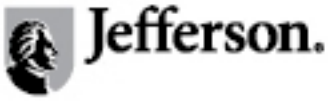

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Nitrous oxide myelopathy posing as spinal cord injury.

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Nitrous Oxide Myelopathy posing as Spinal Cord Injury

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ABSTRACT
INTRODUCTION: A patient presented with acute tetraparesis and a proposed acute traumatic spinal cord injury which was the result of nitrous oxide myelopathy.
PRESENTATION A 19 year old male sustained a traumatic fall off a 6 foot wall. His examination was consistent with a central cord syndrome with the addition of dorsal column impairment. Cervical MRI illustrated isolated dorsal column signal was suggestive of a non-traumatic etiology.
COURSE His symptoms resolved entirely over the course of 48 hours.
CONCLUSIONS Nitrous oxide abuse is increasing in prevalence. Its toxic side effects can mask B12 and folate deficiency and central cord syndrome. History and radiographic presentation are key to establishing a diagnosis.

Introduction

Central cord syndrome is a common presentations after cervical hyperextension injuries . Nitrous oxide(NO) is utilized as an anesthetic agent, however it is also available as a propellant in pressurized containers such as whipped cream. It is frequently abused for its euphoric and anesthetic effect.[8,24] The effects of nitrous oxide on the spinal cord have been reported in the literature,[4,6,7,15,16,18,19,25,31] however, the unique and insidious presentation after cervical trauma has not been documented. Since the abuse of nitrous oxide is increasing in prevalence, this may be an important mechanism for spinal cord injuries and should be considered in the differential in the acute care setting.

Case Report

A 19 year-old male presented with complaints of progressive numbness and weakness in all extremities after a fall over a six foot fence onto his back, one day prior. He denied back pain, bowel or bladder incontinence. Additionally, he denied any relevant past medical or surgical history. Over the course of

twenty-four hours, his symptoms progressed to an inability to ambulate and numbness in the distal extremities. The patient stated he occasionally abused oxycodone, alcohol, and "huffed" nitrous oxide canisters several times per week. Moreover, he stated he would "huff" as much as twenty pound canisters of nitrous oxide at one setting.

On initial assessment, the patient had minimal neck pain, was hemodynamically stable, and had a Glascow Coma Scale of 15. He was alert and oriented, without apparent cognitive or language deficits. On motor exam however, proximal weakness of 3/5 in the deltoids bilaterally and distally of 2/5 was found. In the lower extremities, the iliopsoas muscle groups were weak (4-/5) and distal groups were 3/5. Hyperreflexia was noted in the bilateral lower extremities. Sensation to light touch and pin prick was absent in the distal extremities as well as vibration and propioception. The sensation loss was not dermatomal. The remainder of the neurological exam was insignificant.

Investigations

Due to the history of NO abuse a vitamin B-12 and folate level were sent and returned within the normal reference ranges. A MRI scan of the cervical spine did not demonstrated any acute traumatic signs such as bony fracture, ligamentous injury, or subluxation. However, the spinal cord parenchymae had significant T-2 signal abnormalities in the posterior columns from C2-7. (Fig. 1-2)

Clinical Course

Intravenous methylprednisolone was started at an outside institution according to the NASCIS II criteria and since the patient was making progressive improvements was maintained. Thirty-six hours after injury, the patient noted complete resolution of his symptoms. The following day, the patient was discharged home.

Discussion

The National Institute of drug abuse has reported a significant increase in the number of nitrous oxide abuse cases- from 1.5 to 18 million across the span of 2000 to 2001.[31] Similarly there has been a decrease use as this drug as an anesthetic agent .[11,21]The biochemical effects of NO as an anesthetic agent have long been studied, but not entirely understood. [9,10,12,13,20,29,30] It's effects on the spinal cord have been recognized since 1959 by Randt, in laboratory studies in cats.[26]

NO abuse tends to present clinically with a predominant effect on the dorsal columns. Vitamin B12 deficiency and folate deficiency can exacerbate this effect.[28] This effect is hypothesized to occur due to the oxidizing effect of NO on the cobalt group of cobalamin.[31] In the presence of NO, the covalence of cobalt will change from monovalent to bivalent, which then reacts to irreversibly inactivate vitamin B12. The formation of tetrahydrafolate (THF) is halted by the further inhibition of methionine synthase, which requires B12. THF is vital in DNA synthesis, cell division, and ultimately myelination in the spinal cord. This biochemical pathway is hypothesized to be most vital in the posterior columns.[14]

Regarding evaluation, there are no specific tests to document nitrous oxide exposure. A detailed history is the basis of diagnosis of NO myelopathy.[10] Vitamin B12 deficiency has been shown to affect vision, hearing, sensory, and motor pathways on evoked studies.[10] Excluding Vitamin B12 and folate deficiency is important as potential causes of this dorsal column pathology, but do not exclude NO abuse.

The resolution of clinical symptoms in this patient was dramatic and thus atypical for SCI patients. Likewise, a central cord SCI pattern of injury is atypical in NO myelopathy. One explanation is that a central cord injury did occur in the setting of nitrous oxide intoxication, resulting in a superimposed posterior column myelopathy. Regarding the imaging, the strong hyperintensity of the posterior columns on T2-weighted MR could have obscured the otherwise faint central cord signal that might have been noticed in the absence of posterior column signal change. NO may also manifest as weakness

due to deficits in proprioception and sensation. This finding in previous literature could explain the rapid recovery that would not typically be observed in central cord myelopathy.[6,8,15,16,18]

Nitric oxide, a rapid and interchangeable form of nitrous oxide in the body, is implicated in the pathophysiology of secondary injury in spinal cord trauma. Liu and associates demonstrated a rapid increase of nitric oxide within 60 minutes of injury[17]. Further increases in nitric oxide were seen in the first 72 hours. In central nervous system inflammation, monocytes and macrophages are the chief supply of cells releasing nitric oxide. In spinal cord injury, nitric oxide synthase, a required enzyme in nitric oxide production, is seen in macrophages, neurons, astrocytes, and oligodendrocytes.[22] The oxidative properties of peroxynitrite (ONOO-), a nitric oxide-like compound formed readily in vitro from NO may accelerate local cell destruction.[1-3]Peroxynitrite can damage tissue in various ways, all from radical formation and tissue destruction. Studies of pretreatment of nitric oxide synthase inhibitors, such as nitro-L-arginine (L-NNA) in the rat have been shown to limit the effects of spinal cord injury through comparisons of histological cord sections when compared to controls.[27]

The duration of effect of nitrous oxide is another consideration. An inhalational anesthetic, nitrous oxide is highly lipid soluble, traversing the blood brain barrier easily with a brief duration of onset and is cleared from the body by still unknown mechanisms in a matter of hours. Diffusion into lipid soluble tissues and then further breakdown into oxidative byproducts is the most likely candidate for clearance.

The use of steroids in spinal cord injury is highly controversial.[23,32] Given the concern for an additional pathology contributing to the dramatic motor and sensory deficits, solumedrol was given in hope of reducing neurologic injury without a clear etiology. Steroids were not given for concerns of injury secondary to trauma, and the administration was not in accordance with the NASCIS guidelines. The mechanism for the mitigation of symptoms in spinal cord injury by steroids is still not completely understood. One credible theory is that steroids limit lipid peroxidation in secondary injury. Also, steroids are thought to have a decreased migration of leukocytes to damaged tissue, leading to a lower population of cells containing nitric oxide synthase, further limiting cell destruction.[5] Ultimately, given the short half-life of nitrous oxide, one would expect the symptoms to have a short half-life.

Conclusions

We present a case of nitrous oxide myelopathy referred to our spinal cord injury center. History and radiographic presentation are key to establishing a diagnosis.

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Figures

Figure 1

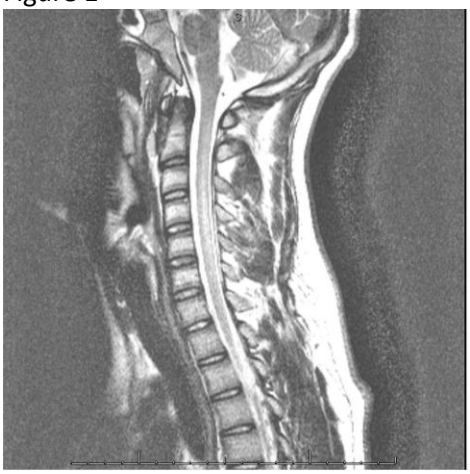

Figure 2