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Lead Poisoning in a Family Due to Cocktail Glasses

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An occurrence of lead poisoning in the adult members of a family was investigated clinically, epidemiologically and in the toxicology laboratory. The occurrence was noteworthy in that (1) the lead exposure was from commercially manufactured and distributed cocktail glasses; (2) machine dishwashing initiated the process of lead dissolution; (3) extremely high doses of lead ingestion were estimated; (4) these doses were related to sequential clinical measurements.

An occurrence of lead poisoning in a family due to cocktail glasses is described here primarily for two reasons: (1) the glasses are of commercial manufacture and represent a public health threat; (2) dose and time relationships could be quantified better than in some previous reports. Review of the literature suggests that although lead poisoning from ceramic beverage containers is an international problem and widely recognized [1–9], commercially manufactured and distributed glasses have heretofore not been implicated in the United States. Furthermore, in English language publications there has been insufficient specification of dosage and time in relation to clinical measurements, matters of concern to clinician, epidemiologist and toxicologist.

CASE REPORT

A thirty-nine year old Caucasian woman was first seen by her physician on January 29, 1970 (time noted as day one on Figure 1), because of a two day history of abdominal cramps, vomiting and constipation. She had been in good health except for an appendectomy sixteen years previously. On initial examination her blood pressure was 138/80 mm Hg and pulse rate 88/minute. She appeared slightly flushed and uncomfortable from the abdominal pain. The abdomen was soft, slightly distended and tympanitic on percussion. There was slight tenderness in the right upper quadrant, and bowel sounds were hypoactive. Flat and upright roentgenograms of the abdomen showed no remarkable abnormalities. There was a normal complement of gas in the large bowel and stomach, with no evidence of intestinal obstruction or air fluid levels. Initial blood count showed a hemoglobin level of 9.7 gm per cent, a red blood cell count of 3.42 million/cu mm and a hematocrit value of 32 per cent. On smear the red blood cells showed polychromasia and basophilic stippling. The white blood cell count was 9,900/cu mm, with a normal differential except for 9 per cent stab forms. Urinalysis was normal except for the presence of ketones. The patient denied any knowledge

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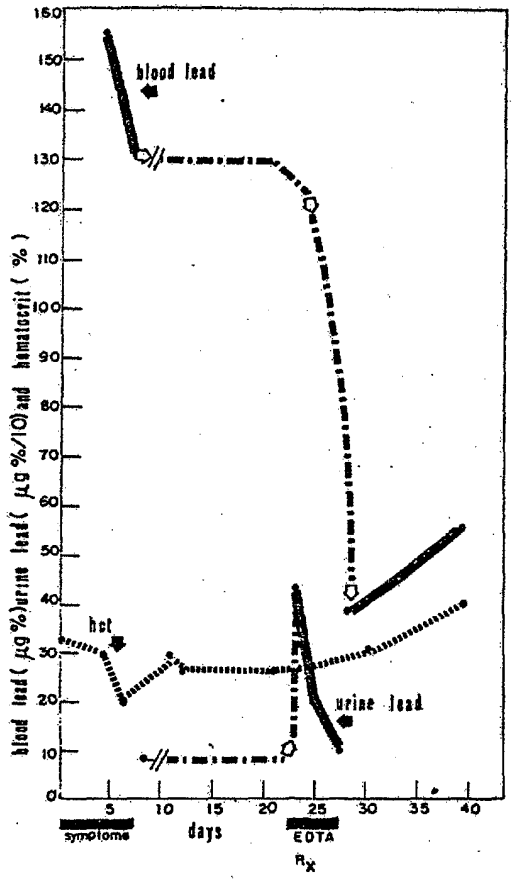

Figure 1. Clinical measurements by day of illness. Note: The dotted lines in the lead curves are not based on data, and merely indicate general direction.

of contact with lead so that this diagnosis was temporarily dismissed. She was treated symptomatically, but the symptoms persisted. Further investigations on the fifth day of observation showed a persistent anemia with a hemoglobin level of 9.4 gm per cent. The reticulocyte count was 9.6 per cent. Serum total bilirubin was 2.1 mg per cent with 1.1 mg per cent direct and 1.0 mg per cent indirect reacting. An oral cholecystogram was negative. A blood sample drawn on that day was later reported as showing a lead level of 156 µg/100 ml (normal up to 60 µg/100 ml). The patient was admitted to the hospital for further substantiation of the diagnosis and was later treated with EDTA.

The blood lead level on February 5, 1970 (eighth day of observation) was 131 µg/100 ml. In a twenty-four hour urine specimen obtained on the same day the lead content was 86 µg/twenty-four hours (normal up to 100 µg/twenty-four hours). Serum iron was 126 µg per cent, total iron-binding capacity 264 µg per cent; percentage iron saturation was 48 per cent. Qualitative tests for urinary coproporphyrins were positive, urinary uroporphyrins were negative. Urinary porphobilinogen test was negative. Direct and indirect Coombs' tests were negative.

Treatment was started with 1 gm of EDTA given intravenously twice daily for five days beginning on February 20, 1970. The results of therapy are shown in Figure 1. Urinary excretion of lead was 420 µg per cent the first twenty-four hours, 270 µg per cent the second, 200 µg per cent the third and 100 µg per cent during the fifth twenty-four hour period. Considering the respective urine volumes, these values are equivalent to total lead excretions of 11.8 mg/twenty-four hours the first day after treatment, 7.1 mg the second, 4.4 mg the third, and 2.8 mg the fifth day. The patient's condition improved, and she was discharged from the hospital on February 25, 1970.

By February 26, 1970, the blood lead level had returned to a normal value of 38 µg/100 ml. The hemoglobin returned to a normal value of 12.5 gm per cent by March 9, 1970. On this date the reticulocyte count was 4.6 per cent. The hemoglobin level rose further to 15.8 gm per cent by May 6, 1970; reticulocyte count at that time was 1 per cent, and the blood lead level was 62 µg/100 ml. The patient has since felt completely well and has resumed her normal activities.

EPIDEMIOLOGY

The wife was not the only member of the family to be affected. After her initial blood analysis confirmed the diagnosis of lead poisoning, considerable anxiety was aroused with respect to environmental exposure, and other family members were examined. Blood samples obtained on day 12 (Figure 1) contained the following lead levels: husband, age forty, 88 µg per cent; son, age fourteen, 25 µg per cent; son, age five, 25 µg per cent; daughter, age four, 25 µg per cent. It appeared that the husband also had had unusual exposure to lead, but the three children had not. The older child attended school, but the younger children

Figure 2. Lead-bearing cocktail glass.

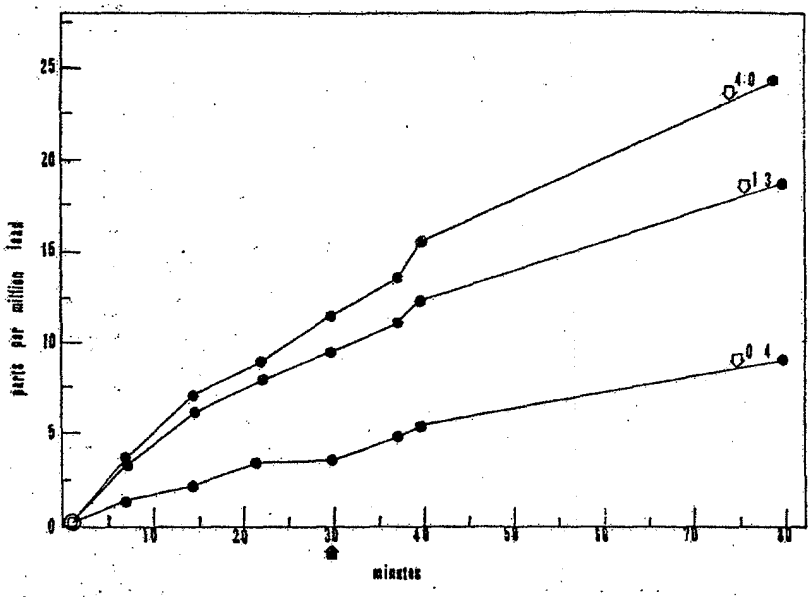

Figure 3. Lead concentration (ppm) by time in three different cocktails (7-Up : water = 4:0, 1:3 and 0:4).

were constantly at home with the wife. The husband had not felt ill recently.

Search of the home produced no suspicious materials, and the water supply was tested and found to be normal. Enquiry into possible domestic and recreational exposures consistent with the epidemiologic pattern revealed only a single likely source: a set of cocktail glasses decorated with antique American cars and frosted white on the inside. The glasses (Figure 2) had been purchased in an Illinois department store about 1953 and had been used only occasionally until about six months prior to the wife's illness, when use became more frequent and automatic machine dishwashing was begun in place of hand washing. During this six months the white interior coating was noticed to be wearing off, but the glasses were used nevertheless, about "half the time" together with other plain glasses.

The habits of alcohol consumption were investigated next. It had long been customary for both husband and wife to consume about five drinks each evening before supper over a three hour period. The husband's drink was always whiskey and water "slightly sweetened" with 7-Up, the proportions of whiskey:7-Up:water thus probably being somewhere between 1:0:4 and 1:1:3. The wife preferred her drink mixed entirely with 7-Up, the proportions of whiskey:7-Up:water for her thus being 1:4:0. The suspect glasses had been used until six days before the wife's admission to the hospital for EDTA therapy (i.e., day 14 on Figure 1).

TOXICOLOGY

The deteriorated white coating on the inner surface of the glasses was found to be made of a lead compound, probably lead oxide since no carbon dioxide was evolved upon treatment with dilute nitric acid. Drinks of the mixtures described were prepared in comparable glasses, each drink containing the same amount of whiskey (1 part or 30 ml) and ice (melted volume of 5 parts, or 150 ml), making a total volume of 300 ml. The concentration of lead at intervals up to eighty minutes was determined for each drink using the method of atomic absorption spectrophotometry.

The results (Figure 3) were as follows (ratios refer to whiskey:7-Up:water:ice; the series of decimal numbers refers to times, in minutes, viz., 1.5, 7.5, 15.0, 22.5, 30.0, 37.5, 40.0 and 80.0 minutes): for control drinks mixed 1:4:0:5 and 1:2:2:5 in lead-free control beakers, no lead was detected by 80 minutes; for drink 1:4:0:5 in cocktail glass, concentrations in μg per cent were 0, 3.7, 7.2, 8.9, 11.5, 13.6, 15.6 and 24.4; for drink 1:1:3:5 in cocktail glass, concentrations in μg per cent were 0, 3.3, 6.2, 7.9, 9.5, 11.1, 12.3 and 18.8; for drink 1:0:4:5 in cocktail glass concentrations in μg per cent were 0, 1.4, 2.2, 3.4, 3.4, 4.8, 5.3 and 9.0.

It is apparent that the wife's drink (1:4:0:5), which was more acidic due to the citric acid and carbonic acid in 7-Up, was the better solvent for lead and yielded higher concentrations of lead at thirty minutes, the approximate average consumption time per drink. Selecting the mixture 1:1:3:5

for the husband's drink and 1:4:0:5 for the wife's, an evening's five drink dose of lead would thus be 14.2 and 17.3 mg for husband and wife, respectively. It should be noted that the husband's value is probably overestimated, since his curve probably lies somewhere between the 1:3 and 0:4 curves of Figure 3; and the experiment is based upon the dissolution of a uniform lead-bearing surface area over time, somewhat inflating the estimates in both instances, since the area would diminish with consumption.

If the lead-containing glasses were randomly selected for use about half the time over the six month period, the total dose of lead for the husband (on the 1:3 assumption) would amount to approximately 1.28 gm, whereas for the wife it would be approximately 1.56 gm. If the husband's mix had been purely water (mix 0:4), his total dose would have been only about 0.46 gm.

COMMENTS

Several features of this occurrence of lead poisoning deserve brief comment. It has been reported that the glaze of a small mug may contain up to 20 or 30 gm of lead [4]. It is likely that these commercially manufactured cocktail glasses contained amounts of similar magnitude, since the wife's total dose was estimated at over 1 gm, most of the white frosting was still intact, and some of the frosting removal probably occurred in the dishwashing process.

The role of machine dishwashing here should itself be emphasized. Although the lead glaze may have been insoluble enough in ordinary dishwashing, the temperature and chemical rigors of machine dishwashing were apparently sufficient to start the process of dissolution.

The severity of the wife's exposure in this report should be pointed out. Assuming that the wife used lead-glazed glasses about half the time, her average daily dose would have been about 8.6 mg. In Kehoe's experiment [10], which administered 3.0 mg of lead daily to a human male subject, premature termination of the study at the end of four months was necessary because the blood lead level had reached 50 μg per cent. He states [10] that "daily intake of 3.27 mg (3.00 administered, 0.27 in food and beverages) of lead per day by an experimental subject would be expected to reach the point of danger [80 μg per cent] in approximately eight months." In this instance, about 8.6 mg of lead/day produced a blood lead level of 160 μg per cent in six months.

Attention should also be called to the fact that although this severe exposure produced a very high blood lead level, the urinary lead concentration before treatment was within the normal range. Normal urinary lead output in cases of acute lead encephalopathy in children has been described [11]. The instance reported here appears to be an example of a misleading urinary lead result in an adult. The urinary excretion of lead after treatment was very high at 11.8 mg the first twenty-four hours, considering that any value in excess of 1.5 mg the first twenty-four hours of chelation therapy is said to be diagnostic of plumbism [11].

Finally, the reasons for the disparity in the blood lead level between husband and wife should briefly be considered. Depending upon assumptions with respect to the proportions of his mix, the husband's average daily dose could have been as little as 2.5 mg or as much as 7.1 mg. The lower part of this range would be quite consistent with Kehoe's experiment [10], in which 3 mg daily produced a blood lead level of 50 μg per cent in four months. The husband's blood lead level reached 88 μg per cent in six months. If, however, the husband's mix approached a 7-Up:water ratio of 1:4, the disparity in blood lead levels between husband and wife might be explained by inaccuracies in alcohol consumption history, or a possible sex difference in lead absorption and/or metabolism. Since Kehoe's subjects [10] were all male, this latter possibility remains to be explored.

REFERENCES

1. Harris RW, Elsea WR: Ceramic glaze as a source of lead poisoning. JAMA 202: 544, 1967.
2. Leonard AR, Lynch G: Dishware as a possible source of lead poisoning. Calif Med 89: 414, 1958.
3. Meiklejohn A: The successful prevention of lead poisoning in the glazing of earthenware in the North Staffordshire Potteries. Brit J Industr Med 20: 169, 1963.
4. Beritic T, Stahuljak D: Lead poisoning from lead glazed pottery. Lancet 1: 669, 1961.
5. Jensen R, Jensen H: Risks of saturnism in decoration on glass. Arch Mal Prof 18: 673, 1957.
6. Primakov SV: Clinical aspects of acute poisoning with glazing material. Klin Med 42: 128, 1964.
7. Beritic P, Djuric D: Lead poisoning caused by pottery glazing. Higijena 8: 12, 1956.
8. Fromke VL, Lee MK, Watson CJ: Porphyrin metabolism during versenate therapy in lead poisoning. Ann Intern Med 70: 1007, 1969.
9. Berk PD, Tschudy DP, Shepley LA, Waggoner JG, Berlin NI: Hematologic and biochemical studies in a case of lead poisoning. Amer J Med 48: 137, 1970.
10. Kehoe RA: Metabolism of lead under abnormal conditions. Arch Environ Health (Chicago) 8: 235, 1964.
11. Chisolm JJ: Treatment of lead poisoning. Mod Treat 4: 710, 1967.

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