ORIGINAL ARTICLE

FREQUENCY OF CONSERVATIVELY MANAGED TRAUMATIC ACUTE SUBDURAL HAEMATOMA CHANGING INTO CHRONIC SUBDURAL HAEMATOMA

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Background: Traumatic brain injury represents a significant cause of mortality and permanent disability in the adult population. Acute subdural haematoma is one of the conditions most strongly associated with severe brain injury. Knowledge on the natural history of the illness and the outcome of patients conservatively managed may help the neurosurgeon in the decision-making process. **Methods:** We prospectively analysed 27 patients with age ranges 15–90 years, in whom a CT scan diagnosis of acute subdural haematoma was made, and in whom craniotomy for evacuation was not initially performed, to the neurosurgery department of Ayub Teaching Hospital Abbottabad (2008–2011). Patients with deranged bleeding profile, anticoagulant therapy, chronic liver disease, any other associated intracranial abnormalities, such as cerebral contusions, as shown on CT, were excluded from this study. All patients were followed by serial CT scans, and a neurological assessment was done. **Results:** There were 18 male and 9 female patients, Cerebral atrophy was present in over half of the sample. In 22 of our patients, the acute subdural haematoma resolved spontaneously, without evidence of damage to the underlying brain, as shown by CT or neurological findings. Four patients subsequently required burr hole drainage for chronic subdural haematoma. In each of these patients, haematoma thickness was greater than 10 mm. The mean delay between injury and operation in this group was 15–21 days. Among these patients 1 patient required craniotomy for haematoma removal due to neurological deterioration. **Conclusion:** Certain conscious patients with small acute subdural haematomas, without mass effect on CT, may be safely managed conservatively, but due to high risk of these acute subdural haematoma changing into chronic subdural haematoma these patients should be reinvestigated in case of neurological deterioration.

Keywords: Acute subdural haematoma, conservative treatment, traumatic brain injury

INTRODUCTION

Traumatic brain injury represents a significant cause of mortality and permanent disability in the adult population. Significant health care costs involve hospitalizations, rehabilitation, and loss of independence. Acute subdural haematoma (SDH) is one of the conditions most strongly associated with severe brain injury with an incidence of 12–29%. The mean age reported in the literature ranges from 31–47 years, mostly involving males.[1–4]

Subdural haematoma occurs due to haemorrhage into the dura-arachnoid interface. Small, acute SDH resolves spontaneously or progress to chronic SDH over a period of three weeks. Mortality and outcome in patients with acute SDHs using the Glasgow Outcome Scale (GOS)[5] and the Glasgow Coma Scale (GCS)[6] have not changed significantly over the last decades. Mortality ranges from 50–90%,[4,7–9] but increases to 90–100% in patients receiving anticoagulatants.[8,10] Associated injuries have been reported to occur in 47–57% of patients, accounting for a higher morbidity and mortality.[1,11] Management decisions should take into account guidelines and recommendations for all the lesions involved. Mortality has been demonstrated to drop to 30% if adequate surgical intervention for evacuation of haematoma is performed within four hours of the trauma.[2,8] Other authors have not found a correlation between the timing of operative intervention and the outcome of the patient.[4,12] The time from the onset of neurological deterioration to surgery is significantly related to the outcome.[1,13]

Despite available guidelines for surgical[1] or conservative[7] management of acute SDHs, management of the patients has to be individualised. The knowledge of natural course of illness and outcome of patients conservatively managed may help tailor the strategies and algorithms in those cases where management has not yet been clearly defined. Dent, *et al*[2] reviewed all patients with an acute SDH admitted over a 6-year period to a single trauma centre and found that 61% of the patients received conservative management.

We evaluated the clinical course and outcome of a group of patients with traumatic supratentorial acute SDHs conservatively treated, and their frequency to change into chronic subdural haematoma.

MATERIAL AND METHODS

This descriptive study was carried out in Department of Neurosurgery, Ayub Medical College, Abbottabad, over a period of two and half years from January 2008 to June 2010. This study evaluated patients who arrived to our emergency room with a diagnosis of acute traumatic supratentorial SDH. Head computed tomographic (CT) scans were used to evaluate all the patients. Patients with a midline shift greater than 10 mm in the head CT scan or with neurological deterioration due to the haematoma were excluded from the study. Patients younger than 15 years were not included. Patients with bleeding disorders, deranged liver functions, patients on anti-coagulants, and with any other associated intracranial abnormalities, such as cerebral contusions, showing on CT, were excluded from this study. Adult patients between the ages of 15 and 90 years conservatively treated were evaluated. Informed written consent was taken. Radiological assessment was provided using the maximum thickness of the haematoma and the amount of midline shift. Those with GCS score >13 were observed for neurological changes and a follow-up head CT scan was done immediately in case of neurological deterioration. Demographic data, co-morbid conditions, associated extracranial injuries, GCS score after initial resuscitation, radiological findings (thickness of the haematoma and midline shift on the head CT scan), course of illness were documented. Follow-up was done at the neurosurgery clinic.

RESULTS

A total of 27 patients with traumatic acute subdural haematomas were included in this study, out of which 21 (77.7%) were men and 6 (22.2%) were women. Age ranged 15–90 years, (Mean=51.4 years). Cerebral atrophy was present in 14 (51.8%). Main cause of trauma were road traffic accidents 23 (85.1%), fall from height 3 (11.1%), and assaults was 1 (3.7%). Fracture of the parietal and temporal bone were seen in 5(18.5%) cases. All 27 patients were conservatively treated with steroids, mannitol, and hyperventilation. Mean follow-up was for 6.4 weeks

In 22 (81.4%) of our patients, the acute SDH resolved spontaneously, without evidence of damage to the underlying brain, as shown by CT or neurological findings. Four (14.8%) patients subsequently required burr-hole drainage for a hypodense liquid chronic subdural haematoma (Table-1). In each of these patients, haematoma thickness was greater than 10 mm. The delay between injury and operation in this group was 15–21 days. We saw no mortality in our study and none of the patients in our study developed seizures. Among these patients, 1 patient (3.7%) required surgical evacuation of haematoma through craniotomy due to neurological deterioration.

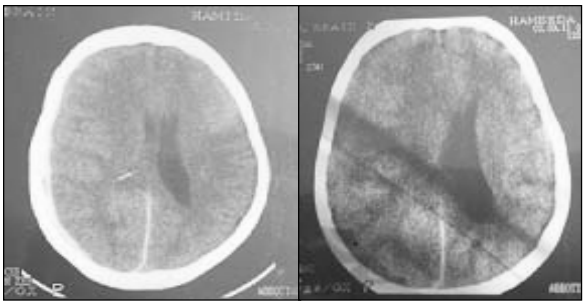

Figure-1: CT Scan of a patient showing hyperdense shadow on right side which changes into hypodense shadow with significant midline shift suggesting acute SDH changing into chronic SDH.

Table-1: Outcome of conservatively managed acute subdural haematomas (n=27)

Acute subdural haematoma	Cases	%
Spontaneously resolved	22	81.4
Craniotomy in case of neurological deterioration	1	3.7
Changed into chronic subdural haematoma	4	14.8

DISCUSSION

SDH are divided into three types depending on the presentation and chronological age: Acute SDH presentation with 72 hours, Subacute SDH presentation with 3 days to 3 weeks, and Chronic SDH presentation with 3 weeks to months. The aetiological factors for the origin and development of chronic SDH has been debated since Virchow reported '*pachymeningitis haemorrhagica interna*' in 1857.[14] In order to explain the mechanism the osmotic gradient theory was proposed by Gardner.[15] However, Weir[16,17] proved that there is no significant difference in osmotic pressure between haematoma fluid, venous blood and cerebrospinal fluid and in the oncotic pressure between haematoma fluid and venous blood.

Recurrent haemorrhage from haematoma capsule was proposed. The vessels of haematoma capsule were reported to have marked proliferative potential and are fragile.[18–20] However, recurrent haemorrhage from haematoma capsule theory does not explain how haematoma fluid accumulates during the early stage before the formation of vascular outer membrane. In addition simple drainage of haematoma leaving the entire outer membrane in situ is curative in most cases.

A recent study of the coagulation and fibrinolytic profile of evacuated clot has indicated that there is excessive activation of clotting system, thrombin generation and increased fibrinolytic activity occurring in the haematoma fluid as compared to intravascular blood. However, the exact reason of this localised manifestation of altered clotting fibrinolytic system within the subdural space in the absence of generalised manifestations remains an enigma. This could be a possible aetiological factor for origin and

development of chronic SDH as follows. Damage to cerebral endothelia, blood cells and brain tissue releases abundant tissue thromboplastin in subdural space. The clotting systems are excessively activated resulting in marked consumption of clotting proteins. An excessive activation of the intrinsic clotting system leads to excessive activation of intrinsic fibrinolytic systems. The cross-linked fibrin polymer is degraded to form fibrinogen degradation products (FDP) and defective clot formation causes recurrent-haemorrhage in the damaged area. As the process is repeated the dura reacts non-specifically to form the vascularised outer membrane gradually. As the outer membrane proliferates, the extrinsic fibrinolytic system is activated and a self-perpetuating vicious cycle is accelerated. The burr hole drainage removes these self-perpetuating factors and restores normal haemostatic mechanism in the subdural space, leading to a cure.[21]

The chronological evaluation of pathological events correlated with CT scan is well-documented.[23,24] The density of subdural haematoma decreases with increasing age of haematoma. As the process of lysis of red blood cells (RBC) and liquefaction of clot progresses over weeks the CT appearance changes through hyperdense (acute), isodense (subacute) and hypodense (chronic) extra cerebral collection.

Approximately 14.8% of patients with conservatively treated acute subdural haematomas changed into chronic subdural haematomas. Clinically our patients deteriorated gradually in approximately three weeks, due to an abnormally rapid increase in the size of the clot. The CT scan appearance changed from hyperdense to predominantly hypodense in 15–21 days. The earliest change in appearance from hyper to isodense documented in literature is two weeks. The patients had no coagulopathy. This rapid increase was not due to haemorrhage in the clot, as a fresh haemorrhage would have been hyperdense. The patient were not anaemic (haemoglobin: 14 gm%) to render the clot hyperdense due to reduced haemoglobin content. Thus it appears that in our patients the fibrinolytic activity has acted abnormally along with the association of brain atrophy, to liquefy the clot. The burr-hole drainage of liquid 'crank case oil' appearance haematoma verified the fact. Furthermore the haematomas had the typical peripheral membrane encountered in chronic SDH.

Our study shows that 14.8% of conservatively treated acute SDH changing into chronic subdural haematoma, especially if brain atrophy is present on CT scan in elderly patients and specifically patients with large acute subdural haematoma, i.e., thickness up to 10 mm associated with GCS >13. So there should be emphasis on the need for reinvestigating a patient who had a subdural haematoma and has neurological deterioration under observation.

CONCLUSION

Conservative management in patients with traumatic acute subdural haematoma can be a valuable alternative in some cases but due to high incidence of these acute subdural haematoma changes into chronic subdural haematoma, proper guideline should be set to re-evaluate such patients in case of neurological deterioration and also patient's attendants should be counselled regarding the possibility of developing chronic subdural haematomas in such patients.

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