USWR 18: Outcome measure: Complications or Side Effects among patients undergoing Treatment with HBOT

MEASURE STEWARD:
US Wound Registry and the Undersea and Hyperbaric Medical Society (UHMS)

DESCRIPTION:
Percentage of patients aged 18 years or older undergoing HBOT for any diagnosis who experience a complication or side effect of therapy. This is a per visit measure.

NUMERATOR:
Patients who experience a specifically defined complication or side effect of HBOT.

DENOMINATOR:
Patients aged 18 years or older who
- Have undergone HBOT for any Medicare covered diagnosis.

DENOMINATOR EXCLUSIONS / EXCEPTIONS
EXCLUSIONS: NONE
EXCEPTIONS: NONE

RATIONALE:
Hyperbaric oxygen therapy is a safe medical therapy with a low risk of side effects and complications. Hyperbaric oxygen therapy involves placing the entire patient in a pressure vessel (hyperbaric chamber) which is then compressed with air or oxygen, and allowing the patient to breathe oxygen at an atmospheric pressure greater than 1.3 times sea level pressure. Due to the mechanical effects of the atmospheric pressure change and the unique physiologic effects of some gases at increased partial pressure, certain side effects are known to occur. These side effects are not due to improper use of HBOT, to equipment malfunction, or a failure of care. These are known risks associated with HBOT. Certain underlying medical conditions may make patients more likely to experience side effects from HBOT. The potential for side effects and complications from HBOT can be mitigated by screening patients for specific underlying medical problems. It is possible to modify the way in which HBOT is provided (e.g. slowing the rate of chamber compression for patients with known Eustachian tube dysfunction), or providing patients with additional information as to the risk vs. benefit of HBOT in their particular case. HBOT may be limb or lifesaving depending upon the indication for which it is recommended. Thus, even a patient with an increased risk of grand mal seizure due to oxygen toxicity may feel this risk is acceptable if, for example, the reason they are being offered HBOT is for carbon monoxide poisoning. In such a case, the benefit of HBOT to protect from the long term neurological sequelae of CO poisoning still outweighs the relatively low risk of hyperoxic seizure from which there are no reported long term neurological effects. Thus, the risk(s) of HBOT side effects must always be evaluated in the context of the potential benefit of HBOT for a given condition.

Middle ear barotrauma is the most common side effect of hyperbaric oxygen (HBOT) therapy. This is usually manifested as ear pain but tympanic membrane rupture can occur. The incidence of otic barotrauma is reported between 2% and 10% of patients undergoing HBOT. It is prevented in most patients by teaching autoinflation techniques or by use of tympanostomy tubes for those who cannot autoinflate their middle ear compartment. Sinus barotrauma or pain is seen less frequently than middle


ear barotrauma. Both of these problems are due to the change in volume of gas with atmospheric pressure.

Claustrophobia or confinement anxiety, present in about 2% of the general patient population, may cause some degree of anxiety among patients undergoing HBOT. Mild sedation may be required for patients to tolerate treatments. Progressive myopia has been observed in some patients undergoing prolonged periods of daily HBOT therapy. Although the exact mechanism remains obscure, it is apparently lenticular in origin and usually reverses completely within a few days to several weeks after the last therapy.

Vision changes as a side effect of HBOT therapy have been well documented since first described by Anderson and Farmer in 1978. The etiology remains unknown, but is likely related to lenticular changes, and usually reverses completely within weeks to months after the last HBOT treatment. Lyne showed that 18 of 26 patients (including all 4 diabetics) treated for 60 minutes at 2.5 ATA while oxygen breathing with a 30 min. compression and a 30 min. decompression over a course of 4-52 weeks developed myopia ranging from 0.5 – 5.5 diopters. Post-HBO2, vision recovery was initially rapid, then slowed, lasting up to one year. No new lens opacities formed, and in patients with pre-existing opacities, none progressed. In a Swedish study, all but 1 of 25 patients treated with 150-850 exposures at 2-2.5 ATA 7 days/week showed myopic refractive changes; 7 of 15 developed new, well-defined nuclear cataracts with the earliest appearing at 150 sessions. The nuclear cataracts were not reversible after cessation. In a study of 96 HBOT patients treated an average of 26 times (range 6-59) at 2.0 or 2.5 ATA for 90 minutes, 5 times/week, visual acuity decreased an average of 2.13 lines on the Snellen chart. There was a greater-than-average change in 31%, no change in 10%, and change > 3 lines in 46% of 50-60 year-olds. Four % more diabetics had a greater-than-average change, and 8% more non-diabetics had no change. A plateau in myopic shift was noted around treatment 25. In a retrospective review of 52 patients treated with > 20 HBOT sessions, 81% experienced vision change at the 20th treatment (compared to pretreatment). Myopic change was the most common, but 25% experienced hyperopia. With one possible exception to date, new cataracts are not reported during the 20-50 treatments commonly used in U.S. centers. Even when progressive myopia occurs during HBOT, the visual changes almost always reverse completely. However, extension of a series beyond 100 sessions has been associated with an increased risk of irreversible refractive changes or development of new cataracts. Information about vision change should be a part of the informed consent for each HBOT patient.

Breathing oxygen at increased atmospheric pressure can cause pulmonary oxygen toxicity, manifested in severe cases as pain on inspiration and reduction in FEV₁. Pulmonary symptoms are not produced by daily exposures to oxygen at the treatment pressure and duration used for clinical hyperbaric oxygen therapy (e.g. 2.0 or 2.4 atm abs for 120 or 90 min). There are no cases of pulmonary oxygen toxicity resulting from the routine clinical use of HBOT with the single exception of a patient having undergone Bleomycin chemotherapy which is a known sensitizing agent. Pulmonary barotrauma with pneumothorax during decompression from increased atmospheric pressure may rarely occur. Patients with airway obstruction probably are at an increased risk for pulmonary barotrauma during decompression. Significant air trapping and a history of spontaneous pneumothorax are also causes for concern and mandate a careful analysis of potential benefit from hyperbaric oxygen therapy versus the associated risk. Only 2 cases of pulmonary barotrauma in association with clinical hyperbaric oxygen therapy have been reported in the literature.

The incidence of central nervous system oxygen toxicity varies widely depending on the atmospheric pressure, breathing apparatus, and acuity of the patient. Among stable patients treated at 2.0 ATA the
incidence is approximately 1:30,000. However, among very unstable patients treated at higher pressures
the incidence increases to approximately 2%. When oxygen convulsions do occur, there are no residual
effects if mechanical trauma can be avoided.

A less well understood side effect of HBOT is “flash pulmonary edema”. This is presumed to be due to an
increase in peripheral vascular resistance from oxygen induced vasoconstriction, increasing intracardiac
pressures and leading to cardiac overload among patients with pre-existing cardiac disease and
decreased ejection fraction (EF). However, no EF limit has been identified that would exclude patients
from HBOT. This problem is usually manifested as the sudden onset of shortness of breath during HBOT.
However, fulminant congestive heart failure symptoms have been reported.

For many years, individual hyperbaric centers, management programs, and other groups have
attempted to pool data to ascertain the incidence of side effects and the impact of specific patient risk
factors. However, no national database has ever been created to track these data. The UHMS believes
that the creation of a clinical quality measure for the reporting of side effects, combined with the
transmission of a Continuity of Care Document (CCD) will allow the USWR and the UHMS to better
understand incidence and risk factors, and then use these data to create more patient specific informed
consent for HBOT.

**CLINICAL RECOMMENDATION STATEMENTS:**
Patients undergoing HBOT should be monitored throughout the treatment for complications and side
effects of HBOT. These side effects are not due to improper utilization of HBOT. In those patients who
have persistent change, consider ophthalmology referral to assess for other causes.

**REFERENCES:**

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