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Lead Poisoning and Neuro-Psychiatric manifestations a need for screening of at risk population - A Case Report

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Abstract : A case report of a 16 years old adolescent boy with Schizophrenia like Psychosis secondary to severe lead intoxication due to use of traditional ayurvedic medicinal preparations is presented. This case report attempts to revisit the etiological association between toxic metal poisoning and psychiatric disorders. Implications of screening and treatment in low and middle income (LAMI) countries, like India, are reviewed.

Keyword : Lead Poisoning, Psychosis, Ayurvedic Medications

Introduction

Normal Brain Functioning and Physiological Homeostasis are known to be a result of a complex interplay of Neurotransmitter systems. Certain toxic substances are known to disrupt this equilibrium in the Brain and cause variety of symptoms ranging from cognitive and memory problems to subtle as well as overt alterations in thoughts, emotions and behaviors (1). Lead is the commonest metal involved in chronic poisoning. This metal is a natural component of the earth's crust with trace amounts existing in soil, water and plants. Environmental sources of lead may be from air, food and water pollution. Leaded gasoline and paints were the main sources of lead pollution in the environment. Ayurvedic Herbal Medications uses natural Plant based preparations to treat a variety of human ailments ranging from headache to Cancer. This system believes that minerals like Copper, Gold, Lead, Sulfur and Arsenic are vital molecules within the human body and hence these minerals are often added in the Ayurvedic preparations (2). One of the common presentations of lead and heavy metal poisoning in India is often following use of Ayurvedic Herbal medicine preparations (3). Neurotoxicity from heavy metals is most commonly studied in two groups: acute or chronic exposure. Acute exposure often involves rapid onset of nausea, headaches, cognitive changes, and emotional disruptions. In chronic exposure, neurodegeneration and psychiatric manifestations are more common. Psychiatric manifestations commonly seen may include increased depression, anxiety, and irritability (4). **Epidemiology**

Lead poisoning affects persons of all ages. Some of the special populations identified are young children, aged 1-6 years, whose primary source of exposure is deteriorated lead paint in their home (6). The second large, affected group is adults, whose occupation involves lead smelting or reclamation, construction or demolition, or the manufacture or repair of lead containing materials. General environmental exposures from contaminated air, water and food are uncommon but not unheard of. Exotic sources are also reported sporadically including exposures to contaminated folk medications, cosmetics, ingested lead foreign bodies, retained bullets, artists or other hoppy materials, firing ranges illicit distilled alcoholic beverages, and substances (7). We report a case of a 16 years old adolescent boy with Seizure disorder who developed neuropsychiatric manifestations secondary to high levels of lead in the body-the source of lead was found to be from Ayurvedic medications.

Case Report

Master K, 16 year old, single male currently dropped out from school from a semi-urban locality in North India, was brought for treatment of epilepsy and behavioral problems by his mother and sister. There was no family history of neuropsychiatric morbidities. There was past history of delay in isolated personal socio-adaptive mile stones and other features of pervasive developmental disorder like preference for solitary play, abnormal interest in spinning objects during childhood and an unusual interest in electronics. She also gave history of precocious and excessive interest in sexuality from about 6 years of age, poor academic performance since 4th standard, poor conduct in school over the last 3 years evident by lying, stealing and using abusive language and cruelty towards animals from childhood. He had his first episode of seizure in April 2014, semiology was suggestive of generalised tonic clonic seizures (GTCS) with duration of 10 minutes and frequency of one episode per month. He was started on ayurvedic medications from August 2014. This was continued till about June 2015.

During this period, the patient was noticed to gradually develop symptoms of increased irritability, assaultiveness, fear, persecutory and referential delusions with hypersexuality and impaired biological functions along with decreased need for sleep. All these symptoms increased in severity and the patient became assaultive towards family members threatening and attempting to physical and sexually assault his mother and sister. He also started roaming the streets at night which he claimed was secondary to his decreased need for sleep. At this time, the patient was observed to have inattention, forgetfulness, delayed recall and repeated questioning. There is also history of multiple episodes of abdominal pain with constipation, loss of appetite and weight loss in that year. The worsening of symptoms was from August 2014 with maximum symptom severity in November 2014 and continuing till July 2015. On general physical examination, Master

K was moderately built and poorly nourished with body mass index of 19.77. Multiple flat hypopigmented macular lesions suggestive of pigmentary mosacism were seen over the anterior, lateral and posterior aspects of the trunk (as shown in Figures 1 A and B). Central nervous system examination was normal other than for fine tremors of outstretched hands. His mini-mental score (MMSE) was 28 out of 30. Fundus examination did not reveal any abnormality. Other systemic examination was normal. Mental status examination revealed a guarded individual with persecutory and referential delusions.

Figure 1A : Flat hypopigmented macular lesion over the anterior aspect of chest (pigmentary mosacism)

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Patient underwent detailed evaluation both in the departments of psychiatry and neurology. His blood Investigations are as shown in the table below (Table 1):

Table 1: Blood parameters

Investigation	Result	Normal values	Inference
Total counts	10700 /cu.mm	4000-10,000/cu.mm	
Peripheral smear	Hypochromia+, Occasional target cells. Basophilic stippling not present		
Serum Iron	49 ug%	**60-160**	Decreased
Total Iron binding capacity	**453 ug%**	**300-400**	Elevated
Urine Porphyrin	Positive	Negative	Elevated
24 hour 5 delta aminolevulinic acid	6.6 mg	<2	Elevated
Whole blood arsenic levels	20.3ug/L	0 – 13	Elevated
Blood lead levels	**87.3 µg/Dl**	**<10.0**	Toxic level

Radiograph of his knees (Figure 2) showed opaque metaphyseal bands in the lower femur, upper tibia, and upper fibula suggestive of 'lead lines'.

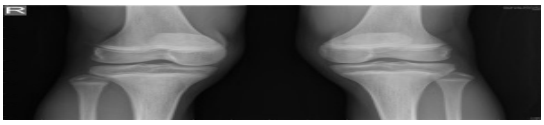

Figure 2 : Opaque metaphyseal band in lower femur, upper tibia, and upper fibula

The patient was started on oral valproate for management of seizures, and also considering its benefits as a mood stabilizer. He was also started on oral olanzapine for his psychiatric symptoms. Following this there was improvement in terms of psychotic symptoms and control of seizures and improvement in biological functions. However he continued to be guarded and hypersexual although overt expressions came down. Following the diagnosis of lead toxicity, he was also given two courses of calcium sodium EDTA 1000 mg in 200 ml normal saline twice a day for 5 days. Interestingly the ayurvedic medications, he was on when subjected to analysis revealed high lead levels.

The family sought a speedy discharge following this due to domestic reasons and lack of care givers. At the time of discharge he did show improvement in his agitation and psychotic symptoms and MMSE revealed a score of 29/30.

Discussion

In the mentioned case, the pointers which made us suspicious of an organic aetiology included early age of onset without a family history, prolonged duration of symptoms, comorbid symptoms of abdominal complaints and seizures. The persistance of mood symptoms following control of epilepsy and the history of use of indigenous medications also prompted us to think beyond epilepsy being the probable organic aetiology. Following chelation therapy, In the short period of hospital stay, the changes initially noticed were mild improvement in symptoms of irritability at the time of discharge from the hospital. Lead poisoning was first recognized as a pediatric disease in Australia over 100 years ago—A series of 10 cases were reported from Queensland in 1892 (8). Children are often the vulnerable population and the sequalae of brain damage caused by chronic low levels of lead exposure is often irreversible and untreatable (9).The mechanism through which lead causes its lethal effects is hypothesized to be its ability to substitute for other polyvalent cations like zinc and calcium. These interactions allow lead to affect different biologically significant processes, including metal transport, energy metabolism, apoptosis,
ionic conduction, cell adhesion, intercellular and intracellular signaling, diverse enzymatic processes, protein maturation, and genetic regulation. Membrane ionic channels and signaling molecules seem to be one of the most relevant molecular targets that contribute to lead's neurotoxicity; the developing central nervous system is particularly susceptible(10). In view of the propensity of lead to cause severe and irreversible brain damage, the United States Department of Health and Human Services Centers for Disease Control and Prevention (CDC) in the 1990's has recommended cut off values of more than 10ug/dl in the human body as unsafe (11). The CDC in the year 1991 in the background of paucity of Community data of Lead poisoning, recommended universal screening for all children aged 9 to72 months. Later, the guidelines have been revised. The present recommendation suggests screening to begin at 9 to 12 months of age and be considered again at 24 months of age of the child. In communities where universal screening is recommended, the physicians are asked to follow that recommendation. In communities where targeted screening

is recommended, a possible history of exposure has to be regularly assessed between 6 months to 6 years of age using community specific risk assessment questions. Blood lead testing also should be considered in abused or neglected children and in children who have conditions associated with increased lead exposure (12).

In India, The George Foundation in Bangalore in 1997 made pioneering efforts to target children between 1 to 6 years of age, pregnant women and emergency referrals from doctors and hospitals for

testing and treating elevated levels of lead—however, there are no clear guidelines to suggest universal screening/at risk screening in the Indian Community.

Conclusion

This case report highlighted our knowledge of the importance of a high index of suspicion for organic causes for psychiatric presentation and also the significance of a heavy metal screening especially in a developing country like ours for more reasons than one. This case report highlights the need for at risk screening of vulnerable population; children with neuropsychiatric manifestations not responding to usual line of management with history of exposure to Ayurvedic Medicaitons are clearly vulnerable and a probable red flag to recommend blood lead level assessment and subsequent management.

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