European Journal of Preventive Medicine
2021; 9(3): 75-78
http://www.sciencepublishinggroup.com/j/ejpm
doi: 10.11648/j.ejpm.20210903.11
ISSN: 2330-8222 (Print); ISSN: 2330-8230 (Online)

Explore of Hyperbaric Oxygen Therapy on the Patients with Acute Encephalopathy Scondery Myocardial Damage Following Carbon Monoxide Poising

Zhang Yanping[1, *], Zhang Pan[2], Lv Changchun[1]

[1]Department of Hyperbaric Oxygen, Central Hospital of Zibo Mining Group, Zibo City, China

[2]Emergency Department, Linzi Qidu Hospital, Zibo City, China

Email address:
zkzhyp@163.com (Zhang Yanping)

[*]Corresponding author

To cite this article:
Zhang Yanping, Zhang Pan, Lv Changchun. Explore of Hyperbaric Oxygen Therapy on the Patients with Acute Encephalopathy Scondery Myocardial Damage Following Carbon Monoxide Poising. *European Journal of Preventive Medicine*. Vol. 9, No. 3, 2021, pp. 75-78.
doi: 10.11648/j.ejpm.20210903.11

Received: April 14, 2021; **Accepted**: April 30, 2021; **Published**: May 14, 2021

Abstract: Objective: To investigate the myocardial damage and hyperbaric oxygen treatment of acute encephalopathy in CO poisoning. Methods: Since March 2015 - March 2020 admitted to 125 patients with acute CO poisoning encephalopathy, emergency admission GCS score 7-8 points or less, and the intracranial pressure, myocardial damage markers enzymes (CK - MB, CK, LDH, MYO, BNP) and troponin (cTnl), arterial COHb quantitative and electrocardiogram (EKG) dynamic inspection, row CT, MRI examination can at the same time, using air pressure cabin HBO treatment, Each patient choose 2.5-3 period of treatment (Every10 times is a course of treatment), 99.5-100% oxygen purity (liquid oxygen gasification), oxygen supply time 60 minutes or 80 minutes, interlude 10 minutes. Results: The higher the COHb, the deeper the coma, the increased intracranial pressure, the intracranial hypertension, and the morphological changes such as diffuse cerebral edema and symmetrical globulin necrosis were observed in 125 patients. However, the myocardial damaging enzymes and troponin markers were significantly increased and the EKG abnormalities were basically consistent. HBO treatment achieved remarkable curative effect, the incidence of delayed encephalopathy was significantly reduced, and no death occurred. Conclusion: Increased intracranial pressure induced by acute encephalopathy induced by CO poisoning can significantly increase the incidence of myocardial damage, subendocardial myocardial infarction, left heart failure and various arrhythmias. HBO is a treatment for etiology and should be preferred.

Keywords: CO Poisoning, Acute Encephalopathy, Myocardial Damage, HBO Treatment

1. Introduction

Acute carbon monoxide poisoning (ACMP) is a whole world problem. The mortality rate is highest in posioning diseases. Incidence rate is the forty thousand people every year in USA. Its mortality rate is in the first one. Incidence rate is high which industrial and nonindustrial occupation in china. Afterwards, decreases rate of the incidence and mortality had made remarkable. The carbon monxiide poisoning encephalopathy leads to increasd intracranial pressure and diffuse cerebral edema, resulting in severe damage of myocardium and its hear, easily leading to subendocardial myocardial infarction, acute heart failure, delayed encephalopathy and so on, which is the main cause of severe disability and death. How to treat the myocardical damage has one of the world, smost difficult problem. Belong to critical disease secondary to acute CO poisoning encephalopathy is the most typical clinical anoxic encephalopathy in a type, mainly located in parenchymal damage and become a diffuse, then appear vascular source sex brain edema, and cause of diffuse intracranial pressure or lead to increasing pressure in a coma, is in different parts and coronary artery myocardial damage is one of the reasons for the heavier. However, clinical practice is easy to be ignored, which is the key to a high case fatality rate [1]. From March 2010 to March 2020, a total of 551 cases of CO poisoning were admitted to our department, among which 125 cases of acute encephalopathy were treated with severe myocardial damage secondary to HBO treatment. The report is as follows:

2. Materials and Methods

There were 85 males and 40 females in this group, with an average age of 45.5Y, with a history of close contact with CO; Coma state: GCS score was 3-4 in 70 cases, 5-6 in 45 cases, and 7-8 in 10 cases. 13 cases of acute left heart failure; The duration of coma was 3h-17h, with an average of 4-5h; No history of coronary heart disease, other cardiomyopathy and various arrhythmias before poisoning.

2.1. Biochemical Tests of All Blood

In this group, arterial blood and venous blood were taken immediately before no oxygen inhalation after admission to the hospital, among which arterial blood was measured for COHb quantification. The venous blood was detected by troponin (cTnI), myocardial enzyme spectrum creatine kinase (CK), myocardial type creatine kinase (CK-MB) and lactate dehydrogenase (LDH), myoglobin (MYO)，brain natriuretic peptic (BNP).

2.2. Electrocardiogram and Intracranial Pressure Detection

The electrocardiogram (EKG) was recorded by 12-lead electrocardiogram machine and the dynamic changes of EKG were observed of cerebral spinal fluid (CSF) pressure was measured by lumbar puncture.

3. Results

(1) After CO poisoning, the COHb content was measured in time and ≥45-55%, the intracranial pressure was increased and the pressure was between 210cmH2O, the myocardial enzyme spectrum and troponin were significantly increased to ≥5-10 times of normal, and the abnormal changes of ECG were also significant. 170cm It is confirmed that the increase of intracranial pressure caused by CO poisoning acute encephalopathy is the main cause of inducing myocardial cell and its substructure damage, so the clinical myocardial damage, subendocardial myocardial infarction, acute left heart failure, various cardiac rhythm disorders, etc.

(2) After treatment with HBO for 2.5-3 courses (10 times for each course), the increased intracranial pressure was gradually reduced, and the myocardial enzyme spectrum and troponin in blood were also decreased, all of which were acceptable 5 to 13 days to reach normal; At the same time, 125 abnormal ECG cases also gradually returned to normal, among which 8 cases of 10 cases sustained myocardial insufficiency, which could basically return to normal within 7-15 days. 5 cases of atrial premature beats and 2 cases of ventricular premature beats; It also reduced the incidence of delayed encephalopathy.

4. Discussion

Effects of acute encephalopathy with CO poisoning on myocardial damage.

Acute severe CO poisoning can lead to the damage of the body's vital organs, among which the brain and heart damage are the most prominent. However, the diffuse brain edema induced by the brain hypoxic damage can produce high cranial pressure or intracranial hypertension, which is the main cause of the highest and serious secondary myocardial damage, which has rarely been reported in the literature. In this study, the coma time after poisoning was more than 3 hours, and the COHb quantification was 45% or more than 55%. The imaging results showed acute diffuse brain edema, necrosis of the globus pallidus area and great artery infarction, and typical brain morphologic changes were called CO poisoning acute encephalopathy [2]. All patients in this group met the requirements

In the diagnosis of CO poisoning acute encephalopathy, after the occurrence of encephalopathy, the myocardial damage is more serious and all the tissues in various parts of the heart can be involved. All of them were examined by myocardial enzyme spectrum, cTnI and EKG, and combined with clinical

Changes in neurological symptoms and signs were confirmed. In addition, it was found that the detection results and clinical manifestations of this kind of myocardial damage were significantly abnormal, which was significantly higher than those of the patients without encephalopathy. Therefore, the view that the myocardial damage caused by CO poisoning is only directly caused by toxicity is not perfect at present. Acute encephalopathy is the whole brain damage, in which hypoxic damage of thalamus, hypothalamus and brainstem is more obvious, easy to lead to severe myocardial damage. In this study, the incidence of rapid atrial fibrillation, acute left heart failure and subendocardial infarction were significantly increased, and the EKG pathological amplitude was also significantly higher. The other patients did not have acute encephalopathy and did not have subendocardial myocardial infarction or acute left heart failure, but had more arrhythmias mainly caused by mild myocardial ischemia or sinus tachycardia, which was still due to the absence of intracranial pressure changes in the early stage.

It has been proved to be closely related to the anatomical basis of the central nervous system controlling cardiovascular function. However, early clinical manifestations of patients with acute encephalopathy are mainly coma, vomiting, even dilated pupil, reduced or disappeared light response, and incontinence, etc., which are the clinical typical manifestations of elevated intracranial pressure or high intracranial pressure, although the duration is relatively short, usually within 24h-72h. But enough to make the hypothalamus of the plant nerve integration center damage, make sympathetic and parasympathetic nerve function disorder, release neurotransmitters, cause myocardial and cardiac function further damage, serious person lead to acute failure of cardiac function. At the same time, the blood-brain barrier function is obviously impaired, and diffuse vasogenic brain edema is more likely to occur in the early stage, which usually reaches its peak within 3-72h, and then causes the increase of cerebrospinal fluid pressure [3]. It is the basic cause of inducing extensive myocardial damage and subendocardial myocardial infarction or fatal left heart failure.

5. Biochemical Reaction of Myocardial Damage in Acute Encephalopathy

The diagnosis of myocardial damage secondary to CO poisoning is based primarily on the simple and rapid determination of blood enzymes

Spectrum, troponin and COHb examination results, because the level of some indicators in the examination results plays an important role in the incidence of acute encephalopathy, but these indicators are also directly related to the determination of the degree of myocardial damage caused by secondary acute encephalopathy. Although there is no specific diagnostic basis for certain enzymes, it is of great significance to determine the prognosis in the diagnosis and treatment of patients after poisoning. Through clinical observation in this group, cTnI is specific for myocardial infarction. The higher the measured cTnI, the wider the area of subendocardial infarction, and the more severe the patient's performance, and the more severe the cardiac insufficiency. However, the high concentration of COHb in the above indicators leads to diffuse vasogenic brain edema and increases in intracranial pressure is the key. It will inevitably lead to the increase of the enzyme profile and cTnI. The abnormal increase of myocardial enzyme profile and troponin is only a biochemical response to myocardial damage and subendocardial myocardial infarction, but not to others organ damage was of no significance. All patients in this group were in emergency treatment, and their GCS score was ≤8. Below, the oxygenated hemoglobin (Hb02) and COHb in the body under the condition of hypoxia must produce significant biochemical reactions, which can involve the myocardium and related different anatomical sites. That is, in acute encephalopathy, not only the oxygen carrying capacity of Hb02 decreased significantly, but also the release of oxygen and the disintegration of COHb also had some special changes, and the transport and release of oxygen were severely restricted. This is because the disintegration of competitively bound COHb in blood is more slow, and its disintegration intensity is significantly slower than that of Hb02, even more than 3,600 times, which significantly reduces the oxygen uptake of cardiac tissue and causes serious damage to the subendocardial myocardial tissue which is most sensitive to hypoxia, leading to an increased incidence of subendocardial myocardial infarction in our group. Myocardial structural protein of striated muscle at the same time, all significant damage to only intramyocardial cTnI release in the quantity is higher than in the normal range in the blood, through this kind of cTnI in the change of blood content, confirmed after the poisoning Hbo2 not only damage to normal function, muscle fibers and myocardial cell damage is more serious, and intracranial pressure high myocardial tissue hypoxia continues to play an important role in the process of poisoning. Through observation in this group, it was found that cTnI was significantly elevated in the blood of patients with subendocardial myocardial infarction or severe myocardial necrosis, or even acute coronary syndrome or myocardial infarction. However, cTnI was a highly specific marker of myocardial injury [4]. This has specific diagnostic value for subendocardial myocardial infarction or severe myocardial damage secondary to CO poisoning, and this biochemical index is closely related to the diagnosis and treatment prognosis.

6. HBO Treatment

Because of still not clarify of specific mechanism

Which is myocardium damage on acute carbon monoxide poisoing [5]. When acute encephalopathy is caused by CO poisoning, the basic pathology is diffuse vasogenic brain edema, which leads to acute intracranial hypertension or high intracranial pressure, which leads to severe myocardial damage and various arrhythmias. The incidence is high and has certain characteristics, and HBO is often chosen for treatment at present. In the group, HBO treatment was firstly selected. HBO treatment improved the blood oxygen concentration in the brain tissue, increased oxygen tension [6], and could rapidly and effectively reduce intracranial pressure [7], thus significantly improving the efficacy of myocardial damage and various arrhythmias. Due to elevated COHb levels within the normal red blood cells of Hbo2 oxygen function significantly reduced, and because of the combination of COHb are not of the protein denaturation, but configuration sexual union, and under the condition of high oxygen partial pressure COHb can disintegrate the attributes, can make the Hb to more oxygen, and cause the oxygen dissociation curve moves to the right, thus enhanced tissue oxygen supply. We believe that HBO is an effective treatment for the cause of the disease, blocking the vicious cycle of brain tissue edema and hypoxia [8]. Therefore, HBO treatment not only has a protective effect [9], but also has a positive effect. HBO treatment can further improve the increase of intracranial pressure caused by cerebral hypoxic cerebral edema, and promote the gradual recovery of myocardial fine structure. It is more important to improve the complete recovery of acute cardiac dysfunction. HBO also plays an important therapeutic role in increasing the physical dissolved oxygen in the body, which is helpful to increase the oxygen content in the tissue space and force the disintegration of free COHb in the tissue space, which is conducive to the recovery of tissue oxygen supply and myocardial damage as soon as possible and reduce the occurrence of delayed encephalopathy [10]. However, HBO reduces the duration of brain edema and maintains the stability of intracranial pressure by increasing the content of physical dissolved oxygen in the blood and increasing the repair of the damaged blood-brain barrier. At the same time, the relative increase of physical dissolved oxygen content in HBO treatment increases the blood oxygen content in myocardium myoglobin microcirculation as well as the oxygen content in tissues and cells, promotes the recovery of mitochondrial energy generation function in myocardium cells, and restores the physiological function of sodium potassium pump in myocardium membrane. Reduce the heart's dependence on HB2 to carry oxygen. The elevated myocardial enzyme spectrum can be reduced to normal or close to normal within a short time, usually within 3-5d [11].

However, in this group, not only the enzyme spectrum decreased to normal after 4-5 days, which was slightly longer than reported in the literature, and was associated with increased intracranial pressure. However, the ECG also recovered accordingly. It is suggested that HBO is the only rapid and effective method for the treatment of CO toxic myocardial damage, myocardial infarction and various arrhythmias. At the same time, after receiving oxygen from the heart cavity, the capillaries of the myocardium can get sufficient oxygen supply, which can reduce the viscosity of exudation and plasma, and can quickly correct local acidosis. The gradual decline of intracranial pressure to reduce the sympathetic nerve excitability is conducive to the restoration if membrane potential polarization and depolarization function of cardiac cells to normal, more important is to improve the rhythm of the sinoatrial node, reduce the excitability of the ectopic rhythm point, which is the key to correct various arrhythmias. Because binding CoHB can be rapidly dissociated and excreted under 2.2ATA pressure conditions, the overall cardiotoxicity is rapidly reduced, and the repair of cardiomyocytes and cardiac physiological conduction are enhanced. Therefore, a short period of hyperbaric oxygen treatment can make cTnL, CK, MYO, BNPand CK-MB rapidly and significantly reduced or close to normal, and ECG changes significantly improved or normal, which is a sign of the recovery of damaged cardiomyocytes, thus further supporting the unique advantage of HBO in the treatment of myocardial damage [12]. However, the patients with scattered patchlike necrosis or subendocardial myocardial infarction, or even descending coronary artery infarction, could be treated in time by entering the capsule, while the patients with acute cardiac insufficiency chose a good time for entering the capsule and were treated with HBO actively. Therefore, not only no fatality rate occurred in this group, but also good clinical efficacy was achieved.

References

[1] Gao Chunjin, Ge Huan, Zhao Liming, et al. Guidelines for Clinical Treatment of Carbon Monoxide Poisoning (Compilation). Chinese Medical Association of Hyperbaric Oxygen Medicine. 2012, 2.

[2] Zhang Yanping, He Changling, Xie Yingli, et al. New classification of carbon monoxide poisoning encephalopathy and clinical hyperbaric oxygen therapy [J]. China Coal Industry Medical Journal, 2015, 10 (18) 1677.

[3] Xiang BJ, Wang BJ, Xue H, et al. Relationship between elevated cerebrospinal fluid pressure and delayed encephalopathy caused by carbon monoxide poisoning in acute stage. Chinese Journal of Neuropsychiatric Diseases. 2015, 5 (41): 269. [J].

[4] Wang M, Dai YX: Protective effect of hyperbaric oxygen on myocardial acute carbon monoxide poisoning [J]. Journal of Tongling Polytechnic, 2015, (4), 27.

[5] Huang Chunhua, Guo Yan, Zheng Qinliang et al. Effect of hyperbaric oxygen combined with sodium fructose diphosphate on myocardial damage in children with acute carbon monoxide poisoning [J]. Chin J Marine Med & High Pressure Medicine, 2020, 27, (6) 707.

[6] Liu Hongmei.. Effects of hyperbaric oxygen on cerebral hemodynamics, serum NT-pronBP and intracranial pressure in patients with severe head injury [J]. Chinese Journal of Practical Medicine, 2017, 44 (13) 24.

[7] Qiu Lili, Hu Yinghua, Feng Keyu. And so on. Study on the relationship between myocardial injury and serum inflammatory factors in acute carbon monoxide poisoning, Industrial Health and Occupational Diseases, 2013, 39 (6) 349.

[8] Fan Pengtao, Yang Fei, Liu Baocheng, et al. Effects of hyperbaric oxygen on CAT/SOD/GSH-Px and Nrf2 in rats with craniocerebral injury [J], Journal of Neuroanatomy, 2017, 33 (3): 283.

[9] LIU bin, WANG Xia, XU Jinyuan, et al. Hyperbaric oxygen combined with alprostol on carbon monoxide poisoning.

[10] Clinical study of toxic myocardium injury [J], China Emergency Medicine, 2019, 39 (1): 42.

[11] Cui jian hua, Wu Peifeng, Gao Liang, et al. Effects of hyperbaric oxygen preconditioning on the ultrastructure of heart, brain and lung in acute hypoxia rats [J]. Chinese Journal of Marine Medicine and High Pressure Medicine, 2014, 21 (3): 380.

[12] Zhou HM, Li JK, Zhao XH, et al. Effect of Danhong injection combined with sodium phosphate creatine in the treatment of myocardial injury caused by acute carbon monoxide poisoning, Journal of Modern Integrated Traditional and Western.

[13] Jun YS, LeeJS, Min YG, et al. Carbon monoxide-induced Cardiomyopathy [J]. Cire J, 2014, 78 (6): 1437-1444.