PROGNOSTIC PREDICTORS OF 30 DAY OUTCOME IN PATIENTS WITH NON TRAUMATIC INTRAPARENCHYMAL HAEMORRHAGE

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MARCH 2010

CERTIFICATE

This is to certify that the dissertation titled **"PROGNOSTIC PREDICTORS OF 30 DAY OUTCOME IN PATIENTS WITH NON TRAUMATIC INTRAPARENCHYMAL HAEMORRHAGE"** is the bonafide original work of **Dr. ELAVARASI MANIMEGALAI. E** in partial fulfillment of the requirements for M.D. Branch – I (General Medicine) Examination of the Tamilnadu DR. M.G.R Medical University to be held in MARCH 2010. The Period of study was from January 2009 to June 2009.

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DECLARATION

I, **Dr. ELAVARASI MANIMEGALAI. E** solemnly declare that dissertation titled **"PROGNOSTIC PREDICTORS OF 30 DAY OUTCOME IN PATIENTS WITH NON TRAUMATIC INTRAPARENCHYMAL HAEMORRHAGE"** is a bonafide work done by me at Madras Medical College and Government General Hospital, Chennai, during January 2009 to June 2009 under the guidance and supervision of **Prof. K.Sivasubramanian , M.D.,** Professor of Medicine, Madras Medical College and Government General Hospital, Chennai.

This dissertation is submitted to the Tamilnadu DR. M.G.R Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree (Branch – I) in General Medicine.**

Place : Chennai **(Dr. ELAVARASI MANIMEGALAI. E)**

Date :

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A special mention of thanks to all the patients who participated in this study for their kind cooperation.

I would like to thank my colleagues and friends who have been a constant source of encouragement.

CONTENTS

INTRODUCTION

Cerebrovascular diseases rank first in frequency and important among all neurologic diseases. It is the second cause of mortality in the World. A stroke or cerebrovascular accident is defined by the abrupt onset of a neurologic deficit that is attributable to a focal vascular cause.

80% of stroke is ischaemic and remaining 20% is due to haemorrhage. Intracranial haemorrhage includes:

1. Intraparenchymal haemorrhage
2. Intraventricular haemorrhage
3. Subarachnoid haemorrhage

There is 2 – 3 fold increased risk of intraparenchymal haemorrhage in Asians and Blacks.

Many controversies surround the management of patients with ICH in large part because of paucity of prospective randomized controlled trial data that might more rationally guide therapy. These controversies include management of hypertension, treatment of raised ICP and appropriate use of surgical techniques. Since there is no sufficiently efficacious therapy for haemorrhage induced cerebral injury, prevention is the mainstay of treatment particularly hypertension. It is appropriate to think in terms of pathophysiology to guide management decisions. It would be useful, therefore, to know something about the time, course and prognosis of ICH.

Various factors have been identified as predicting outcome including age, gender, race, initial MAP, temperature, volume of bleed, site of bleed, intraventricular extension, mass effect and initial level of consciousness.

This study is done to evaluate and discuss the significances of various factors in predicting 30 day mortality and morbidity of patients with non-traumatic intraparenchymal haemorrhage in hypertension and non-hypertension haemorrhage stroke.

AIM OF THE STUDY

1. To evaluate the prognostic factors in predicting 30 day outcome in patients with non traumatic intraparenchymal haemorrhage admitted to medical wards in the Government General Hospital, Chennai.

2. To find out the prognostic significance of:

    a. Age

    b. Temperature

    c. Mean arterial pressure

    d. GCS score at admission

    e. Volume of Bleed

    f. Location of Bleed

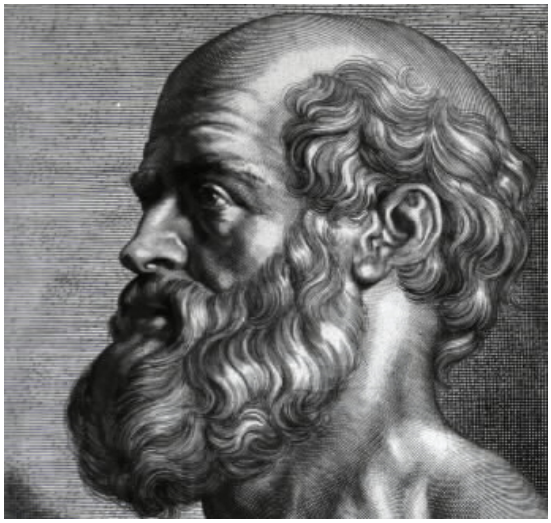
    g. Intraventricular haemorrhage

    in predicting 30 day outcome assessed by NIHSS score in patients with non-traumatic intraparenchymal haemorrhage admitted to the medical wards in the Government General Hospital, Chennai.

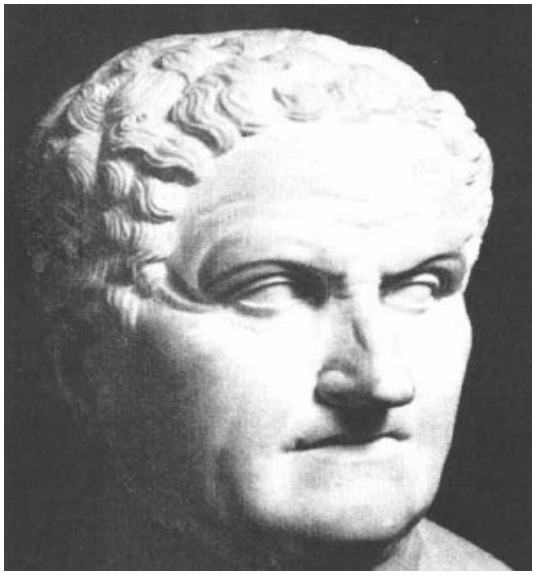
3. To compare the results with that one reported in literature.

REVIEW OF LITERATURE

HISTORICAL REVIEW OF LITERATURE



HIPPOCRATES

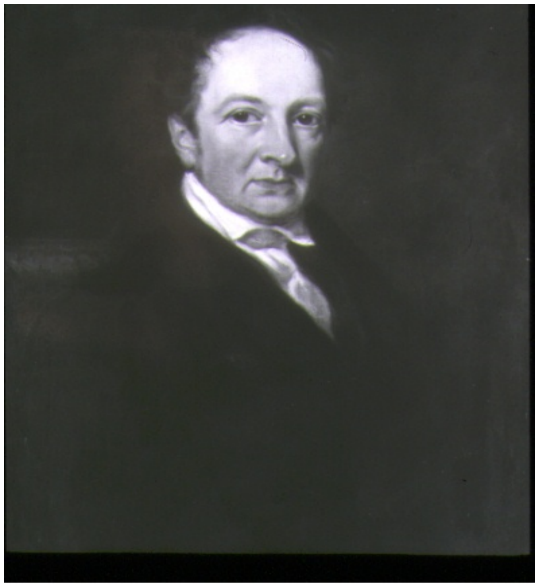


GALEN

Stroke history dates back to Hippocrates and Galen who named at 'apoplexy' a term that physicians applied to anyone suddenly struck down with paralysis but could not explain its pathology.

JOHANN JACOBB WEPFER

The first person to investigate the pathological signs of apoplexy was **Johann Jacobb Wepfer**. Born in Schaffhausen, Switzerland, in 1620, Wepfer studied medicine and was the first to identify postmortem signs of bleeding in brains of patients who died of apoplexy. From autopsy studies, he gained knowledge of carotid and vertebral arteries that supply the brain. He was the first person to suggest that apoplexy in addition to being caused by bleeding in the brain, could be caused by blockage of one of the main arteries supplying blood to the brain. Thus, stroke came to be known as CVD – Cerebrovascular disease, Cerebro-part of brain, Vascular – blood vessels.

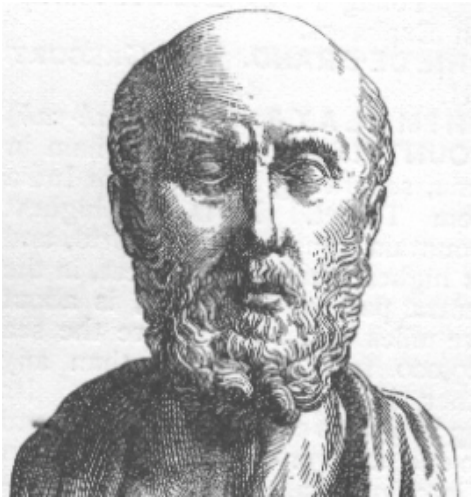


JOHN CHEYNE

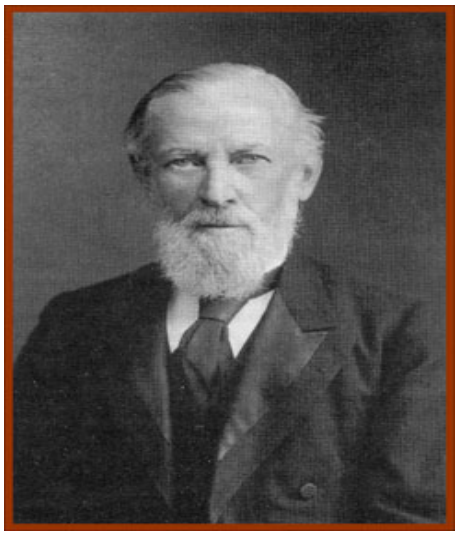
CHARLES BOURCHARD

John Cheyne (1777 – 1836) in his cases of apoplexy and lethargy distinguished SAH and ICH probably the first physician to provide an illustration of SAH.

Charles Bourchard (1827 – 1915) was one of the first to provide evidence that arterial hypertension might be an important cause of haemorrhages in brain. He also found small leisions resembling tiny globules of grain along many penetrating arteries so called military aneurysms were described as one of the cause of brain haemorrhage.



HENRY DURET



SIR WILLIAMS GOWERS

Henry Duret (1849 – 1921) a French Neurosurgeon described the anatomy of arteries to brain and identified the common sites of brain infarct and haemorrhage along with **Charcot** he described the anatomy of arteries to brain and identified the common sites of brain infarct and haemorrhage along with charcot he described the **lenticulostriate artery** and called it the artery of cerebral haemorrhage. He also studied traumatic brain haemorrhages and became aware that many had haemorrhages in upper brainstem near midline so called **DURET HAEMORRHAGES.**

Sir Williams Gowers Began his work of ICH by "Haemorrhage is always due to rupture of a vessel". **Sir Miller Fischer,** during the middle years of 20th century made new clinical and pathologic observations about ICH. He analysed the clinical findings in patients with haemorrhages found at common sites for hypertensive ICH such as putamen, Thalamus, Pons and Cerebellum.

Marcello Malpighi
(1628-1694)

The autopsy on **Marcello Malphigi,** the famous Italian anatomist fortuitously solved the mystery of his long standing Right hemiplegia from stroke for some years before his death as it revealed haemorrhage into his lateral ventricles (Fanyio 1983).

The advent of **CT Scanning** into clinical neurology in 1970s helped to greatly expand our knowledge about ICH. It proved to be an ideal technology for the diagnosis of brain haemorrhages.

The introduction of MRI in 1980s helped in detection of old haemosiderin containing cavities, vascular malformation and three dimensional reconstruction of location and dimensions of bleed.

CURRENT REVIEW OF LITERATURE

In a healthy functioning brain, neurons do not come into direct contact with blood. The vital oxygen and nutrients the neurons need come from the blood across the thin walls of cerebral capillaries. The glia (nervous systems cells that support and protect neurons) form a blood brain barrier an elaborate meshwork that surrounds blood vessels and capillaries and regulates which elements of the blood can pass through the neurons.

When an artery in the brain bursts, blood spurts out into the surrounding tissue and upsets not only the blood supply, but the delicate chemical balance neurons require to function. This is called a haemorrhagic stroke.

Haemorrhages are classified by their location and underlying vascular pathology. Haemorrhagic stroke can be **diffuse** i.e. bleeding into subarachnoid spaces (or) **focal** (intraparenchymal).

CAUSES OF ICH

I. Diffuse haemorrhagic stroke (SAH & IVH):

This is due to rupture of vessels on or near the surface of brain or ventricles.

1. Aneurysms – Berry, Fusiforus, mycotic
2. AV malformation
3. Trauma

4. Vasculitis

5. CNS neoplasms

6. Haematological disorders like Haemophibia, leukemia, TTP

II. Focal haemarrhagic stroke

This is due to rupture of arteries that are within the brain substance.

1. Hypertension

2. Vascular malformation

3. Amyloid angiopathy

4. Anticoagulant therapy

5. Haematologic disease – leukemia, aplastic anaemia

6. Drug abuse – cocaine, amphetamine

7. Neoplasms – Glioblastoma multiforme

8. Head Injury

9. Sepsis

10. Moya moya disease

The most important modifiable risk factor for IPH is hypertension particularly people <55 years, smokers and poor compliance with antihypertensives. People with hypertension have 4 – 6 times increased risk than

patients without hypertension. Excessive alcohol consumption, anticoagulation, thrombolysis and various haematologic abnormalaities increase the risk. Warfarin anticoagulation to INR 2.5 – 4.5 is associated with ICH – 1%/year. Patients treated with tPA have 6.5% risk of ICH compared to placebo – 0.5%. A less well established risk factor is a loco serum cholesterol concentration <160mg%. Advanced age is an independent risk factor.

PATHOPHYSIOLOGY OF NON-TRAUMATIC IPH

It usually results from spontaneous rupture of a small penetrating artery deep in the brain occurs predominantly as consequence of chronic uncontrolled hypertension. The extravasation forms a roughly circular or oval mass that disrupts the tissue and grows in volume as the bleeding continues. Adjacent brain tissue is distorted and compressed. The displacement of normal brain tissue by haematoma and dissection of blood along fibre tracts account for much of the pathology.

If the haemorrhage is large, midline structures are displaced to the opposite side and reticular activating and respiratory centres are compromised by herniation leading to coma and death. The location and size of the clot and the oedema that surrounds the clot determines the degree of upper brainstem compression.

Blood may dissect into ventricular space and CSF becomes blood in more than 90% cases. A haemorrhage of this type never ruptures through the corten, the blood reaching the subarachnoid space via ventricular system.

When haemorrhage is small and located at a distance from ventricles, the CSF may remain clear even on repeated examinations. In first hours and days following the haemorrhage oedema accumulates around the clot and adds to the mass effect. Hydrocephalus may occur as a result of bleeding into the ventricular system of basal disterns or compression of third ventricle.

If the patient survives, the extravasated blood undergoes a predictable series of changes. At first, it clots within hours. Before the blood clots, red cells may settle in the dependend part of the haematoma and form a meniscus with the plasma above. This is particularly prone to occur in anticoagulant induced haemorrhage. Only mass of RBCs and protein are found within the haematoma rarely one sees a few remnants of brain tissue. The haematoma is surrounded by petechial haemorrhages from arterioles and venules.

Within a few days, Hb products mainly haemosiderin and haematiin begin to appear. The haemosiderin form within histiocytes that have phagocytosed RBCs and takes the form of ferritin granules which stain positively for iron. As Oxy Hb is liberated from RBCs and becomes deoxygenated Meth Hb is formed. This hegan within a few days and imparts a brownish hue to the periphery of the clot. Phagocytosis of RBCs begin within 24 hrs and haemosiderosis is first observed around the margins of the clot in 5 to 6 days.

The clot changes colour gradually over a few weeks from dark red to pale red and the border of golden brown haemosiderin in due. The oedema

disappears over many days or weeks. In 2 – 3 months, larger clots are filled with a chrome coloured mesh which is slowly absorbed leaving a smooth walled cavity. (slit haemorrhages) or a yellow brown scar. The iron pigment (haematin) becomes dispersed and studs adjacent astrocytes and neurons. It may persist well beyond the border of the haemorrhage for years.

Most hypertensive intraparenchymal haemorrhages develop over 30 to 90 minutes whereas those associated with anticoagulant therapy may evolve for as long as 24 – 48 hours. The most common sites of non-traumatic intraparenchymal haemorrhage in order of frequency are:

i. Basal ganglia (Putamen and adjacent deep white matter)

ii. Thalamus

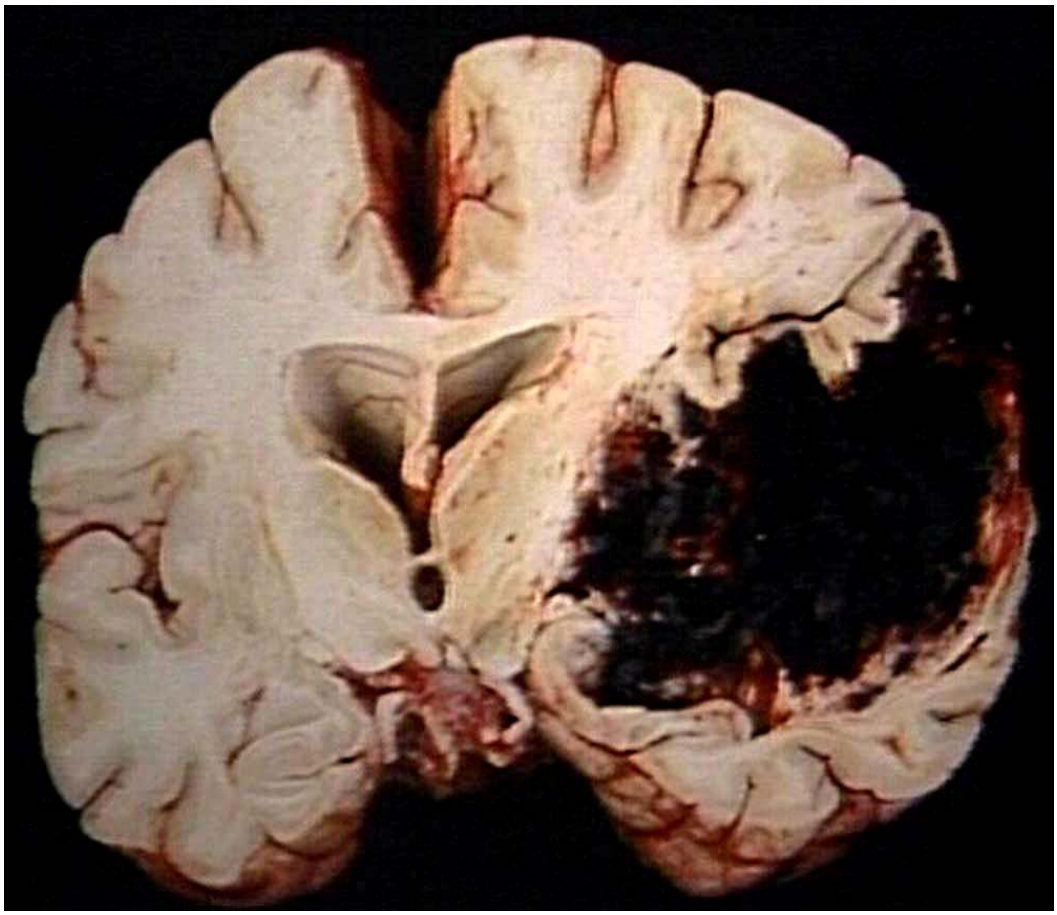
iii. Central white matter of temporal, parietal, frontal lobes (lobar haemorrhage).

iv. Deep cerebellum

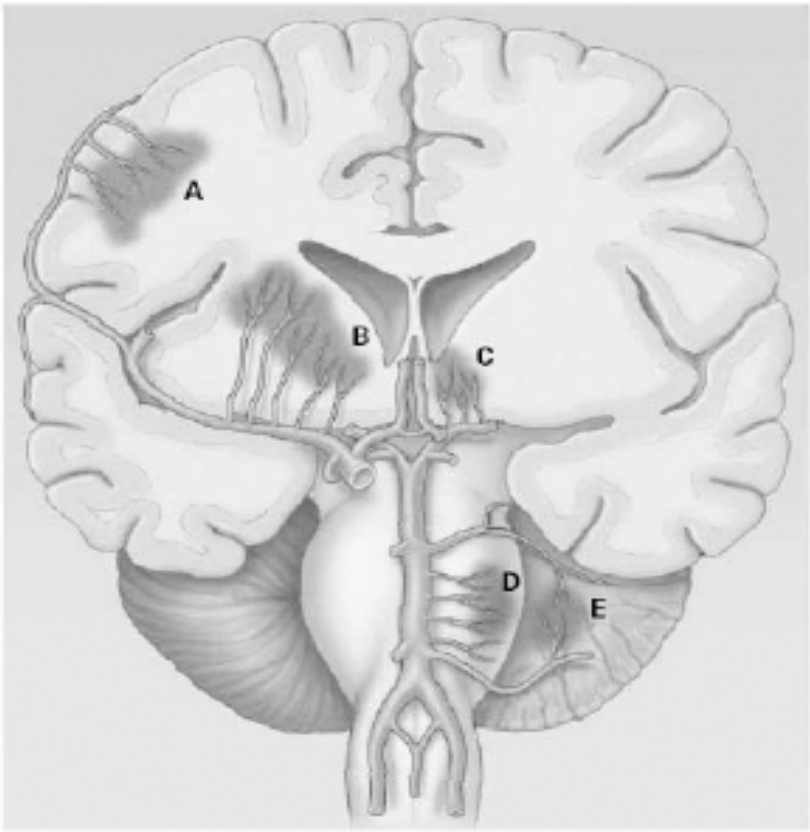
v. Pons

About 2% primary haemorrhages are multiple. Rarely the bleeding is solely intraventricular possibly from choroid plexus. When haemorrhage occurs in other brain areas or in non-hypertensive patients greater consideration should be given to haemorrhagic disorder, anticoagulant use, neoplasm, vascular malformation and causes other than hypertension.

POSTMORTEM BRAIN SPECIMEN OF A CASE OF INTRACEREBRAL HAEMARRHAGE



COMMON SITES OF INTRACEREBRAL HAEMORRHAGE



(A) Cerebral lobes, originating from penetrating cortical Branches of the anterior, middle, or posterior cerebral arteries.

(B) Basal ganglia, originating from ascending lenticulostriate branches of the middle cerebral artery.

(C) The thalamus, originating from ascending thalamogeniculate branches of the posterior cerebral artery.

(D) The pons, originating from paramedian branches of the basilar artery

(E) The cerebellum, originating from penetrating branches of the posterior, inferior, anterior inferior, or superior cerebellar arteries.

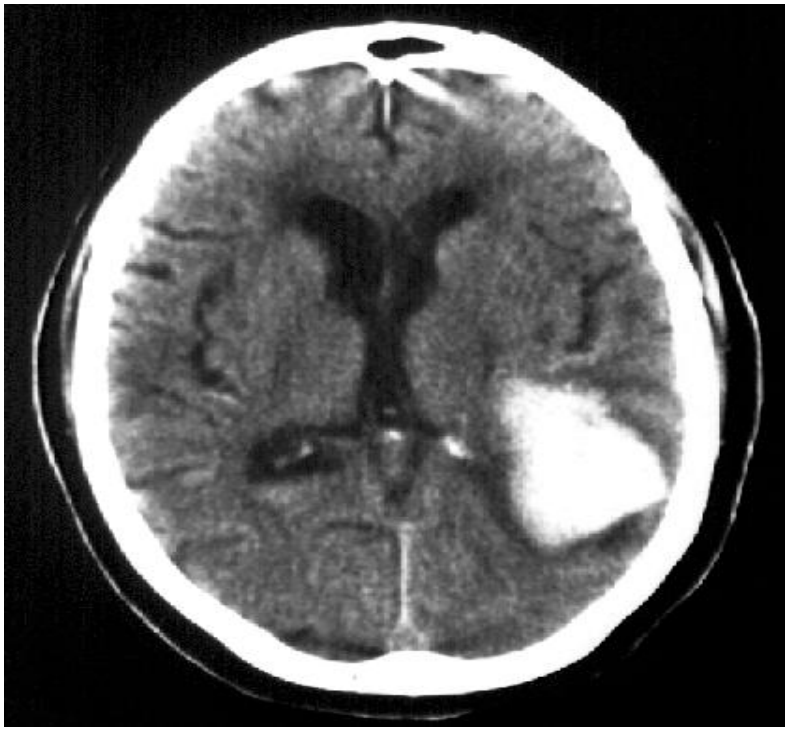
RADIOLOGICAL FINDINGS IN ICH

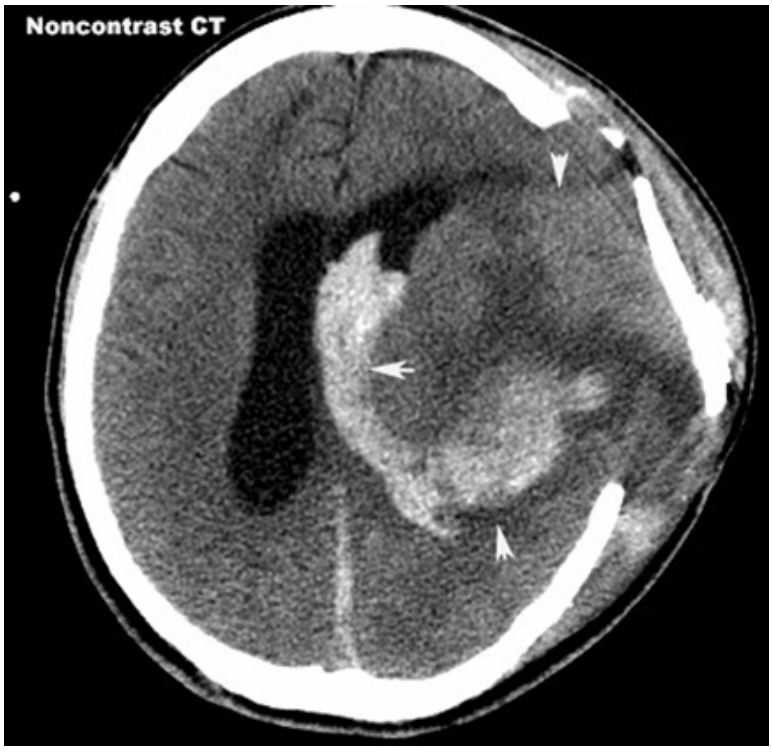
CT SCAN:

CT occupies the foremost position among the lab methods for diagnosis of ICH. This procedure has proved totally reliable in the prediction of haemorrhages that are 1 cm or more in diameter. Smaller pontine haemorrhages are usually visualized with less certainity. At the same time, co-existing hydrocephalus, tumors, cerebral swelling and displacement of intracranial contents are readily appreciated.

In CT scan, fresh blood loss is visualized as a white mass as soon as it is shed. The mass effect and the surrounding extruded serum and oedema are hypodense. After 2 – 3 weeks, the surrounding oedema begins to recede and the density of haemoatonea decreases first at the periphery. Gradually the clot becomes isodense with the brain. There may be a ring of enhancement from the haemosiderin filled macrophages and the reacting cells forming the capsule of the haemorrhage.

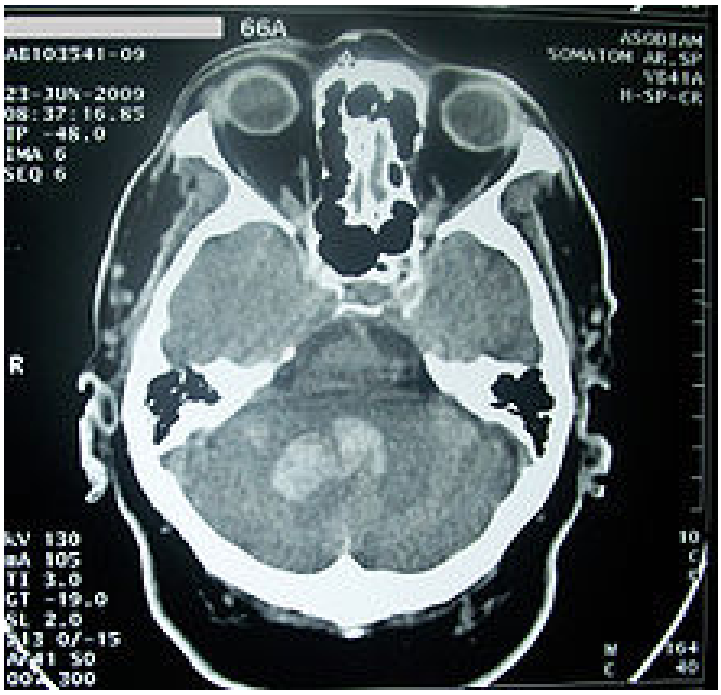
LEFT PARIETO OCCIPITAL BLEED

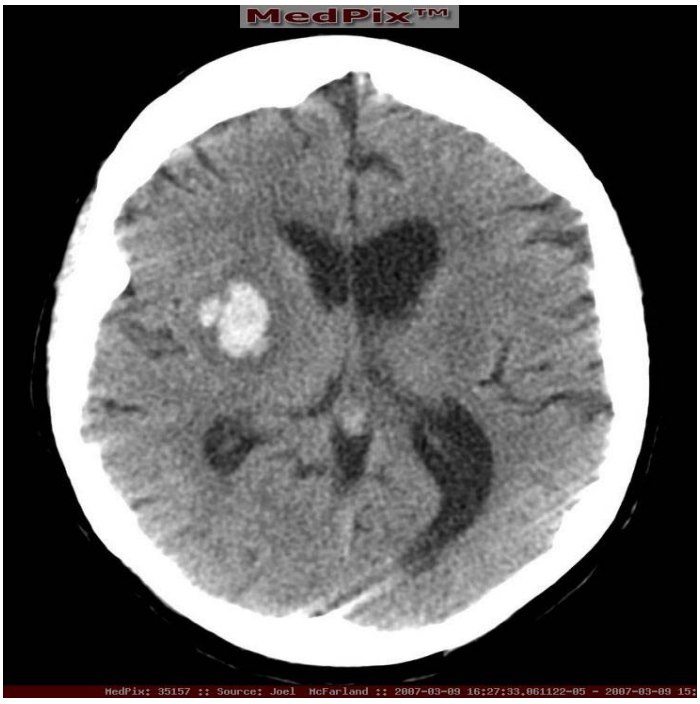




LEFT BASAL GANGLIA BLEED SQUASHING LEFT LATERAL VENTRICLE

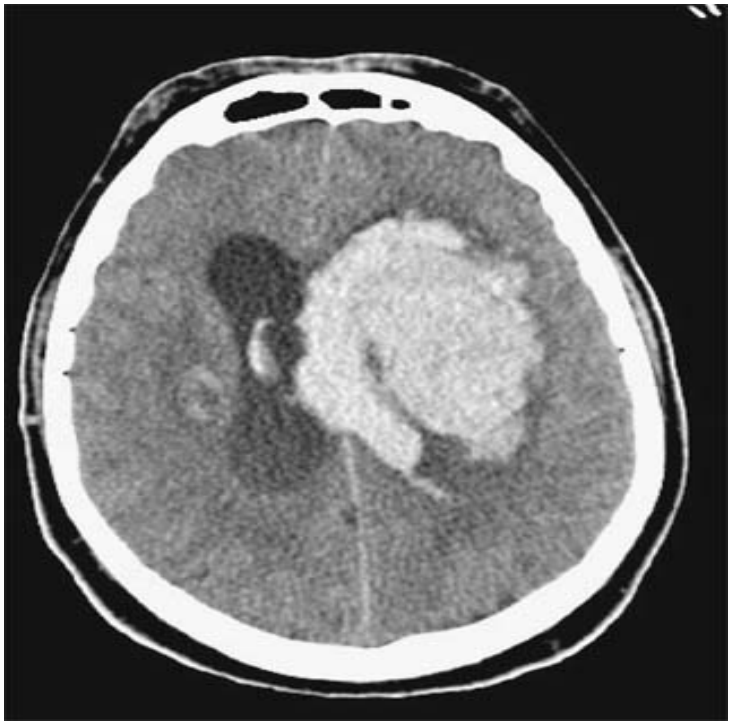
CEREBELLAR BLEED

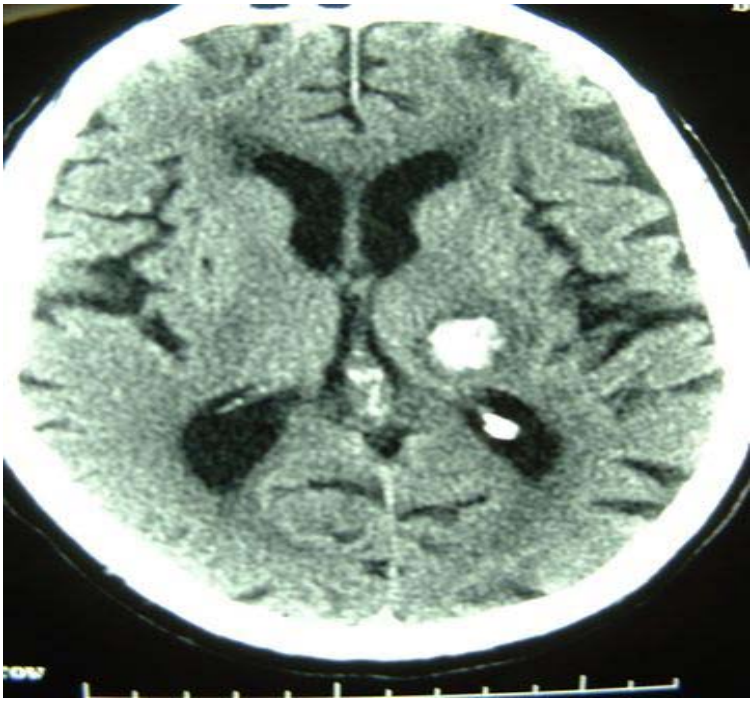




RIGHT THALAMIC BLEED

LEFT PUTAMEN BLEED EXTENDING INTO LATERAL VENTRICLES





LEFT PUTAMEN BLEED

MRI

By MRI, either in T1/T2 weighted images, haemorrhage is not easily visible in 2/3 after bleeding since Oxy Hb is diamagnetic or at most is slightly hypointense so that only the mass effect is evident. After several days, the surrounding oedema is hyperintense in T2 weighted images. As deoxy Hb and Meth Hb form, haematoma signal becomes bright on T1 weighted images and dark on T2. As the haematoma becomes subacute, the dark images gradually brighten when Meth Hb disappears and only haemosiderin remains, the entire remaining mass is hypodense onT2 weighted images as are the surrounding deposits of Fe.

MRI is particularly useful in demonstrating brainstem haemorrhages and residual haemorrhages which remain visible long after they can no longer be seen by the CT scan (4 – 5 weeks).

Intraparenchymal haemorrhages may be described as massive, moderate, small, slit and peticheal.

1. Massive refers to haemorrhage with 7 – 10 cms diameter (or) volume >60 ml.
2. Small referes to haemorrhage with 1 – 2 cm diameter (or) volume <30 ml.
3. Moderate refers to haemorrhage in between the above two both in diameter and volume.
4. Slit refers to old collapsed HT or traumatic haemorrhage that lies just beneath the cortex.

COURSE AND PROGNOSIS OF IPH

The immediate prognosis of large and medium sized cerebral clots is grave i.e. nearly 50% patients die, half of which occurs within first 48 hours either the haemorrhage extends into the ventricular system or intracranial pressure is elevated to levels that preclude normal perfusion of brain. Sometimes the haemorrhage itself seeps into vital centres such as hypothalamus or mid brain and results in poor outcome but others may have a good to complete recovery if they survive the initial haemorrhage …….. various factors are used to predict the outcome of IPH.

VOLUME OF BLEED AND PROGNOSIS

Supratentorial haemorrhages with volume <30 ml have a good prognosis. 30 – 60 ml intermediate and >60 ml poor prognosis during initial hospitalization. Supratentorial haemorrhages smaller than 30 ml rarely produce death unless they are located in the thalamus. Extenion into ventricles, especially the fourth ventricle worsens the prognosis. Haematomas may expand for several hours following the initial haemorrhage, so treating severe hypertension seen reasonable to prevent haemotoma progression.

Infratentorial haemoatomas >3 cm in diameter have worse prognosis and needs surgical evacuation. In recent series, 30 day case fatality rate of IPH averaged 30 – 50%. Long term prognosis for various degrees of recovery is similar to or better than cerebral infarct of same severity.

METHOD TO CALCULATE VOLUME OF BLEED

Lisch and associates and Kothari and colleagues have estimated the volume of IPH. The **formula for an ellipsoid** is as follows:

$$4/3\ \pi\ (A/2)\ (B/2)\ (C/2)$$

Where A, B, C are the three dimensions of an ellipsoid that approximates the IPH. This formula can be simplified by acknowledging that π is approximately 3 and then reducing the formula to:

$$ABC / 2$$

For the bedside ABC / 2 method, CT slice with the largest area of haemorrhage is identified. The longest diameter (A) of the haemorrhage on this slice is measured. The diameter perpendicular to A on the same slice is measured as (B). Finally the number of slices upon which haemorrhage is seen is multiplied by slice thickness is calculated as the third dimension (C). If the haemorrhage area for a particular slice is greater than 50% of the area seen on the slice where the haemorrhage is largest, the slice is considered haemorrhage slice for determining C. If the area is approximately 25 – 75% of the area, the slice is considered half a haemorrhagic slice and if the area is <25% of the largest haemorrhage, the slice is not considered a haemorrhagic slice. The CT haemorrhagic slice values are then added to determine the value for C. Multiplying A,B and C together and dividing by 2 will give the approximate volume of IPH. This method has been shown to be extremely accurate method compared with other methods.

MAP AND PROGNOSIS

Dhandapani et al in a retrospective study examined the relationship between blood pressure and outcome in 87 patients after ICH. Outcome was assessed using Glassgow coma score. Case fatality and combined morbidity and mortality rates were reported based on a stratification using cut off MAP of 145 mmHg in first two hours. Morbidity and mortality were significantly greater among those with MAP greater than 145 mmHg during first two hours after presentation.

According to Lisch D.Pasteur – W – Rhoades H et al, the three most useful indicators in determining the prognosis of patients with IPH appears to be:

1. Volume of haemorrhage
2. Level of consciousness upon arrival at emergency department
3. Presence / absence of intraventricular extension

On the basis of these three parameters, emergency physicians assist in early decision making both for family members and in concert with consultants.

GCS AND PROGNOSIS

The next two predictors are the patients **GCS and Pre/absence of IVH.** Broderich et al developed 9 simple models to predict 30 day mortality after IPH. It is mainly applicable to putaminal and thalamic haemorrhage (i.e.) deep haemorrhages. They reviewed the clinical data and CT scans of 188 patients with intraparenchymal haemorrhage. Neurological function at presentation was

documented using GCS score and clinical outcomes were graded using a modified Oxford Handicap scale.

Using 30 day mortality as the dependent variable, they analyzed age, race, gender, initial systolic BP, volume of IPH, volume of IVH, initial GCS score, location of haemorrhage and surgery using univariate regression analysis and found that volume of IPH, volume of IVH and initial GCS score were significant predictors of 30 day mortality. Using the data from their study, the emergency physician can determine whether the patients with IPH are at very high risk or low risk.

In patients with IPH volume >60 ml GCS <8, 30 day mortality = 91%

In patients with IPH volume <30 ml, GCS >9, 30 day mortality = 19%

Although 19% mortality is still high, it represents a good prognosis for IPH.

Notably morbidity remains quite high for intraparenchymal haemorrhage. In the study of Broderick and colleagues only 1 of 71 patients with IPH volume >30 ml could function independently at 30 days.

LOCATION OF BLEED AND PROGNOSIS

As remarked earlier, it is the location of the haematoma, not simply its size that determines the clinical effects. A clot of 60 ml is almost uniformly fatal, if situated on basal ganglia but may be relatively benign if located in frontal and occipital lobe.

HYDROCEPHALUS AND PROGNOSIS

From the studies of Biringer and colleagues, it appears that hydrocephalus is also an important predictors of poor outcome.

NIHSS SCORE AND PROGNOSIS

Initial severity of stroke often measured by NIHSS score is one of the major predictors of poor outcome including mortality after stroke in many studies. Adams H Davis P Leira I et al in their study have shown that NIHSS score of 16/higher predicts a high probability of death or severe disability and that a score of 6 or less predicts a good recovery.

In patients who survive there can be surprising degree of restoration of function since in contrast to infarction, haemorrhage has to some extent pushed brain tissue aside rather than destroyed it. Function may return very slowly because the extravasated blood takes time to be removed from the tissues. Also since rebleeding from the site is unlikely, patient may live for many years.

In some instances, medium sized cerebral and cerebellar haemorrhages, patient survives but papilloedema appears after several days of ↑ ICP. This does not mean that the haemorrhage is increasing in size / swelling only that papilloedema is slow to develop. Healed scars impinging on the cortex are liable to be epileptogenic.

A Prospective Study was done by Yair Lampl, MD., Ronit Gilad; Yehiel Eshel Ida Sarova-Pinhas to evaluate neurological and functional outcome after spontaneous supratentorial bleeding. The aim of the study was to determine whether clinical or neuroradiological parameters could predict the outcome of these patients during the first hours of hospitalization.

Two hundred seventy-nine patients—52 with thalamic, 87 with putaminal, and 140 with lobar hemorrhages—were followed prospectively and examined on admission and at 2 weeks, 3 months, and 6 months after onset. The patients underwent clinical (according to the Glasgow Coma Scale) and neuroradiological examinations on admission and were scored clinically and functionally (according to Stroke Severity score and Barthel Index) on the follow-up periods. Risk factors and the correlation between findings on admission and the latest clinical and functional results were calculated with the χ^2 test, Pearson correlation test, and Student's *t* test. Multivariate analysis was calculated with the stepwise regression test.

In all of the bleeding locations, lethal outcome was significantly correlated with size of the hematoma ($P<.001$) and Glasgow Coma Scale score on admission ($P<.001$). Intraventricular blood expansion was found to have a better prognosis in thalamic bleeding ($P<.007$) and a worse prognosis in lobar hemorrhage ($P<.01$). The functional outcome after 6 months was directly correlated with the size of the bleeding area in lobar and putaminal hemorrhages. No correlation was found in thalamic bleeding. A worse functional outcome was

found in putaminocapsular bleeding (P=.004) and in patients with ischemic heart disease. A limited better recovery prognosis was found in patients with lobar hematoma in the temporal lobe (P=.052).

The probability of lethal outcome can be calculated on admission in all patients with supratentorial bleeding and in correlation with the location and size of the bleeding area and level of consciousness. Intraventricular expansion of blood is a better prognostic factor in thalamic bleeding and a worse one in lobar hematoma. Functional outcome is correlated with size of the bleeding area and level of consciousness on admission in putaminal and lobar hemorrhages but has no correlation to thalamic hemorrhage.

All patients were hospitalized in the neurological department 15 minutes to 4 hours (mean, 90±140 minutes) after the onset of the first symptoms. The patients included had supratentorial bleeding, were of both sexes, and were older than 20 years. Patients with recurrent intracerebral hemorrhage, previous stroke, or neuroradiological evidence of arteriovenous malformation, brain tumors, or predominant subarachnoid hemorrhage and patients receiving anticoagulant therapy were excluded from the study.

All patients were examined clinically and evaluated by two neurologists. The follow-up period of each patient observed in this study ended after 6 months or by a fatal outcome. Neuroradiological measurements were estimated by a neuroradiologist who was not aware of the clinical status of the patients.

The following clinical parameters were noted: vascular risk factors; hypertension (>170/90 mm Hg; diabetes mellitus (glucose level >140 mg/dL preprandial on two examinations, glucose level >200 mg/dL postprandial, or HbA_{1c} >8.5%); and ischemic heart disease (proven myocardial infarction, existence of multiple lesions on thallium heart isotope screen, or evidence of coronary disease on coronary artery catheterization).

No patient underwent any neurosurgical procedure. All patients were treated similarly during hospitalization. Therapy was based on the preservation of essential life functions and the prevention of secondary medical complications. Intracranial pressure was treated with intravenous mannitol or furosemide.

Clinical evaluations of all patients were done according to the Glasgow Coma Scale (GCS) score on admission and after 48 hours, according to the Stroke Severity (SS) score, and according to the Barthel Index (BI) on admission and at 2 weeks, 3 months, and 6 months after the event. The SS score consisted of an examination of mental function (0 to 11 points); motor nerve function, including involuntary movements and cerebellar ataxia (0 to 11 points); cranial nerve function (0 to 11 points); sensation (0 to 6 points); and reflexes (0 to 5 points).

Location and volume of the hematoma were estimated by brain CT scan. The examination was made with the use of Elsint CT 2400 Elite with a matrix of 512. We calculated volume by measuring the areas of high absorption with a

tracing cursor in each cut demonstrating bleeding and multiplying by the cut thickness. The results were expressed in cubic centimeters. The blood volumes of the bottom and top pools were additionally calculated as a special segment.[4] [5] The location of the hematoma in thalamic hemorrhages was classified as midthalamic, thalamocapsular, paraventricular, posterior thalamic, and holothalamic bleedings. The classifications of putaminal hemorrhage included pure putaminal, putaminocapsular, putaminothalamic, putaminohemispheric, and putaminocapsulohemispheric bleedings. Lobar hematomas were classified as frontal, parietal, temporal, parieto-occipital, and huge lobar bleedings. Extension of blood into the ventricles was noted.

The data were analyzed with the χ^2 test, Pearson correlation test, and Student's *t* test. Multivariate analysis was calculated with the stepwise regression test. No significant exceptions were found.

Fifty-two patients (28 men and 24 women) with thalamic bleeding were included in the study (mean age, 66.6±10.6 years). The bleeding locations were as follows: 36 holothalamic or diffuse bleeding, 11 thalamocapsular, 8 midthalamic, 2 posterior thalamic, and one purely paraventricular thalamic bleeding.

The mortality rate was 36.6% (19 patients) during the 6-month follow-up period. Causes of patient mortality were as follows: direct result of the bleeding (10 patients), rebleeding (1 patient), late complications (8 patients), secondary infection (6 patients), and myocardial infarction (2 patients).

Eighty-seven patients (60 men and 27 women) were included (mean age, 69.9±13.6 years). The bleeding locations were as follows: 26 pure putaminal, 24 putaminocapsulohemispheric, 20 putaminocapsular, and 7 putaminothalamic.

Among the patients with fatal outcome, the putaminal location was as follows: putaminocapsulohemispheric (15 patients), putaminohemispheric (7 patients), and putaminothalamic (5 patients). No lethal outcome was observed in pure and putaminothalamic bleedings. The mortality rate after 6 months was 31% (27 patients). Causes of mortality were as follows: direct result of the bleeding (23 patients) and late complications or secondary infection with sepsis (4 patients).

One hundred forty patients (66 men and 74 women) with lobar hematomas were included in this study (mean age, 66.8±11.1 years). The locations were as follows: 49 frontal lobe, 42 parietal lobe, 17 temporal lobe, 9 parieto-occipital lobe, and 23 massive multilobar bleeding.

The mortality rate after 6 months was 52% (73 patients). All patients with massive multilobar hematomas had a fatal outcome. The causes of mortality were a direct result of the bleeding (60 patients) and late complications (13 patients), including intercurrent infectious disease (5 patients), myocardial infarction (4 patients), pulmonary embolus (1 patient), and pulmonary edema (1 patient). In 2 cases the cause could not be determined. (No postmortem examination was performed.)

In putaminal and thalamic bleedings, no significant correlation was found between age or sex and the BI or SS score (*P*=.29 to .45). In lobar hematomas, higher SS scores were found in men (*P*=.02). No sex or age dependence in the BI results was observed.

Risk factors found in this study were as follows: hypertension (165 patients; 59%), cigarette smoking (146 patients; 52%), diabetes mellitus (84 patients; 30%), ischemic heart disease (62 patients; 22%), and hyperlipidemia (47 patients; 18%).

No significant correlation was found between each of the vascular risk factors and the outcome. Hypertension and ischemic heart disease had a greater tendency toward a reciprocal correlation with poor outcome. Ischemic heart disease was found to have a limited reciprocal correlation with BI after 6 months only in patients with lobar hematomas (*P*=.053).

In thalamic hemorrhage, the mean value of hemorrhage volume was 16.2±12.0 cm^3 (range, 2.8 to 46 cm^3). In putaminal hemorrhage, the mean value of hemorrhage volume was 26.1±20.1 cm^3 (range, 3.1 to 61 cm^3). In lobar hematomas, the mean value of hemorrhage volume was 55.3±55.2 cm^3 (range, 2.7 to 239 cm^3) (frontal lobe, 36.6±22.8 cm^3; parietal lobe, 44.9±35.5 cm^3; temporal lobe, 16.2±13.2 cm^3; and parieto-occipital lobe, 16.4±8.2 cm^3).

Significant correlation was found between low GCS score or size of hematoma and lethal outcome (*P*<.001 for each) and between intraventricular

penetration and better prognosis for survival (*P*=.02), especially during the first 48 hours (*P*=.0007). The relative risks of probability of death in bleeding without intraventricular expansion versus bleeding with intraventricular expansion were 1.16 for blood volume of 7 cm^3 and 2.73 for blood volume of 25 cm^3.

In thalamic hemorrhage no correlation was found between GCS score, size of hematoma, or intraventricular expansion and SS score and BI in any stage of examination (*P*=.1 to .54).

A significant correlation was found between low GCS score or size of hematoma and lethal outcome in putaminal haemorrhage. A high significance for mortality was found in putaminocapsulothalamic bleeding. Intraventricular extension was not found to be a risk factor for poor outcome. The relative risks of probability of death, according to a GCS score of 5 versus 11, were 5.3 for a blood volume of 5 cm^3 and 1.0 for a blood volume of 25 cm^3.

A significant correlation was found between size of bleeding and BI or SS score in putaminothalamic, putaminocapsular, and putaminocapsulothalamic locations (*P*=.04, *P*=.02, and *P*=.02, respectively). A poor functional outcome was found in putaminocapsular and putaminothalamic bleedings (*P*=.044 and *P*=.051, respectively). No correlation was found between the expansion of blood into the ventricle and SS score or BI.

In lobar haemorrhage size of bleeding area (*P*<.0036), GCS score (*P*<.0001), and intraventricular extension were significantly correlated with

lethal outcome. The relative risks of probability of death, according to a GCS score of 5 versus 11, were 4.2 for a blood volume of 30 cm^3 and 1.7 for a blood volume of 100 cm^3.

Significant correlations were found between GCS score or size of bleeding on admission and SS score ($P<.001$ for each) or BI ($P<.05$ and $P<.001$, respectively). The correlation between intraventricular extension and SS score or BI was also significant ($P<.01$ for each). Temporal lobe bleeding was noted to have a somewhat better prognosis compared with other locations ($P<.052$)

MATERIALS AND METHODS

All patients with acute stroke admitted in the medical ward of Government General Hospital, Chennai from April 2009 to July 2009 were investigated for haemorrhagic stroke by obtaining a CT brain.

INCLUSION CRITERIA

Patients with non traumatic intraparenchymal haemorrhage admitted to medical wards.

EXCLUSION CRITERIA

1. Patients with history of Trauma
2. Patients with suh arachnoid haemorrhage alone
3. Patients with intraventricular haemorrhage alone
4. Patients with previous history of intraparenchymal haemorrhage

The following data were collected from the patients.

1. Age
2. Temperature in °F at admission
3. Mean arterial pressure. This is calculated by:
    a. Mean arterial pressure = Diastolic pressure + 1/3 pulse pressure

4. Glassgow coma score at admission to assess the severity of the bleed. CT brain was done on the day of admission and the following data were collected

5. Volume of Bleed

6. Location of Bleed

7. Presence / Absence of Intraventricular haemorrhage.

Volume of bleed was calculated using the formula used by Lisch and colleagues and Kothari and colleagues.

i.e Formula for ellipsoid = ABC / 2. A,B,C are the dimensions.

CT slices taken in our Government General Hospital are calibrated from 10 mm / 1 cm thickness. The slice with largest haemorrhage was found out and the largest diameter measured and is taken as 'A'. The diameter perpendicular to 'A' is taken as 'B' and 'C' is calculated by multiplying the number of haemorrhagic slices (n) and thickness of slice (1 cm). n was calculated by adding:

1 for haemorrhage area of a slice >75% of the largest haemorrhage area

0.5 for haemorrhage area of a slice 25 – 75% of the largest haemorrhage area.

Haemorrhage area of a slice <25% is not included. For example:

n = slice (1) + slice (2) + slice (3)

= 1 + 0.5 + 0

n = 1.5

C = n x slice thickness

C = 2.5 x 1

C = 2.5 cm

By this method, volume of haemorrhage was calculated for all patients included in the study.

100 patients were studied similarly to know the outcome at 30 days. They were treated with the same regimen in our hospital. 30 days outcome i.e. the mortality or the morbidity status using the NIHSS score was done at our review OP.

RESULTS AND OBSERVATIONS

Total No. of cases studied	=	100
No. of deaths in 30 days	=	51
No. of patients survived after 30 days	=	49
No. of male cases	=	72
No. of female cases	=	28
No. of Hypertensive cases	=	32
No. of non-hypertensive cases	=	68

ANALYSIS OF OBSERVATIONS

TABLE – 1

AGE AS A PROGNOSTIC PREDICTOR OF IPH

Age group	No. of patients	No. of deaths	Mortality rate (%)
0 – 10	0	0	0
11 – 20	1	1	100
21 – 30	0	0	0
31 – 40	7	4	57.14
41 – 50	16	4	25
51 – 60	34	14	41.17
61 – 70	23	14	60.86
71 – 80	16	12	75
81 – 90	3	2	66.66
91 – 100	0	0	0
Total	100	51	

- The most frequent age group affected is 51 – 60 years in wide range 41 – 70 years.
- Mortality is high in extremes of age groups, particularly older age group.
- There is no persistent relation between the age and the mortality.
- This may be due to diverse aetiology/coexistent morbidities
- So, age cannot serve as a definite prognostic predictor.

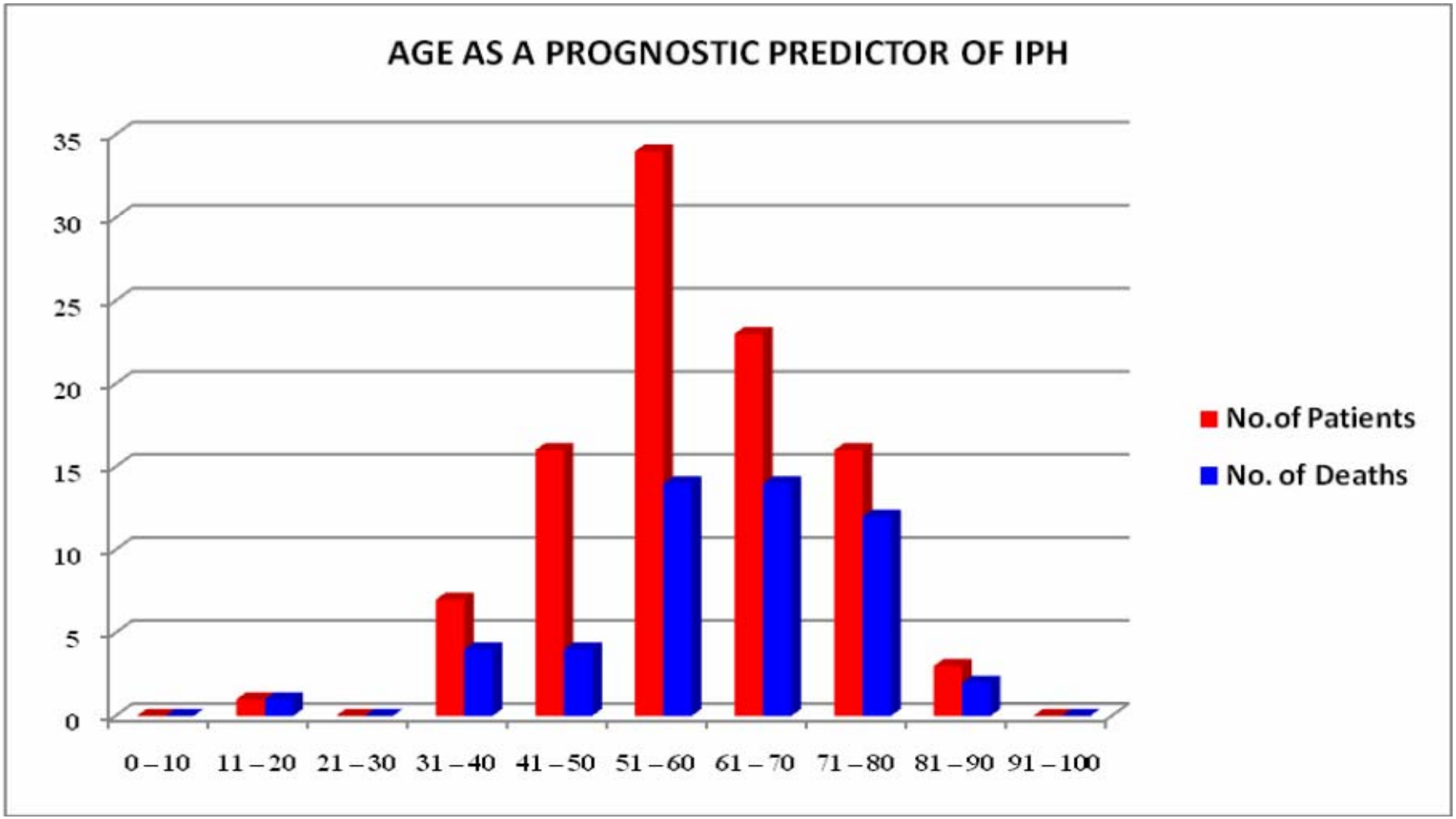

AGE AS A PROGNOSTIC PREDICTOR OF IPH
35
30
25
20
15
10
5
0
0 – 10
11 – 20
21 – 30
31 – 40
41 – 50
51 – 60
61 – 70
71 – 80
81 – 90
91 – 100
No.of Patients
No. of Deaths

TABLE – 2

GENDER AS A PROGNOSTIC PREDICTOR OF IPH

Patient Type	No. of patients	No. of deaths	Mortality rate (%)
MALES	72	35	48.61
FEMALES	28	16	57.14
TOTAL	100	51	

- Incidence of intraparenchymal heamorrhage is higher in males.
- Male : Female ≈ 3 : 1.
- But the mortality rate is higher in the females.

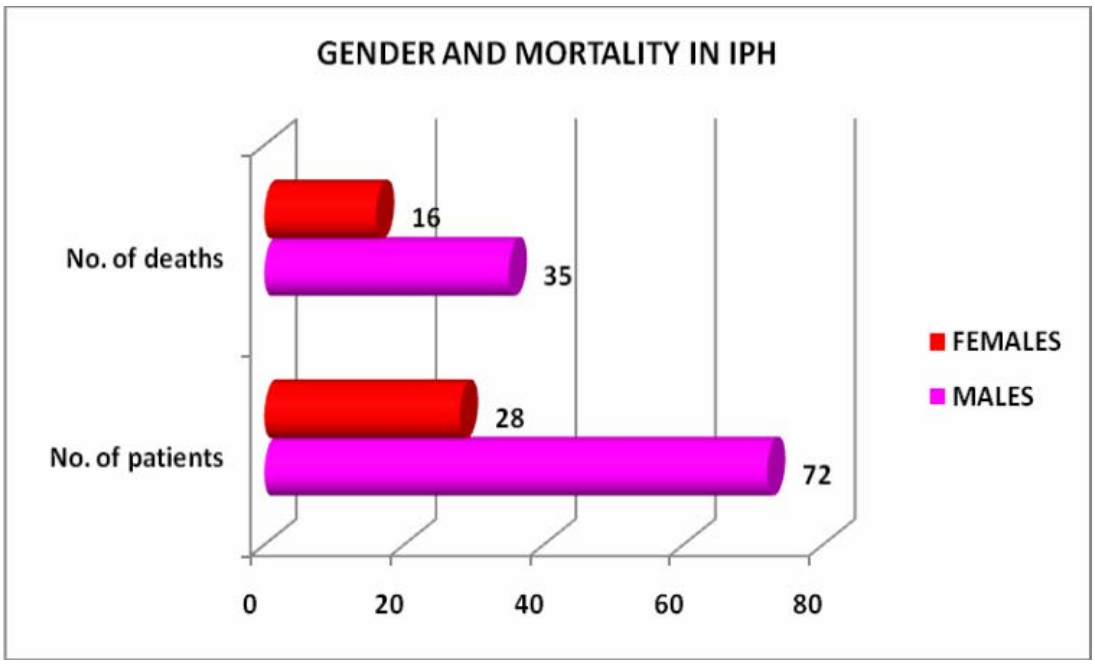


TABLE – 3

TEMPERATURE AS A PROGNOSTIC PREDICTOR OF IPH

Temperature	No. of patients	No. of deaths	Mortality Rate (%)
Temperature >103°F	15	10	66.66
Temperature ≤103°F	85	41	48.23
Total	100	51	

- Patients with brainstem bleed had temperature more than 103°F.
- Temperature cannot serve as a prognostic predictor as the values were not applicable for bleed in all locations.

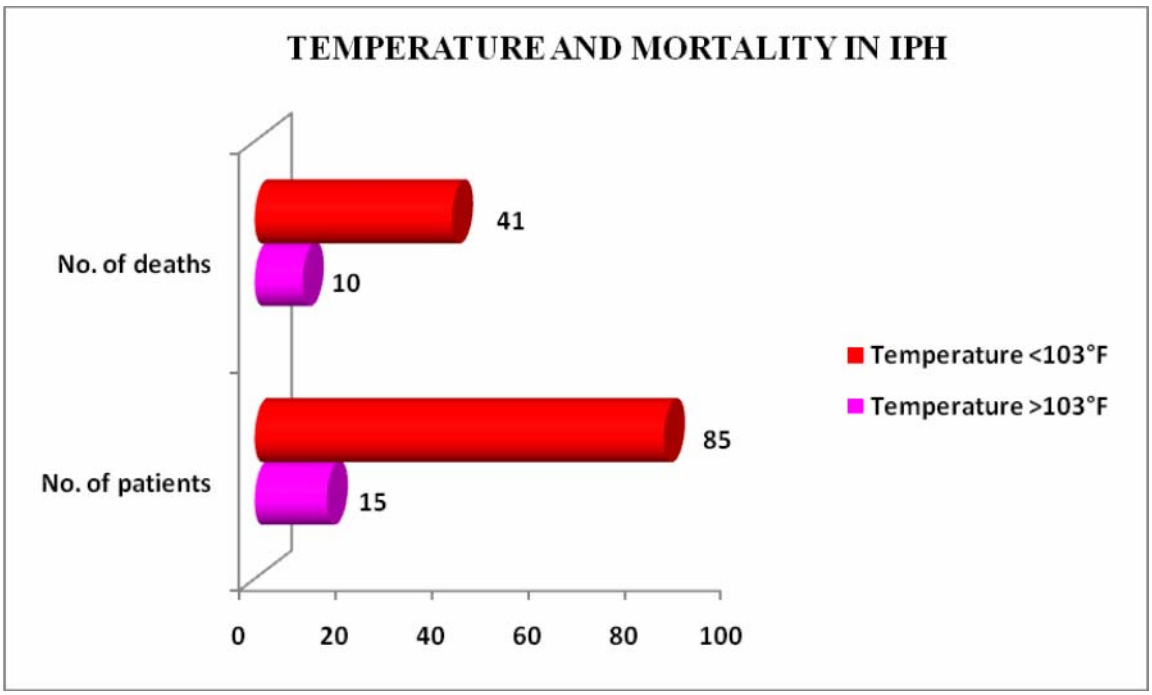


TABLE – 4

HYPERTENSION AS A PROGNOSTIC PREDICTOR OF IPH

Type of Patient	No. of patients	No. of deaths	Mortality rate (%)
Hypertension	**32**	**10**	**31.25**
Non-Hypertension	**68**	**41**	**60.29**
Total	**100**	**51**	

- Incidence of IPH is high in patients who are not known hypertensives than in known hypertensives.
- Mortality is higher in non-hypertensive IPH.
- This may be explained by:
    i. They are hypertensives not detected earlier.
    ii. Adaptation of the cerebral vessels to the increased pressure in known hypertensives.

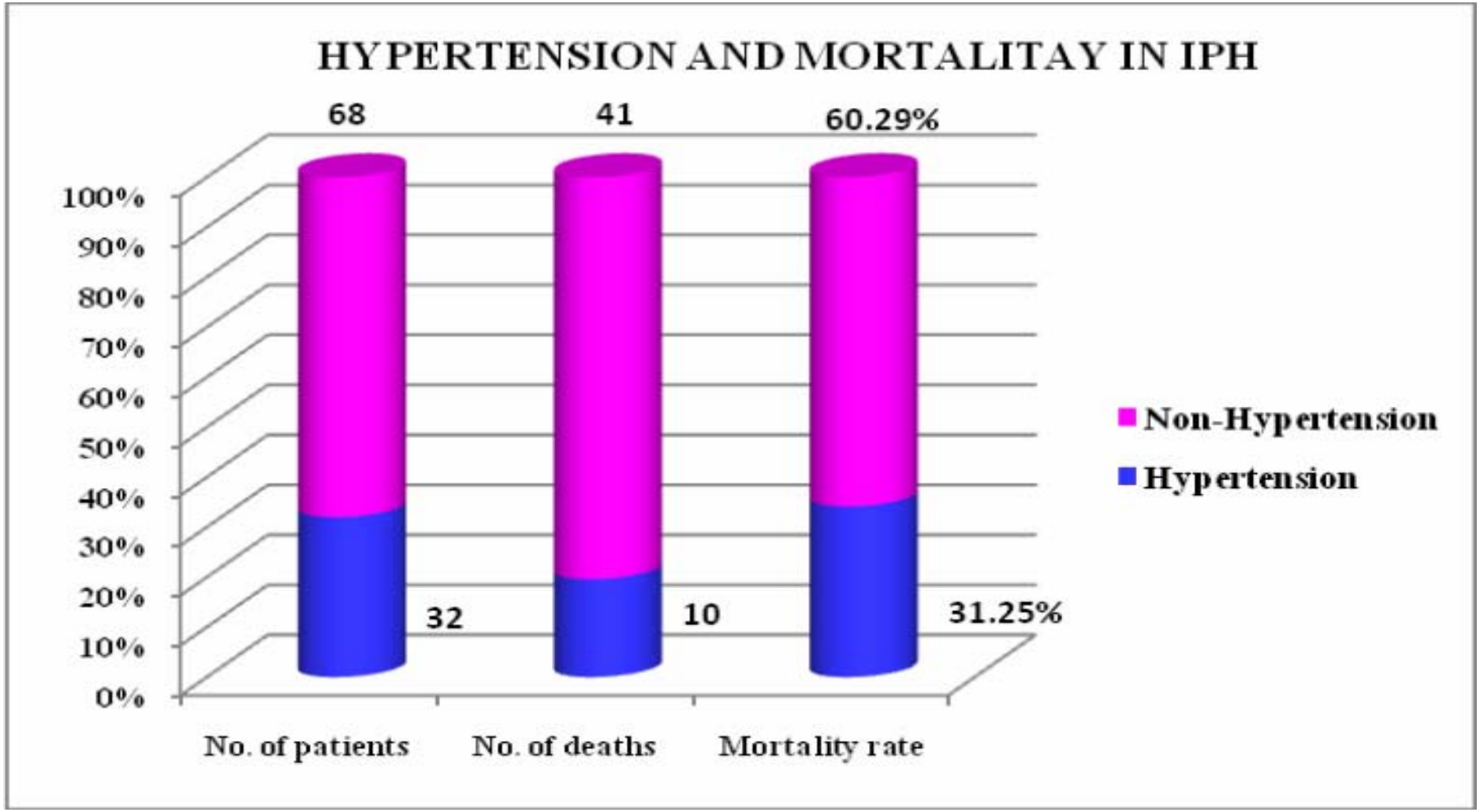

HYPERTENSION AND MORTALITAY IN IPH
68
41
60.29%
100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
32
10
31.25%
No. of patients
No. of deaths
Mortality rate
Non-Hypertension
Hypertension

TABLE – 5

MAP AS A PROGNOSTIC PREDICTOR OF IPH

Type of Patient	No. of patients	No. of deaths	Mortality rate (%)
MAP ≥145	26	21	80.76
MAP <145	74	30	40.54
Total	100	51	

- The mortality rate in patients with MAP ≥145 is almost double that of patients with MAP <145.
- So, MAP serves as an important prognostic predictor of 30 day outcome in non-traumatic intraparenchymal haemorrhage patients.

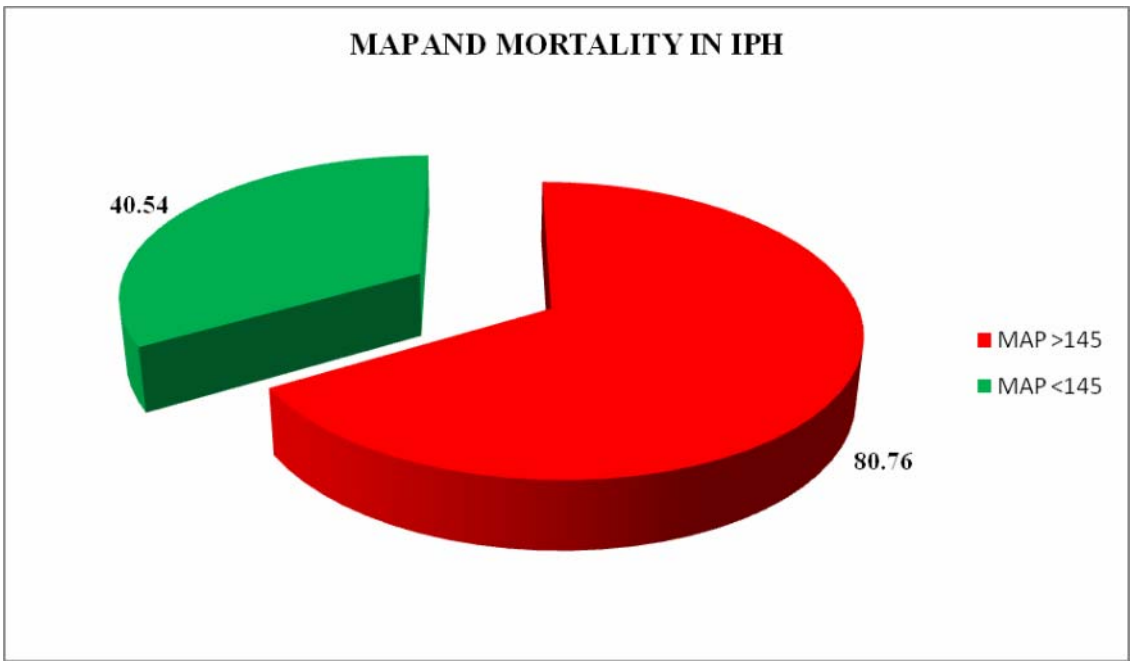


TABLE – 6

GCS AS A PROGNOSTIC PREDICTOR OF IPH

Type of Patient	No. of patients	No. of deaths	Mortality rate (%)
GCS ≤8	57	49	85.96
GCS >8	43	2	4.65
Total	100	51	

- Higher the GCS score, higher is the survival rate.
- GCS at admission also correlates with the morbidity rate.
- Patients with ↑ GCS score has lesser morbidity i.e. NIHSS <6/42.
- So, GCS can serve as **an important predictor** of both mortality and morbidity of non traumatic IPH.

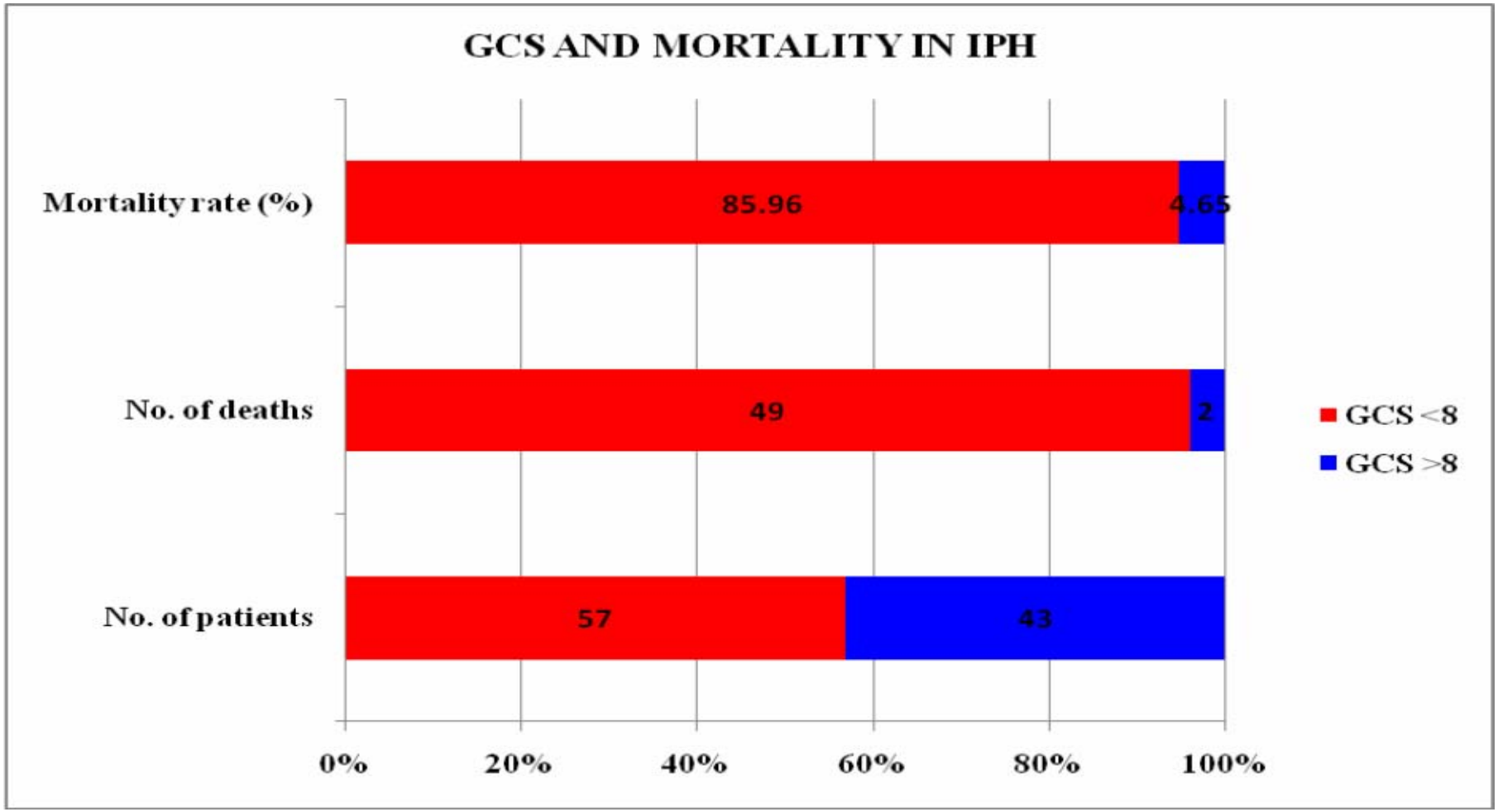

GCS AND MORTALITY IN IPH
Mortality rate (%)
No. of deaths
No. of patients
85.96
4.65
49
2
57
43
GCS <8
GCS >8
0%
20%
40%
60%
80%
100%

TABLE – 7

VOLUME OF BLEED AS A PROGNOSTIC PREDICTOR OF IPH

Type of Patient	No. of patients	No. of deaths	Mortality rate (%)
Volume <30 ml	51	10	19.6
Volume 30 - 60 ml	37	30	81.08
Volume >60 ml	12	11	91.66
Total	100	51	

- Mortality rate is less when the bleed is less than 30 ml.
- Mortality rate for bleed more than 60 ml and 30 – 60 ml is more or less nearer.
- Bleed less than 30 ml has a good prognosis and more than 30 ml has a bad prognosis.
- So, volume of bleed is an important prognostic predictor of non-traumatic IPH.

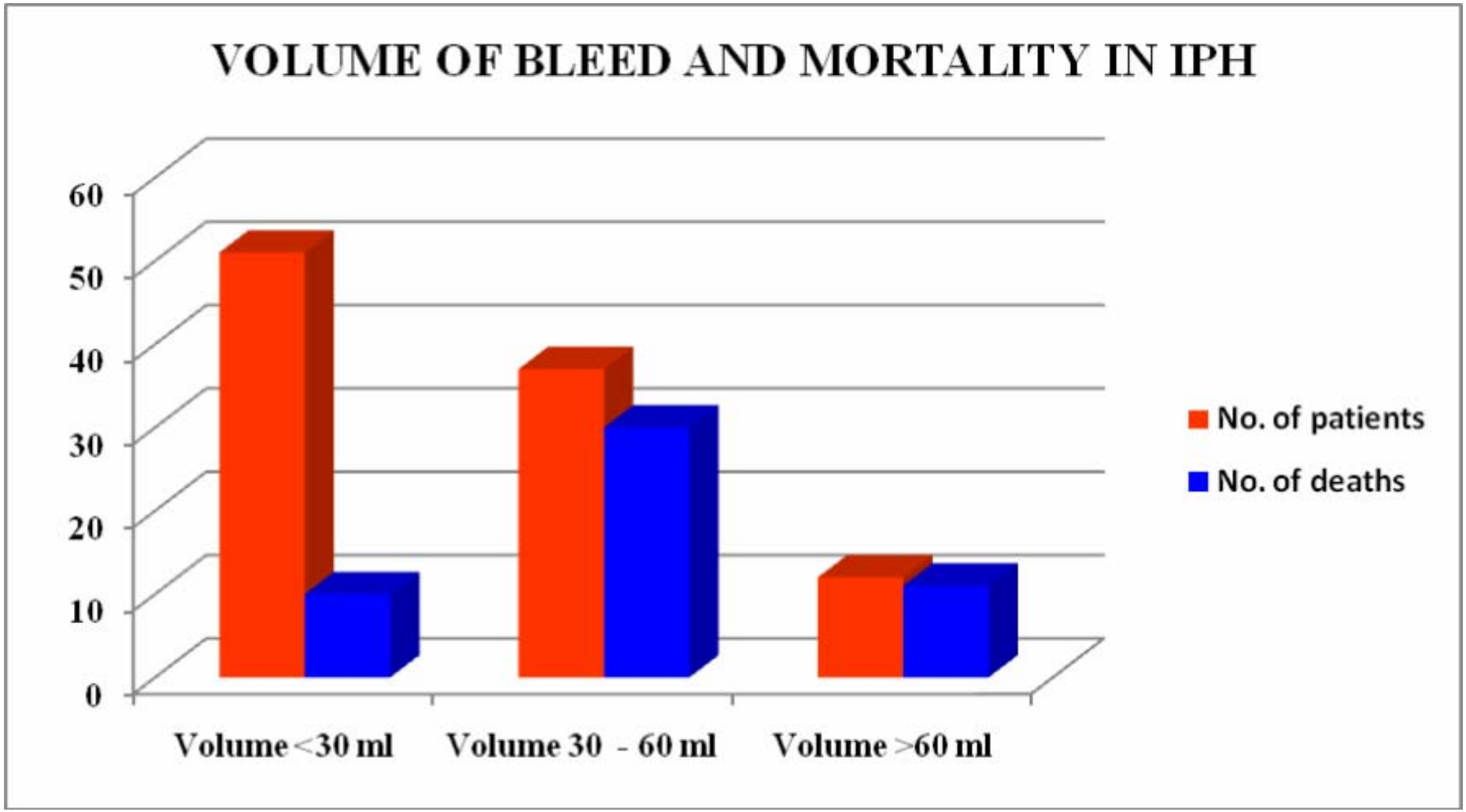
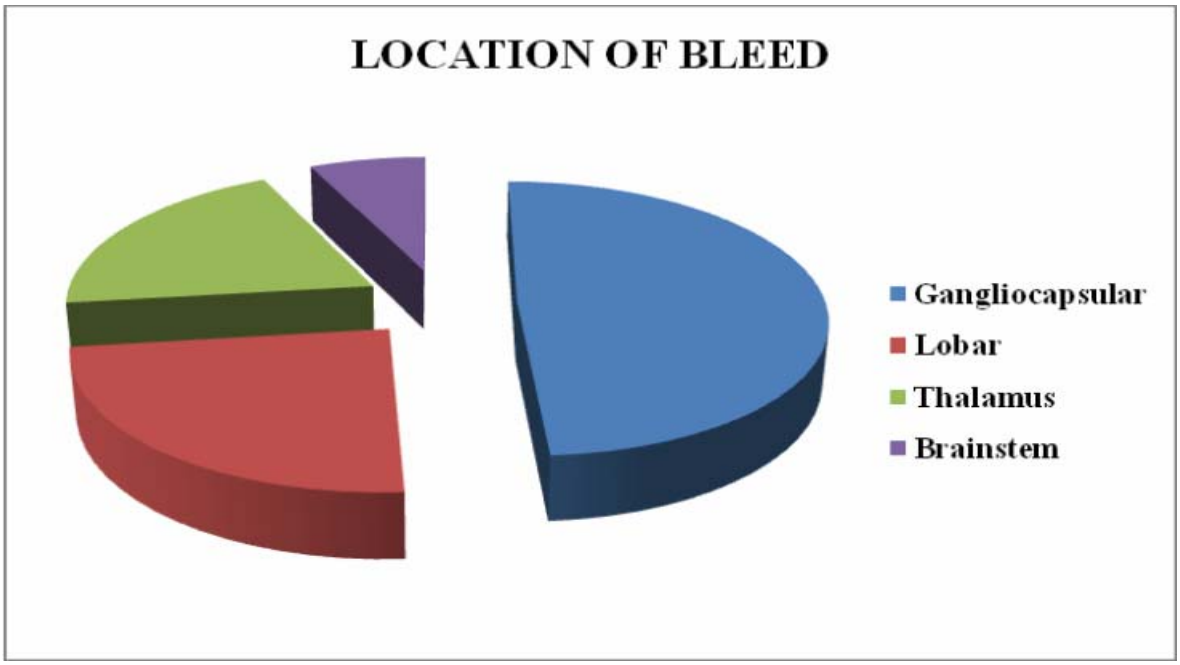

VOLUME OF BLEED AND MORTALITY IN IPH
60
50
40
30
20
10
0
Volume <30 ml
Volume 30 - 60 ml
Volume >60 ml
No. of patients
No. of deaths

TABLE – 8

LOCATION OF BLEED AS A PROGNOSTIC PREDICTOR OF IPH

Type of Patient	No. of patients	No. of deaths	Mortality rate (%)
Gangliocapsular	49	23	46.94
Lobar	24	12	50
Thalamus	20	10	50
Brainstem	7	6	85.71
Total	100	51	

- Basal ganglia is the most common site of bled followed by the lobar, thalamus and brainstem.
- Mortality is high with brainstem haemorrhage.


LOCATION OF BLEED
Gangliocapsular
Lobar
Thalamus
Brainstem

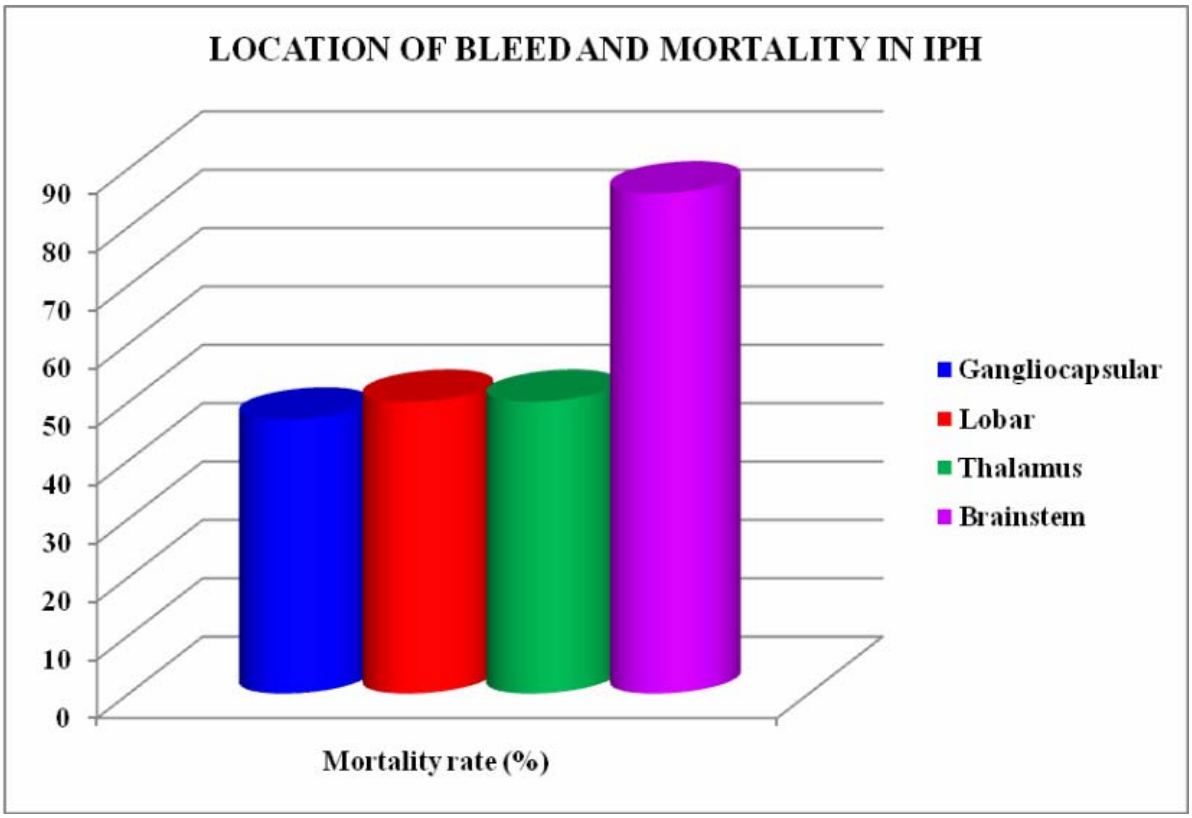

LOCATION OF BLEED AND MORTALITY IN IPH
90
80
70
60
50
40
30
20
10
0
Mortality rate (%)
Gangliocapsular
Lobar
Thalamus
Brainstem

TABLE – 9

IVH AS A PROGNOSTIC PREDICTOR OF IPH

Type of Patient	No. of patients	No. of deaths	Mortality rate (%)
IVH (+)	48	40	83.33
IVH (-)	52	11	21.15
Total	100	51	

- Presence of intraventricular haemorrhage carries a poor prognosis.

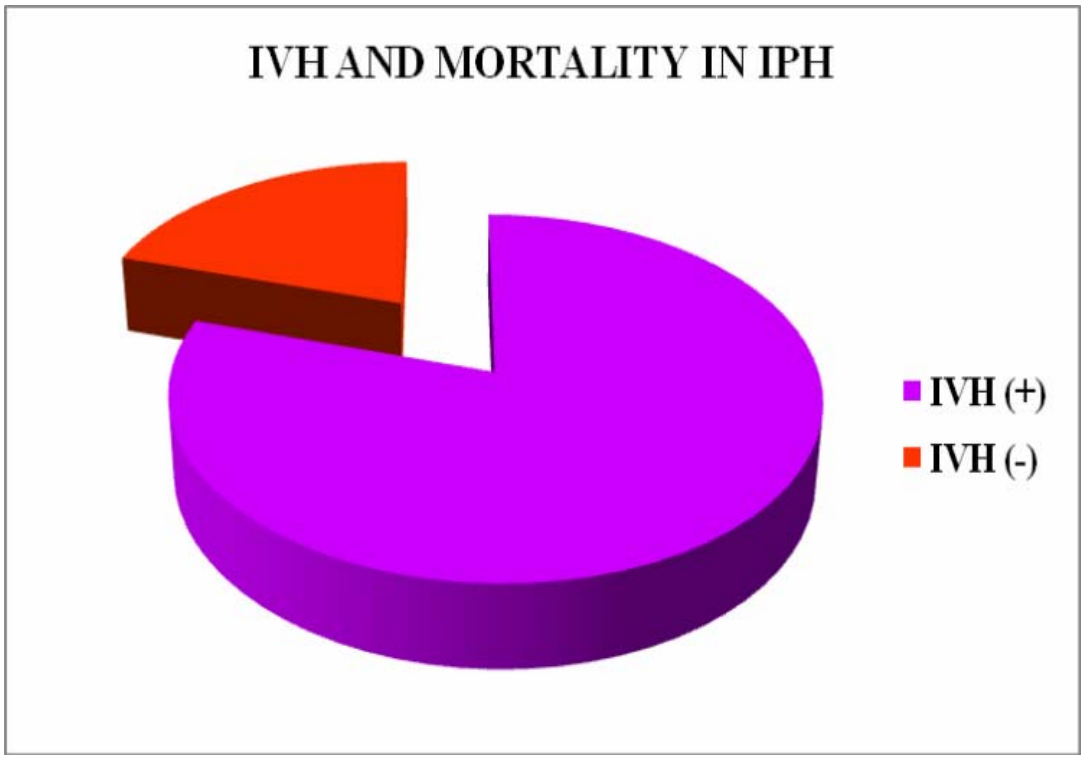


TABLE – 10

RELATION BETWEEN GCS AND NIHSS SCORE AT 30 DAYS

Type of patient	No. of patients alive	Patients with NIHSS ≤6	Patients with NIHSS >6	Morbidity Rate (%)
GCS ≤8	8	0	8	100
GCS >8	41	35	6	15
Total	49	35	14	

- GCS is directly related to decreased NIHSS score.
- Higher the GCS, lower the NIHSS score.

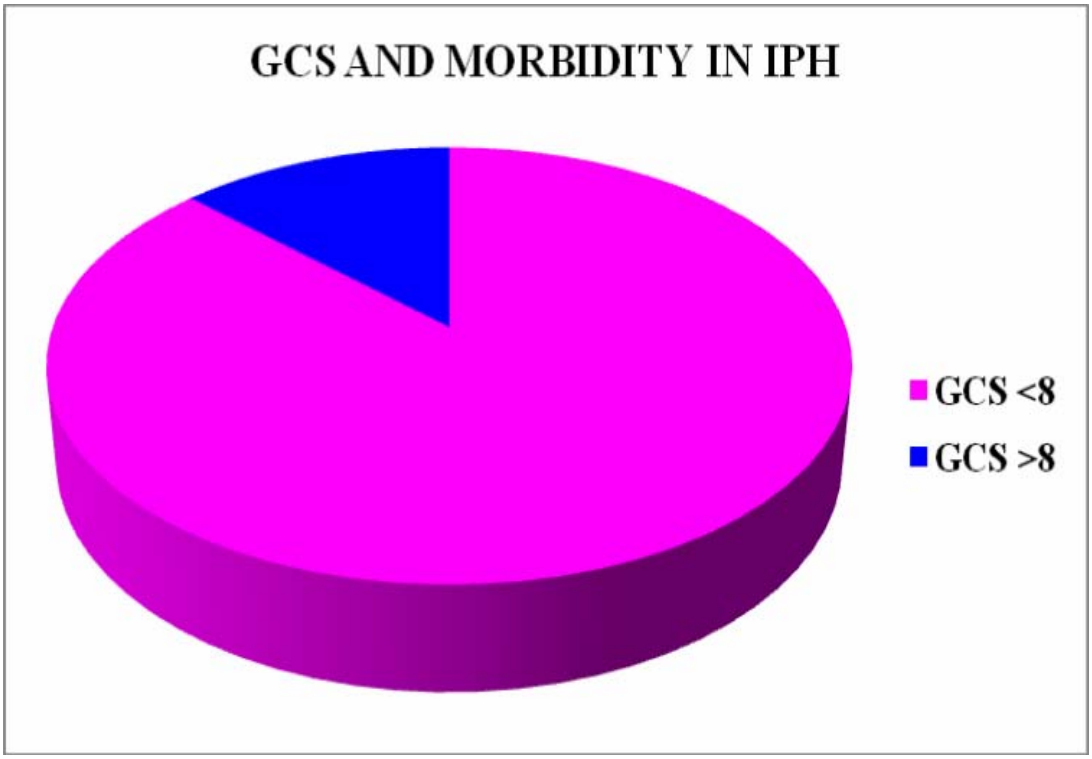


TABLE – 11

PROGNOSTIC PREDICTORS OF 30 DAY MORTALITY IN NON-TRAUMATIC IPH

FACTOR	NUMBER OF PATIENTS	NUMBER OF DEATHS	MORTALITY %
MAP ≥ 145	**26**	**21**	**80.76**
GCS ≤ 8	**57**	**49**	**85.96**
Volume > 60ml	**12**	**11**	**91.66**
IVH (+)	**48**	**40**	**83.33**

The prognosis predictors of 30 day mortality in non-traumatic IPH patients in order of importance according to this study are

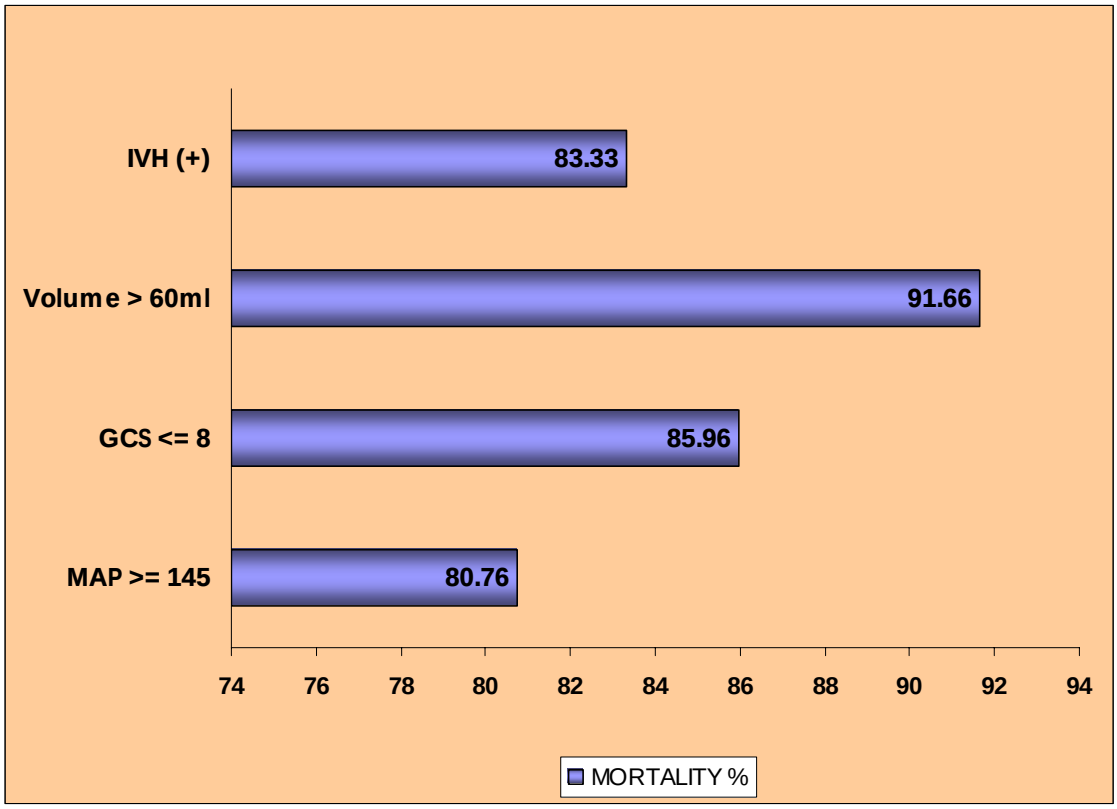
i) Volume > 60ml

ii) GCS ≤ 8

iii) Presence of IVH

iv) MAP ≥145mm of Hg.

PROGNOSTIC PREDICTORS OF 30 DAY MORTALITY IN NON-TRAUMATIC IPH



DISCUSSION

MORTALITY RATE:

100 patients were included in this study and 51 patients died at the end of 30 days. The 30 day mortality rate was 51% compared to literature which varies from 20 to 60%.

AGE:

Intraparenchymal haemarrhage was common in the 50-70yrs age group and death was also common in this age group. But Age as such was no found to have direct correlation with 30 day mortality.

GENDER:

In our study, IPH was common in males and it was found that a male has a 3 times rish of developing IPH. But the mortality rate was high in females. Again this was not a significant prognostic factor in determining 30 day mortality.

TEMPERATURE:

Only 15 patients out of 100 patients had temperature above 103F and out of them 100 patients died – 8 of them where of Brainstem Bleed. So, temperature cannot be applied as a significant prognostic factor in determining 30 day mortality for IPH in all locations.

HYPERTENSION:

In 100 patients, 32 patients were known hypertensives and the mortality rate was half in the patients compared to the non hypertensives. This may be either due to the reason that they are undiagnosed hypertensives or due to the difference in the pathogenesis of a acute rise in BP and a chronic rise in BP. Further studies regarding the endothelial damage in these patients may clarify these queries.

MAP:

Among the 51 dead patients 21 had a MAP of more than 145mm Hg. MAP is found to be a more significant predictor than the systolic or diastolic BP.

GCS:

Out of 51 dead patients nearly 49 patients had a GCS score < 8 at admission. GCS has got a very strong correlation over the 30 day mortality than any other factor.

VOLUIME OF BLEED:

Though a difference is made as mild, moderate and massive bleed, there was no significant mortality difference between moderate and massive bleed. This may be changed by increased availability if neurosurgical care to some of these patients and the mortality can come down. But there was a significant reduction in the mortality in patients with bleed < 30ml.

LOCATION OF BLEED:

Basal ganglia was the common site of Bleed. Nearly 50% cases were Basal ganglia Bleed. 25% Lobar Bleed,, 20% Thalamic Bleed and 5% Brainstem bleed. According to literature also this was the approximate incidence. In this study mortality was higher in Brainstem bleed similar to literature but the thalamic bleed did not show a better prognosis as literature. The mortality rates of Thalamic Gangliocapsular and Lobar bleed were similar.

IVH:

In literature, Thalamic Bleed with IVH has got a good prognosis compared to lobar bleed with IVH. In my study, presence of IVH in any bleed carried a poor diagnosis.

GCS and NIHSS:

GCS is the single most important factor in predicting both the mortality and morbidity. Patient admitted with high GCS had a low NIHSS score and low GCS had a high NIHSS score.

ADDITIONAL DATA FROM THE STUDY

1. Left side bleed is more common than right side.
2. Diabetes Mellitus is not a common co-morbid condition with intracerebral bleed.
3. Patients who survive the acute stroke in haemorrhage do well than the infarct patients.
4. The common age group is 40 – 60 years.
5. Incidence of ICH is high in males but the mortality is high in females.

CONCLUSION

- Age, Gender, Temperature do not have a significant role in detecting the 30 day mortality rate in patients with IPH.
- i) Volume of Bleed

  ii) GCS at admission

  iii) Presence of IVH

  iv) Mean Arterial Pressure

  have a significant role in detecting the 30 day mortality in IPH patients.
- Location of bleed at sites other than Brainstem did not have a difference in mortality in this study. Studies with larger number of patients with different locations of Bleed may throw some light on this aspect
- A case of stroke with ICH with MAP < 145mm Hg, GCS > 8, Volume of Bleed < 30ml, in the absence of IVH has 80% chance of survival after 30 days with the best NIHSS score.
- Early detection of HT and prompt management can prevent the occurrence of event.

ABBREVIATIONS

IPH – Intraparenchymal Haemarrhage

IVH – Intraventricular Haemarrhage

GCS- Glasgow Coma Scale

NIHSS – National Institute of Health System Score

BP – Blood Pressure

MAP – Mean Arterial Pressure

SAH – Sub Arachnoid Haemorrhage

ICH – Intracerebrak Haemorrhage

CSF – CerebroSpinal Fluid

CT – Computed Tomography

MRI – Magentic Resonance Imaging

HT – Hypertension

PROGNOSTIC PREDICTORS OF 30 DAY OUTCOME IN PATIENTS WITH NON TRAUMATIC INTRAPARENCHYMAL HAEMORRHAGE

PROFORMA

S. No : **IP No:**

NAME : **AGE:**
SEX: M / F

DATE OF ADMISSION: **DATE OF DISCHARGE:**
WARD/UNIT:

ADDRESS: **OCCUPATION:**

CONTACT NUMBER:

ADMISSION DIAGNOSIS:

BRIEF HISTORY:

COMORBID ILLNESS: DM / HT / IHD / BA / EPILEPSY

PERSONAL HISTORY: Smoker / Alcohol / Betel leaf

PHYSICAL EXAMINATION

VITAL SIGNS

PR : **BP :** **MAP= DP + (1/3)PP =** **TEMP:**

CENTRAL NERVOUS SYSTEM : GCS SCORE / 15

EYE OPENING		VERBAL RESPONSE		MOTOR RESPONSE	
NEVER	1	NO RESPONSE	1	NO RESPONSE	1
TO PAIN	2	INCOMPREHENSIBLE SOUNDS	2	EXTENSOR RESPONSE TO PAINS	2
TO VERBAL STIMULI	3	INAPPROPRIATE WORDS	3	FLEXOR RESPONSE TO PAINS	3
SPONTANEOUSLY	4	DISORIENTED CONVERSES	4	FLEXOR WITHDRAWAL	4
		ORIENTED AND CONVERSES	5	LOCALISES PAIN	5
				OBEYS COMMANDS	6

CVS : **RS :**

ABD :

INVESTIGATIONS

CBC

TC :

DC :

Hb % :

PCV :

ESR :

PLATELETS :

RFT

BLOOD SUGAR :

UREA :

SERUM CREATININE :

Na+

K+ :

LFT

TOTAL BILIRUBIN

AST :

ALT :

SAP :

FASTING LIPID PROFILE

TOTAL CHOLESTEROL:

TGL :

HDL :

LDL :

TOTAL PROTEIN :

BT :

CT :

ECG :

CHEST XRAY PA view :

ECHO : (If done)

CT BRAIN

SITE OF H'AGE :

DIMENSIONS OF H'AGE : a -

b -

c -

volume of ICH - abc/2 =

INTRAVENTRICULAR EXTN : + / -

MIDLINE SHIFT : + / -

OUTCOME OF THE PATIENT : ALIVE / EXPIRED

DATE OF DISCHARGE / DEATH :

ASSESSMENT OF 30 DAY OUTCOME BY NIHSS

ITEM	NAME	RESPONSE	SCORE	
1a	Level of Consciousness	Alert	0	
		Not Alert – responding to minimal stimuli	1	
		Not Alert – responding to repeated stimuli	2	
		Unresponsive	3	
1b	LOC Questions	Answers both correctly	0	
		Answers one correctly	1	
		Answers neither	2	
1c	LOC Commands	Performs both tasks correctly	0	
		Performs one task correctly	1	
		Performs neither	2	
2	Gaze	Normal	0	
		Partial gaze palsy	1	
		Total gaze palsy	2	
3	Visual Fields	No visual loss	0	
		Partial hemianopia	1	
		Complete hemianopia	2	
4	Facial Palsy	Normal	0	
		Minor Paralysis	1	
		Partial Paralysis	2	
		Complete Paralysis	3	
5a,5b	Motor Arm	ARM	R	L
		No drift	0	0
		Drift before 10 sec	1	1

		Fall before 10 sec	**2**	**2**
		No drift against gravity	**3**	**3**
		No movement	**4**	**4**
6a,6b	**Motor Leg**	**LEG**	**R**	**L**
		No drift	**0**	**0**
		Drift before 5 sec	**1**	**1**
		Fall before 5 sec	**2**	**2**
		No drift against gravity	**3**	**3**
		No movement	**4**	**4**
7	**Ataxia**	**Absent**	**0**	
		One limb	**1**	
		Two limbs	**2**	
8	**Sensory**	**Normal**	**0**	
		Mild loss	**1**	
		Severe loss	**2**	
9	**Language**	**Normal**	**0**	
		Mild aphasia	**1**	
		Sever aphasia	**2**	
		Mute/Global aphasia	**3**	
10	**Dysarthria**	**Normal**	**0**	
		Mild	**1**	
		Severe	**2**	
11	**Extinction/inattention**	**Normal**	**0**	
		Mild	**1**	
		Sever	**2**	
		TOTAL		

TOTAL NIHSS SCORE : _____ / 42

MASTER CHART

S.No.	Name	IP No.	Age	Sex	Comorbid Illness	BP	MAP	Temp °F	GCS (Out of 15	Site of Bleed	Volume	IVH	SAH	Midline Shift	Hydrocephalus	NIHSS Score/42	30 days outcome Alive/Expired
1	George	41767	60	M	HT	190/100	130	99	10	R Ganglio capsular	20	-	-	-	-	10	ALIVE
2	Vasantha	43851	40	F	-	160/100	120	98.6	10	R Ganglio capsular	14	-	-	+	-	11	ALIVE
3	Arulsamy	43925	50	M	-	200/110	140	99	7	L Ganglio capsular	40	-	-	+	-	-	EXPIRED
4	Veerappan	44819	35	M	HT	200/100	133	98.6	5	Brainstem	30	+	-	+	-	-	EXPIRED
5	Ramachandran	45424	75	M	CAD	200/100	133	99.2	6	L Ganglio capsular	37.5	-	-	+	-	-	EXPIRED
6	Karunanidhi	46101	50	M	HT	180/100	126	98.2	9	L Ganglio capsular	8	-	-	-	-	10	ALIVE
7	Ramayee	46912	40	F	HT	170/100	123	98.4	10	L Thalamus	10	-	-	-	-	12	ALIVE
8	Kattan	47101	55	M	-	190/100	130	98.6	8	L Parietal lobe	6	-	-	-	-	10	ALIVE
9	Selvam	47504	70	M	DM/HT/DVT	150/70	97	99.4	15	L Parietal lobe	36	-	-	-	-	-	EXPIRED
10	Samsudeen	47580	65	M	-	220/140	166	99.6	12	R Parieto occipital	23	-	-	+	-	6	ALIVE
11	Chinnaiya	44071	65	M	-	220/110	140	98.6	10	L Ganglio capsular	14	-	-	-	-	10	ALIVE
12	Francis	48289	43	M	-	200/100	133	98.6	8	L Ganglio capsular	14	-	-	+	-	9	ALIVE
13	Ravi	49580	35	M	HT	220/140	166	99	15	L Temporal Lobe	10	-	-	-	-	0	ALIVE
14	Mohana	49587	60	F	HT/CAD	220/140	166	99.6	7	R Thalamus	32	+	-	+	-	-	EXPIRED
15	Thiruvengadam	49524	70	M	HT	200/110	140	98.2	10	R Ganglio capsular	24	-	-	+	-	7	ALIVE
16	Saroja	50039	76	F	-	160/100	120	98.6	8	R Ganglio capsular	8	-	-	-	-	9	ALIVE
17	Chali	50233	55	M	-	200/130	153	100.4	4	Brainstem	25	+	-	+	-	-	EXPIRED
18	Sainathprasad	50108	64	M	HT/CKD	200/120	146	98.2	5	L Ganglio capsular	64	+	-	+	-	-	EXPIRED
19	Anbarasan	50709	48	M	-	180/100	126	98.2	9	Brainstem Cerebellum	18	-	-	-	-	6	ALIVE
20	Palani	50850	53	M	-	210/110	143	100	5	R Ganglio capsular	40	+	-	+	-	-	EXPIRED
21	Ganesh Babu	50943	33	M	HT	200/100	140	100	5	L Ganglio capsular	50	+	-	+	-	-	EXPIRED
22	Neelavathy	50926	65	F	HT	250/110	153	104	3	L Thalamus	44	+	-	+	+	-	EXPIRED
23	Ganapathy	51250	55	M	-	220/120	153	98.4	5	R Ganglio capsular	62	-	-	+	-	-	EXPIRED
24	Kasthuri	51482	50	F	HT	190/100	130	100	4	L Ganglio capsular	62	+	-	+	-	-	EXPIRED
25	Ramadurai	52101	40	M	HT	170/100	123	98.2	15	R Parietal lobe	4	-	-	-	-	0	ALIVE
26	Kannan	52098	56	M	HT	150/100	116	98.6	10	R Ganglio capsular	6	-	-	-	-	10	ALIVE
27	Saravanan	52916	55	M	HT	190/100	130	98.6	10	L Ganglio capsular	6	-	-	-	-	6	ALIVE
28	Munusamy	53250	80	M	-	160/80	113	100	5	L Thalamus	46	+	-	+	+	-	EXPIRED
29	Munusamy	53302	72	M	-	180/100	133	100	3	R Parieto occipital	15	-	+	-	-	-	EXPIRED
30	Ramanaiya	53363	50	M	-	160/90	113	98.4	4	L Thalamus	62	+	+	+	+	-	EXPIRED
31	Thirumalai	53554	45	M	-	200/110	140	98.6	9	R Ganglio capsular	10	-	-	+	-	3	ALIVE
32	Prabhu	53984	50	M	HT	200/100	133	98.4	11	R Ganglio capsular	8	-	-	+	-	4	ALIVE
33	Loganathan	54174	69	M	DM/HT/OLD CVA INF	180/100	123	99.8	3	L Ganglio capsular	64	+	-	+	-	-	EXPIRED
34	Kalyani	54516	50	F	HT	220/130	160	99	15	L Parietal lobe	6	-	-	-	-	0	ALIVE
35	Mohamed Anwar	54860	62	M	HT	150/90	110	98	3	L Fronto temporo parietal	64	+	-	+	+	-	EXPIRED
36	Valli	55303	50	F	HT	190/80	113	98.8	7	L Ganglio capsular	45	+	-	+	-	-	EXPIRED

S.No.	Name	IP No.	Age	Sex	Comorbid Illness	BP	MAP	Temp °F	GCS (Out of 15	Site of Bleed	Volume	IVH	SAH	Midline Shift	Hydrocephalus	NIHSS Score/42	30 days outcome Alive/Expired
37	Bharathy	55405	60	F	-	170/100	123	98.8	3	L Ganglio capsular	40	-	-	+	-	-	EXPIRED
38	Vasudevan	55415	62	M	-	200/100	133	98.6	8	L Ganglio capsular	38	+	-	+	-	11	ALIVE
39	Ananda Rao	55825	50	M	HT	180/100	123	98.6	9	R Parietal lobe	40	+	-	+	-	10	ALIVE
40	Haneef	55865	65	M	-	220/100	140	100	5	L Ganglio capsular	38	-	-	+	-	-	EXPIRED
41	Andal	56206	75	F	-	200/140	160	100	3	R Thalamus	50	+	-	+	-	-	EXPIRED
42	Ponnusamy	56601	40	M	HT	200/100	133	98.6	9	L Thalamus	32	+	-	+	-	14	ALIVE
43	Palani	57297	65	M	-	180/110	133	98.6	4	R Ganglio capsular	52	+	-	+	+	-	EXPIRED
44	Chidambaram	56775	51	M	DM	200/110	140	100	4	R Ganglio capsular	50	-	-	+	-	-	EXPIRED
45	Anaresan	55275	67	M	HT	210/100	136	98.6	3	R Fronto parietal	36	+	-	+	-	-	EXPIRED
46	Jegadeesan	57247	70	M	-	200/100	133	100	7	L Ganglio capsular	60	+	-	+	-	-	EXPIRED
47	Ponnammal	58251	63	F	-	230/130	163	99	4	Brainstem	16	+	-	-	-	-	EXPIRED
48	Munusamy	59044	33	M	-	70/40	50	97	3	L Ganglio capsular	50	+	+	+	+	-	EXPIRED
49	Dhayalan	59207	41	M	-	200/100	133	99	10	R Ganglio capsular	15	-	-	+	-	8	ALIVE
50	Babu	59478	37	M	HT	180/120	140	99	6	R Thalamus	34	-	-	-	-	15	ALIVE
51	Kamala	59914	65	F	-	190/120	153	100	7	Brainstem	10	+	-	-	-	-	EXPIRED
52	Kothandapani	60105	50	M	HT	180/110	143	98.2	15	L Fronto parietal	62	-	-	+	-	0	ALIVE
53	Ramalingam	60904	54	M	-	180/100	123	99	9	L Ganglio capsular	8	-	-	-	-	7	ALIVE
54	Sivaraman	61112	50	M	HT	190/120	143	98	8	R Ganglio capsular	15	-	-	+	-	8	ALIVE
55	Durai	61267	45	M	-	200/120	146	100	6	L Ganglio capsular	48	+	-	+	-	-	EXPIRED
56	Ramesh	61298	39	M	-	200/130	163	101	3	L Parietal	64	+	-	+	-	-	EXPIRED
57	Ranganathan	61388	45	M	-	200/100	133	98.6	8	L Ganglio capsular	10	-	-	+	-	-	EXPIRED
58	Kannagi	61986	50	F	HT	190/100	130	98.6	11	L Ganglio capsular	8	-	-	-	-	5	ALIVE
59	Jaganathan	62705	55	M	HT	190/100	130	101	4	L Thalamus	35	+	-	+	-	-	EXPIRED
60	Ponniah	63101	56	M	HT	180/100	123	98.4	11	R Ganglio capsular	8	-	-	-	-	10	ALIVE
61	Pappammal	36746	72	F	HT	200/110	140	99.6	4	L Ganglio capsular	63	+	-	+	-	-	EXPIRED
62	Raja	63276	53	M	HT	180/130	146	99	5	L Ganglio capsular	40	+	-	+	-	-	EXPIRED
63	Ramar	63998	49	M	-	190/100	130	98.4	9	R Thalamus	35	+	-	+	-	11	ALIVE
64	Vijaya	64381	50	F	HT	150/100	116	98.6	10	R Ganglio capsular	10	-	-	-	-	12	ALIVE
65	Asokan	64894	52	M	HT	180/120	140	98.6	15	L Parietal	32	-	-	+	-	0	ALIVE
66	Pushpa	65204	58	F	-	220/120	153	98.2	4	L Parieto occipital	38	+	-	+	-	-	EXPIRED
67	Maragatham	65209	65	F	DM/HT	180/100	123	98.4	10	R Fronto parietal	8	-	-	-	-	13	ALIVE
68	Thangavel	65089	50	M	-	200/130	163	100	6	R Ganglio capsular	14	-	-	+	-	-	EXPIRED
69	Chitra	65655	45	F	HT	170/110	130	98.6	9	L Ganglio capsular	12	+	-	+	-	7	ALIVE
70	Parimala	65935	51	F	-	200/100	133	100	9	R Ganglio capsular	10	-	-	-	-	4	ALIVE
71	Arumugam	65862	52	M	-	230/120	156	99.2	12	R Ganglio capsular	20	-	-	+	-	5	ALIVE
72	Parthasarathy	66259	85	M	PT	200/90	126	98.4	10	L Ganglio capsular	43	-	-	+	-	6	ALIVE
73	Rani	61218	55	F	-	190/140	156	98.6	5	R Temporo parietal	34	+	-	+	-	-	EXPIRED
74	Kothandam	67268	40	M	-	180/120	140	98	5	L Ganglio capsular	60	+	-	+	-	-	EXPIRED

S.No.	Name	IP No.	Age	Sex	Comorbid Illness	BP	MAP	Temp °F	GCS (Out of 15	Site of Bleed	Volume	IVH	SAH	Midline Shift	Hydrocephalus	NIHSS Score/42	30 days outcome Alive/Expired
75	Babyammal	67347	70	F	-	230/110	150	100	3	Brainstem pons	32	+	-	+	-	-	EXPIRED
76	Aayeesha Bee	67669	49	F	HT	150/100	113	99	6	L Thalamus	35	+	-	+	-	-	EXPIRED
77	Prabhakaran	67991	35	M	HT	220/110	146	98.2	15	L Putamen	6	-	-	-	-	0	ALIVE
78	Aayeesha Bee	68019	80	F	-	200/120	146	98.4	8	L Thalamus	13	+	-	+	-	-	EXPIRED
79	Devaraj	67986	50	M	HT	150/90	110	98.6	15	L Ganglio capsular	8	-	-	-	-	0	ALIVE
80	Rajamani	67836	60	M	-	190/90	123	98.4	15	L Parieto occipital	7	-	-	-	-	0	ALIVE
81	Rajeeh	68070	64	M	HT/OLD CVA/INFARCT	200/100	133	105	13	R Thalamus	9	+	-	+	-	3	ALIVE
82	Pachaiyammal	68171	65	F	HT	170/100	123	102	3	R Thalamus	36	+	-	+	+	-	EXPIRED
83	Abdul Basheed	68606	55	M	HT	180/100	123	98.6	10	R Thalamus	10	+	-	+	-		ALIVE
84	Malakondiah	68600	72	M	DM	180/90	120	98.4	9	R Thalamus	8	+	-	-	-		ALIVE
85	Punniyakodi	68866	57	M	HT	150/110	123	103	3	R Ganglio capsular	40	+	-	-	-	-	EXPIRED
86	Velayudham	68879	45	M	HT	160/100	120	99	15	L Ganglio capsular	6	-	-	-	-	0	ALIVE
87	Raghupathy	69124	70	M	-	200/120	176	100	3	L Thalamus	30	-	-	-	-	-	EXPIRED
88	Parthasarathy	69342	54	M	-	180/100	126	98.6	7	L Ganglio capsular	8	-	-	-	-	9	ALIVE
89	Sulochana	69672	47	F	DM/HT	160/100	120	98.8	7	L Parietal	10	-	-	-	-	8	ALIVE
90	Chandra Babu	70368	45	M	-	200/100	133	98.6	9	R Parietal	6	-	-	-	-	11	ALIVE
91	Mariammal	71205	72	F	-	180/130	146	98.8	6	R Temporo parietal	64	+	+	+	-	-	EXPIRED
92	Munusamy	71407	40	M	-	200/130	153	98.6	3	L Temporal parietal	30	-	-	+	-	-	EXPIRED
93	Durairaj	71411	70	M	-	200/120	146	98.6	6	R Temporo parietal	50	+	-	+	-	-	EXPIRED
94	Vairam	71443	65	M	-	150/94	112	99.2	11	R Thalamus	34	+	-	+	-	-	EXPIRED
95	Vairm	71880	60	F	HT	150/90	110	98.6	9	L Parietal	10	-	-	-	-	5	ALIVE
96	Vijayarangam	72224	60	M	-	220/120	153	98.8	3	Brainstem Cerebellum	15	+	+	-	-	-	EXPIRED
97	Sandhya	72589	13	F	-	110/80	90	98.2	7	R Ganglio capsular	36	+	-	+	-	-	EXPIRED
98	Moorthy	72421	95	M	HT	150/90	110	99	15	R Ganglio capsular	10	-	-	-	-	0	ALIVE
99	Elumalai	73145	50	M	-	200/100	133	98.6	10	L Ganglio capsular	12	-	-	+	-	3	ALIVE
100	Richard	74636	40	M	-	220/140	163	105	3	Brainstem	10	+	-	+	-	-	EXPIRED

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INSTITUTIONAL ETHICAL COMMITTEE
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L.Dis.No. 14597/MES/EthicsDean/MMC/2009 Dated .09.2009

Title of the work : "prognostic predictors & 30 day outcome in patients with non-traumatic parenchymal hemorrhage"

Principal Investigator : Dr. E. Elavarasi postgraduate student - M.D General medicine

Department : Madras medical college - Ch-3

The request for an approval from the Institutional Ethical Committee(IEC) was considered on the IEC meeting held on 23rd September 2009 at 2.00P.M. in Madras Medical College, Deans, Chamber, Chennai-3. / pharmacology seminar hall - madras medical college Ch-3.

The members of the Committee, the Secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The principal investigator and their term are directed to adhere the guidelines given below:

1. You should get detailed informed consent from the patients/participants and maintain confidentiality.
2. You should carry out the work without detrimental to regular activities as well as without extra expenditure to the Institution or Government.
3. You should inform the IEC in case of any change of study procedure, site and investigation or guide.
4. You should not deviate form the area of the work for which I applied for ethical clearnance.
5. You should inform the IEC immediately, in case of any adverse events or serious adverse reactions.
6. You should abide to the rules and regulations of the institution(s).
7. You should complete the work within the specific period and if any extension of time is required, you should apply for permission again and do the work.
8. You should submit the summary of the work to the ethical committee on completion of the work.
9. You should not claim funds from the Institution while doing the work or on completion.
10. You should understand that the members of IEC have the right to monitor the work with prior intimation.

SECRETARY
IEC, MMC,CHENNAI

CHAIRMAN
IEC MMC CHENNAI

21/10/09
DEAN
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