Pediatric Anesthesia and Critical Care Journal 2026;14(1):18-21
doi:10.14587/paccj.2026.3

PACCJ

Hyperbaric treatment for acute carbon monoxide poisoning in a patient with beta-thalassemia

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Keypoints

This article describes the case of a pediatric patient with acute carbon monoxide poisoning and beta thalassemia, treated with off-label hyperbaric oxygen therapy.

Abstract

Carbon monoxide (CO) poisoning is a rare but potentially devastating event and remains the most common cause of poisoning in developed countries. Diagnosis is often missed or delayed, as the clinical presentation can be highly variable and characterized by non-specific signs and symptoms that may lead to misinterpretation. This article describes the case of a pediatric patient with acute carbon monoxide poisoning and underlying beta-thalassemia, treated with hyperbaric oxygen therapy.

Keywords

Beta thalassemia, carbon monoxide poisoning, hyperbaric treatment, carbon monoxide, CO, microcytic anemia.

Introduction

Carbon monoxide (CO) poisoning is a rare but potentially devastating event and remains the most frequent cause of poisoning in developed countries. Diagnosis is often missed due to a highly variable clinical presentation and the presence of non-specific signs and symptoms, which can lead to diagnostic errors. Coexisting conditions can significantly increase clinical risk and the likelihood of complications.

In particular, the presence of anemia of various origins may exacerbate the effects of carbon monoxide poisoning, further reducing the amount of oxygen delivered to tissues and worsening tissue hypoxia and its consequences.

Case report

COVID-19 proved to be a determining factor in decision-making regarding access to healthcare, including emergency and urgent care. During the last cold spell before the arrival of the SARS-CoV-2 infection, the Diving & Hyperbaric Medicine Città di Brescia was alerted to the case of a child (under 12 years of age) of African descent who had been accidentally exposed to carbon monoxide (CO). The patient's personal and family medical history was positive for beta thalassemia. In this case, the patient did not show symptoms directly attributable to beta thalassemia. Hemoglobin levels were slightly below normal,

as were MCV and MCH values. Clinically, the child presented as asymptomatic, with a COHb level of 8%. The likely source of the poisoning was a brazier used by the family for indoor heating. Upon arrival at the emergency department, the attending physician contacted the Poison Control Center in Pavia, which referred the patient to the nearest hyperbaric center for treatment. Given the clinical picture and the presence of microcytic anemia, and in agreement with the Poison Control Center, the patient underwent hyperbaric oxygen therapy at 2.8 ATA for 23 minutes, followed by a gradual decompression to 2 ATA for 50 minutes. No significant clinical events were recorded during the session or at its conclusion. The patient was returned to the referring department with a follow-up COHb level and the usual recommendations for monitoring, particularly for early signs of delayed neurological syndrome.

Discussion

Current guidelines recommend hyperbaric oxygen therapy for patients under 12 years of age with COHb levels greater than 10%[1]. The patient had no contraindications to hyperbaric therapy, which could therefore be safely administered.

The personal and family medical history was positive for beta thalassemia. Hemoglobinopathies are the most widespread genetic disorders in humans, with an estimated carrier rate of about 7% of the global population and between 300,000 and 500,000 affected newborns each year (2-3).

The highest prevalence is found in Southeast Asia (including southern China and Indonesia), the Middle East, the Mediterranean basin, and North-Central Africa, with a carrier rate ranging from 2% to 25%. However, due to recent migration flows, Northern Europe has also seen a carrier frequency between 0% and 19%. The term "thalassemias" refers to a group of hematological disorders characterized by reduced or absent synthesis of the normal globin chains of the hemoglobin molecule. In beta-thalassemia, the defect lies in reduced or absent synthesis of beta globin chains, resulting in a relative excess of alpha chains[2-6]. The decreased synthesis of one of the globin chains leads to reduced hemoglobin formation, resulting in hypochromic microcytic anemia. For this reason, thalassemic syndromes are also referred to as "microcytemias" (Fig.1). Most thalassemias are inherited in an autosomal recessive manner.

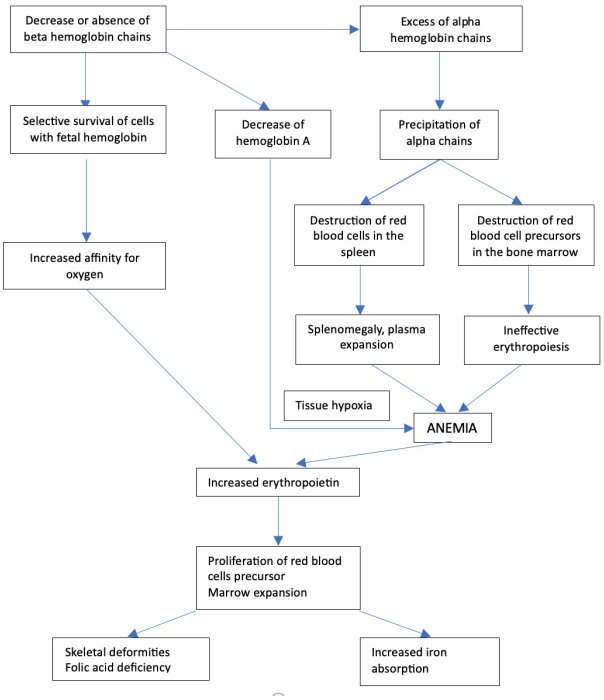

Figure 1. Pathophysiology of Beta Thalassemia - PDTA of Puglia Region (Italy)

The phenotypic expression of thalassemic syndromes, particularly beta forms, is extremely heterogeneous, ranging from asymptomatic carrier states to intermediate severity presentations (thalassemia intermedia), up to the most severe forms represented by thalassemia major (TM) (Tab. 1).

• Homozygosity • Significant imbalance of alpha/beta globin chains • Severe anemia presenting at an early age • Requirement for regular transfusion support • Death within the first decade of life if untreated	BETA THALASSEMIA MAJOR
• Variable genetic compounds • Mild imbalance in globin chain production • Mild anemia, diagnosed in childhood or adolescence • Need for occasional transfusions	BETA THALASSEMIA INTERMEDIA
• Heterozygosity • Asymptomatic condition • May require genetic counseling	BETA THALASSEMIA MINOR

Table 1. Disease severity and symptomatic manifestation of Beta Thalassemia - PDTA of Lombardy Region (Italy)

The clinical complications that develop over the years in clinically significant forms of the disease (thalassemia major and thalassemia intermedia) are partly due to the pathophysiological mechanisms of the disease itself, but largely secondary to transfusion therapy, which causes significant iron overload. Transfusion-related siderosis leads to severe organ damage, especially affecting the heart, liver, and endocrine organs[2-6].

Cardiomyopathy remains the leading cause of death in thalassemic patients. It has a multifactorial etiology, resulting from anemia, iron accumulation, and, in some cases, other events such as pericarditis and myocarditis. Clinically, cardiac involvement often manifests only when ventricular function is already severely compromised or when symptomatic arrhythmias appear. Once clinical signs emerge, progression can be rapid, sometimes resulting in refractory heart failure.

Liver damage in thalassemic patients is associated with infections from hepatotropic viruses (HCV and HBV — often related to transfusion of infected blood units) and iron overload.

The most common endocrine complications include hypogonadotropic hypogonadism, growth and developmental delay, diabetes mellitus, and, less frequently, hypothyroidism, hypoparathyroidism (secondary to hemosiderosis), osteoporosis, and adrenal insufficiency. Endocrine abnormalities are among the most frequent complications in patients with severe forms of beta-thalassemia and are mainly due to iron overload, both endogenous (from hemolysis) and exogenous (from transfusion therapy). The pituitary gland is particularly susceptible to iron toxicity, which is why early initiation of appropriate iron chelation therapy is crucial.

Typical carriers of β-thalassemia usually present with reduced mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH), normal or slightly reduced hemoglobin levels for age, and elevated HbA2 levels.

In the present case, the patient did not show symptoms attributable to beta-thalassemia. Hemoglobin levels were slightly below normal, as were MCV and MCH values.

Inhaled CO binds to hemoglobin to form carboxyhemoglobin (COHb), with a binding affinity approximately 200–300 times greater than that of oxygen. As a result, CO interferes with the normal transport of oxygen to peripheral tissues. This causes a leftward shift in the O_2-Hb dissociation curve, leading to tissue hypoxia[7-8].

The toxicity of CO is related to two mechanisms: tissue hypoxia (indirect damage) and tissue inflammation (direct damage).

The initial injury is due to indirect tissue hypoxia caused by reduced arterial oxygen content, resulting from impaired oxygen transport and worsened by reduced oxygen release to tissues. Hypoxia itself induces oxidative stress, leading to the generation of reactive oxygen species and subsequent inflammation and tissue injury.

Tissue hypoxia is further exacerbated in the presence of microcytic anemia, as seen in this case. The combination of microcytic anemia and carbon monoxide poisoning may also increase the incidence of complications, particularly cardiovascular ones.

In light of all this — and considering the clinical picture of acute CO poisoning — hyperbaric oxygen therapy was chosen, despite a COHb level of less than 10%.

The multidisciplinary consultation between hyperbaric medicine specialists and the poison control center proved to be a key factor in reducing complications and preventing long-term sequelae.

Conclusion

The interdisciplinary collaboration in this case proved essential for the evaluation and "off-label" treatment of the patient (at a further proof that, if they are not paired with the patient's medical history and one's clinical evolution, numbers often remain just plain numbers, without any specific value and a real possibility to help in proper therapeutical choices). Anemia represents an additional risk factor in carbon monoxide poisoning. Currently, clinical guidelines do not address the presence of anemia as a criterion for hyperbaric oxygen therapy. Therefore, careful individual case assessment is critical when deciding whether to initiate hyperbaric treatment.

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