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Case report

Co-existence of peripheral neuropathy secondary to lead poisoning and chronic polymyositis: Case report☆

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ABSTRACT

A case is presented on a 47-year-old man with chronic lead poisoning with typical symptoms after 5 years of occupational exposure. He consulted for generalized muscle weakness, early dysphagia, cephaloparesia, and paresthesias in upper and lower limbs. He also had atrophy and decreased proximal muscle strength (deltoid and medial vast) and in both flexor and extensor muscles of the neck. He had a history of high blood lead levels and peripheral neuropathy documented by electromyography. In addition to the diagnosis of lead poisoning, inflammatory myopathy was confirmed based on muscle enzyme elevation, muscular inflammation in magnetic resonance imaging, and typical findings in a muscle biopsy. To the best of our knowledge, this is the first report where both conditions are documented in one patient.

Coexistencia de neuropatía periférica secundaria a intoxicación crónica por plomo y polimiositis: Reporte de caso

Palabras clave:
Intoxicación por plomo

RESUMEN

Se reporta el caso de un hombre de 47 años con intoxicación crónica por plomo, secundaria a exposición laboral de 5 años, con sintomatología típica de saturnismo. Consultó por

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Enfermedades del sistema nervioso periférico
Polimiositis

debilidad muscular generalizada, disfagia y parestesias en extremidades. Se documentaron altos niveles de plomo en sangre, asociados a neuropatía periférica, confirmada por electromiografía, y disminución de la fuerza muscular en cintura escapular y pélvica (deltoides y vasto medial), así como atrofia de músculos del cuello (flexores y extensores) manifestada como cefaloparesia. Adicional al cuadro de saturnismo se diagnosticó miopatía inflamatoria con base en la elevación de enzimas musculares, miositis por resonancia magnética nuclear y biopsia muscular compatible, siendo, hasta donde se sabe, el primer reporte conocido de la coexistencia de estas 2 enfermedades.

Introduction

Exposure to lead, as a result of the industrial activity and its massive use in household products, emerges as one of the problems of higher prevalence and impact on public health.[1–4] Currently, the main sources of exposure are circumscribed to an occupational setting (manufacturing of paint, glass, ceramics, among others) and in the environment they are derived from the combustion of gasoline, industrial emissions and food contamination; both sources can generate toxicity in human beings.[5] Patients with lead poisoning may have clinical manifestations of acute or late onset; in adults, the clinics becomes more evident when the lead serum levels are within moderate or high ranges (between 40 and 80 μg/dL), with gastrointestinal symptoms (abdominal pain, vomiting, constipation) being the most frequent.[6–8] In individuals with plumbemias exceeding 100 μg/dL, serious manifestations such as nephropathy, hepatic necrosis and anemia, and varied neurological manifestations such as peripheral neuropathy, carpal tunnel syndrome and encephalopathy occur.[9] Regarding other symptoms, for example musculoskeletal, those which have been more often mentioned in the different case series are: arthralgia (14–28%) and weakness of the extensor muscles of wrists and ankles (13%).[10] After an extensive literature search, as far as is known, no clinical articles reporting the simultaneous presence of inflammatory myopathy and peripheral neuropathy in patients with lead poisoning were found.

Case report

A 47-year old-man with chronic lead poisoning secondary to occupational exposure for 5 years, with typical symptoms of saturnism: headache, hyporexia, diffuse abdominal pain and persistent decreased libido despite multiple treatments, among them: BAL dimercaprol, succimer and ethylenediaminetetraacetic acid. He consulted for generalized muscle weakness and paresthesias in upper and lower limbs, associated with high dysphagia for solids and liquids. On physical examination was found decreased muscle strength, symmetrical, with proximal predominance, accompanied by significant atrophy of the deltoid and vastus medialis muscles, as well as of the neck flexors and extensors, with secondary cephaloparesia.

Table 1 – Relevant results of paraclinical analyses in a patient with lead poisoning with muscle weakness and paresthesias.

Paraclinical	Value	Reference range
Blood lead levels	97.61 μg/dL	0–10
Creatine phosphokinase	1000 U/L	0–153
Lactic dehydrogenase	556 U/L	0–250
Aldolase	8.4 U/L	0–6

The main findings in the laboratory tests carried out are in Table 1. It drew the attention, clinically, the weakness as cardinal symptom, the inability of the patient to get up from sitting position, not being able to raise his arms above his head and the cephaloparesia; these characteristics compelled to explore causes of weakness of muscle origin. Toxic (no consumption of alcohol, glucocorticoids or lipid lowering agents), endocrine (hormonal and electrolytic profiles without alterations), malignant (normal thoracoabdominal CT scan, digestive endoscopy and colonoscopy; serum and urine immunofixation with no evidence of monoclonal gammopathy) and infectious (negative serologies for bacteria and fungi) causes were ruled out.

An electromyography was performed as part of the diagnostic process, finding, in addition to the expected neuropathic pattern, polyphasic potentials, early recruitment and membrane irritability, findings highly suggestive of muscle fibers disease (Figs. 1 and 2).

In order to define the degree of muscle atrophy, a nuclear magnetic resonance imaging of the girdle (scapular and pelvic) was performed to the patient, finding predominance of atrophy, but with areas of active myopathy (hyperintensity in the right lateralis vastus until the formation of the lateral retinaculum) (Fig. 3). With these results, it was decided to take the patient to muscle biopsy, confirming the diagnosis of inflammatory myopathy (endomysial commitment and expression of HLA-1) (Figs. 4–6).

Discussion

Lead poisoning can have a broad spectrum in its clinical presentation and a variable intensity, depending mainly on the serum levels of this metal. A case of concomitance of peripheral neuropathy and polymyositis is presented in this report. As mentioned above, in several series of cases have