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Research Article

Setting Protocol for Management of Traumatic Brain Edema

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Abstract

Traumatic brain edema is a serious complications that might affect patients with cranial injury, it may be associated with aggressive morbidity and mortality , the traditional management of the brain edema does not concentrate on controlling the amount of the fluid reaching the brain , and concentrate only in maintaining adequate brain tissue perfusion neglecting the possible harmful effect of the excess fluid on the edematous brain tissue.

Aims of the study are to:

1- Study the effect of using a combination of ways to control the fluid content of the brain and body to achieve good outcome in the patients of traumatic brain edema .

2 - Set a protocol to manage the patients using different ways of controlling the fluid amount in the brain.

***Patients and methods**: All patients admitted to the author's unit in the neurosurgical departments in Alshaab teaching hospital , Ibn Khaldon hospital , and Doctors hospital all based in Khartoum Sudan , with history of recent head injury and loss of consciousness and then CT scan diagnosis of brain edema , in the period from August the first 2006 to the end of September 2010 (four years period) .*

Criteria of patient's admission to the study are:-

1- History of recent head injury either direct or indirect.

2- Loss of consciousness immediately following head injury with Glasgow coma scale of 12 or below .

3-Absence of any other primary apparent intracranial brain injury clinically or radiologically.

4- I.C.U. patient admission.

5- Specific protocol of 15 steps was followed in all patients consisting of administration of Furosemide , Oxygen , controlled amount of fluid ,Piracetam and other specific drugs with specific follow up

Results

40 patients were included in this study , 32.5% were below the age of 18 years and the mean age was 21 years , most of the patients 82.5% were males.

In 92.5% of the patients the cause of the trauma was road traffic accidents while in 7.5 % it was fall from heights.

The main presenting symptom was loss of consciousness following the insult in 97,5%, scalp lacerations in 37.5%, vomiting in 37.5% bleeding from nose mouth or ears in 32.5% and seizers in 15% of the patients.

In 27.5% the Glasgow coma scale on admission was below 8, while it was from 9 - 12 in 55% and above 12 in 17,5%.

The CT brain finding was severe brain edema in 55% or moderate brain edema in 35% and mild in only 10% of the patients.

The final outcome of the patients was Complete cure in 75%.

Partial improvement in 15%, 10% died the causes of death are mostly not directly related to head injury or to its sequel.

***Conclusions**:*

1- Fluid control in brain edema is a flexible method in managing this condition with good outcome .

2- Multidisciplinary approach is very valuable way in managing traumatic brain edema.

3- Close and frequent follow up with flexible adjustments of the various drugs and amount of the fluid used in this method is the key factor in achieving good outcome in those patients.

Introduction

Brain edema is defined as an abnormal accumulation of fluid in the brain tissue accompanied by an increased volume of the brain. It usually results in the increase of intracranial pressure that may affect patient's life[1]

Brain edema may compromises maintenance of the cerebral blood flow. But primary blood flow disturbances may lead to brain edema. The mechanisms underlying the blood flow impairment by brain edema are associated with an increased water accumulation in the parenchyma[2].

Brain edema that usually follows blood-brain barrier disruption, is called vasogenic edema, this is present in most patients of brain edema. According to the Starling's law, water, ions and plasma proteins cross the blood-brain barrier toward the interstitium if the driving forces for transmural bulk flow are excessive and/or if the blood-brain barrier permeability is increased. Both mechanisms are present in most patients. Excessive increment of the gradient of hydrostatic pressure with loss of cerebral auto regulation has been incriminated as a cause of ischemia in traumatic brain edema patients[3].

Water acts as a solvent for electrolytes to influence the molecular configuration, this enhance enzymatic function in particular of polypeptide chains in biological systems. Water and electrolytes determines the osmotic regulation of cell volume this play great role in establishment of the transmembrane ion concentration gradients which in turn induce nerve excitation and impulse conduction. Fluid in the central nervous system is distributed in the intracellular and extracellular compartments of the brain parenchyma, the cerebrospinal fluid, and the vascular compartment of the brain arteries and veins and small capillaries. The exchange of fluid occurs across blood-brain barrier, at the ventricular Ependyma , choroid plexus and, on the brain surface, at the pia mater. The normal blood-brain barrier is relatively permeable to water, but considerably so less to ions [4].

The capillary endothelium isolates the extracellular fluid space in the central nervous system from the plasma. The combination of physical and enzymatic mechanisms which prevent macromolecules, polar solutes, neurotransmitters, peptides, and electrolytes from passively entering the brain is the blood-brain barrier. Specific mechanisms facilitate transport of ions across the blood-brain barrier and active secretion of extracellular fluid and cerebrospinal fluid maintaining homeostasis for nutrients and for cation and hydrogen ions respectively. Consequently, interstitial fluid volume in the central nervous system does not increase when the total extracellular fluid volume is increased. Total tissue volume is sensitive to osmotic forces, while oncotic forces are relatively unimportant[5].

The appropriate administration of intravenous fluids in neurosurgical patients remains an area of disagreement between neurosurgeons and anesthetists. Fluid restriction has long been advocated by the neurosurgeons in cerebral edema and is believed to reduce or prevent the formation of cerebral edema

But such restriction may lead to hypovolemia which in turn can result in hemodynamic disturbances. The brain homeostasis should be aimed for through adequate fluid administration and normal or slightly elevated mean arterial pressure. The properties of the endothelium differ between the brain and the reminder of the body[6].

Late posttraumatic circulatory changes are caused mainly by brain edema. This has an important influence on cerebral blood flow and cerebral microcirculation.

Mortality is high among patients developing post-traumatic brain edema and increased intracranial pressure following severe head injury. Although routine treatment varies from one center to another it often includes one or more of such measures as hyperventilation, high-dose barbiturate therapy, osmotherapy or the drainage of cerebrospinal fluid[7].

The preservation of high cerebral perfusion pressure is fundamental to traditional treatment of cerebral edema , this is usually combined with inotropic support, as ischemia is considered to be a crucial factor with regard to the secondary injury and development of brain edema [8, 9]

Piracetam was found to have great role in recovery of the cortical bioelectrical activity disturbed by traumatic brain edema. The beneficial effect of Piracetam is probably due to an optimization of the functional state of the brain since Piracetam enhances brain excitability[10].

Aims of the study is to:

Study the effect of using a combination of ways to control the fluid content of the brain and body to achieve good outcome in the patients of traumatic brain edema .

Set a protocol to manage the patients of traumatic brain edema.

Patients and Methods

All patients admitted to the author's unit in the neurosurgical departments in Alshaab teaching hospital , Ibn Khaldon hospital , and Doctors hospital all based in Khartoum Sudan , with history of recent head trauma (within 72 hour) and loss of consciousness and then CT scan diagnosis of brain edema , in the period from August the first, 2006 to the end of October 2010 (4 years period) .

Criteria of patient's admission to the study:-

1- History of recent head injury either direct or indirect.

2- Loss of consciousness immediately following head injury.

3- Absence of other primary apparent major intracranial brain injury clinically and radiologically.

4- I.C.U. patient admission.

Radiological features of brain edema:-

1- Obliteration or effacement of brain surface sulci. (Mild brain edema)

2- Obliteration or effacement of the ventricular system. (Moderate brain edema)

3- Obliteration or effacement of the basal cisterns around the brain stem. (Severe brain edema)

Patients management protocol

1- I.C.U. Admission.

2- Oxygen therapy

3- Elevation of the head of the bed by 30 – 45 degrees.

4- Ceftriaxone injections in case of presence of features of fracture base of the skull , wounds, or chest crepitation.

5- Nasogastric fluids feeding 2500 - 3000 ml per day for adults groups, and 1500 to 2000 ml per day for pediatric group or according to age.

6- Daily check for input and output of the fluids and correction of the deficits to be around 1000 ml either positive or negative .

7- Furosemide injections according to patient's age and severity of the edema for adults it was 40 mg intravenously twice a day .

8- Basal and frequent check of serum urea and electrolytes once or twice a day.

9- Piracetam and multivitamins after patient's recovery to more than 13 G.C.S. and there after till the maximum recovery .

10- Ranitidine 50 -100 mg or Omeprazole 40 intravenously twice a day to guard against stress ulcers .

11- Chloramphenicol eye drops 3-4 times a day.

12- Haloperidol 1.5 – 5 mg twice to three times a day may be prescribed in cases of severe irritability.

13- follow up CT scans in cases of delayed recovery or deterioration of the patient's neurological state.

14- Glasgow Coma Scale (G.C.S) and Glasgow outcome score at discharge and follow up at 2 weeks after discharge and then at monthly follow up .

15- Discharge when reaching G.C.S. 14 or 15 and with free oral feeding.

16- follow up two weeks after discharge and then at monthly interval till full recovery .

Results

In the study period (from 01. 8. 2006 to 27. 9. 2010) forty patients with traumatic brain edema were admitted under the author's care in public and private hospitals representing all patients of traumatic brain edema under author's care during that period.

Patients age was ranging from 2 to 44 years with mean age , median age was 22.50 and mean age was 21.89 . Table (1)

The sex distribution was as follows 33 patients were males 82.5% and 7 patients 17.5% were females . Figure (1)

The main presentation was Loss of consciousness in 39 patients 97,5% mostly from one day to less than three days in 33 patients 82.5% some lost consciousness for less than one day but more than

one hour 5 patient 12.5% only one patient lost consciousness for more than one month . Table (4)

Other presenting symptoms include scalp wound in 15 patients 37.5% and vomiting in 15 patients 37.5% , bleeding from either nose or mouth or ears in 13 patients 32.5% and seizers in 6 patients 15% , rare symptoms include fever and psychological changes .

Vital signs and systemic review

Vital signs were normal in most patients 38 patients 95.0% , but in some patients there were associated systemic problems such as chest problems in 8 patients 20% which is mainly crepitation due to inhalation or injuries , limb injuries were present in 7 patients 17.5% , abdominal injuries in 4 patients 10% , there were also associated backache and cranial nerves palsies each in 3 patients 7.5% . Abnormal pupils were found in 8 patients 20%.

The CT findings in the patients

The main CT findings in the patients was brain edema as the sole finding in 33 patients 82.5% , the degree of the edema was severe in 22 patients 55%, moderate in 14 patients 35% , or mild in 4 patients 10% .Table (5&6)

On the other hand it was observed that in 7 patients the edema was associated with other intracranial pathology like subdural or epidural hematomas or subarachnoid hemorrhage , cerebral contusions or aerocele.

Five patients 12.5% were subjected to surgical interventions mainly for the complications or the associated pathology burr holes evacuation of hematomas in 2 patients or orthopedic fixation of fractures in 3 patients 7.5%.

The final outcome

The final outcome in the 40 patients was as follows

Full cure in 30 patients 75% (fully conscious GCS 15, without any residual neurological deficit).

Improvement in 6 patient 15% of them 4 were independent 10% and 2 were dependent 5%.

Death in 4 patients 10% , the causes of death were found not directly related to head injury in most of the cases as it was as follows: gastrointestinal bleeding , pulmonary embolism , cardiac arrest may be due to cerebral herniation and respiratory failure .

Complications

No complications were encountered in 30 patients 75% , the complications occurred were manumitted limb fracture in 2 patients 5% , and facial palsy in 2 patients 5% , and other rare sporadic complications . till the present time 29 patients 72.5% were in follow up while 7 patients 17.5% lost the follow up and 4 patients died . See table (7).

Discussion

Brain edema is a very serious insult that might affect some of the patients with head trauma , it might be associated with severe of damage to the brain with subsequent morbidity and mortality[8].

The seriousness of brain edema lie in the fact that it increases the intracranial pressure which in turn impair the cerebral perfusion which leads to accumulation of the metabolites which in turn cause more edema [1, 2].

The cause of brain edema in head injured patients is proved to be due to disruption of blood-brain barrier[3] ,and to dysfunction of the aqua purine water channels in the brain , and vascular endothelial growth factor these changes lead to escape of the intravascular fluid to the interstitial space with resultant compression on the tiny blood vessels which in turn lead to tissue and cells ischemia and accumulation of tissue metabolites[5, 11, 12].

Although the most recent radiological investigation for diagnosis and follow up of the patients of brain edema is M.R.I. of the brain , CT proved a role since it is the mode of investigation usually available for head trauma patients [13, 14] .

The changes that might be noticed in the C.T scans of the Patients with Brain edema are effacement or obliteration of the subdural space and brain sulci in mild brain edema , ventricular narrowing in the moderate brain edema , and finally basal cisterns obliteration in the severe type .

Although there are many managements for brain edema like hyperventilation , oxygen therapy , Barbiturates osmotherapy and diuretics, cerebrospinal fluid drainage and even surgical decompression[8], but it is a fact that the principle cause of brain edema is water disturbance secondary to disturbed blood-brain barrier and the main effort in managing patients with brain edema should be directed toward the cause and not toward the secondary effects , although one should not neglect those secondary effects since they are sometimes more serious than the primary cause .

The insult to the brain caused by brain edema is most probably not present from the start and it might occur as the brain edema advance and compress the brain , this insult is most probably preventable if there is a way to control the edema so as not to reach a serious level threatening the perfusion of the vital areas of the brain.

In this study the author concentrates on fluid control and not fluid restriction[8]. So the patient is given the daily requirement not more not less with nasogastric tube or orally if possible so as the fluid should contain the enough nutrients for the patient at the same time the vital parameters specially the urine output (which should be more than 0.5 mL/kg/hour for the adults or more than 1 mL/kg/hour for the children) and the blood pressure (not less than 100/60 for the adults) should be kept close to the normal as much as possible by drugs administration in the different situations.

The role of Furosemide here is to decrease the watery part of the circulating volume so as not to accumulate in the brain with disturbed blood-brain barrier at the same time to help the wash of the metabolites by creating the need for an excess of fluid.

The fluid chart of the patient should be observed and only a difference of +\- 500 -1000 ml per day between the input and the output is allowed otherwise is either to increase the amount of the fluid by an amount equal to the positive balance or to decrease it by an amount equal to the negative balance for that day. the close monitoring of the fluid chart is the most vital part of this study so six or at least 12 hourly check and observation for the chart although the author used not to interfere to adjust the fluid imbalance except every 24 hours.

In general a flexible balance should be kept between the following factors:

The need for the fluid to maintain the perfusion for the brain and the other vital organs specially the renal system and to counteract the harmful effect of the presence of an excess amount of water in the brain with disturbed blood-brain barrier and other side effects of the administered drugs like electrolytes

imbalance.

Other combinations of physical factors such as head elevation by 30 – 45 degrees and oxygen administration should be utilized, to insure the maximum oxygen delivery to the brain regardless of the peripheral oxygen pulse oximeter readings, since those readings reflect the peripheral tissue saturation and not the cerebral saturation which is expected to be less due to the effect of the cerebral edema on the cerebral perfusion .

Concentration on the meticulous and close observation of the brain edema patients and the immediate and appropriate intervention when there are any disturbances as regard the fluid and electrolytes balance and the renal functions is crucial, the consciousness and pulmonary functions of the patients using the biochemical, radiological and drug therapy to correct any abnormalities .

Other drugs to prevent the complications due to head trauma like Chloramphenicol eye drops to prevent conjunctivitis, which might also lead to misinterpretation of the eye response in Glasgow coma scale.

The use of the Glasgow outcome score as a universal method to check the outcome of the patients was used to access the outcome achieved in this study is satisfactory since from the 40 patient who represent the total number of the patients followed during the study period and satisfying the study criteria and included in this study there four mortalities due to causes which are not directly related to head trauma, while on the other hand the morbidity condition are minor problems and does not endanger patient's lives severely.

Conclusions

Brain edema following head trauma is a serious condition that require aggressive management.

Fluid control in brain edema is a flexible method in managing this condition with good outcome.

Multidisciplinary approach is very valuable way in managing traumatic brain edema.

Close and frequent follow up with flexible adjustments of the various drugs used in this study is the key factor in achieving good outcome in those patients.

Tables and Diagrams and Images

Mean	21.89	years
Median	22.50	years

Table (1) Age distribution

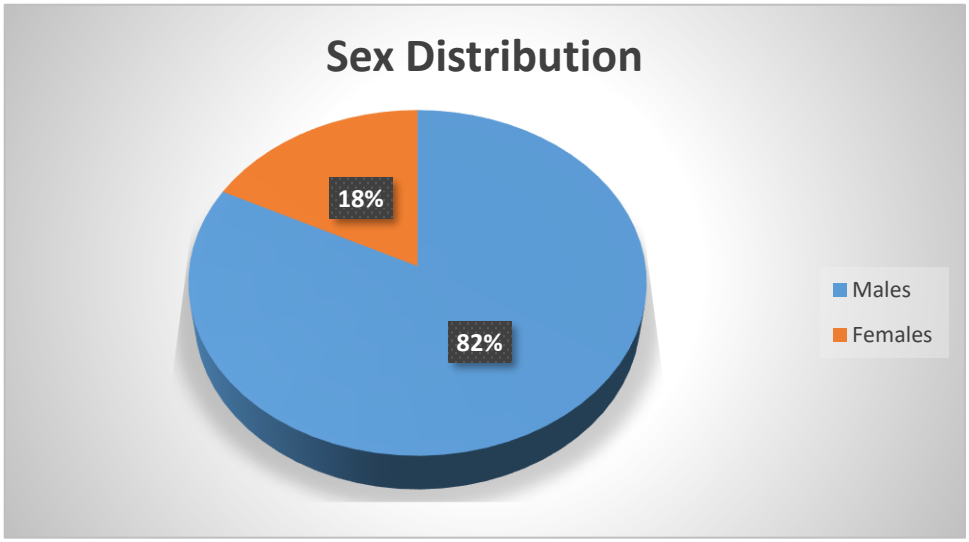

Figure (1): Sex distribution

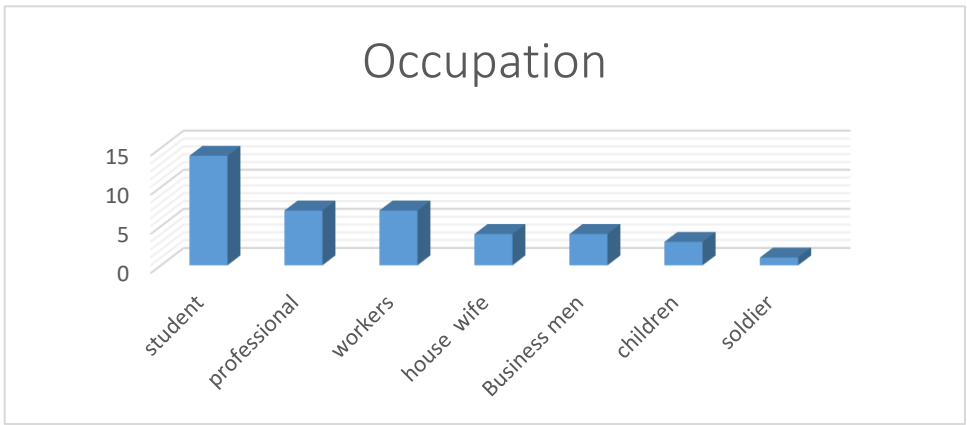

Figure (2): Occupation distribution

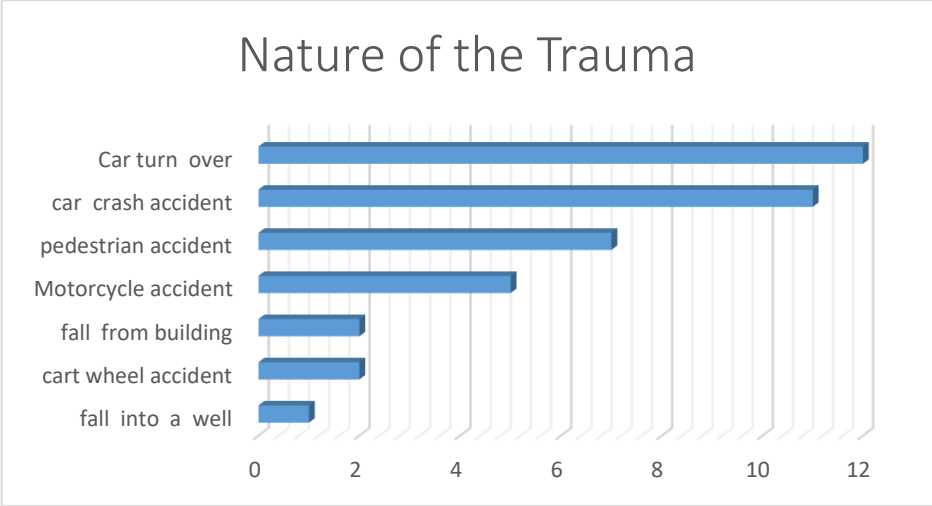

Figure (3): Nature of the trauma , 92.5% of the victims were Road traffic accidents victims other types of trauma patients were only minority

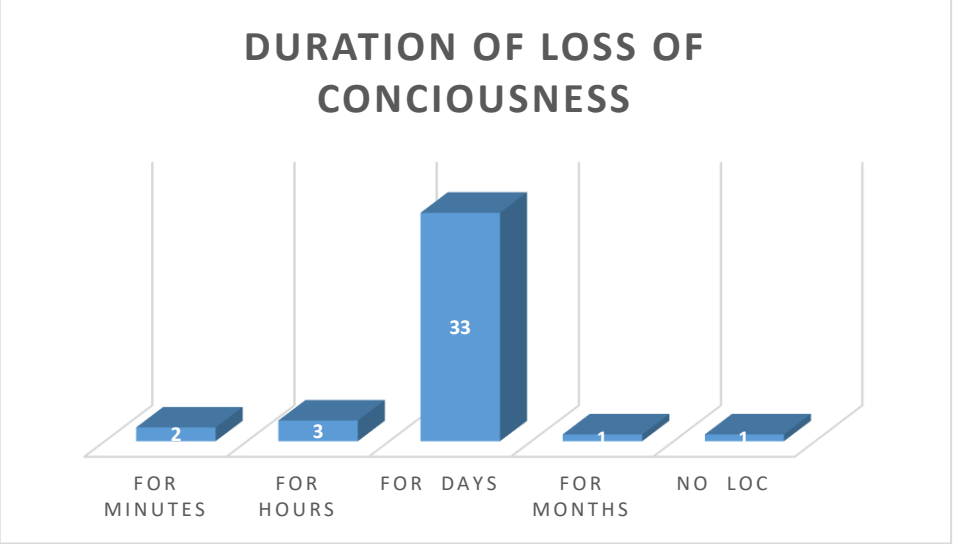

Figure (4): Duration of loss of consciousness

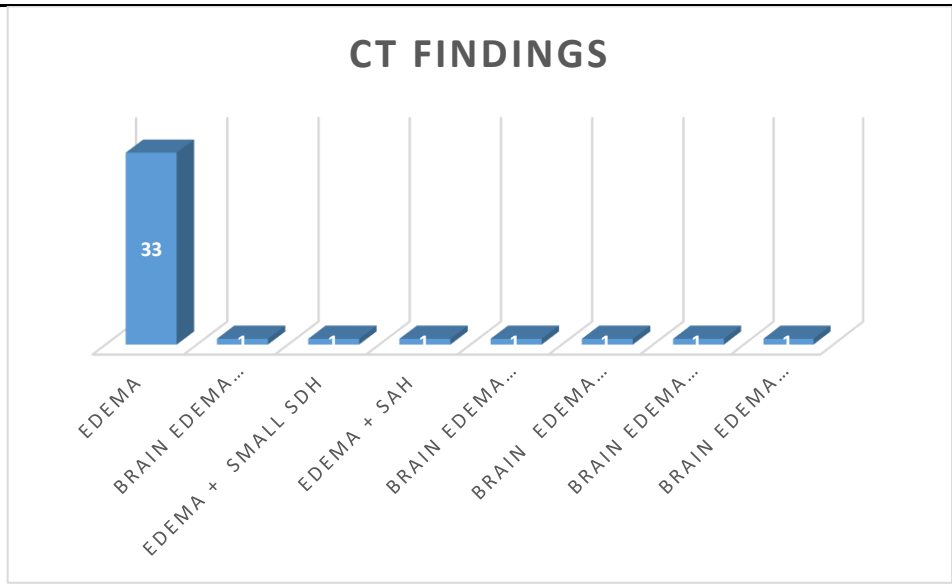

Figure (5): CT Brain findings

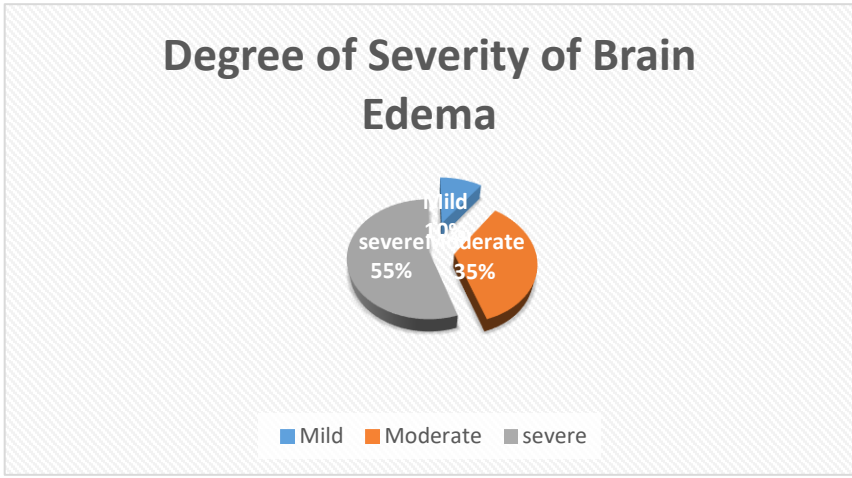

Figure (6): Severity of edema in CT Brain

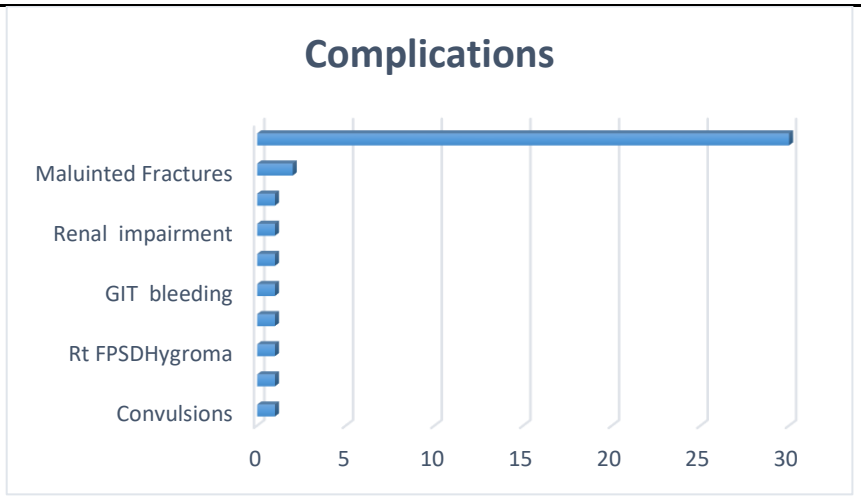

Figure (7): Complications encountered

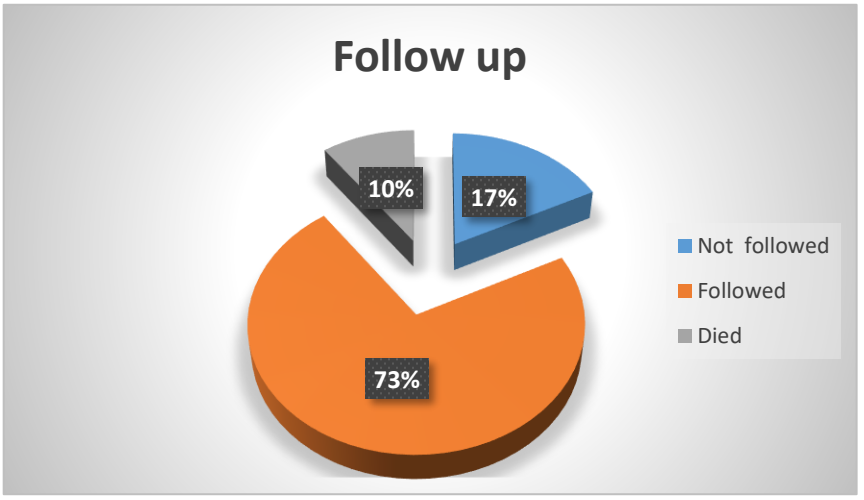

Figure (8): Follow up

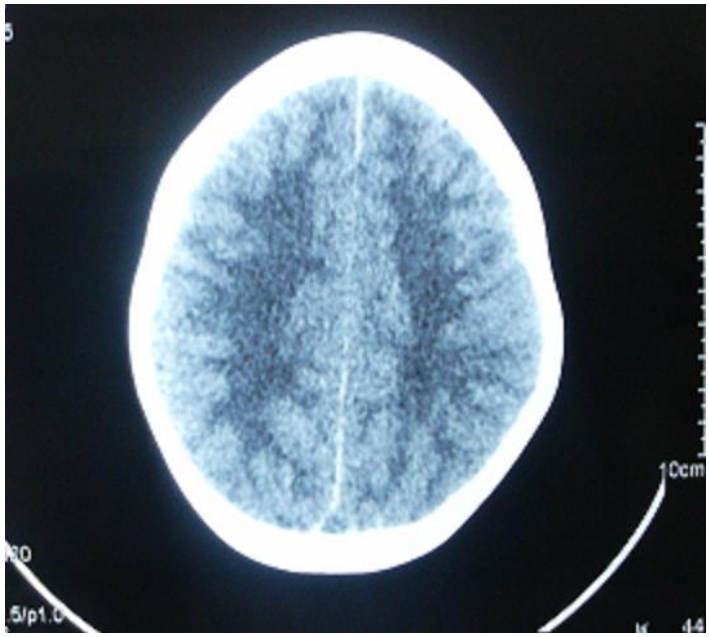

Image (1): CT of one of the patients with severe brain edema with effacement of sulci of the brain

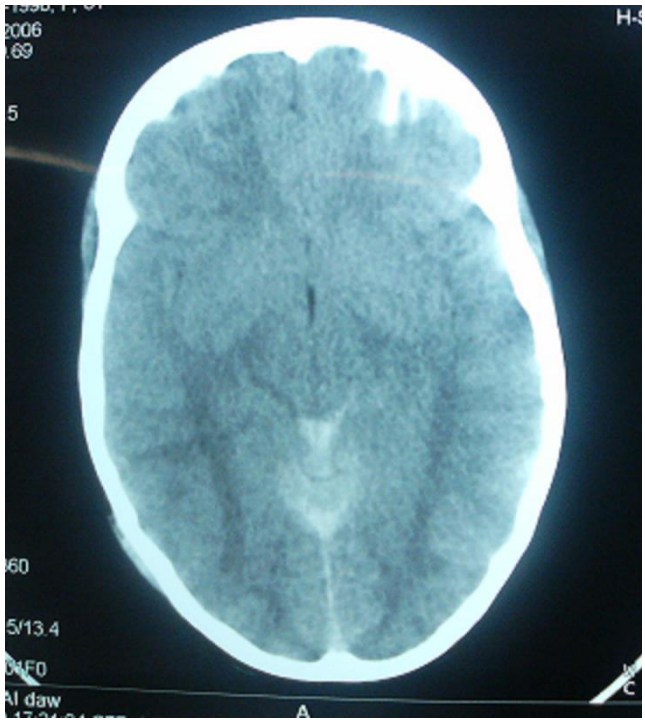

Image (2): CT of another patient with obliteration of Sulci, Ventricular system and basal cisterns around brain stem.

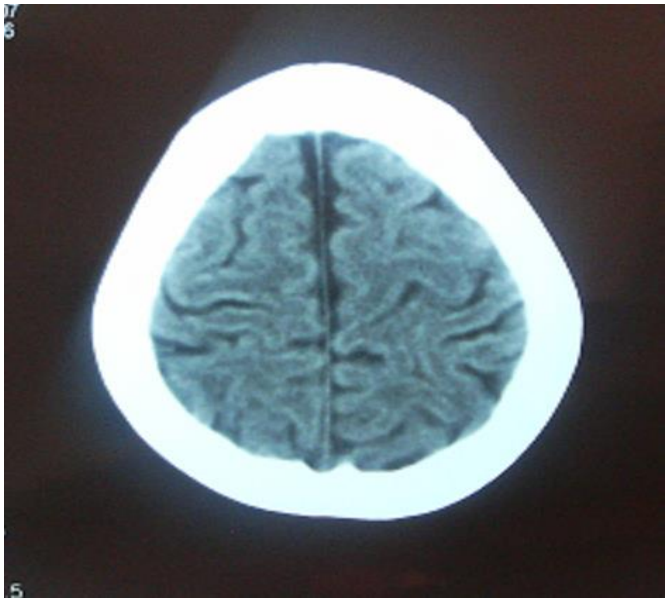

Image (3): Subsequent CT of the patient in image (2) after recovery the sulci has recurred back

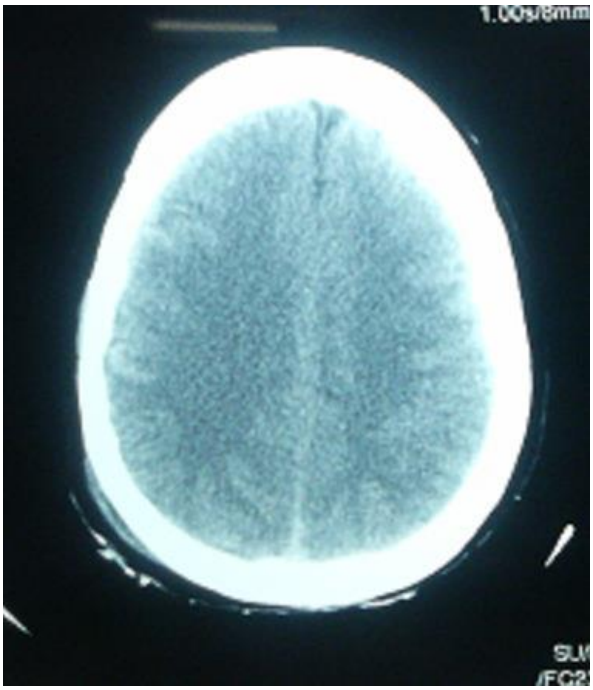

Image (4): image of another Patient showing severe brain this patient has fully recovered after the treatment.

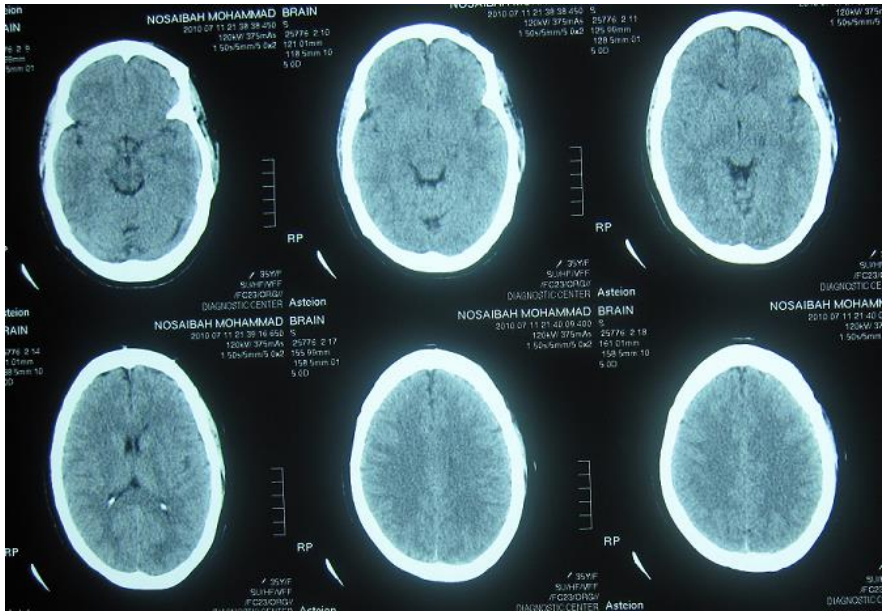

Image (5): another patient with features of ventricular obliteration and sulci effacement following head trauma.

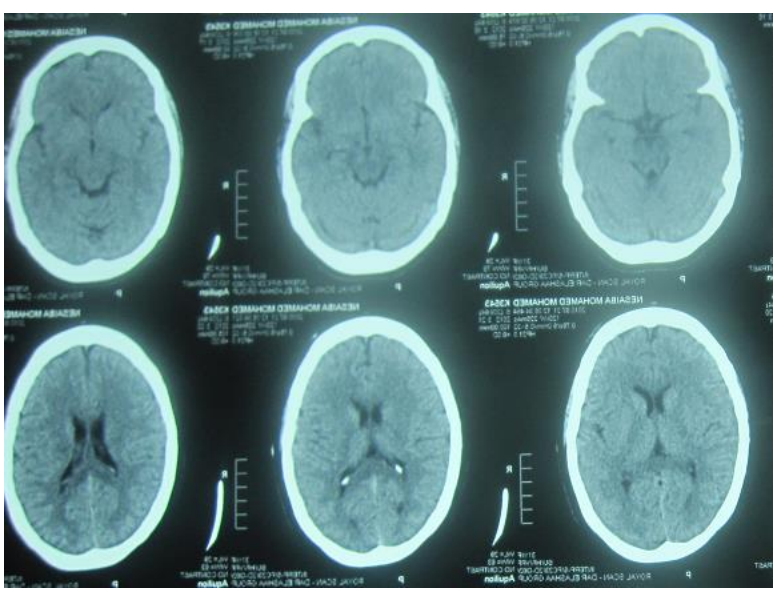

Image (6): subsequent images of the same patient in image (5) 12 days later notice appearance of ventricular system and some sulci.

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