Nitrous Oxide Poisoning Complicated by Syringomyelia: a Case Report

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Case report

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Abstract

Background: Nitrous oxide is a colorless, aromatic, inert organic gas with low toxicity and is currently abused in many recreational areas. Nitrous oxide abuse can lead to vitamin B12 deficiency, which can lead to neurological damage such as spinal neuropathy and peripheral neuropathy. Its abuse has not been associated with syringomyelia.

Case presentation: We described the case of a 21-year-old young girl who presented with progressive weakness in both legs and numbness in four limbs after excessive recreational inhalation of nitrous oxide and her imaging findings were considered syringomyelia.

Conclusion: Nitrous oxide abuse may be associated with syringomyelia, and that patients with nitrous oxide abuse should be further performed cervical and thoracic MRI to determine whether they have syringomyelia and give timely treatment.

Background

Owing to nitrous oxide can make people feel relaxed, happy and even hallucinate when inhaled, and it is easily available, so the abuse of nitrous oxide among young people has increased gradually in recent years. It is reported that nitrous oxide has become the seventh most popular drug abuse in the world by the 2016 World Drug Survey ^[1]. Long-term use of nitrous oxide can cause damage to the nervous system, including myelopathy, subacute combined degeneration (SCD) and peripheral neuropathy, and may even lead to paralysis ^[2]. There were few reports of nitrous oxide poisoning complicated with syringomyelia in the past. Here, we report a young girl who presented with progressive weakness in both legs and numbness in four limbs after excessive recreational inhalation of nitrous oxide and her imaging findings were considered syringomyelia.

Case Report

Our patient, a 21-year-old woman without significant past medical history, presented to the neurology with complaints of progressive weakness in both legs, numbness in four limbs and reduced the sensory function of the bladder. Physical exam was notable for positive bilateral Babinski sign, level 4 bilateral lower limb muscle strength, and weakened tendon reflex of limbs. Relevant social history included frequent consumption of large amounts of nitrous oxide for recreational. There were multiple peripheral nerve injuries, involving all motor sensory fibers, mainly motor fiber myelin with axonal involvement in her electromyography. No abnormalities were found in the head Magnetic Resonance Imaging (MRI) plain scan (Fig.1). Cervical spine MRI scan showed strip T2WI and STIR high signal shadow and low signal shadow of T1WI in C6-7 level spinal cord, considering syringomyelia (Fig.2). Psychological scale: Clinical Dementia Rating was 0.5, Mini-mental State Examination was 28, Montreal Cognitive Assessment was 23. Since the patient's education level was 13 years, it is considered that the patient has mild cognitive impairment. During the period of admission, Patients were mainly given vitamin B₁₂, vitamin B₁, rat nerve

growth factor and other neurotrophic drugs, and told to stop inhaling nitrous oxide. After 9 days of treatment, The patient was discharged after improvement in limb strength and numbness.

Discussion

Nitrous oxide poisoning can lead to hypoxia followed by syncope, heart attack, and even fatal events such as suffocation^[3]. Nitrous oxide does not bind hemoglobin in the blood but only dissolves in physical form in the blood. Inhaled nitrous oxide with vitamin B₁₂ (also known as cobalamin) after contact, cobalt atoms are oxidized and inactivated, and the inactivated cobalamin recovers slowly^[4]. Patients with normal vitamin B₁₂ levels usually develop neurological syndromes after prolonged, repeated exposure to nitrous oxide, while patients with subclinical vitamin B₁₂ deficiency may develop myelopathy after only one abuse of nitrous oxide^[5]. The neuropathologic changes in the spinal cord involved area mainly include initial swelling of the myelin sheath around the axons of nerve cells, followed by demyelination and axonal degeneration. It has been reported in many literatures that the nerve damage caused by nitrous oxide is mainly SCD affecting the posterior cord of the spinal cord, which may include ataxia, loss of sense of posture and vibration, and unstable gait^[6]. The corticospinal tract may also be involved, leading to weakness, spasms, hyperreflexia, clonus, and positive Babinski sign^[7]. Peripheral and optic nerves may also be involved. MRI of the spine showed T2-weighted posterior cord reverse v-shaped high signal characteristics^[8], and similar cases of nitrous oxide poisoning with syringomyelia have not yet been reported.

Syringomyelia (SM) is a chronic, progressive disease characterized by tubular cavity formation and gliosis in the spinal cord. Its lesions are mostly found in the cervical spinal cord. Most lesions are located between C2 and T9, but may extend down to the medullary cone or up to the brain stem (the bulb). The onset is insidious and progressive. At present, the causes and pathogenesis of SM are not completely clear. There are congenital developmental abnormality, cerebrospinal fluid dynamics abnormality, blood circulation abnormality and other theories. The pathogenesis can be divided into congenital SM and acquired SM. The most common congenital causes are neural tube defects (myelomeningocele and tetanus syndrome) and Chiari malformations. Acquired SM is thought to be caused by disturbance of normal cerebrospinal fluid circulation. There are many causes of acquired SM, including hydrocephalus, infection, inflammation, trauma, spinal cord tumors, extramedullary tumors, arachnoid cysts, spinal stenosis, etc.^[9]. In this case, syringomyelia is thought to be associated with nitrous oxide poisoning, but cannot be determined. Since nitrous oxide with syringomyelia has not been previously reported, this paper provides some evidence for the possibility of abuse of nitrous oxide with syringomyelia.

To sum up, Inhaled nitrous oxide abuse is common and should be given high clinical attention because it is a treatable and possibly reversible cause of myelopathy. While healthy young people in subacute or chronic have disease of the nervous system performance, a history of nitrous oxide abuse should be considered, such as the symmetry of the distal limb numbness, weakness, tendon reflex disappears, pathological character, etc. At the same time, the physician should give patients with cervical and

thoracic MRI in order to make clear its related to whether the acquired syringomyelia. In order to avoid irreversible damage of the nervous system, the patient should stop contact with harmful substances and be treated with drugs as soon as possible.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Written informed consent for publication was obtained from the patient. A copy of the written consent is available for review by the editor upon request.

Availability of data and materials

Data has not been made accessible in the interest of protecting the patient's privacy.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

LT is first author, who is responsible for the manuscript draft and revision. CY is responsible for the design. ZW is responsible for medical history inquiry. WZ is responsible for critical revision of the manuscript for important intellectual content, study supervision. All authors contributed to the drafting of the manuscript and approved the final manuscript.

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Figures

Figure 1

MRI plain scan

Figure 2

Cervical spine MRI scan showed strip T2WI and STIR high signal shadow and low signal shadow of T1WI in C6-7 level spinal cord, considering syringomyelia