

Subacute combined degeneration secondary to nitrous oxide abuse

BY MARK GORMLEY III

Vitamin B12 (cobalamin) plays an important role in various biochemical pathways, including hematopoiesis and oligodendrocyte growth.¹ Deficiency can lead to subacute combined degeneration (SCD) of the spinal cord, a condition due to multifocal demyelination, particularly affecting the dorsal columns and lateral corticospinal tracts.^{2,3} B12 deficiency can occur from dietary causes, pernicious anemia, and malabsorptive conditions, but may also be seen in nitrous oxide abuse. We report a case of SCD in a previously healthy young man due to nitrous oxide abuse.

Case report

A 32-year-old previously healthy man presented with bilateral lower-extremity numbness and weakness as well as numbness in his bilateral fingertips. The patient reported chronic injury-related joint pain and had used marijuana, Kratom, and “whippits” (nitrous oxide) for pain control. He had no previous medical or surgical history, was on no medications, and had no family history of neuromuscular disease.

On physical exam, he demonstrated full muscle strength throughout upper and lower extremities, except for 4+/5 on dorsiflexion and plantar flexion bilaterally. Sensation to light touch and vibration was diminished in his bilateral lower extremities starting at the mid-shin and worse distally. Pinprick sensation was intact throughout. Sensation of bilateral upper extremities was normal except for diminished light touch sensation in distal fingers bilaterally. Proprioceptive sense was absent in bilateral great toes. He had diffuse hyperreflexia and an extensor response (positive Babinski test). His gait was spastic and wide-based.

Further data revealed a normal basic metabolic panel, creatine kinase, liver functions tests, and a negative urine drug

screen. Complete blood count showed a normocytic anemia with a hemoglobin of 9.1 g/dL and MCV of 96 fL. B12 was low at 170 ng/L, and homocysteine and MMA were elevated at 99 μ mol/L and 5.2 μ mol/L, respectively. Peripheral smear showed hypersegmented neutrophils. No imaging was obtained.

Discussion

Our patient’s presentation is consistent with SCD due to B12 deficiency. Etiology of B12 deficiency can be broadly distributed into three categories: poor intake, malabsorption, and dysfunction.³ B12 dysfunction is the rarest of these three categories, but it is often related to heavy nitrous oxide (N₂O) use, as in our patient who was using high doses of whippits daily for three months.

N₂O oxidizes cobalamin’s essential co-factor, cobalt, rendering it non-functional.⁴ N₂O-related SCD typically develops in those with heavy whippit use, but it has been documented rarely after one-time nitrous oxide anesthesia as well.⁵

Diagnosis of SCD includes clinical signs of dorsal column and lateral corticospinal involvement. Absolute B12 deficiency may or may not be present in patients with N₂O use, as this causes a functional deficiency, and high homocysteine and MMA levels support the diagnosis.^{6,7} Spinal MRI is not necessary for diagnosis, but imaging may reveal T2-weighted hyperintensity in the posterior cord.⁸ In our patient’s case, the history of nitrous oxide abuse along with exam findings and low serum B12 confirmed the diagnosis without need for imaging.

Partial recovery is most common in patients with SCD, but full recovery is often seen when B12 replacement is initiated promptly.⁹ It can take months to years for maximal recovery, and this process requires the support of a multidisciplinary team. Our patient was started on intra-

muscular B12 injections and discharged to a rehab facility for ongoing physical therapy and support for nitrous oxide abuse.

Conclusion

Subacute combined degeneration of the spinal cord is a multifocal myelopathy related to B12 deficiency that typically affects the dorsal columns and lateral corticospinal tracts. It often presents in those with malnutrition or malabsorptive disorders, but should be considered in patients with sensory deficits and a history of nitrous oxide abuse. **MM**

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