# Lead Poisoning due to Traditional (Ayurvedic) Medication

P. Mishra, P. Shinde, S. N. Shinde, S. Y. Wagh, A. Atre, R. S. Wadia

**Dr. R. S. Wadia**
M.D.

It is a common belief of the lay public that allopathic medicine is prone to multiple side effects, and that traditional and herbal medicines are therefore better as they are free of such harmful effects. This belief is also much fostered and encouraged by advertisements in the media. Even the law requires an allopathic medication to be marketed only after proof of its efficacy and freedom from adverse effects, but allows a traditional or herbal medicine to be used without any such proof and on the word of any practitioner. Recently we came across a case of lead poisoning due to use of ayurvedic medication. Later on, during discussion amongst colleagues, several narrated similar experiences they have had in the past. It may be mentioned at this stage, that a 2003 US CDC report found 12 cases of lead poisoning due to traditional medicines in the US[1]. 11 of the 12 cases were Indians, the 12th case being a Nepali. All twelve had been taking ayurvedic medications obtained from India. If 12 cases could be found in the US, the number of such cases occurring in India can well be imagined, and suggests that we are missing several cases.

We are describing 3 cases that developed various manifestations of lead poisoning viz. anaemia, abdominal pain and encephalopathy from the use of ayurvedic drugs. It is clear this is a frequently missed medical diagnosis, where clinical manifestations reach severe proportions and occasionally may be fatal.

**Case 1**

Mr. TS, a 41 year old bank officer, was admitted in early September 2004 at Ruby Hall Clinic (RHC) with complaints of imbalance while walking, apathy, inattentiveness, poor sleep and poor appetite. On interrogation the wife revealed that he had been well and asymptomatic until late March when he had an episode of vertigo which lasted 10-15 minutes, this episode was neglected by them. About fifteen days later he had vertigo again. This time he consulted a doctor who found his BP to be 160/120 mmHg and started him on antihypertensive medication. Two days later the wife found him lying on the floor; he was sweating profusely and was in altered sensorium. He recovered from this state but remained drowsy. He was admitted to a hospital where a CT-scan was done and reported an infarct in the basal ganglia. During his stay in the hospital he developed inability to pass urine. An Uroflowmetry examination revealed no obstruction and he was advised intermittent self- catheterization. It was for these urinary complaints that he was admitted to an Ayurvedic hospital; renal function tests and urine examination were found to be within normal limits; Hb was 14.1. He was started on Ayurvedic medication in early May and by late May he could pass urine on his own; the catheter was removed. He continued with the medication and was apparently free of any serious disability for the next three months. In early September he developed imbalance [? to the right] and relatives complained of his apathy, inattentiveness, poor appetite and poor sleep. He was examined outside for the progressive deterioration of mentation and ataxia; a MRI brain done, showed multiple patchy gray and white matter lesions in occipital, frontal, cingulate gyrus and cerebellum. The radiology opinion was either a Vasculitis or Encephalitis. It was at this stage that he sought medical care at RHC.

MMSE was done: It was noted he was slow in answering, inattentive, but the score came to 26/30. He could do 100-7 only up to 93 orally, and required pen and paper for the remaining calculations, but he eventually got these with three mistakes. He was confused about his son's name. His writing was poor. Power in all four limbs was good, but he had hypertonia and hyperreflexia. Both plantars were extensor. He had marked gait ataxia and tremor of the extended hand.

Multiple Tests were Done - His Hb was 7.1, much reduced from original. Indirect bilirubin 1.5mg%, Reticulocytes 3.5%. Blood Sugar, Blood Urea and Sr. Creatinine levels were normal. The EEG showed excess bilateral theta activity.

A repeat MRI-scan of the brain showed the same grey and white matter changes, (Fig 1) The lesions did not enhance MR-angiography done on suspicion of a vasculitis was also normal. Collagen workup showed normal ANA level, anti ds-DNA 6 and ESR 23mm at 1 hour. Routine CSF revealed normal cells, Protein 315 and Sugar 68. Nerve Conduction, Vitamin B12 and Folate levels were within normal limits.

Blood samples were sent to the National Referral Centre in Bangalore, which recorded that the blood lead level was 161 micrograms/dl. The Centre receives 200 blood samples for lead level estimation every month and we were told this is highest they have received from any patient.

With the report in hand the patient was reassessed. He had no leadline, no basophilic stippling on peripheral blood smear, no urine porphobilinogen. He was taking Mahayograg Guggulu 12 tab daily, Laxmivdas Rus 3 daily and a Mixture of Rajat Sindhoor 125 mg, Jatta mansi 1 TSF, Dhanara 1 TSF, Khurasani ova and Ashwargandha 1 TSF regularly for the last 5-6 months.

The wife's blood lead level was also done and was 9 microgram/dl. Following the report he was given BAL injections 250mg IM 8 hourly for 4 days and then oral penicillamine. He clearly improved; ataxia was minimal, understanding had cleared and there were no significant signs by the end of one month. At the end of 6 weeks the scan repeated was almost normal. His blood lead level at this time was 68 ug/dl. Subsequently it has been reported 38 ug/dl after 4 months therapy.

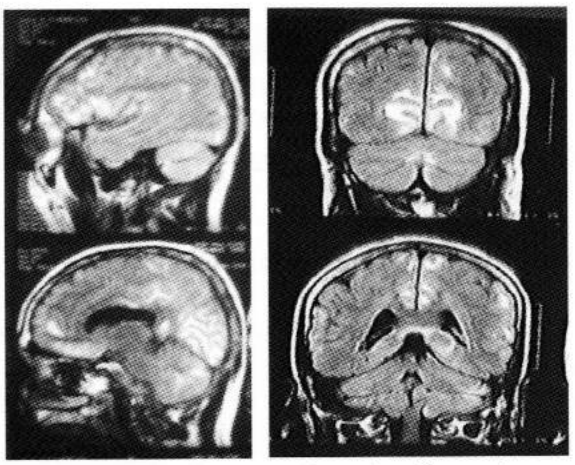

Fig 1. T2 weighted images of case 1 at diagnosis

**Patient Statistics for the period Nov.04 to May 05**

| | Nov'04 | Dec'04 | Jan'05 | Feb'05 | Mar'05 | Apr'05 | May'05 | Total |
|---|---|---|---|---|---|---|---|---|
| 1. Total No. of OPD Patients | 8555 | 9645 | 9790 | 9354 | 10236 | 9523 | 10445 | 67548 |
| 2. Total No. of Indoor Patients Admitted | 1725 | 18632 | 1756 | 1684 | 1874 | 1741 | 1945 | 29357 |
| 3. Total No. of Free and Concessional cases in OPD (All Depts.) | 468 | 488 | 510 | 503 | 571 | 491 | 505 | 3536 |
| 4. Free cases treated (General Ward Patients Only) Indoor | 58 | 57 | 53 | 42 | 53 | 53 | 73 | 389 |
| 5. Concessional cases treated (General Ward Patients Only) Indoor | 76 | 96 | 78 | 83 | 68 | 78 | 78 | 557 |

**Case 2**

A 55-year-old male diabetic was admitted with H/o abdominal pain & mental changes. He started with recurrent severe abdominal pain lasting ½ - 1 hour more than once a day. After 15-20 days of pain he developed mental changes. He had become extremely violent, abusive, and rowdy and had not slept for several days. He was more violent at night. Apparently the mental changes were more severe when he had the episodes of abdominal pain. He was seen by a neurologist who found no significant deficit, except for depressed deep tendon reflexes. It was difficult to communicate with him, but there were indications that his memory was probably impaired.

Routine laboratory investigations, CSF examination and MRI brain were normal. Urine was tested for Porphyria; one lab gave a normal report while another lab gave a weak abnormal report. A differential diagnosis of Porphyria and Lead poisoning was made. Some days later his Blood lead level was done and reported to be 75 micrograms/dl.

The source of lead was unclear; he works in a chemical factory [which does not use lead] he also had been taking Ayurvedic medicines for diabetes for several months. After Penicillamine he is well and has no problems today.

**Case 3**

A male aged 66 has been under treatment for apparently diverse complaints since March 1999. In July 97 he was started on Mahayograg Guggulu 500mg BD for osteoarthritis. In March 99 he was seen for abdominal pain, which subsided with symptomatic treatment. In March 2004 he was seen for excess fatigue; investigations revealed that his Hb was 6.8 and that he had hypochromic anaemia with anisopoikilocytosis; he was promptly put on haematinics. Repeat haemogram done in July 04 showed Hb to be 8.7, and another done in August 04 showed it to be 9.4. However in November 04 he had a TIA like episode; his Hb had fallen to 8.0 and peripheral smear again showed hypochromic anaemia with anisopoikilocytosis.

Bone marrow examination done in Dec 04 demonstrated hypochromia and the bone marrow did not show significant depletion of iron stores. Hb Electrophoresis was normal. All routine lab investigations were normal. By this time the case 1 had been discussed in a clinical meeting; basophilic stippling was looked for on a peripheral blood smear, it was not seen. But blood lead level came to 56.1μ/dl. The ayurvedic medicine has been stopped, and he has been started on Penicillamine.

**Discussion**

In the modern day, safe blood lead level is taken to be under 10 microgram/dl (equal to 0.48 mmol/litres). **The level is measured in whole blood as most of the lead is inside the RBCs, serum level is unimportant**. Levels above 25 microgram/dl are considered significantly elevated and are to be kept under surveillance and levels above 40-45 microgram/dl deserve treatment with DMSA even if the individual is asymptomatic. Levels above 60 are usually symptomatic and levels of 80-100 cause CNS manifestations.

In the US surveillance study[2] in 2002, 10,658 persons had lead levels above 25μ/dl which comes to 10.1/100,000 employed personnel and 1854 (1.7 /100,000 employed) had levels greater than 40 microgram/dl. 5% of all cases were non-occupational, most commonly from shooting firearms, or remodeling houses. 7% of the non occupational cases were from PICA and 4% was from the use of non traditional medicines. Five states reported 12 cases of lead poisoning from traditional remedies....in all cases Ayurvedic medicines prescribed from India were incriminated[1]. In a recent US study the heavy metal content of Ayurvedic drugs

available at stores inside a 20-mile radius of Boston[3] was examined; Of the 70 Ayurvedic medcine tested 14(20%) contained heavy metals including 13 containing lead [median conc. 40 µ/G with highest level 37000 µ/GM], 6 contained mercury [median 20,225 µ/Gm] and 6 contained arsenic [median 430 µ/gm max 8130 µ/gm].

Table 1 shows the lead content of some important ayurvedic preparations.

Lesser levels are present in Keshar, Punarvadi Trifla Guggal, Arogyavardhani 63/ppm & Chavanprash 7.3 ppm

Surma and Kohl used as eye cosmetics contain lead. Surma actually has 34% or more of lead but market samples mixed with Talc etc and Pb % falls to 1%. Sindhoor also has high levels and a family has been found poisoned due to addition of Sindhoor in food for colouring. In Indian and Pakistani children using eye cosmetic blood Pb measured 13 µ/dl. Sood et al[6] described 11 cases of lead poisoning in North India presenting as diffuse pain in abdomen, anaemia and mild abnormal liver function tests. Eight of these had history of taking indigenous and herbal medicines for diabetes and psychosexual disorder.

Of course there are other sources of lead toxicity in India other than Ayurvedic drugs[4]. The main sources are contaminated soil and dust, usually due to heavy vehicular traffic. Other causes include mining, smelting, highway construction, country liquor, adulteration of food. In India there are other occupations known to be associated with lead poisoning which are relatively peculiar to India. These are listed in Table 2.

Of interest is that lead absorption from the GI tract is increased in children (5 times as compared to adults), pregnant females and those with iron and Ca deficiency. Also lead is seen in herbal remedies across the world; most famous of these are Greta and Azarcon from Mexico and Pay-loo-Ah from South East Asia. Each of these may contain 90% or more of lead.

**Table 1: Lead levels in some ayurvedic medications[4,5]**

| Drug | % Lead | Usual Indication |
|---|---|---|
| Trivanga Bhasma | 26% | Diabetes & sexual |
| Pusppadhanva | 7-8% | Fertility pill |
| Brahmi vati | 2% | Brain tonic |
| Diabline Bhasma | 3.7% | Diabetes |
| Ghasard | 1.6% | Dyspepsia |
| Mahayograg Guggulu | 1.0% | Osteoarthritis |
| Vatvidhvans | 3.3% | Analgesic / arthritis |

**Table 2 - Unusual Occupations where lead poisoning may occur in India**

Indian perspective:

1. Battery recyclers unorganized outside pollution control board. One study 53% have level > 10 µ/dl.
2. Working at petrol pumps, auto repairs.
3. Removing impurities from silver jewellry
4. Bangle making
5. Soldering units. High incidence of women.
6. Village grinding mills.
7. Hand compositors in printing 1960-70

## References


1. CDC Lead poisoning Associated with Ayurvedic Medications Five States 2000-2003 MMWR 2004. 53; 582-584.
2. CDC adult blood lead epidemiology and Surveillance United States 2002. MMWR 2004; 53: 578-582.
3. Saper RB, Kales SN, Paquin J, Burns MJ, Eisenberg DM, Davis RB, Phillips RS. Heavy metal content of ayurvedic herbal medicine products. JAMA 2004; 292: 2868-73.
4. JAMA Report (1984) Lead poisoning associated death from Asian Indian Folk Remedies Florida. J Am. Med. Assoc. 252, 3118-3120.
5. Nambi KSV, Raghunath R, Tripathi RM, Khandekar RN (1997) Scenario of Pb pollution and children in Mumbai: current air quality standard vindicated. Energy Environ. Monitor 13, 53-60.
6. Sood A, Midha V, Sood N. Pain in abdomen-do not forget lead poisoning. J. Gastroenterol 2002; 21: 225-6.


**Editors Note**:- We showed these cases at an Association of physicians conference in Pune. At the same meeting-

a. Dr. U P Divate described a case of resistant abdominal pain shown to be due to lead toxicity. The ayurvedic medicine was shown by HPLC to contain lead and Arsenic.

b. Dr. V Shah described a doctor with resistant anaemia whose blood lead levels were shown to be high in US. He was on ayurvedic medicine and showed basophilic stippling.