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Case Report

Subacute combined degeneration of spinal cord due to chronic nitrous oxide abuse

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Abstract

Subacute combined degeneration (SCD) of the spinal cord is a known clinical manifestation of vitamin B12 deficiency. We present a case of a young patient with undifferentiated neurological symptoms secondary to a rare complication of chronic N2O cannister (laughing gas) usage for recreational purposes resulting in subacute combined degeneration (SCD) of the spinal cord.

Keywords:

Nitrous oxide; inhalant abuse; sub-acute combined degeneration

Introduction

Nitrous oxide (N2 O) is known to have potential for recreational abuse, although controversial debate regarding the toxic effects of such abuse continues. Clinically, it is widely used as an inhaled anaesthetic gas in operation theatres, analgesics and anxiolytics mainly in emergency department for various indications. Recently recreational abuse of N2O has become increasingly common, especially among adolescents age group between 16 to 24, but many of them lack awareness of the potential side effects associated with nitrous oxide. Our main aim was to highlight the clinical presentation and review the neurologic, psychiatric and medical consequences of N2O abuse.

Case History

An 18-year-old boy was brought to the Emergency department by his mother complaining of a 1-week history of worsening weakness of both legs, a tingling sensation in both hands and an unsteady gait. He denies any fever, bowel or bladder abnormalities, recent travel or recent trauma.

His past medical record was unremarkable, and he denied taking any alcohol or recreational drugs within the last 4 weeks. His initial vitals, ECG and GCS were stable. On examination, he had bilateral lower limb power of 4/5, an ataxic gait, decreased deep tendon reflexes, mildly decreased sensation in the lower limbs and a positive Romberg's test. The rest of the systemic examination did not show any significant findings. Bedside investigations include routine bloods /ECG /urine toxicology screen and ABG were within limits.

In view of ongoing neurological symptoms, a CT head was done to rule out raised intracranial pressure and space occupying lesion which was normal. A lumbar puncture was done which did not show any findings for meningitis or albumin cytologic dissociation suggestive of Guillain Barre syndrome. A further detailed neurological examination revealed loss of vibration and proprioception in bilateral lower extremities. An MRI of head and spine was done after discussion with the neurologist in view of symptoms which showed multi-level high density signal along the posterior column typically described as "Inverted V sign or Rabbit ear's sign" as shown in fig (A) and (B) suggestive of subacute combined degeneration of spinal cord. MRI head did not show abnormalities. Viral screening were negative. Later patient admitted that, he has been using

N2O canisters for euphoria for almost 6 months (but not in the last 4 weeks). Patient was treated with vitamin B12 supplements, neuro rehabilitation, anticoagulation for DVT prophylaxis and supportive care as per neurologist advice.

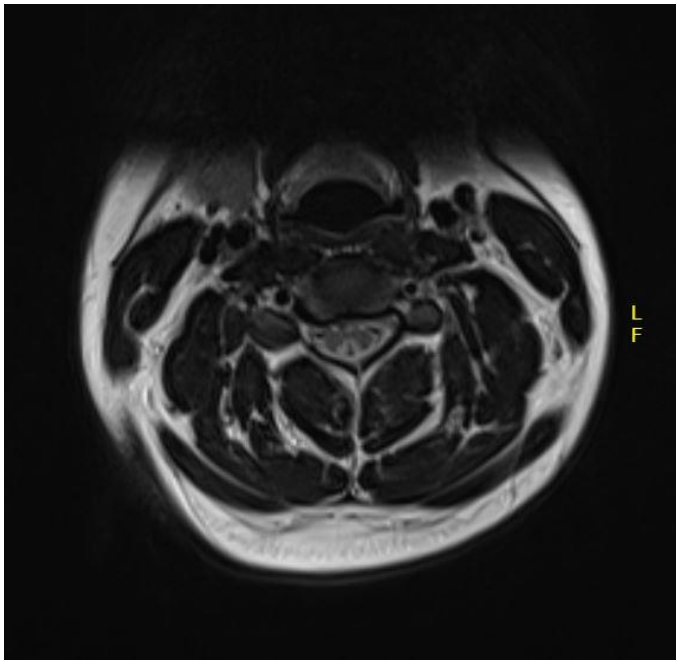

Figure A: MRI T2 showing inverted sign along the dorsal column of the spinal cord

Figure B: Typical inverted V or rabbit ear sign

Discussion

N2O (laughing gas) is an inhalational anaesthetic agent commonly used for dental procedures. Industrial use includes as a propellant in whip creams and as an engine accelerant. It works as an N-methyl-D-aspartate antagonist. Because of its additional opioid receptor activity, it results in analgesic and euphoric effects.[1] The recreational use of N2O is rapidly increasing especially among adolescents in the recent times.

As per the 2016 Global Drug Survey, 38.6% of N2O abuse occurred in the United Kingdom. The sale of nitrous oxide for recreational use is illegal in the UK under the Psychoactive Substances Act 2016. The UK Home Office estimates prevalence of nitrous oxide use at 2.3% in England and Wales, with the highest prevalence (8.8%) in the adolescent age group between 16–24 years.[2]

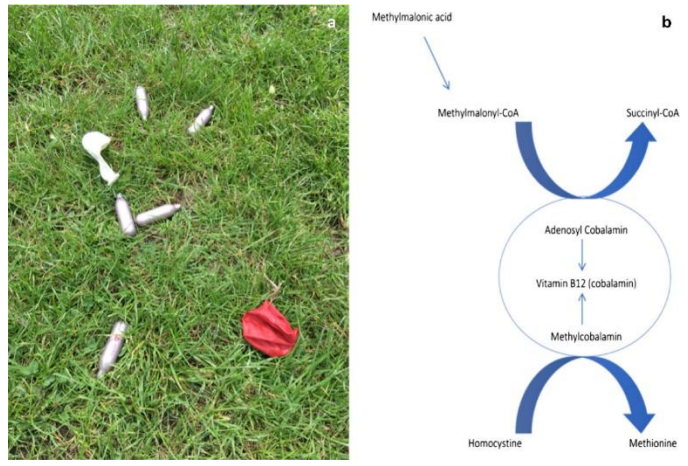

Figure C: Mechanism of N20 toxicity causing vit b12 deficiency and SCD

Nitrous oxide causes adverse neurological effects such as myelopathy, peripheral neuropathy, or Subacute combined degeneration (SCD) of the spinal cord. Clinical features include paraesthesia, gait disturbance, unsteadiness, or weakness. Psychiatric disorder and medical sequelae such as irritability, reduced sleep and anxiety also have been reported.[3] Nitrous oxide (laughing gas) binds to cobalamin which prevents conversion of homocysteine to methionine in vitamin B12 synthesis as shown in fig (C) which is required for myelination and thereby results in demyelination, neurological symptoms and myelopathy.[4]

Symptoms include patient describing as ‘pins and needles’, clumsiness of hand, ataxic gait, restlessness and agitation. Signs include decreased power in lower extremities, sensory ataxia, decreased or absent deep tendon reflexes

in bilateral lower limbs, loss of vibration and proprioception (posterior column signs) and a positive Romberg sign.[5,6]

Investigations include routine blood including vit B-12, homocysteine, folate levels and iron studies to rule out malabsorption and malnutrition which can cause vit B12 deficiency. Most people with subacute combined degeneration of cord will have low vit B12 levels but it can be normal in fewer population.[4] MRI spine is the investigations of choice which shows increased signal in the cervical and dorsal columns with inverted V sign or rabbit ears sign highly suggestive of subacute combined degeneration of spinal cord.[7]

Differential diagnosis includes HIV-1 associated myelopathy, neurosyphilis, Guillain-Barré syndrome, demyelinating (multiple sclerosis), nutritional copper deficiency and neoplastic (paraneoplastic syndrome) disorders should be considered.[8]

Treatment includes cessation of exposure, supplementation of vitamin B12 and neuro rehabilitation for SCD. Vitamin B12 therapy can be administered orally or as injections (IM) along with supportive care.[9] Few cases of agranulocytosis after 4 days of anaesthetic exposure and megaloblastic changes have been reported after 24hrs exposure. Pre anaesthetic considerations include – appropriate scavenging unit to limit environmental exposure, checking all fittings and tubes to prevent leakage, ensuring adequate operatory ventilation in theatre and air circulation (above 10/hr recommended), to ensure appropriate gas flow and proper mask fit, and, to ensure that just before procedure ends terminate N2O flow and administer 100% oxygen 5 minutes before removing the nasal hood.[10]

Conclusion

The incidence of N2O-induced neurotoxicity is increasing recently due to N20 cannister abuse particularly in younger generation and should be considered as an important differential diagnosis especially in young patients presenting with undifferentiated neurological symptoms.

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