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Case Report

Cognitive Complications in a Case of Chronic Non-Occupational Lead Intoxication: A Case Report

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ABSTRACT

Cases of lead poisoning in adulthood are predominantly of occupational origin while, in childhood, poisoning from domestic sources is more common, especially in the presence of behavioral disorders (e.g., pica). This case report examines the case of a 52-year-old man with non-occupational lead exposure and a silent neuropsychiatric history. The onset clinical picture was characterized by asthenia, generalized malaise, abdominal pain and anemia, which appeared within the preceding 2 months. He was admitted following the appearance of neurological symptoms (confusional state, balance disturbances, memory disorders) and initially evaluated to rule out myeloproliferative diseases and gastrointestinal causes of blood loss. Among the required investigations were performed: A total-body CT scan which showed "hyperdense punctiform images, with bone density, probably attributable to ingestion" and a peripheral venous blood smear that showed "anisocytosis and anisochromia compatible but not pathognomonic for lead anemia". It was only then that the patient's hobby became known: He produced lead fishing sinkers himself, which were attached to the fishing line with his teeth. Some of these were accidentally ingested and led to chronic intoxication (first detected lead value 546 ug/l). Neuroradiological and neurocognitive examinations performed documented selective deficits in verbal memory, logorrhea and lightly incoherent and inappropriate behavior.

Keywords: Lead; Lead poisoning; Neurocognitive deficits; Fishing sinkers; Cognitive complications; Non-occupational lead intoxication

INTRODUCTION

Lead is a soft, dense, ductile, and malleable grey-blue metal. Lead occurs widely in nature, both in organic and inorganic forms, and has been widely used in numerous contexts. Exposure to lead can occur through ingestion or inhalation, mainly, but dermal and prenatal exposures have also been described.

Exposure in adulthood occurs mainly in occupational settings through inhalation of vapors and dust, with lead being used in battery production, recycling plants, construction for renovation and demolition, welding and painting, plumbing, the rubber and plastics industry, munitions production, and automotive and radiator repair. Non-occupational exposure, mainly from domestic sources, is more frequent in the pediatric age, also about the greater propensity of children to carry objects in their mouths; numerous objects may contain lead, including: Lead paint, water, soil, food, children's toys, cosmetics, alternative medicine or natural remedies, etc.[1].

CASE PRESENTATION

The toxic effects of lead are related to its concentration in the

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blood [1]. Lead absorption occurs mainly through the respiratory tract and the gastrointestinal tract, from where it enters the bloodstream and is distributed to various organs, in particular the nervous system, the haemopoietic system, the kidneys, and the gastrointestinal system; it can also cause hypertension and infertility [2]. Clinically, lead poisoning manifests itself with non-specific signs and symptoms including abdominal pain, constipation, anemia, irritability, short-term memory impairment, myalgia, and neuropathy.

Neurological symptoms include ataxia, stupor, coma, convulsions, and hyperirritability, reduced IQ, reduced attention span, increased antisocial behavior, reduced educational attainment, and even death [3]. The nervous system is the most sensitive tissue to lead toxicity, both in children and adults. Long-term exposure in adults may result in decreased performance in certain cognitive tests that measure nervous system function [4].

A previously healthy 52-year-old man, who smoked from 18 to 52 of 20 cigs/day, was admitted to a peripheral hospital due to the onset of confusion, balance disorders, and amnesia; He also reported asthenia, muscle weakness, retrosternal pyrosis, and abdominal pain in the lower quadrants of the abdomen in the last three months. The blood tests showed anemia (Hb 7.5 g/dL), severe hyponatremia (Na^+ 116 mEq/l), and increased inflammation indexes (PCR 4.57, neutrophilic leukocytosis). The patient was therefore treated with empiric antibiotic therapy, hypertonic solution and, on suspicion of myeloproliferative disease, was performed instrumental examinations including colonoscopy, gastroscopy, a total body CT scan with evidence of “hyperdense punctiform images, with bone density, probably attributable to ingestion” (Figure 1), and a peripheral venous blood smear that highlighted “anisocytosis and anisochromia compatible but not pathognomonic for lead anemia”. The patient was initially discharged with a diagnosis of "anemia, leukocytosis, and acute hepatitis in lead blood accumulation”.

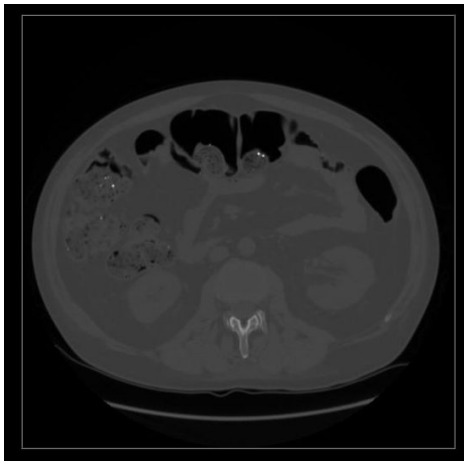

Figure 1: Radiopaque ingestions in the bowel found on CT scan.

The first peripheral venous blood sampling after the discharge revealed high blood lead levels (Pb-B) 546 ug/l (n.v. 0-350 ug/l). At the check-up, carried out at the toxicology clinic, a Pb-B 93 ug/dl (n.v. 1-16), urinary lead (Pb-U) 111.2 ug/g (n.v. 1-18), Porphobilinogen (PBG) 0.94 mg/g (n.v. 0-2), delta-Aminolevulinic Acid (dALA) 23.5 mg/g (n.v.<6), Zinc-Protorphyrin IX (ZPP) 146.3 ug/dl (n.v.<40). It was therefore decided to carry out a scheduled admission to the occupational medicine clinic and clinical toxicology for further diagnostic and therapeutic investigations. The blood tests carried out on admission showed normochromic normocytic anemia (Hb 12.3 g/dL, MCH 29.1, MCV 85.1), Pb-B 66 ug/dl (n.v. 1-16), ZPP 183.9 ug/dl (n.v.<40), Pb-U 1166.0 ug/g (n.v. 1-18), dALA 2 mg/g (n.v.<6). Clinical objectivity was normal, except for logorrhea.

Finally, the anamnesis revealed that the patient was in the habit of using lead pellets for fishing, which were crushed with his teeth to be attached to the thread, leading to the unintentional ingestion of some of them. This activity, was reported to have intensified in frequency during March/April 2020, due to the lockdown, and was performed for approximately 3-4 hours per day. During the hospital stay, the patient underwent various instrumental examinations: Cardiac Doppler ultrasound showed no significant changes, the electroencephalogram was normal, as was the electromyography of the lower limbs. An encephalic MRI was also performed, which was negative and an evaluation of the cognitive state, revealed the presence of selective deficits in verbal memory, from interference sensitivity, with intrusion phenomena. The patient was therefore treated with chelation therapy by administering EDTA for the four days following admission to the ward, obtaining the following values on discharge: Pb-B 39 ug/dL (n.v. 1-16), Pb-U 133 ug/g, ZPP 185.9 ug/dL.

After discharge, the patient reported giving up the fishing hobby; periodic checks of lead and effect indicators were performed, with evidence of gradual reduction of these values until they normalized. After two months Pb-B were 56 ug/dl (n.v. 1-16), Pb-U 76,7 ug/g (n.v. 1-18), PBG 0,63 mg/g (n.v. 0-2), dALA 1,1 mg/g (n.v.<6), ZPP 133,2 ug/dl (n.v.<40). After three months Pb-B were 38 ug/dl (n.v. 1-16), Pb-U 20,7 ug/g (n.v.<6, 0), dALA 2,2 mg/g (n.v. <6, 0), ZPP 56,6 ug/dL (n.v.<40).

After six months Pb-B were 28 ug/dl (n.v. 1-16), Pb-U 26,1 ug/l, dALA 3,7 mg/g. Creat (n.v.<6), ZPP 26,4 ug/dl (n.v. 0-4). One year later the discharge Pb-B was stable at 28 ug/dl (n.v. 1-16), Pb-U 18 ug/l, dALA 3 mg/g. Creat (n.v.<6), ZPP 21 ug/dl (n.v. 0-4). After eighteen months Pb-B was 11.97 ug/dl (n.v. 1-16) and on June 2022 Pb-U was 56.6 ug/g. Crea (n.v. 1-18), dALA 5.4 mg/g. Crea (n.v.<6) and ZPP 28.3 ug/dL (n.v.<40). Finally, an assessment of the cognitive status was repeated more than a year later, which showed an improving trend in verbal memory tests compared to the previous in-patient assessment.

RESULTS AND DISCUSSION

Thus, the clinical case presented is interesting because of the peculiar etiology; non-occupational lead intoxication is not uncommon but is typically pediatric, mostly related to the phenomenon of pica [5], and/or environmental, related to reduced socioeconomic conditions, delineating poverty as a risk factor [6]. The case described, however, cannot be placed in

either of the above two categories, going to highlight a mainly "hobbyist" cause of lead poisoning not yet well explored in the literature. Few cases of pica in adulthood have been described [7]. Cases of accidental intoxication in adulthood are mainly related to hunting (because of the ingestion of animals, e.g., fish, containing lead elements [8]), or in domestic settings [9]. The second element of interest is the clinical presentation. Typically, lead intoxication manifests at the onset with gastrointestinal complaints, such that it is among the less frequent differential diagnoses of acute abdomen [8,10]. In this case, the most prominent complaints were cognitive deficits. The peculiarity of these deficits is the onset in the adult subject who, unlike the cases observed in pediatric age, did not suffer the harmful effect of lead on the nervous system, including the central nervous system, during the age of development. The greatest risk in pediatric age is the possibility of developing irreversible disabilities and cognitive defects, up to, in the worst case scenario, seizures, coma, and exitus [11].

CONCLUSION

The occurrence of neurocognitive deficits in chronic lead intoxication is typical of childhood with the possibility of developing irreversible disabilities and deficits. In our case, however, there was reversibility of the neurological impairment following removal from the source of exposure, chelation therapy with Ethylenediaminetetraacetic Acid (EDTA) and normalization of lead values (last value 11,97 ug/dL). In contrast, the case described showed reversibility of the above deficits, described as amnestic deficits, concerning the normalization of Blood Lead Level (BLL).

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

REFERENCES

1. Dapul H, Laraque D. Lead poisoning in children. Adv Pediatr. 2014;61(1):313-333.
2. Marginean CO, Melit LE, Moldovan H, Lupu VV, Marginean MO. Lead poisoning in a 16-year-old girl: A case report and a review of the literature (CARE compliant). Medicine. 2016;95(38):e4916.
3. Hosseini MS, Salimi A, Phillips S, Zamani N, Hassanian-Moghaddam H. Adult lead poisoning caused by contaminated opium: A two-year longitudinal follow-up study. Ann Glob Health. 2021;87(1):89.
4. Ara A, Usmani JA. Lead toxicity: A review. Interdiscip Toxicol. 2015;8(2):55-64.
5. Jouhadi Z, Bensabbahia D, Chafiq F, Oukkache B, Guebessi NB, Najib J, et al. Lead poisoning in children: A case report. Pan Afr Med J. 2016;24:316.
6. Wheeler W, Brown MJ. Blood lead levels in children aged 1-5 years-United States, 1999-2010. MMWR Morb Mortal Wkly Rep. 2013;62(13):245-248.
7. Sabouraud S, Testud F, Descotes J, Benevent M, Soglu G. Lead poisoning following ingestion of pieces of lead roofing plates: Pica-like behavior in an adult. Clin Toxicol. 2008;46(3):267-269.
8. Grade T, Campbell P, Cooley T, Kneeland M, Leslie E, MacDonald B, et al. Lead poisoning from ingestion of fishing gear: A review. Ambio. 2019;48:1023-1038.
9. Nicolli A, Mina GG, De Nuzzo D, Bortoletti I, Gambalunga A, Martinelli A, et al. Unusual domestic source of lead poisoning. Int J Environ Res Public Health. 2020;17(12):4374.
10. Yang Y, Li S, Wang H, Liu M, Tuo B, Wu H, et al. Chronic lead poisoning induced abdominal pain and anemia: A case report and review of the literature. BMC Gastroenterol. 2020;20(1):1-5.
11. Needleman HL, Gunnoe C, Leviton A, Reed R, Peresie H, Maher C, et al. Deficits in psychologic and classroom performance of children with elevated dentine lead levels. N Eng J Med. 1979;300(13):689-695.