E-Poster

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Severe Neurological Complications of Chronic Nitrous Oxide Abuse: A Case Series

Topic: Health Conditions (Nervous System, Musculoskeletal System, Cardiopulmonary System)

Gonçalo Martins e Pereira[1], David Oliveira Cordeiro[1], João Ventura Luís[1], Sandra Palma[2], João Afonso Morgadinho[2], Marta Nascimento[2], Alyne Cordeiro[2], Augusto Rachão[2], Margarida Mota Freitas[3], Inês Mendes Ribeiro[3], Pedro Pereira[4]

[1]*Physical and Rehabilitation Medicine, ULSAS, Almada, Portugal*
[2]*Neurology, ULSAS, Almada, Portugal*
[3]*Physical and Rehabilitation Medicine, ULSAS, Almada, Portugal*
[4]*Neurology, ULSAS, Almada, Portugal*

BACKGROUND

Nitrous oxide (N_2O) is widely used for its anesthetic and analgesic properties, but its increasing recreational use poses serious neurological risks. N_2O toxicity is linked to vitamin B12 deficiency and demyelination, affecting both the central and peripheral nervous systems. Despite its growing clinical significance, literature on N_2O-induced neurological disorders (NI-NDs) remains scarce. This study presents a case series of severe NI-NDs, focusing on pathophysiology, clinical presentation, and treatment. It also highlights the role of Physical and Rehabilitation Medicine (PRM) in patient recovery.

METHODS

A retrospective case series was conducted, analyzing NI-ND patients treated at a Portuguese tertiary hospital from January 2021 to December 2024. Data were extracted from hospital records, assessing demographics, neurological and systemic manifestations, laboratory, imaging, and electrophysiological findings, as well as therapeutic strategies, functional outcomes, and the collaboration of PRM departments.

RESULTS

Eight NI-ND cases required hospital care (six hospitalized, one ER discharge, one outpatient). Patients were mostly women (75%), aged 17-28 years. Half worked in the food and beverage industry, while 25% were unemployed, and the rest were students. Only 50% had completed high school, and none had higher education. Three patients smoked tobacco, one used cannabis, and all had chronic N_2O use (average 2.88 ± 1.79 years), with five reporting increased recent consumption. All patients presented sensory deficits (100% proprioception; 87.5% temperature-pain) and gait disturbances, with symptoms progressing for 26.25 ± 40.63 weeks before seeking hospital care. Vitamin B12 deficiency was present in 75% absolute and 25% relative (average level of 191.25 ± 86.64 pg/ml), and all patients had homocysteine levels > 50 µmol/L (indeterminable). Additionally, three had folic acid deficiency, and two had macrocytosis. All magnetic resonance imagings (MRIs) showed T2 hypersignal in the posterior cervical cords. Electromyography identified axonal sensorimotor polyneuropathy in 71.4%, and all somatosensory evoked potentials showed central deep sensory pathway dysfunction. Transcranial magnetic stimulation in four patients revealed pyramidal tract dysfunction in 75%. All patients received vitamin B12 supplementation. All hospitalized patients were evaluated by the PMR team, and 75% continued treatment in PRM department, with one requiring rehabilitation centre admission. Functional status was assessed using the FIM, improving from 114 ± 18.42 to 124.38 ± 3.86 over six months.

CONCLUSION

Chronic N_2O abuse leads to severe neurological consequences, primarily due to vitamin B12 deficiency. Early recognition and multidisciplinary care, including PRM, are crucial for recovery. Despite functional improvement, some symptoms persist. Increased awareness, early screening, and patient education are essential to reduce long-term disability and improve patient outcomes.

Statement of Disclosures

None

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Corresponding Author:
Gonçalo Martins e Pereira
Physical and Rehabilitation Medicine, ULSAS, Almada, Portugal
Email: gmartinsepereira@gmail.com