Delayed Onset of Neurological Symptoms in Carbon Monoxide Poisoning

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A 75-year-old old man with a history of progressive gait problems due to severe knee arthritis and a family history of Parkinson's disease (PD) presented to the clinic five months after carbon monoxide (CO) poisoning. He had been working alone at his glassblower studio for about 80 minutes when his wife found him stuporous and called 911. Upon arrival to the hospital, his temperature was 92°F. The carboxyhemoglobin (COHb) level was 22.5% within four hours of arriving. During this time he was minimally verbal. He was immediately given oxygen by mask and transported to a hospital where hyperbaric oxygen (HBO) therapy was administered about 10 hours after being found. He received one treatment of HBO for 90 minutes at 2.5 atmospheres absolute (ATA), with 10-minute breaks, for a total treatment time of 140 minutes. Brain MRI showed global atrophy, a left frontal white matter infarct, and an old, right basal ganglia infarct, with computed tomography angiogram and carotid ultrasound unrevealing.

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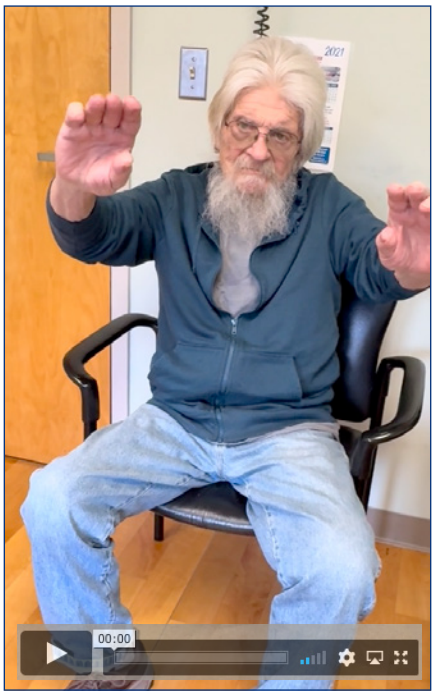

Video 1. https://vimeo.com/1191560659 [0.27]

Video 2. https://vimeo.com/1191560657 [0.536]

In the following two weeks, his gait and mental status improved, approaching baseline, but was followed by deterioration of both cognition and gait, which progressed for about four months, after which he again showed mild improvement.

In the clinic, he was alert but offered no spontaneous speech, although naming, clock drawing, handwriting, speech, and memory were normal. He exhibited marked bradykinesia and rigidity. Deep tendon and Babinski reflexes were absent. He had a stooped posture, a widened base, very small stride and needed support [**Videos 1, 2]** to walk.

Despite the lack of brain MRI abnormalities, this patient suffered delayed neurological sequelae (DNS) of CO poisoning. DNS after CO poisoning occurs in 15–40% of patients,[1] with a biphasic clinical course, where patients show a period of recovery followed by neurological symptoms typically developing two to 40 days after the exposure (sometimes even later).[2] Mild symptoms of CO toxicity include headaches, nausea, and lightheadedness, whilst severe symptoms include neurological deficits or coma.[3] Cardiac ischemia may also occur with severe toxicity.[4] Permanent movement disorders such as parkinsonism, as seen here, are not uncommon.

Predictors of DNS include loss of consciousness (LOC), low Glasgow Coma Scale, COHb >25%, long exposure , and increased serum troponin.[2] Current guidelines suggest immediate administration of normobaric oxygen.[5] HBO is indicated for patients with severe symptoms such as LOC, any neurological deficits, or signs of cardiac ischemia. COHb >25% in adults without severe symptoms may also qualify for HBO. Pediatric and pregnant patients are at increased risk and have looser criteria for HBO.[5] Standard protocol includes administration of three sessions within the first 24 hours, followed by reevaluation and further daily sessions if required.[5] Best results from HBO are when it is administered within six hours of exposure.

An alternative to HBO when it is not available is oxygen delivered with high flow nasal cannula (30 liters/min or more). This reduces the half-life of COHb to 50 minutes and may show reduce the risk of DNS. The length of treatment with this method is not known, but would likely be 24 hours or longer to match HBO.[6]

The efficacy of HBO therapy for long-term cognitive impairment is debated, but all authorities recommend early administration. Recognition of possible delayed neurological symptoms after initial improvement must be considered when providing a prognosis.

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Disclosure

The patient has signed a consent form to have this Video published.

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