EARTHENWARE CONTAINERS AS A SOURCE OF FATAL LEAD POISONING*

Case Study and Public-Health Considerations

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Abstract Two young children suffered lead poisoning as a result of drinking juice stored in a modern handmade earthenware jug. One of the children died. Subsequent testing of 264 contemporary earthenware glaze surfaces revealed that 50 per cent released sufficient lead to make them unsafe for culinary use. Between 10 and 25 per cent of the pieces tested would have been capable of causing severe lead poisoning. Compounding of safe earthenware glazes is essential.

LEAD poisoning in children is well known. It is usually associated with pica under conditions of social deprivation found in large tenement dwellings constructed before World War II, when leaded paints were routinely used. Lead poisoning in Canada is rare and is almost unheard of in the children of middle-class suburbs. The cases that follow are unusual, but they resulted in the identification of a serious public-health hazard.

CASE REPORTS

CASE 1. J.L., a 2-year-old boy, was admitted to the Montreal Children's Hospital in a comatose state. Respiratory arrest occurred in the Emergency Room, and assisted ventilation was instituted. Three weeks previously, afebrile nausea and vomiting had begun. A physician diagnosed gastritis and advised rest and forced fluids. There was no improvement, and 2 days before admission to a suburban hospital, the parents noted lethargy, anorexia and a marked increase in irritability. On admission anemia and basophilic stippling of erythrocytes were noted. The diagnosis of lead poisoning was considered, but no history of pica was obtained. Growth and development had been entirely normal, and there was no previous history of serious illness. On the next day, the patient had a grand-mal seizure followed by deep coma; he was transferred to the Montreal Children's Hospital. On examination, he was in deep coma and areflexic; the pupils were fixed and dilated, and corneal reflexes were absent. The liver edge was palpable 6 cm below the right costal margin. Laboratory findings included a hemoglobin of 7 g per 100 ml and a white-cell count of 8100, with a normal differential. A peripheral blood smear revealed anisocytosis, poikilocytosis and marked basophilic stippling. On urinalysis the sediment contained coarse granular casts, 35 to 40 red cells per high-power field and moderate glucose by glucose-oxidase paper; screening for coproporphyrins was positive, and paper chromatography demonstrated generalized aminoaciduria. Examination of the spinal fluid showed 1000 red cells and 10 lymphocytes per cubic millimeter and a protein of 435 mg and glucose of 121 mg per 100 ml. Serum electrolytes, blood urea nitrogen and creatinine were normal. The serum glutamic oxalacetic transaminase was 92 U, and the serum glutamic pyruvic transaminase 117 U. The urinary lead level was 5 mg per liter on admission and 138 mg per liter† on therapy (normal, 0.02 to 0.08 mg per liter). The diagnosis of lead poisoning was established within an hour of admission, and therapy with intramuscular BAL (8 mg per kilogram of body weight every 8 hours) and intravenous calcium disodium EDTA (16.5 mg per kilogram every 8 hours) was instituted in an alternating fashion according to the method of Chisolm.[1] The course continued unremittingly downhill despite chelation therapy and the use of cerebral dehydrating agents. The patient died on the 3d hospital day while still on the respirator. Electroencephalograms for the 2 days before death were isoelectric. Autopsy revealed severe brain swelling, with herniation of the cerebellar tonsils and uncinate portions of the temporal lobes and ischemic necrosis of the pituitary gland.‡ Tissue levels of lead are shown in Table 1.[2]

Table 1. Tissue Lead Levels.

TISSUE	LEVEL OF PATIENT	NORMAL LEVEL[2]
	mg/100 ml	
Liver	1.33	0.05 - 0.94
Kidney	1.82	0.02 - 0.08
Spleen	1.13	0.01 - 0.07
Brain:		0.01 - 0.10
Basal ganglions	0.196	
Cortical gray matter	0.218	
Cortical white matter	0.037	

Careful questioning of the parents disclosed that during the 4 weeks before admission, the patient had been drinking large quantities of apple juice that had been stored in a hand-crafted earthenware jug (Fig. 1). With the onset of the symptoms, the patient's juice consumption was further increased, on medical advice. Subsequent testing revealed that apple juice stored in the jug for 3 hours contained 157 mg per liter (ppm) of lead. Juice stored for 3 days contained 1300 mg per liter (ppm) of lead.§

CASE 2. C.L., the four-year-old brother of J.L., was admitted to the Montreal Children's Hospital on the same day. He had been drinking apple juice from the same container. He also had a 3-week history of intermittent afebrile nausea and vomiting, but at the time of admission he was asymptomatic. On examination he appeared well. The only positive finding was a liver edge palpable 4 cm below the right costal margin. The hemoglobin was 8.5 g per 100 ml, the hematocrit 26.5 per cent, and the white-cell count 8600, with a normal differential. Anisocytosis, poikilocytosis and basophilic stippling were seen on the peripheral blood smear. Urinalysis revealed only occasional fine granular casts in the sediment. Urine screening for coproporphyrins was positive, and generalized aminoaciduria was present. The serum glutamic oxalacetic and pyruvic transaminases were both 110 U. Blood urea nitrogen, creatinine and serum electrolytes were normal. The urinary lead level was 62.6 mg per liter. Examination of spinal fluid gave normal results. Electroencephalography showed only slight abnormality.

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†Twenty-hour sample collected in a lead-free plastic container.

‡Pathology courtesy of Dr. F. W. Wigglesworth.

§Lead determinations in urine and apple juice were performed by atomic absorption.

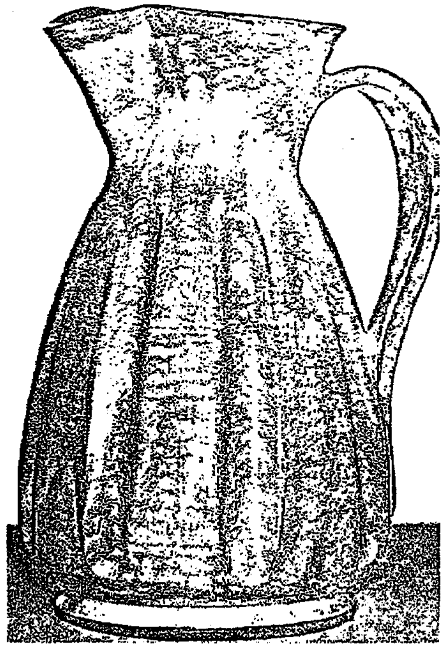

Figure 1. Hand-Crafted Pitcher Used for the Storage of Apple Juice.

Therapy with BAL and calcium disodium EDTA was instituted according to the same schedule used in Case 1. The patient was asymptomatic throughout and was discharged on the 5th hospital day.

The whole-blood lead level (normal less than 40 μg per 100 ml) at the end of the 5-day course of BAL and calcium disodium EDTA was 8 μg per 100 ml.* Three days after this therapy was completed the blood lead level had rebounded to 75 μg per 100 ml. At this point d-penicillamine therapy was started (100 mg per kilogram for 5 days and 40 mg per kilogram for 60 days). Blood lead levels were 47 and 27 μg per 100 ml 1 and 2 months after the institution of this therapy, respectively. The patient has remained well without treatment for 5 months.

Epidemiology

The solubility of lead glazes on earthenware designed for household use has long been a matter of great importance, and in some countries is regulated by legislation.[3] At present in Canada, no governmental standards exist setting the levels of safety for ceramics intended for culinary use. The United States Potter's Association and the United States Food and Drug Administration have recently defined 7 ppm as the maximum lead release of glazes recommended for use on ceramic items intended for food and drink.[4] The high extractable lead content of the jug implicated in the two cases resulted in the following study by one of us (R.N.).

Samples

One hundred and seventeen samples of earthenware pottery of unknown glaze composition, both domestic and imported, handcrafted and commercial, were obtained from handicraft shops and department stores.

One hundred and forty-seven samples were prepared in the ceramics laboratory, with the use of 49 different glaze compositions in use by various schools, clubs, studios and potters. These were fired at 1750, 1850 and 2050°F on containers of identical size and clay composition. These are the three temperatures at which most hand-craftsmen fire their earthenwares.

Procedure

The testing procedure was standardized with that outlined by the Department of Consumer and Corporate Affairs, Standards Branch, Ottawa, Ontario, and was as follows: pottery samples were washed with dilute alkaline-detergent solution, rinsed with distilled water; samples were filled to the top with 4 per cent acetic acid solution, and the volume recorded; samples were covered with plastic film and allowed to stand at room temperature for 18 hours; and lead was determined by atomic absorption technic,† reported as metallic lead in micrograms per milliliter (ppm).

The report of lead extractions from 264 earthenware glaze surfaces is shown in Table 2.

Discussion

The fact that lead-glazed pottery could result in poisoning was known in antiquity and has been periodically rediscovered as certain cultural changes allowed lead glazes to be produced improperly or used under inappropriate conditions. The Greeks used earthenware vessels for cooking, but under carefully controlled conditions.[5] In the second and first century B.C. Greek cookery was introduced into the aristocratic class of Roman civilization, but the taboos of the Greeks were lost and acidic solutions were stored in earthenware. Wine was stored in this way, and relaxations on the drinking of wine by aristocratic women may have resulted in chronic lead poisoning and sterility. It is believed that this in turn substantially reduced the size of the next generation and may very well have contributed to the rapid extinction of the aristocratic class. From this reasoning, lead poisoning has been alleged to have contributed to the fall of the Roman Empire.[5]

In 1723 the Massachusetts Bay Colony forbade rum distillation from leaded stills, a practice that was known to result in a condition of abdominal

*Blood lead determinations were carried out by the Baltimore Health Department laboratory with the use of a dithizone method.

†Perkin-Elmer Model 303 Atomic Absorption Spectrophotometer with the following operating conditions: wavelength 283, range u.v., slit 4 (1 mm, 7A); source, lead hollow cathode 8-10 ma; fuel flow (acetylene) 9.0, and airflow 9.0.

Table 2. Report of Lead Extractions from 264 Earthenware Glaze Surfaces.

Sample Group*	No. of Glaze Surfaces	Lead Extraction						
		<0.5 PPM	0.5-7 PPM	7-20 PPM	20-100 PPM	>100 PPM	100-500 PPM	500-1000 PPM
A								
Imported pottery	29	9	5	6	6	3	–	–
Domestic commercial ware	48	17	7	4	15	5	–	–
Domestic handcraft	40	10	4	4	6	16	–	–
Totals	117	36	16	14	27	24	–	–
B	147	36	36	32	14	–	10	19

discomfort called "dry gripes."[6] In 1754, the year after his treatise on scurvy was published, John Lind warned that lemon or wine juices should not be stored in earthenware jugs.[7] In 1767 Sir George Baker read his "Essay on the Cause of the Endemic Colic of Devonshire" at the College of Physicians and Surgeons of London, in which he blamed the use of lead-lined troughs in the production of apple cider, a discovery for which in 1772 he was denounced from the pulpit as a faithless son of Devon.[7]

In 1958, some Japanese tableware was found to be unsafe.[8] Two years later, in Britain, lead poisoning resulted from the drinking of homemade wine stored in earthenware.[9] In 1961 Yugoslavian authors described 40 patients poisoned in six years by earthenware pots used for pickling and wine making.[10] In 1967 a case similar to ours was reported in a physician who had ingested 3.2 mg of lead per night for two years by drinking a cola from an earthenware mug made by his son in a university ceramics class.[11] Two years later Mexican pottery was implicated in serious poisoning in the five members of a physician's family, and the suggestion was made that such pottery might be a source of serious disease and disability in Mexican peasants.[12]

Within the past 15 years there has been an impressive revival of interest in handcrafts in North America. In the field of ceramics, this has resulted in an increasing number of amateur and professional craftsmen and small pottery industries. The pursuit of pottery for recreation or as an artistic expression demands little knowledge of chemistry. Most of the textbooks and periodicals available for the amateur are nontechnical. Glaze "recipes" are obtained from the literature, and commercial glazes – unlabeled for chemical composition – are readily available. A basic understanding of the chemical and physical interactions of the compounds composing a glaze is essential.

Earthenware

Most of the world's pottery has been earthenware because of the abundance of these clay deposits and the relative ease of reaching the temperatures needed for their manufacture. The term "earthenware" is correctly used to name pottery that has been fired between 1085 and 2174°F, either glazed or unglazed.

Ceramic Glazes

A glaze is a thin layer of glass fused onto the surface of clay wares. The basic glass former in ceramic glazes is silica, an element that combines freely with other oxides to form a variety of complex silicates. Silica has a very high fusion point and is therefore used in combination with oxides with low melting points. This mixture when fired forms a glass or glaze suitable for earthenware clays.

Lead is a common constituent of earthenware glazes. In the past lead has often been used as the sole glass former or glaze on low-fired wares. Despite the toxicity of lead compounds, they are invaluable to the potter, for they impart characteristics to a glaze unequaled by other oxides.

Earthenware – Glazes of Low Solubility

In compounding lead-bearing glazes of low solubility the following principles apply:

Lead compounds such as lead oxide, lead carbonate and lead monosilicate are highly soluble, even when fused. The lead bisilicate and lead trisilicate have comparatively low solubility. In all cases the ratio of silica to lead should not be less than 2:1 (2 parts silicon dioxide to 1 lead monoxide) in molecular equivalents.

Alumina (aluminum oxide) is effective in decreasing the solubility of lead silicate mixtures, and is always used.

Alkalies, particularly boric oxide, increase the solubility of lead silicate mixtures. This effect must be counteracted by large additional increases of alumina and silica. The alkaline earths (calcium oxide and barium oxide) are also instrumental in decreasing lead solubility.

Lead-bearing glazes, properly compounded to yield low lead release, may be profoundly affected by the addition of various oxides. Introduced for color and textures, these oxides may sufficiently alter the final fired glaze composition to render it soluble.

Glaze compositions are designed for specific temperatures and clays.

Because of the toxic nature of lead, all pottery designed for table use must have a glaze that does not dissolve at all. Absorption and inhalation of lead compounds used in the workshop present an additional hazard. Lead can be converted into compounds that are nontoxic to the workers handling them by a process called fritting.

Lead Frits

A *frit* is a glassy material made by fusion of various glaze components. A frit is designed for use as a constituent of glaze, although entire glazes may be fritted. There is a common misconception that commercial frits are "safe," and they are often used as the complete glaze. Frit compositions vary extensively, and many lead-bearing frits are highly soluble. The use of frits of low solubility, alone or in combination with other compounds or frits, does not guarantee a fired glaze of low solubility. Unfortunately, commercial frits are not labeled for chemical composition, nor are they classified for solubility.

For example, the glaze on the earthenware jug referred to in the case histories was a lead-bearing frit. This frit is highly soluble and difficult to use safely even as a minor glaze constituent.* The craftsman had repeatedly used this frit as the glaze, believing all frits were "safe" and purchasing it by catalogue number from a well known North American chemical company.

Conclusions

There is a considerable health hazard due to lead release from earthenware glaze on handcrafted, commercial domestic wares and some imported pottery. Fifty per cent of all glaze surfaces tested were unsafe for table use (more than 7 ppm). Twenty-five per cent of all domestic handcraft, and 10 per cent of imported and commercial earthenware released over 100 ppm of lead.

Lead release in the range of 100 ppm would be expected to result in severe acute poisoning in small children under the conditions reported in the two cases presented above. Continuous use of containers releasing lead in the range of 7 to 20 ppm could give rise to chronic lead poisoning. In that event, the symptoms would be more insidious, and diagnosis difficult because of their similarity to common functional complaints.

Though the reported frequency of lead poisoning from pottery has been low, the true figure may be considerably higher. Fortunately, relatively few earthenware containers are used for storage of acidic solutions, and many are used only for decorative purposes. Public demand for hand-made pottery is leading to increased production and availability. Unless this is matched by an increased awareness of the problem by potters and governments, one can expect to see an increase in lead poisoning from this source.

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*$\left.\begin{matrix}0.7\ PbO \quad 0.4\ B_2O_3 \quad 0.41\ SiO_2 \\ 0.3\ CaO \end{matrix}\right\}$ in molecular equivalents.