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Rapid Progression of Hydrocephalus following Hyperbaric Oxygen Therapy for traumatic brain injury patients: An unreported adverse effect

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Abstract

Traumatic brain injury presents a major health problem with significant effect on quality of life. Hyperbaric oxygen therapy (HBOT) has been suggested to improve oxygen supply to the injured brain, this may result in a reduction in patient death and disability because of these additional brain-preserving effects. HBOT is associated with its share of complications; most of them are result of supra-normal atmospheric pressure, we would like to share our observation of few of these patient developing rapidly progressing hydrocephalus which need CSF diversion.

Keywords: Hydrocphalus; Decompressive Craniectomy; Ventriculo Peritoneal Shunt; Decompressive Craniectomy

Abbreviations

HBOT: Hyperbaric Oxygen Therapy; TBI: Traumatic Brain Injury; CBF: Cerebral Blood Flow.

Introduction

Traumatic brain injury presents a major health problem with significant effect on quality of life. Hyperbaric oxygen therapy (HBOT) improves the oxygen supply to injured brain through increased atmospheric pressure and reduces the parenchymal damage in viable but damaged brain (penumbra). It is postulated that the addition of HBOT to the standard intensive care regimen may result in a reduction in patient death and disability because of these additional brain-preserving effects. There is no class I evidence as off yet but HBOT is used as an adjunct; as a part of multidisciplinary approach. HBOT is associated with its share of complications; most of them are result of supra-normal atmospheric pressure (predominantly on ear and lungs) [1,2]. No case reports have been published on adverse or untoward effects of HBOT on brain or ventricular system (apart from seizures). We, at our institute, have been using HBOT frequently, as a part of adjunctive treatment in severe TBI cases. We want to share our experience of following four cases of operated severe traumatic brain injury (TBI). All four had undergone decompressive craniectomy for severe TBI and received HBOT as a part of adjunctive treatment. These patients deteriorated neurologically following couple of sessions of HBOT. CT brain was done which showed progression of hydrocephalus; subsequently these patients underwent Ventriculo-peritoneal Shunt which resulted in dramatic improvement.

Case Report 1

A 55 year old male was brought following history of vehicular accident, 24 hrs following injury. Patient had received primary treatment at a peripheral centre; on arrival GCS was E1V1M2 with both pupils sluggishly reacting to light. CT scan of brain showed Left Fronto-temporal subdural hematoma with significant mass effect and midline shift (Figure 1a). Patient underwent Left sided Decompressive craniectomy with evacuation of hematoma and duraplasty. Post procedure patient was electively ventilated and gradually weaned off over 5 days. Tracheostomy was done in view of need of prolong airway control. Post op CT scan done on day 5 was satisfactory, showing adequate decompression. Patient developed insignificant CSF collection beneath the flap , CT scan showed mild hydrocephalus. (Figure 1b) As per institutional policy HBOT was started with protocol as described in table 1. Patients GCS before starting HBOT was E2M5Vt, which deteriorated to E1M3Vt following 5 sessions of HBOT. Electrolytes and other parameters were checked and CT scan of brain was repeated. It showed progression of hydrocephalus with significant periventricular ooze (Figure 1c). Patient underwent ventriculopleural shunt, following which patient improved neurologically. (Figure 1d) Patient went home with GCS of E4M5Vt after receiving 15 sessions of HBOT (Table 1).

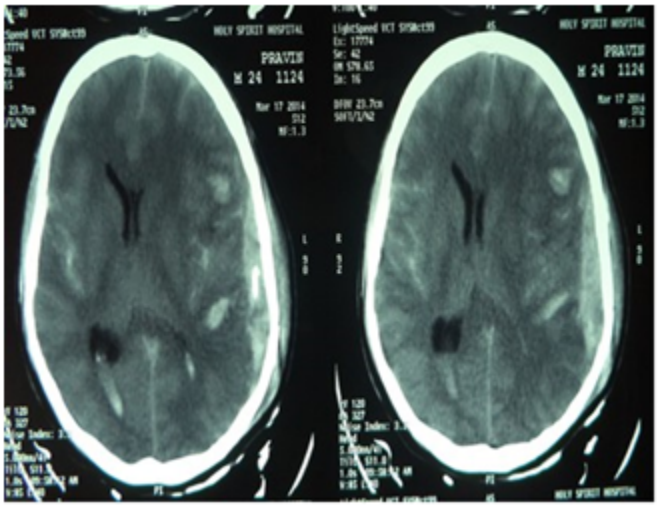

Figure 1a: CT Brain at the time of admission showing left subdural haematoma.

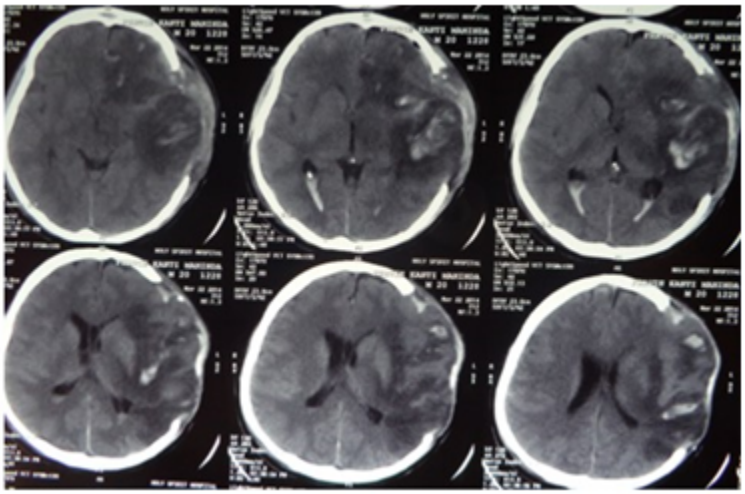

Figure 1b: Post-operative CT Brain showing decompression with brain edema.

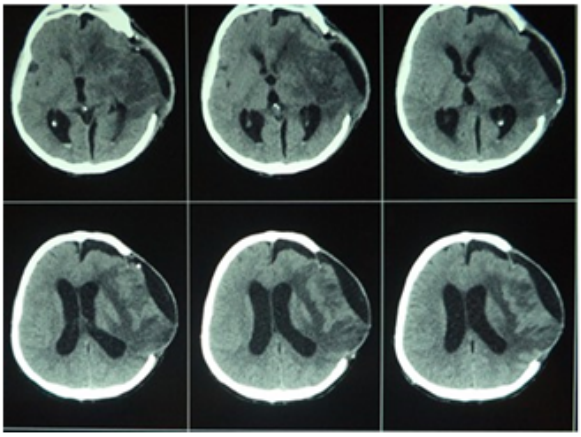

Figure 1c: CT Brain Showing mild hydrocephalus after HBOT 5 sessions.

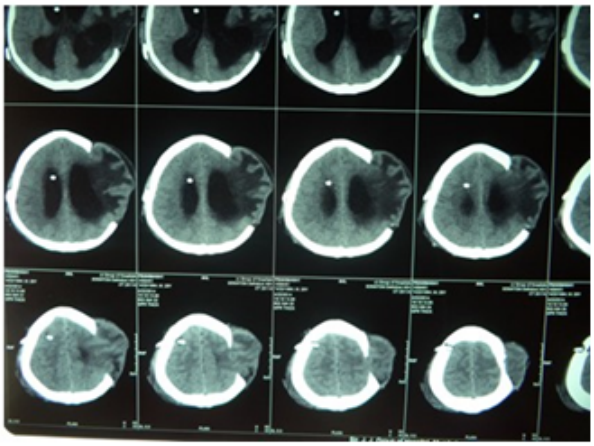

Figure 1d: CT Brain showing significant hydrocephalus after HBOT 10 sessions, post CSF diversion.

All case of severe TBI and Moderate TBI with neurological deficits are given at our institute as a protocal.	
1	100% of O2
2	Pressure used was variable
3	In most of the patients 2.4 atm pressure was used
4	10 sessions were administered and neurological status was assessed, and further 10 sessions were given based on the scan

Table 1: Protocols.

Case Report 2

A 48-year-old female was brought to the casualty with history of fall from 2-wheeler 2 days prior to admission. She was managed at a peripheral centre and was referred to us on admission was after primary management. Her GCS on admission was E1M4Vt; pupils were reacting to light bilaterally. CT brain showed large left sided frontal and right parietal contusions with mass effect and midline shift. Patient underwent left Frontotemporal Decompressive craniectomy with evacuation of contusions and duraplasty. Patient was gradually weaned off ventilator over period of 7 days. Postoperative scan was satisfactory, and showed CSF collection beneath the skin flap. Patients GCS before starting HBOT was E2M5Vt which deteriorated to E1M3VT after 2 sessions of HBOT. CT brain showed progression of hydrocephalus with periventricular ooze. Patients improved neurologically to pre-HBOT status following VP shunt; and were discharged with GCS of E4M5Vt (Figures 2a-d).

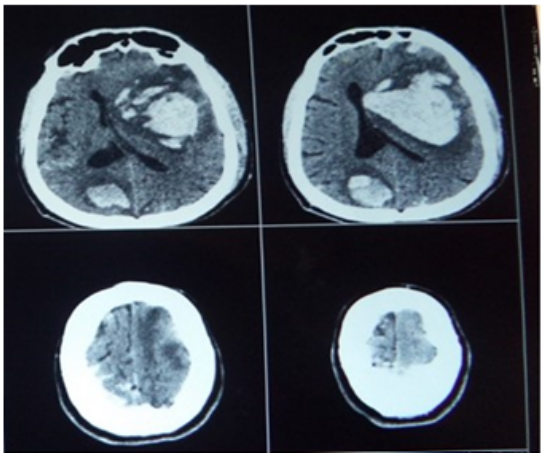

Figure 2a: CT Brain showing large left sided large frontal contusion with right parietal small contusion.

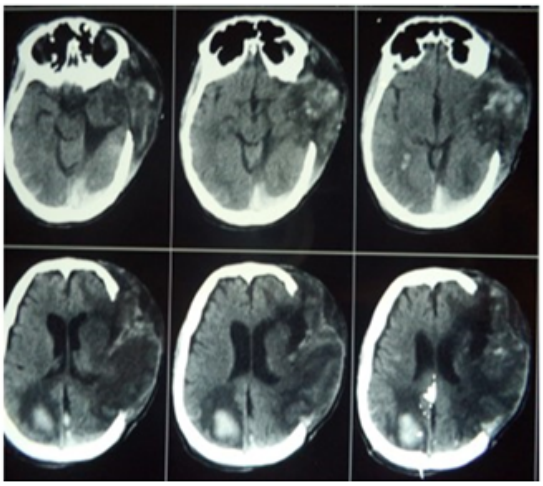

Figure 2b: Post-operative CT Brain showing adequate decompression with clot evacuation.

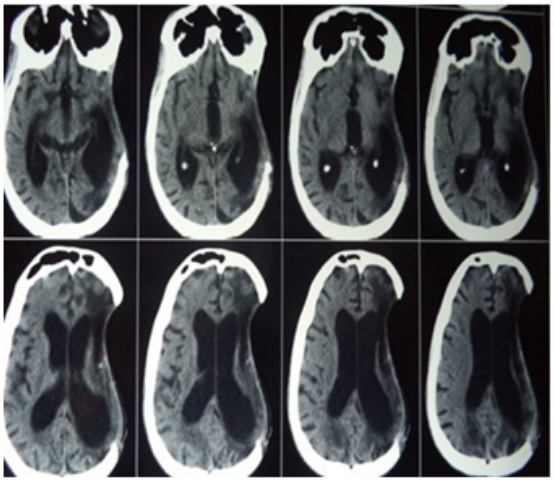

Figure 2c: CT Brain after 10 sessions of HBOT showing moderate hydrocephalus.

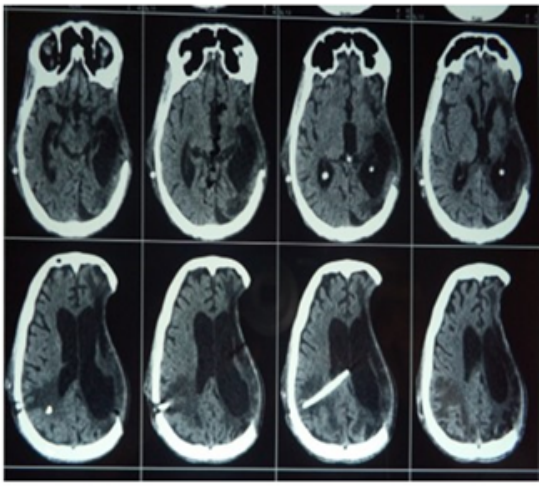

Figure 2d: CT Brain after CSF diversion (Ventriculoperitoneal shunt).

Case Report 3

A 34-year-old male was brought within 8 hours of injury, which he sustained after falling from 15 feet height. Patients GCS on admission was E1M5Vt and pupils were briskly reacting to light.

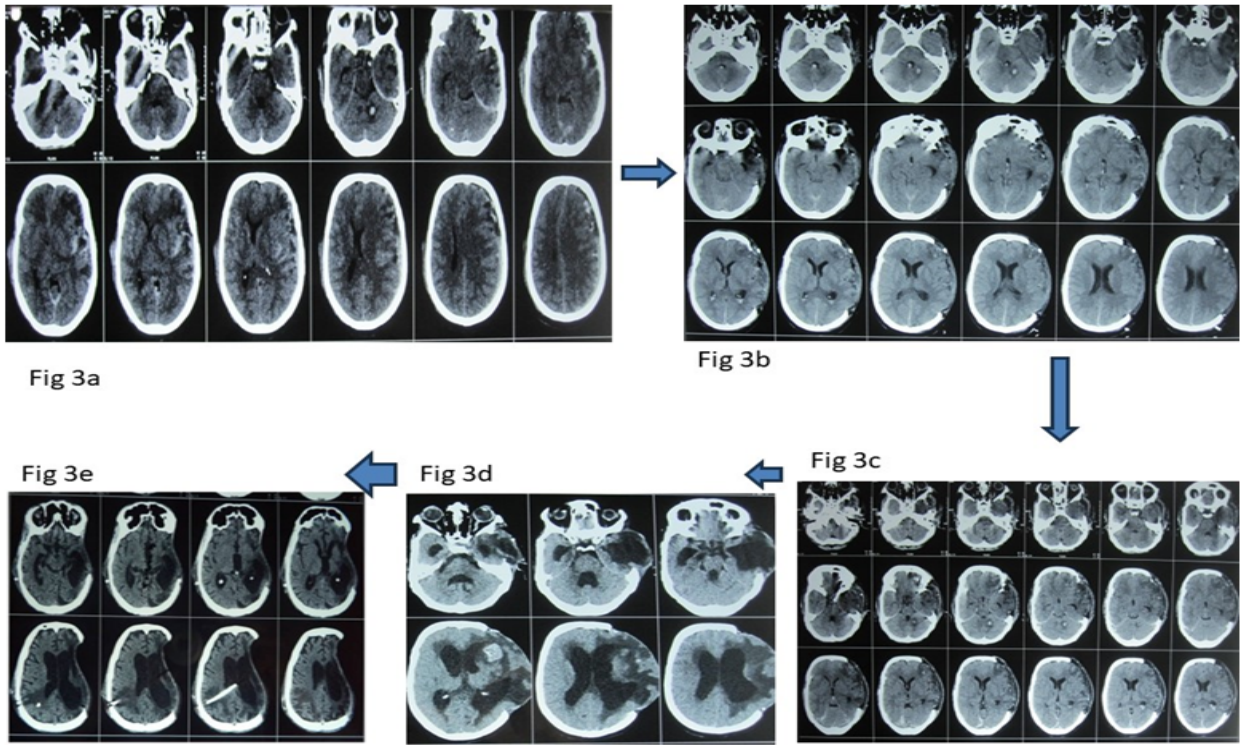

Figure 3a: CT brain showing Left sided acute SDH with SAH
Figure 3b: Post-operative CT brain showing adequate decompression
Figure 3c: Ct brain after 5 sessions of HBOT showing mild hydrocephalus
Figure 3d: CT brain showing moderate hydrocephalus after 10 sessions of HBOT
Figure 3e: CT brain after CSF diversion

CT scan of brain showed acute SDH on left side with subarachnoid hemorrhage and edema with significant mass effect and midline shift (Figure 3a). Patient underwent left side Decompressive craniectomy with duraplasty. Patient was electively ventilated for 3 days and then gradually weaned off. CT scan of brain done after surgery showed adequate decompression (Figure 3b). Patient was scheduled for HBOT after 15 days of injury. As the flap was full; CT scan was done which revealed mild ventriculomegaly (Figure 3c). HBOT was continued as scheduled. Patient's GCS deteriorated to E1M5 VT which was E3M5VT before starting HBOT. CT scan was repeated which showed progression of hydrocephalus with periventricular ooze (Figure 3d). Patient underwent VP shunt following which patient improved; patient was subsequently discharged after giving 10 sessions of HBOT with GCS of E4M6VT (Figure 3e).

Case 4

Another 56-year-old male was brought with history of vehicular accident about 18 hours post injury, GCS of E1M3V1 and pupils bilaterally reacting to light. CT scan of brain revealed Left frontotemporoparietal acute SDH with mass effect. Patient underwent Left side Decompressive craniectomy and was gradually weaned off over 8 days period Check CT scan was satisfactory apart from mild ventriculomegaly. Following 3 sessions of HBOT patients GCS deteriorated to E1M4VT from E2M5Vt. CT brain showed moderate hydrocephalus, for which V P shunt was done. Patient improved following surgery and was discharged with coma scale of E4M5Vt.

Discussion

"Man can survive without food for weeks and without water for days, but only minutes without oxygen. Oxygen is the basis of life"

The concept of hyperbaric oxygenation has been in driven by this statement as early as 1662 but has only gained recognition in conventional medicine over the past 40 years. Hyperbaric Oxygen Therapy (HBOT) delivers oxygen under increased pressure compared to atmospheric pressure thus increasing the absorption. This increased pressure allows more oxygen to reach the cells within the body therefore contributing to the many healing and therapeutic benefits. This principle is based on Henrys law which states that:

- **Henry's Law**
- The amount of gas dissolved in a liquid is directly proportional to its partial pressure at the liquid/gas interface.
- Increasing the partial pressure of oxygen results in more oxygen dissolved in the blood
- Blood oxygen carriage therefore increases, in addition to saturation of available hemoglobin

In acute TBI, hypoxia and hypotension are independent risk factors associated with increased mortality and morbidity1. Thus, secondary ischemia and oxygen deficiency are thought to be important mechanisms of cell death in TBI. Aggressive management of trauma is found to significantly reduces the hypoxic and ischemic episodes and ultimately affect the outcome; this forms the basis of the proposed golden hour. Since the primary insult is sometimes significant enough, there is renewed interest in finding more effective strategies for ensuring adequate oxygenation and redistributing cerebral blood flow (CBF) to injured areas of the brain. Immediately after a brain injury, brain cells can be inactivated temporarily by ischemia and edema which compromise local perfusion. This observation forms part of the rationale for the use of HBOT, which increases blood flow to the damaged areas enriched with oxygen. Adverse events can occur during compression and decompression, and are related to the increased pressure and the increased oxygen concentration. Complications such as pulmonary Barotrauma or seizures can occur immediately, but more subtle adverse effects may emerge after a series of treatments Table 2 Adverse effects of HBOT.

Delivery of O2 to hypoperfused tissued	
1	Limit ischemic damage, cell death, and inflammation
2	Promotes collagen synthesis and angiogenesis
3	Decreases lactate production and tissue acidosis
Generation of oxygen free radicals	
1	Aids in oxygen-dependent killing of antibiotica
2	Facilitates oxygen dependent transport of antibiotics
Vasoconstriction	
1	Limits leukocyte adhesion and de-granulation
2	Decreases tissue edema

Table 2: Effects of HBOT.

Most of the side effects of HBOT have been attributed to delivery of oxygen at increased pressure; lungs and ear bear the brunt, whereas its effect on brain has not been explained in literature. All the patients described in this article were middle to old age (mean age- 48 yrs) and had undergone Decompressive craniectomy for severe TBI. Patients were given HBOT as part of adjunctive treatment. What is of importance was that all of them had mild hydrocephalus on postoperative CT scan, with presence of subgaleal CSF collection in 3 patients. We postulate that with increased atmospheric pressure the CSF from pseudo-meningocele is driven into the ventricle, and further drainage of CSF from

ventricles is also hampered. To add to this, the CSF absorption from the subarachnoid spaces is also hampered due to loss of drive across the arachnoid granulation, which results from increased pressure during HBOT. All four patients improved with ventriculo peritoneal shunt.

Decompressive craniectomy itself is associated with development of hydrocephalus, but usually not an acute hydrocephalus unless there is an associated intraventricular bleed. Its development is a result of alterations in CSF dynamics, which occurs over a period. In 2021, a total 298 cases of severe TBI were operated in our institute, all of them underwent decompressive craniotomy. Out of these 27 cases developed hydrocephalus. (Table 3) A total of 1876 HBOT sessions were given in 2021, out of which 209 cases were of severe TBI. Average duration between development of hydrocephalus from the day of surgery was 26 days, where as in cases that were given HBOT, there was development of acute hydrocephalus in approximately 2 days. These statistics point out to the contribution of HBOT in acute progression of hydrocephalus [2-4].

Conclusion

Administration of HBOT may lead to acute progression of hydrocephalus in patients with mild hydrocephalus and subgaleal collection; a word of caution is advised. A pre-HBOT CT scan should be done to rule out borderline or mild hydrocephalus in such patients. More prospective studies are required for further interpretation.

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