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Polyneuropathy with subacute combined degeneration of the spinal cord following nitrous oxide abuse

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Introduction.
Nitrous oxide (N2O) is known to induce vitamin B12 deficiency, leading to myeloneuropathies. This report describes two cases of subacute combined degenerations (SCD) of the spinal cord following N2O abuse, concomitant with motor dominant polyneuropathies.

Case 1.
A 22-year old female with a history of N2O abuse for nearly 6 months, complaining weakness of both lower limbs and gait disturbance for 2 weeks, was admitted. Primary physical examinations showed motor grades of 3 (Fair), bilateral hypoesthesia of T4 dermatomes and below, decreased proprioception, and hyperactive knee jerks. Spine magnetic resonance imaging demonstrated high signal intensities on the dorsal columns of the spinal cord at C2-C5 on T2 weighted images (Figure 1. A, B), suggestive of SCD. Initial electrodiagnostic studies (EDX) Resulted in demyelinating and axonal polyneuropathies, mainly involving the motor nerves (Table 1). Chart reviews revealed a history of vitamin B12 deficiency, which was normalized after treatment. Additional vitamin B12 supplements were administered and comprehensive rehabilitation programs were initiated. After 3 weeks, motor grades of the lower extremities and gait performance showed considerable ameliorations. Follow up EDX also exhibited some improvements: normalization of the H-reflex latencies, amplitude increments of compound motor action potentials (CMAPs) of the median nerve (Table 1).

Case 2.
A 33-year old male with a history of N2O abuse for 7 months complained gait difficulties, numbness of the trunk and below, and weakness of the lower limbs for 3 weeks. He was diagnosed with SCD of C2-C6 levels of the spinal cord (Figure 1. C, D) in a local hospital. The patient was transferred to our hospital for further evaluation since symptoms continued in spite of vitamin B12 supplements. Initial physical examinations showed motor grades of 2 (Poor) for both ankle dorsiflexors and toe extensors, bilateral hypoesthesia of the T3 dermatomes and below, hyperactive knee jerks, and a positive Romberg's sign. The patient was able to walk independently, but the dynamic standing balance was impaired. EDX suggested motor dominant polyneuropathies (Table 2). After rehabilitation, dynamic standing balance and ankle strengths were improved. Parallel to clinical improvements, follow up EDX also displayed some improvements in CMAPs (Table 2).

Conclusion.

In these cases, vitamin B12 deficiency due to N2O inhalation was suspected as the primary cause for SCD. Vitamin B12 deficiency, however, is mainly known to affect sensory nerves, and therefore difficult to account for the motor dominant polyneuropathies in our cases. Based on such fact, it may be suggested that N2O induced polyneuropathy can occur independently from vitamin B12 deficiency. We, therefore, suggest for possible polyneuropathies in SCD patients with a history of N2O abuse, even with normal vitamin B12 levels.

Table 1. Motor and Sensory Nerve Conduction Study Results of Case 1.

				Stimulation site	Recording site	Latency (msec)	Amplitude (mV)	NCV (m/sec)	F wave (msec)
Initial (after symptom onset of 2 weeks)	**Motor NCS**	Right	M	APB	Wrist	2.7	8.2	52	24.8
			U	ADM	Wrist	1.9	15.5	56	25.8
			T	AH	Ankle	**5.8***	**2.3***	44	**55.8***
			P	EDB	Ankle	**6.4***	**1.0***	42	**54.6**
		H-reflex			Right	**36.09***			
					Left	**36.15***			
				Stimulation site	Recording site	Peak latency (msec)	Amplitude (uV)		
	Sensory NCS	Right	M	Digit II	Finger	2.7	28.0		
			U	Digit V	Finger	2.4	36.5		
			S	Leg	Ankle	2.6	9.9		
			S.P	Leg	ankle	2.3	7.4		
				Stimulation site	Recording site	Latency (msec)	Amplitude (mV)	NCV (m/sec)	F wave (msec)
Follow up (after symptom onset of 5 weeks)	**Motor NCS**	Right	M	Wrist	APB	3.5	13.2	52	28.0
			U	Wrist	ADM	2.9	12.4	50	27.0
			T	Ankle	AH	**5.5***	**2.6***	47	**54.0***
		H-reflex			Right	29.3			
					Left	30.4			
				Stimulation site	Recording site	Peak latency (msec)	Amplitude (uV)		
	Sensory NCS	Right	M	Wrist	Digit III	3.6	49.0		
			U	Wrist	Digit V	**3.9***	44.0		
			S	Leg	Ankle	4.0	14.0		
			S.P	Leg	ankle	4.0	6.0		

NCS: nerve conduction study, M: median nerve, U: ulnar nerve, T: tibial nerve, P: peroneal nerve, S: sural nerve, S.P: superficial peroneal nerve, APB: Abductor pollicis brevis, ADM: abductor digiti minimi, AH: abductor hallucis, EDB: Extensor digitorum brevis, NR: no response

Abnormal values are presented with an asterisk (*).

Table 2. Motor and Sensory Nerve Conduction Study Results of Case 2

				Stimulation site	Recording site	Latency (msec)	Amplitude (mV)	NCV (m/sec)	F wave (msec)
Initial (after symptom onset of 4 weeks)	**Motor NCS**	Right	M	APB	Wrist	**4.5***	**1.9***	52	29.5
			U	ADM	Wrist	2.5	5.5	56	28.3
			T	AH	Ankle		**NR***		**NR***
			P	EDB	Ankle		**NR***		**NR***
		H-reflex			Right		**32.34***		
					Left		**32.92***		
				Stimulation site	Recording site	Peak latency (msec)	Amplitude (uV)		
	Sensory NCS	Right	M	Digit II	Finger	2.9	48.3		
			U	Digit V	Finger	2.7	20.3		
			S	Leg	Ankle	2.7	8.5		
			S.P	Leg	ankle	2.3	5.8		
				Stimulation site	Recording site	Latency (msec)	Amplitude (mV)	NCV (m/sec)	F wave (msec)
Follow up (after symptom onset of 8 weeks)	**Motor NCS**	Right	M	Wrist	APB	3.9	**3.7***	50	28.6
			U	Wrist	ADM	3.5	6.1	55	29.0
			T	Ankle	AH	**4.8***	**0.2***	40	**NR***
			P	Ankle	EDB		**NR***		**NR***
		H-reflex			Right		**32.2***		
					Left		**32.0***		
				Stimulation site	Recording site	Peak latency (msec)	Amplitude (uV)		
	Sensory NCS	Right	M	Wrist	Digit III	3.3	26.0		
			U	Wrist	Digit V	3.7	27.0		
			S	Leg	Ankle	3.9	5.0		
			S.P	Leg	ankle	3.9	5.0		

NCS: nerve conduction study, M: median nerve, U: ulnar nerve, T: tibial nerve, P: peroneal nerve, S: sural nerve, S.P: superficial peroneal nerve, APB: Abductor pollicis brevis, ADM: abductor digiti minimi, AH: abductor hallucis, EDB: Extensor digitorum brevis, NR: no response

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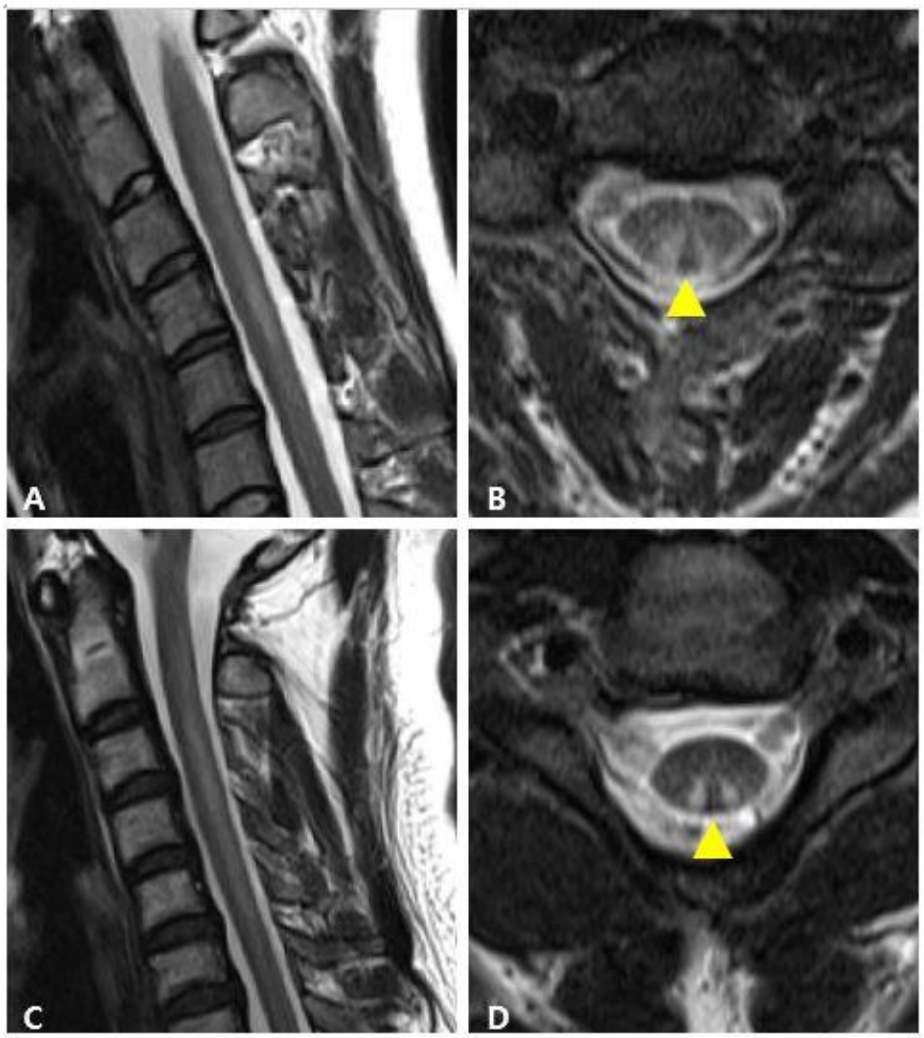

Figure 1. T2 weighted images of cervical MRI.

Sagittal (A), (C) and axial (B), (D) images of case 1 and case 2.

High signal intensity lesions are evident in the posterior column, presented as 'Inverted V sign' (arrow head).