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Case Report
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Rare Inducement of Syndrome (GBS) - Carbon Monoxide Poisoning: A Case Report and Literature Review

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Abstract

Carbon monoxide poisoning is a public health event of global concern, and the disability rate of delayed encephalopathy is 10% to 30% among surviving patients. However, relatively little attention has been given to its symptoms of complicated nerve root and peripheral nerve injuries. Although the incidence of Guillain-Barré syndrome (GBS) is low, the disease progresses rapidly, and the prognosis is poor. There are still many inducing factors that have not been found; "poisoning" is relatively rare; GBS induced by carbon monoxide poisoning has not been reported abroad at present; and the diagnosis and treatment process are more likely to be missed or misdiagnosed. This document reviews the diagnosis and treatment of a patient with GBS induced by carbon monoxide poisoning and reviews the relevant literature, hoping to determine the possibility of GBS complications or "poisoning" in the diagnosis and treatment of GBS in clinical work and avoid missed diagnoses or misdiagnoses.

Keywords: Carbon monoxide poisoning; Guillain-Barré Syndrome (GBS); MRI

Case Presentation

A 62-year-old male patient was found unconsciously by his family in a closed charcoal-burning room at 02:00 on December 25th, 2020. After approximately 10 min, he recovered consciousness without convulsions, vomiting or incontinence. He was then sent to the emergency department of the Affiliated Hospital of Zunyi Medical University by his family; "acute carbon monoxide poisoning" was considered; and he was actively given hyperbaric oxygen, nutrient nerves, steroids and other treatments. Head MRI revealed multiple demyelinating lesions in the bilateral white matter of the brain. Because of the patient without limb weakness, cognitive impairment or other symptoms, the doctor decided to continue original treatment plan. On Day 7 after poisoning (December 31st, 2020), the patient developed right eyelid ptosis, an oblique mouth, and cough even when not drinking water. On Day 12 after the poisoning (January 1st, 2021), the left limb gradually weakened, and the left index finger was limited in dorsiflexion. The next day, he gradually progressed to progressive weakness of his limbs, unstable walking, inability to walk independently, and inability to hold objects, but he no obvious numbness or pain in his extremities. He was in good health in the past; denied a history of diabetes, vaccination, trauma or surgery; and denied a recent history of diarrhea and infection. Physical examination revealed the following: Clear consciousness, normal hearing, air leakage at the right corner of the mouth, slight limitation of eye closure, symmetrical existence of the bilateral frontal and nasolabial grooves, middle extension of the tongue, muscle strength of limbs of Grade 3, normal muscle tension, disappearance of the limb tendon reflex, negative pathological signs and no obvious hypoesthesia. Auxiliary Examination: On January 07th, 2021, the MRI imaging of the spinal cord show no abnormalities. Electromyography revealed abnormal SEPs in the radial nerve, ulnar nerve, posterior tibial nerve and common peroneal nerve in both upper limbs. The cerebrospinal fluid test was performed, showing a pressure of 110 mmH_2O; a white blood cell count of 1×10^6/L was found in the CSF; the fluid was clear and transparent, and the protein concentration was 397 mg/L. Because the separation of cellular proteins in the cerebrospinal fluid of patients is not obvious, further detection of peripheral nerve antibodies in the patient's blood and cerebrospinal fluid revealed positivity for the anti-sulfatide antibody IgG and the anti-GM1 antibody IgG. In summary, the patient was diagnosed with Guillain-Barré syndrome (GBS) and carbon monoxide poisoning. Due to gastrointestinal bleeding after hormone therapy and after hospitalization and the family members refused human immunoglobulin therapy, the patient was given comprehensive treatment, such as mecobalamin, hyperbaric oxygen, acupuncture and rehabilitation. After treatment, when the patient was discharged on January 28th, 2021, the prolapse of the right eyelid and corners of his mouth had recovered, the muscle strength of his proximal

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Table 1: Reports of peripheral neuropathy secondary to carbon monoxide poisoning in China and abroad.

Investigator	Year	Literature Type	Peripheral Nerve Involved	Diagnostic Method	Occurrence Time (After Poisoning)	Prognosis
Renfert H Jr	1955	Case Report	Sciatic Nerve Root	EMG	6 Weeks	Sensory Abnormalities Confined to the Knee after 10 Months
Snyder RD	1970	Case Report	Facial Nerve/Ulnar Nerve	EMG	4 Weeks	Unreported
Choi IS	1982	Retrospective Study	Nerve of Lower Extremity	EMG	Unreported	Basic Recovery in 3-6 Months
Thomas A. Joiner	1990	Case Report	Unilateral Phrenic Nerve	Chest X-ray	2 Days	12 Days
Rao Shouxin	1994	Case Report	Right Common Peroneal Nerve	EMG	1 Days	Complete Recovery in 2 Years
Chen Xuelin	2004	Case Report	Ulnar Nerve/Median Nerve	EMG	12 Hours	Complete Recovery in 2 Months
Gi-Young Park	2018	Case Report	Unilateral Brachial Plexus	Bone Imaging/ Electromyography	2 Days	Unreported

Table 2: GBS-induced "Poisoning" factors.

Investigator	Publication Year	Age (Years)	Gender	Disease Onset Area	Disease Onset Season	Poison Types	Poisoning Mode	Speculation Mechanism	Symptoms Appeared after Poisoning	Prognosis
Chusid JG	1946	None	Male		7 Months	mustard gas	Purification Pit Explosion	Mustard Gas Contact on Tissue that Activated a Local Virus	55 Days	In the prior 5 months, all symptoms resolved except the disappearance of the deep reflex
Yongan W	1976	12	Male	Jiangxi	7 Months	Toadstool	Oral	Not Conjectured	3 Days	Recovered after 1 month
Fisher JR	1977	28	Male		Summer	Organophosphorus (merphos)	Farming Skin Contact	Cholinesterase inhibition	4 Days	The patient could walk with external force at approximately 6 weeks
Peter D. Donofrio	1987	60-63	Male (3 Cases)		Unreported	Inorganic Arsenic	Take by Mistake/ Murder	Demyelination	6 Days to 2 Weeks	Undescribed
Romain K. Gherardi	1990	26	Female	Central Africa	3 Months	Melarsoprol (Organic Arsenic)	T. gambiense Treatment	Loss of Spinal Cord Anterior Horn Cells	11 Days after the First Treatment	He died 68 days after the first treatment
Pang KA	1993	39	Male	Majorca	7 Months	Jellyfish Sting	Hang Out	Venom Immune Response	1 Weeks	Nerve conduction was normal after 3 months
Chuang TY	1996	36	Male	Taiwan	7 Months	Gold-banded Krait Venom	Bite Wound	Residual Bungarotoxin Binds Silently to Receptor Site	4 Weeks	Reliable Walking with Crutches for 4 Weeks
Arushi Gahlot Saini	2012	6	Female	India	Unreported	Bees	Sting	Bee Venom Protein Immune Response	3 Days	Walking independently at 20 weeks, not running.
Dra. Graciela M. Gaioli	2020	14	Male	Argentina	Unreported	Mercury	Grinding fluorescent lamps in the patient's yard	Degeneration of front end and axon of direct acting neurons	4 Weeks	Complete recovery after 2 months
Hailing Liu	2021	46	Male	Guangdong	Unreported	Thallium	Poisoning	Direct Action	30 Weeks	Recovered 25 months

upper limbs had recovered to grade 5, the muscle strength of his distal upper limbs was grade 4, the muscle strength of both his lower limbs was grade 4, his muscle tension was normal, and the tendon reflex had disappeared.

Discussion and Literature Review

GBS is considered to be an immune-mediated acute multiple peripheral neuropathy with myelinated nerve involvement that often involves nerve roots and peripheral nerves. According to the diagnostic criteria for GBS proposed by the National Institute of Neurological Diseases and Stroke, the main clinical manifestations are progressive symmetrical retardation, tendon reflex weakening or disappearance, etc. In addition, symptom progression from several days to four weeks, associated with cranial nerve injury symptoms and signs, autonomic nerve or sensory dysfunction, "protein-cell separation" in cerebrospinal fluid, and EMG detection meeting GBS standards can be used as supporting features [1]. For patients with atypical symptoms or atypical Cerebrospinal Fluid (CSF) and Electromyography (EMG) results, imaging or autoantibody detection is required to support the diagnosis of GBS and exclude other diseases. The complication of carbon monoxide poisoning—"delayed encephalopathy of carbon monoxide poisoning", which is widely considered—mainly involves

the core system, extrapyramidal system and other parts, but little attention has been given to whether nerve roots and the peripheral nervous system are involved. At present, there are no reports of carbon monoxide poisoning complicated with GBS abroad, but only one case has been reported by Qi Qi [2] in China. However, there are many differences between the cases reported in this paper and those reported in Qi: (1) The disease development process of this case is not typical: The first symptom of this case involved cranial nerve involvement on Day 7 after the poisoning, and unilateral limb paralysis first occurred approximately two weeks after the poisoning. The next day, the paralysis had progressed to quadriplegia with no sensory disturbance, so it was easily misdiagnosed as "stroke" in the early clinical stage. (2) The phenomenon of "protein-cell separation" in cerebrospinal fluid was not obvious: in this case, the protein in cerebrospinal fluid was at the upper limit of the normal value, and there was no typical protein-cell separation phenomenon of GBS. Considering that this may be explained by the findings in the literature report showing that only 50% of GBS patients can have obvious protein separation one week after symptoms appear [3], GBS could not be completely ruled out in clinic, and other related auxiliary tests need to be further checked up similar to this case to confirm the diagnosis. This patient was clearly diagnosed by further examining his Cerebrospinal Fluid (CSF) anti-GM1 antibody concentration and electrophysiological electromyography. (3) GBS is more likely to occur in this case: According to epidemiological studies, the incidence of GBS increases by nearly 20% with the increase of age from 10 years old, male patients are more than female patients [4], and age plays an important role in the study of significant correlation factors of carbon monoxide poisoning complicated with nerve injury [5]. Therefore, this patient is more prone to GBS. (4) There are many factors related to the poor prognosis of this case, but the outcome is good. According to the literature [6], it is reported that there are 5 risk factors for adverse prognosis, and this patient meets two of them: Age >60 years old and time from symptoms to admission of less than 7 days, categorizing the patient into a high-risk patient. However, this patient has a good outcome and prognosis; which may be related to the comprehensive treatment with hormones and hyperbaric oxygen due to carbon monoxide poisoning in the early stage. This measure is also the preferred treatment for this syndrome and could also benefit patients.

At present, little attention has been given to peripheral nerve injury caused by carbon monoxide poisoning, and most of these studies involve case reports. According to a large-scale clinical study of peripheral nerve injury caused by carbon monoxide poisoning abroad, of the 2,759 cases included, 23 patients were diagnosed with peripheral neuropathy through electromyography and nerve conduction research, and the incidence of peripheral neuropathy was 0.8% [7]. Although its incidence is low, it has been reported in relevant studies in China and abroad that its long-term functional recovery time is long (Table 1). It has a great impact on the recovery of patients' physical and psychological functions and society and family and economy. Thus, it is worth asking clinicians to raise awareness of this kind of complication in their usual work. In addition, some patients with carbon monoxide poisoning, such as those with mental disorders, long durations of coma, autonomic nervous dysfunction as the main manifestation in the early stage, and symptoms similar to "stroke", are more likely to be overlooked and missed, and may even be misdiagnosed as having other diseases in clinical work; therefore, clinical workers should increase their awareness of such complications.

The mechanism by which GBS is caused by carbon monoxide poisoning has not been identified at present, and this topic is worth further exploration. In this paper, it is speculated that the following mechanisms may be involved. (1) According to the autoimmune theory, the blood-nerve barrier is destroyed after carbon monoxide poisoning, and the myelin sheath of myelinated nerves is damaged, which leads to the release of a large amount of myelin protein into the blood under physiological conditions. It has neuro-antigen specificity and can be recognized to induce an immune cascade reaction, produce demyelinating lesions and cause nervous system symptoms [8]. (2) Hypoxia–reperfusion theory: It is extremely difficult to dissociate the carboxyhemoglobin that is formed after carbon monoxide poisoning, and hypoxia affects the metabolism of tissues and cells. After the organism breaks away from the poisoning environment, normal pressure oxygen or hyperbaric oxygen treatment improves the hypoxia state, leading to a pathological state of hypoxia-reperfusion. When oxidative stress causes the production of a large number of free radicals, nerve cells can be damaged or die, and corresponding nervous system symptoms appear.

Most of the precursor factors of GBS are related to upper respiratory tract or digestive tract infections. In addition, vaccination, trauma, surgery, systemic lupus erythematosus and Hodgkin's lymphoma are also rare inducing factors of GBS [9], but many inducing factors are still unknown. Poisoning, a common public health event in the emergency department, is a relatively rare inducing factor of GBS that is not given enough attention clinically and is easy to miss or misdiagnose, which has a significant influence on its clinical diagnosis and treatment. At present, it has been reported in China and abroad that the factors that induce GBS are mustard gas, snake venom, bee stings, mushrooms, jellyfish, mercury, thallium, phosphorus, arsenic and other metallic substances (Table 2). Additionally, clinicians should be alerted to patients with GBS symptoms. If the diagnosis and treatment process, treatment effect and prognosis are not as expected, they should be alert to rare incentives such as "poisoning". Additionally, for poisoned patients, clinicians should be alert to complicated GBS in the diagnosis and treatment process. Although the incidence of this disease in these patients is low, some patients experience progression and poor prognosis.

In summary, the author has the following thoughts: (1) Because ordinary clinicians have a deep understanding of delayed encephalopathy caused by carbon monoxide poisoning but have little understanding of the secondary peripheral nerve injuries and nerve root injuries caused by carbon monoxide, which are easy to misdiagnose or miss in the clinic, should the treatment guidelines for carbon monoxide poisoning provide corresponding diagnosis and treatment guidance for such patients? Is it necessary that delayed encephalopathy be the focus in such patients? Everyone should have a more comprehensive and in-depth understanding of carbon monoxide poisoning. (2) Although the incidence of clinical GBS is low, "poisoning" is the focus of clinical public health events, and it is less of a concern as an inducing factor of GBS, which is more likely to be missed or misdiagnosed clinically. Therefore, it is necessary to guard against the situation of "poisoning" complicated with GBS in clinical diagnosis and treatment. (3) The mechanism of GBS induced by this rare induction is still unclear and needs further study and discussion to make the diagnosis of GBS more extensive and comprehensive.

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