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Predictors of Outcome in Acute Subdural Hematoma with severe head injury- A Prospective Study

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Abstract: Acute subdural hematoma(SDH) is an uncommon condition, however, SDH associated with cerebral contusion or laceration is more frequent. Acute SDH still carry a high mortality and morbidity. This paper is a prospective analysis of 61 acute SDH patients admitted over 1 year period. Glasgow Coma Score of all the patients was 8 or less. Thickness of SDH, midline shift, associated pathology along with clinical parameters were correlated with outcome. While all the patients were ventilated, patients with significant mass lesion and midline shift were subjected to surgery. Forty percent patients were under 40 years of age and 85% patients were involved in vehicular accidents. Nine patients were M1 (motor response), 30 patients had bilateral fixed dilated pupils and 8 patients had unilateral dilated pupils. Overall survival was recorded in 21 patients (34.6%) and16.6% patients with bilateral dilated pupils survived. Young age, bilaterally reacting pupils, normal cold caloric response and midline shift less than 5 mm were good prognostic factors. Only 2 out of 21 patients with diffuse cerebral swelling survived. None of our patients with shock survived. Our study brings out the factors, which influence the outcome in patients with acute SDH.

Keywords: acute subdural hematoma, Glasgow outcome scale, severe head injury, traumatic subarachnoid hemorrhage

Introduction

Head injury as a result of accidents are the leading cause of death; and acute subdural hematoma is one of the common types of intracranial hematoma which continues to have a high mortality[1]. Acute SDH is generally only the tip of iceberg and has misled the neurosurgeons in evolving an appropriate management. It represents a challenging clinical entity and is associated with poor prognosis despite advances in head injury management .The mortality rate in comatose patients (GCS<8) is 50-80%. Acute SDH is seldom isolated ,even microscopic changes of DAI have been seen in patients seeming to have pure SDH on CT scanning[2-3]. Besides natural factors such as age, other factors like pupillary abnormality, initial coma scale, associated extracranial injury and raised intracranial pressure significantly influence the outcome. Finding associated on CT scans such as hematoma volume, midline shift, associated lesions, presence of subarachnoid hemorrhage and basal cisterns compression also have been related to outcome. Other factors like interval between trauma and evacuation of hematoma and type of surgery are also significant prognostic factors.[4] A prospective study was therefore carried out to study the role of various clinical/radiological and management related factors in influencing outcome in severe head injury patients with acute subdural hematoma.

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Materials and methods

A series of 61 consecutive patients of acute subdural hematoma with a post resuscitation GCS of < 8, admitted and treated at our institute over one year period, were evaluated. All patients had a supratentorial acute subdural hematoma with or without associated parenchymal lesions (contusion, hematoma, subarachnoid hemorrhage, and infarct) on CT scan. Patients expiring within six hrs of admission were not included in this study. Clinical profile of patients was recorded which included age, sex, mode of injury, post resuscitation GCS, pupillary response, oculocephalic reflex, associated medical illness, hypotension and any other associated systemic injury. All patients underwent an immediate CT scan following admission and CT findings (site, thickness of acute subdural hematoma, degree of midline shift, associated parenchyma pathology, cisternal effacement, presence of subarachnoid hemorrhage, intraventricular hemorrhage, infarcts) were noted (Figs 1-5). All patients were intubated on admission. Patients were taken up for surgery depending on the mass lesion (SDH/contusion), mass effect and clinical status. Moribund patients, those with GCS 3 with thin subdural hematomas, no significant midline shift and diffuse hemispheric swelling were not operated. Large Falconers flap craniotomy followed by subdural hematoma evacuation and lobectomy was done depending on the

presence of contusion and condition of the brain in those who were operated. A note was made on the condition of the brain at the beginning and end of surgery. In case brain continued to be tense after evacuation of SDH and lobectomy, then duroplasty was done and bone flap was not replaced. In some patients, postoperative intracranial pressure monitoring was done with the help of an intracavitary catheter(Figs 1-5). The time gap between time of admission to surgery and time of injury to surgery, were recorded. GCS at admission and just prior to surgery was recorded to take note of preoperative deterioration if any. All the patients were electively ventilated and received intravenous mannitol/glycol for initial 48-72 hrs followed by oral glycerol and frusemide, according to the requirement. All the patients also received intravenous prophylactic anticonvulsants (diphenylhydantoin). Antibiotics were given according to our protocol and those requiring prolonged hospitalization received culture based antibiotics subsequently. Patients deteriorating or not showing expected recovery had repeat CT scanning. All the complications were treated accordingly. Patients needing prolonged intubation underwent tracheostomy. Patient's outcome was recorded at discharge, at 3 months and at 6 months following injury (according to Glasgow outcome scale). The data obtained were analyzed and clinical features, radiological parameters and management protocol were then correlated with outcome using Chi square, Fischer's exact test analysis and paired t-test wherever applicable.

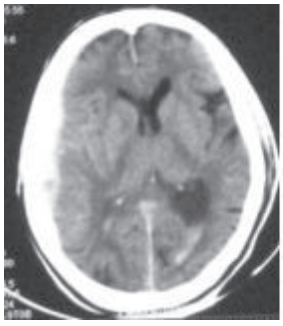

Fig.1a

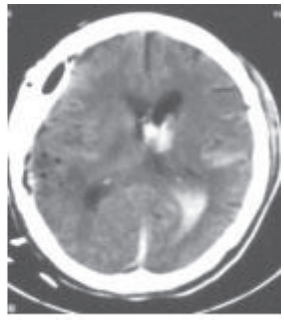

Fig1b.

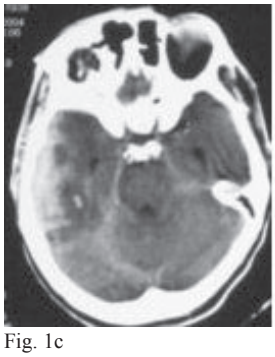

Fig. 1c

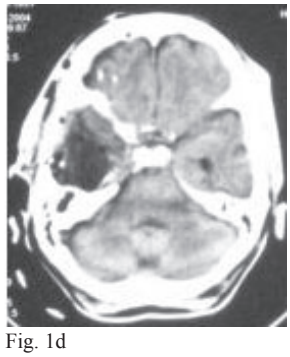

Fig. 1d

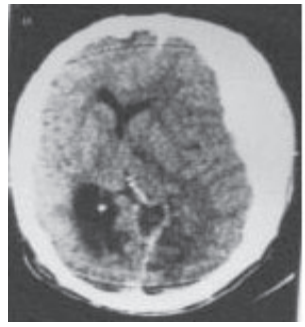

Fig. 2

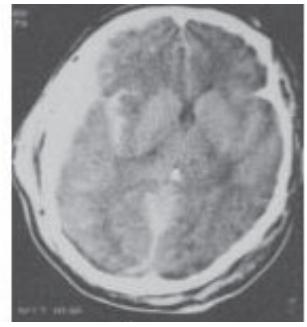

Fig. 3

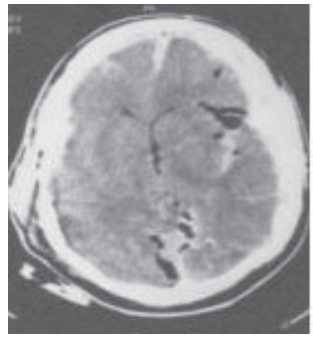

Fig. 4

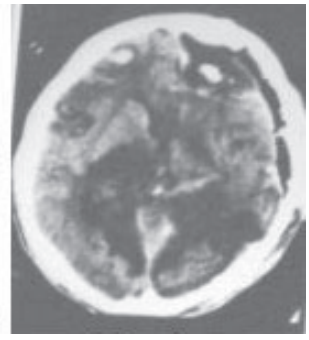

Fig. 5

Observations

The study consisted of 61 consecutive patients of acute SDH with GCS<8. Age of patients ranged from 17 -81 years (57% of these were in their 3rd and 4th decades). While 41% of patients with age less than 40 years survived, only 22% above age of 40 years survived and this difference was statistically significant. (p value: 0.044). Mean age in survivors was 32.7±11.57 years and 40.4±14.95 years in those who had expired. There were 51 males out of which 19 survived while 2 of 10 females survived. Mode of injury was road traffic accidents in 85% patients and fall from height in 15% of patients.

All patients except one were unconscious at the time of admission. Poor motor response (M1-M3) was seen in 60% of patients. All 9 patients who had M1 motor respnse and the only patient of M6 group expired. Survival was better in those with good preoperative motor scores (62.5% of patients of M4-M5 score survived while only 28% with M2-M3 scores survived and this difference was statistically significant). Overall 21out of 61 patients (34.4%) survived. Mean motor response in-patients who survived were 4.1±1.044 and in those who expired were 2.4±1.215. (Table 1). Pupillary abnormality correlated well with the outcome. Out of 22 patients who had bilateral reacting pupils, 15(68%) survived whereas among 30 patients with bilateral non-reacting pupils only 5 survived (16.6%). Eight patients had unilateral reacting pupils, out of which only

one survived. (Table 1)

Table 1: Clinical/Radiological/Surgical factors affecting outcome in Acute Subdural Hematoma patients.

Factor	Number of Patients	Survival	Percentage	
Motor response				
M1	9(13.1%)	0	0	
M2	20(32.9%)	2	10	
M3	7(13.1%)	4	57	(P<0.001,Significant)
M4	14(22.9%)	5	35.7	
M5	10(16.4%)	10	100	
M6	1(1.6%)	-	-	
Pupillary abnormality				
BL reacting	22(36%)	15	68	
BL norreacting	30(49.2%)	5	16.6	(P<0.001, Significant)
Unilateral reacting	8(13.1%)	1	12.5	
Oculocephalic response				
Present	26(42.6%)	15	57.7	
Absent	32(52.5%)	3	9.4	(P=0.003, Significant)
Not recorded	3(5%)	-	-	
Subdural hematoma thickness(mm)				
1-5	18	6	33	
6-10	21	12	57	
11-15	19	2	10.5	(P=0.147, Not Significant)
16-20	2	0	-	
21-25	1	1	100	
Midline shift(mm)				
1-5	6	2	33	
6-10	21	9	42	(P=0.147, Not Significant)
11-15	25	8	32	
16-20	9	2	22	
Status of basal cisterns				
BL Effaced	53	16	30.1	
UL effaced	7	4	57	(P=0.5, Not Significant)
Normal	1	1	100	
Traumatic SAH on initial CT				
Present	31	7	22.5	
Absent	30	14	44.6	(P=0.047, Significant)
Associated Cerebral contusion				
Single	22	10	45	
Multiple	18	9	50	(P=0.012, Significant)
No contusion	21	2	9.5	
Presence of infarct on initial CT				
Present	17	3	17.6	(P<0.001, Significant)
Absent	44	17	38.6	
Duration between injury to surgery(hrs)				
0-5	21	6	28.6	
6-10	22	9	40.9	(P=0.203, Not Significant)
>10	9	4	44.4	
Condition of brain At start of surgery:				
Tense	50	18	35.2	
Lax	2	1	50	
At end of surgery				(P<0.001, Significant)
Tens.e	17	0	0	
Lax	35	19	54.2	

Presence or absence of oculocephalic response was also predictive of the outcome in the present study. Among 26 patients who had positive oculocephalic response,57% survived while only 3 of 32 patients with absent oculocephalic response (9.4%) survived. (Table 1). Major associated injuries were seen in 17(28%) patients (10 had associated long bone fractures, 6 had associated chest injuries with hemo- or pneumothorax and fracture ribs, one patient had severe faciomaxillary injury) . Thirty percent of patients who had major such injuries survived while 37% patients without associated major injuries survived (p value: 0.83, not significant).

SDH was seen on right side in 31(22.6% of these survived) and on left side in 40 patients (44.6% of these survived). Though survival may appear significantly better in left-sided SDHs, but out of the 9 patients who were with M1 motor response, 8 had right sided SDH and all expired. So the side of SDH did not influence the outcome significantly; though the mean motor response of patients, with right-sided SDH was 2.7, and that of left side was 3.5.

Subdural hematoma thickness of 1-5 mm was seen in 18 patients out of whom 33% survived whereas 57% patients with 6-10mm hematoma thickness survived. In patients who had thickness of 11-15 mm (n=19) , only 2 (10.5%) survived. One patient who had SDH thickness more than 20 mm survived; this was in a 57 year old male with atrophic brain and no underlying contusion but outcome was poor. Mean thickness of SDH in-patients who survived was 7.76±3.99mm and in those who expired were 9.38±4.11mm. (Table 1).

Survival was found to be inversely related to midline shift (42% in-group of patients with 6-10 mm shift and 22% in those with 16-20 mm shift). Bilateral basal cistern effacement was seen in 87% patients and of these only 30% survived while 62% patients with unilateral or no cisternal effacement survived. A total of 31 cases (50.8%) patients had SAH and it was found to correlate with survival as 22.5% patients (7of 31 with presence of SAH) survived while only 46.4% patients without subarachnoid hemorrhage (14 of 30 patients without SAH) survived. (Table 1).

Associated contusion was present in 40 patients (65.6%). Only 2 out of 21 patients with diffuse brain swelling survived. All except one patient with contusions (who had a posterior temporal contusion, was managed conservatively and survived) underwent surgery. Brain stem/basal ganglia/PCA territory infarcts were seen in 17 patients and of these only 3 (17.6%) survived and this was found to be significant (p value<0.001). Two patients had pneuomocephalus and both expired. One patient had thin parietal EDH on opposite site, which was managed conservatively, and another patient had a posterior fossa EDH for which she was operated upon. Repeat CT was done in 36 patients of which 15 patients survived. In 10

out of 15 (66.7%) repeat CT had shown an improvement in mass effect in the form of decrease in midline shift, opening of ventricles or basal cisterns. However only in 3 of the 21 patients who expired, serial CT scan had shown an improvement in mass effect. Fresh contusions or hemorrhages were seen in 6 cases, flap site EDH was seen in 2 cases and opposite side EDH seen in one case. All of them required surgery. Fresh infarct was seen in 10 cases out of which only 3 survived. (Table 1). Nine patients (14.8%) deteriorated preoperatively (average time gap between admission and surgery was 1-8 hrs in these patients) and 66.6% of these died. Condition of the brain at the end of surgery was lax in 35 (67.3%) patients and amongst them 19(52.8%) survived. Five patients (10%) required redo surgery. Two patients had flap site EDH, one patient had opposite side EDH. One patient was operated for large opposite side frontal contusion and one of them underwent resurgery at same site for residual contusion and mass effect.

Chest infection was the commonest postoperative complication seen in 32.8% patients. Hypotension was noticed in 21% patients and was 100% fatal. Eight patients (15%) had meningitis and among them 4 (50%) survived. Other complications seen were septicaemia, acute renal failure, and persistent hypothermia and CSF rhinorrhea.

Out of 61 patients, 21 survived (34.4%). At discharge, according to Glasgow outcome scale , 5 patients (23.8%) had moderate disability, 9 patients (42.8%) had severe disability and 7 patients (33.3%) were in vegetative state. At 3 months follow-up available in 16 patients, 8 were moderately disabled, 6 had severe disability and two continued to be in vegetative state. More than 6 months follow up was available in 8 patients of whom 4 showed good recovery, 2 were moderately disabled and 2 were severely disabled. (Table 2) In-patients, who survived, mean duration of hospital stay was 21.7 days (range 5-60 days). Overall outcome as last recorded is 8 (13%) have favorable recoveries and 13 patients (21.3%) have unfavorable recoveries.

Table 2 : Overall outcome in Acute SDH patients

Outcome	At discharge	At end of 3 months	At end of 6 months
Good recovery	-	-	4
Moderate disability	5	8	2
Severe disability	9	6	2
Vegetative	7	2	-
Dead	40	-	-

Table 3: Mortality/functional recovery correlation with Glasgow coma scale (Wilbenger)[9]

Glasgow Coma Scale	Mortality (%)	Functional Recovery (%)
3	90	5
4	76	10
5	62	18
6-7	51	44

DISCUSSION

A strong correlation exists between age and outcome in patients with acute subdural hematomas and in general, increasing age is associated with poorer outcome in adults, although for children the opposite may be true[5-7]. Survival in our patients was found to be better in those below 40 years of age (22% patients older than 40 years survived while 41% patients younger than 40 years survived) and this difference was found to be statistically significant. Howard et al observed 18% mortality in young and 66% mortality in old patients and reported a good correlation between age and outcome[6]. Acute SDH in young patients may be a epiphenomenon, whereas in elderly patients , the mass itself is probably the most important pathological process and poor outcome in elderly may also be because of impaired regenerative capacity of the atrophic aging brain. Seeling et al did not find any difference in age between survivors and non survivors[1]. Acute subdural hematoma in infants is distinct from that occurring in older children or adults because of differences in mechanism, injury thresholds, and the frequency with which the question of nonaccidental injury is encountered. Loh et al[8] in their series of 21 infants with acute subdural hematoma, treated 8 patients (with large subdural hematomas) with craniotomy and evacuation of the blood clot. None of these patients developed chronic subdural hematoma. Thirteen patients with smaller subdural hematomas were treated conservatively. Among these patients, 11 developed chronic subdural hematomas. All patients with chronic subdural hematomas underwent burr hole and external drainage of the subdural hematoma. At follow-up, 13 (62%) had good recovery, 4 (19%) had moderate disability, 3 (14%) had severe disability, and 1 (5%) died. Based on GCS on admission, one (5%) had mild (GCS 13-15), 12 (57%) had moderate (GCS 9-12), and 8 (38%) had severe (GCS 8 or under) head injury. Sixty-three percent (5/8) of those patients undergoing operation for acute subdural hematomas and 62% (8/13) of those patients treated conservatively had good outcomes.

In a study by Wilberger et al the most common mechanism of injury was automobile accidents (53%)

followed by falls (37%)[9]. The worse outcome was recorded in patients involved in motor cycle accidents with a 71% mortality rate and there were no survivors with functional recovery. In our study, majority of patients (85%) had road traffic accidents, out of them 37% of riders and 23% of pedestrians survived. This difference was not statistically significant. Massaro et al[12] found that the most common mechanism of injury was road traffic accidents and fall was related to worst outcome. There was no statistically significant difference in outcome related to sex in our study and other studies have also recorded similar results[9-13].

Outcome has been shown to be related to GCS/Motor score in several studies and expectedly outcome worsens as the motor response progresses from normal to posturing to flaccidity. Wilbenger et al[9] reported 74% mortality in-patients with GCS of 3-5 and 36% mortality in-patients with GCS of 6-8. The average GCS score for their patients who survived was 4.8 and 4.4 among those who died[9] (Table 3). In our study , 28.5% of patients with motor response M2-M3 and 62.5% of patients with M4-5 motor response survived. Mean motor response in patients who survived was 4.1±1.044 and those who expired was 2.4±1.215. Massaro et al[12] reported 68% mortality and 11% functional recovery in acute SDH patients with GCS<8.Similar outcomes in acute subdural hematomas were reported by Servadei et al[13] and Kotwica & Bizezenski[22].

The presence of abnormal pupillary finding in-patients of acute head injury has predictive value. Braakman et al reported 54% mortality in-patients with one reactive pupil, compared with 90% mortality in-patients with both pupils reactive[14]. In a study by Tandon, the mortality was 84.3% when both pupils were non-reactive in comparison to 16.6% when pupils were normal[15]. In our study, 68% of patients with bilaterally reacting pupils and 16.6% of patients with bilaterally nonreacting pupil survived. Nakorchai et al[7] and Sarvadei et al[16] have also reported similar outcomes in their series of traumatic acute subdural hematomas.

The nuclei and tracts involved in oculocephalic and oculovestibular reflexes span much of the brain stem and injury to these deep structures often is associated with severe injury to more superficial structures. Abnormal eye movements, including abnormal oculocephalic and oculovestibular reflexes, are associated with poor outcome[1, 14,17-19]. In patients with absent/impaired dolls eye movement, 70-73% mortality have been reported.[17,19]. In the present series, 90% mortality was observed in those with absent oculocephalic response.

Bowers and Marshall[20] opined that the presence of chest or abdominal injuries requiring surgery had a significant impact on the outcome, i.e. polytraumatized patients with acute SDH exhibited a higher mortality rate. The IDB study however failed to demonstrate any significant correlation[18]. In our study, 30% of patients with major associated injury survived compared to 37% without it.

Yanaka et al[21]and Servadie et al[16] indicated worse outcomes with increasing hematoma thickness and reported favorable outcomes in 56% patients with hematoma thickness 0-10mm and 22% in patients with thickness>20mm. In the present study too, 33% patients with hematoma thickness of 1-5 mm and only 10% patients with hematoma thickness of >10 mm survived.

Thickness of hematoma, associated unilateral cerebral edema and associated parenchyma damage contribute to midline shift. Kotwica and Brezezinski[22] showed 42% favorable outcome and 39% mortality rates with less than 1.5 cm midline shift and 52% mortality when this shift was from 1.5-3.0 cm. In our series there was some correlation between midline shift and outcome i.e. survival of 42%, 32% and 22% in-patients with midline shift of 6-10mm, 11-15mm and 16-20 mm respectively. Servadie et al did not find any significant relationship between outcome and brain swelling factor (difference between midline shift and hematoma thickness)[13]. Wang et al[23] retrospectively studied influence of hematoma thickness and midline shift on survival in a series of 95 patients with acute subdural hematoma. The hematoma thickness ranged from 5.0 to 40.0 mm and midline shift was from 0 to 35.0 mm. Among these patients, 51% died and 49% survived after surgery. 18 patients (19%) showed good or satisfactory results. Kaplan-Meier analysis proved that the survival for patients with hematoma thickness approximately equal to l7 mm and a midline shift 15-mm or whose midline shift exceeded hematoma thickness by 2.2 mm, the survival rate was 50%. Glasgow outcome scale scores were correlated significantly with this parameters.

Basal cisterns effacement is associated with poor favorable outcome (12-22% favorable outcome in-patients with absent basal cisterns) [6,16,21,24,25]. In the present study too, similar trend was observed as 62.5% patients with unilaterally effaced cisterns survived while only 30% patients with bilateral cisternal effacement survived.

The presence of subarachnoid hemorrhage is also shown to be associated with poor outcome in head injury patients recently. Unfavorable outcome was seen in 65-75% of patients with presence of subarachnoid hemorrhage by Servadei et al and was a powerful predictor of bad outcome[16]. Similarly, 22.5% patients with SAH and 46.4% patients without SAH in initial CT scan survived in the present series and this was found to be statistically

significant.

In an autopsy study of head injury, Kristiansen and Tandon[3] concluded that acute subdural haematoma is seldom an isolated lesion. Furthermore, there are patients in whom the extensive associated contusion and laceration may be the dominant feature, SDH being only a relatively unimportant coexistent pathological finding. Concomitant brain injuries are of greater importance to the outcome than the effect of acute subdural hematoma itself. The reported incidence of brain contusions associated with an acute SDH ranges from 7%-82%[9,22]. The rate of favorable outcomes ranged from 32 to 58% for patients with isolated SDH and from 12%-32%for those with associated multiple contusions[22, 26.] Servadei et al[16] reported 57% favorable outcome in patients with only SDH and 37% favorable outcome in-patients with SDH and contusion. Massaro et al[12] reported 62% mortality in pure acute SDH, 52% in satellite acute SDHs (subdural hematomas with ipsilateral/ contiguous contusion) and 63% in patiens with associated acute SDH (associated with other lesion/opposite a distant sites or multiple contusion). Contrary to the published literature considering contusion to be an independent factor, 45% patients with single contusion and 50% patients with multiple contusions survived, whereas only 10% of patients without any contusions survived. This is due to the fact that these were the patients who had diffuse brain swelling and were with poor GCS. Seelig et al[1] did not report significant differences in outcome of patients with and without associated contusion.

A recent study showed that preservation of the subarachnoid spaces on preop CT was an extremely favorable prognostic factor[25]. Conversely when the clotted blood entered a damaged pia/arachnoid (traumatic SAH), cytotoxic substances were released into the extracellular space. These biochemical substances (cytokines, oxygen free radicals, or excitatory aminoacids) may cause enlargement of the parenchymal damage, ischaemia and swelling[26]. The shearing forces responsible for producing acute SDH may also damage the cerebral vasculature and disturb the cerebral autoregulation. Such cerebral vascular damage may compound the ischaemia and swelling produced by traumatic SAH and the mass effect due to SDH and any contusion if present. The resultant increased intracranial tension and brain swelling gives rise to obliteration of cisternal spaces and vascular compromise leading to infarcts in various regions. All above factors are not mutually exclusive and are in a vicious cycle. Servadei et al[16] reported infarcts in 10% of patients and among them 30% of patients had poor outcome. In our study 29% patients had associated infarct on the initial CT scan and among them only 17% survived.

The very high mortality associated with surgery of acute SDH prompted a number of neurosurgeons to delay operating on such patients. Seelig et al reported a dramatic reduction in mortality to 30% with early surgery (within 4 hrs of injury) as compared to 85% with delayed surgery[1]. Stone et al[27] found no significant difference in outcome in those undergoing surgery within 4 hrs of injury (60%mortality) than in those operated between 4-12 hrs postinjury (75%) and similar results were observed in our series too. While the debate regarding the benefits of early surgery (within four hrs) continues, it is generally agreed that the denial of operation during the first 24 hrs or delaying surgery in the hope of improving the outcome has no basis[28]. Early surgery should be performed in patients with more than 5 mm midline shift, without waiting for deterioration in the condition or rise of intracranial pressure. Failure to observe progressive improvement and not evidence of deterioration is an itself an indication for surgery[15, 28].

Believing the subdural clot as the real culprit responsible for the clinical condition of the patient, the standard surgical procedure earlier was to evacuate SDH through burr hole. This not only proved to be futile in majority of cases, it also prevented the surgeon from appreciating the associated brain lesion. Tokutami et al compared different surgical treatment in a series of 120 patients and observed craniotomy to be associated with higher rate of good result (48%) than in burrhole evacuation group wherein 75%mortality rate was observed[29]. In our study, 52 of 61 patients underwent craniotomy with hematoma evacuation and lobectomy according to requirement and we observed 36.5% survival rate. Kotwika & Bizenzenski[22]recorded 23% favorable recovery and 55% mortality while Marshal et al[30] and Wilberger et al[9] recorded 14%/19% favorable recovery and 50%/60% mortality respectively. We observed favorable recovery in 8 patients (13%), 13 patients (21.3%) had unfavorable recovery (severe disability and vegetative state) and in 40 patients (65.5%) expired. Huang et al[31] compared the therapeutic effect and indication between standard large trauma craniotomy and routine craniotomy in acute subdural hematoma patients. There were 97 patients in the standard large trauma craniotomy group and 110 patients in the routine craniotomy group. Fifteen patients (15.6%) died in the standard large trauma craniotomy group and 30 (27.7%) in the routine craniotomy group. The rate of complication was lower in the standard large trauma craniotomy group, but no obvious difference in long-term therapeutic effect was found between the two groups. They concluded that standard large trauma craniotomy can attenuate brain hernia and the mortality of the patients with acute subdural hematoma.

CONCLUSIONS

Adults in their 3rd and 4th decades of life are the commonest victims of acute subdural hematoma and there is a significant difference in mortality between patients below and above 40 years of age. Road traffic accident is the commonest cause of acute subdural hematoma followed by fall from height. Initial motor response, pupillary abnormality and presence or absence of oculocephalic response correlated well with outcome while presence or absence of associated injuries does not influence outcome significantly. We observed that thickness of subdural hematoma and midline shift though affected the outcome but the difference was not significant. Majority of patients with acute SDH have associated focal (contusion/ laceration/infarction) or global (diffuse axonal injury, subarachnoid hemorrhage) involvement of the brain or both of these. Presence or absence of subarchnoid correlated well with outcome. Presence of contusion altered the mode of management and this group had a better outcome than in those where there was no contusion and not only diffuse brain swelling or diffuse axonal injury was present. In addition to SDH and contusion; presence of infarct on initial CT scan was associated with a poorer outcome. Though timing of operation does not affect significantly the overall outcome in-patients of acute subdural hematoma; but patients do deteriorate even after admission and this does affect the outcome. Postoperative infections (meningitis, chest infection, and septicemia) are also detrimental in the ultimate outcome.

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