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Hyperbaric oxygen therapy in post-stroke patients

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Abstract

Stroke is regarded as the major cause of brain damage, chronic neurological impairments, functional deficits, and long-term disability worldwide. Hyperbaric oxygen therapy (HBOT) might be taken into consideration as relatively novel method for brain repair. This article aims at analysis the extent to which the available ooprtunities are being exploited, including limitations associated with therapeutic use of HBOT in the therapy of post-stroke patients. Despite HBOT constitutes promising method in post-stroke patients further research is needed to better define the role of HBOT in the therapy of post-stroke patients.

Introduction

Stroke is regarded as the major cause of brain damage, chronic neurological impairments, functional deficits, and long-term disability worldwide. There is lack of agreement concerning effective intervention for stroke patients with chronic neurological deficit. Recovery may persist for years, but some damages may be orreversible. Thus current research focus on methods salvaging and protecting brain tissue. Researchers and clinicians are open to novel ways of treatment and rehabilitation providing quicker restoration of function. Hyperbaric oxygen therapy (HBOT) might be taken into consideration as relatively novel method for brain repair.

This article aims at analysis the extent to which the available ooprtunities are being exploited, including limitations associated with therapeutic use of HBOT in the therapy of post-stroke patients.

HBOT theory and practice

HBOT refers to the medical use of 100% oxygen at a level higher than atmospheric pressure ($\geq$ 1.4 atmospheres absolute pressure) [1, 2, 3, 4, 5]). HBOT increases the delivery of oxygen to damaged local tissues, stimulates angiogenesis, immune response, collagen synthesis, and stem cell migration, this way accelerating healing of damaged tissues [4, 5]. Although pathophysiology of HBOT is relatively clear its clinical application is still under research [1, 2, 3, 4, 5]. HBOT has two basic mechanisms of action: hyperoxygenation and decrease in bubble size. But whole protective effect is more complex. In damaged tissue cellular metabolism and cell survival may be improved, inflammation and swelling may be decreased – as a result cell death may be postponed and reduced.

Clinical applications of HBOT in post-stroke patients

Role of the HBOT in the therapy of post-stroke patients is multidimensional. We should take into consideration at least following factors:

- location, size and dynamics of the damage (rapid rate of injury progression),
- time after damage,
- response of brain to damage,
- possibilities of natural recovery processes,
- potential of brain plasticity,
- associated interventions (anticoagulation and antiplatelet),
- inclusion and exclusion criteria,
- optimal timing of HBOT,
- dosage of HBOT,
- prognostic factors and future directions (e.g. way of the further therapy),
- prevalence (70-80%) of ischemic stroke caused by impairment of blood flow to the brain, reduction of oxygen delivery and cell death [6].

In very early stage single HBOT intervention immediately after temporary middle cerebral artery occlusion (MCAO) in mouse followed by 24 hours reperfusion may significantly decrease edema and improve perfusion [7]. HBOT may increase oxygen delivery reducing cell death i.e. decreasing brain swelling and volume of brain that may die. Aforementioned reduction can influence better outcomes of the therapy. HBOT up to 3 hours after ischemia may stabilize CREB and reduce infarct size in rats [8, 9]. Despite weak evidences possibility of positive influence to clinical outcomes is not excluded [10]. Lin et al. described a case of air embolism resulting in cerebral infarction related to angioinvasive cavitary aspergillosis.

Emergent HBOT improved clinical status of the patient [11]. Improvement of function and life quality in chronic post-stroke patients following HBOT were conformed by CT and SPECT medical imaging. True may be an assumption that HBOT may activate important mechanisms of brain plasticity in such patients, even after long term lack of activation in selected brain areas [12]. This evidence, if confirmed, may change course of short-, middle-, and long-term neurorehabilitation where exercise-induced neuroplasticity seem be basis for functional recovery in patients after stroke. Adding of HBOT-induced neuroplasticity may increase functional outcomes and shorten period of the rehabilitation (including both hospital stay and outpatient phase of the therapy). Studies by Mu showed another benefit: HBOT may influence synthesis and degradation of hormones, e.g. leptin [8, 9]. Study by Chen et al. showed (long-term) HBOT as effective and safe adjunctive therapeutic method for patients with acute ischemic stroke [13, 14]. There is need to investigate influence of HBOT features to results of the neural restoration. Bigger dose of HBOT proved high efficiency in reducing infarct volume and improving outcome in permanent middle cerebral artery occlusion (MCAO) in rats in early stage of stroke, but longer duration of HBOT do not [15].

We should be conscious possible limitations e.g. diabetic foot patients with coronary artery diseases or stroke and non-proliferative or proliferative retinopathy might resist HBOT [16]. HBOT, if necessary, may also decrease blood glucose level in patients with diabetes mellitus, traumatic brain injury or stroke thus this factor should be monitored is such cases [17]. We should take into consideration that patients after stroke, especially elderly, may show many complications or co-occured diseases significantly influencing possible way of the therapy. Current therapies for ischemic stroke such as thrombolysis with recombinant tissue plasminogen activator also have their own limitations [18] – HBOT may be feasible is such patients. HBOT combined with thrombolysis provides neuroprotection in acute ischemic stroke in rats by decreasing volume of infarct [18].

Discussion

Despite studies and analyses role of HBOT in the treatment of post-stroke patients is still controversial. Current evidences are insufficient to provide clear guidelines for HBOT use in post-stroke patients. Some effects of HBOT has been studied only in animal models of stroke. But clinical application of HBOT is still in the beginning of its development and need continuous effort of scientists and clinicians.

Basic studies should focus on healing mechanism of HBOT and associated occupations. Dramatic increase of oxygen level within body tissues may not only spare and maintain damaged brain areas, but also (temporary) remove problems in blood flow, inhibit selected bacterias, etc.

Brain sparing effect of single treatment of HBOT applied immediately after stroke onset is rather clear but it needs further research as adjunctive therapy in cases when “traditional” methods fail. There is also need to explain if HBOT combined with other therapies may extend the window of treatment with tPA. Success of such combined therapy may singificantly increase survival rates thanks to the synergistic effect.

Studies on detailed parameters of single/multiple HBOT application in post-stroke patients may significantly influence guidelines toward everyday clinical practice. HBOT still needs individually shaped administration.

Improvement of diagnostic tests and medical imaging causes more accurate and quicker possibility of safe intervention and therapeutic success. Application of HBOT allows for further increase aforementioned safety margin to early reaction, and use of combination of methods within longer therapeutic window.

Further studies are required also in the area of HBOT-related effects within long-term neurorehabilitation including partial re-integration of damaged tissues. Extended efficacy, better functional outcomes, and increases patients' wuality of life are the ultimate goals of our efforts.

Conclusions

HBOT constitutes promising method in post-stroke patients. Further research is needed to better define the role of HBOT in the therapy of post-stroke patients.

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