Severe Neurological Manifestation of Type II Decompression Sickness in a Traditional Diver: A Case Report

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BACKGROUND

Type II Decompression Sickness (DCS) is a serious condition resulting from nitrogen bubble formation due to rapid ascent after deep or prolonged dives. Traditional divers using compressors are at high risk because of inadequate decompression procedures

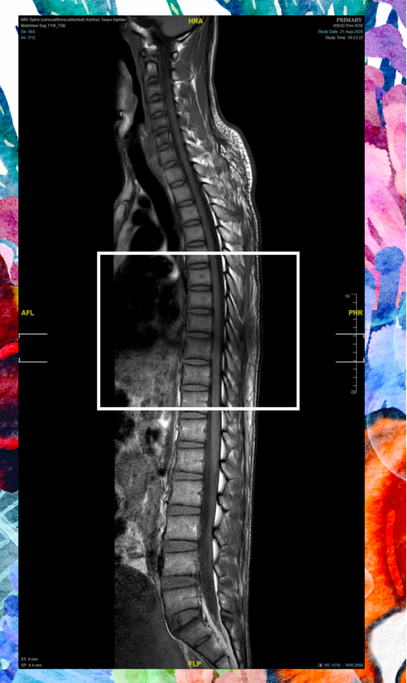
CASE PRESENTATION

A 32-year-old male traditional diver developed bilateral lower limb weakness, numbness below the umbilicus, and urinary retention approximately 20 minutes after surfacing rapidly from a 30-meter dive lasting one hour using a compressor

Neurological examination

- Paraplegia
- Hypoesthesia at the level of the T10 myelum segment

- Intramedullary hyperintense lesion at T8–T11 consistent with spinal cord edema



Therapy

- Recompression delayed >24 hours after symptom onset
- Hyperbaric Oxygen Therapy (HBOT): 3 sessions
- Supportive care and physiotherapy

Outcomes

- Persistent paraplegia and sensory deficit below T10

DISCUSSION

- Type II DCS occurs when nitrogen bubbles obstruct spinal cord microcirculation, leading to ischemia, inflammation, and edema.
- Rapid ascent and absence of safety stops increase bubble formation.
- Delayed recompression (>24h) allows irreversible neuronal injury.
- MRI findings (spinal cord edema) confirm central nervous system involvement.
- Traditional divers face high morbidity due to limited awareness and lack of hyperbaric facilities in rural areas.
- Early recognition and HBOT within 6 hours improve outcomes significantly

CONCLUSION

Type II DCS can cause severe neurological deficits if not promptly treated. Immediate HBOT is the definitive therapy to prevent permanent spinal cord injury. Education and preventive training for traditional divers are essential to reduce morbidity and mortality

REFERENCES