Journal of Pathology and Toxicology Research
JCMR, 1(1): 5-8
www.scitcentral.com

Case Report: Open Access

Unusual Cases of Nervous System Damage: Diagnostic and Therapeutic Problems

Włodzisław Kuliński[1,2*]

[*1]*Department of Rehabilitation, Military Institute of Medicine, Warsaw, Poland*

[2]*Faculty of Medicine and Health Science, Jan Kochanowski University, Kielce, Poland.*

Received November 29, 2019; Accepted December 09, 2018; Published June 26, 2020

ABSTRACT

Introduction: Peripheral nervous system damage, or peripheral neuropathy, is a difficult clinical problem. The most common causes include traumatic injuries, inflammation and metabolic disorders. Traumatic injuries, both acute and chronic, usually affect single nerves (mononeuropathies) while inflammation and metabolic disorders damage multiple nerves (polyneuropathies). The paper presents two rare cases of severe nervous system damage.

Case 1: The first part describes the clinical presentation of a female patient with yew needle extract poisoning. The predominant neurological symptom was severe spastic tetraparesis with a bilateral positive Babinski sign and total aphasia. After physical therapy and rehabilitation, the patient's mental status and ability to maintain contact were considerably improved and further increases in muscle strength and physical function were seen, which helped the patient walk unassisted using a walking frame and perform the activities of daily living.

Case 2: A quality inspector working at a gas station presented with distal paresis of both lower limbs after chronic, long-term, 20 years' occupational exposure to gasoline fumes. He had memory gaps, psychomotor retardation, a gait with bilateral foot-dragging, diminished knee and ankle reflexes and glove- and stocking-type superficial sensory disturbances. The patient underwent physical therapy and rehabilitation at the Department of Rehabilitation.

The treatment resulted in partial improvements with respect to the weakened muscle groups. A further six months' follow-up showed increasing central and peripheral nervous system deficits.

Results and conclusion: Toxic polyneuropathy and encephalopathy are irreversible in patients with chronic hydrocarbon poisoning caused by gasoline and result in permanent nervous system damage. Physical therapy and rehabilitation must be patient-specific.

Keywords: *Taxus baccata*, Gasoline poisoning, Rehabilitation treatment

INTRODUCTION

Physical medicine is an interdisciplinary specialty. Physical therapy is used in patients with initial diagnostic problems and later therapeutic problems.

MATERIALS

Two rare cases of patients with very severe nervous system damage.

CASE 1

Yew needle extract poisoning

A 20 year old woman ingested yew needle extract. Common yew (*Taxus baccata*) is an ornamental and a long-living tree with medicinal properties. Yew needle extract used to be given to people with hypotension. The chemotherapy drug docetaxel is manufactured using substances isolated from yew trees [1-6].

Yew wood does not contain resin. All parts of the plant contain the toxic alkaloid taxine, which has mostly cardiotoxic and hepatotoxic effects. The whole plant is poisonous, but the needles are the most poisonous, followed by seeds. The main toxic substances affect the heart, the central nervous system and the respiratory center, damage

Corresponding author: Włodzisław Kuliński, Department of Rehabilitation, Military Institute of Medicine, Warsaw, Poland, E-mail: wkulinski52@hotmail.com

Citation: Kuliński W. (2020) Unusual Cases of Nervous System Damage: Diagnostic and Therapeutic Problems. J Pathol Toxicol Res, 1(1): 5-8.

the kidneys and may induce miscarriage [7-12]. Symptoms of poisoning develop 30 to 90 min after ingestion and include vomiting and severe abdominal pain. Patients may experience dizziness, giddiness, somnolence, muscle weakness, dyspnea, seizures, functional cardiac disturbances and cardiac arrest.

The patient was initially treated at an intensive care center due to severe arrhythmia in the form of tachycardia and ventricular fibrillation, which occurred a total of 11 times. As a result, the patient was defibrillated multiple times. She was then transferred to and treated at an acute poisoning center. At the time, the predominant neurological symptom was severe spastic tetraparesis with a bilateral positive Babinski sign and total aphasia. She no longer showed cardiovascular abnormalities. When the patient was transferred to the Department of Rehabilitation of the Military Institute of Medicine, intensive physical therapy and rehabilitation were initiated. The treatment program was gradually expanded as her motor and cognitive functions improved. The patient's progress during rehabilitation was assessed based on time to recovery of motor functions. At the end of her 3 month stay at the Department of Rehabilitation, she showed marked improvements in her clinical status (maintaining contact, considerable reduction in limb spasticity and contractures, good sitting tolerance).

During her next stay at the Department of Rehabilitation, the patient's management was focused on verticalization and active ambulation, especially gait education, maintaining better contact with the patient and improving her cognitive functions. The patient's mental status and ability to maintain contact were also considerably improved and increases in muscle strength and physical function were seen, which helped the patient walk unassisted using a walking frame and perform the activities of daily living.

Conclusion

Effective management after acute yew poisoning is possible only with:

- Regular exercises and a long-term rehabilitation process,
- Physical therapy procedures adjusted to the patient's condition,
- The intensity of exercise changing depending on improvements in motor functions.

CASE 2

Chronic gasoline poisoning

Cases of chronic poisoning with compounds found in gasoline are rare. Gasoline poisoning may develop due to inhalation or ingestion. Gasoline is a mixture of aliphatic hydrocarbons with various boiling temperatures, with low amounts of aromatic hydrocarbons. Gasoline contains the toxic substances benzene, methyl alcohol and tetraethyl lead. Inhaling air with 1-2% gasoline for several minutes may result in severe poisoning symptoms, including coma [13-19]. Tetraethyl lead is added to gasoline as an antiknock agent. It may be absorbed into the body by the lungs, the skin, or the gastrointestinal tract and is then converted to triethyl lead, which is responsible for the symptoms of toxicity. A lead ion is the end metabolite, which accumulates in the brain, liver, kidneys, muscles and bone. The half-life of lead is 32 years in bones and 7 years in kidneys. Lead poisoning is usually associated with hematopoietic and nervous system damage (polyneuropathy and encephalopathy). Lead is toxic, because it inhibits the activity of certain enzymes and damages multiple cellular proteins due to its ability to bind to sulfhydryl groups. In the hematopoietic system, lead inactivates δ-aminolevulinic acid dehydratase, thus inhibiting porphobilinogen synthesis, and reduces hemoglobin synthetase activity, thus inhibiting the insertion of iron into the protoporphyrin IX ring. Impaired conversion of coproporphyrinogen III into protoporphyrin IX results in higher coproporphyrin excretion in urine. Moreover, it impairs ribonucleic acid metabolism in erythrocytes; damage to erythrocyte membrane through the inhibition of membrane ATPase leads to reduced blood cell survival and hemolysis. In the gastrointestinal tract, lead may damage the autonomic system, causing peristalsis abnormalities. The neurological toxicity of lead stems from the fact that it is highly lipid-soluble; poisoning results in degenerative changes in the cerebral cortex, the cerebellum and subcortical nuclei, and the hypothalamus autonomic centers as well as segmental demyelination of peripheral nerve fibers. After the absorption of a large amount of tetraethyl lead, patients present with acute poisoning; typically, there is a latent period, which may last between several hours and a few days and is followed by headache and dizziness, loss of appetite, insomnia and considerable weakness. A physical examination reveals decreased blood pressure and heart rate values. Patients show signs and symptoms of nervous system damage, such as paresthesia in the limbs, nystagmus and euphoria. Next, they develop mental disorders such as delirium, delusions, and sometimes schizophrenic syndrome. A period of agitation is followed by obtundation and sometimes death [20-25].

Toluene is a colorless liquid and has an odor similar to benzene. It may be absorbed by the lungs and skin. Toluene is metabolized to benzoic acid by methyl group oxidation and is excreted in urine within 15 h of the end of exposure. Symptoms of acute toluene poisoning include irritated mucous membranes, headache and dizziness, somnolence, and, rarely, loss of consciousness. Chronic poisoning is associated with pseudoneurotic disorders and possibly liver and kidney damage.

Case description

A 50 year old male patient was admitted to the Department of Rehabilitation to undergo rehabilitation due to distal paresis of both lower limbs. He was previously diagnosed at the neurology department, where imaging studies were performed. An NMR of the brain conducted at the time showed no pathological changes. An EMG revealed the following: in the upper limbs, there was bilateral carpal tunnel syndrome, involving both the median and the ulnar nerves, with only some motor fiber damage, extensive nerve conduction impairment, and prolonged sensory fiber latency; in the lower limbs, the EMG showed axonal sensorimotor polyneuropathy. On admission, history-taking showed the following: complaints of headache and dizziness, memory impairment, difficulty concentrating, sleep problems, excessive somnolence during the day, emotional irritability, mood changes, and tingling in the fingers and toes. A physical examination showed depressed mood, memory gaps, psychomotor retardation, impaired two-point discrimination in the fingertips, a positive Phalen's test, a positive Tinel's sign, reduced grip strength in both hands, a gait with bilateral foot-dragging, diminished knee and ankle reflexes, glove- and stocking-type superficial sensory disturbances, reduced muscle strength in feet flexors (3) and feet extensors (3 in the Lovett scale).

Physical therapy and rehabilitation

The physical therapy and rehabilitation program during the patient's stay at the Department of Rehabilitation was adjusted to his condition and gradually modified as clinical improvements were observed [26].

The physical therapy procedures used in the patient included:

- Four-cell baths, twice daily,
- Electrical stimulation of the fibular and tibial muscle groups of both feet,
- Cathode galvanization to the hands and feet, twice daily,
- Whirlpool massage of the upper and lower limbs, once daily,
- Low-frequency alternating magnetic fields with the following parameters: field intensity of 5 mT, frequency of 30 Hz, bipolar sine wave, duration of 15 min, twice daily to the wrists and hands and to the feet and lower legs.

The kinesiotherapy program included:

- Active exercises with no weight-bearing on the hip and knee joints,
- Active exercises with regulated resistance for the extensors and flexors of both feet,
- Exercises with regulated intensity on an ergometer,
- Self-assisted exercises of the ankles,
- Gait training and correction.

Kinesiotherapy procedures were performed twice daily, in the morning and in the afternoon.

Discussion

Physical therapy and rehabilitation used in the patient working as a quality inspector at a gas station, performed 6 days a week for 6 weeks, resulted in an improvement in the form of reduced nocturnal acroparesthesia, improved muscle strength in the weakened muscle groups (feet flexors, 3; feet extensors, 4 in the Lovett scale). Taking into account 20 years' occupational exposure to hydrocarbons from gasoline fumes, i.e., tetraethyl lead and toluene, specialist consultations at an occupational medicine center, and the clinical findings, the patient was diagnosed with toxic polyneuropathy and encephalopathy in the course of chronic gasoline poisoning. A further six months' follow-up showed increasing central and peripheral nervous system deficits. This was probably caused by permanent, chronically progressive damage to the nervous system.

At work, authors from Sweden, Helander et al. [25], who examined the concentration of Pb in the liver of dead birds , eagle owls, stated that in these birds in the north of Sweden, the concentration of Pb in the liver was love/low population, few cars.

In central Sweden and the south of the country, Pb levels were high in the liver of birds. These are more densely populated areas of Sweden and there are more cars.

After treatment conducted in the patient with yew needle extract poisoning, her mental status and ability to maintain contact were considerably improved and further increases in muscle strength and physical function were seen, which helped the patient walk unassisted using a walking frame and perform the activities of daily living.

Conclusion

In order for physical therapy and rehabilitation to be effective in patients with axonal sensorimotor polyneuropathies, it is crucial to establish the causal factor. In the cases presented above, the causes were discovered as a result of detailed occupational history-taking, which included talking both to the patients and to their family members, clinical examination findings, and accessory investigations (NMR, EMG). Physical therapy and rehabilitation must be patient-specific and guided by the patient's current clinical status, including their perception abilities (toxic encephalopathy) and muscle fatigue resulting from permanent peripheral nerve damage (polyneuropathy).

The above examples show that even in patients with permanent, chronically progressive damage to the central and peripheral nervous system, painstaking efforts of the team responsible for their physical therapy and

rehabilitation, lasting many weeks, help improve the patients' mobility.

REFERENCES

1. Thomas PA, Polwart A (2003) *Taxus baccata* L. J Ecol 91: 489-524.
2. Cragg GM, Schepartz SA, Suffness M, Grever MR (1993) The taxol supply crisis. New NCI policies for handling the large-scale production of novel natural product anticancer and anti-HIV agents. J Nat Prod 56: 1657-1668.
3. Cope EA (1998) Taxaceae: The genera and cultivated species. Bot Rev 64: 291-322.
4. Jaziri M, Zhiri A, Guo YW, Dupont JP, Shimomura K, et al. (1996) *Taxus* sp. cell, tissue and organ cultures as alternative sources for taxoids production: A literature survey. Plant Cell Tissue Organ Cult 46: 59-75.
5. Sabater-Jara AB, Tudela LR, López-Pérez AJ (2010) In vitro culture of *Taxus* sp.: Strategies to increase cell growth and taxoid production. Phytochem Rev 9: 343-356.
6. Zhang CH, Xu HB (2001) Improved paclitaxel production by *in situ* extraction and elicitation in cell suspension cultures of *Taxus chinensis*. Biotechnol Lett 23: 189-193.
7. Zhong JJ (2002) Plant cell culture for production of paclitaxel and other taxanes. J Biosci Bioeng 94: 591-599.
8. Onrubia M, Moyano E, Bonfill M, Cusidó RM, Goossens A, et al. (2013) Coronatine, a more powerful elicitor for inducing taxane biosynthesis in *Taxus media* cell cultures than methyl jasmonate. J Plant Physiol 170: 211-219.
9. Sabater-Jara AB, Onrubia M, Moyano E, Bonfill M, Palazón J, et al. (2014) Synergistic effect of cyclodextrins and methyl jasmonate on taxane production in *Taxus media* cell cultures. Plant Biotechnol J 12: 1075-1084.
10. Croteau R, Ketchum REB, Long RM, Kaspera R, Wildung MR (2006) Taxol biosynthesis and molecular genetics. Phytochem Rev 5: 75-97.
11. Bruňáková K, Košuth J, Katkovčinová Z, Lázárová M, Čellárová E (2010) Expression of two genes of paclitaxel biosynthetic pathway during germination of *Taxus baccata* zygotic embryos. Biol Plant 54: 515-519.
12. Ramirez-Estrada K, Osuna L, Moyano E, Bonfill M, Tapia N, et al. (2015) Changes in gene transcription and taxane production in elicited cell cultures of *Taxus media* and *Taxus globosa*. Phytochemistry 117: 174-184.
13. Hernberg S (2000) Lead poisoning in a historical perspective. Am J Ind Med 38: 244-254.
14. Kalra V, Sahu JK, Bedi P, Pandey RM (2013) Blood lead levels among school children after phasing-out of leaded petrol in Delhi, India. Indian J Pediatr 80: 636-640.
15. Sawalha AF, Wright RO, Bellinger DC, Amarasiriwardean C, Abu-Taha AS, et al. (2013) Blood lead level among Palestinian school children: A pilot study. East Mediterr Health J. 19: 151-155.
16. Papi L, Chericoni S, Bresci F, Giusiani M (2013) Fatal acute poisoning from massive inhalation of gasoline vapors: Case report and comparison with similar cases. J Forensic Sci 58: 552-555.
17. Afolayan JM, Edomwonyi NP, Esangbedo SE (2014) Carbon monoxide poisoning in a Nigerian home: Case reports. Niger Postgrad Med J 21: 199-202.
18. Etchevers A, Bretin P, Lecoffre C, Bidondo ML, Le Strat Y, et al. (2014) Blood lead levels and risk factors in young children in France. Int J Hyg Environ Health 217: 528-537.
19. Mitri S, Fonseca AS, Otero UB, Tabalipa MM, Moreira JC, et al. (2015) Metabolic polymorphisms and clinical findings related to benzene poisoning detected in exposed Brazilian gas-station workers. Int J Environ Res Public Health 12: 8434-8447.
20. Wang L, Li Z, Huang SX, Chuang DU, Wang H, et al. (2015) A systematic assessment of blood lead level in children and associated risk factors in China. Biomed Environ Sci 28: 616-619.
21. Vorvolakos T, Arseniou S, Samakouri M (2016) There is no safe threshold for lead exposure: A literature review. Psychiatriki 27: 204-214.
22. Emmerich SJ, Wang L, Persily AK (2017) Measured carbon monoxide concentrations from stock and reduced-emission prototype portable generators operated in an attached garage. J Air Waste Manag Assoc 67: 889-898.
23. Murthy AS, Das S, Hanuman SB (2018) Fatal diesel poisoning: a case report and brief review of literature. Am J Forensic Med Pathol 39: 169-172.
24. Carpenter C, Potts B, von Oettingen J, Bonnell R, Sainvil M, et al. (2019) Elevated blood lead levels in infants and children in Haiti, 2015. Public Health Rep 134: 47-56.
25. Helander B, Sundbom M, Runkel AA, Bignert A (2019) Temporal changes in concentrations of lead and other trace metals in free-ranging Eurasian eagle owls *Bubo bubo* in Sweden. Arch Environ Contam Toxicol 77: 377-389.
26. Kuliński W (2012) Physical therapy in medical rehabilitation. Wrocław, Wyd. Elservier Urban Partner, pp: 351-411.