Hyperbaric oxygen ameliorates worsening signs and symptoms of post-traumatic stress disorder

Benjamin Eovaldi¹ and Claude Zanetti²

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Abstract

Hyperbaric oxygen therapy at 2.4 atmospheric pressure absolutes for 90 minutes per day ameliorated the signs and symptoms of agitation, confusion, and emotional distress in a 27-year-old male seven days following a traumatic accident. Hyperbaric oxygen was used to treat the patient’s crush injury and underlying nondisplaced pelvic fractures which were sustained in a bicycle versus automobile traffic accident. Its effect on the patient’s neuropsychiatric symptoms was surprising and obvious immediately following the initial hyperbaric oxygen treatment. Complete cognitive and psychiatric recovery was achieved by the seventh and final hyperbaric oxygen treatment. We propose that hyperbaric oxygen was effective in improving the patient’s neuropsychiatric symptoms by reducing cerebral oxidative stress, inflammation, vasogenic edema, and hippocampal neuronal apoptosis. Further investigation into the use of hyperbaric oxygen as a novel therapy for the secondary prevention of post-traumatic stress disorder that often accompanies post-concussive syndrome may be warranted. We acknowledge that hyperbaric oxygen therapy has been shown to have a strong placebo effect on neurologic and psychiatric diseases.

Keywords: hyperbaric oxygen, post-traumatic stress disorder, neuropsychiatric, acute stress disorder
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Introduction

Post-traumatic stress disorder (PTSD) is a global problem that continues to grow as our understanding and use of simple explosive chemistry becomes more prevalent. The general population is now beginning to view PTSD as more of a public health concern, largely because the media has increased their focus on the impact that PTSD has on military personnel returning to the US from service in Iraq. In the civilian population, PTSD following motor vehicle accidents, the most common cause of PTSD, is less likely to be recognized by the general population and medical community than PTSD caused by exposure to military combat.¹,² Research and evidence related to the pathogenesis of PTSD suggests that the dysfunction is not merely present in the mind, but that organic changes in the hippocampus and limbic system are also involved.²-⁴ Humans are regularly exposed to painful or difficult situations, and
manageable stress is considered to play a positive role in our lives. However, uncontrollable stress is the primary etiologic factor in the development of acute stress disorder and PTSD.\textsuperscript{2}

Acute stress disorder and PTSD are the same constellation of symptoms involving intense fear, sleep abnormalities, behavioral changes, and intrusive thought processes that may develop following exposure to a life-threatening event. The duration of disturbance must be between two days and four weeks in acute stress disorder and greater than one month in acute PTSD as per the \textit{Diagnostic and Statistical Manual of Mental Disorders}, fourth edition (DSM-IV) criteria. Because PTSD has been shown to be particularly difficult to treat and has a profound negative impact on the affected patient’s quality of life, it is important to recognize the signs and symptoms early in at-risk patients in order to attempt to stop progression from acute stress disorder to PTSD. Several studies have shown hyperbaric oxygen therapy to be effective in preventing or treating acute psychiatric disturbances that are associated with stress and disease.\textsuperscript{4,5}

We suggest that it is reasonable to investigate if hyperbaric oxygen therapy is an effective modality for treating acute stress disorder and if it may serve potentially as secondary prevention of PTSD in patients who have experienced life-threatening trauma resulting in crush injuries because these victims of trauma are at increased risk for developing PTSD, and hyperbaric oxygen is an indicated therapy for their crush injuries.\textsuperscript{6,7} Hyperbaric oxygen therapy has been used with varying degrees of success in treating chronic psychiatric and neurologic diseases and is currently being investigated for the potential treatment of autism, multiple sclerosis, and chronic traumatic brain injury.\textsuperscript{8} Acute neurologic and psychiatric disturbances such as acute traumatic brain injury, lacunar infarcts, acute stress disorder, and acute PTSD may respond better than chronic neurologic and psychiatric conditions to hyperbaric oxygen therapy.

Currently eight of the 13 disorders that hyperbaric oxygen is approved for treating are acute conditions.\textsuperscript{6,9–12} There is strong evidence that hyperbaric oxygen therapy decreases vasogenic edema and inflammation in soft tissue injuries.\textsuperscript{6} The mechanisms involved for hyperbaric oxygen treating soft tissue injuries are related to the extreme elevations in tissue oxygen tensions generated under supra-atmospheric oxygen pressures and are discussed further in this report. Because the brain is an electrical organ and its functional networks and supportive structures are made up of different cells than soft tissues, the mechanisms involved in treating nervous tissue with hyperbaric oxygen are probably also different but may act similarly.\textsuperscript{13,14}

The pharmacologic mechanisms of hyperbaric oxygen therapy appear to work against more than one pathophysiologic process. We propose that the overall therapeutic effect of hyperbaric oxygen therapy on acute traumatic neuropsychiatric dysfunction is to reduce vasogenic edema and limit hippocampal dysfunction.\textsuperscript{20–24} Following traumatic neurologic injury, it is important to achieve optimal clearing of the proinflammatory cytokine storm associated with acute central nervous system injury. It is well known that reducing cerebral vasogenic edema improves neurologic outcomes following trauma. Oxidative stress and inflammation together disturb laminar blood flow by altering macrophages and T lymphocyte function which results in cell accumulation and adherence to the endothelium.\textsuperscript{6,7,11,12} Hyperbaric oxygen therapy generates tremendous tissue oxygen tensions which reverse in vivo electrochemistry to a reductive state and has been shown to prevent cerebral lipid peroxidation following carbon monoxide
exposure. The anti-inflammatory effects of hyperbaric oxygen may involve reduction of glutathione and suppression of tumor necrosis factor-α, interleukin-6, and tissue myeloperoxidase.

Damage to the hippocampus and limbic system appears to be important in the development of PTSD, the postconcussive syndrome, and postdepressive syndromes. The hippocampus is uniquely positioned among the vascular territories of the posterior cerebral artery, anterior cerebral artery, anterior choroidal artery (a branch of the internal carotid artery), and penetrating branches of the middle cerebral artery. The rich collateral blood flow to the hippocampus and limbic region is a therapeutic target. Damage to the surrounding microvasculature and endothelium in the injured tissue increases capillary permeability and displaces fluid to the interstitial space. Increased interstitial osmotic pressures force fluid into surrounding cells which results in concomitant intracellular edema. Intra- and extracellular edema together promote hypoxia, cell death, and tissue ischemia. Hyperbaric oxygen therapy counteracts the vasogenic edema complex of edema, hypoxia, and vasodilation by acting as a robust vasoconstrictor. Vasoconstriction reduces blood flow to edematous tissue and effectively reverses the hydrostatic pressure gradient to favor lymphatic and microvasculature fluid reabsorption. Reducing vasogenic edema improves venous outflow and ultimately restores functional vascular flow.

Damage to the hippocampus has been repeatedly implicated as a causal factor in the development of PTSD, the postconcussive syndrome, and postdepressive syndromes. PTSD is associated with hippocampal atrophy in humans. A proposed mechanism for the hippocampal dysfunction involves the activation of mitochondrial apoptotic pathways mediated via pathophysiologic crosstalk elements, ie, reactive oxygen species, cytochrome c, and caspases. Exposing rats to a single prolonged stress, a proposed method for inducing PTSD in animals, reproducibly kills hippocampal neurons via apoptosis. Preconditioning rats with induced PTSD using hyperbaric oxygen prevented the loss of hippocampal neurons by inhibiting mitochondrial apoptotic pathways. Hyperbaric oxygen preconditioning also improved the animals’ neurologic and behavioral outcomes. Preconditioning with hyperbaric oxygen has also been shown to reduce postpump depression significantly following cardiopulmonary bypass. Directing therapy to serve as secondary prevention of PTSD for at-risk patients or patients with early cognitive and psychiatric disturbances following trauma may be useful. There is recent evidence suggesting that the use of morphine in acute trauma care may reduce the risk of developing PTSD following serious injury by blocking memory consolidation mediated via adrenergic pathways.

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Conclusion

Several other case studies have been reported in which hyperbaric oxygen therapy resulted in a dramatic reduction or abolition of psychiatric symptoms in individuals suffering from postconcussive syndrome with and without associated PTSD features. Remarkably, hyperbaric oxygen therapy was effective in eliminating psychiatric symptoms when initiated six months to three years post-trauma. We report the first case of hyperbaric oxygen ameliorating the signs and symptoms of acute stress disorder following a life-threatening traumatic injury. We
suggest that hyperbaric oxygen therapy was effective in improving our patient’s acute psychiatric disturbance by effectively counteracting the pathologic processes of oxidative stress, acute inflammation, vasogenic edema, and hippocampal neuronal apoptosis. Our case may support further investigation into the use of hyperbaric oxygen therapy as a novel treatment for the secondary prevention of PTSD and postconcussive syndromes.29,32

Footnotes

Disclosure

The authors report no conflicts of interest in this work.

References


