

Severe Carbon Monoxide Poisoning with Central and Peripheral Neuromuscular System Injury : A Case report

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Background

- Carbon monoxide (CO) poisoning remains a significant cause of morbidity and mortality worldwide, commonly resulting from accidental exposure in confined environments
- Neurophysiological assessments serve as a critical tool for quantifying neuromuscular and central nervous system damage
- However, there are **limited reports that contains both peripheral and central electrodiagnostic studies**

Case presentation (50/M)

C.C	Tetraplegia		Laboratory results		
Onset	Found after estimated loss of consciousness of 2 days (2026.02.07)		CK	▲ > 22000 U/L	
Vector	After using a butane gas stove for heating in a parked car		Myoglobin (urine)	▲ > 3000 ng/mL	
Associated symptom	Pressure injuries involving right shoulder, both lower extremities, and the coccyx		Myoglobin (serum)	▲ 112.9 ng/mL	
Physical examination	Mental status : Alert MMT : Upper Extremity : T/G Lower Extremity : F/P- ROM : Passively full	Sensory : - Anesthesia (-) - Hypesthesia : Rt. Upper, both Lower extremity - Paresthesia : Both upper extremity Pathologic reflex : Hoffman -/+, Ankle clonus -/ Direct tenderness on Rt. Triceps, both lower GCM Functional ability : MBI 12, BBT 5, MMSE 21/30	eGFR	▼ 58.7 mL/min/1.7	
			LD	▲ 1159 U/L	
			CRP	▲ > 271.86 mg/L	
			AST	▲ 51 U/L	
			ALT	▲ 44 U/L	
			Past history	No chronic disease	

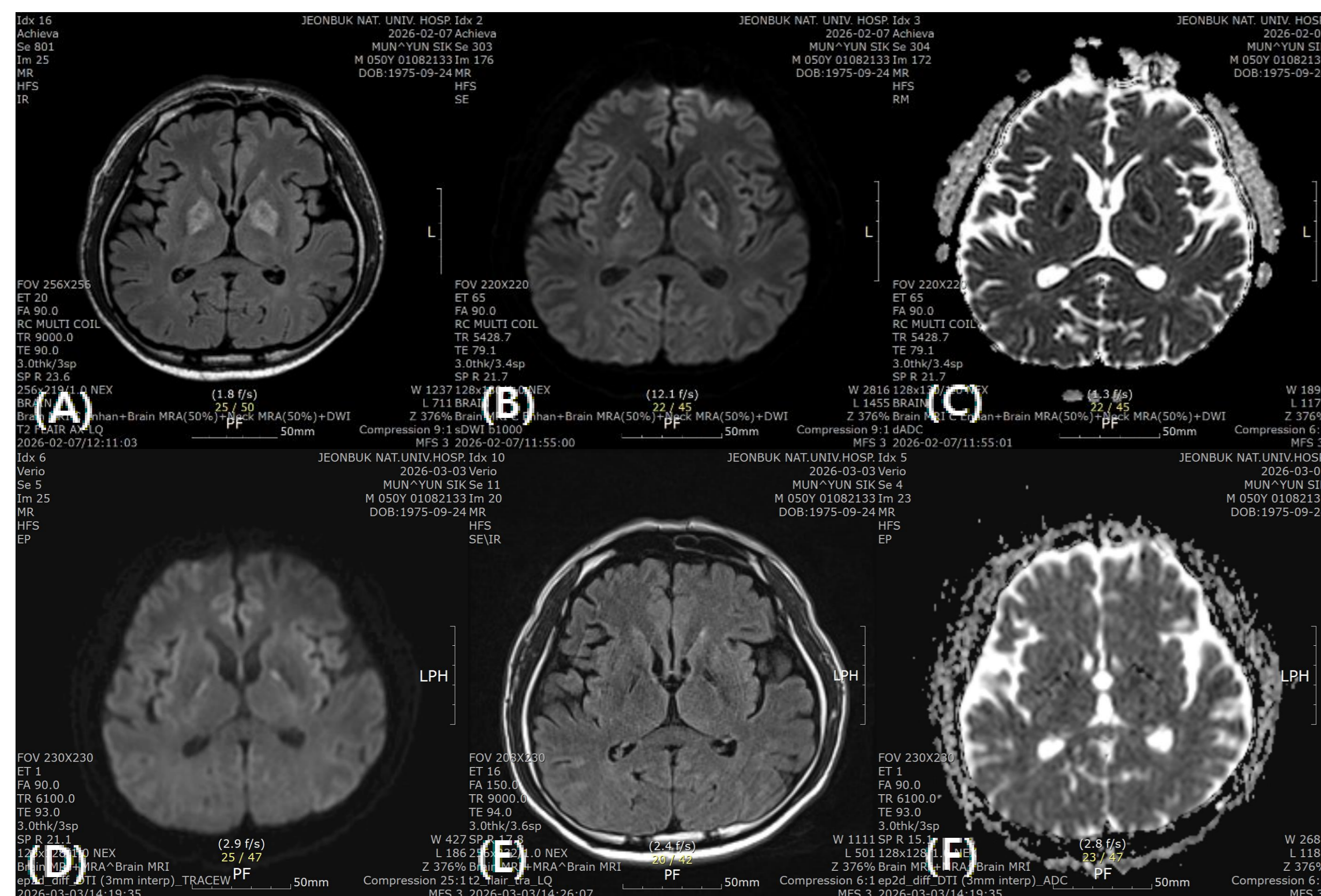


Figure 1. Figure 1. Brain MR imaging. (A) T2 FLAIR image. (B) DWI b1000 image. (C) ADC image. MR imaging showed bilateral globus pallidus lesion. Follow up study showed resolution state of bilateral globus pallidus lesion. (D) T2 FLAIR image, (E) DWI b1000 image, (F) ADC image.

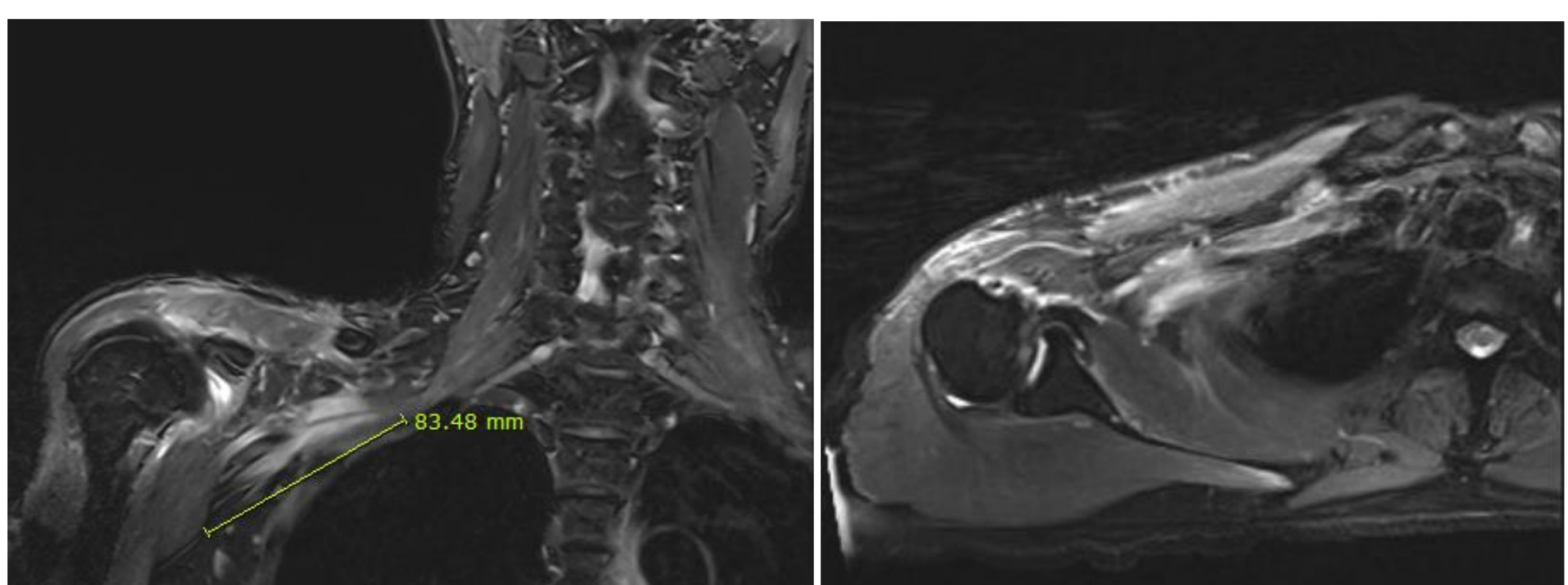


Figure 2. Highly suggestive of brachial neuroplexopathy, right in brachial plexus MR. Increased signal intensity and swelling in 8.3cm from division to cord level.

Nerve Conduction Study	Right Latency (ms)	Right Amp	Right Vel (m/s)	Left Latency (ms)	Left Amp	Left Vel (m/s)
Motor NCS (mV)						
MEDIAN - APB	NR	NR	NR	2.56	15.4	57.7
ULNAR - ADM	2.73	0.3	42.3	2.38	14.9	51.1
RADIAL - EIP	NR	NR	NR	1.88	12.1	67.0
MUSCULOCUTANEOUS - BB	NR	NR	-	4.94	20.8	-
AXILLARY - Deltoid	10.90	0.4	-	3.65	23.4	-
LONG THORACIC - SA	3.50	0.3	-	3.33	7.7	-
DORSAL SCAP - Rhomboideus	6.81	1.4	-	3.48	5.3	-
SUPRASCAP - Supraspinatus	1.60	8.5	-	2.56	17.3	-
SUPRASCAP - Infraspinatus	1.90	5.1	-	2.62	11.3	-
TIBIAL (KNEE) - AH	3.35	15.9	52.7	NR	NR	NR
DEEP PERONEAL - EDB	NR	NR	NR	NR	NR	NR
TIBIAL (KNEE) - GCM	3.98	12.9	-	6.92	1.3	-
DEEP PERONEAL - TA	4.00	1.5	56.8	NR	NR	NR
Sensory NCS (µV)						
MEDIAN - 3rd finger	NR	NR	NR	2.56	59.2	56.9
ULNAR - Digit V	NR	NR	NR	2.60	27.8	53.8
SUPERFICIAL RADIAL - Thumb	2.12	7.3	47.1	1.77	41.1	56.5
MEDIAL ANTE BRA CUT - Forearm	NR	NR	-	1.98	14.7	60.6
LATERAL ANTE BRA CUT - Forearm	2.44	6.5	49.2	1.85	63.1	64.7
SUP PERONEAL - lower leg	NR	NR	-	NR	NR	-
SURAL - Lat Malleolus	2.48	13.3	48.4	NR	NR	-

MEP Modality / Site	Latency (ms)	Amplitude (mV)	CMCT (ms)
Right Cortex - Lt 1st DI	21.5	1.63	8.2
Left Cortex - Rt 1st DI	NR	NR	-
Right Cortex - Lt BB	14.6	0.22	-
Left Cortex - Rt BB	NR	NR	-
Right Cortex - Lt TA	33.2	0.07	19.7
Left Cortex - Rt TA	33.5	0.11	19.9
Right Cortex - Lt VM	25.0	0.33	-
Left Cortex - Rt VM	25.5	0.24	-

SEP Nerve / Modality	Latency (ms) - Rt	Latency (ms) - Lt
Median Nerve SEP (N19)	NR	18.96
Posterior Tibial SEP (P1)	39.69	NR
Pudendal Nerve SEP (P1)	42.08	

Electrodiagnostic studies (Post-event 1 month)

- Needle EMG : abnormal spontaneous activity in bilateral FDI, APB, BF short head, TA, TP, EDB, right SS, serratus anterior, rhomboideus major, deltoid, PT, EIP muscles

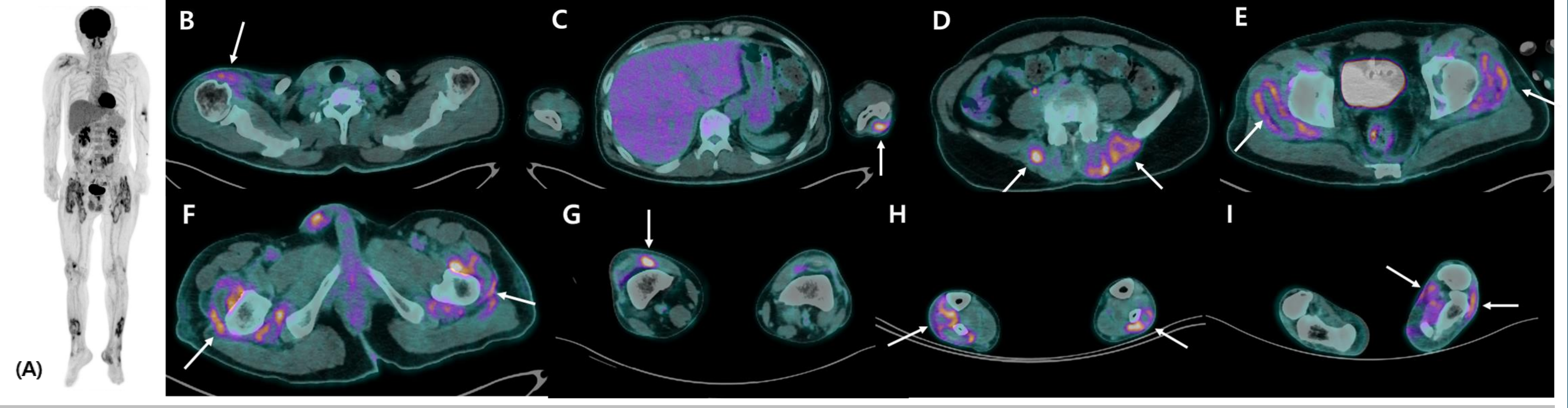


Figure 3. (A) FDG PET/CT Total body view (A). Abnormal hypermetabolism was observed in right shoulder (B), left elbow (C), erector spinae muscles at the lower lumbar level (D), both hip joints (E and F), right knee (G), left hind foot, pressure injuries (H and I), suggesting hypoxic myopathy.

Conclusion

- This case highlights that severe CO poisoning can induce toxic encephalopathy, necrotizing myopathy, rhabdomyolysis and peripheral nerve injury through hypoxia and direct cellular toxicity
- These findings suggest that corticospinal tract involvement can be localized to lower extremities, possibly due to the vulnerability of longer fibers
- Clinicians should perform multimodal neurophysiological assessments to identify the central and peripheral neuromuscular system injury in patients with CO poisoning.
- The patient showed impaired sitting balance at the time of admission, but after intense individualized rehabilitation based on electrodiagnostic findings, he was able to walk 50m using mono cane at discharge. FDG PET/CT and follow up NCS, EMG will take place in near future