	P					
18	LAKSHMI NARAYANAN	49	M	U	6							*		p		p	p	yes	yes	p	p	GRADE II	1+	p						P	
19	SUBRAMANIYAN	75	M	U	7	p	p			p			*							p						P	P				P
20	MADATHIAMMAL	50	F	R	9		P			P		*								P	P	GRADE II		p				P			
21	PONSELVI	55	F	U	10	p						*								P	P	GRADE II		P			P				
22	MUNIYASAMY	72	M	R	15	p					*									p	p	GRADE I				P	P				
23	GANDHIMATHI AMMAL	51	F	R	3			p				*		P		P	P			p	p	GRADE IV	2+	p					P		
24	RAMACHANDRAN	49	M	U	15	p						*						yes		p		GRADE I			p	P					P
25	MALAIYAMMAL	80	F	R	7	p	p			p			*							p			2+	p		P	P				
26	GOMATHIAMMAL	39	F	R	14	p			P			*		p		p				p	p	GRADE II	2+	p			P				
27	ESAKKIPANDI	71	M	R	5	p		p					*	P	P				yes	p	p	GRADE III	2+	p	p	P					
28	VEERAMMAL	56	F	U	9	p		p				*								p	p				p	P					P
29	KANNAN	45	M	R	11	p						*		P	P					p	p	GRADE II	1+	p	p	P					
30	AYYAKANI	49	M	R	12		p			p			*					yes	yes	p	p			p				P			
31	RAMAR	56	M	R	6	p							*	p				yes		p	p		TRACE			P	P				
32	ABUBAKKAR BIETHOESH	72	M	R	4								*	P	P					p	p	GRADE III	2+	p	p	P					P
33	ARUNACH ALA VADIVU	58	F	U	15		P				*									p								P			
34	PUSHPARAJ	34	M	U	3			p		p		*				P				p	p	GRADE IV	2+	p					P		
35	SEETHALAKSHMI	67	F	R	15	p				p	*								yes	p				p	P	P					
36	YESAIAH	66	M	R	12	p							*					yes	yes	p	p			p	p	P					P
37	THANGAMMAL	78	F	U	7	p						*					p									p	P				
38	ESAKKIPANDI	70	M	U	13	p						*			P			yes		p	p	GRADE II	TRACE	p	p	P					
39	RAJESH	61	M	R	4			p					*	p		P				p	p	GRADE III	2+	p	p	P					P
40	LAKSHMI	58	F	R	12	p				p		*								p	p			p	p	P					
41	SELVAM	65	M	R	5		p			p		*						yes	yes									P			
42	PUITCHAMMAL	59	F	U	15	p							*	p						p	p	GRADE I	TRACE	p			P				
43	MANIKANDAN	72	M	R	5							*								p	p				p	P					P
44	GOMATHI	71	F	R	14	p						*		p		p	p			p	p		2+	p	p	P					P
45	ESAKKIMUTHU	79	M	U	6	p		p					*							p	p				p	P					p
46	MANI	67	M	U	13	p	p			p		*		P	P					p	p	GRADE II	2+	p	p	P					
47	PARAMASIVAM	53	M	U	7	p							*					yes		p	p				p	P					P
48	LAKSHMIAMMAL	60	F	R	3							*		P	P					p	p	GRADE III	2+	p				p			
56	MURUGAN	74	M	U	3							*		p		p		yes	yes	p	p	GRADE IV	3+	p					P		
50	NATARAJAN	62	M	R	5		p			p			*							p										P	
51	DURAICHI	59	F	R	12	p			p	p	*			P		P				p	p	GRADE II	1+	p		P	p				P
52	ARUNACHAL;AM	72	M	R	6	p	p			p		*		p		P	p		yes	p	p	GRADE III	2+	p			P				

53	NOORJAHAN	51	F	U	15	p			p		*									p	p						p				
54	MURUGAN	47	M	U	4							*		p	p					p	p	GRADE III	2+	p	p	P					P
55	SUDALAIKANNU	55	M	R	11	p							*							p	p				p	P					
56	CHELLAMAL	79	F	R	5							*								p	p								P		
57	DHARMAKANI	68	F	R	6	p						*					p							p			P				
58	LAKSHMANAN	72	M	R	6		p			p		*						yes	yes					p				P			
59	SELVARAJ	76	M	U	8	p	p			p		*		p			p			p	p		TRACE		p	P					
60	ASIRVATHAM	58	M	U	4	p	p			p		*		P	P			yes		p	p	GRADE III	2+	p	p	P					P
61	THANGADURAI	66	M	U	10		p			p			*					yes		p								P			
62	CHANDRA	51	F	R	3							*		p		P				p	p	GRADE III	1+	p	p	P					P
63	MAHARAJA	74	M	R	15	p					*			p			p			p	p	GRADE I				P	P				
64	CHERMAKANI	76	F	U	12	p						*								p	p			p	p	P					
65	KALIMUTHU	59	M	R	3			p					*			P		yes	yes	p	p	GRADE III	2+	p	p	P					P
66	GOPAL	73	M	R	12	p						*		p						p	p				p	P					P
67	RAJAVADIVU	82	F	U	6	p							*							p	p				p	P					
68	IMMANUVEL	49	M	U	13	p	p		P	p		*			P			yes	yes	p	p	GRADE II	1+	p			P				
69	KALAIAMMAL	54	F	R	5	p	p			p		*								p	p				p	P					P
70	GURUSAMY	49	M	U	4								*	p						p			TRACE						P		
71	SAMUVEL	71	M	R	5	p		p					*					yes		p				p					P		
72	MUPPIDATHY	60	F	R	15	p					*		*				p			p	p				P	P					
73	SUBBUKUTTI	77	M	U	15	p	p			p		*								p	p			p	p	P					P
74	ZAHIRHUSAIN	63	M	R	6		P						*	p				yes	yes	p								P			
75	ESAKKIAMMAL	66	F	R	15	p					*									p	p						P				
76	MUTHUKRISHNAN	48	M	R	7	p						*								p	p				p	P					
77	SHANMUGAKANI	57	M	R	4							*								p	p								P		
78	NARAYANAPILLAI	62	M	U	12	p						*		p	P		p	yes	yes	p	p	GRADE II	2+	p	p	P					P
79	CHINNATHAMBI	54	M	U	3		p			p			*							p	p		1+		p	P					
80	PARAMESHWARI	59	F	R	6	p							*	p		p				p	p		2+	p	p	P					P
81	SUBBAIAH PILLAI	74	M	R	5	p						*						yes	yes	p						P	P				
82	KUMARAVEL	54	M	U	3		p	p		p		*								p	p								P		
83	MALAIAMMAL	72	F	R	9	p						*		p						p	p				p	P					
84	GOMATHINARAYANAN	69	M	R	10	p						*		p		p		yes	yes	p	p		2+	p	p	P					P
85	ABDUL AASAN	39	M	R	11	p							*	p	P		p			p	p	GRADE II	2+	p	p	P					
86	MUTHUPANDI	68	M	R	3							*						yes	yes	p	p								P		
87	PETCHIMUTHU	54	M	R	5	p	p			p		*								p					p	P					
88	JAMILA	63	F	U	3							*		p						p	p								P		
89	PAULRAJ	61	M	U	13	p					*									p	p				p	P					
90	ESAKKI	44	M	R	12	p							*	p		p		yes	yes	p	p		2+	p	p	P					
91	SELVARAJA	58	M	U	9	p	p			p		*								p	p				p	P					
92	ASYAL BEEVI	61	F	R	4		p			p			*	p			p			p	p								P		
93	TINOSAN	47	M	R	5		P			P		*		p		p		yes	yes	p	p		2+	p					P		
94	PETCHI	55	M	U	6	p						*		p						p								P			
95	TAMILSELVI	58	F	R	11	p			P			*								p							P				
96	ABDUL RAHIM	81	M	R	4							*		p		p		yes	yes	p	p		2+	p					P		
97	LEELAVATHY	75	F	R	3		p	p		p			*							p	p								P		
98	MARIYAMMAL	77	F	U	9		P					*		p						p	p			p				P			
99	PAPPA	58	F	U	10	p						*								p	p				P	P					
100	PITCHAMMAL	55	F	R	6		p			p			*	p		p				p	p		2+	p				P			