## Original Article

ISSN (0):2395-2822; ISSN (P):2395-2814

# A Prospective Study to Evaluate Efficacy & Safety of HBOT in Acute Traumatic Brain Injury.

Bankat Phad<sup>1</sup>, Minakshi Gadahire<sup>2</sup>, Mohan Joshi<sup>3</sup>

<sup>1</sup>Assistant Professor, Department of General Surgery, Lokmanya Tilak Municipal Medical College and Hospital, Mumbai, India.

Received: November 2016 Accepted: November 2016

**Copyright:** © the author(s), publisher. Annals of International Medical and Dental Research (AIMDR) is an Official Publication of "Society for Health Care & Research Development". It is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

#### **ABSTRACT**

**Background:** The purpose of this study was to test the effectiveness and safety of Hyperbaric Oxygen HBOT group (HBOT) in improving brain function in Traumatic brain injury patients suffering acute neurocognitive impairments. **Aims:** To study efficacy & safety of hyperbaric oxygen HBOT group on mortality and morbidity in acute traumatic brain injury with respect to degree of recovery, speed of recovery, length of stay in the hospital. **Methods:** In this prospective study we present 100 cases of head injuries. Patients were included in the study according to inclusion criteria. Fifty of them assigned to the control group and 50 to the HBO treatment group. Allocation is done by chit method in control and HBOT group. Glasgow coma score was obtained pre HBOT and post HBOT. The outcome was assessed by two blinded independent examiners. **Results:** All patients were assessed for improvement in GCS score. The average improvement in GCS score was 5.29% and 3.87% in HBOT and control group respectively. Similarly the average hospital stay was 12.26% days in the HBOT and 27% in the control group. **Conclusion:** Hence we would like to conclude that, HBOT is safe and effective for acute brain injury.

Keywords: HBOT, Acute Brain Injury, Glasgow coma scale (GC Scale).

#### INTRODUCTION

Primary head injuries can cause Skull fractures, Focal injuries, Diffuse brain injuries. Skull fractures may or may not cause damage to the brain. Focal Injuries can lead to contusion or hematoma in brain diffuse brain injury is characterized histologically by widespread damage to the axons of the brainstem, parasagittal white matter of the cerebral cortex, corpus callosum, and the gray-white matter junctions of the cerebral cortex.<sup>[1]</sup> The pathophysiological changes that occur after acute brain trauma are divided in two types-primary and secondary. Primary changes that are seen is of brain tissue whereas secondary disruption changes which occurs after initial brain injury are oedema/diffuse brain swelling ,major arterial territory infarction, boundary and terminal zone injury, diffuse hypoxic injury.

## Name & Address of Corresponding Author

Dr. Minakshi Gadahire 1st floor, R No-25, College building, LTMMC & Hospital, Sion, Mumbai- 400022, Maharashtra, India.

Hyperbaric oxygen HBOT group (HBOT) is a treatment by which 100% oxygen is administered to a patient at a pressure greater than atmospheric

pressure at sea level (i.e. one atmosphere absolute, ATA). [2] HBO causes vasoconstriction and decreases edema. In this way it is quite beneficial in compression-dislocation mechanisms. It exerts a preventive and therapeutic antiedematous action, minimises interhemispheric asymmetry of brain edema in edema pathology. [3-5] Hyperbaric oxygen produces vasoconstriction, there by causing decrease in cerebral blood flow. However, when the pressure is gradually increased at a certain pressure the Central blood flow increases. [6] Inadequate oxygen supply to traumatised brain results in conversion of aerobic glucose metabolism to anaerobic leading to acidosis and depletion of energy. As the demands for energy not met, brain cells lose their ability to maintain normal ionic homeostasis. Abnormal high levels of calcium inside cells forms highly reactive free radicals which damage cell membrane when ischemia is immediate and profound. These events occur rapidly (minutes to hours) however there is evidence that ischemia can occur days after head injury. Contreras et al have documented the effect of HBO on glucose metabolism using rat model. [7]

Thus, HBOT induces a much larger oxygen-carrying capacity in the blood that dramatically increases the driving force of oxygen diffusion to tissues. Although HBOT-induced cerebral vasoconstriction appears to be undesirable within the context of ischemic conditions <sup>[8,9]</sup> this may not be necessarily

<sup>&</sup>lt;sup>2</sup>Associate Professor, Department of General Surgery, Lokmanya Tilak Municipal Medical College and Hospital, Mumbai, India.

<sup>&</sup>lt;sup>3</sup>Professor, Department of General Surgery, Lokmanya Tilak Municipal Medical College and Hospital, Mumbai, India.

## Thad et al; HBOT in Traumatic Brain Injury

deleterious due to increased oxygen availability to injured tissues. HBOT may also counter vasodilation of the capillaries within hypoxic tissues, thereby minimizing collection of extravascular fluids (edema) which ultimately reduces brain vasogenic edema and the ensuing decrease in intracranial pressure (ICP). [10, 11]

## **MATERIALS AND METHODS**

In this prospective study we present 100 cases of head injuries. 50 of them assigned to the control group and 50 to the HBO treatment group. Allocation of therapy was by chit method in control and HBOT group. The main and most important part of the study was the HBOT Chamber.

HBO administration can be done in either a Multiplace or a Monoplace chamber.

## A) Multiplace:

Large tanks accommodating 2-14 people.

Achieve pressure up-to 6 atm and have chamber lock entry system that allows personnel to pass through without altering the pressure of inner chamber.

Normal rating 1.5kva peak rating is 3.5kva. Sound earthing is required to prevent leakage of current and accidental electrocution. Recirculation consoles conditions the gas circulating in the system. It removes the co2, obnoxious odours and moisture passing through soda lime activated charcoal filter calcium sulphate respectively. Oxygen concentration is maintained around 95%. Maximum working pressure can be achieved to 3ATA. The compression and decompression pressures are semiautomatic. The operator can set up a pressure lead depending on the condition for which the HBOT group is given. Once the requisite pressure is reached the same pressure is maintained at all times. After required time, pressure in the chamber is lowered gradually over twenty minutes. In case of complication like convulsions etc; decompression is also possible. Compression and decompression are done gradually to prevent discomfort and earache.

#### Inclusion

Patients of all age and sex having traumatic brain injury with GCS scale of 8 to 12 with cerebral oedema, contusions, extradural hematoma, subdural hematoma, subarachnoid hemorrhage.

Patient with GC scale 14/15 on admission but subsequently deteriorated were also considered in the study.

# Exclusion criteria

- 1) Patients with GCS score of above 12.
- 2) Patients who were unstable and required prolonged hemodynamic ventilatory support were excluded from the study.
- 3) Patients with GCS of 8- 12, but associated with other injuries like thoracic, abdominal, long bone, spine fracture injuries.

Patients with major polytrauma were avoided as their other multi-systemic involvement would affect the results of the study. Only cases of isolated head injury were included, with only minor injuries in other systems.

After the initial examination GCS score was determined, then patients were assigned to either a control or HBOT group by picking a chit which mentioned the modality of treatment. Patient were assessed on admission for vital parameters and complete clinical examination and CT scan brain was done in all patient and reassessed after 48 hours.. The patients who were in control group were given conservative treatment with antibiotics, eptoin, mannitol and oral glycerine. Where as patients who were assigned HBOT were send for hyperbaric HBOT group for 3-5 sessions. The changes in GCS, complications, mortality were recorded.

## **RESULTS**

Out of 100 cases studied the distribution of various parameters in control and HBOT groups were as follows:

There were 52% and 40% patients in HBOT and control groups respectively. The patients were in age group of 26 to 40 years of age. The percentage of patients in extremes of ages was small, reflecting the susceptibility of various age groups of trauma. There is male predominance in both HBOT group and control groups, with respective percentage being 80% and 78%. The mode of injury was comparable in both the groups, with predominance of railway and vehicular accidents. In HBOT group they together constitute about 94% and the control group 88%.

**Table 1:** Distribution as Per the Glasgow Coma Scale.

Sr. No	GC Scale	НВОТ	Control
1	8- 10	42(84%)	40(80%)
2	10 - 12	08(16%)	10(20%)

Table 2: Outcome of HBOT

Tuble 2: Gateonic of Tibo 1:						
SR.	Parameter	CASES	CONTROL	P		
NO.	Studied			VALUE		
1.	Mean Duration	12.24	27.00	< 0.0001		
	of Hospital Stay	SD=4.94	SD=12.73			
2.	Improvement in	5.3	3.84	< 0.0001		
	GCS Score	SD=1.24	SD=1.40			

The CT brain findings of head injury was comparable in both the groups, with predominance of extradural hematoma, subdural hematoma. In HBOT group they together constitute about 62% and the control group 54%.

Both the groups were comparable in the severity of injury with the Glasgow coma scale taken as measure of severity. The maximum number of cases in both the groups were in the group of GCS score 8-10 (84%) in HBOT group and (80%) in control

## Thad et al; HBOT in Traumatic Brain Injury

group.16% in the HBOT as well as 20% in the control group belonged to the less severely injured group of GCS score 11 or more [Table 1].

The improvement in GCS score, which is taken as measure of the improvement in neurological status, is more in the HBOT group as compared to control, and is statistically significant (p<0.05) [Table 2].

The duration of hospital stay was found to be significantly reduced in the HBOT group as compared to the control group (p<0.05) [Table 2].

## **DISCUSSION**

The patients in both groups showed a predominance of young male 60% to 70% of patients in both HBOT group as well as control groups were younger than 40 years in age. There was a total male predominance in both the groups, with more than 80% of patients in both groups being males. There was a predominance of extradural hematoma and subdural hematoma in both HBOT group and control groups which consistent with previous studies. [12] The patients with any other major trauma other than head injury, including any chest or blunt abdominal trauma or major which would adversely affect the prognosis and outcome of the patient were avoided. Only patients with isolated head injury or those with single limb fractures were included in the study. The HBOT group was started after the patient were stabilised. i.e, hemodynamically stable, without ventilatory support and enteral nutrition as patients had to be shifted out of the institute for the HBOT group.

The average duration of stabilisation was about of 6 days. On an average our patients received 4.60 sittings of HBOT. Rockswald has described giving 21 sittings.<sup>[12]</sup> The study of Isakov suggest that 7 sittings are appropriate. The major focus of our study was to study the efficacy and safety of HBOT. In our study we found that the patients who were most likely to benefit from HBOT are the patients with GCS from 8-10 & patients who had cerebral oedema, small extradural hematoma, subdural hematoma and cerebral contusion and patients with these CT scan findings with no indication for surgical management.

Regarding the morbidity, the outcome measures studied were the duration of hospitalisation required and improvement in the GCS score in the patients who recovered. Bennet in study have found two trials had shown improvement in GCS of the patients of traumatic brain injury. The average hospital stay was 12.26% days in the HBOT group and 27% in the control group. This difference was found statistically significant. Similarly for improvement in the GCS score, the average in the HBOT group and control group 5.29% and 3.87% respectively. This difference is significant. The effect on morbidity is very important effect of hyperbaric oxygen as it causes improvement in neurological deficit & speeds

up the recovery, with not only the obvious economic advantages to the patients & to health-care systems, but also preventing the complications with associated factors like bed sores, infections, pneumonia which compromise the survival, as well as the quality of life. Barotrauma, pneumothorax, air embolism, neurotoxicity are rare complications of HBOT and are not seen in any of our patients.

#### **CONCLUSION**

Our study shows that improvement was far greater in patients of traumatic brain injury who did not have any operable intracranial lesion & in whom neither was Glasgow coma scale too poor nor too good so that they might have improved even otherwise. In people with traumatic brain injury, the addition of HBOT have reduced the risk of death and improved the final GCS and reduced the hospital stay.

### REFERENCES

- Meythaler JM, Peduzzi JD, Eleftheriou E, Novack TA. Current concepts: diffuse axonal injury-associated traumatic brain injury. Archives of physical medicine and rehabilitation. 2001;82(10):1461-71.
- Gill AL, Bell CN: Hyperbaric Oxygen: Its Uses, Mechanisms of Action and Outcomes. QJM. 2004, 97: 385-395.
- Rockswold SB, Rockswold GL, Vargo JM, Erickson CA, Sutton RL, Bergman TA, et al: Effects of Hyperbaric Oxygenation Therapy on Cerebral Metabolism and Intracranial Pressure in Severely Brain Injured Patients. J Neurosurg. 2001, 94: 403-411.
- Artru F, Philippon B, Gau F, Berger M, Deleuze R. Cerebral blood flow, cerebral metabolism and cerebrospinal fluid biochemistry in brain-injured patients after exposure to hyperbaric oxygen. European neurology. 1976; 14(5):351-64.
- Bennett MH, Trytko B, Jonker B. Hyperbaric oxygen therapy for the adjunctive treatment of traumatic brain injury. The Cochrane database of systematic reviews 2012;12: CD004609.
- Rockswold SB, Rockswold GL, Zaun DA, Zhang X, Cerra CE, Bergman TA, et al: A Prospective, Randomized Clinical Trial to Compare the Effect of Hyperbaric to Normobaric Hyperoxia on Cerebral Metabolism, Intracranial Pressure, and Oxygen Toxicity in Severe Traumatic Brain Injury. J Neurosurg. 2010, 112: 1080-1094
- Contreras FL, Kadekaro M, Eisenberg HM: The Effect of Hyperbaric Oxygen on Glucose Utilization in a Freeze-Traumatized Rat Brain. J Neurosurg. 1988, 68: 137-141.
- Miller JD, Ledingham IM: Reduction of Increased Intracranial Pressure. Comparison between Hyperbaric Oxygen and Hyperventilation. Arch Neurol. 1971, 24: 210-216.
- Lambertsen CJ, Dough RH, Cooper DY, Emmel GL, Loeschcke HH, Schmidt CF: Oxygen Toxicity; Effects in Man of Oxygen Inhalation at 1 and 3.5 Atmospheres Upon Blood Gas Transport, Cerebral Circulation and Cerebral Metabolism. J Appl Physiol. 1953, 5: 471-486.
- Sukoff MH, Ragatz RE: Hyperbaric Oxygenation for the Treatment of Acute Cerebral Edema. Neurosurgery. 1982, 10: 29-38.
- Kohshi K, Yokota A, Konda N, Kinoshita Y, Kajiwara H: Intracranial Pressure Responses During Hyperbaric Oxygen Therapy. Neurol Med Chir (Tokyo). 1991, 31: 575-581.
- Rockswold GL, Ford SE, Anderson DC, Bergman TA, Sherman RE. Results of a prospective randomized trial for

# Thad et al; HBOT in Traumatic Brain Injury

- treatment of severely brain-injured patients with hyperbaric oxygen. Journal of Neurosurgery. 1992; 76(6):929-34.
- Isakov Iu V, Anan'ev GV, Romasensko MV, Aide Kh B. Hyperbaric oxygenation in the acute period of craniocerebral injuries. Zhurnal nevropatologii i psikhiatrii imeni SS Korsakova. 1982; 82(5):7-12.

**How to cite this article:** Phad B, Gadahire M, Joshi M. A Prospective Study to Evaluate Efficacy & Safety of HBOT in Acute Traumatic Brain Injury. Ann. Int. Med. Den. Res. 2017; 3(1):SG20-SG23.

Source of Support: Nil, Conflict of Interest: None declared