



Subacute Combined Degeneration from Nitrous Oxide-induced Vitamin B12 Deficiency: A Case Report

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INTRODUCTION/BACKGROUND

The US SCI Model System's program is adding non-traumatic SCI to the model system program. As a result, it's critical that clinicians understand the spectrum of non-traumatic causes of myelopathy that may require intensive rehabilitation programs.

Subacute combined degeneration (SCD) is a well-described non-traumatic spinal cord injury of the dorsal columns. While most commonly associated with vitamin B12 deficiency (VitB12-Def), the mechanism of this acquired deficiency may vary. We present a case of SCD secondary to VitB12-Def acquired through nitrous oxide (N2O) abuse.

CASE REPORT

Brief History

A 37-year-old male presented in November 2022 with a 1-month history of progressive unsteadiness, inability to walk, and neurogenic bowel and bladder symptoms. The patient reported huffing two 700g commercial N2O canisters daily over 4 months to replace an alcohol dependency.

Pertinent Physical Exam Findings

- Severe truncal ataxia
- Absent lower limb vibratory sensation
- Absent lower limb proprioceptive sensation
- Spastic lower limb paraparesis
- Required moderate assistance of 2 for transfers

Pertinent Laboratory Findings

- Vitamin B12 : 158 ng/L (Ref >180ng/L)
- Methylmalonic acid: 0.76 mcmol/L (Ref <0.40 mcmol/L)

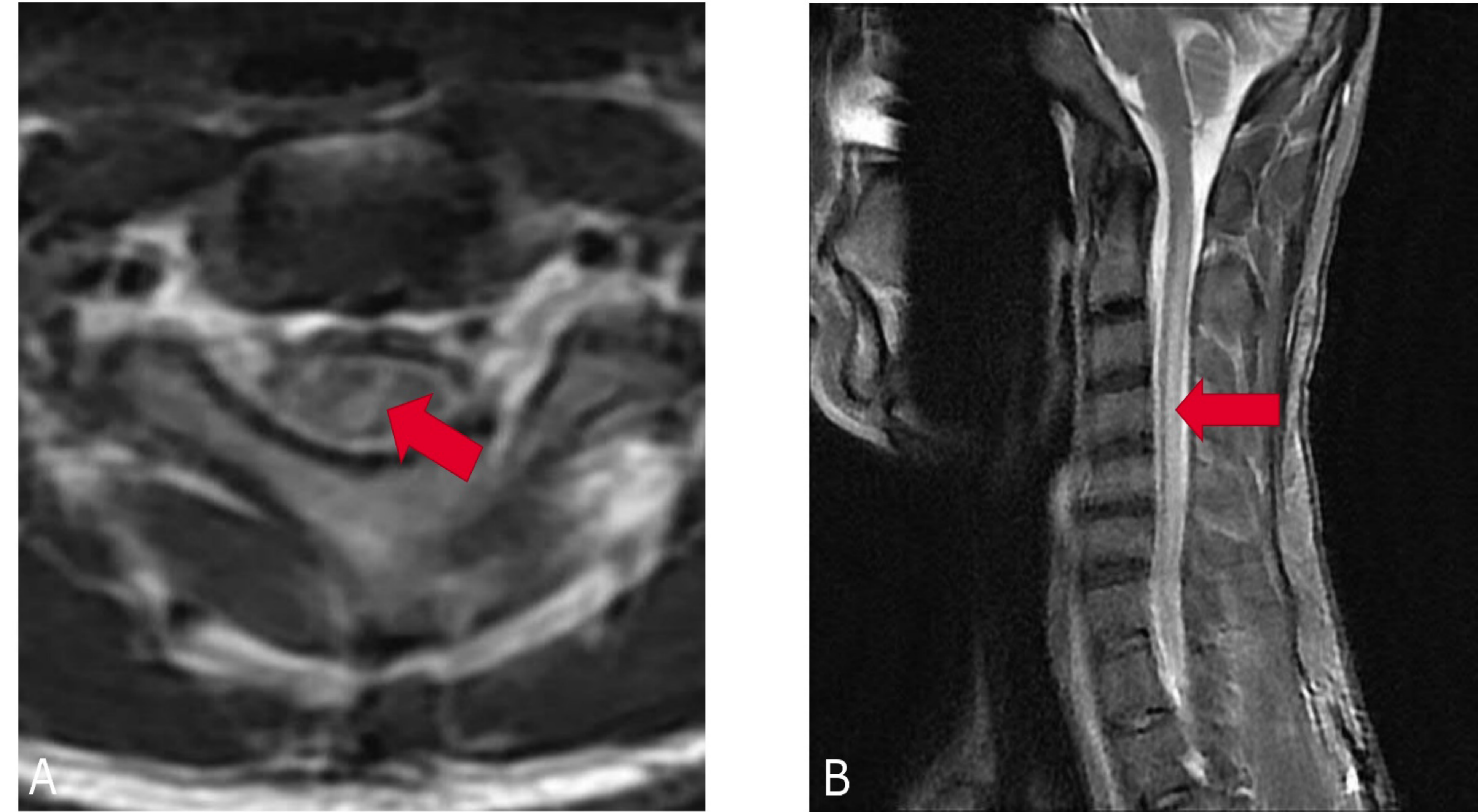


Figure 1: 2 Views Cervical Spine
A) Axial T2 & B) Sagittal T2 STIR featuring increased T2 signal within the dorsal columns of the cord which appears to extend caudally from approximately C2

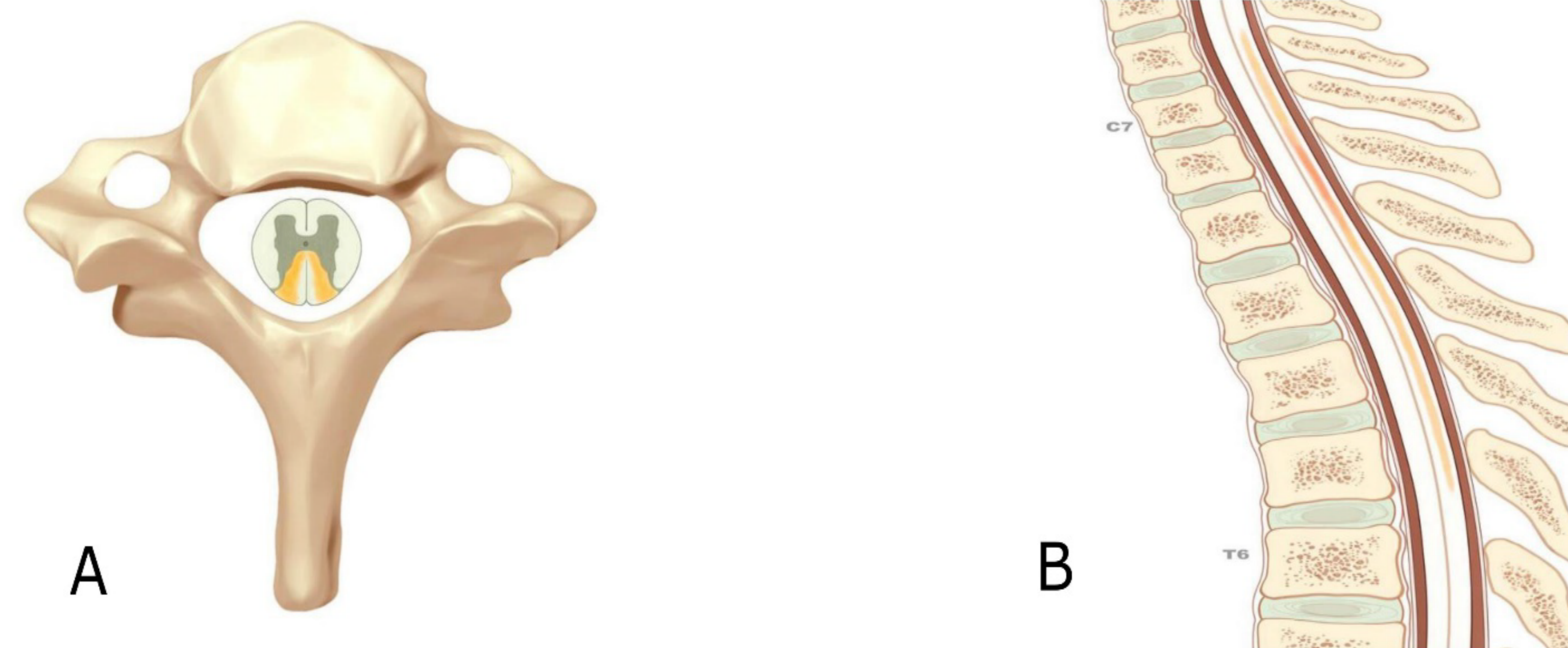


Figure 2: Subacute Combined Degeneration in the Dorsal Columns
A) Axial & B) Sagittal views depicting subacute combined degeneration within the dorsal columns (images retrieved from Mayo Clinic media database at mcmedia.mayo.edu)

TREATMENT

Patient was educated on strict N2O cessation and was given subcutaneous cyanocobalamin 1000 ug daily for 1 week, tapered to monthly injections.

After 28 days of inpatient rehabilitation, he was discharged independent with transfers and daily cares, and he ambulated using a front-wheeled walker. Three weeks later he ambulated with a quad cane.

CONCLUSION

N2O is a recreational drug that can induce SCD through vitamin B12 inactivation and myelin synthesis disruption. N2O abuse should be on the differential diagnosis for patients presenting with myelopathic symptoms and low vitamin B12 levels.

Treatment with N2O cessation, vitamin B12 repletion, and inpatient rehabilitation may provide functional benefits.

REFERENCES

