ORIGINAL ARTICLE

Current Consideration Regarding Operative Versus Non-Operative Outcome of Brain Contusion Patients

Saeed Mazher[1], Shahid Ahmed[2], Abdul Ali[3]

ABSTRACT

Background: Brain contusion surgeries versus conservative treatment are considered to be the typical decisions faced by doctors, resolved usually based on the experience of respective departments. Till date there are no standard guidelines regarding conservative versus surgical management.

Objectives: To determine the outcome of the surgical and medical management in posttraumatic brain contusion patients.

Methods: The study was conducted on patients having small and large brain contusions admitted in the department were selected through custom Proforma. The 41 patients (31 males 10 females) included corresponded to no specific exclusion criteria. The patients who improved by Glasgow coma score, neurologically or resolution was confirmed by Computed tomography scan brain were categorized under conservative management. While the patients, who deteriorated by Glasgow coma score, neurologically, bradycardia or showed expansion in the size of contusion with mass effect underwent standard decompressive craniotomy or cranectomy and evacuation of contusions along with duroplasty. Complications and improvement of the patients were determined at follow up and monitored by the help of interval brain CT scan during their stay in the department subsequently followed by cranioplasties.

Results: There was a male predilection with male to female ratio of 3.1:1. The age group more frequently affected was 20-40 years and the most causative agent was road trauma accident. Complications were found among two patients as intractable seizures, brain abscess formation, hydrocephalus and post operative jaundice in one patient each. Wound infections in four patients. Overall outcome was good in conservative and surgical intervention patients of brain contusions. The total mortality was four patients, two in each group of patients.

Conclusion: Therefore we recommend that nonsurgical and surgical management has comparable results, but surgical decompressive craniotomy is the mainstay of treatment based on the essential monitoring tools as repeated interval scanning and neurological evaluation considering the timing of surgery, volume and size of hematoma, conscious status, bradycardia and hypertension.

KEY WORDS: *Post Traumatic Brain Contusions, Glasgow Coma Score, Outcome.*

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[1] **Saeed Mazher**

Consultant Neurosugeon, Ziauddin University and Hospitals, & Taj Medical Complex, Karachi.

[2] **Shahid Ahmed**

[3] **Abdul Ali**

INTRODUCTION

The traumatic brain mass lesions comprise of approximately about 8.2%, among all traumatic brain injuries.[1] These traumatic lesions were as much as 20% of operative intracranial lesions in respective series[2] while most of the small intracranial lesions do not require surgical evacuation.

Cerebral contusions are areas of trauma induced brain swelling. These are zones of cellular injury where the microvasculature is also disrupted. The contused area becomes an amalgam of blood and necrotic brain, the so-called 'hemorrhagic contusion'. Sometimes bleeding from a small ruptured vessel dominates and an intracerebral hematoma results. The resultant micro-hemorrhages provide the basis of the radiological grading of diffuse axonal injury (DAI). Significant brain swelling may not occur in some cases of diffuse axonal injury.[3]

The genesis of brain swelling in an area of brain contusion is complex.[4] The mass effect of an area of contusion is conventionally considered to result from a combination of vasogenic and cytotoxic edema. Vasogenic edema results from the breakdown of the blood brain barrier and extravasations of fluid into the extracellular space. Cytotoxic edema is the consequence of a hypoxic insult resulting in membrane pump failure and cellular swelling. The early swelling around a contusion which occurs in the first 24 hours and is often life threatening, cannot be explained by either of these factors. Vasogenic edema sets in only after 12-24 hrs.[5] Cytotoxic edema can occur early, but the quantum of cytotoxic edema is insufficient to explain the mass effect that is clinically encountered. There are three clinical phases of brain swelling due to contusion. The ultra early phase occurs within the first 24 hours and is often the cause of clinical deterioration or death. The second, delayed phase sets in after 24 to 72 hrs and progresses for 7-10 days.[6] This swelling rarely contributes to clinically significant intracranial hypertension.

The severe brain swelling which occurs after a traumatic brain injury has three temporally distinct and etiologically disparate phases.[7] In the first phase, there is breakdown of cell membrane proteins and lipids into smaller molecules resulting in an osmotic gradient and an indrawing of fluid into the contusion.

The second phase of brain edema is dominant in 24 to 48 hours after trauma. This phase is mediated by activation of thrombin consequent to triggering off of the coagulation cascades.[8] Thrombin and inflammatory mediators causes the disruption in the blood brain barrier leading to vasogenic edema. The third phase of edema sets in with the lysis of RBC in the intracerebral clot. Hemoglobin breakdown products activate reactive oxygen species, trigger cytokines (esp IL6 & IL10) and activate the complement system (esp C 3d & C9).[9]

Due to the pathological consequences of cerebral edema and other factors like epileptogenic activity, hypercarbia, hypoxia, hypertension, hypotension leading to impairment of cerebral blood flow. This diminution of blood flow causes raised intracranial pressure and fall in cerebral perfusion pressure. This expanding contused brain matter in a compromised setting of auto regulation (Trauma induced)[10] results in further fall in cerebral perfusion pressure and rise in intracranial perfusion pressure.

A variety of surgical strategies had been employed ranging from simple decompressive craniotomy without resection of contusion, resection of contusion combined with decompressive craniotomy, safe cerebral lobe resections with decompressive craniotomy without resection of contusion and simple resection of contusion without decompressive craniotomy. The aim of this grouped retrospective trial, which was carried out in China was to recommend the best strategy to preserve the neurological functions among posttraumatic contusion patients.[11]

The aim of this study was to report our experience with the post traumatic brain contusion management guidelines and recommendations.

METHODOLOGY

The present study focused on the standard protocols of surgical versus nonsurgical decision and their respective outcomes. This study was conducted after the approval of research and ethical committee of the hospital. This prospective study was carried out at the Neurosurgical Department, Abbasi Shaheed

Hospital Karachi Medical and Dental College, from December 2011 to Dec 2012. We included 41 patients regardless of their age and gender who had post traumatic brain contusion secondary to road traffic accident, fall, and assault admitted in our Neurosurgical Unit mostly through casualty and those referred from other hospitals. All the admitted patients were undergone through the CT scan after neurological assessment. They were categorized as mild, moderate and severe head injury based on Glasgow coma score and accordingly shifted to ward, high dependency unit and intensive care unit respectively. Investigations performed included full blood count, serum electrolytes, blood sugar, urea creatinine, prothrombin time (PT) and activated thromboplastin time (APTT)

All the patients having supratentorial brain contusions were kept on prophylactic antiepileptic medications. Similarly patients having cerebral edema, cisternal effacement and not showing extradural or subdural hematoma simultaneously were prescribed on osmotic diuretics. During the initial three days patients were monitored by the neurological exam and vitals, if patient developed seizures, deterioration of neurology or cushing reflex CT scan brain was repeated even earlier. Within the week or initial 2-4 days, based on the CT scan brain findings and improvement versus deterioration of neurology were considering factors for surgical or non surgical decisions. In one patient special investigation MRI brain was also performed to exclude the cause of deterioration. Patients were followed in the outpatient department at 1st week after discharge, 2nd week and finally after 6 month and readmitted with a plan of cranioplasty if required.

Data regarding number of cases, age, gender, time between surgical or nonsurgical management and recovery, surgical procedures performed and postoperative outcome was collected on proforma and processed on Microsoft Excel. The variables were calculated in numbers and percentages.

RESULTS

After exclusion of 3(7.31%) patients who were lost to follow up the study population comprised 38(92.68%) patients with post traumatic brain contusions secondary to road traffic accidents, assaults and occupational injuries.

Out of 38 patients, 30 were males and 8 were females, with a male to female ratio of 3.1:1. The mean age of the patients was 26 years. Thirty six patients had sustained head injury and admitted through emergency room while two patients were referred from other hospitals in the locality. All the patients were admitted in our department at intensive care unit, high dependency unit and ward accordingly depending upon their Glasgow coma score and neurological status. Initially patients were managed conservatively by instituting the physical, pharmacological measures and ventilator support.

The time of presentation of post traumatic brain contusions from other hospitals was up to 24 to 48hrs. The clinical presentation included head ache in 29 (76.31%) cases. Twelve (31%) patients developed seizure episodes while nine patients had more prominent feature of drowsiness and irritability.

Table 1: Site of Injury according to CT scan Brain findings n=41

Management Strategy	Site of Injury	No. of cases	%
Surgical	Frontal Parietal Temporal Post-fossa	14 02 01 01 Total: 18	43.9%
Non Surgical	Frontal Temporal Parietal Post-fossa	12 02 07 02 Total: 23	56.0%

The seven (18%) patients who had Glasgow coma score of less than eight were managed in intensive care unit, about five of them undergone surgical decompression and contusion removal. Remaining patients were managed in high dependency unit as they showed improvement in Glasgow coma score they were shifted toward after rescanning. While subsequently about 12 (31%) patients were in the high dependency unit who had

decompressive craniotomy and contusion removal due to deteriorating Glasgow coma score and expansion of hematoma within one week after injury (Table-2).

Table 2: Operative Procedure n=18

Procedure	No. of Cases	%
Bifrontal decompressive craniotomy with contusion removal	6	33%
Frontal decompressive craniotomy with contusion removal	4	22%
Frontoparietal cranectomy with contusion removal	4	22%
Temporal craniotomy with lobectomy	2	11%
Pos-fossa cranectomy with removal of contusion	2	11%

Table 3: Post-operative Morbidity n=41

Complication	No. of Cases	%
Seizures	02	4.8%
Brain abscess	01	2.4%
Post operative jaundice	01	2.4%
Wound infection	04	9.7%
Hydrocephalus	01	2.4%
Death	04	9.7%

One of the patients who was having GCS of 4/15 had undergone immediate decompressive cranectomy and temporal lobectomy as the patient was having a burst temporal lobe in the initial CT scans, but unfortunately he succumbed to death two days after surgery.

Figure 1: CT Scan Reveals Bifronta; Post-traumatic Brain Contusion

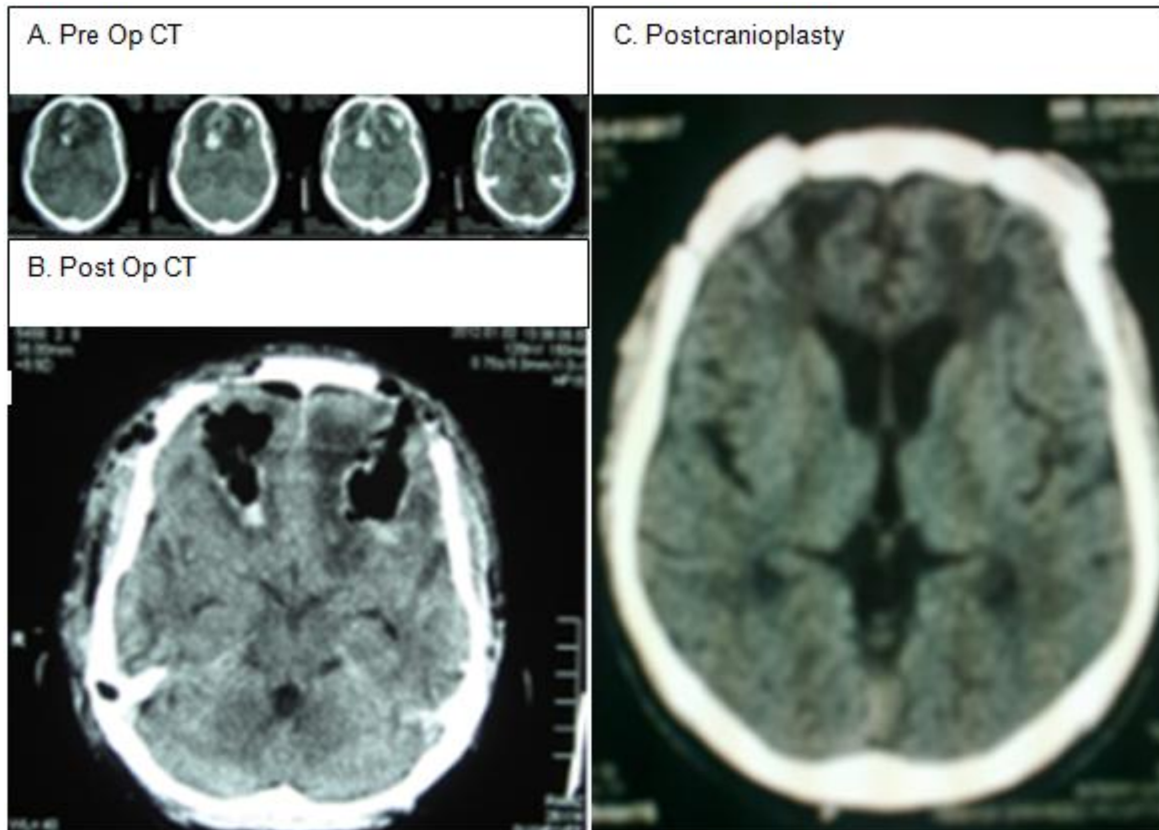
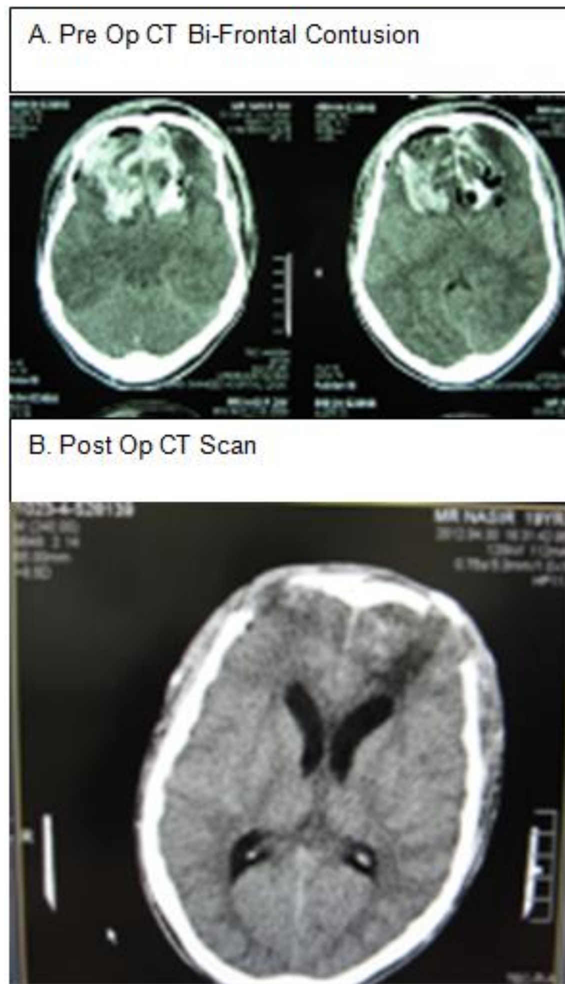


Figure 2: Pre Op and Post OP CT Scans



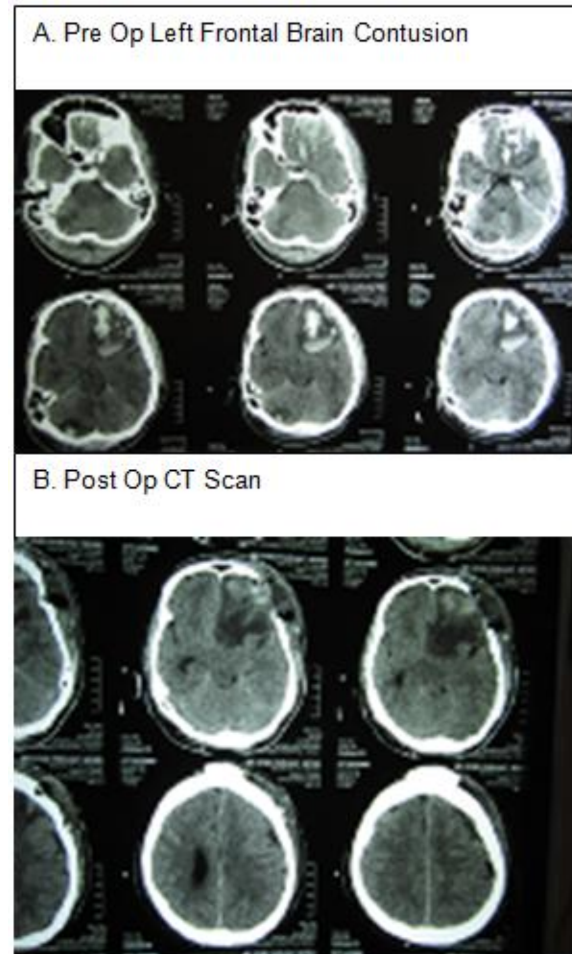
Among 41 patients, 6 (14%) patients underwent bilateral surgery and 10 (24%) patients undergone unilateral surgery Table 2. Post operative complications included hematoma enlargement, seizures, brain abscess formation, hydrocephalus and wound infection Table 3.

After stratification it was found that 23 (56%) patients were managed non surgically (Table 1).

On 6 months follow up there were 8 (19%) patients with good recovery, 12 (29%) with moderate disability, 5(12.1%) with severe disability, 9(21.9%) in vegetative status and 4(9.7%) had died.

Patients has good recovery and moderate disability were considered as good prognosis group and those who had severe disability and vegetative status or died were categorized as poor prognostic group

Figure 3: Pre & Post Op Frontal Brain Contusion



DISCUSSION

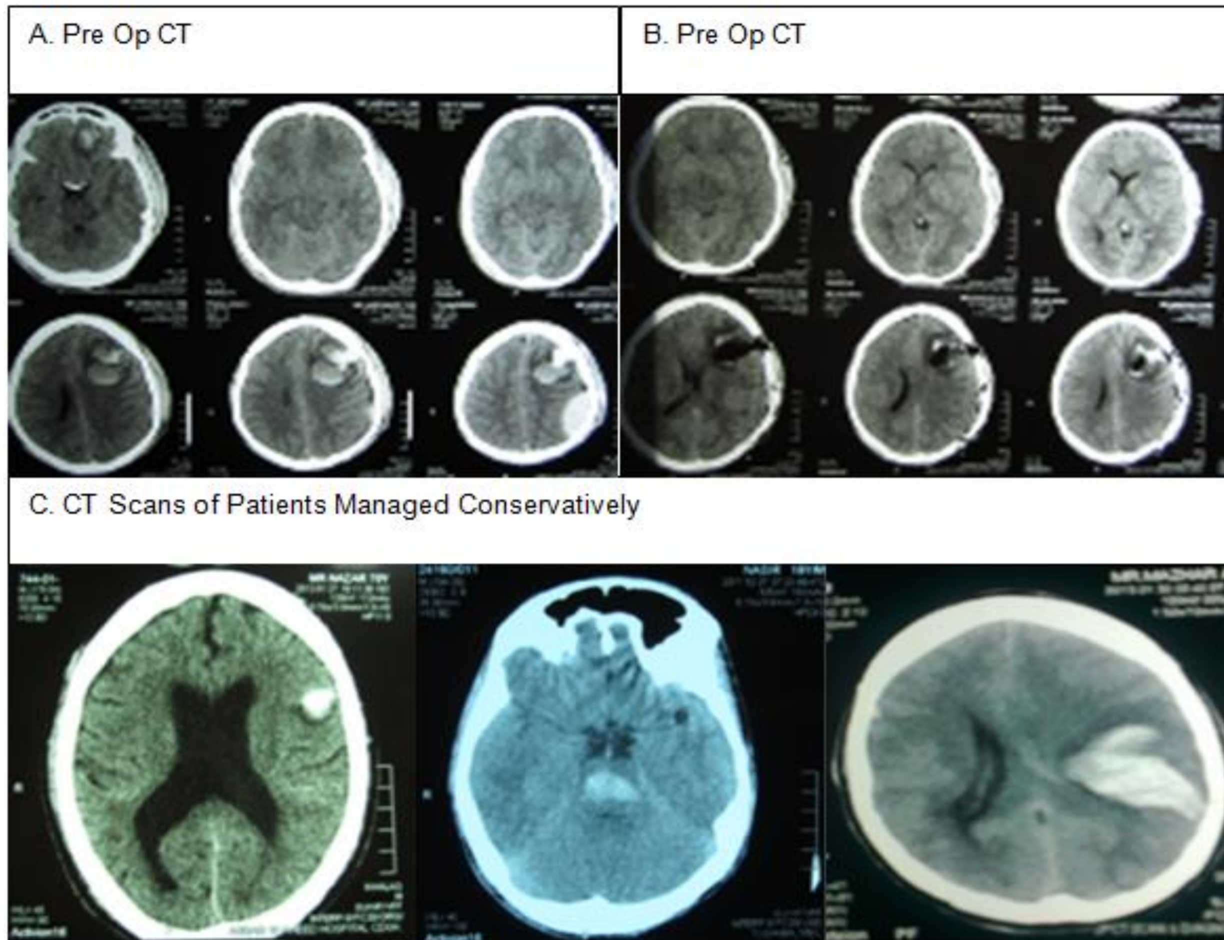
Severe brain contusion is often associated with mass effect and diffuse axonal element or extradural and subdural collection. This mass effect usually progress rapidly after 48 hours. After the breakdown of debris membrane and cytoplasmic structures there is a high osmotic potential generated within the contused brain tissue, which leads to water accumulation and rapid progression.[12, 13]

It has been reported that surgical intervention should be considered as early as possible when surgical indication are met.[14, 15,16]

But the surgical strategy usually employed varies depending on institutional experience.

Kawamata et al. believed that early massive edema is caused by brain contusion, and surgical excision of the necrotic tissue provides satisfactory control of progressive elevation in intracranial pressure and clinical deterioration.

Figure 4: Frontoparietal Contusion and Extradural Hematoma

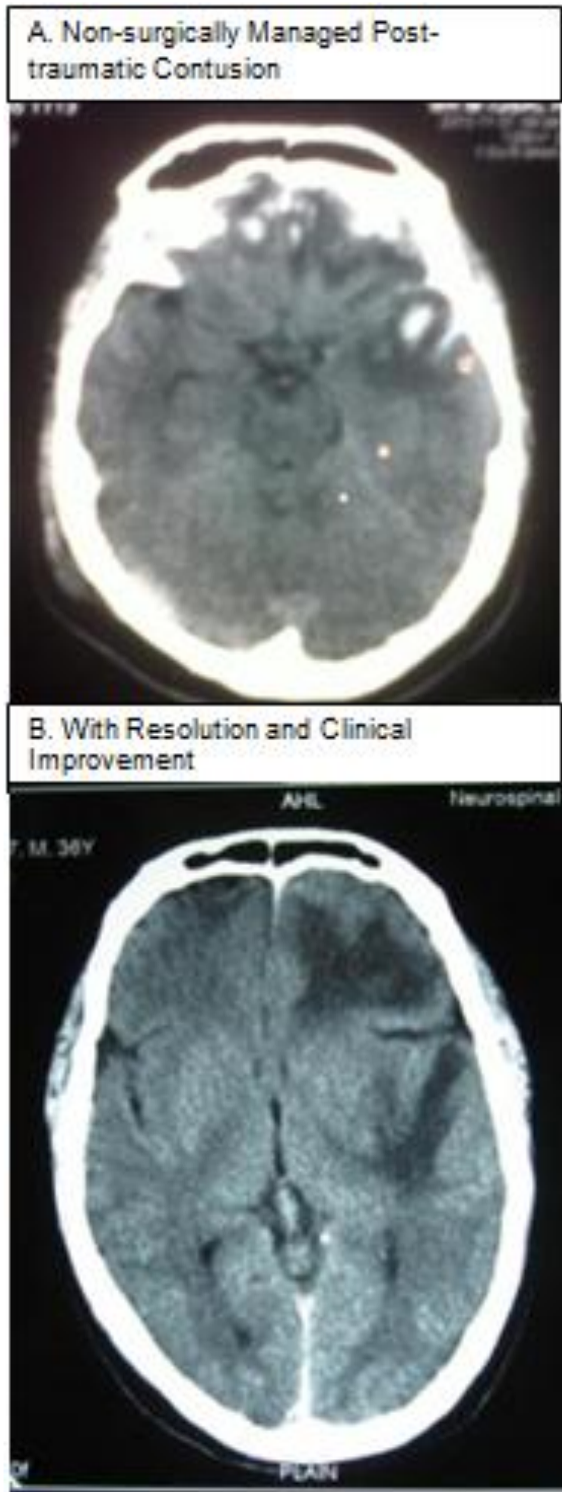


In our series of 41 patients, 18(43.9%) patients were surgically intervened (Figure 1-4), while remaining 23(56.09%) patients were tentatively ready for the intervention (Table 1) but brain contusion resolution continued with the clinical improvement hence managed conservatively (Figure 4).

Before 2007, the surgical strategy for the acute severe brain contusion in one of the university hospital of china reported was primarily resection of the contused tissue followed by Decompressive craniotomy, which was consistent with Rubiano et al and Kawamata et al. It was most commonly employed for the contusions located in a relatively non functional area. As a result the mortality rate was significantly reduced under this type of strategy but the resected region was constantly associated with certain degree of neurological dysfunction.[17, 18, 19, 20]

Seizures should always be considered as confounding factor, expansion of hematoma should be considered timely by repeat CT scanning after excluding the electrolyte imbalance. In few of our patients who came to follow up after 12 months through telephone contact had recurrent episode of seizures due to non compliance to antiepileptic medications. One of the patients who had undergone bifrontal decompressive craniotomy, with expended duroplasty. After 6 months of follow up cranioplasty was performed (Figure 1), had intractable seizures which was treated by combination antiepileptic therapy of sodium valproate and levetracetam.

Figure 3: Non-surgically managed and Resolution with Clinical Improvement



One of the patients of our series who was a known psychiatric patient had brain abscess formation, diagnosed in outpatient clinic at the followup visit after 4 months. As the patient was toxic and had a large abscess cavity therefore planned to drained by burr hole aspiration. He was kept on antibiotics for the period of 3 months. Then he remained well in further frequent follow up visits (Figure 4). Another patient who had undergone unilateral frontal craniotomy developed post operative jaundice which was presumed to be anesthesia related complication and considered to be the cause of delayed recovery. Hence post operatively MRI brain was done to exclude neurological pathology later after consulting with the general surgeon ultrasound abdomen was done and patient was found to have gall stones which were treated by Endoscopic Retrograde Cholangiopanreatography (Figure 3)

Nowadays people are talking about how can we avoid the resection of necrotic brain tissue and achieve better outcome. Hence in our series we tried our best to manage patients conservatively with tentative planning of intervention, and it was best performed by surgical excision of contusion by appropriately placed pial-cortical window [21] with minimal or no trauma to surrounding brain tissue for those patients who deteriorated clinically and radiologically. Further more conservative contusectomies are best combined with decompressive craniotomy. After wards cranioplasty was performed.

To establish standard guidelines, some larger studies or trails will be required, as there are multiple other factors which are directly or indirectly impacts on the overall outcome. Such as GCS, severity of injury, primary brain injury, associated peripheral and visceral injuries and delayed arrival at the tertiary care centers, are far beyond the scoop of this article.

CONCLUSION

Hence the current consideration is to stratify the management of brain contusion patients so that the outcome can be improved and morbidity can be minimized. It is well accepted by evidence that not all patients require surgical intervention. Therefore we recommend that initially if patients are not meeting the criteria of surgical intervention they should be offered physical and

pharmacological measures to decrease the intracranial pressure, if not responding and the patient`s clinical status deteriorating along with expansion of hematoma or increasing mass effect then again surgical decompression should be considered early.

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