DR. F. CURTIS DOHAN
221 MALONEY CLINIC BUILDING
HOSPITAL OF THE UNIVERSITY OF PENNSYLVANIA
36TH AND SPRUCE STREETS
PHILADELPHIA 4

June 26, 1962

Dr. Robert A. Kehoe
Kettering Laboratory
College of Medicine
University of Cincinnati
Eden Avenue
Cincinnati 19, Ohio

Dear Dr. Kehoe:

Thank you for your letter of June 4th about the "possible case of tetraethyllead poisoning". I have enclosed copies of the three discharge summaries of the case. The patient was first admitted to the Hospital of the University of Pennsylvania on June 30, 1960 and twice thereafter. On the first admission he showed signs and symptoms which were interpreted as Infectious Hepatitis. Because of stippling of his red blood cell the blood lead concentration was determined. Post-discharge this was reported as three times the top "normal value" for our laboratory, so he was readmitted for treatment with EDTA.

I would be grateful, if after reviewing the information supplied, you would hazard an opinion concerning the possibility that the hepatitis was related to his lead poisoning. At that time when I was "Ward Chief", we were unable to find in the literature available to us, any reference to hepatitis resulting from lead poisoning. However, we had heard rumors of some evidence to this affect that was accruing in Boston.

The work situation was investigated by the Occupational Health Section of the Department of Health of the City of Philadelphia. It was their opinion and that of the Ward staff and others who saw him, that the history of swallowing leaded gasoline when starting a siphoning procedure for gasoline was the source of the lead. He stated he had practiced this type of siphoning for the past year or two before admission to the hospital.

The welding operation and paint were ruled out. The paint did not contain lead. He worked for only three months in the "shop" which was said, by the experienced investigator from the Department of Health, to be quite ventilated.

If you have any questions, please let me know.

Sincerely,

F. C. Dohan

F. Curtis Dohan, M.D.

FCD:m
encl.

August 6, 1960

SUMMARY:

RE: [redacted]
#17-36-63.
Admitted-6/30/60;Discharged-7/22/60.

DISCHARGE DIAGNOSES: Infectious hepatitis.
Lead [redacted] poisoning.

This was the 1st HUP admission for this 46 year old Negro male who entered with a chief complaint of jaundice, discomfort in the abdomen and abnormal urine.

[redacted] gave a history on admission of intermittent episodes of aching midabdominal pain that would last usually a period of several hours but occasionally would be prolonged to a period of 2-3 days. These pains would come on on the average of 2-3 times a month. He had had the first episode about January of 1960. About one month before admission he had begun to notice a gradual loss of appetite. Nine days before admission right upper quadrant and left upper quadrant feeling of annoyance but not pain had developed. On 6/26/60 he first noticed yellowing of his [redacted] sclerae and also thought his stools had been rather light recently. He had received no injections in the previous six months, and had been exposed to no known toxins. In the past medical history it was noted that he had developed hives after an unknown injection 2-3 years previously. He was on no current medication. He works as a truck rental shop body man and has done this work for the last 15 years. He has smoked two packs of cigarettes per week for several years but in recent weeks has developed distaste for cigarettes. He denied any excess alcohol intake.

In the system review it was noted that he had developed a 12 pound weight loss in the previous three weeks and the rest of the system review was entirely negative. On physical examination his blood pressure was 130/90, pulse 72, respirations 20 and temperature 98.4°. He was noted to be a 46 year old colored male in no acute distress whose sclerae appeared moderately icteric. He had shotty posterior cervical and inguinal nodes. He was tender to palpation in the right upper quadrant of the abdomen but the liver and spleen were not palpable. Neurological exam was entirely negative. He was felt to have infectious hepatitis and was put on infectious precautions.

On admission his hemoglobin was 11.4 with 6% reticulocytes, WBC-17,900, neutrophils 64%, monocytes 10%, lymphocytes 25%. AGOT 77, bilirubin 1.1 direct and 2.6 total. Serum cholesterol 254, albumin 4.1, globulin 3.8, alkaline phosphatase 13, acid phosphatase 0.8, BUN 25, and glucose 80. Over the next few days bilirubin rose to 3.2 direct and 6.2 total and his symptoms seemed to get a worse. His alkaline phosphatase was then noted to be 13. Over the next two weeks his symptoms gradually diminished except for occasional bouts of vague epigastric pain. On 7/20/60 he was noted to have basophilic stippling of his red cells in the peripheral smear and blood was drawn for a serum lead determination. On 7/22/60 it was felt that he was well enough to be discharged but that there were still several unanswered questions. He was then discharged to be followed in Hematology Clinic.

A few days later the blood lead determination was reported as 180 µgm.% which is markedly elevated. Arrangements were then made for re-admission for treatment of chronic lead poisoning.

TFR:es Trans. 8/8

Thomas F. Raymond, M. D.

KE 0015327

[redacted] [redacted]

N20383.01

[redacted]

DISCHARGE SUMMARY

ADMITTED: 8/5/60 DISCHARGED: 8/20/60

Discharge diagnosis: Chronic lead poisoning
Lead nephritis, suspected

This 47 year old negro male was admitted to the University Hospital for the second time on 8/5/60 and discharged on 8/20/60. His first admission had been in June, 1960 because of an acute hepatitis. During his first admission he was noted to be anemic with basophilic stippling of the red blood cells. A lead level was drawn at that time but was not reported until after the discharge of the patient. The blood lead was 130 gamma percent, with a top normal in this hospital of 60 gamma percent. Because of this he was readmitted for therapy.

History reveals the presence of crampy mid-abdominal pain occurring 2-3 times per month since January, 1960. These episodes of pain would last from 3 hrs. to 3 days. These have gradually become more severe and more frequent. In the few weeks prior to this admission he had abdominal pain during, and immediately following, each meal. There has been no history of any metallic taste in the mouth or any particular muscular weakness of the arms or leg, and no known personality changes.

Physical examination on admission revealed the presence of a definite lead line on the lingual surface of the gums. Remainder of the examination including the neurologic evaluation was perfectly normal. He had several episodes ofmid-abdominal pain during the first few days in the hospital; these were relieved almost immediately by intravenous injection of calcium gluconate.

Laboratory work on this admission revealed the following: Repeat blood lead was 125 gamma percent; 24 hour urine collection for lead revealed an excretion of 550 gamma, with a top normal in this hospital of 100 gamma per 24 hrs. Hgb. 10.2 gm., [illegible] with moderate basophilic stippling. There were 7-12 percent reticulocytes, WBC and differential were normal, as were the platelets. Repeat liver function tests on this admission revealed an SGOT of 44 units, normal SGPT, bilirubin .5 mg. direct with .2 mg. total. Total protein A/G ratio and alkaline phosphatase were normal. Thymol turbidity and zinc turbidity were within normal limits. Urinalysis was within normal limits except for the presence of many white cells per hpf as well as 1-4 RBC's per hpf. A trace of protein was present.

A neurologic consultant found no evidence of peripheral neuropathy or encephalopathy. Spinal tap revealed normal manometrics and normal protein cells and glucose. Creatinine and BUN were within normal limits. Bone marrow examination revealed a moderate hypercellularity with increase in red cell activity. Some of the more characteristic changes in lead poisoning were present in the bone marrow. A differential Coomb's test was said to be typical of lead poisoning. After settling the cells in the supernatant and top layer of cells of cells was positive, whereas the bottom layer was negative.

With a firmly established diagnosis of lead poisoning with fairly acute symptomatology in the way of abdominal pain, it was decided to give this patient a course of Versenate. He received 1 gm. daily for 4 days. During this period his urinary lead excretion increased markedly varying from [illegible] 11.5 mg. on the 1st day of treatment to 6 mg. on the 3rd day of treatment. A repeat blood lead at the end of the first course was

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[redacted] [redacted]

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55 gamma percent which is within our normal limits. Following the 2nd day of treatment his abdominal pain disappeared and has not returned since that time.

The investigation of possible sources of lead in this gentleman revealed the following two possibilities: (1) He works at the Ryder Truck Rental Co. and has been siphoning considerable quantities of gasoline for the past year or two. He frequently swallows considerable amounts of gasoline whihc, of course, contains tetraethyl lead, (2) from December until early March he painted trucks in an unventilated room, at the same time there were people welding in this room. This, of course, might cause considerable amounts of lead to be present in the air with the possibility that he might have become intoxicated by this route. The environmental health deporment of the Philadelphia Public Health Service is investigating this job situation and will report to us as ssoon as they have more definite information. In the meantime he is not to return to work.

Repeat liver function studies at the end of his hospital course revealed complete return to normal in all those studies which were abnormal on admission. His Hgb. on discharge was 9.5 gm., however, there was evidence that hemolysis had stopped in view of the normal serum bilirubin. He had a 24 hr. urine collection and a ~~xxxxx~~ repeat blood lead drawn just before discharge and this will be checked in one week when he is seen in the Hematology OPD. As far as his urine is concerned following the course of Versenate, the number of RBC's in his urine increased to 13 - 20 with no change in the WBC's or protein. The possibility that this might bepresent a lead nephritis secondary to large amounts of lead being excreted through the kidneys on Versenate therapy must be considered. Culture of his urine on one occasion showed no growth and on the other a few colonies of non-hemolytic staph aureus.

He was discharged completely asymptomatic and will be seen in the OPD of Hematology in one week.

Arnold S. Roland, M.D.
Resident in Medicine

ASR:ea
Dictated 8/22/60

KE 0015329

[redacted]

September 13, 1960

DISCHARGE SUMMARY

Admitted: 8-31-60 Discharged: 9-10-60

Discharge Diagnosis: Lead Poisoning

[redacted], a forty-six year old Negro male was admitted to the University Hospital for the third time on 8-31-60. He had first been admitted from 6-30 to 7-22-60 with a picture consistent with acute hepatitis. At that time a history of intermittent crampy abdominal pain for the previous seven to eight months was obtained. This was associated with moderate constipation. During this admission he was found to have a hemoglobin of 9.3 grams with basophilic stippling. A blood lead was drawn, but the patient was discharged prior to it being reported. It was later reported to be 130 gamma per cent, the top normal in our laboratory being 60 gamma per cent. Because of this he was re-admitted for the second time on 8-5-60 and discharged on 8-22-60. Re-admission was for study and treatment of lead poisoning. A history of exposure was not well documented, but there were several possibilities: 1. He had [redacted] fairly large quantities of gasoline during the previous several years raising the possibility of tetraethyl lead poisoning. 2. On December 1959 until early March 1960 he had painted an trucks in a poorly ventilated room in which welding was also done. This raised the possibility of exposure to lead fumes. His repeat blood lead was 125 gamma per cent. A twenty-four hour urinary collection for lead was 315 gamma per twenty-four hours, the top normal in laboratory being less than 140 gamma. His urinary coproporphyrin III level was 1350 gamma per twenty-four hours, the top normal being less than 150 gamma. He was given a course of calcium disodium Versenate 1 gram intravenously daily for four days. This resulted in a marked increase in the urinary lead excretion, and by the second day the patient had become asymptomatic. A repeat blood lead at the end of this course of treatment was 55 gamma per cent which was just within our normal range. His hemoglobin during the second admission varied from 9.5 to 10.2 grams, with 7 - 16 per cent reticulocytes, and a bilirubin of 2 mg%. Of this 1.5 mg. was indirect. These findings were considered consistent with a hemolytic component to his anemia.

Following discharge on 8-22-60 he did well for several days and then abdominal pain returned. This was crampy, lasting thirty to sixty minutes, and occurring about one half hour after meals. Mild constipation continued. On 8-29-60 he was seen in the Hematology Outpatient Department. A blood lead and urinary lead which had been collected just as prior to discharge revealed the following: The blood lead had risen to 134 gamma per cent, the twenty-four urinary lead was 336 gamma.. Because of the return of symptomatology and the elevated blood level of lead he was re-admitted for further treatment.

Physical examination at this time was remarkable only in that a lead line was visible on the lingual surface of the gums, but not the labial surface, and there was fairly moderate mucosal and conjunctival pallor. Prior to beginning a second course of therapy repeat blood and urine lead were drawn. The blood was 75 mcg.%, and the twenty-four urinary lead 350 gamma per specimen. He was given a four day course of calcium disodium Versenate 2 grams per day intravenously over a six hour period. At the end of this course a repeat blood lead was 50 mcg.%, and the patient was again asymptomatic.

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The results of the urinary lead output during the course of treatment are not yet available to us. Towards the end of this hospital stay a liver biopsy was performed. This revealed evidence of regeneration of hepatic parenchymal cells thought by the Pathologist to be consistent with the recovery phase of viral hepatitis. However, it was not entirely possible to rule out a toxic hepatitis on this basis. He was discharged on 9-12-60 essentially asymptomatic.

We have contacted the Environmental Health Division of the Public Health Dept. of Philadelphia. A field worker has investigated the working conditions, and we have found that there are several other men now working at the same location who may also have been exposed to lead. We are hoping to obtain blood and urine specimens from them for investigation.

The relationship of this man's hepatitis to his lead poisoning is still problematical. It is entirely possible that this represents a case of toxic hepatitis secondary to lead. On the other hand it may be that a viral hepatitis provided sufficient stimulus to bring out a latent lead poisoning.

His hemoglobin on discharge from the hospital was 10.2 grams, and he has been given a follow-up appointment to the Hematology Clinic.

Arnold S. Roland, M.D.
Resident in Medicine

ASR:jm
Transc. 9-13-60
cc: Dr. A. Lewis - H.U.P.
Dr. A. Proucil - H.U.P.
Dr. George Luferig - H.U.P.