Exposure to Extremely High Concentrations of Carbon Dioxide: A Clinical Description of a Mass Casualty Incident

Clinical reports on unintentional mass exposure to extreme concentrations of carbon dioxide are rare. We describe an industrial incident caused by a container of liquid carbon dioxide that was unintentionally opened in an enclosed working environment. Twenty-five casualties reached our emergency department. Symptoms included dyspnea, cough, dizziness, chest pain, and headache. ECGs (n=15) revealed ST-segment changes in 2 (13.3%) patients, atrial fibrillation in 2 patients, and non–Q wave myocardial infarction in 1 patient. Chest radiographs (n=22) revealed diffuse or patchy alveolar patterns, consistent with pneumonitis, in 6 (27%) patients and pulmonary edema in 2 (9%) patients. Eleven (44%) patients were admitted to the hospital: 8 were discharged 24 hours later and the others within 8 days. No patient died. Exposure to high concentrations of carbon dioxide resulted in significant but transient cardiopulmonary morbidity with no mortality when victims were promptly evacuated and given supportive therapy. Cardiac complications were frequently observed and should be actively sought.


INTRODUCTION

Carbon dioxide is a colorless, odorless, nonirritating gas that has many practical uses, such as carbonating soft drinks and providing a shield during welding. Carbon dioxide is widely used as a fire extinguisher because of its ability to displace oxygen from the incendiary environment. High environmental concentrations of carbon dioxide (>17%) may cause severe and even fatal poisoning in industrial (eg, ice-making factories) and occupational or recreational (eg, diving) settings. The potential severity of toxicity from carbon dioxide was tragically exemplified by the disaster in Cameroon in 1986, when many people were killed by the expulsion of carbon dioxide from a volcano.

Industrial incidents caused by carbon dioxide are well reported in the industrial safety literature, but we were unable to find clinical descriptions of nonfatal mass casualties in these reports. We describe a mass casualty event caused by high concentrations of carbon dioxide after an industrial incident.
A group of workers was exposed to variable and undocumented but extremely high concentrations of carbon dioxide in an ice-making factory when the discharge valve of a truck containing liquid carbon dioxide was knocked open in an enclosed environment. The entire contents of the approximately 10-m³ container were rapidly discharged into a hall roughly 10 × 20 × 4 m. Witnesses described that a white cloud approximately 2 m thick quickly formed. Within seconds, many of the exposed persons were incapacitated and needed to be evacuated by coworkers. Most of the workers estimated that they had been exposed for a few seconds up to 1 minute before successfully reaching open air, but 25 casualties, including 2 rescue personnel, required transport to our emergency department (ED) and arrived there within approximately 10 to 30 minutes of the event.

The patients were 24 men and 1 woman (mean age 39.6 years, range 19 to 76 years). Their symptoms included dyspnea (64%), headache (8%), dizziness (28%), cough (12%), and chest pain (12%). Three (12%) patients had lost consciousness at the scene, and 1 of them was observed to have convulsions. Five patients had tachycardia more than 100 beats/min on admission, and 1 patient had systolic arterial pressures less than 100 mm Hg (and also a finding of atrial fibrillation at 150 beats/min).

ECG tracings were obtained for 15 patients within 30 minutes of ED admission. Of these tracings, ischemic ST-segment changes were observed in 2 (13%) of 15 and atrial fibrillation in 2 (13%) of 15. In 1 patient, ischemic ST-segment changes and atrial fibrillation coexisted (total of 3 patients with abnormal tracings); the remaining ECGs were normal. Chest radiographs were obtained in 22 patients within 1 hour of ED admission. Pulmonary edema was evident in 2 (9%) of 22; diffuse or patchy alveolar pattern, consistent with “pneumonitis,” was evident in 6 (27%) of 22. Fourteen (64%) of 22 radiographs were interpreted as being normal. Arterial blood gases were obtained in 8 patients (all receiving 40% to 95% oxygen by mask): 1 result was normal, 6 patients had respiratory acidosis, and 1 patient had metabolic acidosis (mean PaO₂ for 3 hypoxemic patients was 69 mm Hg, range 45 to 85 mm Hg; mean PaCO₂ of the respiratory acidosis group was 65 mm Hg, range 47 to 107 mm Hg; mean arterial hydrogen ion concentration of this group was 7.26, range 7.09 to 7.34; mean base excess for the group −2.2, range 4.4 to −6.5). Resting pulmonary function tests were obtained in 7 of 25 patients (6 within 2 to 4 hours from admission); all were within normal limits.

For one patient who had been admitted to a medical ward, acute non-Q wave myocardial infarction was eventually diagnosed according to ECG changes (T wave inversion in leads L1, aVL, and V2 to V6 and creatine kinase containing M and B subunits fraction elevation at 6% of 387), but without associated clinical complaints. One patient with radiographic evidence of pulmonary edema underwent pulmonary artery catheterization within 2 hours of admission. The measured values were as follows: mean right atrial pressure 2 mm Hg; right ventricular pressure 18/0 mm Hg; pulmonary artery pressure 20/12 mm Hg; pulmonary capillary wedge pressure 10 to 12 mm Hg; systemic arterial pressure 100/40 mm Hg; cardiac output 6.7 L/min; systemic vascular resistance 800 dyne·s/cm⁵; pulmonary vascular resistance 100 dyne·s/cm⁵. Radio nuclide ventriculography with technetium-99m was performed on the third hospitalization day and revealed good left ventricular function and right ventricular global hypokinesis. Pulmonary function test results were normal. This patient was discharged after 4 days in good clinical condition, with normal ECG and chest radiographic findings.

Eleven patients (44%) were admitted, 1 to the cardiac care unit and the rest to general medical floors, 8 for 1 day, 1 for 2 days, and 1 for 4 days, with 1 patient requiring 8 days of hospitalization for acute myocardial infarction. All patients were treated with high oxygen concentrations by nonrebreather masks and eventually recovered fully and uneventfully.

Carbon dioxide is normally present in the atmosphere at about 360 parts per million by volume. The Occupational Safety and Health Administration and the American Conference of Governmental Industrial Hygienists exposure limit to carbon dioxide is 5,000 parts per million (9,000 mg/m³), or 0.5% for an 8-hour time-weighted average. Acute health effects of exposure to high carbon dioxide concentrations are discussed at length in industrial literature. The Occupational Safety and Health Administration considers a 10% carbon dioxide concentration potentially lethal.
EXPOSURE TO EXTREMELY HIGH CONCENTRATIONS OF CARBON DIOXIDE Halpern et al

dioxide can be conveniently stored and transported in metal cylinders at normal room temperature. It is more compact than the gas form; in fact, if the gas is compressed at constant room temperature, it will eventually condense to the liquid form. The solid form of carbon dioxide is more difficult to work with because it needs to be kept cold for storage and then warmed up for the carbonation process. The liquid form of carbon dioxide can be easily pumped and is used in the beverage industry as a taste enhancer and for carbonation. Assuming that all of the liquid carbon dioxide had escaped from the container, and given that some had solidified into solid carbon dioxide, or dry ice, our calculation yields a quantity of 7,748 m\(^3\) of gaseous carbon dioxide expelled into a volume of approximately 8,000 m\(^3\), almost totally displacing the air in the room where the event occurred (20x4x10 m).

Carbon dioxide closely resembles simple asphyxiants from a toxicologic standpoint. Carbon dioxide in high concentration, however, has direct toxic effects, despite normal atmospheric oxygen concentrations.\(^{10}\) The physiologic effects of hypercapnia are mainly those of sympathetic stimulation, including increased heart rate, cardiac output, mean pulmonary artery pressure, and pulmonary vascular resistance,\(^{11,12}\) and therefore impose excess load on the myocardium.

In the event we describe, 23 industrial workers and 2 rescue workers were exposed to high concentrations of carbon dioxide in an enclosed environment. Carbon dioxide, usually a colorless and odorless gas, was rapidly discharged and formed a potentially hazardous white cloud. The swift evacuation on the part of the coworkers before the medical team arrived undoubtedly reduced the time of exposure to carbon dioxide. Exposure of unprotected rescue teams to carbon dioxide resulted in the intoxication of 2 paramedics. There was no clear correlation between the presence of dyspnea, the most frequent complaint on admission, and extended hospitalization; only 7 (44%) of 16 patients with dyspnea required hospitalization.

A non–Q-wave acute myocardial infarction was diagnosed in one patient: it would be difficult to prove that this condition developed as a direct consequence of his carbon dioxide intoxication, and there are no relevant data on this association in the literature. The ECG changes, including paroxysmal atrial fibrillation and ischemic changes in a number of our patients, serve to raise the issue of a possible causal relationship. Hypercarbia causes sympathetic stimulation and increases cardiac output while it also shortens coronary filling time; thus, cardiac oxygen demand significantly increases, and the potential for relative myocardial ischemia becomes manifest.\(^{13}\)

Chest radiographs varied from normal to pulmonary edema. One patient with a chest radiograph compatible with pulmonary edema, as well as hypotension and atrial fibrillation with diffuse ischemic changes on ECG, underwent pulmonary artery catheterization and isotopic ventriculography. The measured values were consistent with normal cardiac function, so the diagnosis of noncardiogenic pulmonary edema caused by carbon dioxide intoxication was considered. We were unable to find cases of documented noncardiogenic pulmonary edema caused by carbon dioxide intoxication in the literature, except one case report in which clinical suspicion of pulmonary edema was raised solely according to blood gas data during patient resuscitation from severe acute hypercapnia.\(^{14}\) Reports of fatal cases of carbon dioxide exposure\(^{2,3}\) indicate that the lungs were normal on postmortem examination.

The respiratory acidosis that had been revealed in arterial blood gas analysis is likely explained by residual respiratory depression because the rate of excretion of carbon dioxide from the body is so rapid with normal response hyperventilation that arterial \(\text{PCO}_2\) usually returns to normal within a few minutes.\(^{15}\) The blood gas values of the patient with convulsions observed at the scene, however, were compatible with metabolic acidosis (pH 7.19; \(\text{PaCO}_2\) 42 mm Hg; bicarbonate –13.6 mEq/L; base excess –12 mEq/L) on admission, and they returned to normal values within 24 hours. A possible explanation for this result may be lactic acidosis caused by convulsions.

The short hospitalization for the majority of victims, approximately 24 hours for 8 of 11 patients, testifies to relatively rapid recovery.

In summary, carbon dioxide is widely used in industry. Its physical properties (colorless, odorless, and nonirritating) make it a potential toxicologic hazard. The present series documents an unusual event in which recovery from severe symptoms of carbon dioxide intoxication at extremely high concentrations was the rule when rapid evacuation from the affected environment was carried out. The most important action in carbon dioxide intoxication is removal of the victim from the exposed environment and provision of cardiorespiratory function support until spontaneous recovery occurs. Finally, the use of airway protection...
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