Mercury poisoning

A case report and comment on 6 other cases

D. J. SHAMLEY, J. S. SACK

Summary

The diagnosis of mercury poisoning requires a high index of suspicion. Mercury poisoning in a patient involved in illicit gold extraction is reported and 6 other cases considered. Some of the clinical features and treatment of this condition are discussed.

S Afr Med J 1989; **76:** 114-116.

In the last 20 years mercury poisoning has rarely been reported in South Africa, perhaps owing to industrial awareness and control. A case of mercury poisoning is reported and clinical observations of 6 other cases seen in the Neurology Unit at Baragwanath Hospital, Johannesburg, over the past 2 years are discussed. Most of the patients came from goldmining areas, presented to hospital with similar symptoms and their case histories formed a fairly consistent pattern.

Case report

A 24-year-old man, who worked in a post-office on a goldmine, was referred to hospital for investigation of confusion. This had started a few months before and had progressively worsened. There were no recorded previous episodes of confusion and no family history of a similar complaint. There was no significant past medical or surgical history, in particular no history of trauma. The patient was not diabetic or hypertensive. There was a history of moderate ethanol intake over the past few years.

On examination there was no clubbing, cyanosis, pallor, jaundice or oedema. The blood pressure was 110/70 mmHg and pulse 86/min. Heart, chest and abdomen revealed no abnormalities. He displayed abnormal behaviour with outbursts of shouting and screaming, and made sexual advances towards the nursing staff. Formal memory testing was not possible. The pupils were equal and reactive to light. Visual acuity, visual fields and funduscopy were normal.

A slit-lamp examination of the lens revealed no mercuria lentis. The muscle tone was increased with cogwheel rigidity. A resting tremor was present. Muscle power was normal and tendon reflexes were increased. There was no sensory loss. Examination of the cerebellum revealed that the tremor increased on intention. In addition, dysdiadochokinesia, a heel-shin abnormality, gait ataxia and staccato type dysarthria were present. There was no nystagmus.

Chest and skull radiography and computed tomography of the brain were normal. Nerve conduction studies were also normal. Laboratory investigations: haemoglobin 14,6 g/dl; white cell count 5,8 x 10^9/l; platelets 295 x 10^9/l; serum sodium value 139 mmol/l, potassium 4,0 mmol/l, urea 4,8 mmol/l, creatinine 100 mmol/l, calcium 2,4 mmol/l, phosphate 1,0 mmol/l, total protein 69 g/l, albumin 32 g/l, bilirubin (total) 4 μmol/l, aspartate aminotransferase 21 U/l, alanine aminotransferase 32 U/l; γ-glutamyltransferase 50 U/l; and alkaline phosphatase 160 U/l. The erythrocyte sedimentation rate was 10 mm/1st h (Westergren); the test for antinuclear factor was negative; and the serum copper level was 12,8 μg/l. A spot urine mercury test revealed an abnormally high level of 1 693 μg/l (corrected for specific gravity of 1,020) (normal $\leqslant$ 0,5 μg/l). The blood mercury level was 12 μg/100 ml (normal $<$ 0,5 μg/100 ml). A 24-hour urine collection (volume 610 ml) showed that the urinary mercury excretion was 720 μg/24 h.

On further questioning to ascertain the possible source of mercury the patient admitted living in a house where illicit gold extraction took place. The patient was started on penicillamine 10 mg/kg/d in divided doses. On day 21 and day 28

Department of Neurology, Baragwanath Hospital and University of the Witwatersrand, Johannesburg
D. J. SHAMLEY, F.C.P. (S.A.) (NEUROL.) (Present address: Garden City Clinic, Mayfair West, Johannesburg)
J. S. SACK, M.B. B.CH.

Accepted 28 Dec 1988.

after treatment was initiated repeat 24-hour urine collections were taken for estimation of the mercury value, which were 475 μg/24 h and 344 μg/24 h respectively.

The patient's clinical improvement seemed to correlate well with the decrease in urinary excretion of mercury. Fig. 1 shows evidence of improvement of the tremor. Before treatment with penicillamine the patient was unable to draw a spiral (Fig. 1B). On day 21 after treatment (Fig. 1C) there was an improvement, and on day 40 (Fig. 1D) there was marked improvement in the ability to draw a spiral.

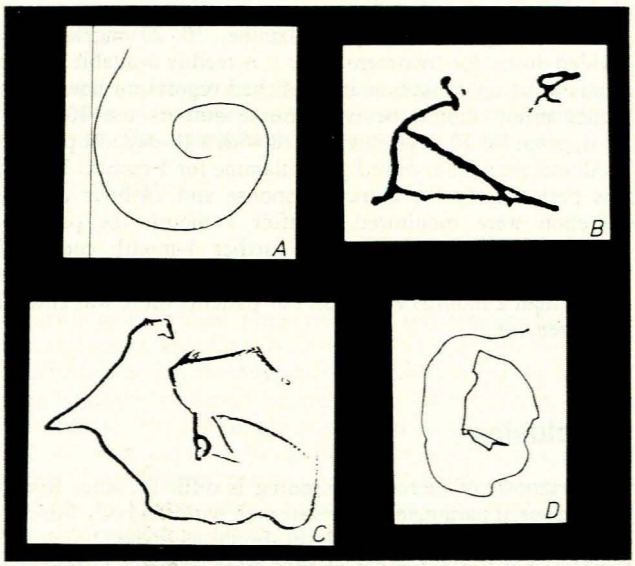

Fig. 1. A temporal improvement in the patient's ability to draw a spiral: A. Control. B. On admission. C. Day 21 after treatment. D. Day 40 after treatment.

Discussion

Over the past 2 years, 6 other patients with similar clinical findings (summarised in Table I) presented to our department. All had raised levels of either blood mercury, random urine mercury or 24-hour urine mercury excretion rates (Table II), and they were all extensively investigated to exclude all other possible causes for their clinical condition.

The normal level of mercury in the urine of subjects who are not occupationally exposed to the metal seems to be below or about 0,5 μg/l. Since mercury in fish is in the form of methyl mercury, which contributes little to mercury in urine, there is little need to take fish-intake habits into account when measuring mercury in urine.[1]

The normal mercury level in the blood is below or about 0,5 μg/100 ml. However, this amount may be influenced by a number of factors including the amount of fish ingested and occupational exposure. In the subgroup of occupationally exposed subjects levels of up to 2 μg/100 ml are acceptable.[1]

In the case reported here, the diagnosis of mercury poisoning was highly probable in view of the patient's clinical signs, raised urine mercury excretion and raised blood mercury levels. A factor in determining whether a patient becomes symptomatic after exposure is the total load of mercury in the body, known as the body burden. Urine and blood mercury levels, although often used, are not good indicators of the body burden, nor are determinations of 24-hour urine excretions since there are large day-to-day fluctuations. Other methods for determining the body burden include cerebrospinal fluid, saliva and hair analyses; the latter being regarded as the best indicator of the body burden. Unfortunately, we were unable to carry out hair analysis on our patients. It has been suggested that weekly averages of 24-hour urine excretions correlate better with the duration of exposure and the severity of the disease.[2]

TABLE I. CLINICAL FINDINGS

Case	Presenting symptoms	Resident in goldmining area	Erethism	Tremor	Ataxic gait	Sensory signs	Type of mercury
1*	**Mental symptoms**	+	+	+	+	+	I
2	**Involuntary movements**	+	+	+	+	-	I
3	**Tremor**	-	-	+	+	+	**Not sure**
4	**Ataxic gait**	+	+	+	+	+	I
5	**Tremor**	+	-	-	-	-	I
6	**Mental symptoms**	+	+	+	-	-	I
7	**Tremor**	+	-	+	-	-	I

*The patient described in this article.
I = inorganic.

TABLE II. MERCURY LEVEL ESTIMATIONS

Case	Level of mercury in blood (μg/100 ml)	Random urine mercury level (μg/l) (corrected for SG 1,020)	Urinary mercury, corrected for volume (μg/24 h)
1*	**12**	**1 693**	**720**
2	**7**	**798**	**Not done**
3	**Not done**	**916**	**736**
4	**12**	**864**	**752**
5	**4**	**701**	**353**
6	**4**	**Not done**	**1 053**
7	**9**	**Not done**	**1 134**

*The patient described in this article.
SG = specific gravity.

The clinical signs of mercury toxicity vary according to whether the mercury is elemental, inorganic or organic, and also whether the exposure to the metal is acute or chronic. Organic mercury is the most toxic of the three due to its lipophilic properties. Elemental and inorganic mercury give rise to a variety of neurological signs, which include erethism and tremor. Erethism is characterised by shyness, irritability, marked outbursts of manic behaviour, hallucinations, insomnia, and difficulty in concentration, which may be associated with vasomotor instability such as excess perspiration, blushing and sialorrhoea. Some of these features were present in patients 1, 2, 4 and 6. The tremor varies in intensity from a mild intention tremor to a severely incapacitating tremor that hampers normal daily activities such as eating and dressing. Some degree of tremor was present in all our patients.

Organic mercury poisoning gives rise to a different clinical syndrome, often referred to as the Hunter-Russel syndrome. It consists of sensory impairment, constriction of visual fields, hearing impairment, ataxia and speech disturbances.[3] Kark *et al.*[4] feel that it is not possible to distinguish between the two syndromes clinically since they may represent the same disease at different ends of the spectrum.

Five of the 7 patients (No. 1, 2, 4, 5 and 6) admitted to having been in the proximity of illicit gold extraction, which is not an uncommon practice in goldmining areas (W. O. Harrison, NIOH — personal communication). Patient 7 had previously worked in a goldmining area.

Mercury is used in extracting gold from quartz. Finely ground ore is mixed with water to form a pulp. The wet pulp is then passed over copper plates coated with mercury. The mercury dissolves the gold to form an amalgam that is heated until the mercury boils off. During the boiling process, mercury vapour is released.[5]

Patients 1, 2, 4, 5 and 6 admitted to living in houses where illicit gold extraction was being carried out. Unfortunately, we were unable to visit the homes of the patients to determine the mercury levels in the home environment or the mercury load of other members of the family. None of our cases was exposed to mercury as a result of inadequate industrial safety standards, and none was referred from industry.

Patient 3 did not live on a goldmine and emphatically denied any involvement with illicit gold extraction. In view of the fact that he showed signs of a neuropathy, with minimal mental changes, he may have been exposed to organic mercury, the source of which is unknown.

Patient 7, a painter, although denying involvement in gold extraction, worked in a goldmining area. Again we are unsure as to the source of the mercury, since paints today do not contain mercury, the exception being nautical paint (W. O. Harrison, NIOH — personal communication).

The treatment of mercury poisoning depends on whether exposure is acute or chronic. In both, removal from the source plays an important role. In acute poisoning dimercaprol and 2-3 dimercaptosuccinic acid are used with good effect. In chronic poisoning a number of chelating agents can be used, including British anti-lewisite (BAL), calcium ethylenediamine tetra-acetic acid (Ca_2 EDTA), penicillamine and the acetylated derivative, D, L, *N*-acetyl penicillamine. The latter agent seems to be the most effective since it is more resistant to metabolic degradation than the parent compound. Although chelating agents are used in chronic poisoning it is uncertain whether the clinical improvement can be attributed to the agent since removal from exposure may bring about the patient's improvement. Several authors do not recommend the use of chelating agents in chronic mercury poisoning.[6]

We elected to use D-penicillamine, 10-20 mg/kg/d in divided doses, for treatment since it is readily available. There seems to be no consensus in published reports on how much penicillamine should be used. Some authors use 100 mg/kg/d, given for 10 days, interrupted with a 10-day rest period.[4]

All our patients received penicillamine for 1 month. During this period both the clinical response and 24-hour urinary excretion were monitored. If after 1 month the patient's symptoms did not improve, a further 1-month course of treatment was given. No patient was given penicillamine for longer than 2 months and in all our patients there was clinical improvement.

Conclusion

The diagnosis of mercury poisoning is difficult, since few of the measured parameters correlate well with the body burden. A raised spot urine mercury level should alert the clinician to the fact that the patient may have been exposed to mercury and that further investigation is necessary.

Six out of the 7 cases discussed came from goldmining areas and patients from these areas with tremors and signs of cerebellar dysfunction should be investigated for evidence of mercury exposure since these symptoms are potentially reversible.

REFERENCES

1. World Health Organisation. Recommended health-based limits in occupational exposure to heavy metals. *WHO Tech Rep Ser* 1980; No. 674: 102-115.
2. Kark RAP. Clinical and neurological aspects of inorganic mercury intoxication. In: Vincken PJ, Bruyn GW, eds. *Handbook of Clinical Neurology*. Vol. 36. Amsterdam: Elsevier-North Holland, 1979: 147-197.
3. Marsh DO. Organic mercury: methyl mercury compounds. In: Vincken PJ, Bruyn GW, eds. *Handbook of Clinical Neurology*. Vol. 36. Amsterdam: Elsevier-North Holland, 1979: 73-81.
4. Kark RAP, Poskanzer DC, Bullock JD, Boylen G. Mercury poisoning and its treatment with *N*-acetyl-D, L-penicillamine. *N Engl J Med* 1971; **285:** 10-16.
5. *World Book Encyclopedia*. Vol. 5. London: Field Enterprises Education Corporation, 1965: 580-585.
6. Twardowska-Saucha K. Evaluation of the chelating agent of methicillin in prolonged experimental metallic mercury poisoning. *Br J Ind Med* 1986; **43:** 611-614.