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Case Report

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# Toxic Polyneuropathy

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## Abstract

Toxic polyneuropathy (TP) is caused by numerous organic and non-organic chemical agents, such as organic solvents, organophosphates, heavy metals (arsenic, talium, plomo), illicit drugs, drugs (chemotherapeutic, immunosuppressive, antibiotics, antiarrhythmic, antiepileptic, vitamin E6 ), marine biotoxins (ciguatera) and alcohol, among others. TP is less frequent than metabolic, inflammatory and hereditary, reporting between 2-4% as a cause of neuropathy. In contrast, preventable causes, and when identified early, can be reversible or prevent their progress, but avoid unnecessary spending and invasive investigations[1-3].

The PT is of axonal type, distal in the lower limbs (LL), involves thick, thick or both fibers, with a predominance of sensory symptoms. In order to establish a causal relationship between the toxic and TP, the following diagnostic criteria have been suggested: characteristic clinical picture; toxic to produce TP; temporal relationship of symptoms and toxic exposure; definite exposure and related to the dose; now and in the progress after stopping the exposure; reproducible neuropathy in experimental animals and exclusion of other causes[1-4]. The record of TP cases motivated us to make this report.

## Introduction

Toxic polyneuropathy (TP) is caused by numerous organic and non-organic chemical agents, such as organic solvents, organophosphates, heavy metals (arsenic, talium, lead), illicit drugs, drugs (chemotherapeutic, immunosuppressive, antibiotics, antiarrhythmic, antiepileptic, vitamin E6 ), marine biotoxins (ciguatera) and alcohol, among others. TP is less frequent than metabolic, inflammatory and hereditary, reporting between 2-4% as a cause of neuropathy. In contrast, preventable causes, and when identified early, can be reversible or prevent their progress, but avoid unnecessary spending and invasive investigations [1,2,3].

The PT is of axonal type, distal in the lower limbs (LL), involves thick, thick or both fibers, with a predominance of sensory symptoms. In order to establish a causal relationship between the toxic and TP, the following diagnostic criteria have been suggested: characteristic clinical picture; toxic to produce TP; temporal relationship of symptoms and toxic exposure; definite exposure and related to the dose; now and in the progress after stopping the exposure; reproducible neuropathy in experimental animals and exclusion of other causes [1,2,3,4].

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## Case Report

### Case 1

37 years old, with personal or family history, consultation for muscle weakness, pares-

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thesias and pain in three months of evolution, slow and progressive. Reported that 30 days before the symptoms, after a psychological stress, intended suicide with ingestion of trichlorphon, being hospitalized at the time. Business use of medicines and toxic habits. The physical examination: good general status, normal vital signs and changes in systems. Neurological examination: Mental state, language and normal cranial pairs, non-cerebellar vestibular signs. In LL presented symmetrically, distal predominance: hypoesthesia, hypopalesthesia, paraparesis with muscular strength for the dorsiflexion of the feet 3/5, patellar and achilleal flexion.

Laboratory exams including liver, thyroid and renal function, metabolic, inflamatory and immunological markers and CSF were normal. Electromyography: signs of denervation of the rectus femoris and vastus lateralis muscles, bilaterally in the anterior tibialis, medial gastrocnemius and extensor cutaway of the fingers. Nervous conduction shows the demining of the amplitude of the potentials of muscular action composed of the bilateral posterior tibial and deep fibular nerves, with slight delay in the distal latency and inaccuracy of bilateral sural and superficial fibular nerves. After a few months of evolution the sensory-motor frame remained stable.

**Case 2**

Man of 20 years, healthy, operator of sterilization, without family history of neurological illness, without use of medication of toxic habits. Consultation by paresthesias and dissolution of muscle strength in LL of 72 hours of evolution. Negative business and previous gastrointestinal or respiratory symptoms. The physical examination: good general status, normal vital signs and changes in systems. Neurological examination: Mental state, language and normal cranial pairs, vestibular or cerebellar signs. In LL, it was symmetrically distal in predominance: hypoesthesia, hypopalesthesia, paraparesis, muscular strength for dorsiflexion of the 4/4 degrees, patellar and Achilles flexion. Laboratory exams were normal. Electromyography: Demining of the recruitment pattern in the bilateral extensor muscle of the cut fingers. The motor and sensory nervous conduction of the four members show retraction of the distal motor latency and the disruption of the amplitudes of the muscular action potentials of the bilateral deep fibular nerve and the disruption of the sensory potential of the bilateral superficial fibular nerve. The evolutionary study was carried out six months after, showing a better amplitude of the sensory potential of the superficial fibular accompanying the clinical improvement.

Case 1 presents a late sensory, motor-distal polyneuropathy, distal in LL, axonal, and central nervous system impairment, following an oral intake of trichlorphon by suicidal intent. The trichlorphon is an anthelmintic used in animals. It is an organophosphate (OF) with highly toxic systemic activity and its effects caused by inhibition of the enzyme acetylcholinesterase, which has the function of hydrolyzing acetylcholine released in the synaptic union of the central nervous system, autonomous system and neuromuscular union. Exposure to OF may be transdermal, respiratory or digestive, manifesting by neurological, behavioral and psychiatric syndromes of acute evolution, subacute or chronic, Among them, late polyneuropathy appears as the most common chronic manifestation, characterized by a latency period of several weeks, with distal axonal degeneration in LL[5-7].

Case 1 meets the clinical criteria for TP by organophosphate. Typically it presents with late symptoms after exposure to OF (4 weeks), with distal sensorimotor symptoms in LL and axonal injury, as described by other authors[5-9]. Similar to the case published by Ergün et al[7] and Vasconcelos et al[9], it is a young adult woman who intended suicide with OF.

Case 2 presents an axonal sensory-motor polyneuropathy and impairment of the central nervous system, following occupational exposure to ethylene oxide and epoxy ethane. Ethylene oxide (OE) is a flammable gas at room temperature, used as a fungicide and in cold sterilization of heat sensitive medical materials such as cardiac pacemakers, tubes, blood oxygenators and kidney dialysis, among others. Its cytotoxicity is based on the alkylating action of sulfhydryl, amine and carboxyl groups, integrating protein molecules, which would explain its multiple clinical manifestations. Chronic exposure to OE can provoke a mixed polyneuropathy of axonal type. This second case, working in contact with the OE, presents a distal TP of the lower limbs, of axonal type and secondary to chronic exposure to the OE, with a clinical and topographic pattern similar to those described above[10-12].

In the cases presented, the clinical history, personal history, family history and complementary examinations will allow excluding other causes of polyneuropathy, the exclusion criterion necessary to diagnose a TP. Nerve biopsy was deferred by the typical clinical setting in both cases, but it was a bloody, high-cost examination because it was estimated as unnecessary.

## Conclusion

The present authors present the rare and preventable cases of polyneuropathy. Concluding that the TP can be preventable, and when diagnosed soon, it will be able to reverse the detention of the evolution of the process as it happened in some cases. On the other hand, workers exposed to substances with potential toxicities to the nervous system should be monitored clinically, and when necessary and possible, to carry out further examinations and dosages of the toxic agent, in addition to ensuring the levels of toxic exposure and compliance. established security measures.

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