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Case Report

Enhancing Cord Lesions with Nitrous-Oxide Toxicity

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A 21-year-old man presented to our institution with 6-months of ascending lower-limb numbness which progressed over the initial 6 weeks and has remained static since. He sought medical attention at his local medical center 2 months following symptom onset. A cervical spine MRI revealed a longitudinal intramedullary cervical cord lesion with increased T2-signal and discontinuous dorsal enhancement following gadolinium administration (Figure A1-A3). Concerns regarding an inflammatory myelopathy prompted his treating physician to obtain a brain MRI with contrast and a spinal fluid study, both of which were unremarkable. Other serological testing from his home institution was reportedly negative. The lesion on the cervical MRI had an expansile, mass-like appearance (Figure A1,A3) and further diagnostic investigation was pursued with a spinal cord biopsy which showed marked macrophage infiltration with a few CD3 positive T-cells but no diagnostic features. Following the cord biopsy he developed transient erectile dysfunction. He was felt to have a spinal cord tumor with a non-diagnostic biopsy and referred to our medical center for further investigation.

A follow-up cervical spine MRI done at our institution, 6 months after the onset of his symptoms, showed a reduction in theintramedullary T2-hyperintensity and resolution of the contrast enhancement (Figure B1-B3). His exam at the time of our evaluation demonstrated mild toe extensor weakness, decreased vibration and position sense in the toes, a positive Romberg, and absent reflexes. It was discovered that his roommate had similar symptoms 6 months previously and that both he and the patient had been abusing nitrous oxide in the form of "whippets" with over 200 "hits" a day.At the time of our evaluation his serum vitamin B_{12} was 186 ng/l (normal: 180-914) and serum methyl malonic acid was 0.37 nmol/l (normal: <0.40 nmol/l). Serum intrinsic factor and neuromyelitisoptica antibodies were negative. His serum copper was normal.

Enhancing cord lesions are typically seen in inflammatory, infectious, neoplastic, and vascular disorders. Enhancement in B_{12}-deficiency-related myelopathy is very rare [1]. This case illustratesthat enhancing lesions can be seen in toxic myelopathies like those due to nitrous-oxide toxicity. The presence of enhancement therefore does not exclude toxic or metabolic etiologies.

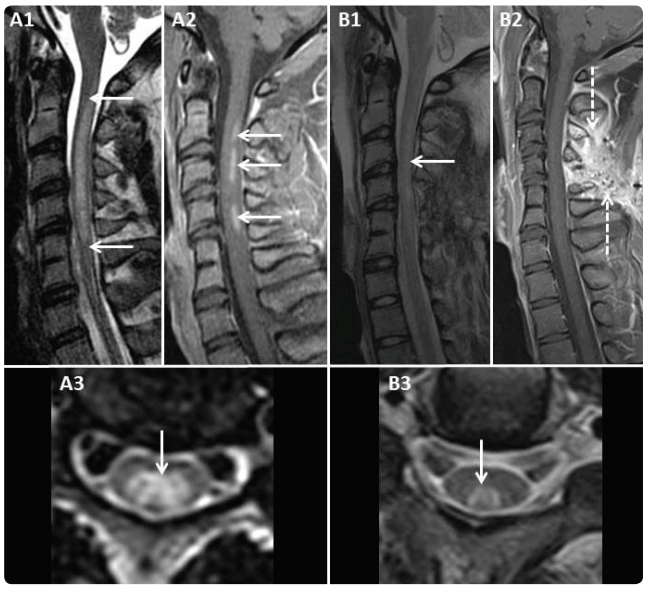

Figure 1: Sagittal (A1, A2, B1, B2) and axial (A3, B3) cervical cord MRIs showing a T2-hyperintensity from C1-5 (A1, A3) which shows multifocal enhancement (A2) 2 months after the onset of his symptoms. Imaging done 4 months laterat our institution shows a reductionin the T2-hyperintensity (B1, B3) and resolution the enhancement (B2). Also noted are post-operative changes related to the cord biopsy (dotted arrow).

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Nitrous-oxideoxidizes the cobalt core of cobalamin (vitamin B_{12}) and renders it inactivate by converting the functional reduced form to the inactive, oxidized form [2]. Nitrous oxide toxicity can therefore causes a myelopathy which is similar to that seen with vitamin B_{12} deficiency [3]. Individuals with subclinical B_{12} deficiency may be prone to develop deficits after more limited exposure. Neurological symptoms seen following nitrous oxide anesthesia has been referred to as "anesthesia paresthetica" [4].

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