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OXYGEN TOXICITY AFTER HYPERBARIC OXYGEN THERAPY

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ABSTRACT:

Hyperbaric oxygen therapy (HBOT) involves breathing 100% oxygen intermittently in a chamber (either mono- or multiplace chamber) at a pressure greater than one atmosphere absolute (1 ATA). Osteomyelitis of the maxilla is very rare due to the high vasculature of this bone. Hyperbaric oxygen therapy is used in the treatment of refractory osteomyelitis. A 24-year-old female received HBOT for treatment of chronic osteomyelitis maxilla. On the 13th day of HBOT she had twitching of her fingers, nausea/headache, blurred vision, and anxiety. Her cutaneous axillary temperature was 40.6 °C. Her pulse was 80/minute and blood pressure was 110/70. In her examination confusion and facial pallor were remarkable. After laboratory samples were taken conservative therapy including intravenous hydration, metamizole sodium, and peripheral cooling was given. In an hour her neurological symptoms had improved and her fever normalized. As all blood tests were normal, septic fever was ruled out. Based on the peripheral vasoconstriction, facial pallor, neurological symptoms, and hyperthermia during the HBOT, oxygen toxicity was considered a possible diagnosis.

Key Words: Hyperbaric Oxygen Therapy, Oxygen Toxicity Fever

HİPERBARİK OKSİJEN TEDAVİSİ ESNASINDA GELİSEN OKSİJEN ZEHİRLENMESİ

ÖZ:

Hiperbaric oksijen tedavisi (HBOT) birden büyük mutlak atmosfer basınç altında, tekli veya çoklu kabinler içinde, %100 oksijenin solutulması esasına dayanmaktadır. Maksillanın osteomyeliti bu kemiğin kanlanmasının oldukça iyi olmasına bağlı olarak nadiren görülmektedir. Hiperbarik oksijen tedavisi osteomyelit tedavisinde kullanılmaktadır. 24 yasında bayan hastaya kronik maksilla osteomyeliti tanısı ile HBOT baslandı. Tedavinin onüçüncü gününde tedavi esnasında hastada parmaklarda karıncalanma, bulantı, bas ağrısı ve görmede bulanıklık sikayetleri basladı. Hastanın vücut ısısı 40,6 C, nabzı 80/dk, tansiyonu 110/70 olarak ölçüldü. Hastada yüzde kızarıklık ve konfüzyon dikkat çekiciydi. Labratuvar testleri alındıktan sonra hastanın atesi intravenöz hidrasyon, metimazol sodyum ve periferik soğutma ile düsürüldü. Tüm labratuvar testlerinin normal olması üzerine hastanın klinik belirtileri göz önene alındığında hastada oksijen toksisitesi tanısı konuldu. HBOT esnasında periferik vazodilatasyon, nörolojik bulgular ve hipertermi varlığında her zaman oksijen zehirlenmesi göz önünde bulundurulmalıdır. Tedavi seansın hemen durdurulması sonrasında diğer ates etkenleri elenmesi ve konservatif olarak atesin düsürülmesidir.

Anahtar Kelimeler: Hiperbarik Oksijen Tedavisi Oksijen Toksisitesi Ates

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INTRODUCTION

Hyperbaric oxygen therapy (HBOT) involves breathing 100% oxygen intermittently in a chamber (either mono- or multiplace chamber) at a pressure greater than one atmosphere absolute (1 ATA). The chamber pressurization should be at least 1.4 ATA according to the Undersea and Hyperbaric Medical Society (UHMS). The pressures used in clinical therapies vary between 2 and 3 ATA. Therapy sessions are 90 to 120 minutes daily.

Osteomyelitis of facial bones, especially the maxilla, is very rare due to the high vasculature of this bone. HBO therapy increases tissue oxygen saturation and growth factors. It aggregates the fibroblast activity, increases the antibacterial effects of the immunity and antibiotics, and decreases inflammatory cytokines. Because of these effects hyperbaric oxygen therapy is used in the treatment of refractory osteomyelitis.

CASE REPORT

A 24-year-old female patient was admitted to our clinic with pallor and edema on the right maxilla that occurred after removal of a tooth. Her clinical findings and CT scans showed an abscess. After debridement and intravenous sulbactam-ampicillin treatment her clinical findings improved and she was discharged with oral sulbactam-ampicillin. Two months later she was admitted again with abscess formation at the same localization. After debridement Klebsiella was cultured from the material. MRI showed osteomyelitis of the maxilla and as her clinical findings had lasted longer than 6 weeks despite antibiotic therapy she was diagnosed with refractory osteomyelitis. Intravenous Ciproflaxacin 2 × 400 mg was given based on the antibiogram. As hyperbaric oxygen therapy has well known effects in the treatment of chronic refractory osteomyelitis, therapy was planned daily at 2.5 ATA with 100% oxygen in a chamber for 120 minutes in each session. After antibiotic therapy and hyperbaric oxygen therapy her clinical and laboratory markers began to improve.

On the 13th day of HBO therapy, in the last 20 minutes of the session the patient stated that she had twitching of her fingers. Ten minutes later, she complained of nausea/headache, blurred vision, and anxiety. Her therapy was stopped and she was taken to the clinic. In her physical examination her cutaneous axillary temperature was 40.6 °C. Her pulse was 80/minute and blood pressure was 110/70. In her examination confusion and facial pallor were remarkable. Venous blood samples were taken for comple-

te blood count (CBC), biochemistry analysis, sedimentation, CRP level, and blood cultures. Thirty minutes later the blood culture was repeated. After samples were taken, conservative therapy including intravenous hydration, metamizole sodium, and peripheral cooling was given. In an hour her neurological symptoms had improved and her fever normalized.

RESULTS

No pathologic bacteria were seen in blood cultures and no abnormality was seen in laboratory tests. As all blood tests were normal, septic fever was ruled out. Based on the peripheral vasodilatation, neurological symptoms, and hyperthermia during the HBOT, oxygen toxicity was considered a possible diagnosis.

DISCUSSION

HBOT increases tissue oxygen saturation and growth factors. It aggregates the fibroblast activity, increases the antibacterial effects of the immunity and antibiotics, and decreases inflammatory cytokines. Because of these effects HBOT is used in the treatment of chronic wounds, refractory osteomyelitis, and osteoradionecrosis.

The CNS effects of oxygen toxicity are called the 'Bert effect' named after Paul Bert, who, in 1878, demonstrated convulsions in larks exposed to 15-20 ATA. The 'Smith effect' is the pulmonary effects of oxygen toxicity, named after J Lorain Smith, who, in 1899, while trying to reproduce the 'Bert effect', noted fatal pneumonia in rats after 4 days of exposure to 73% oxygen at 1 ATA. The clinical settings of oxygen toxicity are broadly divided into two groups; in the first the patient is exposed to very high concentrations of oxygen for a short duration, like in HBOT, and in the second lower concentrations of the gas are used for longer duration. These two can result in the so-called 'acute' and 'chronic' oxygen toxicity, respectively. Acute toxicity has predominant CNS effects, while chronic toxicity has predominant pulmonary effects.

Although Bert originally reported that CNS toxicity occurred at oxygen pressures of >3 ATA, it may, however, occur at lower pressures if exposure is prolonged. An acute increase in oxygen saturation results in peripheral vasoconstriction, which leads to early manifestations of oxygen toxicity like twitching of the fingers and facial pallor. Continuation of exposure can lead to vertigo and nausea, followed by altered behavior, clumsiness, and finally convulsions. Some factors aggravating the CNS toxicity are raised pCO2, stress, fatigue, cold, and dietary deficiency of trace elements such as selenium, zinc, and magnesium.

Pulmonary effects of oxygen toxicity can occur after a prolonged exposure to oxygen >0.5 ATA (chronic toxicity). Symptoms appear after a latent period, whose duration decreases with increases in PO2. In normal humans, the first signs of toxicity appear after 10 hours of oxygen at 1 ATA. Although at first symptoms are mild, reversible prolonged exposure leads to acute respiratory distress syndrome and pulmonary interstitial fibrosis.

Oxygen also has toxic effects on the eyes, ears, and red blood cells. With increased levels of oxygen reversible constriction of the peripheral field of vision results in a progressive but reversible myopia causing blurred vision. Serous otitis media and dysbaric osteonecrosis were seen in astronauts during space flight. Prolonged oxygen exposure can change RBC morphology and hemolysis occurs.

The main pathophysiology of oxygen toxicity is free radical damage (Table 1). Increased free radicals impair lipid peroxidation, protein synthesis, and nucleic acid synthesis, and damage the cellular membrane. Oxygen can also directly decrease glutamic acid decarboxylation and GABA synthesis, resulting in convulsions.

Diagnosis of oxygen toxicity is based on the clinical symptoms. An electroencephalogram (EEG) is of no value in the monitoring of CNS toxicity.

Treatment consists of monitoring the patient and supportive therapy until the symptoms resolve. Exogenous antioxidants, especially vitamin E and C, may be used prophylactically. The recommended dose of vitamin E is 100 mg/kg/day for 4-6 weeks. Adrenalectomy, hypophysectomy, and the hypothyroid state are associated with reduced severity of the toxicity as is the use of alpha adrenergic blockers. As dietary deficiency of trace elements is likely to worsen the toxicity, their supplementation may be helpful in deficient states.

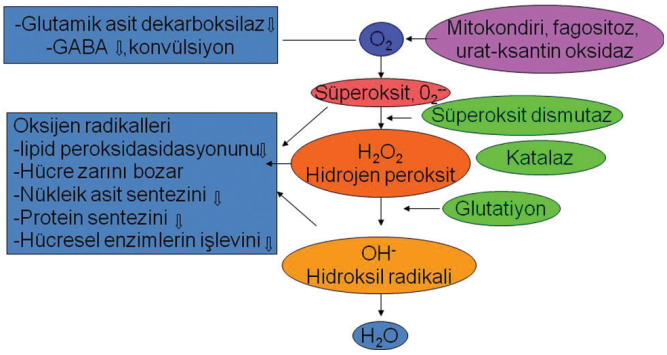

Table 1: Physiological effects of oxygen toxicity.

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