A STUDY ON

" PREDICTION OF OUTCOME IN PATIENTS WITH PRIMARY INTRA CRANIAL HEMORRHAGE USING FUNC SCORE"

Dissertation Submitted to

THE TAMIL NADU DR.M.G.R.MEDICAL UNIVERSITY

CHENNAI

In partial fulfillment of the regulations for the award of the degree of

M.D. BRANCH – I (GENERAL MEDICINE)



DEPARTMENT OF GENERAL MEDICINE

GOVERNMENT STANLEY MEDICAL COLLEGE, CHENNAI.

THE TAMIL NADU DR. M.G.R.MEDICALUNIVERSITY

TAMILNADU, INDIA

MAY 2020

CERTIFICATE

This is to certify that this dissertation entitled " **PREDICTION OF OUTCOME IN PATIENTS WITH PRIMARY INTRA CRANIAL HEMORRHAGE USING FUNC SCORE**" submitted by Dr.M. RAJMOHAN to the faculty of General Medicine, The Tamil Nadu Dr. M.G.R Medical University, Chennai, Tamilnadu, in partial fulfillment of the requirement for the award of M.D DEGREE BRANCH-I (GENERAL MEDICINE) is a bonafide research work carried out by him under my direct supervision and guidance.

GUIDE

HOD

Prof. Dr. A. SAMUEL DINESH, M.D.,Chief, Medical Unit-5,Department of Medicine,Stanley Medical College and Hospital,Chennai - 1.

Prof. Dr. C. HARIHARAN M.D., Head of the Department, Department of Medicine, Stanley Medical College and Hospital, Chennai- 1.

PROF. DR.R.SHANTHIMALAR, M.D., D.A.,

DEAN

Government Stanley Medical College and Hospital,

Chennai-1.

DECLARATION

I, Dr. M. RAJMOHAN, solemnly declare that the dissertation titled "**PREDICTION OF OUTCOME IN PATIENTS WITH PRIMARY INTRA CRANIAL HEMORRHAGE USING FUNC SCORE**" is a bonafide work done by me at Government Stanley Hospital, Chennai during May 2018 to April 2019 under the guidance and supervision of Prof.Dr.A.SAMUEL DINESH, Professor of Medicine, Government Stanley Hospital, Chennai. I also declare that this bonafide work or a part of this work was not submitted by me or any other forward degree or diploma to any other university, board either in India or abroad. This dissertation is submitted to the Tamilnadu Dr. M.G.R Medical University, towards the partial fulfillment of requirement for the award of M.D. Degree (Branch – I) in General Medicine.

Place: Chennai

Signature of the candidate

Date:

(Dr.M.RAJMOHAN)

CERTIFICATE - II

This is to certify that this dissertation work titled titled "**PREDICTION OF OUTCOME IN PATIENTS WITH PRIMARY INTRA CRANIAL HEMORRHAGE USING FUNC SCORE**" of the candidate Dr.M.RAJMOHAN with Registration Number 201711065 for the award of M.D., DEGREE in the branch of BRANCH-I (GENERAL MEDICINE). 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 pages and result shows 4 percentage of plagiarism in the dissertation.

Guide & Supervisor sign with Seal

SPECIAL ACKNOWLEDGEMENT

I gratefully acknowledge and thank

PROF. DR.R. SHANTHI MALAR M.D., D.A.,

DEAN

GOVERNMENT STANLEY MEDICAL COLLEGE AND HOSPITAL, CHENNAI.

For granting me permission to utilize the resources of this

Institution for my study

ACKNOWLEDGEMENT

I am extremely thankful to our beloved Superintendent **Prof.Dr.DHANASEKAR, M.D.,** Government Stanley HospitalChennai-1, for having granted permission to do this dissertation in Government Stanley Hospital, Chennai.

I am very grateful to our Professor and Head of the Department of Medicine

Prof Dr.C.HARIHARAN, M.D., for acceptance to do this dissertation.

I am extremely grateful to my unit chief **Dr.A.SAMUEL DINESH**, **M.D.**, who taught me the basic aspects and clinical skills in internal medicine which is an essential pre requisite for pursuing any dissertation work. The guidance and encouragement they provided need a special mention.

I recall with gratitude the other unit chiefs and Associate Professors of Department of Medicine, Prof . Dr.G.RAJAN, M.D., Prof. Dr. C.SRIDHAR M.D., Prof.Dr.S.GEETA, M.D., Prof.Dr.T.B.UMADEVI M.D., Prof. Dr.A.RAVI M.D., Prof.Dr.I.ROHINI M.D., Prof. Dr. R.THILAKAVATHY M.D., Prof.Dr.P.MALARVIZHI M.D., Prof. Dr. KALPANA RAMANATHAN M.D., for their valuable guidance.

I am extremely thankful to our Registrar **Dr.N.RAVICHANDRAN M.D.**, and our unit **Asst. Professors Dr.R.VIJAY ANAND M.D.**, **Dr. K.LAKSHMI M.D.**, for their valuable suggestions, guidance and support.

I thank **Prof.Dr.S. ARUNAN, M.D.,** Professor and Head of the Department of Neurology, Government Stanley Hospital for guiding me in treating the patients which were very crucial for the study.

I thank **Prof. Dr. M.P. SARAVANAN, M.D.,** Professor and Head of the Department of Biochemistry for providing me with facilities for accurate measurement of the biochemical parameters involved in the thesis work which were very crucial for the study.

I also thank **Prof .Dr. C.AMARNATH M.D.**,(**RD**), Professor and Head of the Department, Department of Radiology, for allowing me to utilize the CT facility of their department to image the brain of my patients, and also in helping to interpret them.

I also thank all our patients and their attenders, without whom the study would not be possible.

I extend my love and gratitude to my family and friends for their immense help for this study.

I owe my thanks to almighty for successful completion of this study.

Date:

Signature of the Candidate

Place:

(Dr.M. RAJMOHAN)

ABBREVATIONS

: Glasgow Coma Scale
: Sub Arachnoid Hemorrhage
: Middle Cerebral Artery
: Anterior Cerebral Artery
: Posterior Cerebral Artery
: Computerised Tomography
: Magnetic Resonance Imaging
: Magnetic Resonance Arteriography
: Magnetic Resonance Venography
: Intra Cerebral Hemorrhage
: FUNCtional score
: Cerebral Amyloid Angiopathy
: Renal Function Test
: Liver Function Test
: Coronary Artery Disease
: Type2 Diabetes Mellitus

TABLE OF CONTENTS

S.NO	CHAPTERS	PAGE NUMBER		
1	INTRODUCTION	10		
2	AIM AND OBJECTIVES OF THE STUDY	11		
3	REVIEW OF LITERATURE	14		
4	MATERIALS AND METHODS	35		
5	STATISTICAL ANALYSIS	45		
6	RESULTS	84		
7	DISCUSSION	87		
8	CONCLUSION	89		
9	BIBLIOGRAPHY	90		
10	PROFORMA	95		
11	ETHICAL COMMITTEE	96		
12	PLAGIARISM CERTIFICATE	97		
13	INFORMED CONSENT	98		
14	MASTER CHART	100		

INTRODUCTION

Intracranial hemorrhage refers to bleeding within the intracranial vault, that includes the brain parenchyma and surrounding meningeal spaces. This study mainly focuses on the primary intra cerebral hemorrhage (ICH) which is non-traumatic.

Intra cerebral hemorrhage (ICH) the most disturbing and least treatable form of stroke, also in addition, it causes severe disability among survivors.^{1–4} Because ICH is considered to be fatal in majority of circumstances, withdrawal of care of such patients commonly occurs early even in the hospital course, in their home that cause a situation that can take away a "fighting chance" to those patients whose prognosis may not be as grave as initially judged.^{5,6,18} Providing proper care to such patients not only help in the survival of those patients but also it results in good independent outcome for those who survive the episode.

Accurate prediction regarding the outcome of the patients presenting with ICH is very important for confronting the members of the family. Since most patients with ICH require advanced treatment and care, many times a need of transfer to higher centre may be required. In such instances an accurate prediction regarding the outcome may help the family members in decision making and proper care.^{19,20}

Basically, it is identification of patients expected to recover functional independence, rather than just survive, which can address the most persistent concern of families, medical teams in regard to direction of care.⁷

10

AIMS AND OBJECTIVES

Aim of the study:

The aim of this study is to determine the **Prediction of Outcome in Patients with Primary Intra cranial hemorrhage using FUNC score** in Stanley Government Medical College Hospital.

Primary objective:

- To calculate the FUNC score in patients admitted with spontaneous intra cerebral hemorrhage confirmed by CT brain.
- 2. To follow up these patients for a period of 90 days.
- To compare the outcome of patients with low FUNC score to those with high FUNC score after 90 days
- 4. Factors determining the outcome are assessed.
- Functional independence of the surviving patients compared according to their FUNC score.
- ^{6.} The importance of a score that can predict the outcome of a patient with primary ICH can help the patient in terms of proper care and support apart from treatment.

Study background :

- Intra cerebral hemorrhage (ICH) is the most serious and disturbing stroke subtype.
- Widely used tools that are used for prediction of mortality and outcome are limited such that they don't take into account of effects of care withdrawal and not planned to predict functional outcome and recovery. ^{2 2-29}
- An acute clinical score to foresee functional independence would help in predicting the outcome.
- Primary (nontraumatic) intracerebral hemorrhage (ICH) accounts for an approximately 10–15 % of strokes.
- Its importance originates from its frequency and its associated high mortality.
- The ICH score and volume is a clinical grading scale that allows risk stratification on patients with ICH.

The use of a scale such as the FUNC score could predict the Outcome in Patients With Primary Intra cranial hemorrhage .

Justification of Study:

- More than 60% of individuals die or are left with severe disability following intra cerebral hemorrhage.
- The accurate prediction of outcome is therefore essential to help families of affected individuals decide on goals of care, as they consider whether their loved one would choose to survive
- FUNC score is based on GCS score, ICH volume, ICH location, Age, Pre ICH Cognitive impairment.
- The score ranged from 0 to 11 with a score of 11 indicating strong likelihood of functional independence.
- Patients revealed no chance of attaining functional independence at 90 days if their score was <4
- Inspite of the effective advances in treatment, the outcome of the patient with Intra cerebral hemorrhage remains poor. The myth that patients presenting with ICH always have a grave prognosis still persists. The care for such patients eventually gets reduced.
- Hence a score or scale that can accurately predict the outcome of such patients maybe helpful for proper care and can result in good functional independence of such patients.

REVIEW OF LITERATURE

HISTORICAL PERSPECTIVE

Stroke is a prehistoric disease. Imhotep, Father of Egyptian medicine, labelled stroke around 3000 BC. In 1600 Thomas Willis termed Circle of Willis and used the term "Apoplexy". Modern era of stroke started when Miller Fisher termed stroke and its features.

In the past few years there was a great change in management of stroke. In 1996, Introduction of IV rt-PA as established effective treatment was a trend setter in the field of medicine. In the field of interventional neurology the use of endovascular therapies is another milestone in treatment of stroke in modern time. A development in neuroimaging and diagnostic services is of supreme importance in the growth of field of neuromedicine.

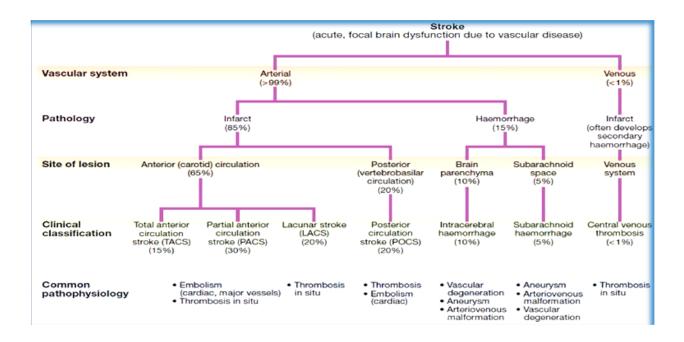
Inspite of the effective advances in treatment, the outcome of the patient with Intra cerebral hemorrhage remains poor. The myth that patients presenting with ICH always have a grave prognosis still persists.

TIME IS BRAIN

The average number of neurons present in human cerebrum is 22 billion. When there is an ischemia, in less than a minute about, 14 billion synapses, 1.9 million neurons and 12 km length of myelinated fibers are lost³. With passing of each hour the ischemic brain eons 3.6 years than normal. Prompt intervention is very vital for maintaining the neuronal cell mass by defending them from the ischemic insult.

STROKE classification:

Stroke maybe arterial or venous. Arterial maybe ischemic or hemorrhagic.



Ischemia

Ischemia is divided into 3 mechanisms: thrombosis, embolism, and diminished systemic perfusion.

Thrombosis :

Thrombosis is stated as obstruction to blood flow which can be due to a localized occlusive disease in the blood vessels. The most common pathology is atherosclerosis, here the fibrous and muscular tissues multiply within the sub intima, and fat forms plaques that can encroach the lumen.

Embolism :

In embolism, the thrombus formed elsewhere in the blood vessels gets blocked in an artery and obstructs its blood flow. Embolic luminal blockage, in disparity to thrombosis, is usually not caused by a local process which is originating in the obstructed artery.

The embolized substance mostly arise proximally, from the heart, or from aorta, vertebral arteries ,carotid, or systemic veins.

Decreased Systemic Perfusion :

In decreased systemic perfusion, reduced blood flow to brain tissue is due to low systemic perfusion pressure. The most common causes are cardiac failure (mostly due to MI or rhythm disturbances) and systemic hypotension (which is due to blood loss or hypovolemia).

Poor perfusion is most prominent in border zone ,also known as watershed regions at the junction of the major vascular territories³¹⁻³³

HEMORRHAGE

Hemorrhage can be subdivided into four subtypes: subarachnoid, intracerebral, subdural, and epidural

Subarachnoid Hemorrhage :

In subarachnoid hemorrhage, the blood leaks from the vessel into the brain surface and is disseminated through the cerebrospinal fluid path into the places around the brain, most often comes from aneurysms or AV malformations.

Intracerebral Hemorrhage :

The intracerebral or parenchymal hemorrhage means the bleeding into the brain parenchyma. The most common cause is hypertension, where the leakage of blood is from small intracerebral arterioles which is damaged by the raised blood pressure¹⁸⁻²²

Subdural and Epidural Hemorrhages :

These are mostly caused by head trauma. Subdural hemorrhages originate from the injured veins which are located between the arachnoid membranes and the duramater.

Epidural hemorrhages are due to tearing of the meningeal arteries, more commonly the middle meningeal artery. Blood collects quickly over minutes to hours in between dura mater and the skull.

STROKE DUE TO ICH

Stroke attributable to intracerebral haemorrhage is around 20-30% of the various causes¹, The importance is due to the high frequency and 30 day mortality and its predilection to affect middle aged hypertensives and elderly on anti-thrombotic therapy. Alleles of apolipoprotein E have an important role in lobar hemorrhages.

BLOOD SUPPLY OF THE BRAIN:

The blood supply of the brain is predominantly from the branches of the internal carotid and vertebral arteries². The two vertebral arteries at the base of the pons unite to form the basilar artery which terminates into the right and left posterior cerebral arteries. The internal carotid artery divides into the smaller anterior cerebral and larger middle cerebral arteries. Around the interpeduncular fossa, in the subarachnoid cistern lies the circle of Willis which is formed by the anterior cerebral and anterior communicating arteries anteriorly, posterior communicating arteries laterally and the posterior cerebral arteries branching off the basilar artery. Aneurysmal rupture induced bleeds may occur in this region.

Branches of the cranial part of vertebral artery:

Anterior spinal artery

Posterior spinal artery

Posterior inferior cerebellar artery

18

Meningeal branches

Medullary arteries

Branches of the Basilar artery:

Pontine branches

Labyrinthine artery

Superior cerebellar artery

Posterior cerebral artery: This artery further divides to form anterior temporal, posterior

temporal branch, parieto-occipital branch and calcarine branch.

Branches of the Internal carotid artery:

Ophthalmic artery

Anterior cerebral artery

Middle cerebral artery

Posterior communicating artery

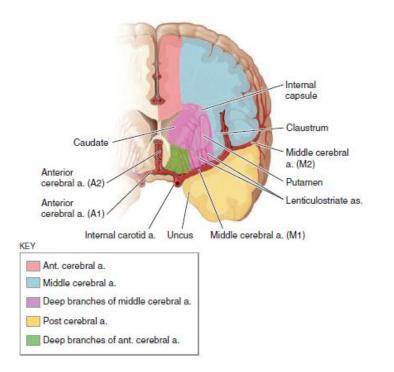
Anterior choroidal artery

ARTERIAL SUPPLY OF THE CEREBRUM:

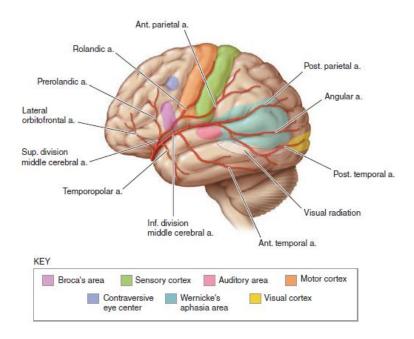
The anterior, middle and posterior cerebral arteries predominantly supply the various cerebral hemispheres. Arterial supply of the supero-lateral surface is by the middle

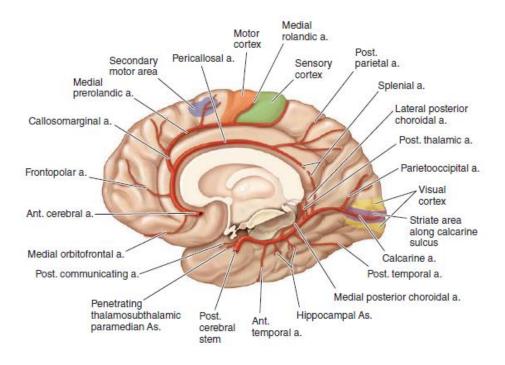
cerebral artery. This covers most of the vital areas including the primary motor and sensory areas, Broca's and Wernicke's speech areas in the dominant hemispheres and the frontal eye field. A portion along the superomedial border upto the parieto-occipital sulcus is supplied by the anterior cerebral artery. Likewise a potion along the lower aspect of the temporal lobe and occipital lobe is supplied by the posterior cerebral artery. The medial supply of the brain is supplied by the anterior cerebral artery predominantly with the middle and posterior cerebral arteries supplying the temporal and occipital lobes respectively. The inferior surface of the brain is supplied by the posterior cerebral artery mainly with contributions from the middle and anterior cerebral arteries. The internal capsule and corpus striatum are supplied by the central branches of the middle and anterior cerebral arteries. The thalamus and midbrain are supplied by the posterior cerebral, basilar arteries alongside the posterior communicating supplying the thalamus and superior cerebellar supplying the midbrain. Pontine supply is from the basilar, superior cerebellar and anterior inferior cerebellar arteries. The cerebellum derives its blood supply from the superior, anterior inferior and posterior inferior cerebellar arteries.

BRANCHES OF INTERNAL CAROTID ARTERY:



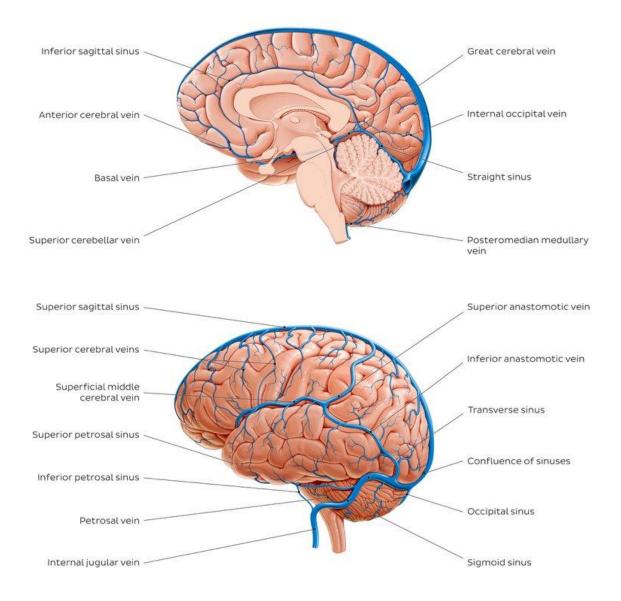
BLOOD SUPPLY OF LATERAL AND MEDIAL ASPECT OF BRAIN:





VENOUS DRAINAGE OF THE BRAIN:

Superior cerebral veins drain the upper portion of the superolateral surface into the superior saggital sinus. Inferior cerebral veins drain the lower potio of the superolateral surface into the superficial middle cerebral vein. The medial surface of the brain is drained by superior, inferior cerebral veins and the anterior cerebral vein. The inferior surface of the brain is drained by the inferior cerebral veins.



CAUSES OF ICH:

Hypertension

Vascular malformations

Vasculitis

Tumoral bleed

Bleeding diathesis

Cerebral amyloid angiopathy

Sympathomimetic agents

Head injury

Haemorrhagic infarction

Hypertension:

Presence of a high frequency of hypertensive population suffering from ICH, high admission blood pressure and echocardiographic evidence of hypertension signifies the importance of hypertension as a causative factor in ICH. Smoking, sedentary lifestyle, dyslipidemia, excess alcohol consumption constitute modifiable risk factors. Lipohyalinosis of the arteries due to chronic hypertension and microaneurysms cause bleeding.

Vascular Malformations:

Intracranial aneurysms, AV malformations, cavernous angiomas at the subcortical white matter. These can be diagnoses by CT or MRI imaging. Cavernous angiomas can be recognised by the presence of a central nidus of irregular bright signal with popcorn pattern and a hypointense ring of hemosiderin on T2-images. Those located in the

posterior fossa present with a progressive course with recurrent small hemorrhages. Recurrence risk is greatest among women and is highest in the first two years.

Intracranial tumors:

Glioblastoma multiforme, metastasis from vaious organs may bleed within resulting in ICH.The unique features include its distinct location from hypertensive bleeds, imaging revealing presence of hyperdense region surrounding a low density area; bleeds at multiple sites, presence of papilloedema, presence of severe cerebral oedema with mass effect.Craniotomy with biopsy of suspected lesion can be done however it carries grave prognosis.

ICH secondary to bleeding diathesis, anticoagulants and fibrinolytic therapy:

Young patients presenting with ICH should be evaluated for the presence of bleeding disorders such as haemophilia. Immune thrombocytopenia, acute leukemia especially acute promyelocytic form and acute lymphocytic leukemia may present as ICH.

9-14% of ICH is due to anticoagulant related causes and is more common in the elderly with history of hypertension, prior cerebral infarction and INR prolongation. Hence maintenance of INR between 2 to 3 may help in reducing the risk of ICH. This type of ICH presents with a slowly progressive course with a large volume bleed and occasionally with other bleeding manifestations. Use of fibrinolytic therapy such as streptokinase or tissue plasminogen activators for thrombolysis may also lead to ICH, a higher incidence being found in those with hyperglycemia, cerebral amyloid angiopathy, use of dual anti-platelets and higher baseline blood pressure post thrombolysis. Presence of cerebral microbleeds also increases the risk.

Cerebral Amyloid Angiopathy:

Deposition of β -amyloid in the cerebral vessel wall especially of those in the leptomeninges and cortex occurs in cerebral amyloid angiopathy. It predominantly affects elderly patients with a lobar location of bleedIt may anifest as focal neurological deficits and seizures weeks prior to onset of ICH. Histology reveals presence of Congo red positive, birefringent amyloid material in the intima media.

Vasculitides:

Polyarteritis nodosa may present as ICH which is characterized by mononuclear inflammation of small and medium sized vessels. There are signs of systemic involvement such as fever, malaise, renal failure, hypertension and elevation of erythrocyte sedimentation rate.

Sympathomimetic agents:

The use of sympathomimetic agents is associated with transient hypertension and areas of spasm and dilatation in the vessels resulting in ICH. Cocaine is most commonly implicated.

Hemorrhagic infarction:

Occurs from embolic stroke with maximal symptoms at onset with a spotted appearance on computed tomography with a cortical distribution around arterial branches.

<u>CLINICAL FEATURES</u>:

Altered level of consciousness

Headache

Type of intracerebral hemorrhage		Hemisensory syndrome Ap	Aphasia	Homonymous Visual Defects	Gaze palsy		
					Horizontal	Vertical	Brainstem Signs
Putaminal	Generally dense	Frequent	Gobabmotor>conduction	In large hematomas	Contralateral	No	No (only present with hemiation)
Caudate	Absent or mild, transient	Absent	No	No	Generally absent	No	No
Thalamic	Generally dense	Frequent, prominent	Occasional, thalamic variety	In large hematomas	Contralateral, occasionally ipsilateral	Yes, upward	Skew deviation, Horner syndrome, Parinaud syndrome
Lobar	Prominent in frontoparietal location	Prominent in frontoparietal location	In dominant temporoparietal location	In occipital hematomas	Contralateral in frontal hematomas	No	No (only present with hemiation)
Cerebellar	Absent	Absent	No	No	lpsilateral	No	Ipsilateral fifth through seventh nerve palsy, Homer syndrome
Pontine	Variable, usually bilateral	Variable, usually bilateral	No	No	Bilateral	No	Pinpoint reactive pupils, ocular "bobbing," decerebrate rigidity, respiratory rhythm abnormalities
Mesencephalic	Variable, usually present	Rare	No	No	No	Occasional, upward	Unilateral or bilateral third nerve palsy
Medulary	Generally absent	Occasional	No	No	No	No	Nystagmus, ataxia, hiccups, facial hypesthesia, dysarthria dysphagia, twelfth nerv palsy, Horner syndrom
Intraventricular	Generally absent	Rare	No	No	Occasional	Occasional	Rare (decerebrate rigidity

Vomiting

Focal neurological deficits that progresses over hours

Specific features pertaining to the site of bleed is as follows:

Putaminal bleed:

Constitutes the most common form of ICH. Presentation varies from hemiparesis to

decerebrate rigidity.

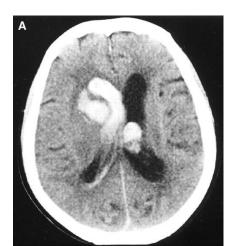


Caudate bleed:

Occurs from rupture of penetrating arteries of anterior and middle cerebral arteries.

Presents with signs of meningeal irritation, focal neurological deficits such as

hemiparesis, Horner's palsy, language and Intraventricular



syndrome, horizontal gaze memory disturbances. extension with hydrocephalous occurs however the overall outcome is good.

Thalamic haemorrhage:

Constitutes 10-15% of the ICH with an abrupt onset and slow progression.Intraventricular clot may cause aqueductal obstruction leading to a hydrocephalous that may be reversed with a ventriculostomy procedure.

Lobar haemorrhage:

Second most common form frequently a result of non-hypertensive causes of bleed like arteriovenous malformations, sympathomimetic agents and amyloid angiopathy. Features pertaining to the lobe involved occur such as hemiparesis involving predominantly the upper limbs in frontal lobe, aphasia in dominant temporal hemisphere, hemianopia with sensorimotor deficit in parietal lobe lesions and homonymous hemianopia in occipital lesions.

Cerebellar haemorrhage:

Presents as vertigo, vomiting, inability to walk with a triad of ataxia, horizontal gaze palsy, facial palsy at the



side of haemorrhage.

Pontine haemorrhage:

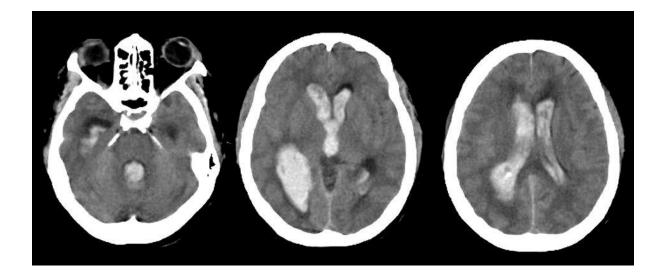
Bilateral involvement of the tegmentum results in quadriplegia, coma, ocular bobbing, horizontal gaze palsy, pin-point pupil and respiratory abnormalities. One and a half syndrome, fifth and sixth cranial nerve palsies and internuclear ophthalmoplegia may occur.

Medullary haemorrhage:

A rare variant that usually affects one half of the medulla resulting in ipsilateral hypoglossal nerve palsy and contralateral hemiparesis thereby differentiating it from Wallenberg syndrome.

Intraventricular haemorrhage:

Commonly occurs in thalamic and caudate haemorrhages. The vasculature of the subependymal layer is thought to be the site of origin of the bleed. Focal neurological deficits are very rare in presentation. Patients who are comatose with brainstem involvement succumb to the illness.



IMAGING:

CT and MRI imaging with angiography form essential components in the diagnosis and prognostification.

Presence of spot sign-contrast extravasation may signify hematoma expansion which occurs in almost 77% of the patients.

TREATMENT:

Rapid evaluation of the patients is to be done with immediate endotracheal intubation for those with a Glasgow Coma Scale score below 8 with the administration of thiopental or lignocaine to prevent increases in intracranial pressure secondary to tracheal stimulation or intubation. Routine evaluation with specific investigations such as use of toxin screening, blood glucose levels, coagulation profile.

If the patient has been previously on heparin anticoagulation protamine sulphate has to be administered while those on warfarin should be given Vitamin K, fresh frozen plasma or prothrombin complex concentrate.¹⁷ If the ICH was secondary to thrombolytic therapy cryoprecipitate or fresh frozen plasma should be transfused.

Initial treatment is aimed at controlling the blood pressure and seizures as hypertension may further worsen the cerebral oedema. The goal should be aimed at around 160/90mmHg; labetalol being the anti-hypertensive of choice. Nicardipine is an alternative.

Raised intracranial pressure may be managed with hyperventilation, osmotic diuretics and steroids. Intravenous mannitol may be given for reducing the cerebral oedema. If the intracranial pressure is not controlled with the above measure, need for surgical intervention should be considered.

Studies comparing the surgical and non-surgical methods of treatment did not find any significant alteration in the mortality rates.¹⁶ Lobar haemorrhage with progressive deterioration in consciousness, ICH due to AV malformation, aneurysm or cavernous angioma are indications for surgery. Cerebellar haemorrhage is another indication for surgery. Radiologically, a hematoma

32

diameter of greater than or equal to 3cm, obliteration of the quadrigeminal cistern or presence of hydrocephalous are indications for surgery.

Surgical options include hematoma evacuation, ventricular drainage for hydrocephalous.

Newer treatment modalities include the use of Recombinant activated factor

VIIa and is under research along with alternative agents such as tranexamic acid

Sample size: 134 cases

Formula

 $n = Z^2 pq/d^2$

Where Z =1.96(statistical constant or 95% CI)

p (prevalence) =62% (incidence of deaths due to ICH volume >60ml in patiens with primary ICH)

q(100-p) = 38%

d = 15% relative precision (i.e 15% of 62)which is 9

Therefore using the formula

 $n = 1.96 \ge 1.96 \ge 62 \ge 38/81$

n = 111

Adding 10% non response rate (i.e 10% of 111 which is 11)

Minimum sample size n = 122

Therefore Sample size n = 134.

Study duration: March 2018 to March 2019 (12 Months)

Inclusion criteria:

- Patients with age ≥ 12 years
- Patients with spontaneous ICH of nontraumatic origin detected on CT/magnetic resonance imaging or angiographic study
- Who have presented with history of acute severe headache, altered sensorium, slurring of speech, acute hemiparesis, and accelerated hypertension—suggestive of acute cerebrovascular stroke

Exclusion criteria:

- Patients <12 years of age
- Patients with history of trauma
- Patient with ischemic stroke and venous thrombosis
- Patients with sub arachnoid hemorrhage
- Patients with epidural hematoma
- Patientswith sub dural hematoma
- Patients with berry aneurysm

METHODOLOGY

During the study period, patients admitted with Intra cranial haemorrhage were performed imaging study. The most basic and useful imaging study was the computed tomography of the brain. Preliminary basic details like name, age, sex, residence were noted. A detailed history was elicited from the patients regarding trauma, road traffic accidents or other modes of injury. All traumatic cases were excluded even when they presented with elevated blood pressures.

In non-traumatic patients, the history of mode of onset, time and place of onset were elicited. Most of the Presenting complaints were like weakness of limbs, difficulty in speaking, altered sensorium, loss of consciousness, seizures, projectile vomiting and all other associated co morbidities were noted.

Since most of the patients presenting as epidural and subdural hemorrhage are due to trauma, they were excluded. Aneurysms and AV malformations being the primary cause of SAH, they may prsent even with modest elevation of blood pressure. Hence EDH, SDH, SAH are all excluded and patients with non traumatic primary ICH(intra cerebral hemorrhage) alone were included in the study group.

Detailed past history of hypertension, diabetes, cognitive impairment, coronary artery disease, prior use of anti-platelet drugs were noted. Recent surgeries or any factors leading to the event are noted. Prior medications and other native

35

medical history were noted. Previous episodes of ICH if any were noted. If so, such patients were excluded.

A family history of hypertension is noted since hypertension is strongly associated with ICH. Any family history of similar episodes were noted. A chance of aneurysm is suspected in patients having with family history of ICH.

Personal history of alcohol intake, smoking, IV drug abuse were elicited in detail. Alcohol being an individual risk factor, also can lead to seizures causing head injury. It may also lead to cognitive impairment, can lead to repeated falls and trivial injury. All these were asked from the presenting patient's attenders in view of reliability.

EXAMINATION:

Patient's vitals including pulse rate, blood pressure, temperature, CBG, saturation were noted and monitored in the zero delay ward. All the patients presenting with ICH had elevated blood pressure levels, indicating uncontrolled blood pressure as the major risk factor for primary ICH.

Patient's level of consciousness, co-operation, orientation, speech, memory, articulation, mood were examined. Furthermore, patient's behaviour, appearance, judgement, power of abstraction, intellect, attention and concentration were checked. MMSE was calculated. The most important aspect of examination in patients with ICH is GCS calculation as it readily tells about the general condition of the patient and it marks as one of the important components of FUNC score

GCS CALCULATION

(I)EYE OPENING:	RESPONSE	SCORE
	Opens spontaneously	4
	Opens to verbal command	3
	Opens to pain	2
	Does not open	1
(ii)VERBAL RESPON	ISE:	
	Oriented	5
	Confused	4
	Inappropriate words	3
	Incoherent sounds	2
	No response	1
(iii) MOTOR RESPON	NSE:	
	Obeys commands	6
	Localising pain	5
	Withdrawal from pain	4
	Decorticate posture	3
	Decerebrate posture	2
	No response	1
TC	TAL SCORE	15

As mentioned earlier, GCS calculation is essential in all patients with ICH. Minimum score of 3 and maximum score of 15 was obtained.

The duration of presentation is noted as timely intervention is the most important aspect in management of stroke irrespective of aetiology. Patients who are referred from primary care health centres to our hospital took longer time for presentation due to various reasons.

INVESTIGATIONS:

CT scan of brain was taken immediately. Along with that additional imaging studies like Doppler study of carotid arteries, angiogram- CT angiogram, MRI brain with MRA and MRV – plain and contrast were taken. The purpose angiogram was to rule out any AV malformations, aneurysms like berry aneurysm. Patients with aneurysmal bleed causing SAH were excluded.

Apart from imaging studies routine blood investigations like complete blood count, renal function tests, and liver function tests were taken. Renal function tests were essential for contrast studies. Liver function tests were important for drug modification in treatment.

The 2 components of FUNC score ICH volume and ICH location is calculated from the CT brain. Besides providing the conclusive diagnosis, CT also show basic features of the hematoma, such as: hematoma location, extension to the ventricular system, presence of surrounding edema, development of mass effect and midline shift.

CALCULATION OF ICH VOLUME:

Estimation of the hematoma volume can be quickly performed in the ED with the help of validated ABC/2 technique⁸ (Figure 1).

Steps to follow using this method are:

- The CT slice with the biggest area of hemorrhage is carefully chosen.
- A is the biggest hemorrhage diameter on the selected cut (in centimeters [cm]).
- B is the biggest diameter perpendicular to A on the same cut.
- C is the approximate number of cuts in which the hemorrhage is noted multiplied by the slice thickness (frequently 0.5cm slices).
- A, B, and C are multiplied and the product is divided by 2.

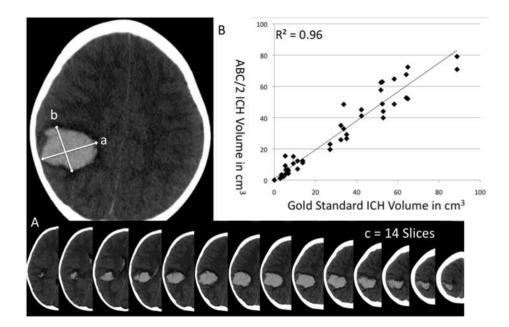


Figure-1: calculation of ICH volume

ICH is considered to be small if the volume is $<30 \text{ cm}^3$. Moderate volume of ICH is between 30-60 cm³. Those having ICH volume $>60 \text{ cm}^3$ are considered to be large ICH.

After calculating ICH volume, it is essential to see the ICH location as location is also a component of FUNC score and is considered to be the most important component of FUNC score apart from GCS. The location may be superficial/lobar, deep or infra tentorial.

ICH was said to be lobar in location if the source of the hemorrhage appeared to be in cerebral hemispheres superficial to deep gray matter structures. Hemorrhages originating in the thalamus and basal ganglia are said to be "deep" in location.^{9,10} ICH is considered to be infra tentorial if the hemorrhage is below the tentorium cerebelli. The infra tentorial region mainly consists of the cerebellum and its peduncles.

Apart from volume and location, midline shift and intra ventricular extension of the bleed are noted. Since the midline shift and intra ventricular extension are life threatening, patient with these complications are intervened surgically depending on patient's condition.

After obtaining all these parameters FUNC score is calculated for all the patients.

41

DETERMINANTS OF FUNC SCORE:

COMPONENT

FUNC SCORE POINT

ICH VOLUME(cm3):

<30	4
30-60	2
>60	0
<70	2
70-79	1
>79	0

ICH LOCATION

AGE

Lobar	2
Deep	1

Infratentorial 0

GCS SCORE

>=9	2
=<8	0

PRE ICH COGNITIVE IMPAIRMENT

NO	1	
YES	0	
TOTAL FUNC SCORE	11	

The prediction of outcome is entirely dependent on FUNC score. Score of >8 is considered good and the patient is expected to be alive after 90 days. A score of <4 is considered poor and the patient is expected to be dead after 90 days. All the patients are followed up for a period of 90 days and the outcome is compared.

FUNC score was once again calculated along with GCS score. 90th day measurement of FUNC and GCS is to determine whether the patient is functionally independent or not. A FUNC score of more than 7 along with GCS of more than 8 is considered to be functionally independent.

In these 90 days patient were evaluated for any re-bleed, seizures and time taken for ambulation with support. Earlier the time taken for ambulation denotes that the person improved well and indirectly tells the patient is functionally independent.

90th day cognitive impairment and bed sores was examined. Bed sores was considered significant as it not only tells about the status of the patient whether he is bed ridden or not, but also the care given for the patient. Improper care results in deep bed sores requiring wound debridement.

43

Informed consent:

Consent form will be written in both English and Tamil and also orally explained in their own language and consent will be obtained from the participants, confidentiality will be maintained.

Statistical analysis:

Data will be entered in excel sheet and the analysis will be done using SPSS version 17. For numerical data mean and standard deviation will be used, for continuous variable chi square will be used and to find out the association of the two variables. Students't' test will be used. Results will be analyzed using students''t' test the probability (p –value) will be calculated.

*Conflict of interest if any - Nil

*Privacy/confidentiality of study subjects - Maintained

*Sponsor details -Not Applicable

*Compensation - Not Applicable

***Insurance -** Not Applicable

STATISTICAL ANALYSIS

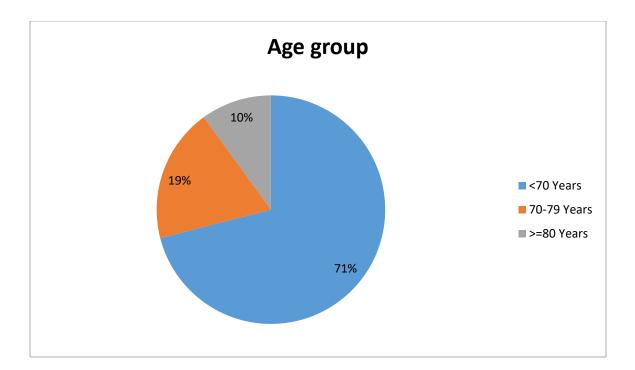
Frequency Table

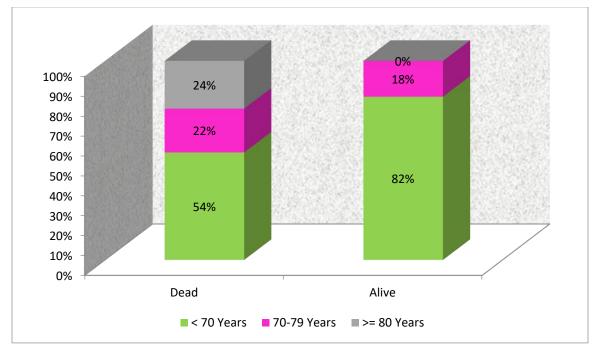
AGE GROUP	FREQUENCY	PERCENT
<70 Years	95	70.9
70-79 Years	26	19.4
>=80 Years	13	9.7
Total	134	100.0

			90th_DAY_		Total
			Dead	Alive	
	70.)/	Count	30	65	95
	<70 Years	%	54.5%	82.3%	70.9%
age_group	70-79 Years	Count	12	14	26
		%	21.8%	17.7%	19.4%
	>=80 Years	Count	13	0	13
		%	23.6%	0.0%	9.7%
Total		Count	55	79	134
TOTAL		%	100.0%	100.0%	100.0%

Pearson Chi-Square=22.471**p<0.001

- Among the total population 71% were of less than 70 yrs
- 19% were between 70 to 79 yrs
- 10% were above 80 yrs of age





- All the patients >80 yrs of age presenting with ICH did not survive at the end of 90 days
- 82% of the people <70 yrs of age survived at the end of 90 days.
- Age has a significant impact on the outcome of patients presenting with ICH.

SEX DISTRIBUTION

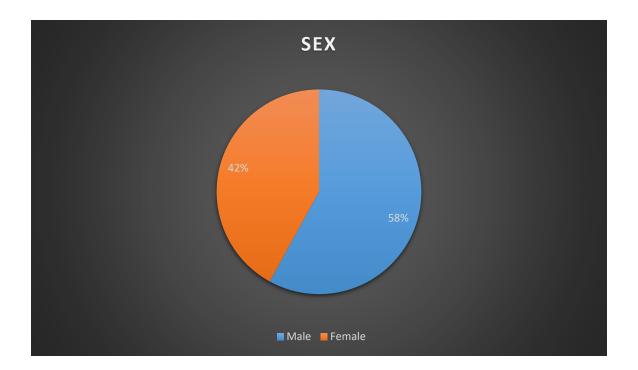
Frequency table:

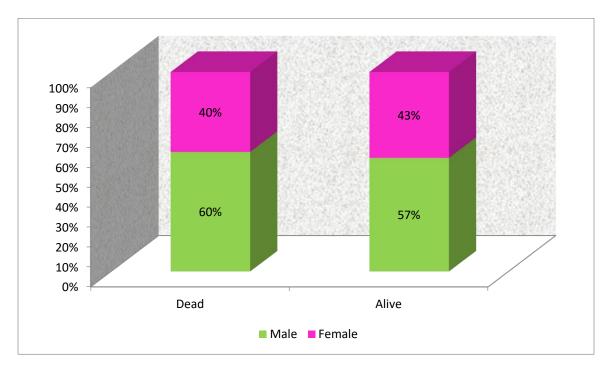
SEX	Frequency	Percent
Male	78	58.2
Female	56	41.8
Total	134	100.0

	Crosstab					
			90th_DAY_	90th_DAY_OUTCOME		
			Dead	Alive		
Male SEX	N. 1	Count	33	45	78	
	%	60.0%	57.0%	58.2%		
	Count	22	34	56		
	Female	%	40.0%	43.0%	41.8%	
Total		Count	55	79	134	
Total		%	100.0%	100.0%	100.0%	

Pearson Chi-Square=0.123 p=0.726

- Of the total 134 population 58% were male and 42% were female indicating a male predominance in the people presenting with ICH
- This can also be attributed to the overall male population more than female population





- The outcome or FUNC score is independent of sex
- <u>Sex doesnot have a significant impact on the outcome of patients</u>

presenting with ICH

ROLE OF HYPERTENSION

Frequency table:

SHT	Frequency	Percent
No	83	61.9
Yes	51	38.1
Total	134	100.0

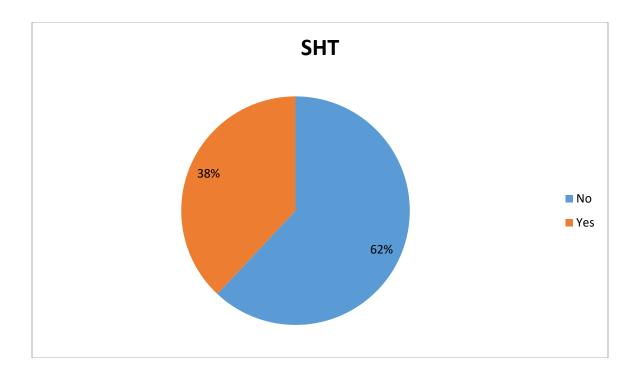
	Crosstab					
			90th_DAY_	OUTCOME	Total	
			Dead	Alive		
SHT	N	Count	28	55	83	
	No	%	50.9%	69.6%	61.9%	
	Vac	Count	27	24	51	
	Yes	%	49.1%	30.4%	38.1%	
Total		Count	55	79	134	
Total		%	100.0%	100.0%	100.0%	

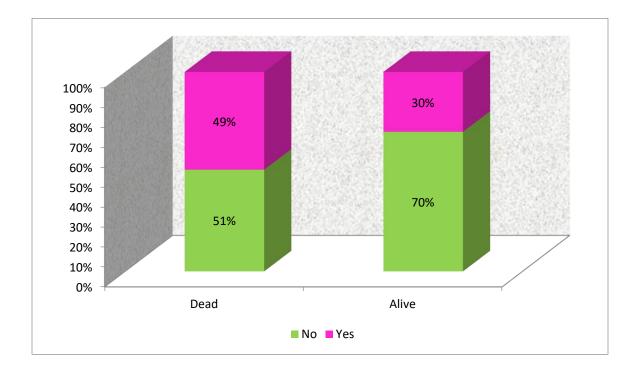
Pearson Chi-Square=4.816* p=0.028

All the patients (100%) admitted for ICH had high blood pressure of whom 28 patients (20%) have systolic BP >200mmHg.

Only 50 patients (38%) of the total subjects were known hypertensive.

If the remaining patients were screened for hypertension in their early life and started on anti hypertensive drugs, they would have prevented the event.





This data suggest the importance of screening for hypertension and an early intervention would have prevented them from this devastating event.

DIABETES MELLITUS

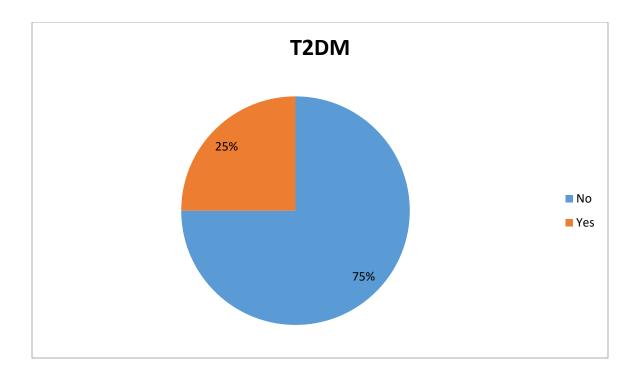
Frequency table:

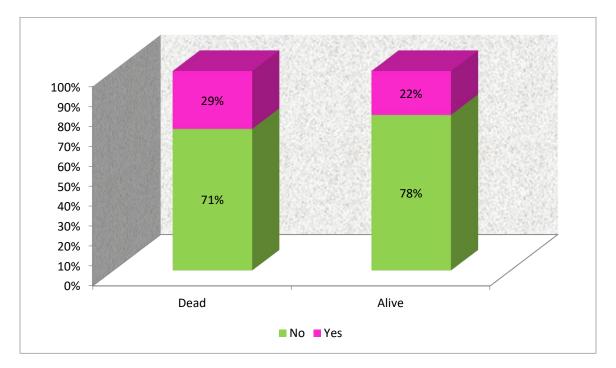
T2DM	Frequency	Percent
No	101	75.4
Yes	33	24.6
Total	134	100.0

	Crosstab					
			90th_DAY_	OUTCOME	Total	
			Dead	Alive		
	Count	39	62	101		
	No T2DM	%	70.9%	78.5%	75.4%	
		Count	16	17	33	
	Yes	%	29.1%	21.5%	24.6%	
Total	Count	55	79	134		
TUIAI		%	100.0%	100.0%	100.0%	

Pearson Chi-Square=1.002 p=0.317

- Of the total population, 24% of the population had a previous history of diabetes mellitus
- Of them 55% expired at the end of 90 days





• Even though there is no significant correlation with diabetes and outcome of the patient, patient's presenting with high CBG at the time of presentation had poor outcome than that of them with normal sugar levels.

CARONARY ARTERY DISEASE

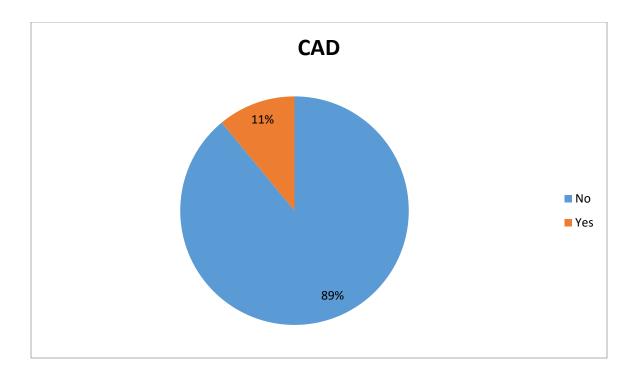
Frequency table:

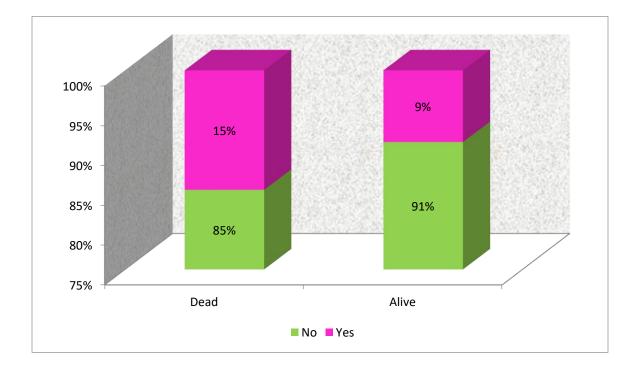
CAD	Frequency	Percent
No	119	88.8
Yes	15	11.2
Total	134	100.0

Crosstab						
	90th_DAY_OUTCOME			Total		
	Dead Alive					
	Na	Count	47	72	119	
CAD	No	%	85.5%	91.1%	88.8%	
CAD	Vac	Count	8	7	15	
	Yes	%	14.5%	8.9%	11.2%	
Total		Count	55	79	134	
		%	100.0%	100.0%	100.0%	

Pearson Chi-Square=1.054 p=0.305

- Of the total population, 11% of the population had a previous episode of coronary artery disease.
- Of them 53% expired at the end of 90 days





• Previous episodes of coronary artery disease doesn't have a significant outcome in patients presenting with ICH

PRIOR ANTI PLATELET THERAPY

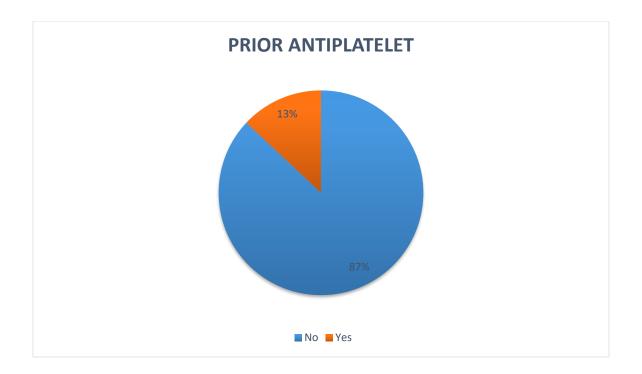
Frequency table

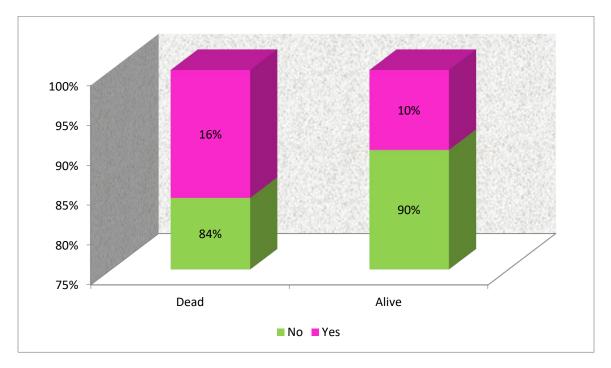
PRIOR_ANTIPLATELET	Frequency	Percent
No	117	87.3
Yes	17	12.7
Total	134	100.0

Crosstab						
			90th_DAY_	OUTCOME	Total	
			Dead	Alive		
	Na	Count	46	71	117	
DDIOD ANTIDI ATELET	No	%	83.6%	89.9%	87.3%	
PRIOR_ANTIPLATELET	Yes	Count	9	8	17	
		%	16.4%	10.1%	12.7%	
Total		Count	55	79	134	
10101		%	100.0%	100.0%	100.0%	

Pearson Chi-Square=1.139 p=0.286

- Of the total population, 12% of the population had a previous history of consumption of anti platelet drugs
- The indication of anti platelet were not considered
- Those taking the drugs daily for atleast 6 months were taken into account.
- Of them 52% expired at the end of 90 days





• Previous episodes of anti-platelet use doesn't have a significant impact on the outcome in patients presenting with ICH

DURATION AT THE TIME OF PRESENTATION

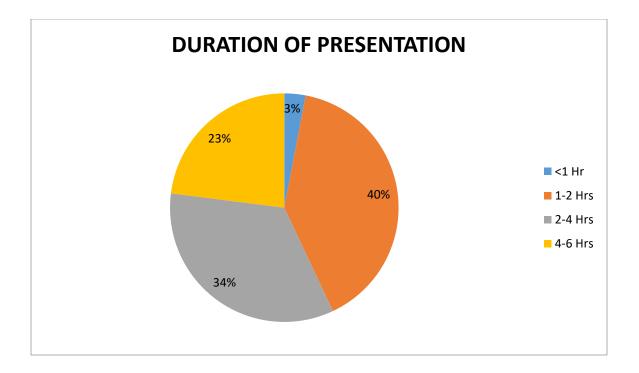
Frequency table:

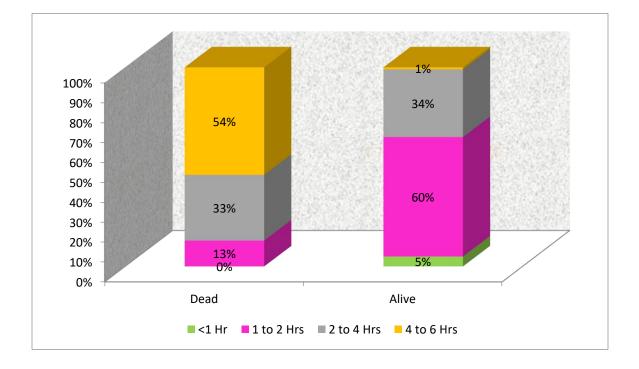
duration_group	Frequency	Percent
<1 Hr	4	3.0
1-2 Hrs	54	40.3
2-4 Hrs	45	33.6
4-6 Hrs	31	23.1
Total	134	100.0

Crosstab					
			90th_DAY_	OUTCOME	Total
			Dead	Alive	
	- _1 II.n	Count	0	4	4
	<1 Hr	%	0.0%	5.1%	3.0%
	1-2 Hrs	Count	7	47	54
Duration moun		%	12.7%	59.5%	40.3%
Duration group	2-4 Hrs	Count	18	27	45
		%	32.7%	34.2%	33.6%
	4-6 Hrs	Count	30	1	31
		%	54.5%	1.3%	23.1%
Total		Count	55	79	134
10(a)		%	100.0%	100.0%	100.0%

Pearson Chi-Square=60.191** p<0.001

- Among the study group >50% of the people presented only after 2 hrs
- 23% of the study population presented very lately of 4-6 hrs
- Of the 58people presenting within 2 hrs only 4 expired at the end of 90days





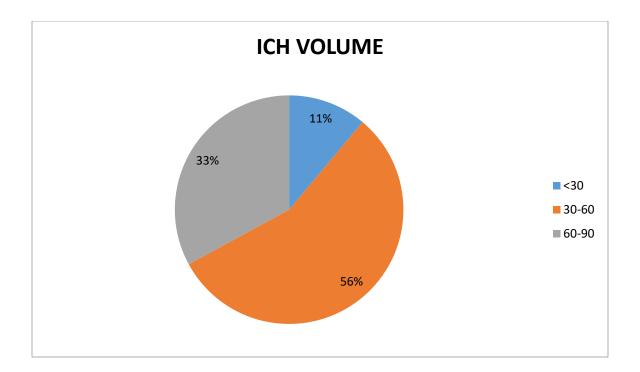
• Duration of presentation has a significant impact on the outcome of patients presenting with ICH, early the presentation better the outcome.

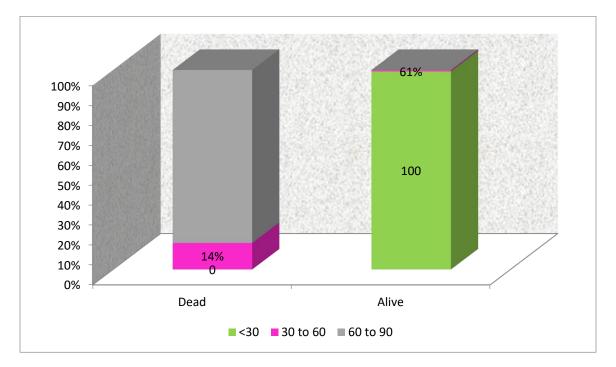
ICH VOLUME

Ich_volume	Frequency	Percent
<30	15	11.1
30-60	75	55.9
60-90	44	32.9
Total	134	100.0

Crosstab					
			90th_DAY_	OUTCOME	Total
			Dead	Alive	
	-20	Count	0	15	15
<30 ICH 20.60	<30	%	0%	100%	11%
	20.60	Count	14	61	75
volume	30-60	%	18.6%	81.4%	56%
	60-90	Count	40	4	44
	00-90	%	90.9%	10.1%	33%
Total		Count	55	79	134
Total		%	100.0%	100.0%	100.0%

- Pearson Chi-Square=70.184** p<0.001
- Among the 134 population, 15 patients had ICH volume of <30ml and all of them survived at the end of 90 days
- Of the 44 patients presenting with ICH volume >60ml, 40(90%)expired





• <u>ICH volume has a significant impact on the outcome of patients presenting with</u>

ICH; smaller the volume, better the outcome.

Ich LOCATION

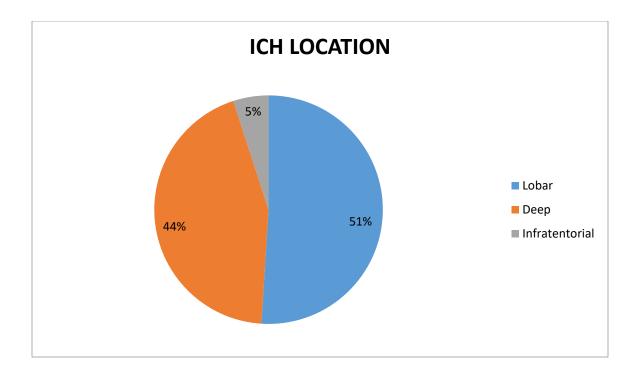
Frequency table

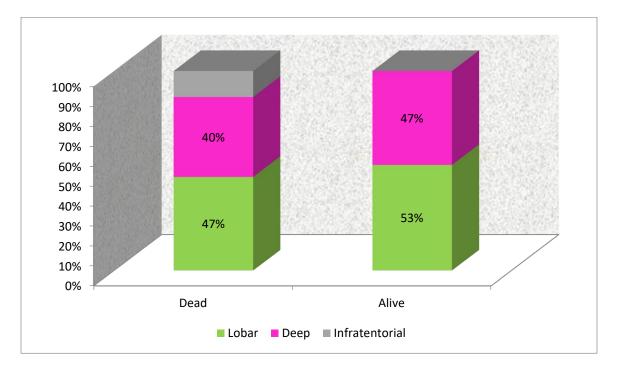
ICH_LOCATION	Frequency	Percent
Lobar	68	50.7
Deep	59	44.0
Infratentorial	7	5.2
Total	134	100.0

Crosstab						
			90th_DAY_	OUTCOME	Total	
			Dead	Alive		
	Lohor	Count	26	42	68	
	Lobar	%	47.3%	53.2%	50.7%	
ICH LOCATION	Deep Infratentorial	Count	22	37	59	
ICH_LOCATION		%	40.0%	46.8%	44.0%	
		Count	7	0	7	
		%	12.7%	0.0%	5.2%	
Total		Count	55	79	134	
10141		%	100.0%	100.0%	100.0%	

Pearson Chi-Square=10.620** p=0.005

- 50% of the study group presented as lobar bleed, of them 61% survived after 90 days, 61% of deep bleed also survived.
- Whereas all the 7(100%) who had infratentorial bleed expired after 90 days
- This data suggest that infratentorial bleed has 100% mortality rate.





• ICH location has a significant impact on the outcome of patients presenting with

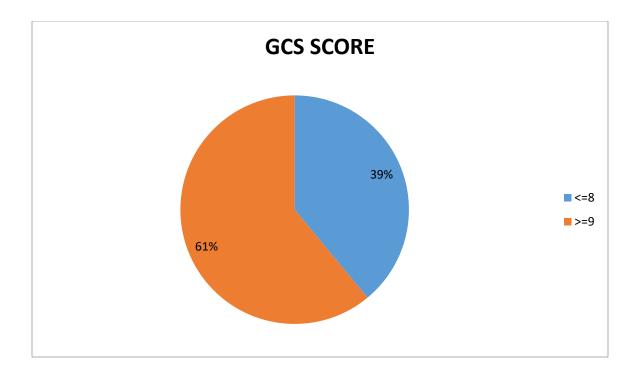
ICH; lobar and deep bleeds have better prognosis than infratentorial bleed.

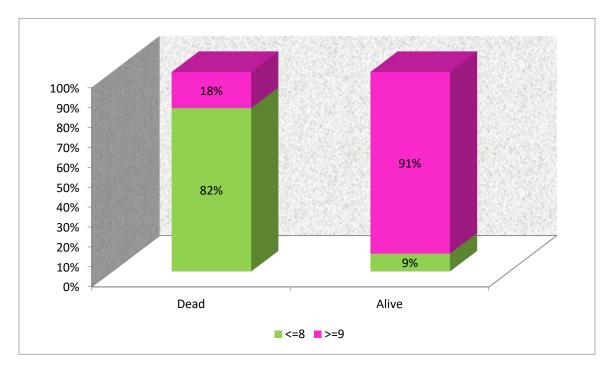
GCS_score	Frequency	Percent
<=8	52	38.8
>=9	82	61.2
Total	134	100.0

Crosstab					
			90th_DAY_OUTCOME		Total
			Dead	Alive	
	0	Count	45	7	52
<=8 gcs_score	<-0	%	81.8%	8.9%	38.8%
	>=9	Count	10	72	82
	>=9	%	18.2%	91.1%	61.2%
Total		Count	55	79	134
10101		%	100.0%	100.0%	100.0%

Pearson Chi-Square=72.680** p<0.001

- Of the total study group around 39% had GCS less than 8, of them 86% expired after 90 days
- Among the 82 people (61% of the study group)presenting with GCS >=9, 87% were alive after 90 days; signifying the importance of GCS score.
- GCS at the time of presentation makes an important predictor in the outcome and is the most important component of the FUNC score





• GCS score has a significant impact on the outcome of patients presenting with

ICH; GCS>=9 have better prognosis and GCS<=8 has poor prognosis.

PRE ICH COGNITIVE IMPAIRMENT

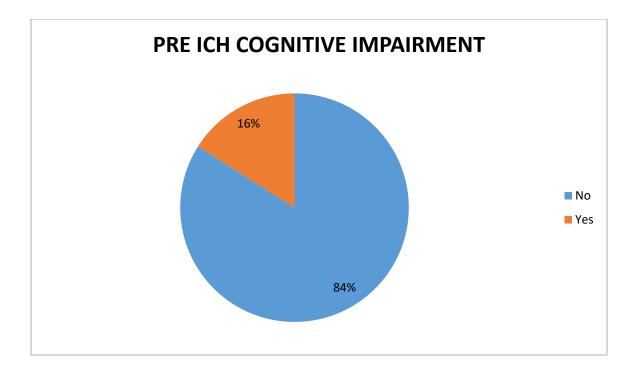
Frequency table

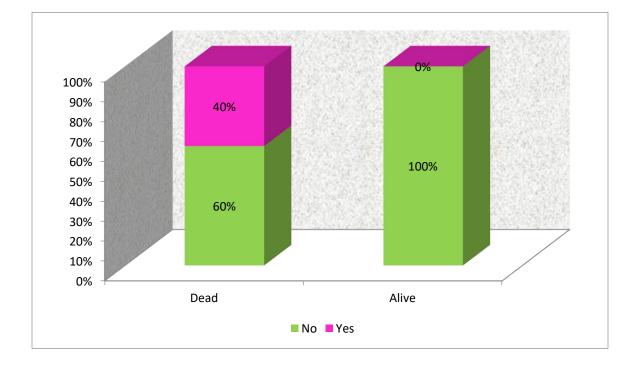
PRE_ICH_COGNITIVE_IMPAIRMENT	Frequency	Percent
No	112	83.6
Yes	22	16.4
Total	134	100.0

Crosstab						
			90th_DAY_	OUTCOME	Total	
			Dead	Alive		
	Na	Count	33	79	112	
PRE_ICH_COGNITI VE_IMPAIRMENT Ye	INO	%	60.0%	100.0%	83.6%	
	Vaa	Count	22	0	22	
	res	%	40.0%	0.0%	16.4%	
Total		Count	55	79	134	
10101		%	100.0%	100.0%	100.0%	

Pearson Chi-Square=37.807** p<0.001

- Among the total population 22 people had pre ICH cognitive impairment, all of them expired at the end of 90 days
- Of the 112 without cognitive impairment, 70% were alive at the end of 90days signifying the importance of pre ICH cognitive impairment





Pre ICH cognitive impairment has a significant impact on the outcome of patients presenting with ICH; patients with cognitive impairment have poor prognosis.

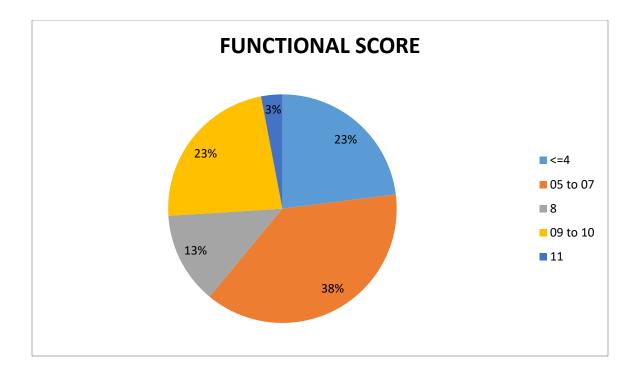
FUNC SCORE

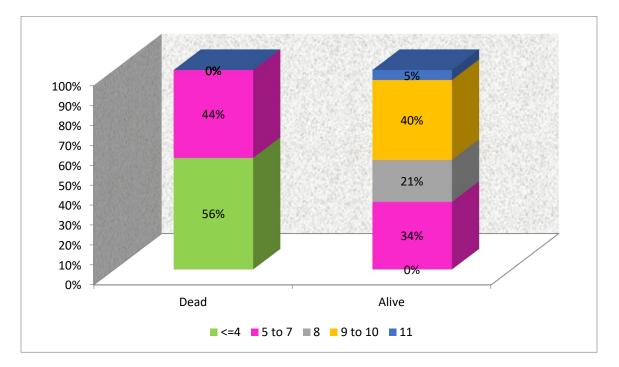
FUNC_score	Frequency	Percent
<=4	31	23.1
5-7	51	38.1
8	17	12.7
9-10	31	23.1
11	4	3.0
Total	134	100.0

Crosstab					
			90th_DAY_	OUTCOME	Total
			Dead	Alive	
		Count	31	0	31
	<=4	%	56.4%	0.0%	23.1%
	5-7	Count	24	27	51
	5-7	%	43.6%	34.2%	38.1%
FUNC	8	Count	0	17	17
FUNCscore		%	0.0%	21.5%	12.7%
	9-10	Count	0	31	31
		%	0.0%	39.2%	23.1%
	11	Count	0	4	4
11	11	%	0.0%	5.1%	3.0%
Total		Count	55	79	134
		%	100.0%	100.0%	100.0%

Pearson Chi-Square=81.492** p<0.001

• All patients with FUNC score<4expired and all patients with FUNC score >8 were alive at the end of 90 days





• FUNC score has a significant impact on the outcome of patients presenting with

ICH; FUNC>=8 have better prognosis and FUNC<=4 has poor prognosis.

MIDLINE SHIFT

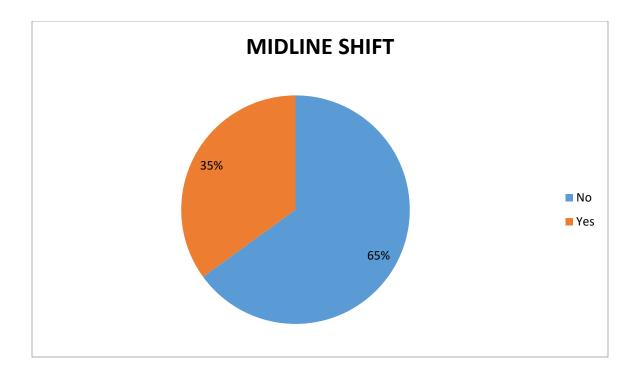
Frequency table

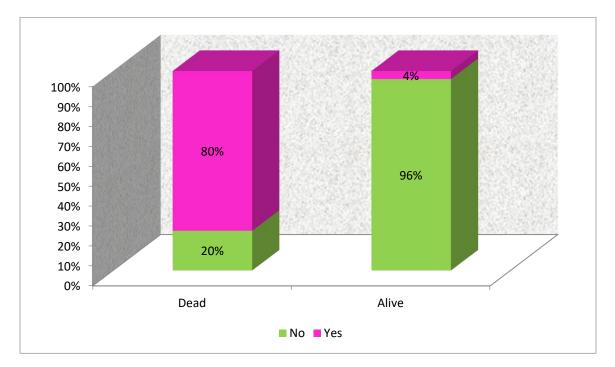
MIDLINE_SHIFT	Frequency	Percent
No	87	64.9
Yes	47	35.1
Total	134	100.0

Crosstab					
90th_DAY_OUTCOME		Total			
			Dead	Alive	
	No	Count	11	76	87
MIDLINE SHIFT	No	%	20.0%	96.2%	64.9%
	Yes	Count	44	3	47
		%	80.0%	3.8%	35.1%
Total		Count	55	79	134
10141		%	100.0%	100.0%	100.0%

Pearson Chi-Square=82.683** p<0.001

- Among the 134 subjects 47 presented with midline shift, among those 47 subjects 44 expired at the end of 90 days
- Among the 87subjects who don't had midline shift, 76 were alive at the end of 90 days





• <u>Midline shift has a significant impact on the outcome of patients presenting with</u> <u>ICH; patients with midline shift has poor prognosis and patients without midline</u> <u>shift have better prognosis.</u>

INTRAVENTRICULAR EXTENSION

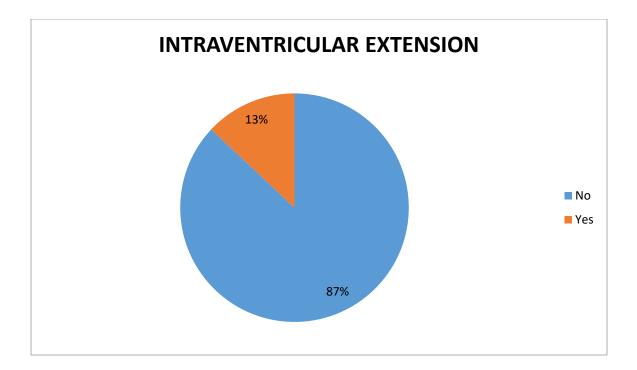
Frequency table:

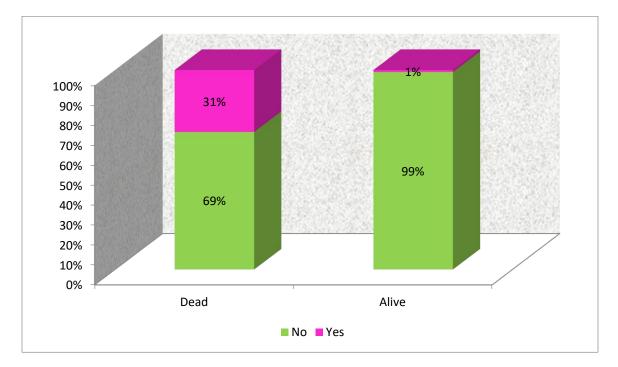
INTRAVENTRICULAR_EXTENSION	Frequency	Percent
No	116	86.6
Yes	18	13.4
Total	134	100.0

Crosstab						
			90th_DAY_	OUTCOME	Total	
			Dead	Alive		
	No	Count	38	78	116	
INTRAVENTRICUL AR EXTENSION	No	%	69.1%	98.7%	86.6%	
	V	Count	17	1	18	
	Yes	%	30.9%	1.3%	13.4%	
Total		Count	55	79	134	
Total		%	100.0%	100.0%	100.0%	

Pearson Chi-Square=24.503** p<0.001

- Among the 134 subjects 18 presented with intraventricular extension, among those 18 subjects 17 expired at the end of 90 days
- Among the 116subjects who don't had intraventricular extension, 78 were alive at the end of 90 days





Intra ventricular extension has a significant impact on the outcome of patients presenting with ICH; patients with Intra ventricular extension has poor prognosis and patients without Intra ventricular extension have better prognosis.

INTERVENTION

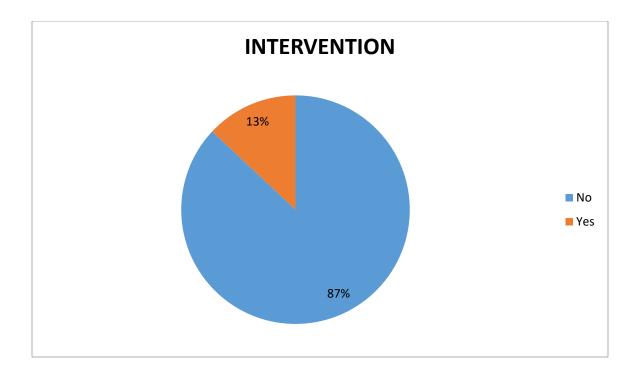
Frequency table:

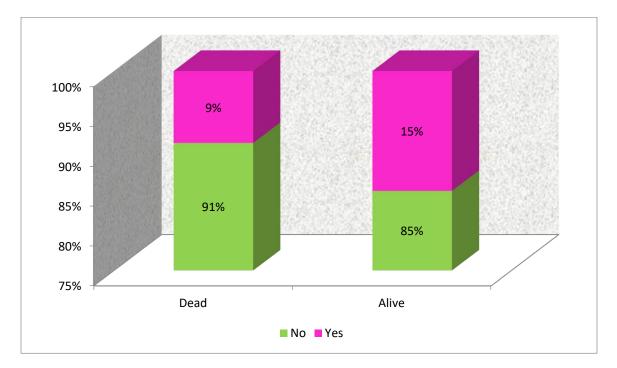
INTERVENTION	Frequency	Percent
No	117	87.3
Yes	17	12.7
Total	134	100.0

Crosstab					
			90th_DAY_	OUTCOME	Total
			Dead	Alive	
INTERVENTIO	No	Count	50	67	117
	No	%	90.9%	84.8%	87.3%
Ν	Vac	Count	5	12	17
	Yes	%	9.1%	15.2%	12.7%
Total		Count	55	79	134
10(a)		%	100.0%	100.0%	100.0%

Pearson Chi-Square=1.089 p=0.297

- Among the 134 subjects 17 underwent surgery as a part of theirtreatment, among those 17 subjects 5patients(30%) expired at the end of 90 days
- Among the 117subjects who don't had intervension, 67(57%) were alive at the end of 90 days
- Inspite of intervension, there happens only a little change in the outcome of the patients.



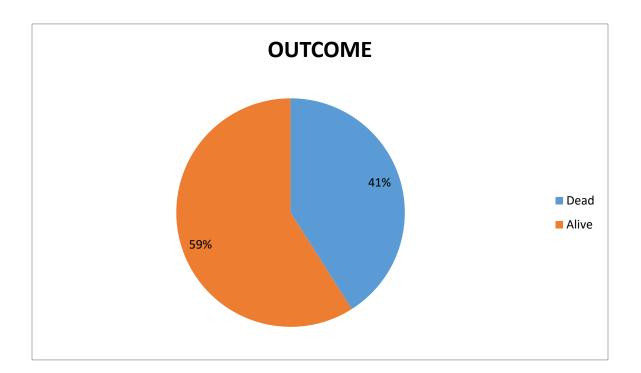


• Surgical intervension doesn't have any major effect on the outcome of patients compared to those who were managed conservatively.

OUTCOME

Frequency table:

	Frequency	Percent
Dead	55	41.0
Alive	79	59.0
Total	134	100.0

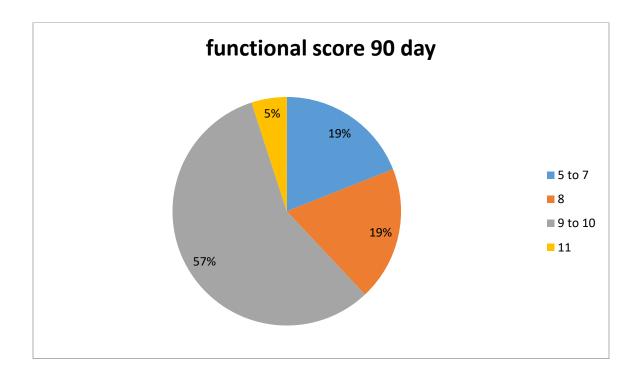


Among the total 134 subjects 79 were alive at the end of 90 days and 55 expired at the end of 90 days. The outcome is independent on the mode of management. Func score at the time of admission is compared to those with dead and those with alive.

90TH DAY FUNC SCORE

Frequency table:

func_score_90_day	Frequency	Percent
5-7	15	19.0
8	15	19.0
9-10	45	57.0
11	4	5.1
Total	79	100.0



At the end of 90 deays FUNC score was allotted for the alive patients. 57% had a score of 9 to 10 and 19% had a score of 8. Overall 81%(score >8) had a good independent outcome.

90TH DAY GCS

GCS_90DAY

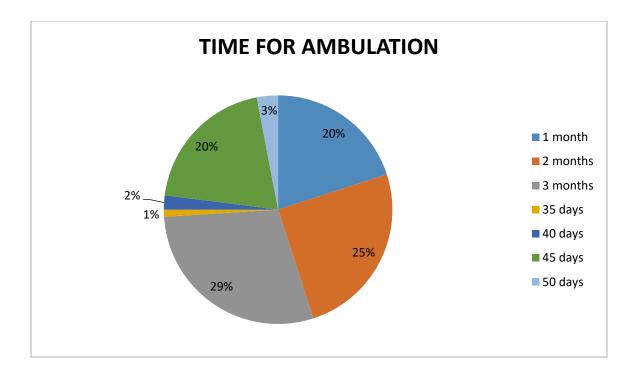
	Frequency	Percent	Valid Percent	Cumulative Percent
Valid	134	100.0	100.0	100.0

90th_DAY_GCS	Frequency	Percent
10	9	11.4
11	9	11.4
12	9	11.4
13	11	13.9
14	16	20.3
15	13	16.5
7	1	1.3
9	11	13.9
Total	79	100.0

 Among the 79 persons who were alive at the end of 90 days, only one had a GCS<=7. 11 people had GCS 9. Remaining 67 had GCS>=10 and had an independent outcome.

TIME FOR AMBULATION

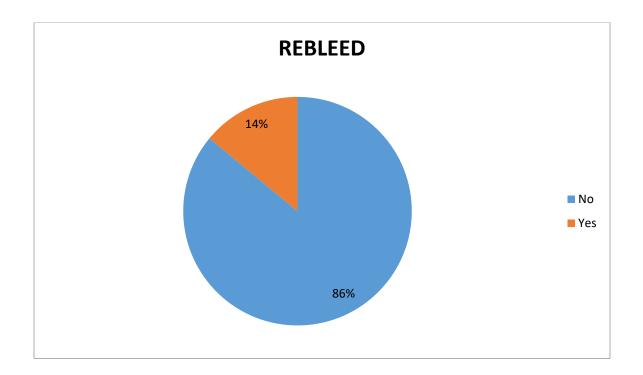
TIME_FOR_AMBULATION	Frequency	Percent
1 month	16	20.3
2 months	20	25.3
3 months	23	29.1
35 days	1	1.3
40 days	1	1.3
45 days	16	20.3
50 days	2	2.5
Total	79	100.0



- 29% of the alive population took 3 months for ambulation with support.
- Ambulation depends on lotof factors and is found to vary from person to person and is independent of the FUNC score or GCS score or ICH volume.
- Younger patients were found to be early ambulant when compared to older people.

REBLEED

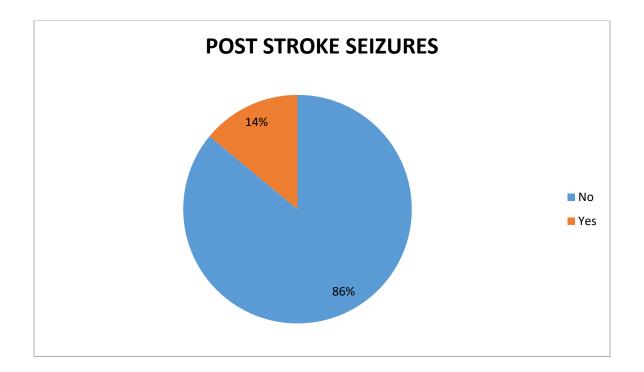
REBLEED	Frequency	Percent
No	68	86.1
Yes	11	13.9
Total	79	100.0



- Among the 79 alive patients, 11 persons had rebleed and found to have poor compliance with treatment such as antihypertensives. All were functionally dependent.
- Strict adherent to drugs would have prevented them from recurrence.

POST STROKE SEIZURES

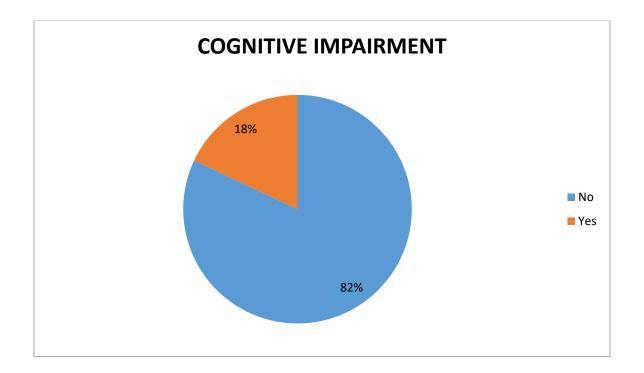
POST_STROKE_SEIZURES	Frequency	Percent
No	68	86.1
Yes	11	13.9
Total	79	100.0



• Among the 79 alive patients, 11 persons had seizures and among them 10 were those with re-bleeding. Only 1 among the non re-bleeders had seizures within the 90 day period. There is a chance of seizures in all the alive patients and were started on anti-epileptic prophylaxis

COGNITIVE IMPAIRMENT

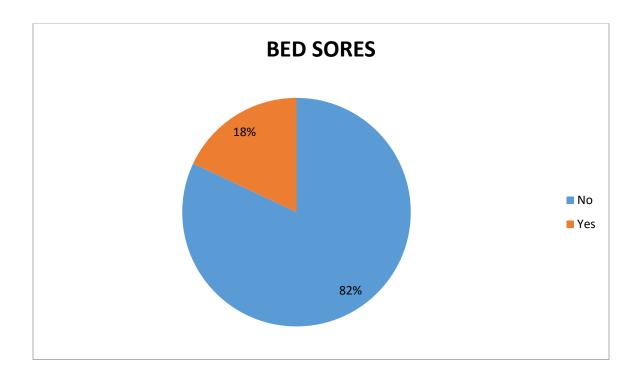
COGNITIVE_IMPAIRMENT	Frequency	Percent
No	65	82.3
Yes	14	17.7
Total	79	100.0



 Among the 79 alive patients, 14 persons had cognitive impairment and among them 9 were those with GCS 8. Only 5 among the GCS>=9 had cognitive impairment within the 90 day period.

BED SORES

BED_SORES	Frequency	Percent
No	65	82.3
Yes	14	17.7
Total	79	100.0



 Among the 79 alive patients, 14 persons had bed sores and among them 9 were those with GCS 8. Only 5 among the GCS>=9 had bed sores within the 90 day period.

RESULTS

Of the total 134 cases in the study, whom were selected as per inclusion and exclusion criteria, 79 patients survived (59 %) and 55 patients (41 %) expired during the hospital stay period or within 90 day period of follow up.

There were 78 males (58.2%) and 56 females (41.8%). Among the expired, majority were males (60%), which did not bear any significance of gender with prognosis (p=0.726)

The mean age of patients in both the survivor and expired group was 52 years. In the age group \leq =70 years, there were 95 patients; 26 patients in age group 70-79 years and 13 patients in age group \geq =80 years.

Among the total expired patients , 54.5 % belonged to the age group of 70<=years, 21.8% belong to 70-79 years, 23.6% belong to >80 years of age. There is 100% mortality in the age group of >80 years and 85.7% mortality in age group of 70-79 years which shows significance of age in prognosis (p< 0.001).

In the analysis of FUNC scoring, among the expired patients 31 patients(56.4%) belonged to score <=4 ; 24 patients (43.6%) belonged to score 5-7; and all the patients presenting with FUNC score of >=8 were alive at the end of 90 days which shows high significance of FUNC scoring(p<0.001) in predicting the outcome.

84

On assessing the volume of ICH 15 patients (11%) were in $<30 \text{ mm}^3$, 75 patients (56%) were having 30-60mm³, 44 patients (33%) were having volume of 60-90 mm³. Of the expired patients 91% were having ICH volume of 60-90 mm³. All the patients having ICH volume of $<30 \text{ mm}^3$ were alive shoeing the significance of ICH volume(p<0.001)

In analysis of ICH location, among patients who expired 26 patients (47.3%%) were having lobar bleed, 22 patients(40%) werehaving deep bleed, 7 patients (12.7%) were having infra tentorial bleed. 100% of people with infratentorial bleed expired and 50.7% of the lobar bleed were alive showing significance(p=0.005)

Of the total study group around 39% had GCS less than 8, of them 86% expired after 90 days. Among the 82 people (61% of the study group) presenting with GCS >=9, 87% were alive after 90 days; signifying the importance of GCS score(p<0.001)

Among the study group >50% of the people presented only after 2 hrs 23% of the study population presented very lately of 4-6 hrs, 33.6% presented after 2 -4 hrs, 40.3% presented within1-2 hrs. Of the 58 people presenting within 2 hrs only 4 expired at the end of 90days signifying the importance of duration of presentation.

85

Among the total population 22 people had pre ICH cognitive impairment, all of them expired at the end of 90 days Of the 112 without cognitive impairment, 70% were alive at the end of 90days signifying the importance of pre ICH cognitive impairment.

Among the 134 subjects 17 underwent surgery as a part of theirtreatment, among those 17 subjects 5patients(30%) expired at the end of 90 days. Among the 117subjects who don't had intervension, 67(57%) were alive at the end of 90 days. Inspite of intervension, there happens only a little change in the outcome of the patients.

DISCUSSION

Flaherty ML, Woo D, Haverbusch M, et al. states that At one year, mortality ranges from 51% to 65% depending on the location of the hemorrhage.¹¹ which slightly correlates with this study that the mortality at 90 days is 41% and may rise at the end of one year.

Brott T, Broderick J, Kothari R, et al. states that Hematoma expansion, highly associated with clinical deterioration and poor outcomes, is evident in nearly 40% of cases within the first 3 hours after onset of symptoms is also well-documented with CT scanning.^{12,13} correlates with this study that longer the duration of presentation poorer the outcome.

Broderick J, Brott T, Duldner JE, et al. states that The volume of the ICH and the clinical grade on the Glasgow Coma Scale on admission are the most powerful predictors of 30-day mortality.^{14,30} which correlates with this study that GCS and ICH volume are the two most important factors contributing to the FUNC score in prediction of outcome after 90 days.

Broderick J, Connolly S, Feldmann E, et al. states that Hemispheric lesions >30 cc have a high mortality rate and Patients with GCS <9 and hematoma >60 cc have a 90% mortality rate.¹⁵

87

Mendelow AD, Gregson BA, Fernandes HM, et al. proposed in the STITCH trial that Surgery doesn't appear to be useful in most cases and is possibly harmful in persons presenting in coma¹⁶ which is similar to this study that surgical intervention doesn't modify the outcome of the patient in such a way that only a very few patients had benefit on the performance of surgery.

Woo D, Haverbusch M, Sekar P, et al. states that proper control of hypertension can result in reduction of re-bleed²⁹ which is similar to this study result that patient who did not take proper anti-hypertensive medication tend to had re-bleed.

CONCLUSION

This study concludes that FUNC score can be used as a reliable tool in predicting the outcome of patients presenting with primary intracerebral hemorrhage. By using this scale withdrawal of care for patients with predicted good outcome can be prevented and can lead to a reduction in mortality and help them in early mobilisation.

Even though a number of factors are involved in predicting the outcome of patients with ICH, GCS of the patient and volume of ICH remains the two most important factors in predicting the outcome.

Other factors that are reliable in predicting the outcome are age, duration of presentation, pre ICH cognitive impairment, location of ICH.

Early screening for hypertension, proper intake of anti-hypertensive medication, adequate control of blood pressure will prevent such catastrophe and can also reduce the episodes of rebleeding in patients presenting with ICH.

BIBLIOGRAPHY

1. Caplan L. Intracerebral haemorrhage. Lancet. 1992;14:656 – 58.

2. Gebel JM, Broderick JP. Intracerebral hemorrhage. Neurol Clin. 2000; 18:419 – 38.

3. Broderick JP, Adams HP Jr, Barsan W, Feinberg W, Feldmann E, Grotta J, Kase C, Krieger D, Mayberg M, Tilley B, Zabramski JM, Zuccarello M. Guidelines for the management of spontaneous intracerebral hemorrhage: a statement for healthcare professionals from a special writing group of the Stroke Council, American Heart Association. Stroke. 1999; 30:905–15.

4. Flaherty ML, Kissela B, Woo D, Kleindorfer D, Alwell K, Sekar P, Moomaw CJ, Haverbusch M, Broderick JP. The increasing incidence of anticoagulant-associated intracerebral hemorrhage. Neurology. 2007;68: 116–21.

5. Hemphill JC III, Newman J, Zhao S, Johnston SC. Hospital usage of early do-notresuscitate orders and outcome after intracerebral hemorrhage. Stroke. 2004;35:1130–34

6. Becker KJ, Baxter AB, Cohen WA, Bybee HM, Tirschwell DL, Newell DW, Winn HR, Longstreth WT Jr. Withdrawal of support in intracerebral hemorrhage may lead to self-fulfilling prophecies. Neurology. 2001;56: 766–72.

 Cordonnier C, Brainin M. Better scoring for better care? J Neurol Neurosurg Psychiatry. 2006;77:571 8. kothari RU. Et al. the ABC of measuring intracerebral haemorrhage volumes, stroke.
 1996;27(8):1304-5

9. O'Donnell HC, Rosand J, Knudsen KA, Furie KL, Segal AZ, Chiu RI, Ikeda D, Greenberg SM. Apolipoprotein e genotype and the risk of recurrent lobar intracerebral hemorrhage. N Engl J Med. 2000;342: 240 –245.

10. Rosand J, Eckman MH, Knudsen KA, et al. The Effect of Warfarin and Intensity of Anticoagulation on Outcome of Intracerebral Hemorrhage. Archives of Int Med. 2004 Apr 26;164(8):880–884.

11. Flaherty ML, Woo D, Haverbusch M, et al. Racial Variations in Location and Risk of Intracerebral Hemorrhage. Stroke. 2005;36(5):934–937.

12. Brott T, Broderick J, Kothari R, et al. Early Hemorrhage Growth in Patients with Intracerebral Hemorrhage. Stroke. 1997;28(1):1–5.

13. Hill MD, Silver FL, Austin PC, et al. Rate of Stroke Recurrence in Patients with Primary Intracerebral Hemorrhage. Stroke. 2003;31(1):123–127.

14. Broderick J, Brott T, Duldner JE, et al. Volume of Intracerebral Hemorrhage: APowerful and Easy to Use Predictor of 30-day Mortality. Stroke. 1993;24(7):987–993.

15. Broderick J, Connolly S, Feldmann E, et al. Guidelines for the Management of Spontaneous Intracerebral Hemorrhage in Adults: 2007 Update: A Guideline from the American Stroke Association Stroke Council. Stroke. 2007;38:2001–23. 16. Mendelow AD, Gregson BA, Fernandes HM, et al. Early Surgery versus Initial
Conservative Treatment in Patients with Spontaneous Supratentorial Intracerebral
Hemorrahge in the International Surgical Trial in Intracerebral Hemorrhage (STICH): A
Randomized Trial. Lancet. 2005 Jan 29;365(9457):387–397.

17. Mayer S, Brun NC, Begtrup K, et al. Recombinant Activated Factor VII for Acute Intracerebral Hemorrahge. New England JMed. 2005 Feb 24;352(8):777–785.

18. Zahuranec DB, Brown DL, Lisabeth LD, Gonzales NR, Longwell PJ, Smith MA, Garcia NM, Morgenstern LB. Early care limitations independently predict mortality after intracerebral hemorrhage. Neurology. 2007; 68:1651–1657

19. Gage BF, Cardinalli AB, Owens DK. The effect of stroke and stroke prophylaxis with aspirin or warfarin on quality of life. Arch Intern Med. 1996;156:1829–1836.

20. Ciccone ASR, Crespi V, Defanti C, Pasetti C. Thrombolysis for acute ischemic stroke: the patient's point of view. Cerebrovasc Dis. 2001;12: 335–340.

21. Gage BF, Cardinalli AB, Owens DK. The effect of stroke and stroke prophylaxis with aspirin or warfarin on quality of life. Arch Intern Med. 1996;156:1829 –36.

22. Hemphill JC III, Bonovich DC, Besmertis L, Manley GT, Johnston SC, Tuhrim S. The ICH score: a simple, reliable grading scale for intracerebral hemorrhage editorial comment: a simple, reliable grading scale for intracerebral hemorrhage. Stroke. 2001;32:891–897.

23. Ariesen MJ, Algra A, van der Worp HB, Rinkel GJE. Applicability and relevance of models that predict short term outcome after intracerebral haemorrhage. J Neurol Neurosurg Psychiatry. 2005;76:839 – 844.

24. Lisk DR, Pasteur W, Rhoades H, Putnam RD, Grotta JC. Early presentation of hemispheric intracerebral hemorrhage: prediction of outcome and guidelines for treatment allocation. Neurology. 1994;44:133–139.

25. Portenoy RK, Lipton RB, Berger AR, Lesser ML, Lantos G. Intracerebral haemorrhage: a model for the prediction of outcome. J Neurol Neurosurg Psychiatry. 1987;50:976–979.

26. Flaherty ML, Haverbusch M, Sekar P, Kissela B, Kleindorfer D, Moomaw CJ, Sauerbeck L, Schneider A, Broderick JP, Woo D. Long-term mortality after intracerebral hemorrhage. Neurology. 2006;66:1182–1186. 2308 Stroke August 2008 Downloaded from http://ahajournals.org by on October 19, 2019

27. Godoy DA, Pinero G, Di Napoli M. Predicting mortality in spontaneous intracerebral hemorrhage: can modification to original score improve the prediction? Stroke. 2006;37:1038–1044.

28. Ruiz-Sandoval JL, Chiquete E, Romero-Vargas S, Padilla-Martinez JJ, Gonzalez-Cornejo S. Grading scale for prediction of outcome in primary intracerebral hemorrhages. Stroke. 2007;38:1641–1644. 29. Takahashi O, Cook EF, Nakamura T, Saito J, Ikawa F, Fukui T. Risk stratification for in-hospital mortality in spontaneous intracerebral haemorrhage: a classification and regression tree analysis. QJM. 2006;99: 743–750.

30. Tuhrim S, Dambrosia JM, Price TR, Mohr JP, Wolf PA, Hier DB, Kase CS.
Intracerebral hemorrhage: external validation and extension of a model for prediction of
30-day survival. Ann Neurol. 1991;29:658 – 663.

PROFORMA

A STUDY ON **"PREDICTION OF OUTCOME IN PATIENTS WITH PRIMARY INTRA CRANIAL HEMORRHAGE USING FUNC SCORE"**

NAME:	AGE:	SEX:
Ip no:	Occupation:	
Address:	Contact no:	
BP:	GCS:	
Other comorbidities:		
Brief History:		
Brief clinical examination:		
TEST	VALUE	
ICH VOLUME		
ICH LOCATION		
PRE ICH COGNITIVE IMPAIRMEN	Г	
DURATION OF PRESENTATION		
FUNC SCORE		
INTERVENTION		

OUTCOME



GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL, CHENNAI -01 INSTITUTIONAL ETHICS COMMITTEE

TITLE OF THE WORK	: PREDICTION OF OUTCOME IN PATIENTS WITH PRIMARY INTRA CRANIAL HEMORRHAGE USING FUNC SCORE
PRINCIPAL INVESTIGATOR DESIGNATION DEPARTMENT	

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 11.05.2018 at the Council Hall, Stanley Medical College, Chennai-1 at 10am.

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 team are directed to adhere to the guidelines given below:

- 1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
- 2. You should not deviate from the area of the work for which you applied for ethical clearance.
- 3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
- 4. You should abide to the rules and regulation of the institution(s).
- 5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
- 6. You should submit the summary of the work to the ethical committee on completion of the work.

IEC, SMC, CHENNAI



Urkund Analysis Result

Analysed Document: Submitted: Submitted By: Significance: plagiarism.docx (D57251465) 10/18/2019 4:46:00 PM drmrajmohan1987@gmail.com 4 %

Sources included in the report:

https://www.researchgate.net/ publication/280391178_Intracerebral_hemorrhage_patients_presenting_with_normal_blood_pr essure https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3443867/

Instances where selected sources appear:

6

98

INFORMED CONSENT

"PREDICTION OF OUTCOME IN PATIENTS WITH PRIMARY INTRA CRANIAL HEMORRHAGE USING FUNC SCORE"

Place of study: Govt. Stanley medical college, Chennai

I have been informed about the details of the study in my own language.

I have completely understood the details of the study.

I am aware of the possible risks and benefits, while taking part in the study.

I agree to collect samples of blood/saliva/urine/tissue if study needs.

I understand that I can withdraw from the study at any point of time and even then, I can receive the medical treatment as usual.

I understand that I will not get any money for taking part in the study.

I will not object if the results of this study are getting published in any medical journal, provided my personal identity is not revealed.

I know what I am supposed to do by taking part in this study and I assure that I would extend my full cooperation for this study.

Volunteer:

Name and address

Signature/thumb impression:

Signature/thumb impression

Witness:

Name and address

INFORMED CONSENT

"PREDICTION OF OUTCOME IN PATIENTS WITH PRIMARY INTRA CRANIAL HEMORRHAGE USING FUNC SCORE"

நான் இந்த ஆராய்ச்சியில் விவரங்களை முற்றிலும் புரிந்து கொண்டேன்.

ஆய்வில் பங்கு எடுத்து போது, சாத்தியமான அபாயங்கள் மற்றும் பயன்களை பற்றி நான்அறிந்துள்ளேன். நான் எந்த வொரு வேளையிலும் ஆய்வில் இருந்து திரும்பமுடியும், அதன்பின்னர், நான் வழக்கம் போல் மருத்துவ சிகிச்சை பெற முடியும் என்று புரிந்து கொள்கிறேன்.

நான் ஆய்வில் பங்கு எடுத்து பணம் எதையும் பெறமுடியாது என்று அறிந்துள்ளேன்.

இந்த ஆய்வின் முடிவுகள் எந்த மெடிக்கல்ஜர்னலில் வெளியிடப்பட இருந்தால் நான் எதிர்க்கவில்லை, என் தனிப்பட்ட அடையாளத்தை வெளிப்படுத்தப்பட்டு இருக்ககூடாது.

நான் இந்த ஆய்வில் பங்கெடுப்பதன் மூலம் நான் என்ன செய்யபோகிறேன் என்று தெரியும். நான் இந்தஆய்வில் என் முழுஒத்துழைப்பையும் கொடுப்பேன் என்று உறுதியளிக்கிறேன்.

தன்னார்வளர்

பெயர் மற்றும் முகவரி

கையொப்பம் /விரல்ரேகை:

பெயர் மற்றும் முகவரி

சாட்சி

கையொப்பம் / விரல்ரேகை:

																		90	9						
									ICH			PRE						th D	0 t	90th			PO		
									VO			ICH						AY	h	DAY			ST		
						PRIO	BP ON	DURA TION	LU ME			COG NITIV			INTRAV		90t h	FU N	D A	FUN CTIO	TIME		STR OK	COG NITIV	BE D
						R	AD	AT	(cu			E			ENTRIC		DAY	C	Ŷ	NAL	FOR		E	E	S
	Α	S	S	T2	С	ANTI	MIS	PRESE	bic	ICH	G	IMPA	TOTAL	MIDLI	ULAR	INTER	OUT	SC	G	OUT	AMB	RE	SEI	IMPA	0
NAME	G E	E X	H T	D M	A D	PLAT ELET	SIO N	NTATI ON	cm)	LOCA TION	C S	IRME NT	FUNC SCORE	NE SHIFT	EXTENSI ON	VENTI ON	CO ME	O RE	C S	COM E	ULAT ION	BLE ED	ZU RES	IRME NT	RE S
	-		Y				100/																		
peruma I	5 4	м	E S	N O	N O	NO	180/ 100	4hrs	64	lobar	3	no	5	YES	YES	YES	DEA D	-	-	-	-	-	-	-	-
Le cile c	c		Y	VE	Y		24.07										554								
kantha n	6 5	м	E S	YE S	E S	YES	210/ 110	5hrs	72	deep	8	no	4	YES	YES	nil	DEA D	-	-	-	-	-	-	-	-
noorull	5		Y E	YE	N		140/										alin		1	inde	3				
noorull ah	5 6	м	S	S	0	NO	140/	2hrs	36	lobar	9	no	9	no	no	nil	aliv e	9	1 0	pend ent	mont hs	no	no	nil	nil
	F		Y	N	N		100/				1														
vijaya	5 5	F	E S	N O	N O	NO	190/ 100	2hrs	61	lobar	1 3	no	7	YES	no	nil	DEA D	-	-	-	-	-	-	-	-
volam	5		N	N	N		160/				1						aliv		1	inde	45				
velam ma	5 4	F	0	0	N O	NO	90	1hrs	54	lobar	1	no	9	no	no	nil	e	9	3	pend ent	45 days	no	no	nil	nil
	2		N	N	N		170/												1	inde	2				
sekar	3 5	м	N O	N O	N O	NO	170/ 100	2hrs	27	lobar	9	no	11	no	no	nil	aliv e	11	1 1	pend ent	mont hs	no	no	nil	nil
thanika	7		Y E	N	Y E		150/										DEA								
thanika chalam	6	м	۲ ۲	N O	۲ ۲	YES	100	6hrs	66	deep	4	no	3	YES	YES	nil	DEA	-	-	-	-	-	-	-	-
kanaka	4		Ν	Ν	Ν		190/				1					_	DEA								
valli	5	F	0	0	0	NO	120	2hrs	69	lobar	1	no	7	YES	no	nil	D	-	-	- inde	- 1	-	-	-	-
	4		Ν	N	N		176/				1						aliv		1	pend	mont				
pushpa	3	F	0	0	0	NO	94	2hrs	31	lobar	2	no	9	no	no	nil	е	9	4	ent	h	no	no	nil	nil
sheik moham	5		N	YE	N		188/				1						DEA								
med	2	М	0	S	0	NO	90	1hrs	66	deep	1	no	6	YES	no	nil	D	-	-	-	-	-	-	-	-
vellaiya	6		Y E	N	N		150/										DEA								
n	1	М	S	0	0	NO	102	3	65	lobar	4	no	5	YES	no	nil	D	-	-	-	-	-	-	-	-
	4		N	N	N		208/										aliv		1	inde pend	3 mont				
joseph	0	М	0	0	0	NO	108	30mts	27	deep	9	no	10	no	no	nil	е	10	1	ent	hs	no	no	nil	nil
suseela	8 1	F	Y E	N O	N O	NO	144/ 100	8	32	infrat entor	3	yes	101	no	YES	nil	DEA D	-	-	-	-	-	-	-	_
0000010	. <u> </u>	. <u> </u>		, J	5			5		001	<u> </u>	,	-		. 25				I	1	1	I	1	1	ـــــ ا

			S							ial															
			Y																	inde	2				
sumath	4		Ē	YE	N		140/										aliv		1	pend	mont				
i	7	F	S	S	0	YES	90	2	54	lobar	8	no	7	no	no	nil	e	9	0	ent	hs	YES	YES	nil	nil
eswara	6	•	N	N	N	125	156/	-	51	10.541	0	110	,	110	110		DEA		Ŭ	ent	115	125	125		
n	7	М	0	0	0	NO	94	4	66	deep	6	no	4	YES	no	nil	D	-	-	-	-	-	-	-	-
			-	-	_		-				-			_	-					inde	2				
saravan	4		Ν	Ν	Ν		210/				1						aliv		1	pend	mont				
an	6	М	0	0	0	NO	110	1	44	lobar	1	no	9	no	no	nil	е	9	3	ent	hs	no	no	nil	nil
			Y		Υ																				
	5		Е	YE	Е		198/										DEA								
gopal	9	Μ	S	S	S	YES	98	5	69	deep	5	no	3	YES	no	nil	D	-	-	-	-	-	-	-	-
																				inde	2				
	6	_	Ν	N	Ν		240/				1		_				aliv		1	pend	mont				
eswari	9	F	0	0	0	NO	120	2	55	lobar	0	no	7	no	no	YES	е	9	1	ent	hs	no	no	nil	nil
	2		Y E				200/				1								1	inde	1				
mahesh	3 9	м	E S	N O	N O	NO	200/ 106	1	47	lobar	1 4	no	9	no	20	nil	aliv e	9	1 5	pend ent	mont h	no	20	nil	nil
Inditesti	9	171	3	0	0	NO	100	1	47	IUDai	4	110	9	110	no	1111	е	9	5	inde	1	110	no	1111	1111
	4		N	N	N		180/				1						aliv		1	pend	mont				
rani	7	F	0	0	0	NO	92	1	25	deep	2	no	8	no	no	nil	e	8	5	ent	h	no	no	nil	nil
Turn		•	0	0	0	110	52	-	23	ucep		110	Ū	110	110		ç	<u> </u>	,	inde	3				
rathnav	4		Ν	Ν	N		170/										aliv			pend	mont				YE
el	1	м	0	0	0	NO	100	1.5	43	lobar	7	no	7	no	YES	nil	е	7	9	ent	hs	no	no	YES	S
			Y		Υ															inde					
peruma	5		Е	Ν	Е		144/				1						aliv		1	pend	45				
1	2	М	S	0	S	YES	80	3	48	lobar	2	no	9	no	no	nil	е	9	3	ent	days	no	no	nil	nil
deenul	5		Ν	Ν	Ν		220/				1						DEA								
akbar	5	Μ	0	0	0	NO	90	4	62	deep	1	no	6	YES	no	nil	D	-	-	-	-	-	-	-	-
moham																				inde	2				
med	5		N	YE	N		186/				1		_				aliv	_	1	pend	mont				
husain	9	М	0	S	0	NO	88	2	59	lobar	1	no	7	no	no	nil	е	7	2	ent	hs	no	no	nil	nil
				••			1001										. 11			inde	1				
notor	4 4	м	N O	N O	N O	NO	196/ 92	1	30	lobar	1 4		9		20	nil	aliv e	9	1 5	pend ent	mont h	-	20	nil	nil
peter	4	171	Ŷ	0	0	NO	92	1	50	IUDai	4	no	9	no	no	1111	е	9	5	ent	11	no	no	1111	
jayakod	8		Ē	N	N		204/										DEA								
i	0	F	S	0	0	NO	102	5	67	deep	3	yes	1	YES	YES	nil	DEA	-	_	-	-	-	-	-	
-		<u> </u>	5	5			102	5	57	чеср	5	,	-				5			inde	3				
vinayak	7		Ν	YE	N		166/										aliv		1	pend	mont				YE
am	1	м	0	S	0	NO	102	2	33	lobar	9	no	8	no	no	nil	e	8	1	ent	hs	no	no	YES	S
			Y																						
valar	5		Е	Ν	Ν		188/				1						DEA								
kodi	3	F	S	0	0	NO	98	3	60	lobar	0	no	7	YES	no	nil	D	-	-	-	-	-	-	-	-
	6		Ν	Ν	Ν		158/										DEA								
lakshmi	5	F	0	0	0	NO	110	3	76	lobar	8	no	5	YES	no	nil	D	-	-	-	-	-	-	-	-

dhooth	8		Ν	YE	Ν		170/										DEA								
amma	5	F	0	S	0	NO	110	6	32	deep	7	yes	3	no	no	nil	D	-	-	-	-	-	-	-	-
			-	-	_	-		-	_			1	-							inde	2				
	3		Ν	Ν	Ν		190/				1						aliv		1	pend	mont				
prabu	8	м	0	0	0	NO	100	1	42	deep	1	no	8	no	no	nil	e	8	2	ent	hs	YES	YES	nil	nil
10 · • • • •	-		-	-	-			_			_						-	-	_	inde					
girithar	4		Ν	N	N		156/				1						aliv		1	pend	50				
an	9	м	0	0	0	NO	88	2	27	lobar	0	no	8	no	no	nil	e	8	2	ent	days	no	no	nil	nil
dii	5	111	N	N	N	NO	182/	L	27	lobul	1	110	0	110	110		DEA	0	2	cite	uuys	110	110		
mythili	5	F	0	0	0	NO	102/	2	66	doon	1	20	6	YES	20	nil	DLA		_	_	-				
mythin	5	Г	Ŷ	0	Ŷ	NO	100	Z	00	deep	T	no	0	TES	no	1111	D	-	-	-	-	-	-	-	-
	c			YE			1761																		
acakan	6	54	E S	S	E S	NO	176/	2	62	lohor	7		-	VEC	20	nil	DEA D	-		-	-				-
asokan	6	Μ				NO	112	3	62	lobar	/	no	5	YES	no	nil		-	-	-	-	-	-	-	-
banum	5	_	N	N	N	NO	188/	2		La la const			-	VEC			DEA								1
athi	4	F	0	0	0	NO	98	3	66	lobar	8	no	5	YES	no	nil	D	-	-	-	-	-	-	-	-
kandha	8		Ν	N	N		244/	_					_				DEA								1
samy	4	Μ	0	0	0	NO	140	5	45	deep	6	yes	3	no	YES	nil	D	-	-	-	-	-	-	-	-
																				inde	2				1
	2		Ν	Ν	Ν		220/										aliv		1	pend	mont				
franklin	7	Μ	0	0	0	NO	120	2	31	lobar	9	no	9	no	no	nil	е	9	2	ent	hs	no	no	nil	nil
	4		Ν	YE	Ν		220/				1						DEA								
ismail	4	Μ	0	S	0	NO	110	1	61	lobar	0	no	7	YES	no	YES	D	-	-	-	-	-	-	-	-
	4		Ν	Ν	Ν		192/				1						DEA								
roja	9	F	0	0	0	NO	100	4	64	lobar	1	no	7	YES	no	nil	D	-	-	-	-	-	-	-	-
																				inde	1				
	5		Ν	Ν	Ν		166/				1						aliv		1	pend	mont				
revathi	1	F	0	0	0	NO	90	2	54	deep	2	no	8	no	no	nil	е	8	5	ent	h	no	no	nil	nil
			Υ																						
	5		Е	YE	Ν		182/										DEA								
nalini	8	F	S	S	0	YES	104	3	78	lobar	6	no	5	YES	YES	nil	D	-	-	-	-	-	-	-	-
																				inde	2				
farook	4		Ν	Ν	Ν		156/										aliv		1	pend	mont				
abdulla	6	М	0	0	0	NO	88	2	27	lobar	9	no	11	no	no	nil	e	11	1	ent	hs	no	no	nil	nil
																Ì				inde					
manoh	4		Ν	Ν	Ν		178/				1						aliv		1	pend	50				
ar	4	м	0	0	0	NO	86	2	42	lobar	1	no	9	no	no	nil	e	9	3	ent	days	no	no	nil	nil
			-	-	-	-						-	-	-	-	ł	-	-	_	inde	3	-	_		1
srinivas	4		Ν	YE	N		154/										aliv		1	pend	mont				
alu	9	М	0	S	0	NO	92	3	48	deep	9	no	8	no	no	nil	e	8	0	ent	hs	YES	YES	nil	nil
	5		Ŷ	~	Ŷ			5		~~~p	2		,				Ĩ			inde	1				
shama	5		Ē	N	Ē		188/				1						aliv		1	pend	mont				
begum	1	F	S	0	S	NO	94	4	33	lobar	2	no	9	no	no	nil	e	9	5	ent	h	no	no	nil	nil
JCBUIII	8		N	N	N	110	182/	-+	55	100001	~	110	5	110	110		DEA	5	5	Crit					
mallika		F	N O	N O	0	NO	82	6	33	doon	7	Nec	3	20	20	nil	DEA	-			-				
mallika	3	r				NU		υ	55	deep		yes	3	no	no			-	-	- indo		-	-	-	<u> </u>
naraya	5	N.4	Y E	YE	N O	NO	188/	n	64	door	1	20	e	VEC	20	p:I	aliv	e	1 5	inde	1 mont	-	22	nil	ا :م
na	4	Μ	E	S	U	NO	120	2	64	deep	2	no	6	YES	no	nil	е	6	5	pend	mont	no	no	nil	nil

moorth			S																	ent	h				
у																									
																				inde	3				
ramara	4		Ν	Ν	Ν		178/										aliv		1	pend	mont				
0	7	М	0	0	0	NO	82	3	31	lobar	8	no	7	no	no	YES	e	9	1	ent	hs	no	no	nil	nil
	_		Y																	inde	3				
	4		E	N	N		168/				-		_				aliv	•		pend	mont				YE
samuel	8	Μ	S	0	0	NO	92	3	43	lobar	7	no	7	no	no	YES	e	9	9	ent	hs	YES	YES	YES	S
krishna	0		Y	YE	N		200/										DEA								
moorth i	8 4	м	E S	S	0	NO	100	7	54	deep	6	yes	3	no	YES	nil	DEA	-	-	-	-	-	-	-	-
1	4	101	Y	3	0	NO	100	/	54	ueep	0	yes	3	110	TLJ	1111			-	inde	-	-	-	-	-
	5		Ē	N	N		190/				1						aliv		1	pend	40				
mythili	8	F	S	0	0	NO	100	2	52	deep	1	no	8	no	no	nil	e	8	4	ent	days	no	no	nil	nil
	8	-	N	YE	N		188/				_		-				DEA	-							
saroja	1	F	0	S	0	NO	100	4	36	lobar	6	yes	4	no	no	nil	D	-	-	-	-	-	-	-	-
										infrat															
noorjah	7		Ν	Ν	Ν		150/			entor							DEA								
an	0	F	0	0	0	NO	100	6	60	ial	7	yes	3	YES	no	nil	D	-	-	-	-	-	-	-	-
			Y																	inde	3				
manick	4		Е	Ν	Ν		168/										aliv		1	pend	mont				
am	7	Μ	S	0	0	NO	90	2	28	deep	9	no	10	no	no	nil	e	10	2	ent	hs	no	no	nil	nil
	_		Y		Y															inde	2				
	5	-	E	YE	E		144/						_				aliv	_	1	pend	mont				YE
jasmin	1	F	S	S	S	YES	90	1	55	lobar	8	no	7	no	no	nil	e	7	0	ent	hs	no	no	YES	S
							200/				1								1	inde	1				
peruma I pillai	4 1	м	N O	N O	N O	NO	208/ 100	1	31	lobar	1 4	no	9	no	no	nil	aliv e	9	1 5	pend ent	mont h	no	20	nil	nil
i pillai	1	101	0	0	0	NO	100	1	51	iobai	4	110	9	110	110	1111	e	3	5	inde	1	110	no	1111	
vanaroj	4		Ν	N	N		158/				1						aliv		1	pend	mont				
a	9	F	0	0	0	NO	100	3	36	deep	1	no	8	no	no	YES	e	10	4	ent	h	no	no	nil	nil
-	-		-	-	-			-			_						-			inde	1				
	4		Ν	Ν	Ν		170/				1						aliv		1	pend	mont				
gopal	2	М	0	0	0	NO	100	2	41	lobar	2	no	9	no	no	nil	е	9	5	ent	h	no	no	nil	nil
																				inde	3				
jayaros	5		Ν	Ν	Ν		164/										aliv			pend	mont				YE
е	2	F	0	0	0	NO	102	1.5	47	lobar	7	no	7	no	no	nil	е	7	7	ent	hs	YES	YES	YES	S
maniga			Y							infrat															
ndapra	7		Е	YE	Ν		190/			entor							DEA								
bhu	1	Μ	S	S	0	NO	90	6	60	ial	6	yes	3	YES	no	nil	D	-	-	-	-	-	-	-	-
velmur	6		N	N	N		180/			1-1			-	VEC			DEA								
ugan	7	Μ	0	0	0	NO	94	4	66	lobar	8	no	5	YES	no	nil	D	-	-	-	-	-	-	-	-
habu	6		N	N	N	NO	176/	-	~	lahar	_		4		VEC		DEA								
babu	3	Μ	0	0	0	NO	86	5	60	lobar	5	yes	4	no	YES	nil	D	-	-	- indo	-	-	-	-	-
poonko	5		N	YE	N		208/				1						aliv		1	inde pend	1 mont				
di	5 9	F		S		NO	100	3	62	deep	2	no	6	YES	no	nil	e	6	1 5	ent	h	no	no	nil	nil
u	9	L 1	0	5	0	NU	100	3	02	ucep	- 2	10	0	ILJ	10	1/11	e	0	5	CIIL		110	10	110	L

			Y		Y									1						inde					
banum	5		Ē	Ν	E		160/				1						aliv		1	pend	45				
athi	0	F	S	0	S	YES	90	3	45	lobar	0	no	9	no	no	nil	e	9	4	ent	days	no	no	nil	nil
sivaku	6	-	N	N	N		220/	-			-						DEA	-							
mar	0	м	0	0	0	NO	120	5	66	lobar	4	yes	4	YES	no	YES	D	-	-	-	-	-	-	-	-
	_		-	-		-	-	-				1				_				inde	3				
	4		Ν	Ν	Ν		190/										aliv			pend	mont				YE
kumar	9	М	0	0	0	NO	100	3	51	lobar	8	no	7	no	no	nil	e	7	9	ent	hs	YES	YES	YES	S
																				inde					
	4		Ν	Ν	Ν		208/				1						aliv		1	pend	45				
ruby	0	F	0	0	0	NO	120	2	44	deep	0	no	8	no	no	nil	е	8	4	ent	days	no	no	nil	nil
										· ·										inde	1				
suresh	3		Ν	Ν	Ν		240/				1						aliv		1	pend	mont				
kumar	9	М	0	0	0	NO	140	25mts	54	lobar	1	no	9	no	no	nil	e	9	5	ent	h	no	no	nil	nil
										infrat															
govinda	8		Ν	Ν	Ν		140/			entor							DEA								
n	0	М	0	0	0	NO	90	1	30	ial	5	yes	4	no	no	nil	D	-	-	-	-	-	-	-	-
																				inde					
manjul	7		Ν	Ν	Ν		170/				1						aliv		1	pend	45				
а	7	F	0	0	0	NO	110	3	41	deep	0	no	7	no	no	YES	е	9	3	ent	days	no	no	nil	nil
			Υ																						
kanakar	5		Е	Ν	Ν		160/										DEA								
aj	8	Μ	S	0	0	NO	96	2.5	78	lobar	7	no	5	YES	no	nil	D	-	-	-	-	-	-	-	-
noor			Υ		Y																				
moham	9		Е	YE	Е		168/										DEA								
med	1	Μ	S	S	S	YES	98	7	76	deep	3	yes	1	YES	no	nil	D	-	-	-	-	-	-	-	-
																				inde	1				
seetha	5		Ν	YE	Ν		170/				1						aliv		1	pend	mont				
lakshmi	9	F	0	S	0	NO	100	3	64	deep	3	no	6	YES	no	nil	e	9	5	ent	h	no	no	nil	nil
																				inde	1				
poova	6	_	N	N	N		154/				1						aliv		1	pend	mont				
mmal	0	F	0	0	0	NO	100	2	46	deep	1	no	8	no	no	nil	е	8	4	ent	h	no	no	nil	nil
narend	7		N	YE	N		188/		C 1		_		-	VEC	2455		DEA								
ra babu	6	Μ	0	S	0	NO	94	1	61	deep	5	no	3	YES	YES	nil	D	-	-	-	-	-	-	-	-
							204/				1								1	inde	2				
maniual	4	64	N	N O	N O	NO	204/	Δ	22	door	1		c	20	20	p:I	aliv	0	1 3	pend	mont	22	20	nil	n il
manivel	9	Μ	0	0	0	NO	104	4	33	deep	0	no	8	no	no	nil	e	8	3	ent	hs 3	no	no	nil	nil
ramana	4		NI	N	N		109/										aliv			inde					YE
ramana than	4 3	64	N O	N O	N O	NO	198/ 98	2	30	lobar	9	20	9	20	20	nil	aliv	9	9	pend	mont	n 0	n 0	VEC	YE S
than rakkam	6	Μ	N	YE	N	NU	98 188/	۷.	50	lobar	9	no	3	no	no	1111	e DEA	3	3	ent	hs	no	no	YES	3
а	0 1	F	0	S	N O	NO	88	3	68	lobar	6	no	5	YES	no	nil	DEA	-	-	-	-	-	-	_	-
a	1	1	0	5	0	NU	00	3	00	iobai	0	110	5	TLJ	10			-	-	inde	2	-	-	-	<u> </u>
	4		Ν	N	N		190/										aliv		1	pend	z mont				
raja	2	м	0	0	0	NO	190/	2	39	lobar	9	no	9	no	no	nil	e	9	0	ent	hs	YES	YES	nil	nil
anthon	7		Y	YE	Ŷ	110	180/	2	55	100001	1	110	5		10		aliv		1	inde	2	123	123		
y antition	0	м	E	S	E	NO	100/	3	33	deep	1	no	7	no	no	YES	e	9	3	pend	mont	no	no	nil	nil
у	0	111	Ľ	5	Ľ	NU	102	3	55	ueep	1	110	1	10	10	IE3	ਦ	9	്	penu	mont	110	110	1111	1 111

			S		S															ent	hs				
																				inde	3				
riyaz	4		N	N	N		180/										aliv			pend	mont				YE
ahmed	3	м	0	0	0	NO	112	2	47	deep	9	no	8	no	no	YES	e	10	9	ent	hs	no	no	YES	S
unneu			Ŷ	•	Ŭ	110	112			uccp	,	110	Ū	110	110	125	C	10	,	inde	3			125	
karpaka	5		Ē	Ν	Ν		170/										aliv		1	pend	mont				
m	3	F	S	0	0	YES	96	40mts	23	lobar	9	no	11	no	no	nil	e	11	0	ent	hs	no	no	nil	nil
parthib	7		Ν	Ν	Ν		188/										DEA								
an	7	М	0	0	0	NO	92	5	64	lobar	5	yes	4	YES	no	nil	D	-	-	-	-	-	-	-	-
angam	7		Ν	Ν	Ν		152/										DEA								
ma	7	F	0	0	0	NO	102	4	66	deep	7	no	3	YES	YES	nil	D	-	-	-	-	-	-	-	-
elango																				inde					
marimu	5		Ν	Ν	Ν		164/				1						aliv		1	pend	45				
thu	0	М	0	0	0	NO	94	1	27	deep	1	no	10	no	no	nil	е	10	4	ent	days	no	no	nil	nil
	_			••			2224													inde	45				
elavara	7		N	N	N	NO	220/	2	42	محمام	1		7			VEC	aliv	0	1	pend	45				
san	0	Μ	0 Y	0	0	NO	120	2	43	deep	2	no	7	no	no	YES	е	9	5	ent	days	no	no	nil	nil
nrathaa	2		Y E	N	N		248/				1						aliv		1	inde	1				
prathee ba	3 1	F	E S	N O	N O	NO	248/ 140	3	31	lobar	1 1	no	9	no	no	nil	aliv e	9	1 4	pend ent	mont h	no	no	nil	nil
Da	1	-	3	0	0	NO	140	3	51	IUDai	1	110	9	110	110	1111	e	9	4	inde	3	110	110	1111	
poovizh	3		N	N	N		190/										aliv			pend	mont				YE
i	9	F	0	0	0	NO	100	2	33	lobar	9	no	9	no	no	nil	e	9	9	ent	hs	no	no	YES	S
	-		Ŷ	-	-						-		-				-	-	-	inde	1				-
karthik	7		E	Ν	Ν		186/				1						aliv		1	pend	mont				
eyan	1	м	S	0	0	NO	88	1	54	deep	1	no	7	no	no	nil	е	7	4	ent	h	no	no	nil	nil
sheik																									
dawoo	6		Ν	Ν	Ν		158/										DEA								
d	2	М	0	0	0	NO	98	5	62	lobar	6	no	5	YES	no	nil	D	-	-	-	-	-	-	-	-
																				inde					
ansar	4		Ν	Ν	Ν		190/				1						aliv		1	pend	45				
fathima	5	F	0	0	0	NO	90	3	47	lobar	0	no	9	no	no	nil	е	9	3	ent	days	no	no	nil	nil
	_		Y		Y															inde	3				
In Laborat	7	-	E	N	E	VEC	178/	2	20		•		-				aliv	-	0	pend	mont			VEC	YE
lakshmi	1	F	S	0	S	YES	108	3	39	deep	9	no	7	no	no	nil	е	7	9	ent	hs	no	no	YES	S
	4		NI	N	N		200/										aliv		1	inde	3				
angol	4 0	F	N O	N O	N O	NO	208/ 108	2	54	deep	9	no	8	no	no	nil	aliv e	8	1 0	pend ent	mont hs	YES	YES	nil	nil
angel	0	г	0	0	0	NU	108	2	54	infrat	9	no	0	no	no	nii	е	0	0	ent	ns	TES	TES	nii	nii
kathirv	7		N	N	N		186/			entor							DEA								
elan	6	М	0	0	0	NO	98	6	62	ial	7	no	2	YES	no	nil	DLA	-	-	-	-	-	-	-	-
5.0.1	-		Ŷ	•				Ű					-	. 20			-								
partha	7		Ē	Ν	Ν		188/										DEA								
sarathi	8	М	S	0	0	NO	108	3	66	lobar	6	yes	5	YES	YES	nil	D	-	-	-	-	-	-	-	-
	3		N	N	Ν		220/				1	-					aliv		1	inde	45				
baskar	3	М	0	0	0	NO	100	2	41	lobar	0	no	9	no	no	nil	е	9	4	pend	days	no	no	nil	nil

																				ent					
mohan	6 6	М	N O	N O	N O	NO	190/ 100	5	66	lobar	6	yes	4	YES	no	nil	DEA D	-	-	-	-	-	-	-	-
kalidas	7 2	м	Y E S	N O	N O	NO	158/ 98	6	66	deep	8	no	3	YES	no	nil	DEA D	-	-	-	-	-	-	-	_
jalal akbar	7 1	м	N O	N O	N O	NO	200/ 110	3	46	deep	1 0	no	7	no	no	YES	aliv e	7	1	inde pend ent	3 mont hs	no	no	nil	nil
saravan a peruma	8		N	N	N O		140/	7									DEA								
settu	8 6 3	M	O Y E S	O YE S	Y E S	NO YES	100 144/ 100	5	66 78	deep lobar	5 8	yes no	5	YES	YES no	nil	D DEA D	-	-	-	-	-	-	-	-
fathima banu	4 2	F	N O	N O	N O	NO	220/ 100	4	44	deep	1 1	no	7	no	no	nil	aliv e	7	1 3	inde pend ent	2 mont hs	no	no	nil	nil
harihar an	7 9	М	N O	N O	N O	NO	188/ 96	6	62	infrat entor ial	3	no	2	YES	no	nil	DEA D	-	-	- inde	- 2	-	-	-	-
sukany a	3 8	F	N O	YE S	N O	NO	186/ 92	4	45	deep	1 1	no	8	no	no	nil	aliv e	8	1 2	pend ent	mont hs	YES	YES	nil	nil
sridhar	7 0	М	N O	N O	N O	NO	184/ 94	2	40	deep	1 2	no	7	no	no	nil	aliv e	7	1 4	inde pend ent	45 days	no	no	nil	nil
anusha	5 4	F	Y E S	N O	N O	NO	188/ 98	6	66	lobar	6	yes	4	YES	YES	YES	DEA D	-	-	-	-	-	-	-	-
rupa devi	8 2	F	Y E S	N O	N O	NO	190/ 90	6	54	deep	6	yes	3	no	no	nil	DEA D	-	-	-	-	-	-	-	_
vijaya	3 7	М	N O	N O	N O	NO	200/ 108	2	36	lobar	9	no	9	no	no	nil	aliv e	9	9	inde pend ent	3 mont hs	no	no	YES	YE S
vimala	4 6	F	Y E S Y	N O	Y E S	YES	186/ 90	4	64	deep	1 0	no	6	YES	no	nil	DEA D	-	-	-		-	-	-	-
fathima banu	4 8	F	F E S Y	N O	N O	NO	176/ 98	2	31	lobar	9	no	9	no	no	nil	aliv e	9	1 0	inde pend ent	3 mont hs 2	no	no	nil	nil
moham med ismail	7 1	М	Y E S	YE S	N O	YES	168/ 92	3	39	deep	1 1	no	7	no	no	YES	aliv e	9	1 3	inde pend ent	z mont hs	no	no	nil	nil

john																				inde					<u> </u>
pravee	7		N	N	N		190/				1						aliv		1	pend	45				
n	2	М	0	0	0	NO	104	3	49	deep	2	no	7	no	no	nil	e	9	4	ent	days	no	no	nil	nil
11	2	141	Ŷ	0	0	NO	104	3	49	ueep	2	110	1	110	110	1111	e	9	4	ent	uays	110	110	1111	1111
	8		Ē	N	N		178/										DEA								
rukiya	0	F	S	0	0	NO	98	5	36	lobar	6	VOC	4	no	no	nil	DLA	-	-	-	-	-	-	-	_
Тикіуа	0	F		0	0	NO	90	5	30	IUUai	0	yes	4	110	110	1111	D	-	-		-	-	-	-	-
	F		Y E	VE	NI		172/				1						aliv		1	inde	45				
	5	-		YE	N	NO	172/	2	24		1		•				aliv	•	1	pend	45				
mary	2	F	S	S	0	NO	92	3	34	deep	3	no	8	no	no	nil	е	8	4	ent	days	no	no	nil	nil
							2001													inde					
	3	_	N	N	N		200/				1						aliv		1	pend	45				
priya	5	F	0	0	0	NO	108	2	36	lobar	2	no	9	no	no	nil	е	9	4	ent	days	no	no	nil	nil
			Y		Y																				
muthuk	5		Е	Ν	Е		178/										DEA								1
umaran	5	Μ	S	0	S	YES	102	3	88	lobar	4	no	5	YES	YES	nil	D	-	-	-	-	-	-	-	-
			Y																	inde	3				1
shanm	6		Е	YE	Ν		166/										aliv			pend	mont				YE
ugam	6	Μ	S	S	0	NO	90	3	64	deep	9	no	6	no	no	nil	е	6	9	ent	hs	no	no	YES	S
			Υ							infrat															
karmeg	7		Е	Ν	Ν		172/			entor							DEA								
am	3	F	S	0	0	NO	90	7	66	ial	3	yes	1	YES	no	nil	D	-	-	-	-	-	-	-	-
			Y																						
thanga	8		Е	YE	Ν		160/										DEA								
m	1	F	S	S	0	NO	100	6	54	deep	3	yes	3	no	no	nil	D	-	-	-	-	-	-	-	-
												- /								inde	2				
kalavat	4		Ν	Ν	Ν		150/										aliv		1	pend	mont				
hi	8	F	0	0	0	NO	100	4	56	lobar	9	no	9	no	no	nil	e	9	1	ent	hs	YES	YES	nil	nil
	•		•		•		100	•	50	10.001	5						2	5	-	inde	2	. 20	. 20		
fernand	4		Ν	N	N		178/				1						aliv		1	pend	mont				
ez	1	М	0	0	0	NO	98	2	45	lobar	0	no	9	no	no	nil	e	9	2	ent	hs	no	no	nil	nil
62	1	141	0	0	0	NO	50	2	45	IUDai	0	110	9	110	110	1111	e	9	2		115	110	110	1111	1111
	3		N	Ν	N		220/				1						aliv		1	inde	45				
chokor	3 7	5.4			0	NO		1	20	lahar		-	9			nil	aliv	0	3	pend		-		mil	mil
shekar	/	Μ	0	0	0	NO	120	1	30	lobar	1	no	9	no	no	nil	е	9	3	ent	days	no	no	nil	nil
	-		Y				100/																		
	5		E	N	N		180/	_					6				DEA								1
baskar	2	Μ	S	0	0	NO	100	5	66	deep	9	no	6	YES	no	nil	D	-	-	-	-	-	-	-	-
viswan	7		N	N	N		180/	_			-		c.				DEA								
athan	6	Μ	0	0	0	NO	100	7	61	deep	6	no	3	YES	YES	YES	D	-	-	-	-	-	-	-	-
			Y																						
simmad	6		Е	YE	Ν		176/										DEA								
ri	1	Μ	S	S	0	YES	98	7	66	lobar	5	no	5	YES	YES	nil	D	-	-	-	-	-	-	-	-
			Y																	inde	3				
rangan	5		Е	YE	Ν		172/										aliv		1	pend	mont				1
athan	2	М	S	S	0	NO	100	45mts	21	deep	9	no	10	no	no	nil	е	10	2	ent	hs	no	no	nil	nil
			Y																	inde					Γ
karthik	5		Е	Ν	Ν		190/				1						aliv		1	pend	45				
а	4	F	S	0	0	NO	100	1	27	lobar	1	no	11	no	no	nil	е	11	4	ent	days	no	no	nil	nil
				-		-		· · · · · · · · · · · · · · · · · · ·				-	L		· · · ·	l		L							

																				inde	2				
	4		Ν	Ν	Ν		200/				1						aliv		1	pend	mont				
menaka	2	F	0	0	0	NO	120	3	56	deep	0	no	8	no	no	nil	е	8	2	ent	hs	no	no	nil	nil
																				inde	3				
	7		Ν	Ν	Ν		190/										aliv			pend	mont				YE
sasi	0	F	0	0	0	NO	100	2	42	deep	9	no	7	no	no	nil	е	9	9	ent	hs	no	no	YES	S
			Y		Y															inde	2				
mathav	7		Е	YE	Е		178/				1						aliv		1	pend	mont				
an	7	М	S	S	S	YES	140	5	51	deep	0	no	7	no	no	YES	е	9	1	ent	hs	YES	YES	nil	nil
																				inde					
vijayala	7		Ν	Ν	Ν		180/				1						aliv		1	pend	35				
kshmi	1	F	0	0	0	NO	90	4	36	deep	1	no	7	no	no	nil	е	7	5	ent	days	no	no	nil	nil
																				inde	2				
elavara	4		Ν	Ν	Ν		240/										aliv		1	pend	mont				
si	1	F	0	0	0	NO	140	3	37	lobar	9	no	9	no	no	YES	е	9	0	ent	hs	no	no	nil	nil
																				inde					
kodees	4		Ν	Ν	Ν		190/				1						aliv		1	pend	45				
waran	7	М	0	0	0	NO	102	2	34	deep	1	no	8	no	no	nil	е	8	4	ent	days	no	no	nil	nil
dhatch																									
ana			Y																	inde	3				
moorth	7	М	Е	YE	Ν		180/										aliv			pend	mont				YE
i	0		S	S	0	NO	100	1	36	deep	9	no	7	no	no	nil	е	7	9	ent	hs	no	no	YES	S