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TOXIC POLYRADICULONEURITIS FOLLOWING N-HEXANE INTOXICATION: A CASE REPORT AND LITERATURE REVIEW

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ABSTRACT

Introduction: Hexane is a highly neurotoxic solvent capable of triggering peripheral neuropathies upon inhalation, either due to dependency or industrial exposure. The first case was reported among industrial workers.
We present a case of acute polyradiculoneuritis associated with N-Hexane.
Case Report: A 28-year-old patient, addicted to N-Hexane (solvent), presented with ascending tetraparesis initially involving the lower limbs. Cerebrospinal fluid (CSF) analysis was normal. Electroneuromyography (ENMG) indicated severe sensorimotor demyelinating polyneuropathy predominantly affecting the lower limbs, consistent with toxic neuropathy. MRI findings supported diffuse toxic leukodystrophy. The condition progressed to respiratory impairment, requiring intubation and mechanical ventilation. Despite immunoglobulin therapy, the patient's condition deteriorated.
Discussion: Diagnosing polyradiculoneuritis induced by N-Hexane remains challenging. Imaging and ENMG are crucial for diagnosis. Management primarily involves cessation of exposure. Limited evidence exists regarding the efficacy of immunoglobulins or plasmapheresis.
Conclusion: This report highlights a rare and severe case of polyradiculoneuritis associated with N-Hexane, emphasizing awareness of its neurotoxic potential.

KEYWORDS

Toxic Polyradiculoneuritis, N-Hexane, case report

MAIN ARTICLE

Introduction

Polyradiculoneuritis is a rare condition characterized by segmental, multifocal, sensorimotor demyelinating neuropathy. This condition is generally immunological in origin (40%) but can also result from infectious, autoimmune, neoplastic, or toxic causes (such as solvents). Neurotoxic syndromes due to solvent exposure represent a significant but little-known health issue with potentially severe consequences for human health. These chemicals, some widely used industrially, pose major and serious intoxication risks to exposed individuals. Certain solvents, notably N-Hexane (C6H14), have been studied for their peripheral nerve toxicity. The development of hexacarbon neuropathy resulting from exposure to N-Hexane was first recognized and reported in the 1960s. N-Hexane is a petroleum product found in solvents and adhesives and has been noted for neurological symptoms in automotive mechanics, shoemakers, handbag manufacturers, and furniture finishers. Toxicological studies have shown that N-Hexane causes peripheral neuropathy in laboratory animals and humans. Electrophysiological findings in patients with polyneuropathy due to N-Hexane exposure correlate with clinical severity and may be related to the duration and intensity of exposure. Through the following case, we aim to highlight the impact of N-Hexane on peripheral nerves and the severity of this condition.

Patient and observation :

Observation:

Mr. K.E., a 28-year-old patient.

Known history of substance abuse (N-Hexane, alcoholism, smoking, and cannabis), experienced acute polyradiculoneuritis linked to N-Hexane consumption in 2010, treated successfully with immunoglobulins over two months. He experienced two relapses (2015-2016) after resuming N-Hexane consumption; hospitalization without immunoglobulin treatment led to improvement after one month post cessation. He resumed N-Hexane use in January 2023.

He was admitted to a neurology consultation on March 9, 2023, with weakness in both lower limbs, progressing to the upper limbs without genito-sphincter or visual disturbances and without respiratory distress. On March 16, 2023, he was admitted to Neurology Department A at Rabat Specialty Hospital for electroneuromyography (ENMG), which confirmed severe sensorimotor demyelinating polyneuropathy predominantly in the lower limbs, consistent with toxic neuropathy. His condition deteriorated, developing genito-sphincter disturbances and

decreased visual acuity on April 2, 2023, leading to readmission to Neurology Department A - HSR on April 17, 2023.

Initial evaluation found a stable patient overall. Neurological examination showed flaccid tetraplegia (1/5 proximal-distal strength), abolished deep tendon reflexes in all four limbs, vibratory hypoesthesia in all limbs, facial diplegia (bilateral Souques' sign), blurred vision, and sphincter disturbances characterized by anal incontinence and urinary leakage.

ENMG indicated severe sensorimotor demyelinating polyneuropathy, predominantly in the lower limbs, consistent with toxic neuropathy (in a worsening phase). Associated spinal involvement was possible due to sphincter disturbances (Figure 2).

MRI revealed diffuse toxic leukodystrophy (Figure 1).

On April 25, 2023, the patient developed respiratory distress with oxygen saturation dropping to 80% under 6L of O2, necessitating transfer to the intensive care unit for further management.

Examination :

- Neurological: GCS 7 (Eyes 4, Verbal 2, Motor 1), pupils equal and reactive, tetraplegia, no convulsions.
- Respiratory: SpO2 at 80% under 15L, respiratory rate: 36/min, bilateral rhonchi, superficial breathing, signs of respiratory distress (suprasternal, intercostal, and subcostal retractions).
- Hemodynamic: Blood pressure at 170/110 mmHg, heart rate 120 bpm, no peripheral hypoperfusion, capillary refill time < 3 seconds.
- General: Blood sugar 1.86 g/L, temperature 37.1°C, sweating.

Decision: Transfer to ICU for further management.

Management plan :

- Establish two 18-gauge peripheral venous lines.
- Fluid resuscitation with 1 L normal saline 0.9%.
- Intubation, controlled ventilation, sedation.
- Post-intubation blood gas analysis showed: pH 7.31, PO2 86.7 mmHg, PCO2 47.6 mmHg, HCO3- 23.8 mmol/L, BE -2.8, SO2 96.3%, hematocrit 52.4%, hemoglobin 17.4 g/dL, Na 139.1 mmol/L, K+ 4.13 mmol/L.
- Immunoglobulin therapy at 0.4g/day for 5 days.

The patient's condition worsened with the onset of a pulmonary infection due to aspiration caused by swallowing dysfunction, without any motor improvement. Further respiratory deterioration led to septic shock requiring vasopressors (Noradrenaline) and targeted antibiotic therapy (Tienam + Amikacin) after bacteriological sampling. The patient did not respond to treatment and subsequently passed away.

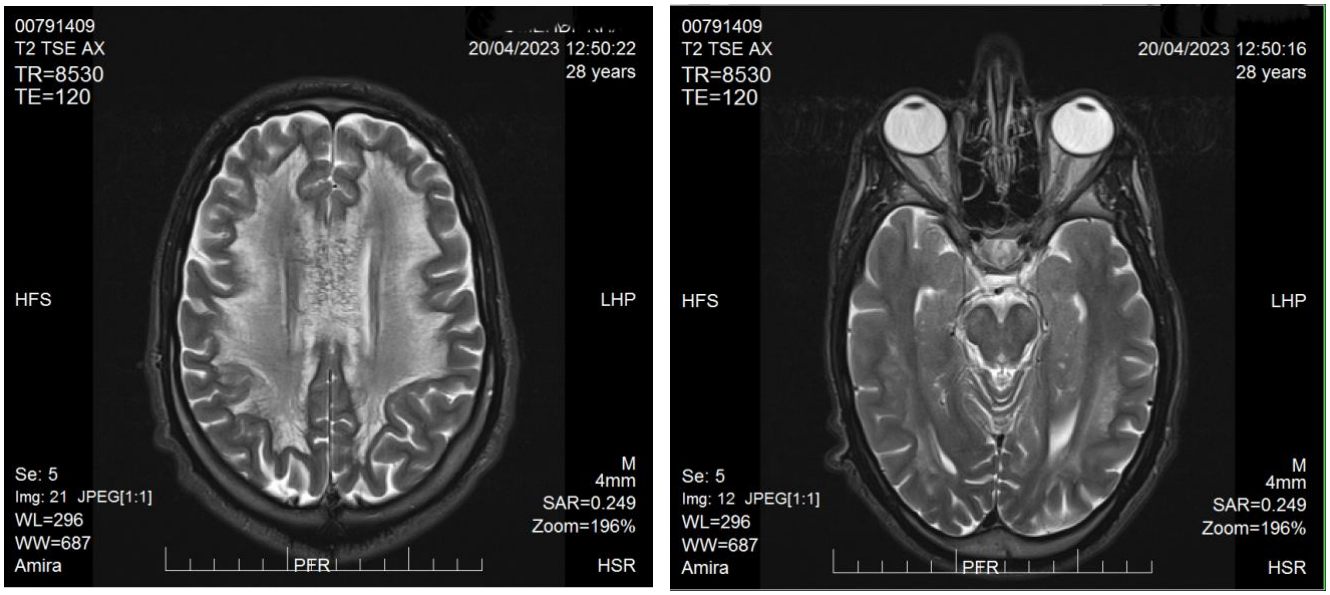
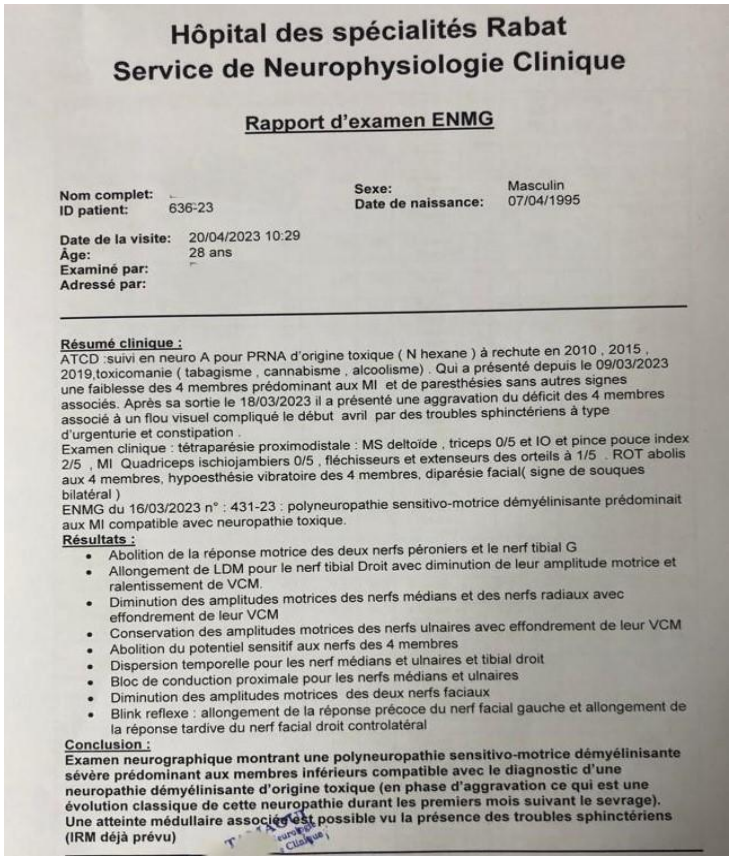


Figure 1 : Marked, diffuse, and symmetrical signal abnormalities, with T2 hyperintensity involving the deep supratentorial and periventricular white matter, extending to the corpus callosum, posterior limbs of the internal capsule, midbrain, and corticospinal tract of the pons.



Hôpital des spécialités Rabat
Service de Neurophysiologie Clinique

Rapport d'examen ENMG

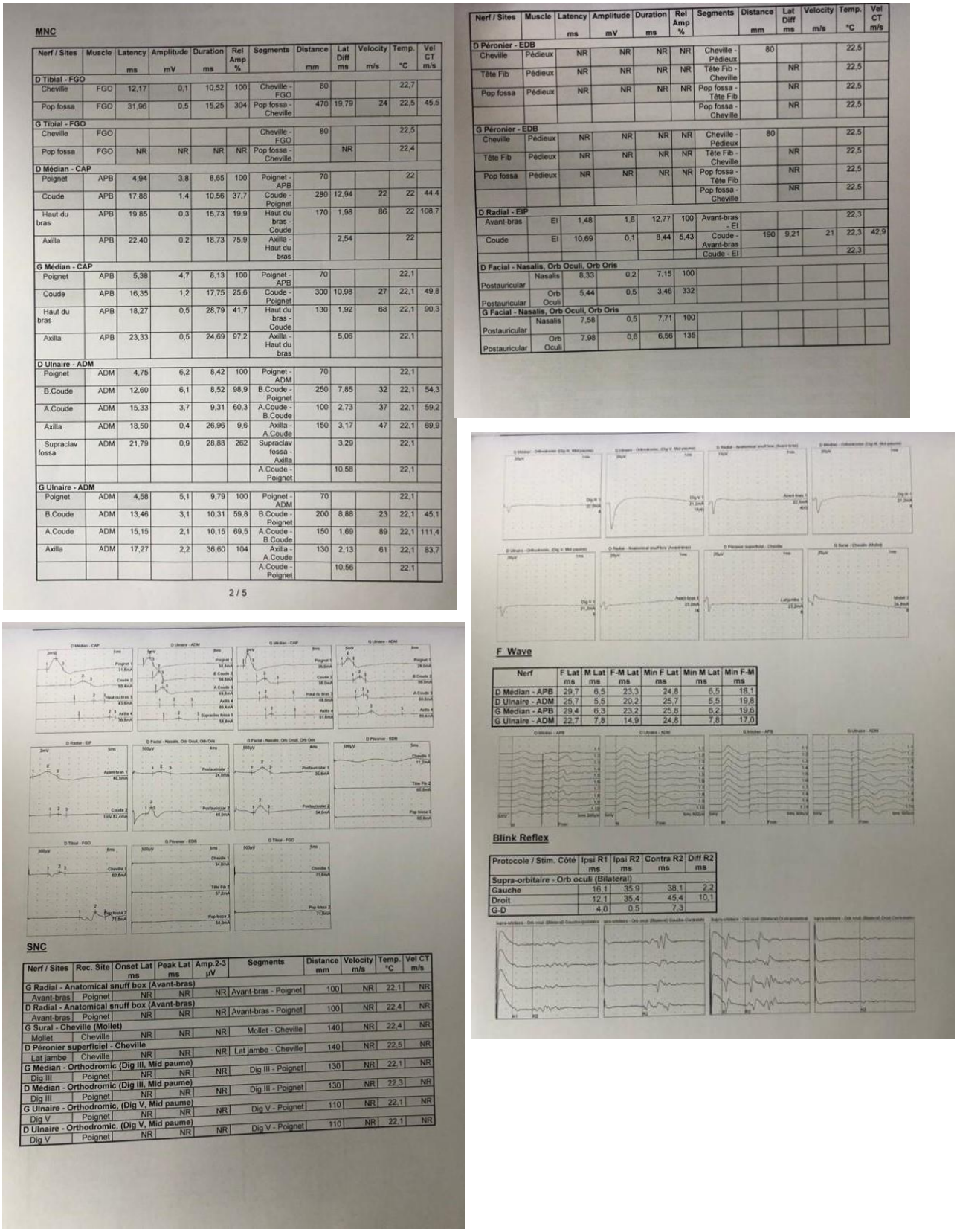
Nom complet:
ID patient: 636-23
Sexe: Masculin
Date de naissance: 07/04/1995

Date de la visite: 20/04/2023 10:29
Âge: 28 ans
Examiné par:
Adressé par:

Résumé clinique :
ATCD :suivi en neuro A pour PRNA d'origine toxique (N hexane) à rechute en 2010 , 2015 , 2019,toxicomanie (tabagisme , cannabisme , alcoolisme) . Qui a présenté depuis le 09/03/2023 une faiblesse des 4 membres prédominant aux MI et de paresthésies sans autres signes associés. Après sa sortie le 18/03/2023 il a présenté une aggravation du déficit des 4 membres associé à un flou visuel compliqué le début avril par des troubles sphinctériens à type d'urgenturie et constipation .
Examen clinique : tétraparésie proximodistale : MS deltoïde , triceps 0/5 et IO et pince pouce index 2/5 , MI Quadriceps ischiojambiers 0/5 , fléchisseurs et extenseurs des orteils à 1/5 . ROT abolis aux 4 membres, hypoesthésie vibratoire des 4 membres, diparésie facial(signe de souques bilatéral)
ENMG du 16/03/2023 n° : 431-23 : polyneuropathie sensitivo-motrice démyélinisante prédominait aux MI compatible avec neuropathie toxique.

Résultats :
- Abolition de la réponse motrice des deux nerfs péroniers et le nerf tibial G
- Allongement de LDM pour le nerf tibial Droit avec diminution de leur amplitude motrice et ralentissement de VCM.
- Diminution des amplitudes motrices des nerfs médians et des nerfs radiaux avec effondrement de leur VCM
- Conservation des amplitudes motrices des nerfs ulnaires avec effondrement de leur VCM
- Abolition du potentiel sensitif aux nerfs des 4 membres
- Dispersion temporelle pour les nerf médians et ulnaires et tibial droit
- Bloc de conduction proximale pour les nerfs médians et ulnaires
- Diminution des amplitudes motrices des deux nerfs faciaux
- Blink reflexe : allongement de la réponse précoce du nerf facial gauche et allongement de la réponse tardive du nerf facial droit controlatéral

Conclusion :
Examen neurographique montrant une polyneuropathie sensitivo-motrice démyélinisante sévère prédominant aux membres inférieurs compatible avec le diagnostic d'une neuropathie démyélinisante d'origine toxique (en phase d'aggravation ce qui est une évolution classique de cette neuropathie durant les premiers mois suivant le sevrage).
Une atteinte médullaire associée est possible vu la présence des troubles sphinctériens (IRM déjà prévu)



MNC

Nerf / Sites	Muscle	Latency ms	Amplitude mV	Duration ms	Rel Amp %	Segments	Distance mm	Lat Diff ms	Velocity m/s	Temp. °C	Vel CT m/s
D Tibial - FGO											
Cheville	FGO	12,17	0,1	10,52	100	Cheville - FGO	80			22,7	
Pop fossa	FGO	31,96	0,5	15,25	304	Pop fossa - Cheville	470	19,79	24	22,5	45,5
G Tibial - FGO											
Cheville	FGO					Cheville - FGO	80			22,5	
Pop fossa	FGO	NR	NR	NR	NR	Pop fossa - Cheville		NR		22,4	
D Médian - CAP											
Poignet	APB	4,94	3,8	8,65	100	Poignet - APB	70			22	
Coude	APB	17,88	1,4	10,56	37,7	Coude - Poignet	280	12,94	22	22	44,4
Haut du bras	APB	19,85	0,3	15,73	19,9	Haut du bras - Coude	170	1,98	86	22	108,7
Axilla	APB	22,40	0,2	18,73	75,9	Axilla - Haut du bras		2,54		22	
G Médian - CAP											
Poignet	APB	5,38	4,7	8,13	100	Poignet - APB	70			22,1	
Coude	APB	16,35	1,2	17,75	25,6	Coude - Poignet	300	10,98	27	22,1	49,8
Haut du bras	APB	18,27	0,5	28,79	41,7	Haut du bras - Coude	130	1,92	68	22,1	90,3
Axilla	APB	23,33	0,5	24,69	97,2	Axilla - Haut du bras		5,06		22,1	
D Ulnaire - ADM											
Poignet	ADM	4,75	6,2	8,42	100	Poignet - ADM	70			22,1	
B.Coude	ADM	12,60	6,1	8,52	98,9	B.Coude - Poignet	250	7,85	32	22,1	54,3
A.Coude	ADM	15,33	3,7	9,31	60,3	A.Coude - B.Coude	100	2,73	37	22,1	59,2
Axilla	ADM	18,50	0,4	26,96	9,6	Axilla - A.Coude	150	3,17	47	22,1	69,9
Supraclav fossa	ADM	21,79	0,9	28,88	262	Supraclav fossa - Axilla		3,29		22,1	
						A.Coude - Poignet		10,58		22,1	
G Ulnaire - ADM											
Poignet	ADM	4,58	5,1	9,79	100	Poignet - ADM	70			22,1	
B.Coude	ADM	13,46	3,1	10,31	59,8	B.Coude - Poignet	200	8,88	23	22,1	45,1
A.Coude	ADM	15,15	2,1	10,15	69,5	A.Coude - B.Coude	150	1,69	89	22,1	111,4
Axilla	ADM	17,27	2,2	36,60	104	Axilla - A.Coude	130	2,13	61	22,1	83,7
						A.Coude - Poignet		10,56		22,1	

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Nerf / Sites	Muscle	Latency ms	Amplitude mV	Duration ms	Rel Amp %	Segments	Distance mm	Lat Diff ms	Velocity m/s	Temp. °C	Vel CT m/s
D Péronier - EDB											
Cheville	Pédieux	NR	NR	NR	NR	Cheville - Pédieux	80			22,5	
Tête Fib	Pédieux	NR	NR	NR	NR	Tête Fib - Cheville		NR		22,5	
Pop fossa	Pédieux	NR	NR	NR	NR	Pop fossa - Tête Fib		NR		22,5	
						Pop fossa - Cheville		NR		22,5	
G Péronier - EDB											
Cheville	Pédieux	NR	NR	NR	NR	Cheville - Pédieux	80			22,5	
Tête Fib	Pédieux	NR	NR	NR	NR	Tête Fib - Cheville		NR		22,5	
Pop fossa	Pédieux	NR	NR	NR	NR	Pop fossa - Tête Fib		NR		22,5	
						Pop fossa - Cheville		NR		22,5	
D Radial - EIP											
Avant-bras	EI	1,48	1,8	12,77	100	Avant-bras - EI				22,3	
Coude	EI	10,69	0,1	8,44	5,43	Coude - Avant-bras	190	9,21	21	22,3	42,9
						Coude - EI				22,3	
D Facial - Nasalis, Orb Oculi, Orb Oris											
Postauricular	Nasalis	8,33	0,2	7,15	100						
Postauricular	Orb Oculi	5,44	0,5	3,46	332						
G Facial - Nasalis, Orb Oculi, Orb Oris											
Postauricular	Nasalis	7,58	0,5	7,71	100						
Postauricular	Orb Oculi	7,98	0,6	6,56	135						

F Wave

Nerf	F Lat ms	M Lat ms	F-M Lat ms	Min F Lat ms	Min M Lat ms	Min F-M ms
D Médian - APB	29,7	6,5	23,3	24,8	6,5	18,1
D Ulnaire - ADM	25,7	5,5	20,2	25,7	5,5	19,8
G Médian - APB	29,4	6,3	23,2	25,8	6,2	19,6
G Ulnaire - ADM	22,7	7,8	14,9	24,8	7,8	17,0

Blink Reflex

Protocole / Stim. Côté	Ipsi R1 ms	Ipsi R2 ms	Contra R2 ms	Diff R2 ms
Supra-orbitaire - Orb oculi (Bilateral)				
Gauche	16,1	35,9	38,1	2,2
Droit	12,1	35,4	45,4	10,1
G-D	4,0	0,5	7,3	

SNC

Nerf / Sites	Rec. Site	Onset Lat ms	Peak Lat ms	Amp.2-3 µV	Segments	Distance mm	Velocity m/s	Temp. °C	Vel CT m/s
G Radial - Anatomical snuff box (Avant-bras)									
Avant-bras	Poignet	NR	NR	NR	Avant-bras - Poignet	100	NR	22,1	NR
D Radial - Anatomical snuff box (Avant-bras)									
Avant-bras	Poignet	NR	NR	NR	Avant-bras - Poignet	100	NR	22,4	NR
G Sural - Cheville (Mollet)									
Mollet	Cheville	NR	NR	NR	Mollet - Cheville	140	NR	22,4	NR
D Péronier superficiel - Cheville									
Lat jambe	Cheville	NR	NR	NR	Lat jambe - Cheville	140	NR	22,5	NR
G Médian - Orthodromic (Dig III, Mid paume)									
Dig III	Poignet	NR	NR	NR	Dig III - Poignet	130	NR	22,1	NR
D Médian - Orthodromic (Dig III, Mid paume)									
Dig III	Poignet	NR	NR	NR	Dig III - Poignet	130	NR	22,3	NR
G Ulnaire - Orthodromic, (Dig V, Mid paume)									
Dig V	Poignet	NR	NR	NR	Dig V - Poignet	110	NR	22,1	NR
D Ulnaire - Orthodromic, (Dig V, Mid paume)									
Dig V	Poignet	NR	NR	NR	Dig V - Poignet	110	NR	22,1	NR

Figure 2 : Electroneuromyography results

Discussion :

Epidemiology:

Peripheral neuropathy is a significant clinical condition with a prevalence of approximately 15% in the U.S. population and 1.9% in the African population.Etiologies include metabolic, infectious, hereditary conditions, and nutritional deficiencies.

Toxic neuropathies resulting from xenobiotics—foreign substances capable of interacting with living cells—represent another significant cause of acquired polyneuropathy.

These neuropathies can be environmental, occupational, recreational, or iatrogenic, with prevalence influenced by geographical and economic factors. In developed countries, the most common cause is drug toxicity, particularly from chemotherapy treatments, affecting up to 68% within the first month post-therapy.

Environmental and occupational exposures to agents such as arsenic, lead, mercury, and organophosphate compounds are also important peripheral neurotoxins.

N-Hexane is one of the toxic agents responsible for toxic polyneuropathy, entering the body via inhalation or skin absorption.

Diabetes mellitus and alcoholism, two common causes of polyneuropathy, may also be considered toxic neuropathies due to excessive glucose and ethanol effects.

In our case, the patient is a 28-year-old substance abuser with documented alcohol and N-Hexane consumption, having experienced three episodes of polyradiculoneuritis linked to N-Hexane.

Clinical evaluation

Neuropathy is characterized by distal central and peripheral neurofilamentous axonopathy. Aggregates of neurofilament proteins accumulate in the subterminal axon, creating axonal swellings accompanied by myelin retraction, often occurring just upstream of axonal constrictions at the nodes of Ranvier (small domains interposed between myelin segments, essential for nerve impulse propagation). Electron microscopy of these swellings is consistent with disrupted axonal transport, showing neurofilament aggregates upstream and vesicle accumulation downstream of nodal constrictions. Continuous exposure leads to axonal atrophy development, followed by axonal degeneration downstream of the swellings.

Clinical manifestations of toxic polyradiculoneuritis vary based on nerve structure damage severity but share common features:

- Sensory symptoms: paresthesia or complete loss of sensation, burning sensations, heightened touch sensitivity.

- Motor symptoms: muscle weakness, coordination difficulties, progressive muscle atrophy.
- Autonomic symptoms: digestive issues, urinary disturbances, cardiovascular dysfunction such as orthostatic hypotension.

Symptom distribution can be symmetrical, affecting both sides of the body, initially favoring distal extremities, especially lower limbs, with ascending progression.

A study by Puri et al. on screen-printing workers showed that 92% had sensorimotor deficits, and only two patients exhibited sensory disturbances exclusively.

In our case, the deficit initially manifested as muscular weakness in the lower limbs without genito-sphincter disturbances, visual impairment, or cardiovascular dysautonomia. The condition progressively ascended to involve the upper limbs, sphincter disturbances, visual blurriness, and respiratory distress.

Paraclinical evaluation :

Electrodiagnostic studies, including nerve conduction studies (NCS) and electromyography (EMG), are the gold standard for evaluating peripheral nerve function, neuromuscular junctions, and muscles, and for identifying demyelinating and axonal damage.

However, electrodiagnostic studies alone cannot conclusively diagnose these disorders; they should always be considered as an extension of the clinical history and physical examination.

Typical electrophysiological findings of sensory or motor polyneuropathy include:

- Low sural/radial amplitude ratio,
- Reduced amplitude of sural sensory nerve action potentials,
- Increased minimum latency or absence of F-waves in median or tibial nerves,
- Reduced motor and sensory conduction velocities,
- Abnormal spontaneous EMG activity and other signs of denervation.

Biological monitoring includes the measurement of urinary 2,5 hexanedione levels.

Cerebrospinal fluid (CSF) analysis typically shows no abnormalities and has limited diagnostic value.

In our case, the patient showed signs of severe sensorimotor demyelinating polyneuropathy predominantly in the lower limbs. Monitoring for 2,5 hexanedione was not performed due to the unavailability of reagents in our facility.

Therapeutic management :

No proven effective treatment exists for toxic neuropathies. Various studies have demonstrated that intravenous immunoglobulins and plasmapheresis are ineffective in treating toxic polyradiculoneuritis caused by N-Hexane. The only intervention known to

potentially improve patient outcomes is the cessation of N-Hexane exposure, though improvement occurs only in certain cases.

In our patient's previous episodes, symptom improvement was observed following the cessation of N-Hexane exposure. However, during his hospitalization, no improvement was noted even after the use of immunoglobulins, with continued deterioration of the patient's condition.

Conclusion

Toxic polyradiculoneuritis remains a severe neurological disorder with an often poor prognosis. Its variable clinical presentation poses a diagnostic challenge, highlighting the importance of detailed patient history. Eliminating exposure to N-Hexane is the only known treatment for this condition, as plasmapheresis and immunoglobulins have shown no therapeutic benefit

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REFERENCES

1. Mendell JR. Chronic inflammatory demyelinating polyradiculoneuropathy. Annu Rev Med. 1993;44: 211-219. https://doi.org/10.1146/annurev.me.44.020193.001235
2. Kanbayashi T, Sonoo M. Acute-onset chronic inflammatory demyelinating polyradiculoneuropathy. Brain Nerve. 2015;67: 1388-1396.
3. Loo LK, Salim O, Liang D, Goel A, Sumangala S, Gowda AS, et al. Acute-onset polyradiculoneuropathy after SARS-CoV2 vaccine in the West and North Midlands, United Kingdom. Muscle Nerve. 2022;65: 233-237. https://doi.org/10.1002/mus.27461
4. Miranda C, Brannagan TH. Chapter 26 - Acute/chronic inflammatory polyradiculoneuropathy. In: Younger DS, editor. Handbook of Clinical Neurology. Elsevier; 2023. pp. 619-633. https://doi.org/10.1016/B978-0-323-98818-6.00026-1
5. Zahlane S, Louhab N, Kissani N. Intoxication with n-hexane mimicking Guillain-Barré syndrome: A case report. Revue neurologique. 2021. pp. 433-434. https://doi.org/10.1016/j.neurol.2020.07.011
6. Karam C, Dyck PJB. Toxic Neuropathies. Semin Neurol. 2015;35: 448-457. https://doi.org/10.1055/s-0035-1558977
7. Centers for Disease Control and Prevention (CDC). n-Hexane-related peripheral neuropathy among automotive technicians--California, 1999-2000. MMWR Morb Mortal Wkly Rep. 2001;50: 1011-1013.
8. Huang J, Kato K, Shibata E, Asaeda N, Takeuchi Y. Nerve-specific marker proteins as indicators of organic solvent neurotoxicity. Environ Res. 1993;63: 82-87. https://doi.org/10.1006/enrs.1993.1129
9. Yamamura Y. n-Hexane polyneuropathy. Folia Psychiatr Neurol Jpn. 1969;23: 45-57. https://doi.org/10.1111/j.1440-1819.1969.tb01441.x
10. Takeuchi Y. n-hexane polyneuropathy in Japan: a review of n-hexane poisoning and its preventive measures. Environ Res. 1993;62: 76-80. https://doi.org/10.1006/enrs.1993.1091
11. Kutlu G, Gomceli YB, Sonmez T, Inan LE. Peripheral neuropathy and visual evoked potential changes in workers exposed to n-hexane. J Clin Neurosci. 2009;16: 1296-1299. https://doi.org/10.1016/j.jocn.2008.12.021
12. Valentine WM. Toxic Peripheral Neuropathies: Agents and Mechanisms. Toxicol Pathol. 2020;48: 152-173. https://doi.org/10.1177/0192623319854326
13. Rizzuto N, De Grandis D, Di Trapani G, Pasinato E. n-hexane polyneuropathy. An occupational disease of shoemakers. Eur Neurol. 1980;19: 308-315. https://doi.org/10.1159/000115166
14. Magy L. [Peripheral neuropathies: Diagnostic strategy]. Rev Med Interne. 2018;39: 27-33. https://doi.org/10.1016/j.revmed.2017.01.015
15. Clark JD. Detection and assessment of xenobiotic-induced sensory neuropathy. Curr Protoc Toxicol. 2005;Chapter 11: Unit11.13.
16. Grisold W, Carozzi VA. Toxicity in Peripheral Nerves: An Overview. Toxics. 2021;9. https://doi.org/10.3390/toxics9090218
17. N-hexane. [cited 25 Oct 2023]. Available: https://wwwn.cdc.gov/TSP/PHS/PHS.aspx?phsid=391&toxid=68
18. Alcohol (ethanol) related neuropathy. 30 Jun 2023 [cited 25 Oct 2023]. Available: https://emedicine.medscape.com/article/1174146-overview

19. Vinik AI, Strotmeyer ES. Diabetic Neuropathy. Pathy's Principles and Practice of Geriatric Medicine. Chichester, UK: John Wiley & Sons, Ltd; 2012. pp. 751-767. https://doi.org/10.1002/9781119952930.ch65
20. Wang C, Chen S, Wang Z. Electrophysiological follow-up of patients with chronic peripheral neuropathy induced by occupational intoxication with n-hexane. Cell Biochem Biophys. 2014;70: 579-585. https://doi.org/10.1007/s12013-014-9959-7
21. Watson JC, Dyck PJB. Peripheral Neuropathy: A Practical Approach to Diagnosis and Symptom Management. Mayo Clin Proc. 2015;90: 940-951. https://doi.org/10.1016/j.mayocp.2015.05.004
22. Puri V, Chaudhry N, Tatke M. N-hexane neuropathy in screen printers. Electromyogr Clin Neurophysiol. 2007;47: 145-152.
23. Koo YS, Cho CS, Kim B-J. Pitfalls in using electrophysiological studies to diagnose neuromuscular disorders. J Clin Neurol. 2012;8: 1-14. https://doi.org/10.3988/jcn.2012.8.1.1
24. Pastore C, Izura V, Geijo-Barrientos E, Dominguez JR. A comparison of electrophysiological tests for the early diagnosis of diabetic neuropathy. Muscle Nerve. 1999;22: 1667-1673. https://doi.org/10.1002/(SICI)1097-4598(199912)22:12<1667::AID-MUS8>3.0.CO;2-W
25. Perbellini L, Marhuenda Amoros DM, Cardona Llorens AC, Giuliari C, Brugnone F. An improved method of analysing 2,5-hexanedione in urine. Br J Ind Med. 1990;47: 421-424. https://doi.org/10.1136/oem.47.6.421
26. Chaqda M, El Mellakh M, Kissani N, Louhab N. Neuropathy caused by addictive inhalation of n-hexane in glue sniffers. Presse Med. 2019;48: 568-572. https://doi.org/10.1016/j.lpm.2019.04.001