Original Article

Expanding traumatic intracerebral contusion/hematoma

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Background and Aims: Delayed traumatic hematomas and expansion of already detected hematomas are not uncommon. Only few studies are available on risk factors of expanding hematomas. A prospective study was aimed to find out risk factors associated with such traumatic lesions. **Materials and Methods:** Present study is based on 262 cases of intracerebral hematomas / contusions out of which 43 (16.4%) hematomas expanded in size. computerized tomography (CT) scan was done in all the patients at the time of admission and within 24 hours of injury. Repeat CT scan was done within 24 hours, 4 days and 7 days. Midline shift if any, prothrombin time, activated partial thromboplastin time, bleeding time, clotting time and platelet counts, Glasgow coma scale at admission and discharge and Glasgow outcome score at 6 months follow up were recorded. **Results:** Twenty six percent, 11.3 and 0% patients developed expanding hematoma in Glasgow Coma scale (GCS) of 8 and below, 9-12 and 13-15 respectively. The chances of expanding hematomas were higher in patients with other associated hematomas (17.4%) as compared to isolated hematoma (4.8%) (Fisher's exact results *P*=0.216). All the cases of expanding hematoma had some degree of midline shift and considerably higher proportion had presence of coagulopathy. The results of logistic regression analysis showed GCS, midline shift and coagulopathy as significant predictors for the expanding hematoma. Thirty nine patients (90.7%) of the total expanding hematomas developed within 24 hours of injury. **Conclusions:** Enlargement of intracerebral hematomas is quite common and majority of them expand early after the injury. These lesions were common in patients with poor GCS, associated hematomas, associated coagulopathy and midline shift.

Key words: Delayed Intracerebral hematoma, head injury, secondary insult

Introduction

Post traumatic intracranial hematoma (ICH) can expand or develop late after head injury.[1-5] Identification of risk factors, early diagnosis and proper treatment of such lesion are important for improving the prognosis of these patients. There are reports on delayed ICH, however very little literature is available on expansion of already existing intracerebral hematoma.[6,7] We are reporting a prospective study of expansion of intracerebral hematoma.

Materials and Methods

A prospective study was carried out in neurosurgery unit of our institute from Jan.2002 to Dec 2003. Total of 326 post traumatic intracerebral contusions (n=294) / hematomas (n=32) were managed, out of these 47 patients were operated after first computerized tomography (CT) scan and were excluded from the study. 17 patients who died within 24 hours of admission (repeat scan could not be done in these cases) were also excluded from the study. Patients reported after 24 hours of injury were also excluded. The distinction between a contusion and an intracerebral hematoma was made by the proportion of blood in the lesion, if two thirds or more of the lesion was hematoma then it was considered as intracerebral hematoma. Increase in volume of 12.5 cc was considered as expansion of hematoma

A detailed history of injury, date and time of accident, age, sex, unconsciousness, ear nose throat bleeding, seizures, vomiting, headache, etc. was asked. A thorough neurological, systemic and general physical examination was done. Glasgow Coma scale (GCS) was obtained after resuscitation. CT scan was done in all the patients at the time of admission [Figures 1, 3 and 5]. Repeat CT scan was done in all the patients of intracerebral contusions / hematomas within 24 hours of admission [Figures 2, 4, and 6]. Another CT scan was done on 4th and 7th day of the admission. 38 times CT scan was done outside the mentioned period due to clinical worsening out of these 29 were in none expanding and 9 in expanding group. Size of hematoma and midline shift was recorded. Hematoma volume was calculated using formula ABC/2, where A was the greatest diameter of hemorrhage in single CT slice, B was the diameter 90^{o} to A and C is number of CT slice with hemorrhage multiplied by the slice thickness. Coagulation tests

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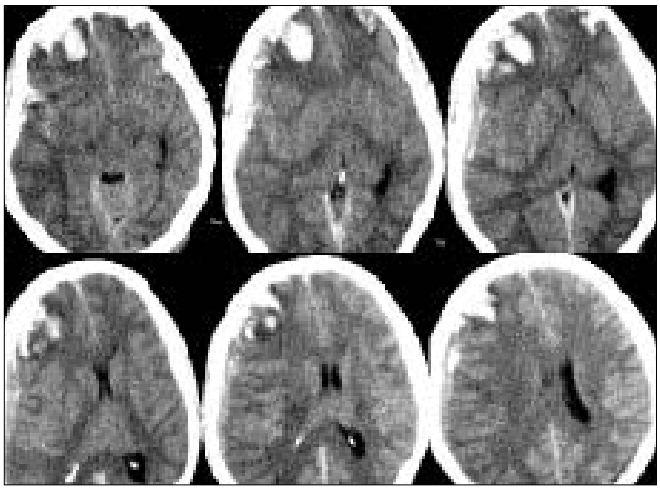

Figure 1: First CT scan of case 1 showing bifrontal ICH more on right side and an acute SDH in right fronto-temporo-parietal region

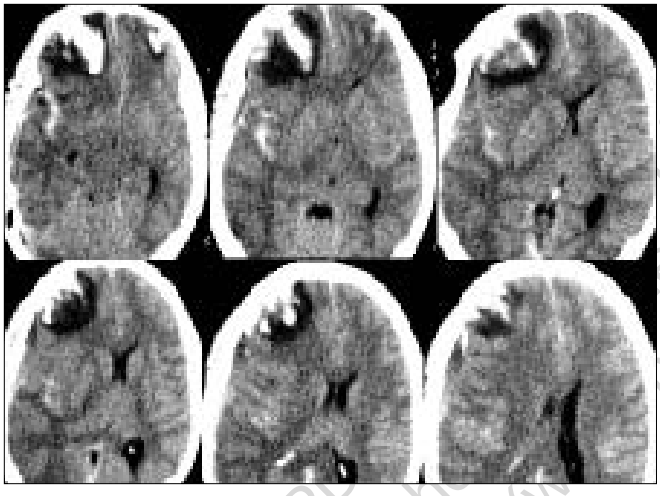

Figure 2: Second CT scan of case 1 showing no significant increase in ICH

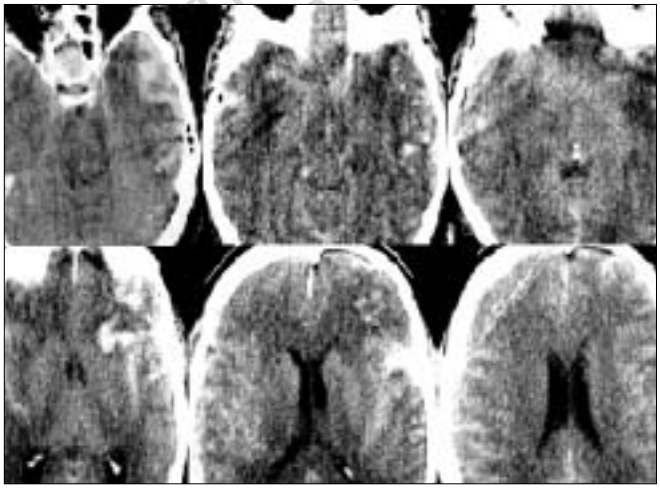

Figure 3: First CT scan of case 2 showing bilateral SAH involving fronto-temporo-parietal region and bilateral temporal contusions, more on left side

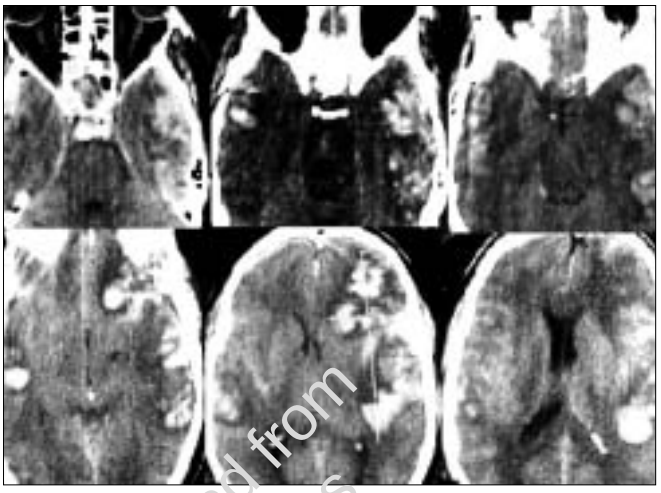

Figure 4: Second CT scan of case 2 showing significant increase in temporal contusions. This patient was operated after 2nd CT scan

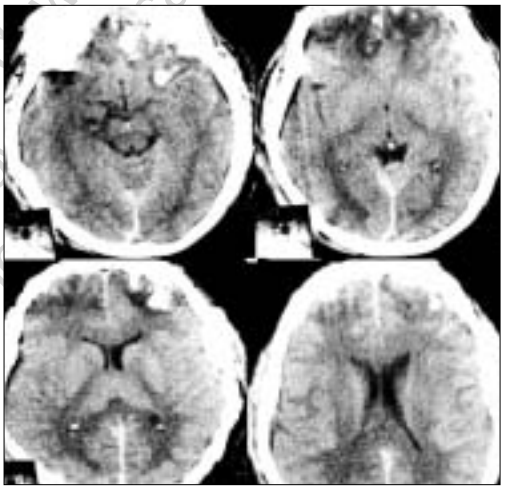

Figure 5: First CT scan of case 3 showing an acute SDH in left fronto-temporo-parietal region and bifrontal contusions more on left side

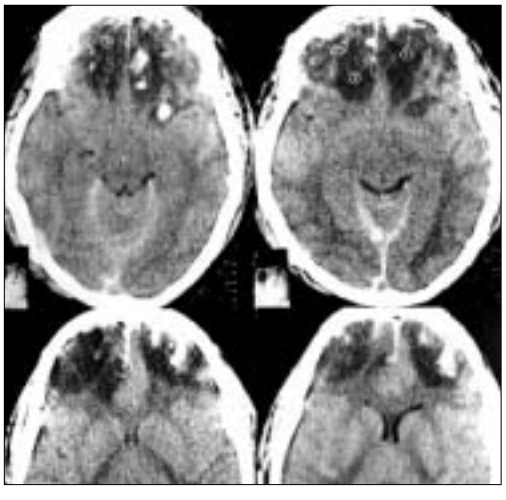

Figure 6: Second CT scan of case 3 showing significant increase in frontal contusions. This patient was treated conservatively, patient was conscious after second CT

like prothrombin time (PT), activated partial thromboplastin time (APTT), bleeding time, clotting time and platelets counts were done in all the patients. Patients were diagnosed to have coagulopathy if PT and APTT were more than one and half times of the control and also when bleeding time or clotting time was more than upper limit and platelet count was less than lower limit of normal. All the patients with GCS of 8 and below were managed in ICU. All other relevant biochemical investigations were performed. Routine management of head injury patients was done. Results of treatment were recorded. Glasgow outcome scale was recorded at 6 months follow up.

The data for this study was analyzed using univariate, bivariate statistical analysis using fisher's exact test, Chi square test and a logistic regression analysis was also applied considering all these factors for the expanding hematoma.

Results

Out of 262 patients, 43 ICH expanded in size. The analysis of 262 prospective cases of post traumatic intracerebral hematomas / contusions which was carried out in Neurosurgery unit of our institution is presented in this section. Majority (66.5%) of the studied cases were in age below 40 years with a median age of 33 years. Age ranged from 2 to 80 years. Overall the mean age was 35.16 (± 17.15) years. Male female ratio was 2.05 but the incidence of expanding hematoma in both the sex was equal having insignificant difference ($P>0.05$). Initial ICH volume ranged from 4-18 cubic cm [cc] with a mean value of 12.06 (± 3.70) cc and this was increased from 12.5 to 58 cc with a mean value of 33.13 (± 10.59) cc and this increase in ICH volume was statistically significant. ($P<0.001$) 34 patients deteriorated in GCS from 1-3. The deterioration in GCS was from 1-3 in operated group as compared to 1-2 in conservatively managed groups. Increase in midline shift in the operated groups was 2-5 mm while it was 1-3 mm in conservatively managed groups.

As depicted in Table 1, out of 262 cases 104 (39.7%) cases were observed with GCS ≤ 8, 141 (53.8%) with GCS 9-12 and 17 (6.5%) with GCS 13-15 and their corresponding findings of expanding hematoma were 26.0, 11.3 and 0.0% respectively. The incidence of expanding hematoma in cases with lower GCS was significantly higher. A linear trend was observed in GCS and expanding hematoma i.e., the cases with lower GCS have higher chances of expanding hematoma. ($P<0.001$) The impact of GCS shows its direct correlation with the development of hematomas.

There were 21(8.0%) patients with single ICH / contusion as compared to 241 (92.0%) with multiple lesions like acute subdural hematoma (SDH) in 134 (55.6%), other ICH in 51 (21.2%), extra-dural hematoma in 9 (3.7%), SDH and other ICH in 47 (19.5%) patients. 1 (4.8%) out of 21 isolated ICH [without any other hematomas] developed expanding hematomas as compared to 42 patients (17.4%) with other associated hematomas [n=241] but this difference was statistically not significant (fisher exact test results $P=0.216$). Twenty four patients (17.9%) out of 134 ICH associated with acute SDH, 10 (19.6%) patients out of 51 multiple ICH and 8 (17.0%) out of 47 both SDH and ICH developed expanding hematomas. This difference in incidence of expanding hematoma among various associated hematomas is not statistically significant ($\chi^2 = 0.12$; $P>0.05$).

The study of midline shift showed that there were 249 ICH / contusions with midline shift while in 13 cases there was no midline shift observed, out of the cases with midline shift 17.3% (n=43) developed expanding hematoma while none of the patients without midline shift developed expanding hematomas.

This difference was also statistically insignificant (fisher exact test results $P=0.135$) while this was one of the significant factor in the logistic regression analysis. There were 41, 87, 108 and 13 patients with midline shift of less than 3 mm, 3 to less than 5 mm, 5 to less than 10 mm and more than 10 mm respectively, out of these 4 (9.7%), 14 (16.1%), 21 (19.4%) and 4 (30.8%) developed expanding hematomas respectively. ($\chi^2 = 3.72$; $P>0.05$ at 3 df)

Coagulopathy was directly related to expanding hematoma. Coagulopathy was seen in 39 patients of ICH. Out of these 41.0 percent developed expanding hematoma while the incidence of expanding hematoma was only 12.1% where the coagulopathy was absent and statistically this was highly significant ($P<0.001$).

Surgeries were required in 37 cases out of 43 expanding hematomas. 19 (51.3%) patients in operative group died, while there was no mortality in conservatively treated group. However they had better grade with small ICH. 4 patients (one operative and 3 conservative group) could not be followed up out of 24 survivors. All three patients of conservative group who could be followed up had good recovery while 5, 5, 4 and 3 patients of operated group had good recovery, mild, moderate and severe disability (35% moderate to severe disability) at 6 months follow up.

The location of the ICH / contusions were 106 in frontal region, 134 in temporal, 12 in parietal region and 10 in more than one region, out of these 18 (17.0%), 23 (17.2%),1 (8.3%),1 (10.0%) in frontal, temporal, parietal and more than one region developed expanding hematomas respectively. 39 (90.7%) expanding hematomas developed within 24 hours of injury while the rest 2(4.6%) patients each developed ICH within 4 days and 7 days.

The result of logistic regression analysis showed positive value for the Cox and Snell R square ($R^2 = 0.216$) and found that GCS ($P<0.001$), midline shift ($P<0.001$) and coagulopathy ($P<0.003$) were highly significant predictors for the expanding hematoma.

Discussion

We used the term expanding traumatic intracerebral contusion / hematoma which expand after the initial detection on first CT scan while in cases of delayed hematomas, initial CT scan was normal. The incidence of expanding hematoma was 16.4% in our

series while it was 42.3% in Oertel series[6] and (68.2%) in Lobato[7] series. The risk factors of delayed hematomas like coagulopathy, presence of preexisting intracerebral contusion / hematoma, traumatic vessel injury, subdural hematoma and development of hematoma after treatment of raised intracranial pressure are described.[1-12] Similar mechanism of development of expansion of hematoma may exist.

Delayed hematomas are known to be associated with coagulation disorder. 47.6% patients of traumatic sub-arachnoid hemorrhage with coagulopathy at admission developed delayed hematoma.[1] In our series also 41% of ICH with coagulopathy developed expansion of hematoma. Delayed and recurrent intracranial hematomas were found to be related to disseminated intravascular clotting and fibrinolysis.[3] These were also reported in patients taking oral anticoagulants.[2] Delayed hematomas were commonly associated with SDH and underlying contusion / hematoma.[13] Expanding hematomas were also more common in ICH associated with other hematomas as compared to isolated ICH in our series although the difference was insignificant. It could be due to venous infarct developing in the contused brain region. Injured draining veins responsible for the acute SDH, could also be responsible for delayed / expansion of hematoma.

Delayed intracerebral hematoma can also develop in patients with raised intracranial pressure. Treatment of such patients by either surgery or medicine relieves temponade effect which can result in bleeding from injured vessel in area of contused brain.[10,12,14] Expanding hematomas were more common in frontal and temporal region. Vascular injury like angionecrosis, intravascular thrombosis could be responsible for expanding hematoma.[5,15] The chances of expanding hematomas were higher in patients with other associated hematomas (17.4%) as compared to isolated hematoma (4.8%) (Fisher's exact results; $P=0.216$) in our series similar observations were made in other series.[16]

Intracerebral haematomas with midline shift were also associated with higher incidence of expansion of hematoma as compared to haematomas without midline shift in our series but the differences were insignificant in fisher's exact results due to small number however it was one of the predictor observed significant in the logistic regression analysis.

Expanding hematomas developed more commonly in patients in poor GCS and with other associated hematomas in our series; this was also observed in other series.[16,17] These were also more commonly associated with midline shift as compared to patients without midline shift. Age was not found to be risk factor in this study and in Stein *et al* series,[16] while it was found as a risk factor in other series.[6] They also found sex as a risk factor which was also not seen in our series or in any other series.

Coagulopathy was also found to be significant predictors for the expanding hematoma in our series; this was also observed in other series.[6,16,17] Thirty nine patients (90.7%) of the total expanding hematomas developed within 24 hours of injury in our study, similarly majority (80.7%) of delayed hematomas were noted within 48 hours after injury[18] and within 72 hours of injury.[8] Okada[19] *et al* observed that the times, when the formations of traumatic intracerebral hematomas were judged as completed showed two peaks: within 6 hours after the trauma and 12 to 24 hours after the trauma. Oertel *et al*[6] also observed progressive hemorrhage in 48.6% of patients who underwent scanning within 2 hours of injury.

Expanding hematoma can be diagnosed by repeat CT scan. Lobato *et al*[7] advised serial CT scanning at 2-4, 12, 24, 48 and 72 hours after injury. Near infrared spectroscopy can also detect such lesions much earlier.[20] Prevention of these hematomas can be done by correction of risk factors like coagulopathy, treatment of raised intracranial pressure etc. Early diagnosis should be done by timely CT scan when patient is at risk like patient with cerebral contusion / hematoma, associated with other hematomas, raised ICP with midline shift, coagulopathy and patients in poor GCS.

Treatment of such lesions consists of surgical removal when patient is in altered sensorium, with large clot and with midline shift. Small lesions in conscious patient can be managed by conservative method. Prognosis is poor with 50- 60% mortality and increased morbidity in majority of patients.

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