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Lead poisoning with low blood lead levels

P. TORKINGTON*
M.A., M.B., M.R.C.P.

*Division of Metabolism,
The Royal Infirmary, Manchester*

K. K. BHALLA
M.D. (LUCKNOW), M.R.C.P.

Bury General Hospital

Summary

Two cases are reported in which symptomatic lead poisoning coincided with normal haemoglobin concentrations and blood lead levels below 80 $\mu\text{g}/100\text{ ml}$. Urinary coproporphyrins and amino laevulinic acid concentrations were elevated. These latter tests are useful for confirmation of clinical diagnoses and for the screening of industrial lead workers.

Introduction

A lead hazard exists in many industries and even where protective measures are enforced, workers have shown indices of exposure only just below danger levels (Gibson, Mackenzie and Goldberg, 1968). Several tests are available for detecting those in danger of becoming intoxicated. Measurements of blood lead and haemoglobin concentration are widely used for such screening. Two cases are described to draw attention to the limitations of these particular tests whether used in a prophylactic capacity in industry or to confirm clinical lead poisoning.

Methods

Blood and urinary lead concentrations were measured by E. King of National Occupational Hygiene Services using the classical monocolour di-

thizone technique (King and Thompson, 1961). Within the relevant range of blood lead concentrations, 50-100 $\mu\text{g}/100\text{ ml}$ whole blood, results are accurate to within $\pm 5\text{ }\mu\text{g}$.

Case 1

A 20-year-old man mixed lead sulphate and lead stearate powders in the manufacture of plastics. An extractor fan was ineffective. Dust readily penetrated his gauze mask. Within 3 weeks of starting the job he developed central abdominal, colicky pain; this was quickly followed by vomiting. The condition was not recognized and after a week at home he resumed work. Within 3 hr he was prostrated by abdominal colic. He was admitted to hospital the next day; at that time the haemoglobin was 14.2 g/100 ml and the blood lead 75 $\mu\text{g}/100\text{ ml}$. The pains diminished over a period of 4 days. Four weeks later at outpatients, the blood lead was 73 $\mu\text{g}/100\text{ ml}$. Against advice, he again resumed work. Within 3 weeks he was re-admitted to hospital, the symptoms had recurred. Vomiting started at the onset of the abdominal colic; it was intractable and was evoked by any oral food or fluid for 3 days after the colic had ceased. He also complained of postural dizziness, parasthesiae in the hands and face and that 'the use had gone out of his legs'. Examination revealed an ill-defined blue line on the upper gum which had disappeared within 5 days but there was no neurological abnormality.

* Present address: Department of Medicine, Sydney Hospital, Sydney, N.S.W. 2001, Australia.

The results of the investigations performed during this second hospital admission are tabulated. After discharge he changed his work and has since been well. Biochemical results obtained at follow up 1 year later are tabulated. The blood lead had fallen to 27 $\mu\text{g}/100\text{ ml}$. Erythrocyte protoporphyrin and urinary amino laevulinic acid levels had fallen appreciably but had not reached the normal range. Other results were normal. The time-course of the biochemical recovery in this patient is typical of lead intoxication.

Case 2

This man was 23 years old. He smeared a paste of inorganic lead salts on to a metal grid in the manufacture of lead-acid batteries and had been doing this work for 2 years. A routine check in 1971 showed him to have a blood lead of 86 $\mu\text{g}/100\text{ ml}$. He consulted the factory doctor in September 1972 because of intermittent abdominal pains, constipation and general lethargy and the lead concentration was 76 $\mu\text{g}/100\text{ ml}$. He was referred to hospital. Considerable domestic stress was evident and made interpretation of his symptoms uncertain. Barium X-ray studies revealed no abnormalities. The results of the laboratory tests are tabulated.

On advice, he was 'removed from exposure to lead' to another part of the factory. Two weeks later his complaints and the blood lead figure were unchanged but thereafter he became symptom-free. However, investigations 4 months later showed a slight fall in haemoglobin concentration and other evidence of persisting intoxication (see Table 1). Enquiry revealed that he was handling dried lead plates and thus was probably still occupationally exposed.

Discussion

In both cases, laboratory tests showed abnormalities consistent with the clinical suspicion of lead poisoning. The possibility of acute intermittent porphyria was considered in view of the evident disturbance of porphyrin metabolism but was ruled out by the absence of neurological or psychiatric manifestations, the absence of porphobilinogen from the urine and the marked increases in erythrocyte protoporphyrin. A co-worker of case 1 was twice admitted to hospital because of colic and vomiting within the space of 3 months. On the second occasion, the diagnosis of acute lead poisoning was made, the haemoglobin being 9.0 g/100 ml, the blood lead 170 $\mu\text{g}/100\text{ ml}$ (corrected for the low haematocrit) and the urinary coproporphyrins 600 $\mu\text{g}/\text{l}$. Bouts of acute illness recurring with re-exposure to lead and ceasing once exposure ceased, together with evidence of similar intoxication in a colleague would seem to put the diagnosis in case 1 beyond doubt. In the second case, it is not possible to be certain that the non-specific symptoms complained of were the result of lead poisoning but such a diagnosis seems the only reasonable explanation for the disturbance of porphyrin metabolism.

Lane and his co-authors (1968) classified lead absorption from occupational exposure as acceptable, excessive or dangerous and defined for several laboratory tests, values they considered characteristic of each level of absorption. Blood lead concentrations of up to 80 $\mu\text{g}/100\text{ ml}$ were regarded as consistent with acceptable absorption; and, although the authors did not intend that this figure should be used in isolation, it has been applied in the screening of industrial workers as an arbiter of safety. The

TABLE 1. Lead poisoning with low blood lead levels; results of laboratory tests

	Case 1		Case 2		Normal range
Interval between investigation and last exposure (days)	3	350	1	?120*	
Haemoglobin g/100 ml	13.9	14.6	15.4	13.6	13.5–18.0
Blood lead $\mu\text{g}/100\text{ ml}$ whole blood	69	27	70	63	<40
Urinary lead (urine S.G.) $\mu\text{g}/\text{l}$	310 (1012)	7 (1011)	288 (1021)	210 (1022)	<80
Erythrocyte protoporphyrin $\mu\text{g}/100\text{ ml}$ whole blood	524	162	341	497	15–40
Erythrocyte δ amino laevulinic acid dehydrogenase i.u./ml	—	—	12	13	28 $\pm$ 11
Urinary coproporphyrin $\mu\text{g}/\text{l}$	1570	70	1720	1460	70–250
Urinary δ amino laevulinic acid mg/100 ml	8.0	0.8	3.0	5.0	0.1–0.7
Urinary porphobilinogen (qualitative test)	Absent	Absent	Absent	Absent	

* Probably still exposed to lead, see text, though he claimed not to have been at risk for 4 months.

cases we report had blood lead values below 80 $\mu\text{g}/100\text{ ml}$ yet both cases were unequivocally suffering from lead poisoning. In addition, both cases had normal haemoglobin concentrations and so would have escaped detection by screening programmes based on the usual haemoglobin or blood lead criteria. Similar cases have been reported (Waldron, 1971; Gibson *et al.*, 1968) and the latter authors recommended that lead concentrations greater than 60 $\mu\text{g}/100\text{ ml}$ should prompt further investigation.

Lead in the blood reflects absorption of the metal. Individuals vary in their susceptibility to intoxication and in practice, indices of absorption do not correlate closely with indices of intoxication (Gibson *et al.*, 1968). It, therefore, seems logical to prefer evidence of intoxication to estimates of absorption both for occupational screening and for confirmation of clinical diagnoses. Of the investigations tabulated for these patients, the measurement of erythrocyte protoporphyrin is too difficult and that of δ -amino laevulinic acid dehydrogenase activity too sensitive (Hernberg and Nikkanen, 1970) for routine use. Regular haemoglobin determinations in all statutory lead workers are required by law (Ministry of Labour, 1965). A decrement in haemoglobin concentration is evidence of significant lead poisoning, other causes having been excluded, but as shown in this report, symptomatic poisoning may coincide with normal haemoglobin values. Measurements of urinary amino laevulinic acid and urinary coproporphyrins are useful in both the industrial and the clinical context (Gibson *et al.*, 1968; Haeger-

Aronsen, 1971). Their sensitivity is such that they become abnormal at an early stage of lead intoxication. Had our cases presented for routine occupational screening, both urinary coproporphyrin and amino laevulinic acid measurements would have recognized a state of lead poisoning; neither haemoglobin nor blood lead measurements would have recognized it.

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Acute barbiturate poisoning in young epileptics

B. W. HANCOCK
M.B., CH.B., D.C.H.

Neurology Unit, Fulwood Annexe, Sheffield

Summary

Two young epileptic patients presenting in coma with focal neurological signs were found to be suffering from unsuspected acute barbiturate overdose. There had been strenuous denial of any possibility of psychiatric disturbance or of self-poisoning in the history obtained from their parents at the time of admission. The importance of excluding deliberate drug overdose in any young epileptic patient presenting with prolonged or atypical coma is re-emphasized.

Correspondence: B. W. Hancock, 42 Winchester Road, Sheffield S10 4EE.

COMA is a common presentation of the known epileptic patient to the acute medical unit. The following two cases are reported to emphasize the importance of excluding deliberate drug overdose in the young epileptic patient whose presenting coma is in any way atypical or prolonged.

Case 1

A 12-year-old girl with a 2-year history of Grand Mal epilepsy was admitted to the Neurology Unit at midnight on 6 December 1972, after being seen at home by her general practitioner. That evening she