ISSN: 2690- 909X

Case Report

Advances in Neurology and Neuroscience

Intoxication Camouflaged: Lesson Learnt. An Interesting Case of Lead Poisoning

Karan Chouhan[1], Manjeet Kumar Goyal[1], Sidakbir Singh Paul[1], Birinder Singh Paul[1,2] and Diljot Singh[1]*

[1]*Dayanand Medical College & Hospital, Ludhiana, Punjab, India.*

[2]*Professor Department of Neurology.*

***Corresponding Author**
Diljot Singh, Dayanand Medical College & Hospital, Ludhiana, Punjab, India.

Submitted: 2023, Aug 14 ; **Accepted:** 2023, Sep 16 ; **Published:** 2023, Sep 28

Citation: Chouhan, K., Goyal, M. K., Paul, S. S., Paul, B. S., Singh, D. (2023). Intoxication Camouflaged: Lesson Learnt. An Interesting Case of Lead Poisoning. *Ad Neur Sci, 6*(2), 257-260.

Abstract

Lead poisoning is a common occupational health hazard and may lead to permanent neurological sequelae or even death. However, there is an emerging trend of new forms of non-occupational lead poisoning which is challenging to diagnose. We report a case of a 40 y/o male who presented with chronic lead neurotoxicity occuring after consumption of over-the-counter ayurvedic medication. While exploring medication for its content, it was discovered that this abused drug had Papaver somniferum (opium) in the alarmingly high ratio of 12:1. A review of literature demonstrated that Lead is usually added to opium during its preparation either as an ingredient or as an adulterant to increase opium weight in its trading for profitability. Additionally, improvement in the neurological syndrome after chelation therapy with D-penicillamine further confirmed that Lead was the culprit responsible for this unusual presentation.

Keywords: Non-occupational, Lead Poisoning, Adulteration, Ayurvedic Medication, Neurotoxicity

1. Introduction

Lead poisoning is a common occupational health hazard that may lead to permanent neurological sequelae or even death. However, there is an emerging trend of new forms of non-occupational lead poisoning which are challenging to diagnose. We report a case of chronic lead neurotoxicity that occurred after consuming ayurvedic medicine.

2. Case Presentation

The 40-year-old gentleman presented with a one-month history of difficulty in walking and maintaining balance followed by restlessness and irritability for seven days before admission to the emergency room. His personal history revealed that he consumed alcohol (375mg/day) for 18 years but for the last seven months he had shifted to the use of ayurvedic medication (12-14 tablets/day) in place of alcohol. On examination, he was inattentive and agitated, his RASS was (+2) and CAM-ICU was positive suggesting acute delirium. There were no meningeal signs, power was 5/5 (MRC), and brisk (3+) deep tendon reflexes. He was shifted to the neurology intensive care unit where he was managed for delirium. By day five, his RASS became zero, CAM-ICU turned negative, and MMSE testing revealed a score of 18/30. As his delirium improved, a detailed higher mental examination revealed recent memory and attention deficits and symmetrical cerebellar signs involving limbs. Laboratory examination showed hemoglobin of 6.5g/dL and microcytic hypochromic anemia with reticulocytosis (5.79%) while the liver, kidney, thyroid, autoimmune and infective workups were normal. MRI brain showed cerebellitis (Figure 1,2). There was also a bluish pigmentation of the gum–tooth border suggestive of the lead line (Burtonian line, Figure 3). The bone marrow biopsy showed microcytic hypochromic anemia, basophilic stippling, and Howell-Jolly bodies (Figure 4). The blood lead levels were sent for confirmation of lead neurotoxicity. Meanwhile, he was treated with chelation therapy with oral D-penicillamine (25 mg/kg/day) for five days and his lead levels came to be 365 µg/dL [normal blood Lead levels (BLL): < 5 µg/dL]. As the blood lead levels started decreasing, his cognitive deficits and ataxia started improving. He was discharged with normal MMSE and cognitive functions. On follow-up after 3 weeks, his neurological examination and MR imaging of the brain were normal.

3. Discussion

Lead neurotoxicity is less common in adults than children and is mainly associated with occupational exposure. We report this interesting and challenging case of lead neurotoxicity. The two reasons that make this case worth reporting are the unusual presentation of lead poisoning with delirium and cerebralitis and secondly, the non-occupational, rare, recreational drug as source of poison.

The blood lead levels (BLL) normally should be < 5 µg/dL but in this scenario, the patient had BLL >350 µg/dL [1]. Our patient also had systemic features suggesting lead poisoning like gum-lead-line, basophilic stippling, Howell-Jolly bodies of RBC with microcytic hypochromic anemia, and elevated blood lead levels which confirmed the diagnosis of lead neurotoxicity. Ataxia was a prominent feature in our patient and has been described as

a feature of lead encephalopathy especially in children. There are two case reports from India where patients presented with neurological features following occupational exposure of lead. In one of the case reports, the author remarked about the difficulty in walking, an unsteady gait, and incoherent speech in addition to T2-weighted bilateral hyperintense lesions in both thalami with no radiological involvement of the cerebellum. While in the other case report, the presenting neurological features were seizure and encephalopathy with no involvement of the cerebellum either clinically or radiologically (bilateral thalamic hyperintense lesions). Our case had an unusual neurological presentation in the form of gross cerebellar edema involving both hemispheres, which clinically presented as ataxia. This isolated cerebellar involvement due to chronic lead intake has not been reported previously. Lead metal acts as a cellular toxin by inhibiting mitochondrial respiration and the cerebellum itself has a very high metabolic rate hence higher lead levels in the blood might have resulted in its cellular toxicity. Once started on chelating therapy with D-penicillamine the patient made a complete clinical, radiological and hematological recovery over 2 months.

Another interesting point of discussion, in this case, was the source of lead poisoning, which resulted in neurological deficits. After reviewing the contents of ayurvedic medication (Kamini, an aphrodisiac drug) abused by our patient, we found out the presence of Papaver somniferous (a derivative of opium) in the ratio of 12:1. Opium poppy (Papaver somniferum) is a medicinal plant known to the human race since the ancient civilizations and continues to be cultivated around the globe for the production of pharmaceutical opiates and heroin. There is literature supporting that this ayurvedic medication is abused for opium kick and our patient consumed 12 tablets of the drug Kamini daily (144 parts of Papaver somniferum/day) [2,3]. A review of 14 studies including case reports demonstrated that non-occupational lead poisoning could be related to the lead contamination of opium. It is yet to be determined whether the lead is added to opium during the process of opium preparation or added as an adulterant to increase opium weight in its trading for profitability [4]. The WHO recommends that a person with a body weight of 68 kg can intake 240 mg lead/day as a tolerable daily intake (TDI). Evidence shows that if a person consumes approximately 30 g of opium, he will receive at least 20% of TDI [4,5]. This concentration of lead in opium if used for long-term may cause toxicity. Our patient had chronic, severe lead toxicity according to the criteria of the Occupational Lead Poisoning Prevention Program [6]. These clinical observations are substantiated by whole blood lead level measurements, which were greater than 25 µg/dl in our patient. This case highlights the non-industrial and non-occupational exposure to toxic levels of Lead with the use of ayurvedic medicine easily available over the counter. One should consider lead poisoning in an adult presenting with unexplained encephalopathy. A detailed history of usage of all medications, along with knowledge about their composition may be a clue for chronic lead poisoning, as any delay in diagnosis may be fatal or cause significant disability.

4. Lesson Learnt

• Lead poisoning can occur as a non-occupational or industrial exposure.
• Alternative forms of treatment may not display the actual contents which could be contaminated with lead.
• Lead poisoning if not diagnosed timely may cause permanent neurological dysfunction and may even cause death.
• A high index of suspicion and eliciting the history of usage of purported drugs is important for diagnosis, especially in cases of subacute onset of encephalopathy or focal neurological dysfunction

References

1. Adult blood lead epidemiology and surveillance (ABLES): Reference Blood Lead Levels (BLLs) for Adults in the U.S. 2021. Centers for Disease Control and Prevention.
2. Basu, D., Gaur, N., Das, P. P., & Mattoo, S. K. (2010). Herbal medicines: perfect garb for opioid abuse? A case series from India. The American Journal on Addictions, 20(2), 174-175.
3. Algora M, Martín-Castillo A, Zabala P, Fernández MN. Saturnismo por drogadicción: nueva fuente de intoxicación con interés clínico e importantes consecuencias epidemiológicas [Lead poisoning due to drug addiction: a new source of poisoning with clinical interest and important epidemiological consequences]. An Med Interna. 1989 Sep;6(9):483-5. Spanish. PMID: 2562725.
4. Alinejad, S., Aaseth, J., Abdollahi, M., Hassanian-Moghaddam, H., & Mehrpour, O. (2018). Clinical aspects of opium adulterated with lead in Iran: a review. Basic & clinical pharmacology & toxicology, 122(1), 56-64.
5. Aghaee-Afshar, M., Khazaeli, P., Behnam, B., Rezazadehkermani, M., & Ashraf-Ganjooei, N. (2008). Presence of lead in opium. Arch Iran Med, 11(5), 553-4.
6. Occupational Lead Poisoning Prevention Program (OLPPP) [Last accessed on 2023 May 10].

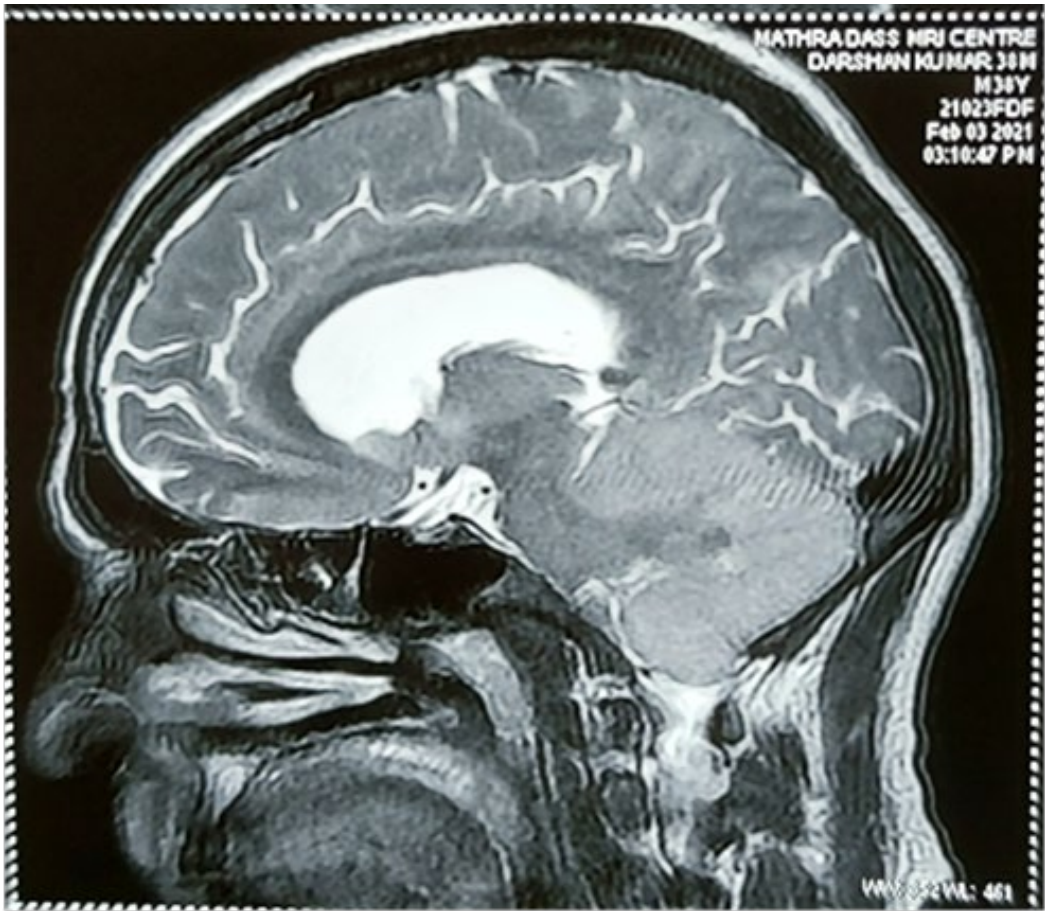

Figure 1: T2W- MRI sagittal section showing cerebellar edema

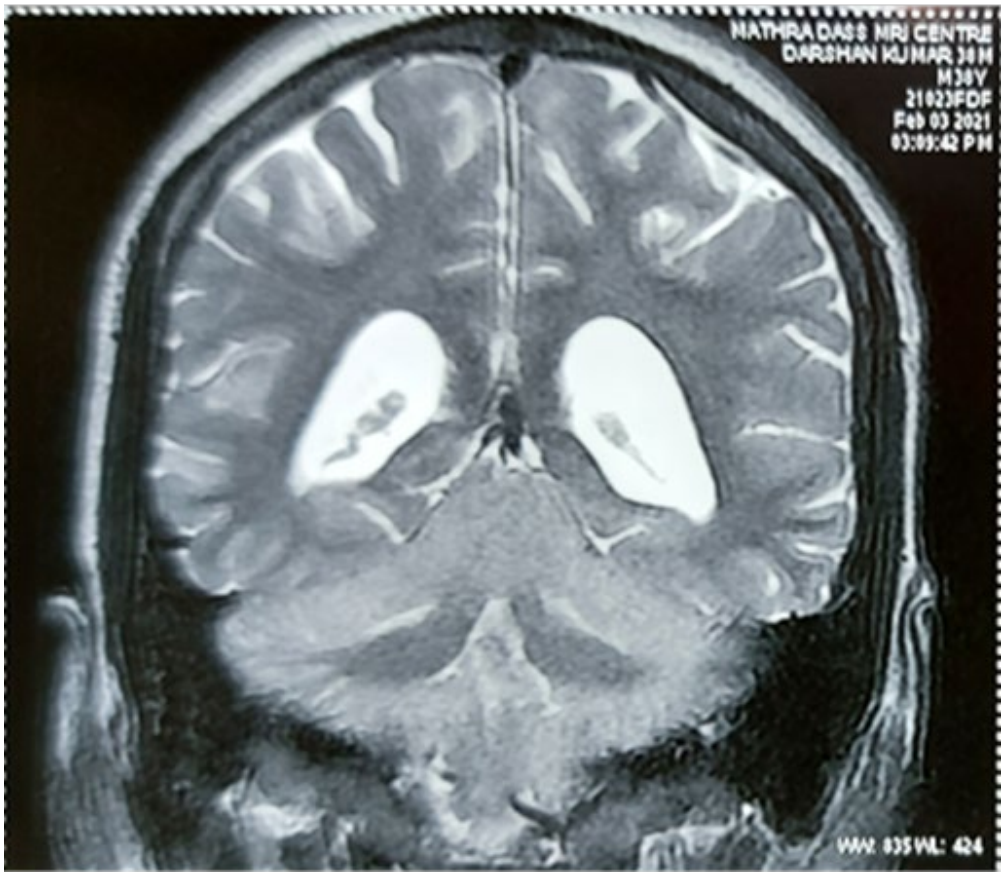

Figure 2: T2W-MRI coronal section showing cerebellar edema with narrowing of the fourth ventricle

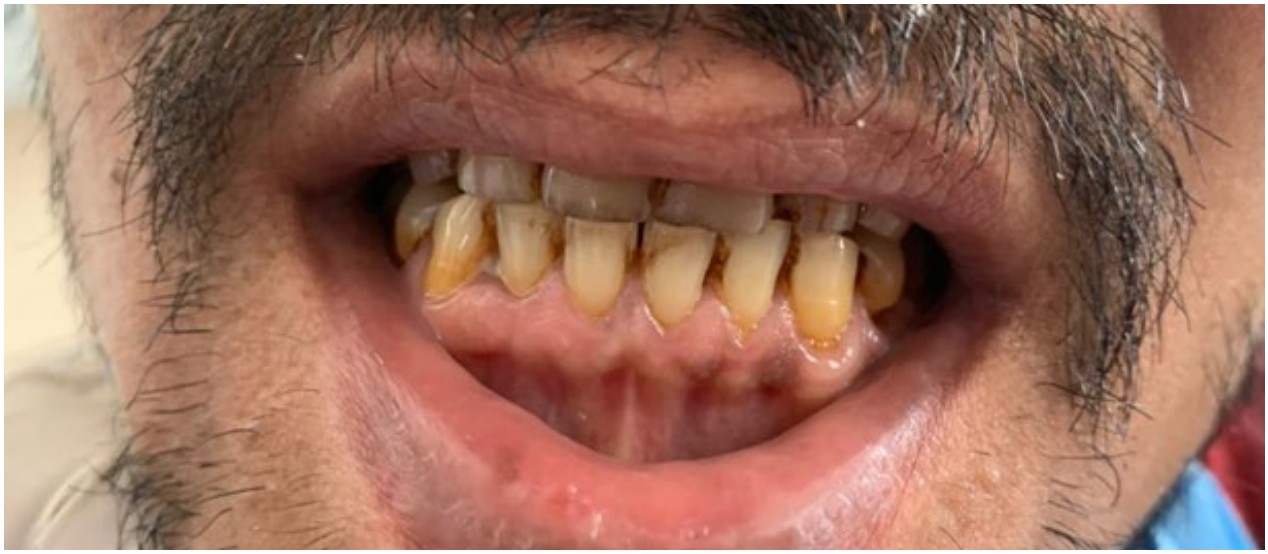

Figure 3: Bluish pigmentation of the gum–tooth border suggestive of the Lead line (Burtonian line)

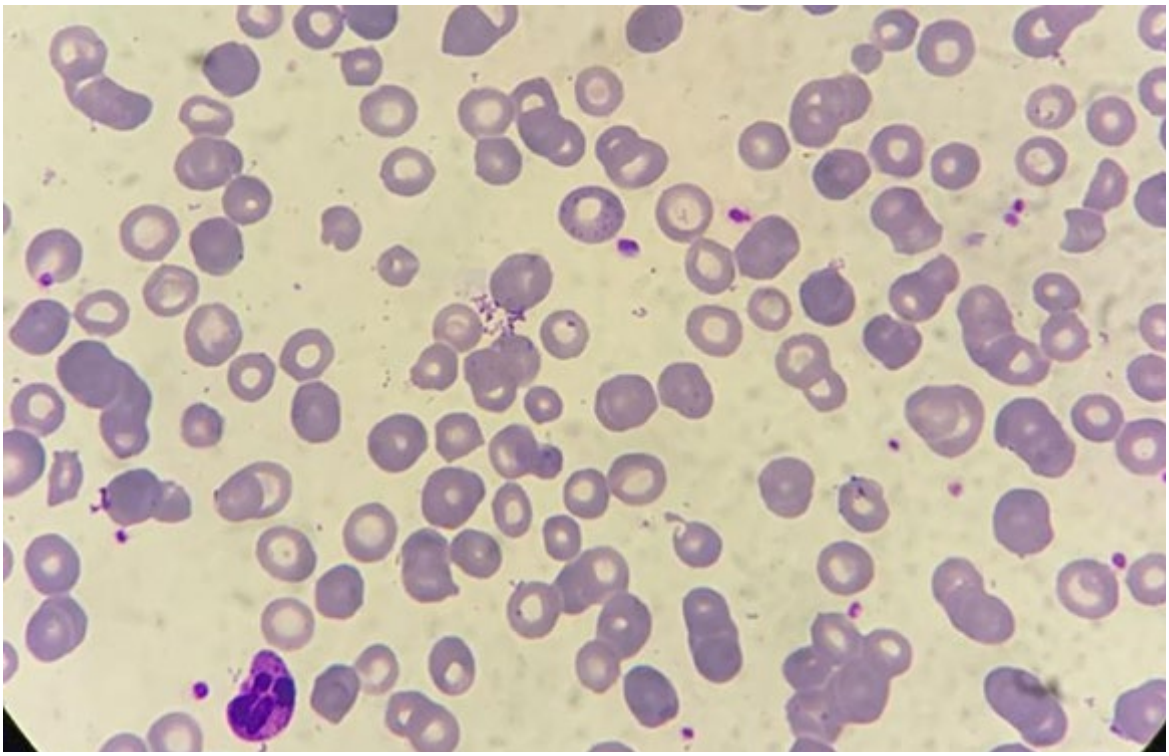

Figure 4: Peripheral blood film showing microcytic anemia with presence of Howell- Jolly bodies.

Copyright: *©2023 Diljot Singh, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.*