Lead Poisoning following Traditional Drug Therapy

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Acute CNS Infection / Acute Stroke

❖Background: 5 yr boy 3rd born to non consanguinous parentage and developmentally normal; Hospitalised for enteric fever found to be anemic (Hb 6.8) 2 months back

❖Admitted with history of

- Headache 3 days
- Vomiting 2 days (6-7 times)
- Paucity of movements of Right upper and lower limbs 2 days
- Aphasia and Deviation of angle of mouth to left side 2 days
- Altered level of consciousness 2 days
- Seizures and Fever1 day

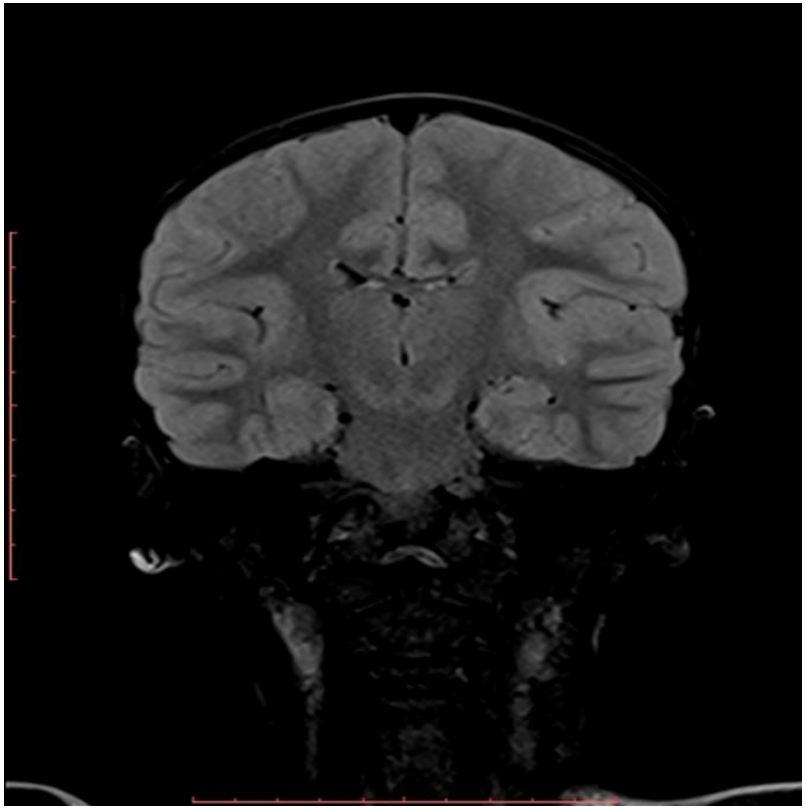
▪He was on Inj.Ceftriaxone and Inj.Acyclovir

Encephalitis + Rt Hemiparesis

- GCS: 11/15
- Rt facial palsy
- Tone: normal
- Power: Rt Upperlimb 2/5; Rt lower limb 4/5. Lt Normal
- DTR : Rt Brisk
- Rt Plantar Extensor
- No meningeal signs
- Spine and cranium normal
- Vitiligo leision in the leg

Diagnostic Dilemma

- He had right focal seizures thrice- Treated with IV lorazepam& Fosphenytoin.
- Became alert after 8 hours of admission and limb power improved dramatically to 4/5
- MRI Brain: Subtle cortical thickening in left parietal & insular cortex without signal enhancement/altered signal intensity.Possible post seizure related changes/ focal encephalitis.
- MRA & MRV: Normal
- CSF: Traumatic, WBC-11,N30 L70, RBC-1700, G- 63, P-126,Cl-123
- CSF Viral markers: Negative



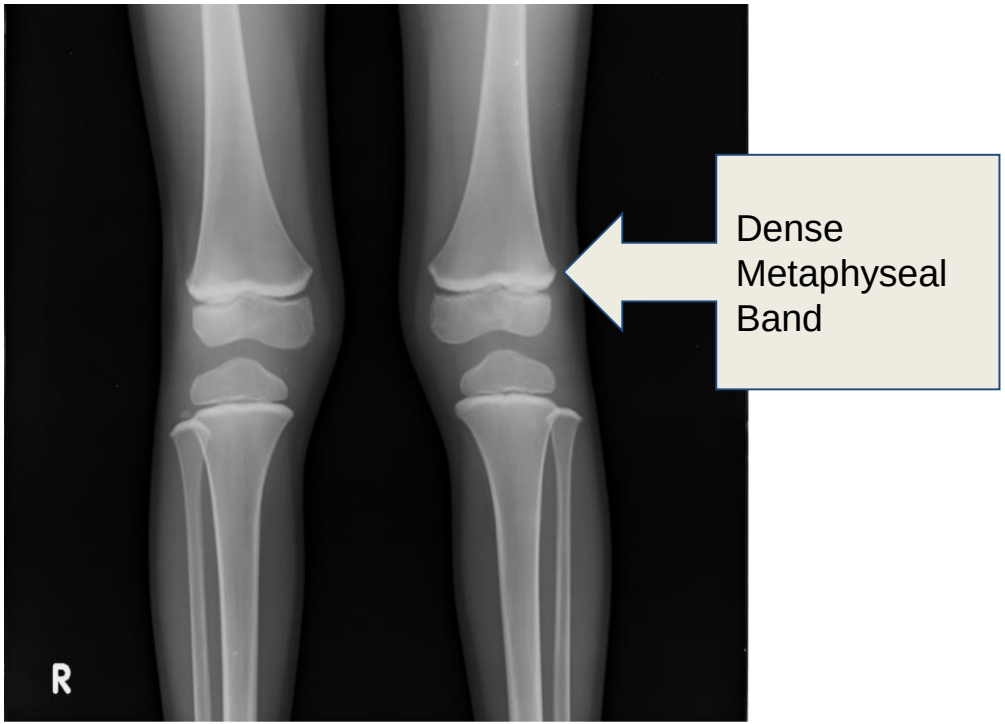
Haemolytic Anemia

- PS: Microcytic Hypochromic Anisopoikilocytosis, Target,Tear drops & Elliptocytosis

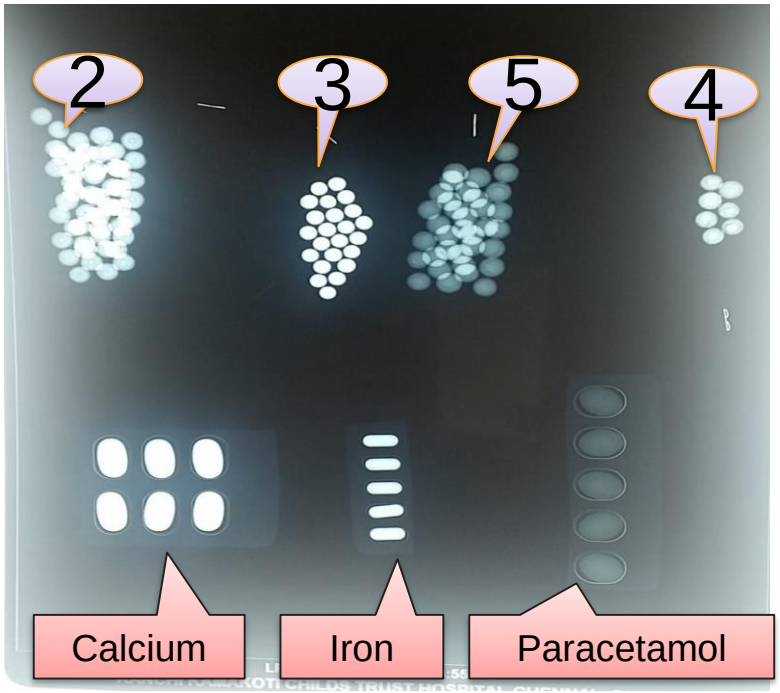
Hb	MCV	MCH	RDW	RBC	Iron	TIBC	Ferritin	Retic	DCT	Urine Hb	Sickle Test	Myco IgM	BLL
6.8	77	23	17.1	2.9	46	292	126	10%	Neg	Nil	Neg	Neg	>100

- H/o Ayurvedic Drug intake for skin lesions for 1 ½ yrs
- Neurologist Opinion: To R/O Plumbism

X ray Both Knees - Lead Lines



Lead Level in Traditional Drugs



X-Ray evidence of Heavy Metals in various medications

National Referral Centre for Lead Projects - India (NRCLPI)

LEAD (Pb) ANALYSIS REPORT

Date – 12-06- 2017

	Sample Name	Pb (ppm)	Pb +/-	Pb Detected
1	YG-60	111	8	Detected
2	ABB-60	649	33	Detected
3	KKS-60	148	36	Detected
4	KKS-30	ND	ND	Not Detected
5	KG-60	71	8	Detected
6	RTO-30	31	5	Detected
7	TRI-30	99	7	Detected
8	BLN-30	30	5	Detected

ND- Not Detected

Reference Range: 0- 10 ppm

The above tests has been carried out by X Ray Fluorescence Technology instrument named FP XRF - Field Portable X Ray Fluorescence.

Note-Any amount of lead in medicines and food is dangerous.

NRCLPI Bangalore 560 034 SJMC

For NRCLPI

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Chelation

- Chelation was started with D-Penicillamine 10mg/kg/day due to unavailability of other agents
- Follow up – BLL 27 mcg/dl – Stopped Chelation

TABLE

Oral Chelators for Childhood Lead Poisoning

Chelator	Dosing Recommendations	Adverse Effects	Monitoring Recommendations
d-PCN	10 mg/kg/dose every day for 7 days, then 10 mg/kg/dose twice a day; maximum dose 30 mg/kg/day	Urticarial rash, GI upset, leukopenia, thrombocytopenia, proteinuria/hematuria	BPb, ZPP, CBC, platelet count, urinalysis, BUN, creatinine pretreatment, then every 2 weeks for 1 month, then monthly until treatment stops
DMSA	10 mg/kg/dose three times a day for 5 days, then 10 mg/kg/dose twice a day for 14 days	GI upset, AST/ALT elevation, rash, neutropenia	BPb, ZPP, AST, ALT, CBC pretreatment, then after 2 weeks on therapy and 2 weeks after therapy

Abbreviations: d-PCN = d-penicillamine, GI = gastrointestinal, BPb = blood lead, ZPP = zinc protoporphyrin, CBC = complete blood cell count, BUN = blood urea nitrogen, DMSA = 2,3-dimercaptosuccinic acid, AST = aspartate aminotransferase, and ALT = alanine aminotransferase.

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Case Report

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LEAD POISONING PRESENTING AS A MIMIC OF ACUTE STROKE

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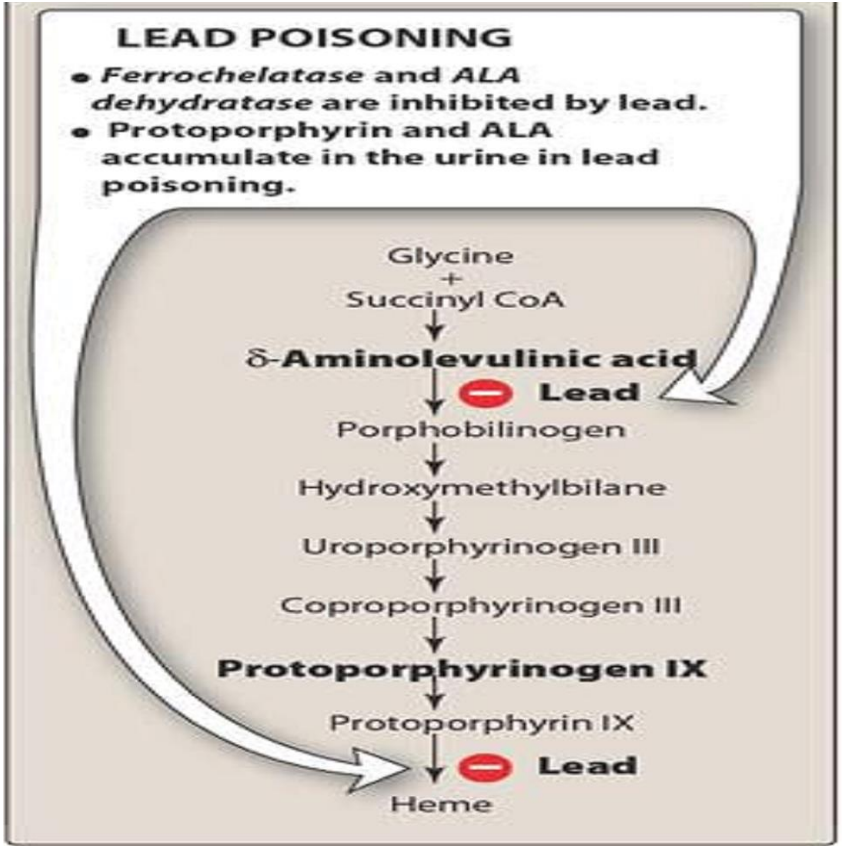
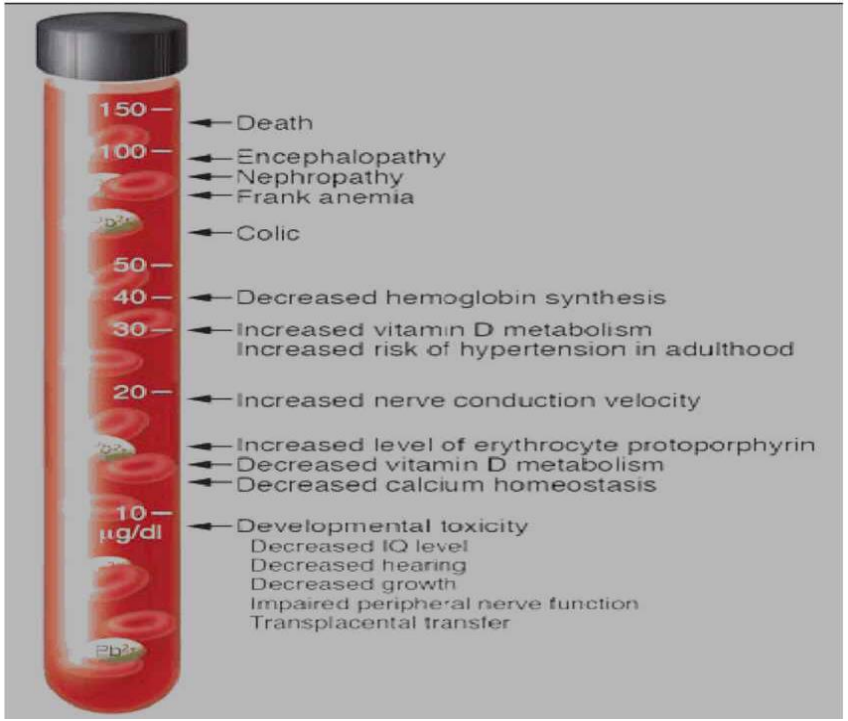

LEAD POISONING
• Ferrochelatase and ALA dehydratase are inhibited by lead.
• Protoporphyrin and ALA accumulate in the urine in lead poisoning.
Glycine
+
Succinyl CoA
δ-Aminolevulinic acid
Lead
Porphobilinogen
Hydroxymethylbilane
Uroporphyrinogen III
Coproporphyrinogen III
Protoporphyrinogen IX
Protoporphyrin IX
Lead
Heme

Clinical Manifestation at various BLL

- Basophilic Stippling: Inhibition of pyrimidine 5'-nucleotidase can prevent the degradation of ribosomal RNA in RBC - at BLLs of ~50 µg/dL
- Anemia develops at >80 µg/dL
- Children are at much greater risk for developing lead encephalopathy at lower lead levels (>70-90 µg/dL) than adults (>150 µg/dL)
- Zn protoporphyrin reflects the last 6-8 weeks of exposures(< 35 µg/dL)



Cochrane - Lead Encephalopathy

- Data collected from Cochrane library from 1966 to 2007 reported 76 cases of Lead encephalopathy, 5% were in adults and 95% were in infants and young children
- In infants and young children, among 72 cases 8 (11%) were fatal, and at least 15 (21%) had residual neurological deficits

Take Home Message

- Lead Poisoning through herbal supplements/treatment still remains a public health problem
- It is the responsibility of the physician to obtain a detailed history of traditional medicine intake and be aware of its harmful effects
- Lead level analysis should be done in the blood and traditional medicines
- The Central Drug Standard Control Organization of India (CDSCO) should vigilantly check the heavy metal levels in all traditional medicines before licensing it for therapeutic use

Don’t be misLEAD by Traditional medicine

Thanks!

Neurologist: Dr.V.Viswanathan
Hematologist: Dr.Deenadhayalan