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ANALYTICAL STUDY OF SURVIVAL OF PATIENTS OF TRAUMATIC ACUTE SUBDURAL HAEMATOMA (RETROSPECTIVE)

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CERTIFICATE

This is to certify that this dissertation titled "**ANALYTICAL STUDY OF SURVIVAL OF PATIENTS OF TRAUMATIC ACUTE SUBDURAL HAEMATOMA**" is an original bonafide work conducted by **Dr. K.BAGATHSINGH** at Madurai Medical College & GRH, Madurai under my guidance and supervision.

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CONTENTS

INTRODUCTION

Head injury defines an injury to head and brain. Head injury is a neuro surgical problem and operation if required should be performed by neuro surgeon. Head injury continues to be a night mare, not only for the public but also for the neuro surgeon, because of high mortality / morbidity study published in journal of neuro trauma June 2001, which was a leading article from neuro trauma conference, concluded that no change in last 30 years in mortality and morbidity of severe head injury. In India problem has become more acute over last two decades, basically due to increased vehicular traffic and poor maintenance of the road. The number of head injuries are expected to increase further, due to urbanisation. Industrialization and increase in vehicular population. Head injury by and large classified as (a) mild (b) moderate (c) severe based on duration of loss of conscious and glasgow coma scale ‘no head injuries minor enough to be neglected nor severe enough to be given up’.

Epidemiological incidence of head injury per 100,000 population per year ranges from 56-430 in various countries in the world. From India, incidences are basically from metropolis and based on medico legal reports which may not be absolutely correct. In India incidence of head injury is steadily increasing with urbanisation and increasing number of vehicular

population. Among the road accidents 70% have head injury among road accident deaths 70% are due to head injury.

Total number of vehicles in India are only 1% of World's total vehicles, however, total number of accidents in India as reported in 1991 were 6% of total accidents, thus making it highest incidence of accident rate in the world. The number of accidents are directly proportional to number of vehicles on the road. Thus today, suppose things have gone worse than what it was15 years back.

Roughly 5-10% of all injuries are fatal. Fortunately, 75-80% of all head injuries and minor (or) moderate.

Large number of factors determine the outcome in a head injury patient. Age, sex, severity of head injury, intracranial pathology, intracranial pressure and associated injuries are significant prognostic factors. Recently genetic basics of head injury outcome is reported. Presence of apolipoprotein E4 alleles is recognised as poor prognostic factor. The American Association of Neurological Surgeons (AANS) and Neurotrauma subcommittee of Neurological society of India (NSI) decided to develop uniform guidelines for management of head injuries.

Acute subdural haematoma is a collection of fresh blood under dura, which compress the brain. Acute SDH occurs in approximately 10-30% of

patients with severe head injury. In 80% of acute SDH it is the extend of the underlying brain injury which determines the outcome. The low incidence of 1.4% reported in 1970 by Ramamurthi as the symptomatic acute SDH. But with increasing speed of modern transport and enormous increase in the number of motor vehicles and two wheelers, severity of accidents has increased resulting in the greater incidence of subdural haematomas.

Acute SDH involves all forms of head injury involving mild, moderate and severe forms. Thus it is clear that there is need for valid, better and reliable guidelines to allow physicians for care of patients with acute SDH.

HISTORICAL REVIEW

Incas of peru probably practised trephination as FAR as back as 3000 B.C. The Edwin Smith Papyrus recording surgical practices in ancient Egypt

(1700 B.C). Recognised that scalp lacerations and fractures of skull with meningeal irritation could be treated. At the time of Hippocrates (460-370 B.C) different types of fracture skull were recognised and trephination was advocated. Extradural haemorrhage without skull fracture and intradural haemorrhage were known in ancient Greek and Rome. Hippocrates felt that 'No head injury was so slight that it could be neglected or so severe that life should be despired of'.

One of the earliest cases of subdural haemorrhage on record was that of Henry Second of France who had sustained frontal wound and died. Hoessly published a translation of Wepfers case notes written in 1657 on subdural haematoma. These were probably the first fully described cases of this condition. However, it was only sixty years ago that Trotter finally established that lesion described as pachymeningitis- haemorrhagicainterna, by virchow and others was in reality a subdural haematoma due to trauma. Ambrose Pare in seventeenth century pointed out that concussion was recognised as distinct clinical entity. Sharp in 1754, distinguished between concussion and intracranial extravasation. Hutchinson, in 1867 gave attention to significance of the unilateral pupil in head injury. John Abernthy a pupil of hunter described extra and subdural haematomas. Burrows and Jalabson also contributed to our understanding of these lesions. Cushing in

1908 advocated subtemporal decompressive (craniotomy) operations for intracranial complications associated with fracture skull. Evidence of the practice of cranioplasty is available in five thousand years old peru vaian skulls. Zander was been the first to perform in 1940, methymethacrylate cranioplasty in human. Prevention of head injuries had been recognised long time. Back since knights of the middle ages wore steel helmets as part of armour. The Tin had evolved in first world war, crash helmet is second world war. To Cairns goes the credit for popularishing the crash helmet for civilan use.

MICRO SURGICAL ANATOMY AND SIGNIFICANCE

The innermost layer of the dura is composed of flattened fibroblasts that are in close contact with arachnoid suggesting that the subdural space is a potential space rather than actual space Penfield suggested that the dura

and arachnoid are separated by a potential space containing small amount of fluid. This compatible plane between the dura mater and arachnoid that is seen in surgery. This concept was challenged by Schachenmayr and Freede and Yama Shima and Freede who investigated the fine structure of dura arachnoid interface in human necropsy material fixed in situ.

Other authors also have described in detail a continuum of cells between the dura and the underlying arachnoid with no naturally occurring subdural space. In accordance with this model, the cleavage of the weakest layer of this continuum and the cellular, reaction to the cleavage result in the formation of a ‘subdural’ space.

The outer layers of continuum between the dura and arachnoid consist of the periosteal dura and meningeal dura. These layers of dura are composed primarily of fibroblasts and extra cellular collagen. The extracellular collagen is oriented in a variety of directions and is internining; resulting in the strength of these layers and their resistance to distruption under stress.

The layers of cells immediately superficial to the arachnoid is also resistant to disruption. This layer termed the arachnoid barrier cell layer, consists of cells tightly bound by numerous tight junctions, gap junctions, and desmosomes. A strong basement membrane forms the inner surface of

this layer and the outer surface of the subarachnoid space. These structures features impart strength to this layer of cells, preventing disruption by minor trauma.

Between dural and the arachnoid barrier layer is a layer of cells referred to by various authors as ‘dural border cell layer’ light cell layer, flake like cell layer. This layer is devoid of the collagen that strengthens the dural layers and has few intracellular junctions compared with arachnoid barrier cell layer mechanism. Separation of the arachnoid from dura causes cleavage with in this layer of cells. Experimental injection of blood between dura and arachnoid causes a cleavage plane that creates space bounded on either side by cells of this layer. Because this layer of cells occupies space immediately under dura. And there is no other space observed ultrastructurally, it follows that a cleavage plain with in this fragile layer creates a subdural space.

Normally pressure in the subrachnoid space keeping the arachnoid against dura, thus oblitrating the space. When the subarachnoid pressure drops due to any cause or in a rapid lowering of intracranial pressure subdural space opens up. The superficial cerebral veins across subdural space before entering the sinuses rupture this fill the subdural space with blood.

ETIOPATHOGENESIS

Acute subdural haematomas are caused by rupture of parasagittal or sylvian bridging veins draining into dural venous sinuses. The parasagittal veins are susceptible. The damage during short duration causes angular acceleration of head as they are superficially located. Rarely acute SDH can be due to rupture of cortical veins. Acute SDH results entirely from inertial forces when the head strikes a board, hard surface (as in fall). The impact energy causes brain to accelerate with in the skull. The strain that occurs during these conditions is confined of the surface, if the acceleration is

present for a brief period of time. The types of injury that can be produced in these circumstances are those of brain surface and those of vascular tissue (bridging veins). If duration of acceleration is prolonged the strains penetrate deeper into brain and cause diffuse axonal injury. Hence acute SDH and DAI often coexist. Another source of bleeding that can result in subdural haematoma is lacertation or rupture of small cortical arteries and veins associated with cerebral cortical contusion or injury. Subdural haematoma's are usually located over cerebral convexities. The most common site of cerebral contusion associated with subdural haematomas in temporal pole, followed by frontal pole and cerebral convexity. Subdural haematoma extend to nearest dural reflection. It may extend along the tentorium. Acute SDH may occur medially between falx and medial surface of cerebral hemisphere due to rupture of veins bridging the medial aspect of hemisphere and superior sagittal sinus. This is commonly called parafalcine sub dural haematoma. Rupture of the surface veins around the cerebellum leads to subdural haematoma in the posterior fossa. In addition to injury at the site of impact. A contrecoup injury may cause SDH in opposite side. Depressed fracture and penetrating wound may cause rupture of surface vessels and SDH. Acute SDH caused by a bleeding traumatic aneurysm of middle cerebral artery has been reported. Bleeding of dural AVM may result in

acute SDH, as well as a whip flush injury of the cervical spine. And also falls from a height and landing on feet or buttocks cause Acute SDH. Acute SDH also reported in battered babies and also in boxing injuries.

PATHOLOGY

In an acute SDH there is no time for the development of a limiting membrane around the haematoma, if there is no spontaneous arrest of the bleeding haematoma continues to enlarge. In many instances ICP raises, bleeding veins get occluded and progression of the haematoma gets arrested. This does not happen if the dural sinuses are torn due to raised intrathoracic pressure. And also due to valveless venous system between right atrium and sinuses. In pure venous bleed the haematoma is likely to consist of mixed liquid and solid clots or liquids in some areas and solid in others. Solid subdural haematoma have been found over the temporal regions in patients with severe head injury with prolonged loss of consciousness. If there is an associated arachnoid rupture CSF enters the subdural space and contents are

mixed with it. Such haematomas spread extensively in the subdural and subrachnoid space above and below the tentorium.

CLINICAL FEATURES

Normally 70-85% of all head injuries are minor or moderate and 15-20% are severe. Acute SDH occurs in approximately 5 to 25 percent of patients with severe head injury, the clinical findings are related to the size and rapidity of growth of the SDH and severity of diffuse injury to the brain.

Alteration in conscious level

When an acute subdural haematoma arises after an uncomplicated head injury, classic picture with a lucid interval may be seen.

Severe

Patients who are rendered immediately unconscious. With decerebrate posturing at the time of injury may be assumed to have sustained diffuse injury to the cerebral parenchyma. Recovery often does not take place

regardless of how rapidly the haematoma is removed or intracranial pressure (ICP) controlled.

Less severe

In patients with less severe injuries the sequence of changes in level of consciousness is determined by the magnitude of the impact injury and rapidity of haematoma accumulation.

Minor

In minor injuries patients may lose consciousness only briefly or not at all, at the time of impact. This have lucid internal. As the haematoma expands in the early post trauma period, however consciousness is gradually lost.

Papilloedema

It is too early for papilloedema to develop in a patient with an acute subdural haematoma, though an occasional case is seen where severe papilloedema with or without retinal haemorrhage may be seen with in a day or two of the injury.

LATERALISING FINDINGS

Localizing signs

Usually the pupillary dilatation will be ipsilateral and motor deficit will be contralateral to the site of subdural haematoma.

False localising sings

a) Contra lateral pupillary dilatation

May be seen as a result of direct trauma to the globe, oculomotor or optic nerve injury and direct mid brain trauma.

b) Ipsilateral motor deficit

This false localising sign is not uncommon and results from cerebral parenchymal injury on the side opposite to the SDH or compression of the contralateral cerebral peduncle against the edge of the tentorium (Kernohan's notch).

Associated injuries

Injuries else where in the body affect the prognosis in acute subdural haematoma and hence careful assessment of associated injuries is called for. Injuries like thoracic injury and long bone injury are looked for.

Skull roentgenogram

The roll of skull x-ray in evaluating patient of an intracranial mass lesion is contravertial. The skull x ray provides little information that will help the neurosurgeon to make therapeutic decisions. However risk of an

intracranial haematoma is increased 50 times when skull fracture is present. Therefore, CT scanning is essential in any patient with an abnormal skull film.

CT Scan

CT scan should be used as first diagnostic procedure for patients suspected of having an acute SDH. CT scanning is rapid, visualizes the entire intracranial compartment, and reliably distinguish the density and thus the nature of intra and extra axial mass lesions. Acute SDH appear as hyperdense cresentric areas over the cerebral hemispheres. They may be localised to one or more lobes or may be holohemispheric, extension over the entire convexity. Acute SDHs are sometimes isodense to brain when patients have low haemoglobin level or when CSF from torn arachnoid dilutes the extravasated blood. The volume of acute SDH may be underestimated by CT scan.

Carotid Angiography

Prior to the widespread use of CT scanner, carotid angiography was used a an alternative means of diagnosis. Now it is of historical interest only.

MRI

MRI is more sensitive than CT at detecting intraparenchymal lesions caused by head trauma, it has been of limited value in the radiographic diagnosis of acute SDH. More over, its great sensitivity is not of immediate value for the diagnosis of surgical lesions that are readily demonstrated by CT. MRI is useful in assessing the intra parenchymal injury associated with an acute SDH and may be preformed in stabilized patient.

Burr holes as diagnostic Aids

When symptoms are developing rapidly and there is no time for investigations, it is best to make exploratory burr holes in frontal, parietal and temporal region on both sides of skull. Acute SDH can be diagnosed by presence of fresh blood or blood clot beneath dura after incising dura in cruciate manner. The advantage of this procedure is that it combines treatment also.

TREATMENT

The initial management of acute subdural haematoma includes maintenance of blood pressure and measures to lower ICP. The aim of decreasing ICP and maintaining BP is to maintain cerebral perfusion pressure (CPP).

The definitive treatment of an acute SDH consists of operative removal using a large craniotomy centered over maximum thickness of the haematoma.

SURGICAL TREATMENT

Indications

1. All acute SDH that are more than 5mm thickness that contribute significantly to the mass effect and shift should be considered for evacuation.
2. Some patients who have exhausted their reserves of intracranial compliance, may be benefited from the evacuation of even small haematomas.

Surgical technique

The mechanism of injury responsible for producing acute SDH also leads to high incidence of temporal and frontal contusions and tearing of midline bridging veins. To achieve all surgical objectives, the operative exposure must provide access to frontal and temporal lobes.

After emergency preoperative preparation and positioning.

Scalp incision

A large fronto temparo parictal skin flap is made using a question mark incision. Incision over temporal region should be made first. Because in patients who are deteriorating rapidly quick decompression can be achieved by making Burr hole craniotomy followed by cruciate dural incision.

Bone flap

Multiple burr holes are placed in the parietal, frontal, and temporal region. The burr holes are enlarged and undermined to allow a Penfield no. 3 dissector to slide between the dura and inner aspect of bone flap gently separating the dura from the skull. Special care is needed at this stage near sagittal sinus. Joining the burr holes completes the craniotomy opening. The bone flap is fashioned so that its medial margin is at least 1.5 to 2cms from the midline if greater medial exposure or further exposure of middle fossa is obtained using Lekshell rongeurs.

Dural opening and evacuation of haematoma

Dura is opened in C shaped manner. The dura is opened by gently curving the incision anteriorly. Care should be taken not to injure cortical vessels particularly in sylvian fissure.

After dura is opened, the clot is removed gently from cortical surface with the help of suction catheter and irrigation. Frontal lobe or temporal lobe contusions, if present, for evacuation, haemostasis should be meticulously done with bipolar.

Closure

Dura should be closed in all cases with or without dural graft. Bone flap is replaced and fixed, multiple tack up sutures should be used to prevent

post operative extradural haematoma. If possible intra cranial pressure (ICP) monitoring should be done in all cases post operatively.

Surgery in not indicated in cases

Such as

1. Patients with good (GCS) no neurological deficit, with small subdural haematoma may not need surgery.
2. Patients with absent brain stem reflexes after resuscitation will almost certainly have poor results and are rarely operative candidates.

COMPLICATIONS AND POST OPERATIVE COURSE

Intraoperative

1. Intraoperative Brain swelling
2. Intra operative haemorrhage

Intraoperative brain swelling

Causes are

1. Cerebral vascular engorgement
2. Epidural haematoma on opposite side
3. Intracerebral contusion
4. Residual haematoma

All the causes looked carefully during operation. Practical problem of massive brain swelling in closure of dura and whether to replace bone flap, most authors strongly advocates closure of dura with grafts and replacement of bone flap.

2. Intraoperative haemorrhage

Venous haemorrhage is an important cause of acute subdural haematoma.

Important veins from which bleeding occur

1. Bridging veins along the sagittal and transverse sinus
2. Veins at anterior aspect of sylvian fissure
3. Inferior cerebral veins and dural sinuses

Post operative course

After evacuation patient does not show improvement in clinical status due various factors that are

1. Primary associated neuronal damage
2. Post traumatic oedema
3. Recurrence of haematoma
4. Associated haematoma
5. Respiratory problems
6. Metabolic problems
7. Infections
8. Associated injuries

PROGNOSIS

The outcome in acute SDH has been generally unsatisfactory. Most series in the literate report mortality of over 50 percent none records a morality of less than 35 percent of the patients who do survive most do not return to normal functioning, and a significant number have disabilities that render them dependent.

The important predictors of outcome include

1. Age
2. Admission GCS
3. Pupillary responsiveness
4. ICP
5. Presence of hypotension and hypoxia
6. Associated brain injury

Poor prognostic factors

1. Patients older than 40 years
2. Glasgow coma Scale <8
3. Pupillary abnormalities
4. Post operative increased ICP
5. Associated brain injury like SAH and parenchymal injury

PART – II

AIM OF THE STUDY

1. To study the various clinical presentations and their significance in the survival of Acute SDH.
2. To study the various radiological findings and their significance in the survival of acute SDH.

3. To study the various modes of treatments and their significance in the survival of acute SDH.

MATERIALS AND METHODS

This a retrospective study conducted over a period of ten months from January 2007 – October 2007 in the head injury ward, Government Rajaji Hospital, Madurai Medical College. 84 consecutive cases of acute SDH case including mild, moderate and severe head injuries were studied in this period.

Inclusion criteria include

1. All patients with acute SDH and underwent CT head with GCS between 3 to 15.
2. Patients who were discharged from head injury ward or transferred to trauma ward.

Exclusion criteria include

1. Patients who went against medical advice or absconded from head injury ward.

All these patients were subjected to thorough history and clinical examinations age and vomiting were obtained by history from the patient or from reliable attendants. Retrograde amnesia for >30 minutes and dangerous mechanism of injury were also obtained by history. Alcohol intoxication was evident through history or looking for breath smell. Neurological deficits and seizures were obtained by history. Evidence of trauma above and below the clavicles was searched by thorough examination. Contusions, lacerations, incised wounds above and below the clavicle were all included. All the patients were investigated with CT head, even for severely injured patients after resuscitation. Presence of intracranial acute subdural haematoma were all taken as cases for the study. All the patients were submitted for treatment after confirming the diagnosis, the modalities of treatment depends upon the factors elucidated like, GCS on admission,

thickness of acute SDH, mass effect, associated parenchymal contusion and skull fracture. The various modalities of treatment used in this study includes conservative and surgical evacuation of hematoma. In our study conservative treatment was indicated for patients with very small thickness of acute SDH, mild mass effect, poor GCS with absent brain stem reflex after resuscitation. These patients were treated with maintenance of airway, fluids, antibiotics, IV mannitol initially for 48-72 hours followed by oral glycerol and frusemide according to the need of the patient. At patients were treated with prophylactic diphenyl hydantion. In our study surgical intervention as the first modality for those with thickness of acute SDH greater than 5mm, mass effect and other parameter like GCS was also considered. Large Falconer's craniotomy followed by subdrual haematoma evacuation and lobectomy done depending on the presence of contusion and conditions of brain. In case brain continue to be tense after evacuation of SDH and lobectomy, a dura plasty was carried out and bone flap not replaced. GCS at the time of admission and just prior to surgery was recorded to take note of any preop. Deterioration if any many of the patients are electively ventilated IV Mannitol / glycol were used initially for 48-72 hours followed oral glycerol and frusemide according to requirements. All patients also received prophylactic diphenyl hydantion. Antibiotics were

given according to our protocol and if patient continues to be hospitalized requiring antibiotics subsequently were treated according to culture and sensitivity reports. Repeat CT scans were carried out in patients who did not show expected recovery or who had signs of deterioration. All the complications were treated accordingly. Patient needing prolonged intubation underwent tracheostomy.

Patients outcome recorded at discharge, at (1 month) following injury (according to glasgow outcome scale).

A. Good recovery (Capacity to reintegrate)

Resumption of normal life even if there may be minor neurological and psychological defects. This does not imply return to previous employment.

B. Moderate disability (independent but disabled)

Independent in so far as daily life is concerned. The disabilities include varying degree of dysphasia, hemiparesis, ataxia, intellectual and memory defect and personality change.

C. Severe disability (Conscious but dependent)

Depend for daily support due to mental and physical disability. May be institutionalised but this is not a criteria.

D. Persistent vegetative state (Wakefulness, without responsiveness)

Unresponsives and speechless after 2-3 weeks may open eyes and have sleep / wake cycles.

E. Death

The data obtained were analysed i.e., clinical features, radiological parameters and management protocol then correlated with outcome. ‘t’ Distribution, chi square, paired t test, used wherever applicable.

RESULTS AND ANALYSIS

This retrospective study consisted of a total 84 consecutive patients of acute SDH with GCS 3 to 15 admitted and treated at head injury ward, GRH, Madurai between January 2007 to October 2007.

The age of the patient ranged from 12 years to 86 years with maximum incidence in the 3^{rd} and 4^{th} decade. A total of 38 patients out of 84 (45.23%) belonged to this age group (Table - I) while 46% of patients with age less than 40 years survived, only 28% of them above age of 40 years survived. And difference was not statistically significant (p=0.22). There were 68 males out of which 25 survived and out of the 16 female 6 survived.

The majority of patients suffered head injury due to RTA i.e. 85% followed by accidental fall from height in 7.5% and assault in 7.5% patients

(Table - II). 83% of patients who had acute SDH due to accidental fall survived which was statistically significant (p=0.01).

Fifty five percent of patients had a initial GCS of <8 followed by 45% had GCS >8 at presentation (Table - III). Only ten percent of patients with GCS <8 survived whereas 73% of patients with GCS >8 survived which was statistically significant (p=0.003).

Sixteen of the 84 patients (19%) had major associated injury. Four patients had chest injury in the form of fracture ribs. Five patients had faciomaxillary injury. Seven patients had long bone fracture (Table - IV). Thirteen percent patients with associated injury survived where as 43% of 68 patients who did not have associated major injury survived which was statistically significant (p=0.03).

Thirty nine patients had left sided subdural haematoma and thirty eight patients had right sided subdural haematoma, seven patients had bilateral haematoma. Nine out of thirty nine patients (23%) with left sided SDH survived where as nineteen out of thirty eight patients (50%) with right sided SDH survived (Table -V). Though this may appear significant but out of the 18 patients who were with GCS at admission 3/15, 12 patient had left sided SDH and all expired. So the side of SDH did not influence the outcome significantly but only GCS at the time of admission.

Almost all the patients had midline shift with 31 patients out of 84 patients survived (36%) where as 64% of patients expired. Although some patients had thin midline shift they had low survival. We cannot make at any significance (Table -VI).

The status of basal cisterns on CT has brought on some outcome. Eighty (80%) percent had cisterns effaced and it had a poor outcome with survival rate of 22% where as 16 patients had no effacement of basal cisterns in them survival rate was 100% (Table - VII) which was statistically not significant (p=0.19).

We also found a correlation between outcome and the presence of subarachnoid haemorrhage on initial CT scan (Table - VIII). Total of 42 cases (50%) had SAH in their initial CT scan, out of which 4 (10%) survived where as in the 42 cases where there was no SAH 27 (64%) patients survived which was statistically significant (p=0.00002).

Associated contusion was present in 27 patients (32%) seen on initial CT scan (Table - IX). Twenty seven patients with associated contusion of which 9 (33%) patients survived whereas twenty two patients (39%) out of 57 were survived who were no contusion on initial CT scan which was statistically not significant (p=0.80).

Eleven patients (13%) underwent surgery. Surgical decision and type of surgery (Lobectomy) depends on the presence of contusion and condition of brain. All patients underwent subdural haematoma evacuation out of which 27% patients survived. The rest of patients are not operated managed conservatively because of their thin SDH, very morbid patient with GCS 3 or haemodynamically unstable and all other expired.

Commonest complication post operatively was chest infection. Other complications were septicemia, meningitis, renal failure, persistent hyperthermia and CSF rhinorrhca.

Out of total 84 patients 31 (37%) survived at discharge.

DISCUSSION

A strong correlation exists between age and outcome. In general increased age is associated with poor outcome, in adults although for children the opposite may be true[15,22,33] as outcome effect can be contributed to other associated variables, age appears to be an independent predictive value. In our study most of the patients were aged to 3rd and 4th decade while 46% of patients with age less than 40 years survived. Only 28% of patients above this survived. In a study reported by David et al[4] there was a slight better survival (42% mortality) than the patient over 40 years of age (49% mortality) children under 10 years of age have strikingly lower mortality around 33%, higher mortality reported in patients over 60 years of age. Howard et al[22] in their study of acute subdural haematoma reported a good correlation between age and the outcome. They concluded that mortality rate for old patients was as high as 66% compared to young patients, whom mortality rate was 18%. Acute SDH in young patients may be a epiphenomenon where as in elderly patients the mass itself is probably the most important pathological process. Our explanation for poor outcome in elderly may be aging brain, may be impaired regenerative capactiy[18]. Seeling et al[24] did not find any difference in age between survivors and nor

survivors. In a series published by Massaro et al[20] patients under the age of 35 yrs had a mortality rate of 45% as compared to mortality of 60% for those above 65 years. Patients aged between 35 and 65 years had a mortality rate of 62% though the difference was statistically significant there was association between age and outcome.

In a study by Nilberger et al[14] the most common mechanism of injury was automobile accidents (53%) followed by falls (37%). The worst outcome was recorded in patients involved in motor cycle accidents with a 71% mortality and to survivors with functional recovery. In our study majority of our patients i.e. 83% suffered head injury due to RTA out of them 31% survived. Massaro et al[20] in their study found that the most common mechanism of injury was RTA and fall was related with worst outcome. There was no statistically significant differences in outcome related to sex in our study and other studies also recorded similar results[4,9,20,28,34].

Outcome has been shown to be related to GCS scores in several studies and expectedly outcome worsens as GCS scores low. Gennarelli (1982)[7] in his scores of comatosed patients with acute SDH reported a mortality rate of 74% in patients with GCS 3-5 and 36% in patients with GCS 6-8. In a series reported by Wilbenger et al (1991)[34] the average GCS

score for all patients who survived was 4.8 and 4.4 among those died. The mortality / functional recovery ratio in each of GCS group was as follows GCS score 3, 90%; 5%, GCS 4, 76%; 10%, GCS 5, 62%; 18%, GCS 6 & 7, 51%, :44%. In our study 7% of patients with GCS <8 and 20% of patients with preoperative GCS <8 survived. Massaro et al (1996)[20] reported mortality of 70% and functional recovery in 11% cases of acute SDH patients with GCS <8 was significant. Similar outcome was reported by Servadei et al[28] and Kotwica[12].

Browers and Marshall[3] opinined that presence of chest or abdominal injuries requiring surgery had a significant impact on outcome, i.e., poly traumatized patients with acute SDH exhibited higher mortality rate[8]. In IDB study, however, failed to demonstrate any significant correction[10]. In our study only 12.5% patients with major injuries survived compared to 42% without it.

Yanaka et al[35] calculated haematoma volume and reported mean volume was 31mL for patients with favourable outcome and 104mL for patients with poor outcome. Calculation haematoma volume is neither practical nor reliable because of SDH is often spread over irregular surface. Servadie et al[25] in their study have indicated worse outcome as haematoma thickness increases.

Three factors contribute to the presence of midline shift, the first factor is haematoma thickness. Servadei et al[28] found a linear correlation between thickness of haematoma and midline shift. The second factor associated unilateral oedema[17,36] and associated parenchymal damage third factor. Kotwica and Bizezenski[12] showed 42% favourable outcome and a mortality of 39% when midline shift was below 1.5 cms : 25% favourable outcome and 52% mortality when this shift was 1.5-3cms. In our study there was some correlation between midline shift and outcome i.e., survival of 36% Servadei et al[28] did not find any significant relationship between outcome and brain swelling (Difference between midline shift and haematoma thickness).

The importance of effacement of basal cistern in proportion to mass effect and increased intracranial pressure has been emphasized[5,22,36]. Servadei et al[25] in their series; observed favourable outcome, only in 12% patients with completely obliterated basal cisterns. In a series by Yanaka et al[35] rate of favourable outcome was only 22% in acute SDH patients with compressed or absent cisterns on admission. In our study, 81% patients had cisterns effaced this may be due to increased severity of injury and presence of mass lesion. 22% of patients with cisternal effacement survived where as with normal cistern 100% survived.

Only recently[5,6,25,28] the presence of sub arachnoid haemorrhage had been identified to be an independent factor for poor outcome in patients with severe head injury. Previously published papers included patients with SAH among the group of patients without associated lesions[12,20,26,34]. In the series published by Servadei et al[25], evidence of SAH on the first CT scan was a powerful predictor of bad outcome. They have shown an unfavourable outcome in 65-75% of patients with presence of SAH in an initial CT scan. In our study 10% patients with SAH and 65% patients without SAH in initial CT scan survived and this was statistically significant.

In autopsy study of head injury Kristiansen and Tandon[14] concluded that acute subdural haematoma is seldom an isolated lesion. Concomitant brain injuries are of greater importance to the outcome than the effect subdural haematoma itself[7]. Tendon et al[31] had studied the importance of temporal lobe lesion in head injury, in that several other cases, coexistence of subdural haematoma was probably not documented in operation notes because the surgeon considered it insignificant in the production of clinical picture. The reported incidence of brain contusion associated with acute SDH ranges from 7% to 82%[12]. The presence of associated brain contusion is a powerful indicator of bad outcome. The rate of favourable outcomes ranged from 32% to 58% for patients with isolated SDH from 12% to 32%

those associated with haematomas. Servedei et al[25] reported 57% favourable outcome in patients with only SDH and 37% favourable outcome in SDH with contusion. In our study associated contusion was present in 32% of cases.

Thirty percent of cases with contusion and thirty nine percent of cases without contusion survived. In a study by Massaro et al[20] (1996) mortality was 62% in pure acute SDH, and 52% to 62% in patients acute SDH associated with contusion. Seeling et al[24] did not report significant differences in outcome of patients with and without associated contusions.

The very high mortality associated with surgery of acute SDH prompted, the neuro surgeons to delay operation on such patients. Futility of operating within 24 hours was a common refrain till 1970s. In a land mark paper by Seeling et al[24] reported dramatic reduction in the mortality of 30% if subdural haematoma was evacuated with in 4 hours of injury as compared to mortality rate of 85% as the operation was delayed. Stone et al[26] found no significant difference in outcome in those undergoing surgery less than 4 hours after injury (60% mortality) and those operated between 4 and 12 hours post injury. Nilberger et al[34] mortality rate of those patients operated on with in 4 hours of injury was 59% versus 69% for operated after four hours. Functional recovery for those group were 26% & 16% respectively.

While the debate regarding the benefits of early surgery (with in 4 hours) continues, it is generally agreed that the denial of operation during the first 24 hours of delaying surgery in the hope of improving outcome has no basics. Early surgery should be performed in patients with more than 5mm midline shift, without waiting for deterioration in the condition or rise intracranial pressure. Failure to observe progressive improvement and not evidence of deterioration is an indication itself for surgery. Believing the subdural clot as the real culprit responsible for clinical condition of a patient, various surgical procedures were used to evacuate the subdural clot. Tokutami et al[29] compared different surgical treatment in a series of 120 patients. In a comatose patient craniotomy had shown to yield a higher rate of good results (48%). The highest rate of death in the group of cases with evacuation and irrigation via Burr holes. In our study 11 patients underwent craniotomy and haematoma evacuation and lobectomy according to their requirement. Among them 27% survived.

In our study 31 patients (37%) survived and 53 patients (63%) expired. Kotwica recorded 23% favourable recovery and 55% mortality, Marshal[21] and Wilberger[34] recorded 14% and 19% favourable recoveries and 50% and 66% mortalities respectively.

CONCLUSIONS

1. Adults in their 3rd and 4th decade of life are commonest victims and there is significant difference in mortality between patients below and above 40 years of age.

2. Road traffic accident is the commonest cause of acute SDH due to head injury followed by accidental falls.

3. Initial GCS score correlated well with out come of patients with acute SDH.

4. Associated chest and long bone injuries had worst outcome in patients with acute SDH.

5. The midline shift and thickness of subdural haematoma though affected the outcome. But was not very significant.

6. Majority of patients with acute SDH have associated focal (contusion) or global (subarachnoid haemorrhage) involvement of brain or both.

7. Presence or absence of subarachnoid haemorrhage correlated well with outcome of patients with acute SDH.
8. Presence of contusion altered the mode of management and this group had worst outcome than in those where there was no contusion.

9. Surgical mode of management does not affect significantly the over all outcome of patient with acute SDH.

TABLES

1. Age Incidence and related

Age Incidence	Number	Survival	Percentage
11 to 20	3	2	66
21 to 30	11	8	72
31 to 40	27	9	30
41 to 50	16	4	25
51 to 60	18	3	16
61 to 70	7	4	57
> 70	2	1	50
Total	84	31	

($p = 0.22$ Not Significant)

2. Mode of Injury out come

Mode of Injury	Number	Survival	Percentage
RTA	72	22	30
Accidental Fall	6	5	83
Assault	6	4	66

(P = 0.01 : Significant)

3. Initial GCS and Outcome

GCS	Number	Survival	Percentage
< 8	47	4	9
9 to 12	13	8	62
13 to 15	24	19	79

(p = 0.0039 Significant)

4. Associated injuries and outcome

Present	Number	Survival	Percentage
Ortho	7	2	13
Chest injury	4	0	0
Faciomaxillary Injury	5	0	0
Absent	68	29	43

(P = 0.03 : significant)

5. Side of SDH Outcome

Side	Number	Survival	Percentage
Right	38	19	50
Left	39	9	23
Bilateral	7	3	43

(P= 0.43: Not Significant)

6. Midline Shift and Outcome

	Number	Survival	Percentage
Present	84	31	37
Absent	0	0	0

(p = 0.15 Not Significant)

7. Status of Basal cisterns and outcome

Status	Number	Survival	Percentage
Effaced	68	15	22
Normal	16	16	100

(p = 0.19 : Not Significant)

8. Traumatic subarchnoid haemorrhage and outcome

SAH	Number	Survival	Percentage
Present	42	4	10
Absent	42	27	64

(p = 0.00002 : Significant)

9. Associated Cerebral Contusion

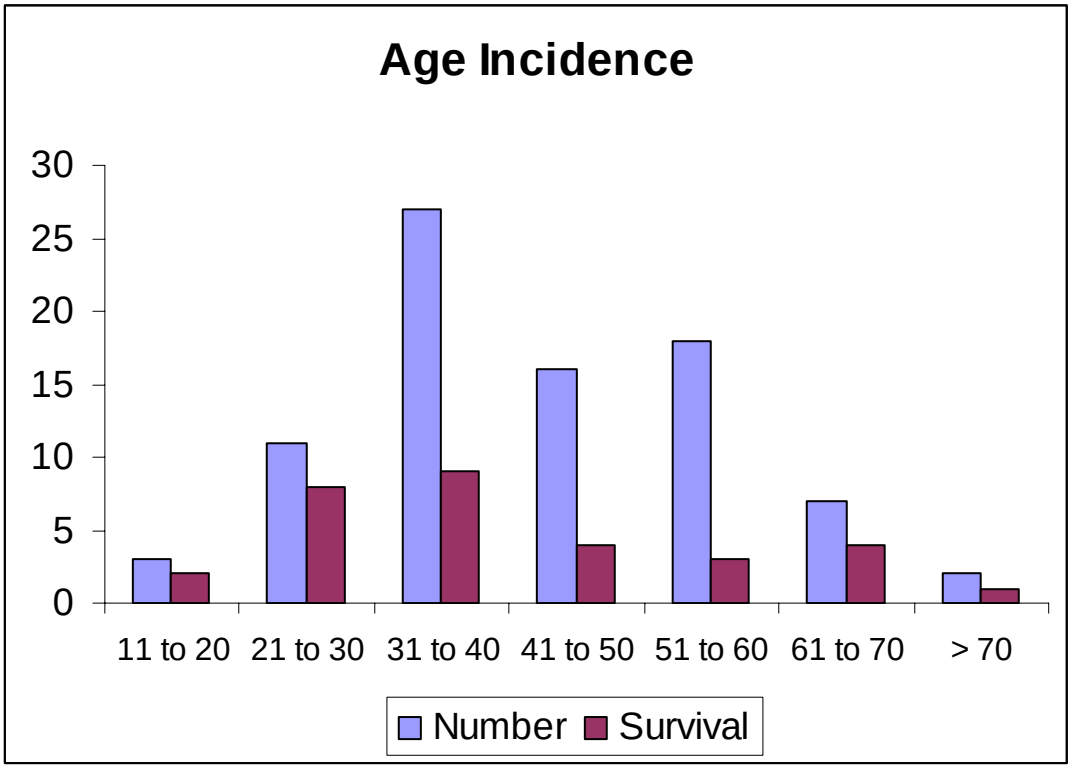
Contusion	Number	Survival	Percentage
Present	27	9	33
Absent	57	22	22

(p = 0.80 : Not Significant)

10. Management Modality and outcome

Mode of Treatment	Number	Survival	Percentage
Operative	11	3	27
Non Operative	73	28	38

(p = 0.56 : Not Significant)


Age Incidence
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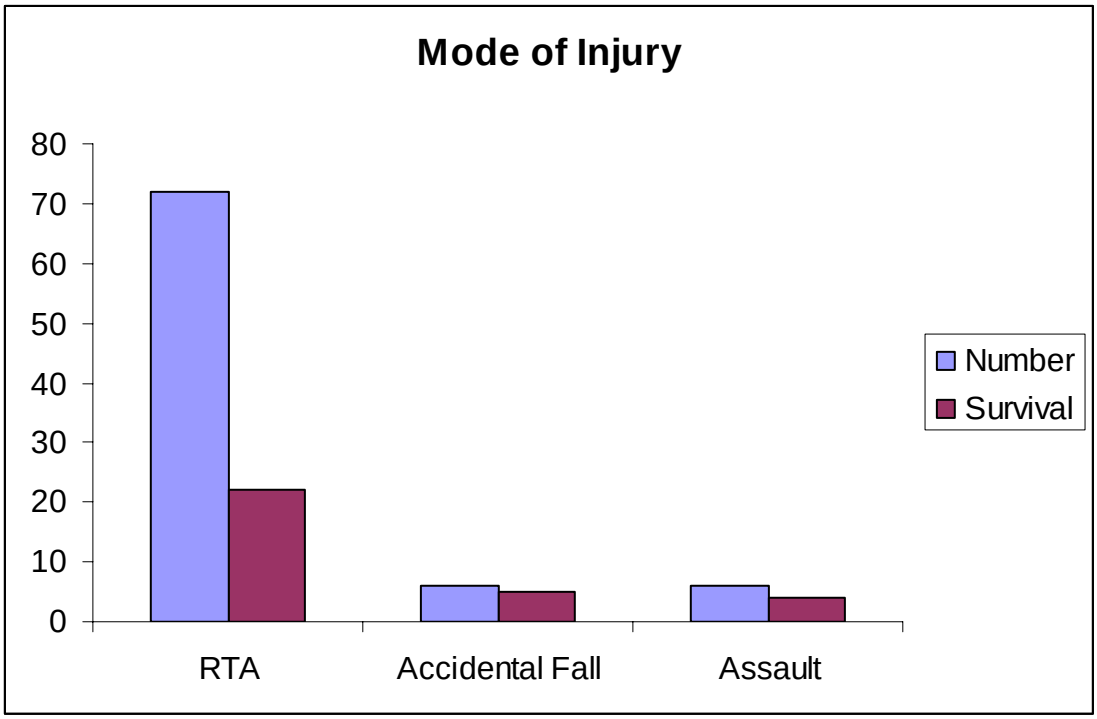
25
20
15
10
5
0
11 to 20
21 to 30
31 to 40
41 to 50
51 to 60
61 to 70
> 70
Number
Survival


Mode of Injury
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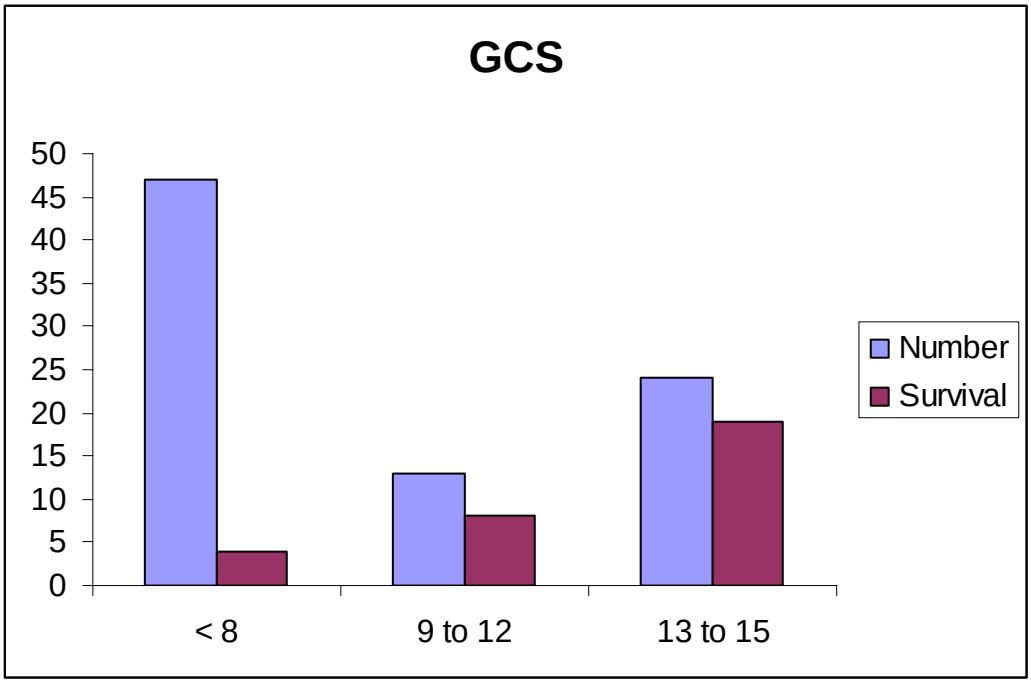
70
60
50
40
30
20
10
0
RTA
Accidental Fall
Assault
Number
Survival


GCS
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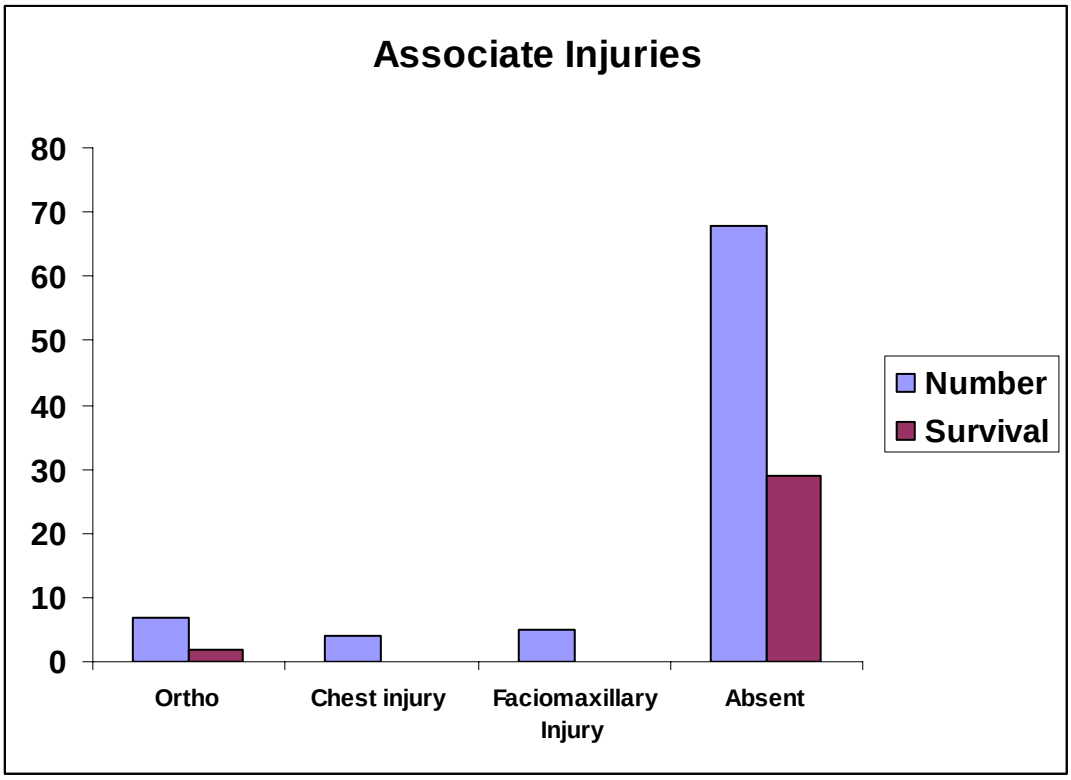
45
40
35
30
25
20
15
10
5
0
< 8
9 to 12
13 to 15
Number
Survival


Associate Injuries
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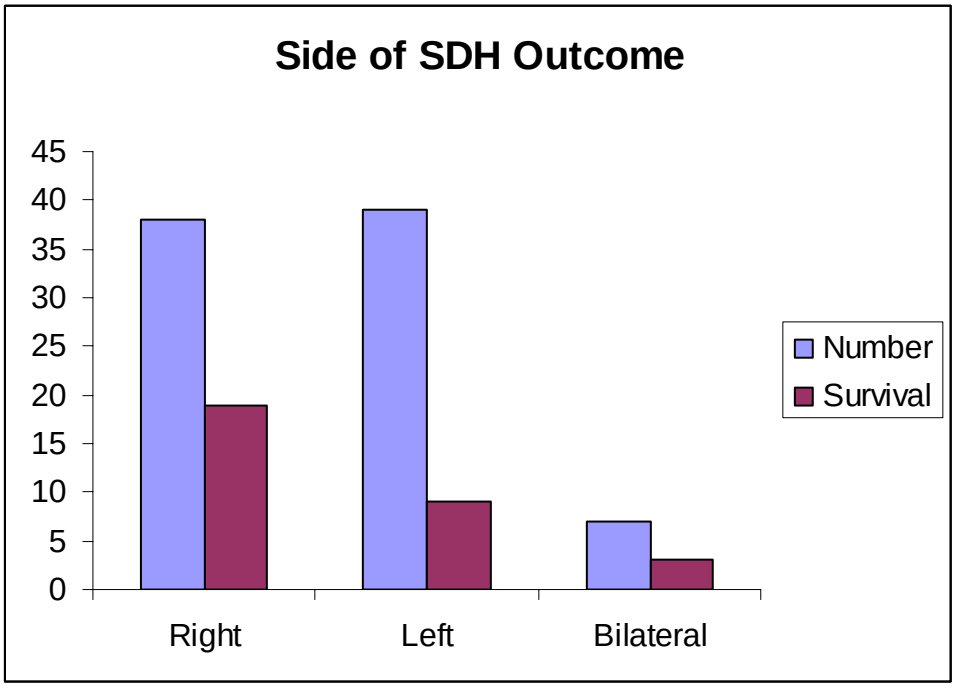
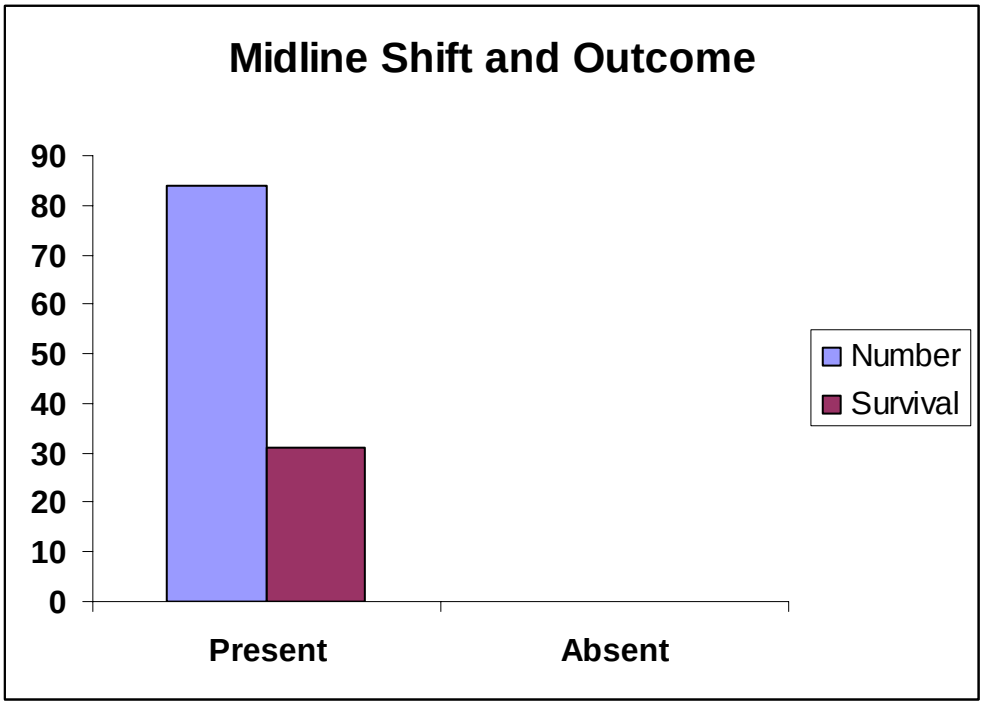
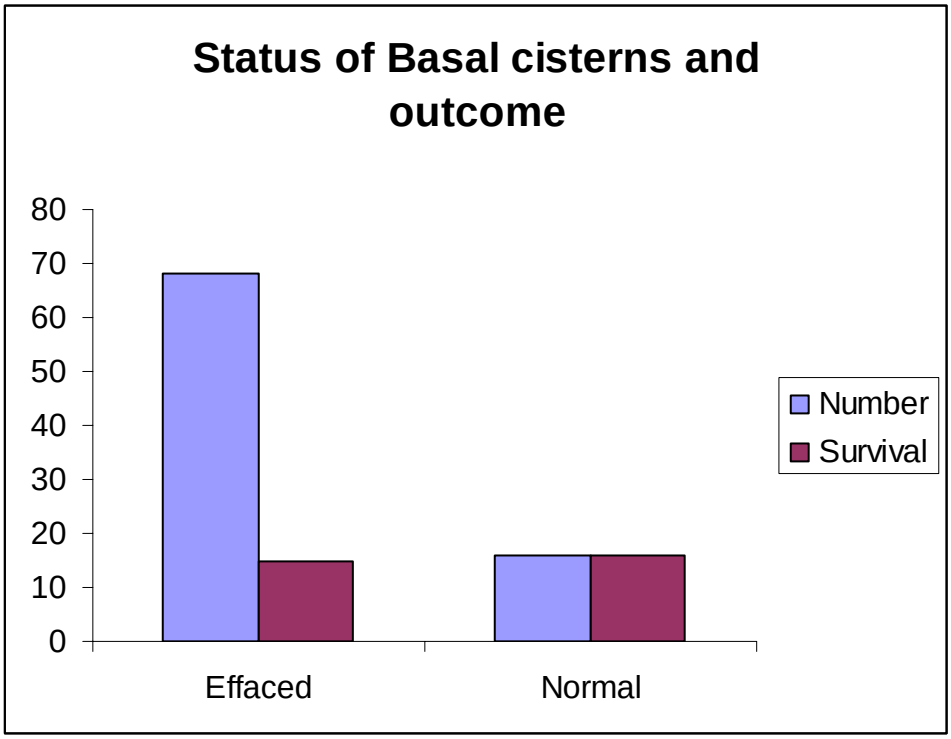
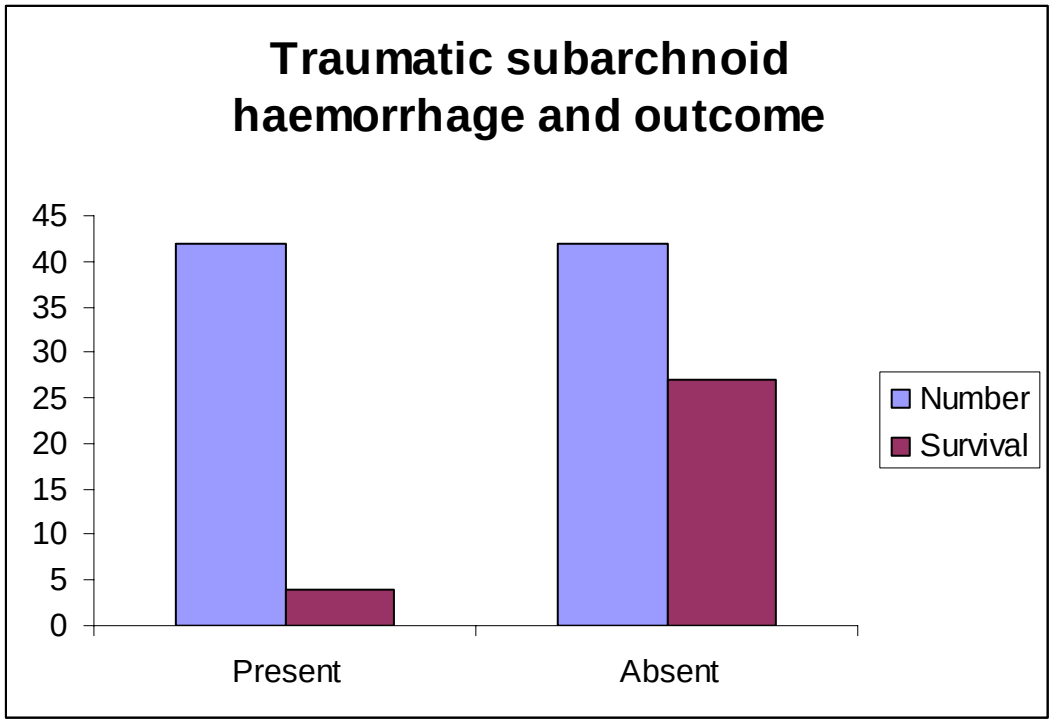
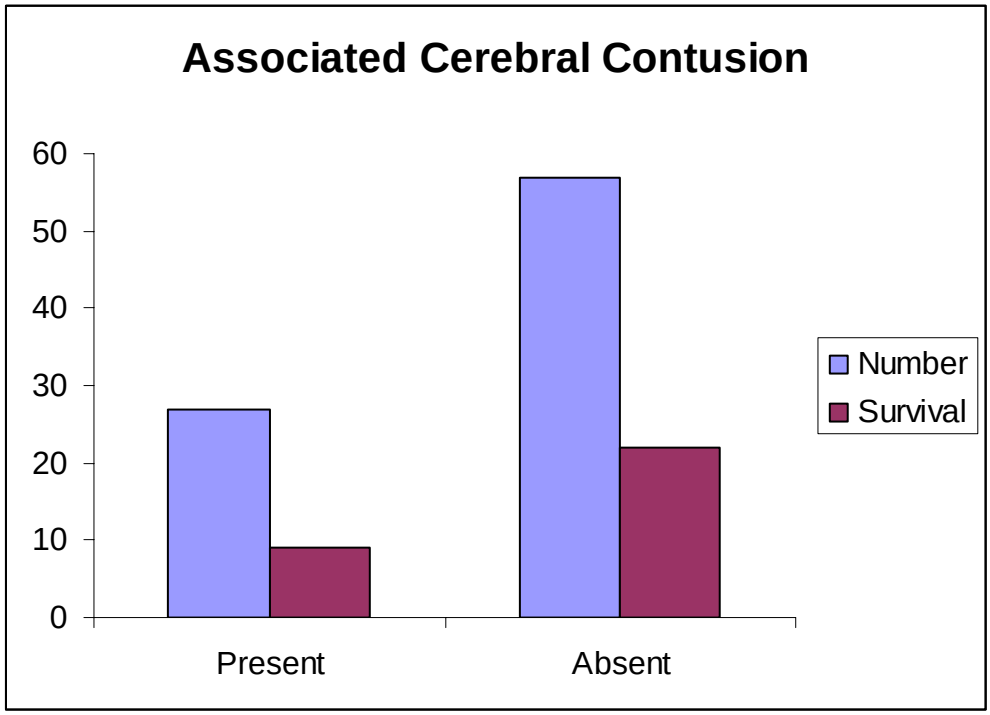
70
60
50
40
30
20
10
0
Ortho
Chest injury
Faciomaxillary Injury
Absent
Number
Survival

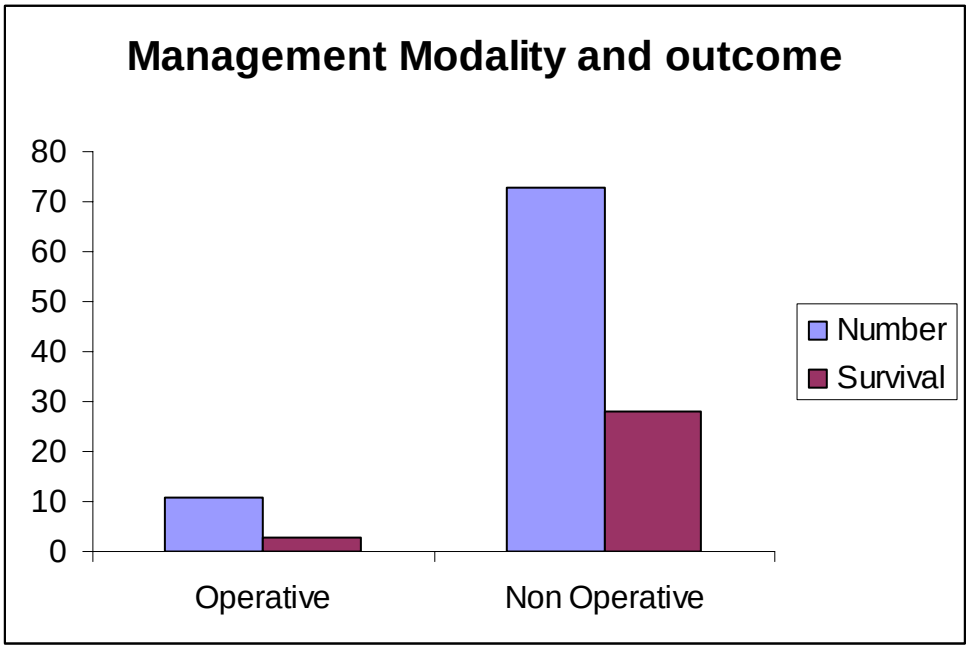

Side of SDH Outcome
45
40
35
30
25
20
15
10
5
0
Right
Left
Bilateral
Number
Survival


Midline Shift and Outcome
90
80
70
60
50
40
30
20
10
0
Present
Absent
Number
Survival


Status of Basal cisterns and outcome
80
70
60
50
40
30
20
10
0
Effaced
Normal
Number
Survival


Traumatic subarchnoid haemorrhage and outcome
45
40
35
30
25
20
15
10
5
0
Present
Absent
Number
Survival


Associated Cerebral Contusion
60
50
40
30
20
10
0
Present
Absent
Number
Survival



		Number	Survival	Prop	SE
Age < = 40	GCS	41	19	0.46	0.0778
	< 8	19	1	0.05	0.05
	> = 8	22	18	0.82	0.0819

Age	GCS	43	42	0.28	0.0685	1.23	82	0.2221
> 40	< 8	25	2	0.08	0.0543	0.29	42	0.775
	> = 8	18	10	0.56	0.117	1.31	38	0.199

Mode of Injury							
Accidental fall	47	4	0.09	0.0417	3.01	58	0.0039
Assault	6	4	0.67	0.19	2.6	76	0.0111

RTA	72	22	0.31	0.05	1.5	76	0.1378
GCS							
< = 8	47	4	0.09	0.0417	3.01	58	0.0039
9 to 12	13	8	0.62	0.1346	5.61	69	0.000001
13 to 15	24	19	0.79	0.0831	0.7809	35	0.4401
Associated Injuries							
Chest Injury	4	0					
Faciomaxillary	5	0					
Ortho	7	2	0.13	0.0827	2.1023	82	0.0386
No Injury	68	29	0.43	0.0600			

Side of SDH							
B/L SDH	7	3	0.43	0.1871	0.7859	44	0.4362
Left	39	9	0.23	0.0674	0.2610	43	0.7953
Right	38	19	0.50	0.0811	1.8182	75	0.073
CT FINDINGS Present							
Midline shift	84	31	0.37	0.0527	1.4426	150	0.1512
Basal cisterns effaced	68	15	0.22	0.0502	1.2986	108	0.1968
SAH	42	4	0.10	0.0463	1.7407	67	0.0863
Contusion	27	9	0.33	0.0905	0.2495	109	0.8034
CT FINDINGS Absent							

Midline shift	0	0					
Basal cisterns effaced	16	16					
SAH	42	27	0.64	0.0741	1.8025	47	0.0779
Contusion	57	22	0.39	0.0646			
			SAH	**Pre X Abs**	**4.4850**	**82**	**0.00002**
Mode of Treatment							
Non – operative	73	28	0.38	0.568	0.5768	82	0.5656
Operative	11	3	0.27	0.1339			

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MASTER CHART

S.No	NAME	Age/Sex	IP/NS NO	Mode of Injury	GCS	Side	CT Midline shift	Basal cistern	SAH	Contusion	Associated	Treatment	DOA	DOD
1	IRULAPPAN	43/M	1287/129	RTA	5/15	RIGHT	P	P	P	P		CONSERVATIVE	05.01.07	EXP I.06.01.07
2	KANNAIAH BABU	12/M	2093/196	RTA	10/15	LEFT	P	P	A	P		CONSERVATIVE	08.01.07	DIS 17.01.07
3	MARIYAMMAL	30/F	2550/224	RTA	10/15	RIGHT	P	A	A	P		CONSERVATIVE	10.01.07	DIS 04.02.07
4	MAYAN	15/M	2998/69	RTA	3/15	LEFT	P	P	P	P	# Left Leg	CONSERVATIVE	12.01.07	EXP 12.01.07
5	PADMA	48/F	3223/293	RTA	7/15	LEFT	P	P	P	P		DECOMPRESSIVE CRANITOMY	13.01.07	EXP 19.01.07
6	PANDIAN	53/M	6797/717	RTA	4/15	LEFT	P	P	P	P		CONSERVATIVE	28.01.07	AMA 01.02.07
7	SELVI	53/M	6851/726	RTA	5/15	LEFT	P	P	P	P		DECOMPRESSIVE CRANITOMY	28.01.07	EXP 31.01.07
8	ANNADURAI	60/M	7907/809	ACCIDENTAL FALL	5/15	LEFT	P	P	A	P		DECOMPRESSIVE CRANITOMY	01.02.07	DIS 12.02.07
9	MUTHU	55/M	11129/1096	RTA	3/15	LEFT	P	P	P	P	# Ribs R	CONSERVATIVE	14.02.07	EXP 15.02.07
10	BALAKRISHNAN	50/M	11162/1121	RTA	15/15	RIGHT	P	A	A	A		CONSERVATIVE	21.02.07	DIS 06.03.07
11	RAKKIAMMAL	25/F	13318/1295	ASSUALT	15/15	RIGHT	P	A	A	P		CONSERVATIVE	23.02.07	DIS 08.03.07
12	SIVAN	40/M	13585/1324	RTA	3/15	LEFT	P	P	P	P		DECOMPRESSIVE CRANITOMY	24.02.07	EXP 26.02.07
13	PERIYAKARUPPAN	38/M	17265/1672	RTA	4/15	LEFT	P	P	P	A		CONSERVATIVE	10.03.07	EXP 11.03.07
14	VIMALA	40/F	17490/1685	RTA	3/15	LEFT	P	P	P	P		DECOMPRESSIVE CRANITOMY	12.03.07	EXP. 17.03.07
15	GOPAL	52/M	18331/1761	RTA	13/15	LEFT	P	P	A	P		DECOMPRESSIVE CRANITOMY	14.03.07	EXP 16.03.07
16	DHANALAKSHMI	60/F	20255/1899	RTA	13/15	LEFT	P	P	A	A	Faciomaxillary	CONSERVATIVE	21.03.07	EXP 23.03.07
17	GANESHAN	52/M	20299/1901	RTA	7/15	RIGHT	P	P	A	A		CONSERVATIVE	21.03.07	DIS 25.03.07
18	THANGADURAI	40/M	20846/1937	RTA	3/15	RIGHT	P	P	P	P		CONSERVATIVE	23.03.07	EXP 27.03.07
19	RAJU	28/M	22263/2065	RTA	3/15	RIGHT	P	P	P	P		CONSERVATIVE	28.03.08	EXP 29.03.07
20	THANGAMMAL	65/M	22281/2072	ACCIDENTAL FALL	4/15	RIGHT	P	P	P	A	# L Femur	CONSERVATIVE	29.03.07	AMA 29.03.07
21	PITCHAI	50/M	22297/2128	RTA	6/15	RIGHT	P	P	P	A		CONSERVATIVE	31.03.07	EXP 02.04.07
22	PRABHAKARAN	40/M	24882/2319	ASSUALT	10/15	B/L SDH	P	P	A	A		CONSERVATIVE	07.04.07	ABS 09.04.07

23	RAMASAMY	55/M	24928/2321	RTA	8/15	LEFT	P	P	P	A		CONSERVATIVE	07.04.07	EXP 27.04.07
24	UNKNOWN	30/M	25406/2374	RTA	7/15	LEFT	P	P	A	P		CONSERVATIVE	09.04.07	ABS 10.04.07
25	PRABHU	20/M	25393/2373	RTA	8/15	RIGHT	P	P	A	A		CONSERVATIVE	09.04.07	DIS 20.02.07
26	AYYASAMY	60/M	25692/2391	RTA	3/15	LEFT	P	P	P	A	Faciomaxillary	CONSERVATIVE	10.04.07	EXP 13.04.07
27	MADHURAJ	43/M	26492/2466	RTA	4/15	LEFT	P	P	P	P		DECOMPRESSIVE CRANITOMY	13.04.07	EXP 17.04.07
28	ALAGU	35/M	26644/2493	RTA	3/15	LEFT	P	P	P	A		CONSERVATIVE	14.04.07	AMA 15.04.07
29	SANNASI	65/M	28304/2627	ACCIDENTAL FALL	15/15	RIGHT	P	A	A	A		CONSERVATIVE	20.04.07	DIS 30.04.07
30	MAYAN	55/M	28514/2652	ASSUALT	10/15	LEFT	P	P	A	A		CONSERVATIVE	21.04.07	EXP 28.04.07
31	VATHAVALLI	45/F	29065/2703	RTA	3/15	RIGHT	P	P	P	A	B/L Ribs #	CONSERVATIVE	23.04.07	EXP 24.04.07
32	KATHIRAVAN	26/M	30564/2823	ASSUALT	15/15	RIGHT	P	A	A	A		CONSERVATIVE	29.04.07	DIS 09.05.07
33	ALAGUMUTHU	40/M	31021/2884	RTA	15/15	RIGHT	P	A	A	A		CONSERVATIVE	01.05.07	DIS 09.05.07
34	VASANTHA	45/M	31907/2950	RTA	9/15	RIGHT	P	P	P	A		CONSERVATIVE	04.05.07	DIS 17.05.07
35	SUBBAIAH	64/M	33233/3070	RTA	4/15	RIGHT	P	P	P	A	R Femur #	CONSERVATIVE	09.05.07	EXP 10.05.07
36	NAGARAJ	23/M	33520/3091	RTA	13/15	RIGHT	P	A	A	A		CONSERVATIVE	10.05.07	DIS 14.05.07
37	VASANTHA	55/F	33548/3095	RTA	14/15	LEFT	P	A	A	A		CONSERVATIVE	10.05.07	DIS 14.05.07
38	NARAYANAN	37/M	33748/3123	RTA	15/15	RIGHT	P	A	A	A		CONSERVATIVE	11.05.07	DIS 16.05.07
39	DHANAM	86/M	34272/3168	RTA	12/15	RIGHT	P	P	A	A		CONSERVATIVE	14.05.07	DIS 26.05.07
40	MANI	26/M	34447/3190	RTA	15/15	LEFT	P	A	A	A		CONSERVATIVE	14.05.07	DIS 17.05.07
41	GANAPATHY	40/M	34428/3190	RTA	3/15	LEFT	P	P	P	A		CONSERVATIVE	14.05.07	EXP 14.05.07
42	KARUPPANAN	55/M	35450/3266	RTA	6/15	LEFT	P	P	A	A	Faciomaxillary	CONSERVATIVE	18.05.07	EXP 19.05.07
43	PUSBHA	30/F	35721/3289	RTA	3/15	LEFT	P	P	P	A		CONSERVATIVE	19.05.07	EXP 22.05.07
44	SUBRAMANI	35/M	35822/3301	RTA	7/15	LEFT	P	P	P	A		CONSERVATIVE	20.05.07	EXP 21.05.07
45	PARANTHAMAN	38/F	35/854/3308	RTA	7/15	LEFT	P	P	P	P		CONSERVATIVE	20.05.07	EXP 23.05.07
46	PUSBHA	40/F	35916/3399	RTA	3/15	LEFT	P	P	P	P		DECOMPRESSIVE CRANITOMY	26.05.07	EXP 27.05.07
47	PALANI	40/M	38628/3578	RTA	4/15	RIGHT	P	P	P	A	# R Ribs	CONSERVATIVE	30.05.07	EXP 01.06.07

48	CHINNA SANKARAN	50/M	38914/3610	RTA	4/15	LEFT	P	P	P	A		CONSERVATIVE	31.05.07	EXP 01.06.07
49	HARICHANDRAN	45/M	42488/3307	RTA	10/15	RIGHT	P	P	A	A		CONSERVATIVE	15.06.07	EXP 15.06.07
50	LAKSHMI	32/F	42502/3310	RTA	14/15	RIGHT	P	A	A	A		CONSERVATIVE	15.06.07	DIS 23.06.07
51	LOORDURAI	50/M	42317/3306	RTA	10/15	LEFT	P	A	P	P		DECOMPRESSIVE CRANITOMY	16.06.07	DIS 26.06.07
52	THANGARAJ	40/M	43700/3369	RTA	4/15	RIGHT	P	P	P	A	# Fore arm	CONSERVATIVE	20.06.07	AMA 21.06.07
53	SIVARAM	45/M	44270/3378	RTA	4/15	B/L SDH	P	P	P	A		CONSERVATIVE	22.06.07	EXP 22.06.07
54	SIVANKALAI	34/M	50498/3579	RTA	7/15	RIGHT	P	P	A	P		DECOMPRESSIVE CRANITOMY	15.07.07	EXP 16.07.07
55	MALAISAMY	32/M	54262/3694	RTA	15/15	LEFT	P	A	A	A		CONSERVATIVE	29.07.07	DIS 02.08.07
56	RAMRAJ	48/M	55338/3704	RTA	7/15	LEFT	P	P	A	A	Faciomaxillary	CONSERVATIVE	30.07.07	EXP 03..08.07
57	ARULSAKTHI	54/M	56506/3746	RTA	3/15	RIGHT	P	P	P	A		CONSERVATIVE	05.08.07	EXP 06.08.07
58	KARUPPASAMY	39/M	56803/3762	RTA	8/15	RIGHT	P	P	P	A		CONSERVATIVE	06.08.07	EXP 11.08.07
59	AYYANKANNU	29/M	58316/3789	RTA	15/15	LEFT	P	A	P	A		CONSERVATIVE	10.08.07	DIS 21.08.07
60	NAGARAJ	40/M	58394/3792	RTA	3/15	RIGHT	P	P	P	P		CONSERVATIVE	10.08.07	EXP 11.08.07
61	MURUGESAN	50/M	61857/3825	RTA	13/15	RIGHT	P	P	A	A		CONSERVATIVE	17.08.07	EXP 25.08.07
62	SUBRAMANI	34/M	62713/3868	RTA	14/15	B/L SDH	P	P	A	A		CONSERVATIVE	21.08.07	DIS 28.08.07
63	RAJYOSINGH	50/M	63109/3874	RTA	3/15	LEFT	P	P	A	A		CONSERVATIVE	22.08.07	EXP 27.08.07
64	RAMAMOORTHY	32/M	64728/3902	RTA	12/15	LEFT	P	P	A	P		CONSERVATIVE	25.08.07	DIS 02.09.07
65	KARUPPASAMY	40/M	66734/5832	RTA	15/15	LEFT	P	P	A	A	# Femur L	CONSERVATIVE	30.08.08	TRANSFER OUT TO 99
66	PALANI	45/M	67012/3958	RTA	9/15	LEFT	P	P	A	A	B/L Ribs #	CONSERVATIVE	01.09.07	EXP 08.09.07
67	KALIAMMAL	58/F	68539/4046	RTA	10/15	RIGHT	P	P	A	P		CONSERVATIVE	09.09.07	EXP 10.09.07
68	UNKNOWN	39/M	68539/4046	RTA	13/15	RIGHT	P	P	A	A	# R Upper limb and R femur	CONSERVATIVE	12.09.07	TRANSFER OUT TO 99
69	ARUNKUMAR	31/M	69220/4064	RTA	11/15	RIGHT	P	P	A	A		CONSERVATIVE	13.09.07	DIS 17.09.07
70	GOPAL	36/M	69414/4067	ASSUALT	15/15	RIGHT	P	P	A	A		CONSERVATIVE	13.09.07	DIS 16.09.07
71	JESINTHA	58/F	69551/4072	RTA	3/15	RIGHT	P	P	P	A		CONSERVATIVE	14.09.07	EXP 14.09.07

72	RAJAGOPAL	42/M	69770/4081	ASSUALT	14/15	B/L SDH	P	P	A	A		CONSERVATIVE	21.09.07	DIS 20.09.07
73	SAKTHIVEL	54/M	71491/4084	RTA	4/15	RIGHT	P	P	P	A		CONSERVATIVE	25.09.07	EXP 22.09.07
74	VELLAITHAI	62/F	72592/4115	ACCIDENTAL FALL	15/15	B/L SDH	P	A	A	A		CONSERVATIVE	02.10.07	DIS 27.09.07
75	SWAMINATHAN	58/M	76483/4185	RTA	3/15	LEFT	P	P	P	A		CONSERVATIVE	10.10.07	EXP 03.10.07
76	PANDIAN	37/M	78427/4247	RTA	3/15	LEFT	P	P	P	A		CONSERVATIVE	10.10.07	EXP 10.10.07
77	ANDAL	70/F	78428/4248	ACCIDENTAL FALL	10/15	RIGHT	P	P	A	P		CONSERVATIVE	12.10.07	DIS 16.10.07
78	PRAMBHAN	75/M	79463/4271	RTA	3/15	RIGHT	P	P	P	A	Faciomaxillary	CONSERVATIVE	14.10.07	EXP 12.10.07
79	RAJARATHINAM	60/M	79776/4286	RTA	6/15	B/L SDH	P	P	P	A		CONSERVATIVE	19.10.07	EXP 14.10.07
80	RANJTHKUMAR	27/M	81073/4335	RTA	7/15	RIGHT	P	P	P	P		DECOMPRESSIVE CRANITOMY	23.10.07	DIS 01.11.07
81	MURUGAPRABHU	26/M	81891/4362	RTA	14/15	LEFT	P	P	A	A		CONSERVATIVE	23.10.07	EXP 07.11.07
82	PONNAIAN	40/M	81801/4367	RTA	15/15	B/L SDH	P	P	A	A	# R Fore arm	CONSERVATIVE	28.10.07	EXP 29.10.07
83	SADAIAN	66/M	83293/4299	ACCIDENTAL FALL	15/15	RIGHT	P	A	A	P		CONSERVATIVE	31.10.07	DIS 06.11.07
84	MEENA	64/F	83940/4318	RTA	5/15	LEFT	P	P	P	A		CONSERVATIVE	31.10.07	EXP 31.10.07