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Coma Reversal and Functional Recovery after Hyperbaric Oxygen Therapy and Rehabilitation in the Subacute Phase of Carbon Monoxide Poisoning: A Case Report and Literature Review

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Hyperbaric oxygen (HBO) effectively decreases the morbidity and mortality of carbon monoxide poisoning. Immediate HBO treatment is indicated as it yields a more rapid and greater neuropsychological recovery. HBO significantly decreases the half life of carboxy hemoglobin (COHb) and increases the tissue clearance of residual carbon monoxide, thus decreasing the neuropsychological deficit. HBO enhances the outcome even in the subacute phase of CO poisoning as in this case. A thirteen year old girl was admitted to our hospital in a deep coma after severe CO poisoning from gas leakage and was referred for HBO treatment one month after carbon monoxide poisoning. She was administered and received the second course of HBO 8 months later. Ten months after carbon monoxide poisoning, she regained consciousness but remained mute. After four weeks of rehabilitation she started talking. Her speech was coherent and relevant. However, her thought content was childish and superficial. She became partially independent in daily activities. Based on this case study, we highly recommend HBO therapy and intensive rehabilitation in the management of an acute and subacute CO poisoning patient with serious neurological sequelae. (J Rehab Med Assoc ROC 2000; 28(3): 163 – 170 )

Key words: hyperbaric oxygen, normobaric oxygen, carbon monoxide poisoning, activities of daily living

INTRODUCTION

Carbon monoxide poisoning accounts for the majority of accidental and suicidal morbidity and mortality worldwide [1-2], including Taiwan [3]. Hyperbaric oxygen (HBO) therapy yields a more rapid and better neuropsychological recovery [3-10]. Thus, immediate HBO treatment [11-13] is preferred. However, the end point of the therapeutic window is unclear. The following question arises: when does HBO become ineffective in managing carbon monoxide poisoning? Previous investigations have examined coma reversal and neuropsychological recovery after HBO therapy in the subacute phase [14-17], up to one month of CO poisoning. In this article, we report on a thirteen year old girl who regained consciousness from a...
deep coma about ten months after carbon monoxide poisoning and regained speech about fourteen months after carbon monoxide poisoning. She received two courses of repetitive HBO and intensive rehabilitation.

**CASE REPORT**

On August 19, 1997, a thirteen year old girl was admitted to the Emergency Department of our hospital after severe carbon monoxide poisoning. She had been found unconscious with urine and stool incontinence. Her family had left her unattended for four hours before discovering her condition. She had not attempted suicide. After advanced cardiac life support ACLS, 100% normobaric oxygen was administered via endotracheal tube. During hospitalization, she had experienced some complications: such as upper gastrointestinal bleeding, increased intracranial pressure, seizure and pneumonia; they were dealt with expeditiously. After remaining in a deep coma throughout her one month hospitalization, she was transferred for HBO therapy with GCS: E,M,V. She received two courses of repetitive HBO in October 1997 and May 1998, respectively.

Around June 1998 she regained consciousness, but remained mute and had minimal facial expressions. She gradually improved and could eventually cooperate a little. She could comply with keeping her nasogastric tube on. She responded to simple commands such as “grasp my hand” and “Close your eyes”. Hence, she began intensive rehabilitation on September 25 1998.

Upon admission, she was a pleasant girl, capable of maintaining good visual contact but having a poor attention span. She was restrained to a wheelchair to prevent catapulsion caused by extension spasticity in her legs. She had no head, trunk or limb control. Her deep tendon reflexes were generally increased with bilateral positive Hoffmann and Babinski signs, as well as sustained ankle clonus. Her feet showed severe planter flexion and supination tightness. She was double hemiplegic with her left side weaker and less coordinated than the right. Her hand functions were limited to a weak and coarse grasp below the shoulder level. She manifested emotional incontinence with a loud, prolonged cry under minimal stress.

The patient then underwent intensive comprehensive rehabilitation with physical, occupational and speech therapies and therapeutic games. A task-oriented approach was adopted. Our long term rehabilitation goal was social reintegration in a wheelchair if she does not regain ambulation. Our short term goal was to increase her mobility and independence in activities of daily living, thus improving her quality of life. Therapy involved stretching her tight ankle joints and soft tissues with modalities such as ultrasound when required. Standing activities were undertaken on tilt able, and progressed to static standing with gaiters. Psychosocial stimulation was also included in her therapy. After four weeks of rehabilitation, she started talking and singing in a low voice. Her other functions also improved. For instance, she could stand with support for forty minutes. She could push a wheelchair while walking with trunk support for about twenty meters with much coaxing. She could perform light hygiene tasks, as well as groom and feed herself with a spoon. She could make very simple additions. However, she remained infantile with poor insight, foresight and coping strategies. She had low tolerance for physical or psychological stress, poor motivation and required much coaxing. Thus, more laborious tasks or evaluations failed. Bladder training by intermittent clean catheterization failed even though she had an upper motor neurogenic bladder. She would cry when seated on the toilet despite coaxing and explanations as to the benefits and pleasures of voiding control. Because her family could not participate in her rehabilitation program, she was discharged partially independent in ADL.

**DISCUSSION**

Carbon monoxide is a colorless, odorless and tasteless gas produced by incomplete combustion of hydrocarbon such as faulty heating systems with poor ventilation, and exhaust fumes in a closed garage. Methylene chloride in a paint remover when adsorbed through the skin is converted to carbon monoxide, possibly causing covert cumulative carbon monoxide poisoning. Thus, CO can kill overtly as in flames or covertly as when the patient unintentionally inhales a large enough dose acutely or a small dose on a long cumulative basis. CO causes widespread tissue damage, most critical in the
central nervous system and the cardiovascular system. CO poisoning may cause cardiac arrest, cardiac ischemia (as in this case) and dysrhythmia. In the central nervous system, CO can cause coma, high cortical dysfunction, e.g. apraxia, agnosia, aphasia, extrapyramidal dysfunction such as choreoathetosis and seizures, and behavioral dysfunction such as psychosis, and abulia apathy. Mild CO poisoning produces ‘Flu’ like non-specific symptoms and signs such as headache, confusion, clumsiness, and hysteria. Thus, carbon monoxide can harm or kill overtly or covertly, acutely or produce late sequelae. CO coma is a unique type of anoxia, and is often followed by recurrent or delayed neurological deficit in 40% normobaric oxygen (NBO) treated subjects, and occurs two to forty days after initial CO poisoning.21-23

Table 1. Relevant data

<table>
<thead>
<tr>
<th>Emergency ward</th>
<th>BP</th>
<th>100/40 mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>RR</td>
<td>24/minute</td>
<td></td>
</tr>
<tr>
<td>GCS</td>
<td>E2M2V1</td>
<td></td>
</tr>
<tr>
<td>Pupils</td>
<td>4.4.5 mm</td>
<td></td>
</tr>
<tr>
<td>L/R +/-</td>
<td>Sluggish</td>
<td></td>
</tr>
<tr>
<td>COHb</td>
<td>62.2% (then down to 1% after 100% normobaric oxygen therapy)</td>
<td></td>
</tr>
<tr>
<td>Glucose</td>
<td>413 mg%</td>
<td></td>
</tr>
<tr>
<td>PH</td>
<td>7.4</td>
<td></td>
</tr>
<tr>
<td>K+</td>
<td>2.8 mEq/L</td>
<td></td>
</tr>
<tr>
<td>CKMB</td>
<td>37U</td>
<td></td>
</tr>
<tr>
<td>CK</td>
<td>&gt;1818 U</td>
<td></td>
</tr>
<tr>
<td>LDH</td>
<td>862 U</td>
<td></td>
</tr>
<tr>
<td>EKG</td>
<td>ST depression in V4 V5</td>
<td></td>
</tr>
<tr>
<td>EEG</td>
<td>Severe diffuse cortical dysfunction</td>
<td></td>
</tr>
<tr>
<td>Brain CT</td>
<td>Diffuse brain edema</td>
<td></td>
</tr>
</tbody>
</table>

Brain CT (fig 1,2) (Eight days later) Intracerebral hemorrhage in bifrontal areas and gray-white matter junctions
SPECT 1997,12/8 Bifrontal perfusion defects compatible with hypoxic encephalopathy
SPECT 1998,10/30 Resolution of bifrontal perfusion defects
MR T2 images 1997,12/10 Bilateral symmetric confluent high intensity in the periventricular white matter, centrum semiovale, may comply with demyelination
(fig 3, 4) 
MR T2 images 1998,10/24 Decrease in extent of above mentioned bilateral high intensity. Increase bilateral ventricles including 3rd ventricle
(fig 5, 6)

Table 2. Summary of the patient’s clinical course

<table>
<thead>
<tr>
<th>Date</th>
<th>Physical findings and symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1997/8/19</td>
<td>E2M2V1 Emergency Department</td>
</tr>
<tr>
<td>1997/9/22</td>
<td>E2M2V1 ≤ 1st Course HBO</td>
</tr>
<tr>
<td>1998/4/27</td>
<td>E2M2V1 ≤ 2nd Course HBO</td>
</tr>
<tr>
<td>1998/6 around</td>
<td>Awakes from coma, mute, spastic double hemiplegia(left&gt;right)</td>
</tr>
<tr>
<td>1998/9/24 10 months</td>
<td>Begin rehabilitation</td>
</tr>
<tr>
<td>&gt; CO poisoning</td>
<td>Mute, no control of head and trunk, severity left &gt; right</td>
</tr>
<tr>
<td>1998/10/21 &gt;1 month</td>
<td>Talks</td>
</tr>
<tr>
<td>Rehabilitation</td>
<td></td>
</tr>
<tr>
<td>1998/10/27 &gt;6 weeks</td>
<td>Discharged from Rehabilitation Dept.</td>
</tr>
<tr>
<td>Rehabilitation</td>
<td>Talks + ADL partially independent, pushing wheelchair for a short distance</td>
</tr>
</tbody>
</table>
SPECT in our patient displayed severe bifrontal perfusion defects that partially resolved with her clinical improvement, which correlates with other reports [24-25].

T2 weighted MR images [26] in delayed encephalopathy of CO poisoning patient reveal bilateral symmetrical confluent high signal intensity in the peri-ventricular white matter and centrum semiovale. This high intensity signal extends through the corpus callosum, subcortical U fibers, external and internal capsules. Ischemia or necrosis of the globus pallidus are also observed in severe CO poisoning. Bilateral symmetrical putamen and thalamus ischemia may also be noted. Such changes decrease with clinical improvement. The above findings imply that CO-related delayed encephalopathy is a reversible demyelination [29]. Our case also demonstrated the above reversible demyelination changes (Fig. 3-6). Globus pallidus ischaemia is the hallmark of ischemic hypoxic process in arterial border zone found on CT scan [27]. Ginsberg [28] postulated that the morphological changes represent gradations in intensity of a pathological process rather than distinct pathologic processes.

The exact pathophysiology of carbon monoxide mediated poisoning is complex and not well understood [5,20]. The relative affinity of carbon monoxide for hemoglobin is two hundred times that of oxygen [9]. Thus, carbon monoxide significantly decreases the oxygen transport and delivery at tissues. However, cellular oxygen supply disturbance by itself can not account for CO poisoning. Haldane [29] in 1927 demonstrated that animals breathing oxygen at 3 atmosphere absolute (ATA) could be poisoned at sufficiently high CO levels. Such poisoning could occur even though these animals were totally oxygenated under hyperbaric conditions with adequate oxygen dissolved in their plasma to support all their metabolic needs. Neither can cytochrome system dysfunction elucidate CO poisoning. In 1951, Ball [30] found that the cytochrome A3 oxidase system preferentially combines with oxygen in a 9 to 1 ratio over CO in beef heart extract. Thus, understanding the pathomechanics of CO poisoning appears to be through defining CO biochemistry in vivo. However, the exact biochemical changes of CO poisoning remains unclear. Related investigations have pointed out the involvement of ischemia related brain damage [27-28]. Thom recently indicated that CO mediated brain injury [32] is at least partly a leucocyte-mediated post-ischemic reperfusion injury. CO mediated endothelial dysfunction attracts leucocytes PMN which adhere to microvasculature. PMN proteases then convert xanthine dehydrogenase to xanthine oxidase, subsequently forming oxygen radicals [30]. Doing so causes lipid peroxidation and subsequent cellular damage and neurological deficit. Thus, evidence suggests that CO-mediated brain injury is both an ischemic and post ischemic anoxic reperfusion injury [27-28,31-32]. Whether CO directly or indirectly aggravates the ischemic and post ischemic reperfusion autacatalytic lipid peroxidation cell damage requires further elucidation of in vivo CO biochemistry.

HBO is more beneficial than NBO in terms of treating a wide range of CO poisoning [5-10]. Immediate HBO therapy is preferred to achieve optimum results [11-13]. HBO may be beneficial, even in the subacute phase of CO poisoning as in our case and others [14-17]. HBO decreases the half-life of CO to 18 minutes under 2.5 ATA [7]. Thom [37] demonstrated one way in which HBO could antagonize CO-mediated brain lipid peroxidation, subsequently reducing brain damage. His in vitro results indicated that HBO could functionally inhibit PMN \( \beta_2 \) integrins, thereby interfering with the ischemia–reperfusion autacatalytic lipid peroxidation pathway. Tomaszewski et al [38] also revealed that HBO prevents hippocampal damage in rats. HBO also results in faster and more extensive recuperation. HBO therapy is becoming an increasingly accepted means of treating CO poisoning, particularly when severe neurologic and cardiac dysfunctions occur [22]. However, the optimal protocol must still be defined. Problems encountered by HBO therapy as oxygen toxicity [39] related seizure risk 0.3% at 2.45 ATA, barotrauma to ear drum, claustrophobia can be all dealt without interfering with HBO therapy.

### CONCLUSION

This case study has demonstrated that a satisfactory outcome can be achieved with aggressive HBO and intensive rehabilitation, even in the subacute phase. The optimal protocol of HBO therapy in CO poisoning must be defined to increase the effectiveness of HBO treatment. Ideally, a network should be set up to provide a scientific patient selection and service provision. Helicopter transport
Fig 1 and 2  Intracerebral hemorrhage bifrontal areas and gray-white matter junctions (8 days later).

Fig 3 and 4  MRI, T2 images (1997/12/10). Bilateral symmetric confluent high intensity in the periventricular white matter and centrum semiovale may comply with demyelinatin.

Fig 5 and 6  MRI, T2 images (1998/10/24). Decrease in quantity of bilateral periventricular white matter change. Dilated bilateral ventricles including 3rd ventricle (white arrow).
and even transportable HBO system could be added if necessary.\textsuperscript{10} Prevention of accidents with grave sequelae such as those occurred after CO poisoning should be minimized by more effective public and health education. This case report further reiterates what rehabilitation enhances quality of life, regardless of how poor the initial prognosis was. Whether rehabilitation facilitates functional recovery by enhancing neuroplasticity needs further study.

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一氧化碳中毒患者在亚急性期接受高圧氧及復健治療後之復甦與功能恢復：病例報告及文獻回顧

王寵惠 溫月英
台北市立忠孝醫院復健科

高壓氧療法能有效地減少一氧化碳中毒之致病率與死亡率。急性期在立即使用高壓氧後有助於神經生理功能之恢復。

高壓氧能有效地縮短一氧化碳血紅素之半衰期，增加組織對廢餘一氧化碳之廓清，以及減少神經心理方面之障礙。

對於一氧化碳中毒亚急性期的患者，高壓氧療法仍有其卓越的效果。在本病例報告中，一位13歲女孩，因嚴重一氧化碳中毒而深度昏迷，1個月後，她接受2段療程的高壓氧治療；10個月後，其意識逐漸改善，但仍舊無法說話。經過4週密集的復健治療後，她開始能夠說話，且其日常生活能力達到部份的獨立自主性。由此病例得知，即使是亞急性一氧化碳中毒的患者，在接受高壓氧療與積極的復健治療後，其功能仍能獲得相當明顯的改善。（中華復健醫誌 2000; 28(3): 163 - 170）

關鍵詞：高壓氧(hyperbaric oxygen)，常壓氧(nomobaric oxygen)，一氧化碳中毒(carbon monoxide poisoning)，日常生活能力(activities of daily living)