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Case Report

Severe Zinc Poisoning in the Peruvian Amazon: A Case Report

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ABSTRACT

Background: Zinc is an essential trace element for humans, although its excessive and prolonged ingestion or inhalation can cause harmful health problems.

Case Presentation: We report the clinical case of a 25-year-old male patient from Yambrasbamba, Amazonas, Peru, who carried out agricultural and livestock activities in an area adjacent to a zinc oxide mining project closed in 2007 and 2008. The patient was admitted to the Lambayeque Regional Hospital with initial symptoms of decreased muscle strength, low back pain, nausea and vomiting, vesicular skin rash and polydipsia. Supporting tests revealed a notable increase in transaminases and total creatine phosphokinase and abdominal radiological image with multiple metallic calcifications along the colon. The history of consuming spring water with unusual organoleptic characteristics suggested environmental exposure to heavy metals. Analysis confirmed the diagnosis of chronic zinc poisoning. Compared with poisonings by other metals, such as mercury, zinc showed distinctive cutaneous symptoms and a favourable response to supportive measures.

Conclusion: This case underlines the importance of considering environmental exposures in the differential diagnosis of patients with non-specific symptoms and highlights the need to raise awareness of the risks associated with zinc and other heavy metal poisoning in rural settings.

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Introduction

Zinc is one of the most important trace elements for human health, as it performs various biological functions: structural, catalytic, and regulatory. The adult body contains between two and three grams of zinc, but only approximately 1% of this is replenished each day [1]. On the other hand, according to the Food and Nutrition Board of the United States of America, the maximum daily intake of zinc for adults is 40 mg [2]. Furthermore, the human body does not have a specific zinc reserve, but rather it is distributed in different parts, with the intracellular environment being the dominant one (95%); and its excretion is mainly via the pancreas, bile, and intestines, and to a lesser extent through urine, hair, sweat, and semen [1, 3].

However, excessive intake can lead to disease [4, 5]. In fact, overexposure to zinc occurs through three main routes: a) inhalation, through smoke generated by industrial processes or military smoke explosives; b) dermal, although this mainly causes skin irritation and is not considered a toxicity risk; and c) oral, where excessive ingestion can cause disease [6]. In fact, cases of zinc poisoning from environmental sources are extremely rare. Intake of more than 15 mg/L in water usually causes nausea, vomiting, diarrhea, epigastric pain, abdominal cramps, and blood lipid abnormalities, among other symptoms, and can even affect several organs due to the formation of phosphine gas that can enter the bloodstream [7].

Currently, the industrialization of zinc has led to a significant increase in its global production. Peru ranks as the second largest zinc producer globally, with 12% of world production, surpassed only by China, which leads the international market with 34%. At the national level, the regions of Ancash, Junín, and Pasco are the most prominent, accounting for 72% of the country's total production [8]. This reality carries significant risks of environmental contamination with zinc if proper protocols are not followed in mining activities. The objective of this study is to describe a clinical case of severe zinc poisoning in a young adult patient exposed to a probable environmental source.

Case Presentation

A 25-year-old male patient, a farmer, from the district of Yambrasbamba, province of Bongará, department of Amazonas, Peru; nine hours by car from the city of Chiclayo, Peru. The patient was admitted to the Lambayeque Regional Hospital through the emergency department on March 3, 2023. Ten days prior to admission, the patient began experiencing decreased muscle strength and pain in his lower limbs and lumbosacral region, associated with nausea and vomiting. Four days later, the pain increased with the appearance of epigastric pain associated with a skin rash on his chest, back, abdomen, and upper limbs.

On the day of admission, the patient had a normal appetite, polydipsia, normal bowel movements, diuresis without macroscopic alterations, and normal sleep patterns. He denied any history of illness but reported temporary consumption of spring water with an unusual taste. The water source is located on a remote property where agricultural activities are carried out sporadically and which is influenced by a closed mining project. He also reported a family history of a recently deceased brother with similar symptoms and exposure.

On physical examination, he was in apparent fair general condition, poorly hydrated, malnourished, and breathing spontaneously without oxygen support. His skin was normochromic, with capillary refill time of less than two seconds, and he had a diffuse vesicular rash on his neck and upper limbs. As for his nervous system, he was awake, lucid, and oriented in time, space, and person.

Laboratory tests showed a significant alteration in transaminases, with values of 2,044 U/L for aspartate aminotransferase and 1,485 U/L for alanine aminotransferase. Similarly, total creatine phosphokinase (total CPK) was 10 times its normal value. The rest of the laboratory tests were within normal values (Table 1). The anteroposterior chest X-ray showed no relevant findings (Figure 1a), while the abdominal X-ray showed multiple calcifications with a metallic density of 19798 HU, rounded in shape and varying in size, throughout the large intestine; no significant findings were observed in the other organs (Figure 1b).

Based on environmental factors reported by the patient, heavy metal poisoning is the presumptive diagnosis, with mercury being one of the most likely elements involved. For this reason, heavy metal levels in urine were tested, yielding the following results: 3,625 ug/24h of zinc (reference = 300 to 800), 0.9 ug/24h of cadmium (reference < 10.0 in occupationally exposed persons), 20.9 ug/24h of mercury (reference < 50), and 11.3 ug/24h of lead (reference < 80). The definitive diagnosis was chronic zinc poisoning.

The first-line treatment was intravenous hydration (1000 mL of 0.9% sodium chloride at 45 to 60 drops per minute for 12 days) and nothing by mouth upon admission, followed by a soft diet according to tolerance until discharge. Symptomatic treatment consisted of intravenous analgesia (100 mg tramadol

Table 1. Laboratory parameters of a patient with Zinc poisoning treated at the Lambayeque Regional Hospital, Lambayeque, Peru.

Laboratory parameters	Reference values	Day 1	Day 6	Day 8	Day 10
Complete Blood Count					
Hemoglobin (g/dL)	13 – 16	14,2	13,7	15,4	15,5
Hematocrit (%)	38 – 50	41,7	41,2	46,1	46,8
White Blood Cells (cells/µL) x 10^3	5,0 – 10,0	7,62	4,48	3,91	5,43
Segmented Neutrophils (%)	40 – 70	73,9	63,7	57,0	63,0
Eosinophils (%)	1– 4	3,3	7,6	5,0	4,5
Basophils (%)	0 –1	0,1	0,2	0,0	0,1
Monocytes (%)	4 – 8	8,5	9,5	8,0	5,4
Lymphocytes (%)	20 – 30	14,2	19,0	30,0	27,0
Platelets (platelets/µL) x 10^3	150 – 400	213	157	184	229
Blood Biochemistry					
Aspartate aminotransferase (U/L)	13,0 – 39,0	2 044	870	445	245
Alanine aminotransferase (U/L)	7,0 – 52,0	1 486	947	599	416
Total bilirubin (mg/dL)	0,30 –1,20	0,40	0,24	0,20	0,29
Direct bilirubin (mg/dL)	0,00 – 0,20	0,13	0,09	0,09	0,11
Indirect bilirubin (mg/dL)	0,20 – 0,80	0,27	0,15	0,11	0,18
Protein (g/dL)	6,60 – 8,30	5,28	5,02	6,54	6,53
Albumin (g/dL)	3,50 – 5,70	3,40	3,24	3,99	3,94
Globulin (g/dL)	2,00 – 3,50	1,88	1,78	2,55	2,59
Alkaline phosphatase (U/L)	30,0 –120,0	73	65	101	102
Gamma-glutamyl transferase (U/L)	9,0 – 64,0	15,2	20,8	31,6	27,4
Total creatine phosphokinase (U/L)	30,0 – 223,0	-	17 757	9 307	4 686

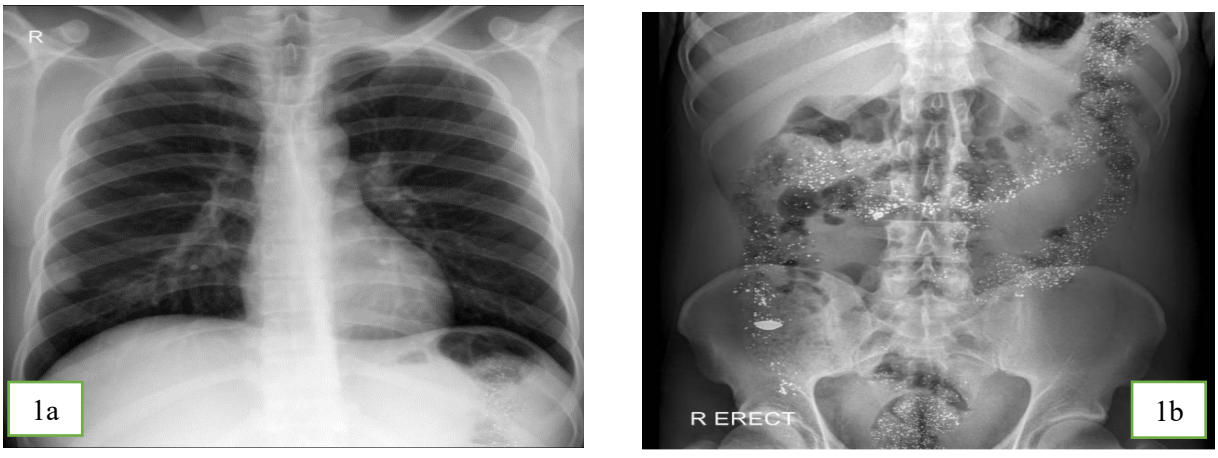

Figure 1. Chest X-ray (1a) and abdominal X-ray (1b) of patient exposed to zinc upon admission, day 1

and 50 mg dimenhydrinate in 1000 mL of 0.9% sodium chloride, intravenously every 8 hours for 12 days) and antihistamine (chlorphenamine 10 mg intravenously for the first 24 hours and then conditional on persistence of skin rash and pruritus).

As the hours passed, the symptoms decreased in frequency and intensity. Additionally, at the suggestion of gastroenterology, an osmotic laxative was prescribed (lactulose 3.3 grams in 5 mL at a dose of 30 mL at night for 7 days). A few days later, an increase in muscle

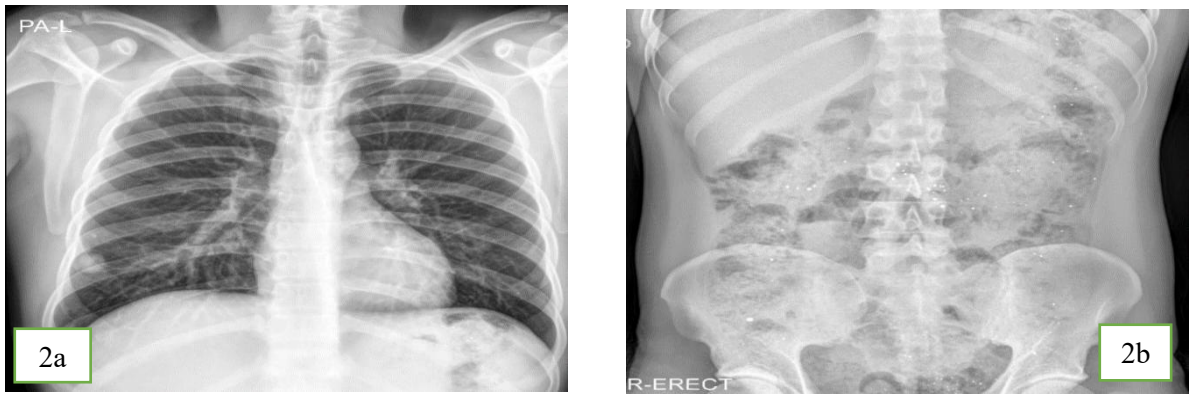

Figure 2. Chest X-ray (2a) and abdominal X-ray (2b) of a patient exposed to zinc on day 10 after treatment.

strength, a decrease in nausea and vomiting, as well as musculoskeletal and abdominal pain, and the disappearance of the skin rash were noted.

On the tenth day of admission, the patient's vital signs were stable, he was tolerating oral intake, and his initial symptoms had disappeared. Furthermore, imaging, specifically the abdominal X-ray, showed few small calcifications with metallic density, suggesting that most of the metal had been eliminated from the colon (Figure 2). In addition, laboratory markers such as transaminases and total CPK showed a significant and gradual decrease, although still above reference values (Table 1). Given this evidence and trends, discharge was considered.

It was not possible to obtain written informed consent from the patient because the decision to report the case was made after the patient had returned to his hometown, and communication was lost. For this reason, authorization was sought from the Research Ethics Committee of the same hospital in order to ensure compliance with ethical principles (Certificate No. 007-2023).

Discussion

The district of Yambrashbamba, in the province of Bongará, Amazonas Region, northern Peru, is known for a zinc oxide mining project processed through a calcination platform that remained in production in 2007 and 2008 [9, 10]. The spring water that the patient reported drinking may have been contaminated from these deposits. In the case presented, it can be observed that, as in other published cases, the clinical characteristics of zinc poisoning are similar: abdominal pain, nausea, and vomiting are among the most frequent symptoms [11, 12]. It is also often accompanied by fatigue, fever, myalgia, dyspnea, as well as hematological alterations such as leukocytosis and anemia, with these symptoms appearing through inhalation [13, 14]. However, given the scarcity of reported cases, the pathognomonic symptom of this type of poisoning is unknown.

Similarities were found in elevated transaminases in a patient with zinc phosphide poisoning, who presented with hypertransaminasemia with maximum values of 544 IU/L for AST and 1058 IU/L for ALT [15]; while in the current case, the maximum value for AST was 2044 IU/L and for ALT was 1486 IU/L. Hypertransaminasemia could be explained by a similar mechanism of action to that seen in zinc and aluminum phosphate poisoning. Phosphine gas is a protoplasmic poison that interferes with the function of cellular enzymes and proteins by blocking cytochrome C oxidase. In addition, it inhibits the entry of amino acids into myocardial protein synthesis, generating a change in the potential of the cardiac muscle cell wall, which could explain the release of CPK [16, 17].

On the other hand, the observed pancytopenia and sideroblastic anemia would be explained by copper deficiency; because the increase in zinc in the system induces greater synthesis of metallothionein (Mt), which, due to greater affinity, forms Mt-Cu complexes. [18]. Therefore, copper is excreted, decreasing its absorption, and since this is an element necessary for hematopoiesis, it produces cellular disorders [19].

Despite the chronic nature of the poisoning, the complete blood count showed no significant variations in the three cell lines. Therefore, it is likely that the ingested zinc did not reach levels that significantly suppressed copper absorption, which is essential for normal hematopoietic function. However, due to the lack of serum copper levels, this hypothesis could not be verified in this patient [6].

It is important to note that there are significant differences in the symptoms and severity of zinc and mercury poisoning. In this case, there was an erythematous rash predominantly on the chest, abdomen, and upper limbs, unlike mercury poisoning, where lesions predominantly occur on the lower limbs

[20]. Another notable difference is the appearance of neurological problems: memory loss and personality changes, manifestations that did not occur in this current case of zinc poisoning; therefore, this clinical aspect could help in the differential diagnosis with respect to mercury poisoning [21].

Acute heavy metal poisoning is usually treated with chelating agents such as ethylenediaminetetraacetic acid (EDTA), dimercaptosuccinic acid (DMSA), and penicillamine [5]. Although chelation with EDTA appears to be less effective with zinc, its administration is subject to a risk-benefit assessment for each patient [22]. In this case, chelating agents were not considered, as symptomatic treatment, hydration, and isolation from the environmental factor resolved the symptoms spontaneously.

This study has some limitations. Because the likely source of exposure is located in an inhospitable place, the chemical composition could not be determined, nor could zinc levels be confirmed. The exact amount ingested could not be established either. Furthermore, the time elapsed between consumption and the onset of symptoms could not be clearly established because the patient did not recall these details.

The low frequency of zinc poisoning poses diagnostic challenges due to the lack of specific symptoms, requiring epidemiological support and diagnostic testing. It is also important to educate healthcare professionals and the general public about the risks of excessive zinc exposure, particularly through dietary supplements and consumer products that may be contaminated. [1].

Conclusion

This clinical case demonstrates the severity and complexity of chronic zinc poisoning from an environmental source, presenting with muscle weakness, nausea, and rash, as well as altered transaminases and total CPK. A comprehensive clinical and epidemiological evaluation is recommended, followed by confirmation through quantification of the chemical element.

Conflicts of Interest

The authors report there are no competing interests to declare.

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