Laughing with serious consequences- a case report of recreational laughing gas use with functional vitamin B12 deficiency

Authors: Mohammed Chowdhury[A] and Onesi Ogedengbe[A]

Introduction

Nitrous oxide (NOS), commonly known by various names including laughing gas, is a common psychoactive substance used recreationally in the UK and other parts of the world. Between April 2019 to March 2020, it was reported that 8.7% of 16–24-year-olds have used it recreationally (equivalent to 549,000 individuals). It was the second most used psychoactive substance after Cannabis used by 16–24-year-olds in this time period.[1]

NOS inactivates vitamin B12, an important co-factor in the enzymes which play a role in maintaining myelin sheath in nerve cells.[2,3] As a result, patients present with a range of different neurological symptoms, such as sensory and motor dysfunction, ataxia, spasticity and in extreme cases psychiatric problems.[4] These patients may have a normal vitamin 12 level when they present with adverse events related to NOS use.

Case presentation

We present a case of an 18-year-old young man who presented with a few months' history of progressive worsening of symptoms including recurrent falls, difficulty walking, bilateral loss of sensation and paraesthesia in lower limbs. He had been using multiple large cannisters of NOS on most weekends but had stopped a month prior to presentation. Neurological examination showed ataxic gait, Romberg test was positive, and he had decreased sensation below the knees bilaterally.

Initial bloods including folate and vitamin B12 levels were normal. He had a normal HBA1c, glucose and thyroid function. Plasma methylmalonic acid and homocysteine came back elevated (several weeks later, due to tests having to be done in external labs), a picture you would find in B12 deficiency.[5,6]

MRI imaging of brain and whole spine was unremarkable.

Due to lack of established guidelines for NOS related neurological complications, expert opinion was sought from neurologists. As per expert advice he was started on a regime of IM hydroxocobalamin initially daily for 7 days, followed by alternative day for seven days, followed by oral vitamin B12.

The patient had input from physiotherapists, occupational therapists and orthotics. At time of discharge, he had improved significantly and was able to mobilise with a Zimmer frame.

Discussion

Establishing the diagnosis of NOS related neuropathy can be tricky. Plasma methylmalonic acid and homocysteine levels which can help make a diagnosis have a long turnaround time.

Therefore, the diagnosis is dependent on a good clinical history and examination and exclusion of other probable diagnoses. Plasma methylmalonic acid and homocysteine levels are useful tests but may not be widely available in NHS hospitals.

Conclusion

There is a compelling duty to educate people of the consequences of recreational NOS use and an urgent need for experts to come together and produce an evidence-based treatment guidelines to ensure better outcomes as current treatment is only based on opinion of experts. We have therefore commenced a systematic review to provide some evidence that can contribute towards the development of treatment guidelines these patients. ■

References

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Authors: [A]Manchester Royal Infirmary, Manchester, UK