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Original Research Paper

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CRITICAL CARE MANAGEMENT OF A CASE OF ACUTE LEAD POISONING - A CASE REPORT

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ABSTRACT **Background:** Humans have been using LEAD in various forms for the day to day requirements. Many factories use different forms of lead and workers who have a chronic exposure in such factories are prone for lead poisoning. They may remain aympotomatic over a long period and exhibit symptoms when blood with lead. Here in this case report we have discussed the acute lead poisoning management in critical care setup. **Case Report:** 38 year old male came with non specific complaints came to ER . Later diagnosed as lead poisoning , from Occupational hazard with elevated methemoglobin and serum lead values. He was found to have end stage renal disease and was started on hemodialysis for the same with symptomatic treatment. **Conclusion:** Acute management in critical care for lead poisoning is patient specific. Not all patients need chelating therapy for lead poisoning in critical care setup. Symptomatic treatment and long term follow up defines a cure for it

KEYWORDS : Lead , methemoglobinemia , chelating therapy , hemodialysis

INTRODUCTION

Lead became a common occupational toxin with its extensive use in paint industry . It is not biodegradable and has remarkable environmental persistence. Due to this nature of lead , the toxic level in blood produces varied symptoms when each organ in the body is affected. Early diagnosis and treatment in critical care management will reduce morbidity and mortality in patients . Not all patients require chelating therapy in a setting of acute lead poisoning. Thus in this case report we have discussed the acute lead poisoning management in critical care setup.

CASE REPORT

A 38 year old male came to ER with complaints of nausea, vomiting, abdominal pain for 4 days. Vomiting was non projectile around 4 episodes per day and the vomitus contained food particles with occasional bile stain. Abdominal pain was dull aching , diffuse and not radiating. No History of loose stools, abdominal distension or constipation. No History of fever, cough and sore throat . No other significant comorbidities.

Normal bowel and bladder habits. Lack of appetite due to nausea and vomiting. Disturbed sleep due to abdominal pain. Occasional smoker and alcoholic : Last drink and smoke one month back.

Occupational history: Works In a factory with exposure to Lead. 5more patient came to ER with similar complaints from same factory.

On examination: Patient was conscious, oriented, afebrile , dehydrated . Pallor present, Icterus present. No clubbing, pedal edema, ascites , lympadenopathy.

PR : 100/min BP: 108 / 76mm hg MAP : 68
GCS : 15/15 Temperature: 98F
Sp02 : 85% on room air .
Systemic examination:
Cvs: S1 S2 heard , no murmurs
RS : Bilateral air entry heard , no wheeze or crepitus.
PA : soft , no tenderness, no organomegaly on palpation. CNS: NFND

On arrival ABG was done in room air : pH : 7.283
PCo2 : 49 mm hg P02: 90.2 mm hg Na: 128 K : 5.8 Lactate : 3.8
Hb : 5 MetHb : 22.8%
Hco3: 16

Blood investigation: Hb: 3.1 TLC : 11570
Platelet count : 1.73L Urea : 422 Creatinine: 11.5
T.B : 6.70 D.B : 4.92 SGOT : 397 SGPT: 144 Albumin : 1.0 Na: 133 K: 5.8
Diagnosis: Acute Lead poisoning- Methemoglobinemia/ Severe Anemia/ Acute on chronic CKD

Patient was shifted to ICU for further management .

CRITICAL CARE MANAGEMENT
On receiving in ICU : Patient was put on NIV with Fio2 of 100% maintaining SP02 OF 94%

Patient was secured with left IJV hemodiaylsis catheter and planned for Hemodiaylsis.
HRCT thorax : Moderate cardiomegaly, atelectasis in basal segment of Bilateral lower lobes

ECHO : EF: 65% concentric LVH
Other blood investigations were done which included: LDH: 5016 Ferritin: 773 uric acid: 4.5 Vit D : < 8.0 Ca: 8.6 phosphorus: 6.8 Serum Magnesium : 2.8.
Sr.Iron: 175
SERUM LEAD LEVEL: 95mcg/dl
Total iron binding capacity : 254ug/dl
Reticulocyte count : 6000
Peripheral smear: Basophilic stipling seen in RBC - normocytic and microcytic anemia . Fragmented RBC seen.

Nil output in Icu prior to HD .Patient was started on heparin free HD immediately on shifting to ICU and blood investigations were followed up with PRBC transfusion.

After 6 hours of HD , Patient had respiratory distress with RR : 40/min and planned for intubation. Despite equal air entry and adequate EtCo2 his saturation was 90% . Hemodiaylsis continued, RFT and other blood investigation were monitored

serially. Patient was put on CPAP and eventually extubated after 3 days. Patient couldn't be started on chelating therapy as he already progressed to END STAGE RENAL FAILURE.

Treatment Given

Inj. Meropenam 500mg bd
Tab.Sodium bicarbonate 500mg tds
Tab Urodeoxycholic acid 300mg bd
Inj.Pantoprazole 40 mg Od
Neb. Salbutamol 6hrly
Inj.Ondensetron 4mg bd
Inj.Recombinant human erythropoietin 4ooounits SC
Calcium correction and haemoglobin correction was done following serial blood report management. Supportive treatment was given .

After 5 cycles of HD : Urea 100, Creatinine: 6.6 , HB: 8.6 , ABG

METHEMOGLOBIN LEVEL: 3.0
Serum Lead level: 25mcg/dl. Patient was transferred to nephrology ward for further management.

DISCUSSION

Humans have been using Lead for variety of applications since millennia and Lead at minor levels is accepted in blood . 70mcg/dl of Lead in blood causes severe symptoms. 100mcg/dl Lead interacts with calcium and interferes with its mechanism. It affects various organs.

CNS : Peripheral neuropathy features , seizures , increased intracranial pressure and encephalopathy.
HEMATOLOGICAL : Anemia , Basophilic stipiling .
RENAL: uremia, increased uric acid production: SATURNINE GOUT , hypertension , renal failure
GI: abdominal pain, constipation , anorexia and vomitting .

Lead poisoning is mainly managed by removing the source. Chelating agents such as EDTA, BAL, calcium disodium can be used. Chelating therapy combined with Hemodialysis causes reduction in serum lead levels by 96hours. Chelator specific adverse effects causes further aggravation of renal derangements , hypertension, tachycardia and aggravates GI symptoms.

In our case report , patient had ended with end stage renal disease induced by lead, therefore chelating therapy could not be started. Symptomatic management with hemodialysis and other blood parameter correction was given. Acute management of such cases in critical care plays a vital role in reducing the morbidity and mortality of the patient.

CONCLUSION

Management of Lead poisoning shows a wide array of treatment modalities. A patient who presented with renal failure induced by lead toxicity can be treated with adequate hemodialysis without chelating agents and adequate supportive treatment with serial blood parameter monitoring.

REFERENCES

1. Mu Muntner P, Menke A, DeSalvo K, Rabito F, Batuman V. Continued decline in blood lead levels among adults in the United States: the National Health and Nutrition Examination Surveys. Arch Intern Med. 2005;165:2155–61.Doi: 10.1001/archinte.165.18.2155. [PubMed] [CrossRef] [Google Scholar]
2. Flora G, Gupta D, Tiwari A. Toxicity of lead: a review with recent updates. Interdiscip Toxicol. 2012;5:47–58. Doi: 10.2478/v10102-012-0009-2. [PMC free article] [PubMed] [CrossRef]
3. Ahamed M, Siddiqui MK. Low level lead exposure and oxidative stress: current opinions. Clin Chim Acta. 2007;383:57–64. Doi: 10.1016/j.cca.2007.04.024. [PubMed] [CrossRef] [Google Scholar]