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Hyperoxia crisis: proposed intervention algorithm

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Keypoints

The article proposes an algorithm for the management of hyperoxic crisis during hyperbaric treatment.

Abstract

Hyperoxic crisis is a relatively rare event that clinically presents as an epileptiform reaction. In most cases, it resolves quickly and without consequences for the patient, provided it is managed correctly. This side effect of hyperbaric medicine can be difficult to manage inside the hyperbaric chamber, especially if medical, paramedical, and technical personnel are not adequately trained. It may pose a safety risk inside the hyperbaric chamber for both patients and healthcare providers. Proper staff training and the adoption of operational protocols are essential.

Keywords

Hyperoxic crisis, oxygen toxicity, hyperbaric treatment, side effects, hyperbaric therapy.
exposure, fetal hypoxia due to CO, paediatric CO intoxication, child, infant.

Introduction

Hyperoxic crisis is a relatively rare event, clinically manifesting as an epileptiform reaction triggered by the excessive production of free radicals. Although it is a highly distressing experience for both the patient and observers, it typically resolves quickly without long-term effects once the noxious agent (oxygen) is removed. There are no Italian case series in the literature documenting its true incidence in pediatric patients or during pregnancy. This may be partly due to the absence of a national registry for adverse events in hyperbaric medicine, leading to incomplete and often partial data. Hyperoxic crisis is a rare event and cannot be entirely prevented—although it can be anticipated. While there is no preventive treatment for hyperoxic crisis, our behaviors can help reduce the discomfort and risk for the patient.

Analysis

The link between seizures and hyperbaric oxygen was first recognized by Paul Bert in 1878. Dr. Lambertsen described it in his publications as follows: “The seizure is

usually preceded by the appearance of localized muscle spasms… Eventually, there is a sudden spread of excitation, and the tonic phase of the seizure begins. Vigorous clonic contractions then occur in the muscle groups of the head and neck, trunk, and limbs, gradually becoming less intense over about a minute."

An oxygen toxicity seizure is relatively rare at the usual clinical treatment pressures (2 ATA–3 ATA). The literature reports a global frequency of about 1 in 10,000 treatments.Over the last 15 years, the worldwide incidence has been around 1 case in every 2,000–3,000 treatments.

The increase in incidence over the last 15–20 years appears to be related to the selection of more critically ill patients with multiple comorbidities and to changes in hyperbaric oxygen treatment protocols.

The individual risk of developing a crisis during treatment is difficult to predict, though several predisposing factors exist (Table 1).

Main Risk Factors
Increased treatment pressure
Brain tumors / radionecrosis lesions
Hypoglycemia / hyperthyroidism
CO_2 retention
Carbon monoxide poisoning
Medication use
Sepsis
Fever
Respiratory acidosis
Epilepsy

Table 1. Risk factors

Sedative-hypnotic drugs (used in critically ill patients) may have a protective effect. Hyperoxic crisis appears to be due to direct oxygen toxicity. The increase in reactive oxygen species (ROS) and other radical compounds may be responsible for lipid peroxidation of neuronal plasma membranes, leading to altered brain electrical activity. Hyperbaric oxygen therapy (HBOT) induces an increase in ROS, with peak production occurring around 2 hours and remaining above baseline for up to 48 hours. The effects of ROS can be counteracted by the action of NRF2, a transcription factor that regulates the antioxidant response. Nitric oxide (NO) also plays a role in oxygen-induced cerebral toxicity through the production of peroxynitrite and cerebral vasodilation. Less than 50% of patients exhibit prodromal signs and symptoms of the crisis (Table 2).

Prodromal Signs and Symptoms	
Profuse sweating	Lip fasciculations
Pallor	Cheek contractions
Bradycardia – Tachycardia	Respiratory difficulties
Mood changes (depression, euphoria)	Vomiting
Drowsiness	Nausea
Visual disturbances, narrowing of the visual field (tunnel vision)	Dizziness
Scotomas	Shouting incoherent speech
Auditory hallucinations	
Unpleasant taste and/or smell sensations	
Epigastric tension	

Table 2. Prodromal signs and symtomps of hyperoxic crisis

Within a few minutes from the onset of prodromal signs and symptoms, the actual hyperoxic crisis occurs, evolving through three phases:

1. **Tonic phase** with generalized muscle rigidity
2. **Clonic phase**, lasting 2–3 minutes, characterized by convulsions, tongue biting, and involuntary loss of feces and/or urine
3. **Postictal depression phase**, lasting about 10 minutes, marked by drowsiness, often stertorous breathing, and profuse sweating

Progressive recovery of consciousness is often accompanied by confusion, agitation, and retrograde amnesia.

Despite its dramatic presentation, hyperoxic crisis usually leaves no lasting effects. The main risks are related to trauma from falls or involuntary movements during the episode.

There is no effective treatment for the crisis itself, which typically resolves fairly quickly once the harmful agent (oxygen) is removed or reduced.

Preventive measures include scheduled air breaks to reduce exposure to free radicals and oxygen toxicity—numerous studies support this approach in the literature.

Proper staff training and the implementation of standardized protocols are essential for the correct management of both the crisis and the patient.

Special attention must be given to properly informing the patient and any accompanying persons. The onset of a crisis inside the hyperbaric chamber may trigger panic attacks and compromise safety.

Thorough pre-treatment evaluation of patients is critically important, including the identification and, if possible, correction of predisposing factors, depending on the clinical condition.

Specialist assessments, such as obstetric-gynecological or pediatric-neonatal consultations, are also important prior to treatment.

Patients should be informed about the importance of promptly reporting the onset of symptoms to the staff.

When a hyperoxic crisis occurs, chamber personnel should:

1. **Clearly and concisely** communicate the onset of the crisis to external personnel
2. **Immediately remove the mask** or stop oxygen administration and switch to air. Note that switching to air reduces FiO_2 and consequently PaO_2, lowering the pathogenic exposure
3. **Ensure patient safety**, preventing trauma and injuries; in pregnant patients, evaluate left lateral positioning
4. **Reassure other patients and companions** in the chamber to prevent panic
5. **Assess and support vital signs** (A-B-C approach)
6. **Ensure airway protection** against the risk of obstruction (e.g., tongue fall, trismus)

The chamber must not be decompressed until the tonic-clonic crisis has resolved, due to the risk of pulmonary barotrauma and glottic closure. Only after the resumption of adequate respiratory rhythm can the hyperbaric chamber be gradually decompressed.

Patients who have experienced an oxygen toxicity crisis can still complete the recommended treatment cycle. Although the risk of recurrence is increased, it remains below 10%. Modifications to subsequent treatments may be considered, such as lower treatment pressures and additional air breaks.

At the end of the crisis, the patient should be carefully monitored until full recovery. A gynecological-obstetric assessment (including fetal well-being) or a pediatric-neonatal evaluation may be important.

The article proposes a management algorithm for use during hyperbaric treatment and in the event of a hyperoxic crisis (Fig. 1).

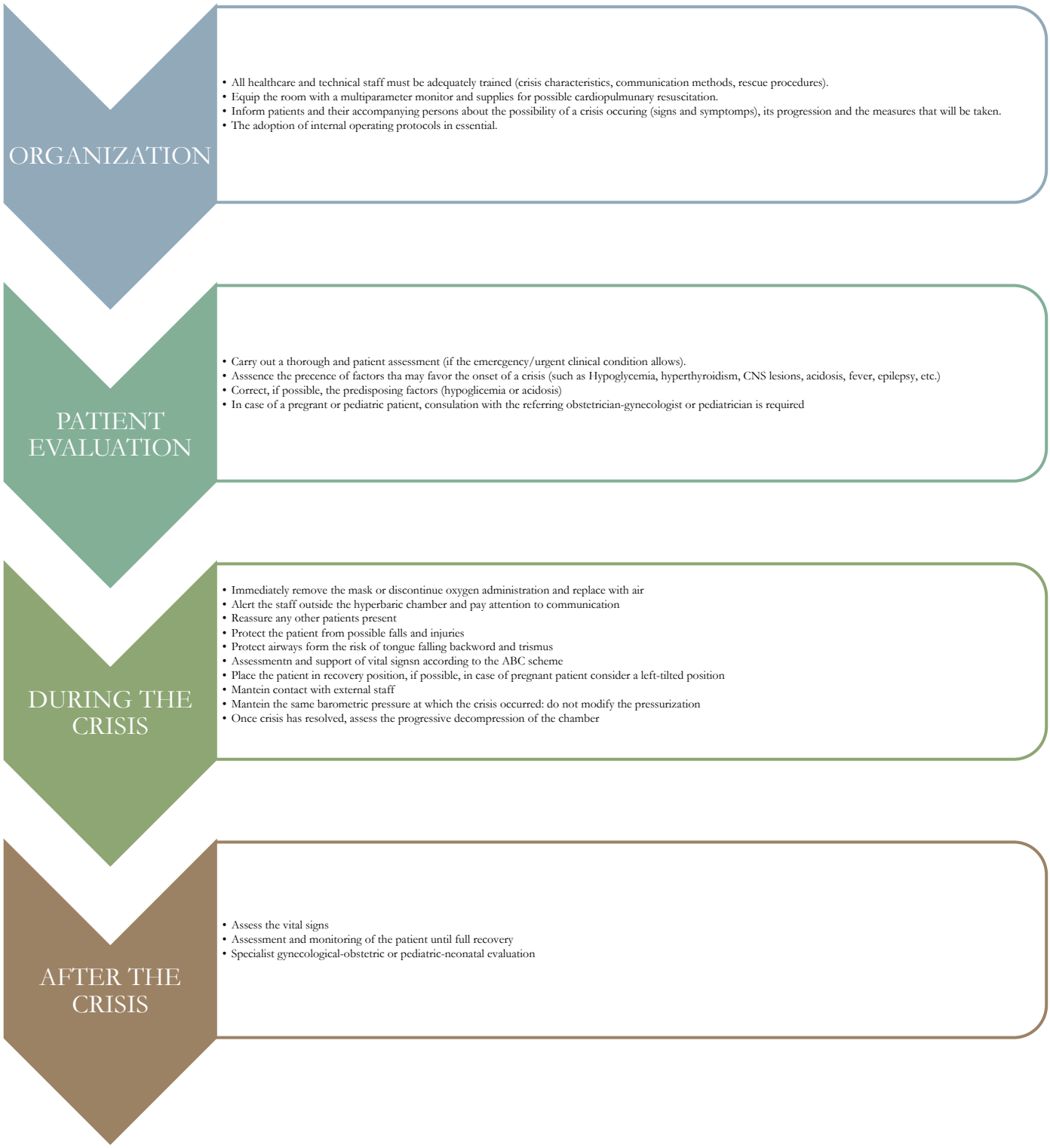

Figure 1. Algorithm for the treatment of hyperoxic crisis

Conclusion

Although oxygen toxicity seizures are one of the most feared side effects of hyperbaric oxygen therapy (HBOT), their incidence remains low, with no evidence of long-term sequelae following an episode. Proper staff training, the adoption of internal protocols, and adequate patient education are essential to reduce the incidence of adverse events and to enhance safety for both patients and healthcare providers.

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