Symptomatic Vitamin B12 Deficiency Secondary to Inhaled Nitrous Oxide: A Case Report

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Case Description

A 36-year-old man, with a history of chronic inhaled nitrous oxide abuse, presented with two weeks of progressive paraparesis, leading to paraplegia, tingling, paresthesia, and pain, pedal edema, and erectile dysfunction. A year before, after repeatedly inhaling nitrous oxide, he had similar deficits, for which he did not seek medical care. He reduced his nitrous oxide use and self-administered B12 injections with symptomatic improvement. However, on his current presentation, he noted that despite self-administering B12 injections, his symptoms did not improve, unlike before, but he had not stopped inhaling nitrous oxide.

Laboratory results along with MRI imaging of the spine confirmed the diagnosis of subacute combined degeneration due to vitamin B12 deficiency. He was admitted and started on thiamine, vitamin B12, and folic acid. One week later, with the cessation of nitrous oxide, therapy, and B12 supplementation, he began to recover clinically with restoration of ambulation with assistance.

Figure 1 common inhalant

Discussion

Nitrous oxide is a chemical compound that can be found in many different molecular make-ups given environmental factors. At room temperature, it is a non-flammable and colorless gas that has euphoric effects upon inhalation. It is widely used in the medical community as an anesthetic but is often abused recreationally. Although it is considered a relatively safe anesthetic, there are extensive side effects including cardiac arrhythmias, hypoxia, and metabolic acidosis. Another serious side effect is vitamin B12 deficiency resulting in subacute combined degeneration. This case demonstrates a typical presentation of vitamin B12 deficiency in chronic nitrous oxide use with a progression from mild numbness and tingling of the extremities to lower extremity paralysis.

Conclusion

Vitamin B12 deficiency can result in subacute combined degeneration, which is the loss of myelin in the dorsal and lateral columns. This case follows the progression of symptoms of weakness with diminished vibration and light touch to a complete lower extremity paralysis with other upper motor neuron signs.

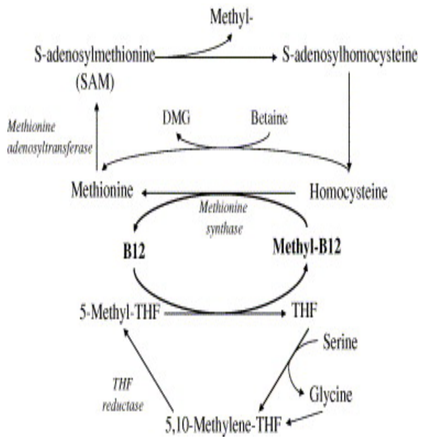

Figure 2 Metabolism of vitamin B12

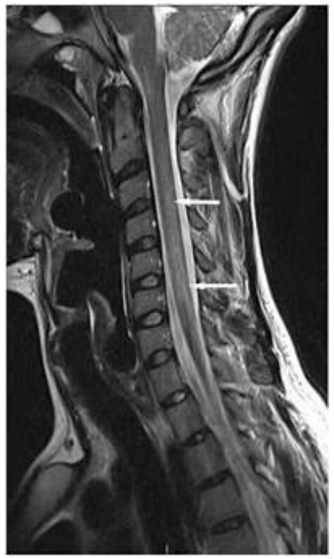

Figure 3 Spine MRI with increased signal density.

References

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