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Original Article

Efficacy of Decompressive Craniectomy in Acute Subdural Hematoma in Head Injury Patients, Madurai Medical College, Madurai

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Abstract

Aims and Objectives: The aims and objectives are as follows: (1) To study the effect of decompressive craniectomy in head injury patient with subdural hematoma (SDH), (2) to compare the outcome of non-operative patients, and (3) to identify the factors contributing the outcome of decompressive craniectomy.

Materials and Methods: This was a retrospective study conducted between November 2015 and October 2016. The patients in trauma head injury ward, Government Rajaji Hospital, Madurai Medical College, Madurai, Tamil Nadu, are grouped as decompressive craniotomy surgery done and conservatively treated with acute SDH. Data regarding mode of accident, GCS - Glasgow coma scale, computed tomography finding, and outcome were collected. Statistical analysis was used to identify factors associated with favorable outcome of the patients.

Results: Statistical analysis were done to identify factors associated with mortality, morbidity, and favorable outcome of the patients, by categorizing the patients with GCS Mild - 13 -15 ,moderate - 9 -12, and severe below 8 with traumatic brain injury. For the patients with GCS moderate 9-12 score better outcome occurs if decompressive craniectomy done. Total 527 patients in which 139 patients were operated.

Conclusion: Age, severity of head injury, neurological status, and timing of surgery are the main factors influencing outcomes. After moderate head injury with acute SDH, surgery with decompressive craniectomy is the better outcome. Mild head injury can be managed conservatively with continuous neuro observation. In severe head injury, the results are poor.

Key words: Acute subdural hematoma, Decompressive craniectomy, Glasgow Coma Scale, Traumatic brain injury

INTRODUCTION

Head injury is one of the important public health problems today. The incidence of head injuries is steadily increasing all over the world, and developing country has the highest incidence in the world of head injuries due to road traffic accidents per 1000 vehicles or deaths per 1000 accidents.[1] The care of head-injured patients forms an important part of neurosurgical management in all countries. The modernization of industries as well as modes of transport have increased the incidence and the severity of injuries.[3] The management of severe head injury is a major challenge to neurosurgeons as the consequent mortality and morbidity is very high. There is a need for an extensive multidimensional effort to improve the prognosis of head-injured patients and provide them a better quality of life.[2]

Acute sub dural hematoma is a hematoma accumulating between the inner layer of the dura matter and the Arachnoid matter, to become clinically symptomatic within 24 - 72 hours. They are usually located over the cerebral convexities conforming to the convex brain surface.

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MATERIALS AND METHODS

This retrospective study was conducted in head injury ward, Government Rajaji Hospital trauma center, Madurai Medical College, Madurai, Tamil Nadu, from November 2015 to October 2016,

- Study done for the patients with ASDH admitted in the head injury ward, Government Rajaji Hospital, Madurai Medical College, Madurai. The head injury patients with or with out poly trauma, irrespective of mode of injury and with radiological findings are selected for study.
- Who are the subjects believed to fulfill all eligibility criteria, and exclusion criteria are participated in the study for conservative management or decompressive craniectomy
- The patients grouped as (1) decompressive craniectomy done and (2) conservatively treated
- Data regarding mode of accident, time of injury, GCS, clinical status of the patient, laboratory investigations, computed tomography (CT) finding, and outcome were collected by GCS outcome score
- Statistical analyses were done to identify factors associated with mortality, morbidity, and favorable outcome of the patients, by categorizing the patients with GCS Mild - 13 -15,moderate - 9 -12, and severe below 8 with traumatic brain injury. For the patients with GCS moderate 9-12 score better outcome occurs if decompressive craniectomy done. Total 527 patients in which 139 patients were operated.
- Patient's follow-up by 1 month, 3 months, 6 months, and 1 year
- In trauma patient with acute head injury with ASDH, inclusion and exclusion criteria are as follows.

Inclusion Criteria

The following criteria were included in the study:

- In trauma patient with acute head injury with ASDH
- No age restriction
- No sex restriction
- No time restriction.

Exclusion Criteria

The following criteria were excluded from the study:

- Chronic SDH
- Extradural hematoma
- Ventricular hemorrhage
- Intracerebral hemorrhage
- Fracture hematoma.

Based on Data

- Basic patient data's name, age, sex
- Vitals, pupils
- Mode of injury
- Time interval
- Glasgow Coma Scale
- Patient with associated injuries
- Laboratory investigations
- CT scan findings
- Time of surgery
- Glasgow Outcome Score (extended)
- Karnofsky Performance Status Scale
- Modified Rankin Scale.

There is about the incidence of acute SDH (29%) as the primary lesion in patients admitted with head injury. Acute SDH more often occurs in the second to sixth decades (mean age 31–40 years) Men are 4 times more likely to be affected than women.[4]

Pathogenesis

Acute SDH resulting from one of the three common causes, namely: (1) Rupture of bridging veins, (2) cerebral contusion, and (3) rupture of small cortical arteries. They termed the bleeding from torn bridging veins and rupture of small cortical arteries as "pure SDHs" as they occurred without any gross (focal or diffuse) damage to the brain itself.[5] They found that the volumes of arterial and venous acute SDH and their relative areas in the horizontal planes were similar irrespective of the causal mechanism. The hematoma thickness and midline shift were higher in arterial SDH. On the other hand, in venous SDH, the difference between the midline shift and the hematoma thickness was lower than in arterial SDH (i.e., in venous SDH, a smaller acute SDH was associated with a greater midline shift) indicating a tendency toward more pronounced midline shift in venous, rather than arterial SDH of similar volumes.

The venous SDH due to bridging vein ruptures was generally located in the central frontoparietal and parasagittal region and had a comparatively smaller length and thickness than the arterial SDH which were more often located in the temporoparietal region.

Poor outcome following acute SDH may also be related to the ischemic damage occurring in the hemisphere underlying the hematoma due to raised intracranial pressure producing impaired cerebral perfusion. Removal of an acute SDH often results in reversal of global ischemia. Decompressive craniectomy is the one of the neurosurgical methods in which part of skull (free bone flap) is removed. Dura opened and the hematoma evacuation done and free bone flap is not replaced. The aim of decompression is to reduce the increased intracranial pressure and prevent coning.

In younger group (18–40 years), 80% were caused by motor vehicle accidents and only 12% were caused by assault. Whereas, acute SDH in the older groups (>65 years), 26% due to fall and only 8% due to assault.[6]

In comatose patients, motor vehicle accidents are responsible for acute SDH in 75% of patients because these are often high-velocity accidents with associated diffuse axonal injury.[7]

Associated intracranial injuries occur in more than 50% of patients with acute SDH and have a significant prognostic implication. Associated lesions occur in 37% of patients presenting with Glasgow Coma Scale (GCS) scores between 13 and 15 and in 45% of patients with GCS scores <8. In patients with acute SDH, contusion and fractures are the most frequent injuries encountered; associated subarachnoid hemorrhage has been seen in 25% of patients with SDH and epidural hematomas in 18% of patients. Extracranial injuries are seen in 48% of patients including facial fractures, limb fractures, and thoracic and abdominal trauma. Around 40% of patients with other associated lesions have a contrecoup injury. Bilateral acute SDH occurs in 13%.

Clinical Presentation

The clinical presentation is non-specific and occurs due to mass effect produced by the acute SDH as well as associated parenchymatous injury. It depends on the severity of the primary injury, the associated parenchymal injuries, and the rapidity of accumulation of the acute SDH. The patients may remain unconscious throughout or may vary in sensorium from being totally unconscious to being lucid to unconscious or may remain lucid throughout. About 40% of patients are semiconscious at the time of their primary injury and remain comatose for prolonged periods. 33% of their patients with lucid interval neurologically deteriorated. Pupillary asymmetry ipsilateral to the side of hematoma with contralateral hemiparesis may be due to transtentorial herniation. However, false localizing pupillary dilatation contralateral to the lesion may occur due to direct optic nerve, oculomotor nerve, or brain stem injury on that side. Ipsilateral hemiparesis may be due to associated brain injury or due to Kernohan's notching produced by compression of the contralateral cerebral peduncle against the tentorial edge. The incidence of associated seizures has present about 18%. Posterior fossa acute SDH is rare and occurs in 3% of patients who underwent surgery within 24 h of injury. Occipital trauma and associated occipital fractures may be responsible. Posterior fossa acute SDH occurs due to tearing of bridging veins, laceration of the tentorium, contusion of the cerebellum, or injury to venous sinuses. Cerebellar signs, neck stiffness, and pain or symptoms of raised intracranial pressure due to the size of the lesion or the development of hydrocephalus may be the presenting features. Despite urgent surgical evacuation, the mortality was about 75%.

Diagnosis: CT Scan Finding

On CT scans, an acute SDH appears as crescentic, hyperdense collections that lie between the arachnoid and the inner meningeal layer of the Dura that conforms to and often exert a mass effect on the surface of the brain. It extends across sutural lines but does not cross the falx or the tentorium. An acute SDH may occasionally be biconvex due to adhesions between the brain and the dura mater or when it is thick. The exact thickness of the crescentic SDH should be measured by taking the CT images with a wide window to distinguish the hyperdense clot from bone. Early CT (within 3 h from injury) underestimates the size of the associated parenchymal contusions and the consequent edema. Patients who show subarachnoid hemorrhage on early CT are those at highest risk for evolving contusions. The worst outcomes previously associated with acute SDH may, in many cases, be due to the concomitant presence of contusions in multiple areas of the brain and consequent development of edema. Thus, the use of sequential CT scan should be included in the routine management of head-injured patients. In the younger population, an associated swollen hypointense, ipsilateral hemisphere indicates a very poor prognosis.[8] In patients with acute anemia and hemodilution (during resuscitation from multiple injuries), the acute SDH may appear as isodense to hypodense on CT.[9] A subacute SDH may also be isodense to the brain.

Surgical Management

The aim of surgery is to evacuate the hematoma and any associated underlying lesions to relieve the mass effect and improve the focal neurological deficits. However, if the patient has no brainstem reflexes and is hypotonic with no motor response, surgery may not be useful. The size of the hematoma that should definitely be removed has not been ascertained. Removal of a very thin acute SDH may not be indicated as the clinical deterioration is usually due to associated lesions in this case and is not likely to improve with acute SDH evacuation. Although the current consensus is to have an acute SDH promptly evacuated through a craniectomy in 97% of patients, conservative treatment of a small acute SDH in patients (approximately 3%) with contraindications for surgery has been reported.

No operative therapy should only be considered in patients who are fully conscious, when the extra-axial mass is the single dominant lesion, that is, there are no multiple contusions or potentially significant contralateral mass lesions (which may be preventing midline shift), and when there are no features of mass effect such as a midline shift >3 mm or basal cistern effacement.

In such cases, and especially, if the lesion is <10 mm at its thickest point, conservative therapy may be successful in most instances. The SDH will usually resorb within

1 month although there are occasional instances of chronic SDH formation. Similarly, a deep seated interhemispheric or tentorial SDH in a stable conscious patient may not need surgical evacuation.

The guidelines for selecting patients for conservative management of the SDH include: (1) GCS score ≥13 since injury; (2) absence of other intracranial hematomas or edema on CT scan; (3) midline shift of <10 mm; and (4) absence of basal cistern effacement. Thickness or the associated midline shift beyond which failure of conservative treatment could be predicted.

The recommendations of the TBI Consortium for the surgical management of acute SDH with a thickness >10 mm or a midline shift >5 mm on CT scan should surgically be evacuated, regardless of the patient's GCS score. All patients with acute SDH in coma (GCS score <9) should undergo intracranial pressure monitoring. A comatose patient (GCS score <9) with an SDH <10 mm thick and a midline shift <5 mm should undergo surgical evacuation of the lesion if the GCS score decreased between the time of injury and hospital admission by 2 or more points on the GCS and/or the patient presents with asymmetric or fixed and dilated pupils and/or the intracranial pressure exceeds 20 mmHg.[10] An increase in hematoma size on CT scan with increasing intracranial pressure and decline in neurological status is also an indication for surgical removal of the lesion. Regarding the timing of surgery, it is recommended that in patients with acute SDH and with indications for surgery, surgical evacuation should be performed as early as possible. If surgical evacuation of an acute SDH is indicated in a comatose patient (GCS <9), it should be performed using a craniectomy with bone flap removal.

RESULTS

The mortality from an acute SDH in all patients shows a wide range (42–90%), in all age groups with GCS between 9 and 12 requiring surgery about 68%, and in comatose patients requiring subsequent surgical evacuation is about 35%. Residual or recurrent hematoma requiring evacuation has been seen in approximately 8% of patients. Occasionally, removal of the mass effect caused by the acute SDH may increase the underlying intracerebral hematoma or contralateral acute or chronic SDH. Post-operative hematomas should be suspected and a post-operative CT obtained in patients who fail to improve or those who deteriorate and in whom the intracranial pressure monitoring shows persistently high intracranial pressures. The post-operative complications following evacuation of an acute SDH may include osteomyelitis, wound infection, meningitis, subdural empyema, abscess formation, and ventriculitis.

DISCUSSION

The Factors Determining Outcome Include

Timing of surgery

Usually, conservative treatment is adopted and surgery deferred in patients with less severe acute SDH, and in better neurological status. Thus, mortality is more whenever timing from injury to surgery increases. In comatose patients, however, there was a significant decrease in mortality and increase in functional recovery in patients who underwent surgery within 4 h of injury as compared to those in whom surgery was delayed beyond 4 h of injury. The mechanism of secondary brain damage is direct compression of the underlying cortex and brain shift that causes local zones of ischemia. If the elevated intracranial pressure is unrelieved, leading to reduced cerebral perfusion pressure, then global ischemic brain damage may occur.

Age

Younger patients have a better outcome than older patients due to less comorbid conditions in the former. There is a significant association between age and functional recovery.

CT parameters

CT parameters include clot thickness, hematoma volume, mid line shift and patency of the basal cisterns. Following surgery for acute SDH, found a significant correlation between poor outcome and the volume of SDH and the midline shift and a correlation between outcome and clot thickness and the status of the basal cisterns. There was a significant relationship between midline shift and outcome in patients with GCS scores lower than 9, who were undergoing surgery for SDH. As per our study reveled a 40% mortality rate in patients with clot thickness of < 10mm and 85% mortality for patients with clot thickness >20mm.[11] Thus, these parameters do seem to influence outcome, but the specific threshold values need to be determined.

The neurological status

This forms the most significant factor in determining outcome. In patients with acute SDH and GCS of 3-5, the mortality was 86% and those with GCS of 9-12 had mortality of 18% and moderate to good outcome in 63%of patients. Pupillary asymmetry correlates with a poorer outcome. In bilateral pupillary abnormalities, the mortality is over 85%; in unilaterally dilated but reactive pupils, the mortality reported is approximately 50%, and in unilaterally dilated non-reactive pupils, the mortality reported is approximately 58%. Decerebrate posturing, flaccid patients (mortality 77–95%), and patients with hemiparesis and hemiplegic (mortality 35–48%) also have a poorer prognosis as compared to intact patients.

Intracranial pressure

Persistently elevated (> 20 mmHg) post-operative intracranial pressure is associated with a poor prognosis.

Associated lesions

An associated intracerebral hematoma or contusion did not influence mortality, but the functional outcome was significantly better in patients without contusions. Associated diffuse axonal injury significantly influences outcome. Acute SDH based on their associated lesions are classified intosimple acute SDH without brain injury (mortality: 22%), acute SDH with contusion (mortality: 40%), and complicated acute SDH (with parenchymal laceration, intracerebral hemorrhage, or burst temporal lobes; mortality: 53%).[12]

Comorbid conditions

Lung injury, meningitis, shock, long bone fractures, and abdominal injury all influence outcome. According to the TBI, the key issues for further investigation in cases of acute SDH include the influence of medical management versus decompressive craniectomy on the outcome; the impact of the timing of surgery, the pre-operative hypotension and hypoxia on outcome; identification of subgroups that do not benefit from surgery such as older patients with low GCS scores, pupillary abnormalities, and associated intracerebral lesions; and investigating whether operating on all comatose patients regardless of their clot thickness would lead to a better outcome.

Isolated acute SDH, acting as a compressive lesion, is an uncommon clinicopathological entity with the majority of patients having associated focal (contusion/laceration/intracerebral hematoma) or global (diffuse axonal injury and subarachnoid hemorrhage) involvement or both. Ischemia underlying an acute SDH and hemispheric brain swelling may be superadded and self-perpetuating and may lead to uncontrollable elevations of intracranial pressure with consequent herniation, brainstem compression, and hemorrhage. The molecular cascade initiated by the injury may lead to secondary brain damage.

CONCLUSION

The future reduction in morbidity and mortality will depend on the effective prevention, arrest, or reversal of the molecular events that are responsible for the secondary ischemia and cytotoxic edema. Acute SDH should therefore be subclassified as SDH with or without associated parenchymal pathology to shift the focus from the hematoma to the brain injuries and the secondary injuries and would permit better comparison of different therapeutic modalities and better prognostication. About 25% of patients died in hospital, 18% survived with unfavorable outcomes, and 57% had favorable outcomes. In moderate to severe TBI with ASDH patients, with successful early decompresive craniectomy and evacuation of clot and aggressive intensive care management gives high functional status and better outcomes.

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