

Nitrous Oxide Induced Subacute Combined Degeneration

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Abstract

Background: Nitrous oxide (N₂O), due to its euphoriant properties is often abused for recreational purpose. **Case Report:** A 38-year-old male who admitted to inhaling recreational nitrous oxide more than five months, presented with a progressive symmetrical tingling sensations and weakness of limbs. Serum vitamin B₁₂ was lower than normal. Magnetic resonance imaging (MRI) of the spine demonstrated hyper-intensity of the posterior cord extending from C1-C5. Nerve conduction velocity test showed slowed motor conduction velocities and delayed or absent F-waves consistent with demyelinating neuropathy. The patient was managed with B₁₂ supplement and his nerve function basically returned to normal after three months. **Conclusion:** This is a rare case of subacute combined degeneration caused by excessive inhalation of nitrous oxide.

Keywords: Demyelinating Diseases, Magnetic Resonance Imaging, Neural Conduction, Nitrous Oxide, Vitamin B12.

Introduction

Nitrous oxide (N₂O) is used commonly for surgical procedures and as a propellant in the food industry [1]. However, due to its euphoriant properties, N₂O now is often abused for recreational purpose. Very few cases of the neurological syndrome caused by excessive inhalation of N₂O were reported [2-6]. We report the clinical presentation, electrophysiological examination results and radiological manifestations of N₂O induced subacute combined degeneration.

Case Report

A 28-year-old previously healthy male presented with progressive inability to walk properly and symmetrical numbness and tingling sensations in his distal extremities within nearly six days. He also complained difficulty to perform fine tasks with his hands. He felt cervicodorsal tingling sensations prior to the onset of symptoms. He had no sphincter dysfunction and other autonomic symptoms and any history of trauma or recent illness was denied.

He admitted to inhaling recreational N₂O in the form of small pressurised canisters for one year. His consumption of N₂O was greatly increased over the last five months and approximately 800 grams (100 whippits) for once or twice a week. Ten days before the symptoms onset, he used 1200 g at a time. Except a history of pneumothorax operation, no special past medical history and family history were recorded.

On examination his cognition, cranial nerve function and muscle tone were normal. He had symmetrical distal weakness of the lower and upper limbs along with a glove and sock-like hyperpathia. He walked with a broad-based gait, and Romberg's sign was positive. Deep tendon reflexes were normal. Position and vibration sense were impaired in both upper and lower extremities. The Babinski's sign was positive.

The results of thyroid stimulating hormone, coagulation testing, liver and kidney function, plasma glucose, routine blood test, routine urine

test, serum folate were normal. Serum vitamin B₁₂ was low at 150.24 pg/mL (normal range = 180-914 pg/mL). Homocysteine was high at 62.9 μmol/L (normal range = 5-15 μmol/L). He underwent magnetic resonance imaging (MRI) scan of his spinal cord, which revealed hyper-intensity of the posterior cord from C1-C5 [Fig.1-4]. No abnormalities were seen on MRI brain. Nerve conduction studies showed slowed motor conduction velocities and delayed or absent F-waves, consistent with demyelinating neuropathy. Based on the history, clinical presentation, image findings and laboratory tests, he was diagnosed as subacute combined degeneration (SCD) caused by N₂O abuse.

The patient was treated with high-dose intravenous vitamin B₁₂ supplementation (1500 μg per day) for the first two weeks. His balance and gait improved gradually as well as the involuntary movement after treatment. With rehabilitation, he could walk independently slowly. After the discharge, the patient has continued to be treated with oral vitamin B₁₂ supplement (500 μg three times a day). After three months, the patient's symptoms basically returned to normal by telephone follow-up.

Discussion

For more than 150 years, N₂O has been widely applied in dentistry and surgery for its analgesic and anesthetic properties. It has lately gained popularity as a recreational drug, and its use is widespread. Acute poisoning of N₂O can cause low blood pressure, lung injury due to hypoxia, and even asphyxia. Chronic poisoning mainly presents as anemia and neurological damage. At present, most countries have not included N₂O under the category of drug administration.

Several case studies have reported neurological damage caused by N₂O abuse, including cognitive impairment, peripheral nerve and spinal damage. In our case, the serum vitamin B₁₂ of patient was lower than normal, while, some



Fig.1-4: Magnetic resonance imaging (MRI) scan of spinal cord, which revealed hyper-intensity of the posterior cord from C1-C5.

case reports reported normal vitamin B₁₂ levels [3]. A normal vitamin B₁₂ level does not rule out the possibility of N₂O-induced subacute degeneration of the spinal cord, given that functional vitamin B₁₂ deficiency can occur in the presence of normal serum vitamin B₁₂ levels. Slowed motor conduction velocities and delayed or absent F-waves also contributes to the diagnosis of subacute combined degeneration. In this case, detecting raised homocysteine, which rely on normally functioning vitamin B₁₂ for their metabolism, can give crucial clues to the diagnosis [7-8]. To best of our knowledge, 19 cases of SCD caused by N₂O abuse have been reported till date. To date, evaluated with MRI and electrophysiological examination at same time, this is the first case of N₂O induced subacute combined degeneration patient reported in the mainland of China.

In European and American countries, the rate of N₂O abuse is as high as 15.8% among teenagers [9]. Although there's no exact statistics

about N₂O abuse in mainland of China, there's a rising trend of the patients who were diagnosed as N₂O poisoning in clinical setups. This phenomenon should attract the attention of relevant government departments, and related measures should be taken.

Conclusion

Although subacute degeneration caused by N₂O is rare, with the increase of N₂O abuse population, early identification of N₂O-induced neuropathy is critical for its property of being reversible.

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