



DEPARTMENT OF HUMAN SERVICES
QUEEN ELIZABETH HOSPITAL

Lead Poisoning from Homemade Wine: A Case Study

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A 66-year-old man suffered the symptoms of severe lead poisoning for 2 years before diagnosis. The man had a blood lead level (PbB) on admission to hospital of 98 µg/dL. A detailed investigation revealed that the poisoning occurred as a result of drinking a homemade red wine, for which analyses showed a lead concentration up to 14 mg/L—70 times the Australian maximum limit for lead in wine. The source of the lead was a highly corroded enamel bathtub in which grape crushings and juice were stored for a week prior to bottling. The corrosion of the enamel surface of the bathtub had resulted in pitted patches up to 1 mm in depth along the side of the bathtub. Powdering of the tub surface was evident below a level where wine had been in contact with the sides of the tub. The homemade wine had a pH of 3.8, which would have greatly contributed to the solubilization of metals from the glaze. We conducted a test in which commercial red wine of similar pH and containing < 0.2 mg/L lead was placed in this tub for 7 days. Subsequent testing revealed a lead level of 310 mg/L. This high lead concentration is consistent with the surface area of enamel on the bathtub being in contact with a small liquid volume as in the case of the leaching test using commercial red wine. This case study highlights the importance of the use of food-grade materials for the preparation and storage of homemade beverages or food. **Key words:** bathtub enamel, food processing, lead, poisoning, wine. *Environ Health Perspect* 109:433–435 (2001). [Online 30 March 2001]

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In South Australia, hospitals and pathology laboratories usually notify the Department of Human Services of any cases of elevated blood lead (PbB) levels. In June 1999 the medical registrar of a public hospital in Adelaide reported a PbB level of 98 µg/dL in a 66-year-old man; this is approximately 10 times the National Health and Medical Research Council (NHMRC) National Goal for all Australians of 10 µg/dL or 0.48 µmol/L (1).

In this paper we outline the medical history of this patient together with the details of the investigation conducted to ascertain the source of the lead exposure.

Materials and Methods

Medical case history. The patient was a diabetic and had been relatively fit and healthy until 1997 when he presented to a number of hospitals on several occasions complaining of abdominal pain. This was extensively investigated with abdominal computed tomography (CT) scans, ultrasounds, endoscopies, colonoscopies, and barium enemas with no positive results. Some tests were repeated several times over a 2-year period, and the patient had been treated for constipation several times. Family members reported that the man's weight had

decreased from 95 to 75 kg over the 2 years before hospitalization in July 1999.

Over the same period the man claimed to have started feeling "different," and his family noted a significant change in his personality and behavior. Although he had previously been relatively healthy and employed, he was no longer able to work. On previous admissions he was noted to be irritable, aggressive, and paranoid. The man also had severe short-term memory impairment, a waxing and waning consciousness, and felt drowsy and intermittently confused.

Despite being only 66 years old, the patient had both physically and mentally deteriorated to the level of not being able to manage at home alone, and at the time of diagnosis he was being considered for nursing home placement.

Arthralgia that responded to nonsteroidal anti-inflammatory medications was also reported in the patient. He appeared to have a peripheral sensory neuropathy that primarily affected his lower limbs, with some decrease in proprioception. From time to time he complained of paresthesia in his feet. During his most recent admission, he had complained of severe headaches. The patient's gait was at times unsteady, and

there was objective proximal muscle weakness in the lower limbs. His most consistent complaint over the 2 years was abdominal pain, aggravated by constipation. The pain was reported as being generalized and "squeezing" and could not be relieved. He also had great difficulty retaining his food, particularly meat.

A routine blood examination on admission revealed severe microcytic anemia. He was not iron deficient and had a normal hemoglobin electrophoresis. A subsequent blood film revealed basophilic stippling, a classic sign of lead poisoning. Other screens (dementia, rheumatologic, and vasculitic) were all negative. The CT scan of the head was not abnormal. On the basis of the above clinical features, the patient was then investigated for heavy metal poisoning.

The patient had been hospitalized for 2 weeks before the diagnosis of lead poisoning, presumably without access to sources of lead exposure while in the hospital. Case notes acquired from other institutions late in the investigation recorded elevated PbB results that had not been followed up.

Previous PbB levels were 52 µg/dL at 7 months before admission (November 1998) and 85 µg/dL at 4 months before admission (February 1999). The initial PbB level 2 weeks after admission (June 1999) was 98 µg/dL; at the time of admission to hospital, the patient probably had a peak PbB level much greater than 98 µg/dL. A week after the test that identified lead poisoning (June 1999) and before chelation was started, his PbB level was 83 µg/dL.

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The patient's zinc protoporphyrin level was 229 $\mu\text{g}/\text{dL}$ (normally $< 40 \mu\text{g}/\text{dL}$). His 24-hr urinary lead level was 1.33 $\mu\text{mol}/24\text{hr}$ [Worksafe occupational standard $< 1.09 \mu\text{mol}/24 \text{ hr}$ (2)]. The PbB level was clearly significantly elevated, and chelation treatment was started after consultation with toxicologists. The agent chosen was oral succimer (dimercaptosuccinic acid) because the patient was not severely encephalopathic. This was given over 18 days. The patient's PbB level had declined to 34 $\mu\text{g}/\text{dL}$ in July 1999 after chelation was completed (Figure 1).

Four months after chelation therapy the patient had recovered his mobility; he was no longer suffering abdominal pain and constipation and could retain his food. Symptoms associated with diabetes relating particularly to the patient's vision and peripheral circulation were present.

The patient subsequently recovered. Most of his symptoms resolved; he had better memory and was less confused. He also felt better and was able to return home and care for himself. His PbB level taken 8 months after he was discharged from the hospital was 31 $\mu\text{g}/\text{dL}$ in March 2000 (Figure 1).

Environmental investigation. The patient's PbB level was sufficiently elevated to suggest a very potent lead source. Strategies for discovering and assessing lead exposure sources for individuals are described by van Alphen (3). Data were acquired to evaluate occupation, hobbies, food items, nonfood items, the patient's home, networks of acquaintances, and past employment. A visit to his home with a family member revealed no indications of lead exposure via paint renovation, occupational exposure, or craft-based hobbies, and the residence was not in close proximity to any lead-associated industries. The patient had previously worked at a tool-making factory and had been involved in general cleaning and maintenance of the machinery. This work involved cleaning the machinery with kerosene and lubricants. A site investigation of the factory was conducted, but no indication of a lead source was apparent. A wipe sample was taken from various items of machinery that the individual had been involved in cleaning.

To evaluate other possible lead sources, we tested the following samples: mains-supply tapwater and bottled springwater, a pewter cup (test for lead leachate), house-dust wipe, imported canned foods (potential for lead soldering contamination), and homemade foods items such as bottled salsa (tomato sauce) and homemade red wine. We also inspected the location where the wine was made.

Sample collection. We collected samples of wine from the 1996 and 1999 vintages from the patient's house. All samples were

sent to the Australian Government Analytical Laboratories (Melbourne, Australia) for lead analysis.

The lead-poisoned patient was involved in wine making with a friend (Mr. B). We initially inspected a grape-crusher and basket-press, but no lead sources were readily apparent. The men stored the crushed grapes in enamel bathtubs for 1 week before initial bottling. The patient used one bathtub for his grapes (bathtub A) and Mr. B used another (bathtub B). After a week, the grape solids were transferred to a basket press, and juice was extracted, transferred into glass flagons, and fermented into wine.

The bathtub used by the patient (bathtub A) was observed to be weathered, the enamel lining was etched and/or corroded, and the surface below the "wine level" readily powdered when wiped with a finger. The other bath surface (bathtub B) was intact with no areas of enamel corrosion.

To test whether lead could be leached from the two bathtubs, we placed 4 L of commercially available cask red wine into each tub. After 7 days (the time normally used by the patient to pre-ferment the crushed grapes), we collected lead leachate samples and measured pH and lead levels.

Results

A variety of samples including water, food, house and factory dust wipes, and a leach-tested drinking cup were shown not to contain significant amounts of lead; however, wine samples from the patient's 1999 batch of wine did contain significant amounts of lead. Lead levels from the leach-test wine sample from bathtub A was also found to be significantly elevated compared to bathtub B (Table 1). Thus, lead was leaching out more readily from the bathtub with the etched and powdering enamel lining. On the basis of the surface area covered by the commercial wine, it is evident that much more lead entered the wine than could be predicted from the surface-wipe analysis. Thus significant leaching

from within the etched bathtub surface had probably taken place.

The Australian maximum limit for lead in wine is 0.2 mg/L (4); the lead levels in the wine samples tested in this case study significantly exceeded this limit. Two samples from the patient's 1999 batch of wine exceeded the lead standard in wine by a factor of 70 (14 mg/L) and 55 (11 mg/L), respectively. The patient's 1996 batch also exceeded the standard by a factor of 4.5 (0.9 mg/L). A 1999 wine sample from Mr. B exceeded the standard by 2.5 times (0.5 mg/L); Mr. B had a PbB level of 11.6 $\mu\text{g}/\text{dL}$, slightly elevated compared to the NHMRC National Goal for all Australians of 10 $\mu\text{g}/\text{dL}$.

The dust wipe from bathtub A demonstrated the presence of lead. The dust wipe result showed that 3.2 mg lead was easily removed from the 400-cm² surface area sampled (Table 1). Assuming that there was a uniform distribution of lead on the 3-m² bathtub surface, we estimate that at least 240 mg of weakly adherent or powdered lead was available on the surface for dissolving into the next batch of wine.

The chips of enamel from bathtub A were rinsed with 5% nitric acid at room temperature for 20 sec, which removed the equivalent of 1,500 mg Pb/m². These results cannot quantitatively account for the rate of addition of lead to wine in contact with the bathtub, but they indicate that the powdered surface of the bathtub was a source of large quantities of lead.

Discussion

The results from this case study demonstrate that the homemade wine of both our patient and Mr. B had been contaminated with lead and that the most likely source was the bathtubs used to store the grape crushings. The dust-wipe and wine-leach tests confirmed the suspicion that the extremely high levels of leached lead in the patient's wine were associated with the extensive etching in bathtub A.

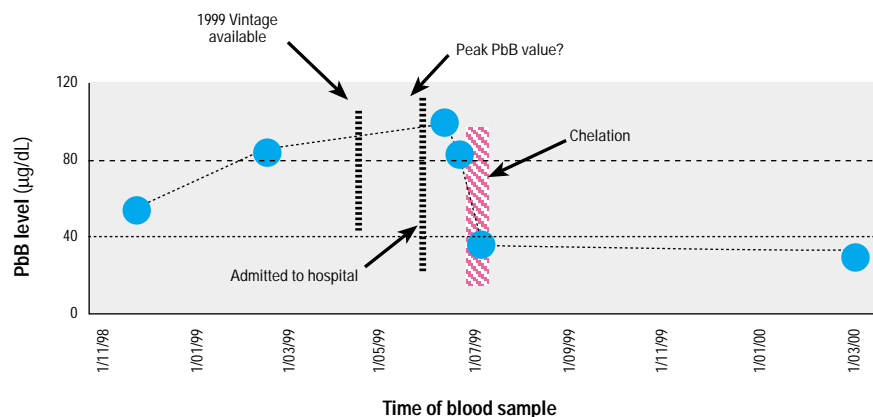


Figure 1. PbB levels of the patient sampled before and after hospital admission.

Both bathtubs were subsequently disposed of in a licensed waste dump. The men now use a stainless steel container to crush and pre-ferment the grapes. The lead level of their most recent batch of wine (year 2000) is <0.2 mg/L.

The patient had substantially depleted his supply of homemade wine in early 1999 and probably had a relatively low lead intake for the first 3–4 months of 1999. For 1–2 months before hospitalization, it is possible that the patient had an intake of lead that was at a higher rate than in previous years and substantially higher than in previous months. This would be consistent with his feeling well again before consuming the 1999 vintage wine. The rapid reduction in the PbB of the patient after chelation may imply that the lead exposure responsible for the PbB elevation to 98 µg/dL was of a high magnitude for a short time before hospitalization. Because the PbB level did not go below 31 µg/dl after 8 months, there is some indication of a longer-term component of exposure that resulted in the storage of substantial lead in bone.

The poisoning and hospitalization of the patient may have also been associated with exposure to high levels of tin and antimony, which were identified late in the investigation as being major components of the enamel in bathtub A; however, we did not evaluate the patient for exposure to these metals.

Grapes are a minor contributor to the low lead content of Australian wine (7). In

the literature there have been a number of incidents of lead contamination of wine, mostly reported from Italy. Contamination of wine with lead may occur in several ways, for example, from lead solder used to repair wine casks (8). In one study it was found that lead poisoning was caused by wine passing through lead-containing outlet tubes of storage casks (9). There have also been cases of lead poisoning from homemade wine where storage and fermentation vats contained lead (10,11). The storage of port and alcohol products in lead crystal decanters is another source of lead contamination (12), as is chewing of the lead foil closures on the neck of wine bottles (13). In Australia, the use of lead foil on wine bottle closures was eliminated in the early 1990s.

The provisional tolerable weekly intake (PTWI) for lead in adults is 25 µg/kg body weight or 250 µg/day for an adult male who weighs 70 kg (14). The average daily intake of lead in Australia is 78 µg (15). In this case study, information gathered suggested that the patient consumed at least three glasses (approximately 0.5 L) of his wine per day (his average weight over 2 years was approximately 85 kg). At a lead concentration of 14 mg/L and with 3.5 L consumed per week (49,000 µg/week of intake), this equates to 23 times the intake at the PTWI for a person weighing 85 kg (2,125 mg intake in 1 week). An intake of 49,000 µg/week is 89.7 times the average daily dietary intake of lead in Australia.

This case study outlines the dangers of lead exposure from making wine using enamel bathtubs. People engaged in home wine making should be aware of the possible sources of lead and take adequate precautions to minimize their exposure. It clearly demonstrates that only food-grade materials should be used for the preparation or storage of homemade beverages or food.

This case study highlights the need for clinicians to be aware of potential lead poisonings from environmental as well as occupational sources and the need for elevated PbB values to be investigated so that lead exposure sources can be identified and eliminated.

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Table 1. Results from the initial investigation of possible lead sources and from the testing of wine leachate.

Samples	Lead level	Standard
Food		
Tomato (whole tomatoes and puree)	0.2 mg/kg	0.5 mg/kg (4)
Water		
Sample 1 (mains water, kitchen)	0.004 mg/L	0.01 mg/L (5)
Sample 2 (mains water, bathroom)	0.006 mg/L	
Sample 3 (bottled spring water)	0.015 mg/L	0.05 mg/L (4)
Dust wipes ^a		
Dust wipe 1 (bathtub A)	3.2 mg/sample	
Dust wipe 2 (house)	< 1 mg/sample	
Dust wipe 3 (factory)	< 1 mg/sample	
Leachate ^b		
Pewter cup	< 0.05 mg/L	4.0 mg/L (6)
Wine		
Patient's 1999 batch	14 mg/L (pH 3.8)	0.2 mg/L (4)
Patient's 1999 batch	11 mg/L	
Patient's 1996 batch	0.9 mg/L	
Mr. B's 1999 batch ^c	0.5 mg/L	
Mr. B's 2000 batch ^d	< 0.2 mg/L	
Wine leachate from bathtubs ^e		
Bathtub A	310 mg/L (pH 3.4)	
Bathtub B	1.8 mg/L	

^aFrom an approximately 400-cm² area with an isopropyl alcohol wipe: control wipes have < 3 µg lead. ^bStandard 24-hr leach test with 4% acetic acid (6). ^cWine made in bathtub B. ^dWine made in a stainless steel container. ^eThe wine added to the bathtubs contained < 0.2 mg lead/L and was allowed to sit in the tubs for 7 days.