RESEARCH ARTICLE

Pneumocephalus as complication from hyperbaric oxygen treatment

Lily Xiao Jing Sun, Andrew Nataraj

ABSTRACT

Introduction: Hyperbaric oxygen treatment (HBOT) is a therapy in which a patient breathes 100% oxygen intermittently while inside a treatment chamber at a pressure higher than at sea level. Barotrauma is the most common cause of side effects with HBOT. In this case report, we discuss the rare presentation of pneumocephalus following HBOT.

Case Report: The patient is a 32-year-old woman with congenital hydrocephalus and an extensive history of shunt revisions, as well as a third ventriculostomy. The patient received two weeks of HBOT given her history of impaired wound healing and continuation of poor skin healing after a shunt revision surgery. This is the first report of pneumoventricle in the context of a non-healing ventriculoperitoneal (VP) shunt wound as a complication of HBOT.

Conclusion: Although pneumocephalus after HBOT is a very rare complication, careful evaluation of the patient is needed prior to initiating HBOT. Increased awareness of this potential complication of HBOT can aid in the decision-making of whether to expose patients with non-healing VP shunt incisions to this therapy.

Keywords: Hyperbaric oxygen, Pneumocephalus, Ventriculoperitoneal shunt

How to cite this article

Sun LXJ, Nataraj A. Pneumocephalus as complication from hyperbaric oxygen treatment. Edorium J Surg 2021;8:100052S05LS2021.

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Received: 22 March 2021 Accepted: 30 April 2021 Published: 03 June 2021 Article ID: 100052S05LS2021

doi: 10.5348/100052S05LS2021CR

INTRODUCTION

Hyperbaric oxygen treatment (HBOT) is a therapy in which a patient breathes 100% oxygen intermittently while inside a treatment chamber at a pressure higher than at sea level pressure (i.e., 0.1 atm) [1]. Indications of HBOT include carbon monoxide poisoning, decompression sickness, arterial gas embolism, radiation-induced tissue injury, acute traumatic ischemic injury, compromised skin grafts and flaps, anemia due to exceptional blood loss, thermal burns, clostridial myonecrosis, necrotizing fasciitis, and refractory osteomyelitis [2]. Ischemia is the most common cause of non-healing infected wounds [3]. Ischemia not only leads to hypoxia but also compromises antibiotic delivery [4]. Primary effects of hyperbaric oxygen treatment are the result of increased tissue oxygen partial pressure. Secondary effects are the result of a controlled oxidative stress producing reactive oxygen species and reactive nitrogen species involved in wound healing, improved leukocyte function and neovascularization [5].

The most common side effects of HBOT is barotrauma explained by pressure equalization problems causing middle ear pain, tooth pain, and cranial/paranasal sinus pain [6]. Other side effects include cranial nervous system oxygen toxicity seizure, hyperoxic myopia, cataracts, pulmonary edema, increase in blood pressure, decrease in pulmonary function, and risk of air trapping in patients with asthma or chronic obstructive pulmonary disease (COPD) [5]. Here, we discuss the rare presentation of pneumocephalus following HBOT for treatment of recurrent postoperative wound infection.

CASE REPORT

The patient is a 32-year-old woman with congenital hydrocephalus and an extensive history of shunt

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revisions, as well as a third ventriculostomy. Most were obstructions complicated by long-standing nonhealing wounds postoperatively. She had a recurrent history of wound dehiscence and leaking of pus over the incision line as well as wound break down exposing the ventriculoperitoneal (VP) shunt and valve. In the past, one month of HBOT had been used for a non-healing VP shunt wound with some success. Her other relevant past medical history includes epilepsy treated with frontal lobe resection and left temporal lobectomy, complete heart block, and postoperative infections including multiple pacemaker infections.

Three months after receiving antibiotics for right parietotemporal osteomyelitis, the patient presented with acute confusion, fever, and headache. Bilateral shunts were removed and a left posterior external ventricular drain (EVD) was inserted after a VP shunt tap showed infection with Escherichia coli. After removal of the EVD, the patient developed symptoms of increased intracranial pressure (ICP). A left frontal EVD was then inserted and ICP was estimated to be 30 cm H₂O. One week later, a VP shunt was inserted through the right frontal area. Because of persistent ventricular dilation, on postoperative day 8 of the VP shunt insertion, revision of the shunt with partial septum pellucidum ostomy was performed using ventriculoscopic guidance. Given her history of impaired wound healing and continuation of poor skin healing, HBOT was started and continued after this operation for two weeks.

The patient was neurologically stable for the next two months until she presented with decreased level of consciousness (LOC), headache, and recurrent episodes of clonic and myoclonic jerking of the upper extremities. There was a significant amount of air distending the right lateral ventricle with midline shift and a hemorrhage involving the left frontal temporal region (Figure 1). The initial shunt tap drew 15 mL of air and the ventricles decreased in size. The shunt was later tapped two more times and no organisms grew. Air in the right ventricle resolved over the next 10 days, but the ventricles kept on dilating requiring a VP shunt revision. After this revision and over the following week, there were two more shunt obstructions requiring revisions. After a new VP shunt system was inserted, one day postoperatively, the right temporal horn became trapped (Figure 2). A right temporal shunt catheter was inserted for the isolated right temporal horn hydrocephalus for decompression (Figure 3A and B).

One week after the insertion of the temporal shunt catheter, the patient deteriorated neurologically to a Glasgow Coma Scale (GCS) of 6, associated with another slight enlargement of the temporal horn of the right lateral ventricle. Both the right-sided frontal and temporal ventricular shunt were externalized, and the patient improved. While the drains were clamped awaiting further shunt revision, there was no subsequent increase in ventricular size. The patient's shunt was removed 12 days after the shunt was externalized and clamped. The patient was discharged after three days of the removal of the shunt. Despite clamping and removing the externalized shunts, her ventricles did not enlarge, nor did she develop symptoms. Ultimately, she stayed stable and was no longer shunt dependent. At fouryear follow-up, the patient's symptoms remained stable with no shunt. In summary, the patient had nine shunt revisions before presentation and seven shunt revisions after presentation.



Figure 1: First head CT after two weeks of HBOT showing large amount of air distending the right lateral ventricle including the temporal horn and causing midline shift. Increased attenuation showed recurrent hemorrhage within the extra-axial collection involving the left frontal temporal region.

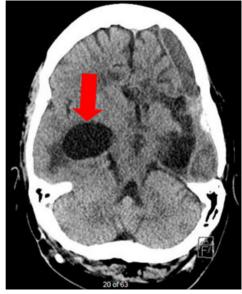


Figure 2: Entrapment of temporal horn of the right lateral ventricle causing midline shift to the left with subacute on chronic left-sided subdural hematoma on postoperative day one of new VP shunt.

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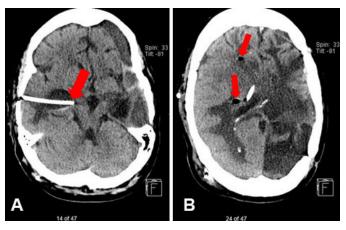


Figure 3: (A) Head CT showing a new shunt catheter was placed through the right temporal bone and extended into the temporal horn of the right lateral ventricle for decompression. (B) There were small amounts of gas and blood within the temporal horn. The right-sided frontal ventricular shunt is still present.

DISCUSSION

The large amount of air distending the right ventricle with midline shift causing decreased LOC and seizure is a possible complication from HBOT. Tension pneumocephalus has been described in the past as a rare complication from HBOT, but this is the first report of air becoming entrapped in the ventricular system as a possible consequence of HBOT of which we are aware [7, 8]. The mechanism is thought to be related to the cyclical pressure change with initiation and completion of each HBOT session [7]. While undergoing HBOT, the patient is in a high-pressure environment creating a negative pressure in the intracranial space causing air to enter the intracranial space through dural and skull defects. Once the patient returns back to atmospheric pressure, intracranial air volume expands to push out cerebrospinal fluid (CSF) through the structural defects [7]. A greater negative pressure will develop intracranially in subsequent sessions of HBOT as the existing intracranial air is compressed during HBOT allowing more air to enter [7]. The cycle repeats itself leading to the buildup of air intracranially. Given the patient's history of multiple shunt insertions and poor wound healing, air likely has reached the ventricle through the tract created by the catheter and postoperative wounds [9].

Another possible but less likely cause of pneumocephalus in this patient includes CSF leak from her multiple neurosurgical operations. Presence of a cranio-dural defect and diversion of CSF are the two requirements for pneumocephalus to occur [10]. Infection with gas-producing organisms can also cause pneumocephalus but unlikely to be the culprit for this patient. No organisms grew from the shunt tap along with normal CSF protein, glucose, and low white blood cell count. In addition, no organisms grew on the catheter specimen. Blood work and blood culture also did not show any signs of infection for this patient during the large accumulation of ventricular air.

Entrapment of the right temporal horn is another interesting presentation. Isolated dilatation of the temporal horn is an extremely rare pathologic condition [11]. There have only been 72 reported cases of temporal horn entrapment from 1947 to 2017 [12]. Possible causes include central nervous system infection, hemorrhage, intra- and extra-axial tumors, postoperative scarring within the trigone and trauma [13, 14]. For the patient in this study, her temporal horn entrapment may be related to the decompression of the large amounts of intraventricular air distending the right ventricle creating a seal in the trigone.

Unexpectedly, the patient became shunt independent after years of VP shunt insertion and revision. One possible explanation is the temporal shunt catheter that was inserted more medially than intended for decompression of the temporal horn entrapment. The temporal shunt catheter was approaching immediately adjacent to the interpeduncular cistern on imaging. A ventriculocisternostomy may have been performed unintentionally leading to the resolution of shunt dependence. Another explanation may be the re-opening of her previous third ventriculostomy with the high intracranial pressure from shunt obstructions.

CONCLUSION

Hyperbaric oxygen treatment is an alternative treatment in neurosurgery for postoperative wound infections. Although pneumocephalus after HBOT is a very rare complication, careful evaluation of the patient is needed prior to initiating HBOT. This is the first report of pneumoventricle in the context of a non-healing VP shunt wound. Increased awareness of this potential complication of HBOT can aid in the decision-making of whether to expose patients with non-healing VP shunt incisions to this therapy.

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Author Contributions

Lily Xiao Jing Sun – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically

for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Andrew Nataraj – Design of the work, Interpretation of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Guarantor of Submission

The corresponding author is the guarantor of submission.

Source of Support

None.

Consent Statement

Written informed consent was obtained from the patient for publication of this article.

Conflict of Interest

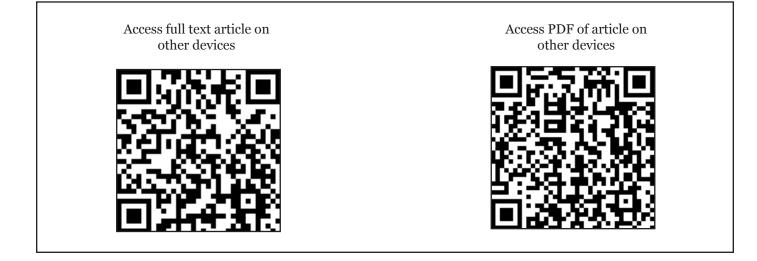
Authors declare no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.

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