CASE STUDY

Patients presenting with Gross Renal and Hepatic Impairment after exposure to lead

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ABSTRACT
Leadis a metal exists in both organic & inorganic forms. Lead toxicity occurs due to occupational & environmental exposure especially in developing countries.
The ways of contamination include ingestion, inhalation, prenatal exposure and dermal exposure. The clinical presentation of toxicity involves nervous, hematologic and renal system with symptoms like ataxia, convulsions, hemolysis, anemia, glycosuria, nephritis, etc. So, it causes potential multi organ damage if present in large amount in blood. In our case series we studied seven patients who came after occupational exposure to lead with severe renal and hematological symptoms. All patients were discharged after normal renal function tests and hematological tests.
Keywords: Lead poisoning, Abdominal pain, Anemia, Hemolysis.

INTRODUCTION

Lead is a soft pliable bluish grey metal resistant to corrosion, that exists in both organic and inorganic forms. The metal doesnot conduct electricity and has its own anti-radiation properties(1,2,3). The toxicity of lead has been known for more than 1000 years, but occupational and environmental lead poisoning occurs endemically especially in developing countries. While there have been many reports about environmental pollution by lead related to battery industries. The victims are mostly confined to workers, families or residentsespecially children of nearby communities (4).

The ways of contamination include ingestion, inhalation, prenatal exposure and dermal exposure but the most important and frequent ones are ingestion and inhalation (5).

The half-life of lead is between 30-40 days in men while in children and pregnant women it can be longer. It binds to sulfhydryl group of proteins leading to toxicity for multiple enzyme systems.The clinical presentation of lead poisoning involves nervous, hematologic and renal system impairment but it can also lead to gastrointestinal disorders (Anorexia, vomiting, constipation, abdominal pain), hypertension and fertility impairment (6,7).

Neurological symptoms include ataxia, stupor, coma, convulsions, hyperirritability, reduced IQ, shortened attention span, increased antisocial behaviour, reducing educational attainment and even death. Impairment of hematological system may involve either disruption of heme synthesis or hemolysis leading to anemia with its specific clinical signs like weakness and fatigue (5).

The effects of lead on renal system consists of proximal tubular function impairment leading to aminoaciduria, glycosuria and hyperphosphatemia, interstitial nephritis on chronic exposure and also impairment of calcium metabolism with activation of vitamin 1,2 dihydroxycholecalciferol (6).

The diagnosis established on blood lead level of more than 40ug% suggestive of occupational exposure and blood lead levels of more than 30ug%suggestive of nonoccupational exposure.

Case 1: 24 yrs male R/o Gangapur, Bhopal working in battery industry came with history of loss of consciousness 2 to 3 days back, vomiting and decrease urine output since 2 to 3 days. On admission patient's GCS was full, vitals were normal. Investigations: Hb-5.7gm%, TLC-5,800, Urea-224mg%, Creatinine-9.05mg%, LDH-4196 U/L, CRP-111.22mg%, PCT-76.88 ng/dl, T. Bilirubin-2.0mg%, SGOT-185 U/L, SGPT-69 U/L, CXR

showed blunting of B/L CP angles with haziness over B/L lung fields most likely pleural effusion. Lead levels were 28ug/dl. Patient received multiple antibiotics, blood transfusions and also received 15 hemodialysis sessions during his hospital stay of 18 days. On discharge patient was self-voiding with adequate urine output. On discharge Hb was 9.1gm%, Urea-141mg%, Creatinine-5.97mg%, CRP in normal range, T. bilirubin 0.5mg%, SGOT-16 U/L, SGPT-18 U/L. Patient came for serial follow ups and after one month Urea was 15mg% and Creatinine 0.93mg%.

Case 2: 52 years male R/o Gangapur Bhopal working in battery industry came with history of hematuria, pain in abdomen, nausea and vomiting since 1 to 2 days. On admission patient's GCS was full, vitals were normal. Investigations Hb-6gm%, TLC-10600, Urea-222mg%, Creatinine-8.10mg%, LDH-4210 U/L, CRP-58.22mg%, PCT-69.66 ng/dl, T. bilirubin-3.6 mg%, SGOT-263 U/L, SGPT-47 U/L. CXR showed blunting of only right CP angle with ill-defined opacity in right lower zone most likely pleural effusion with consolidation right side. Lead levels were 30ug/dl. Patient was anuric from 2nd day of admission for next 20 days. Patient received multiple antibiotics, blood transfusions and also received 20 hemodialysis sessions during his hospital stay of 42 days. On discharge patient was self-vioding with adequate urine output. On discharge Hb was 9.2gm%, Urea = 191mg%, Creatinine = 5.92mg%, CRP in normal range. LFT's were in normal range. Patient came for serial follow ups and after one month Urea was 79mg% and Creatinine 1.77mg%.

Case3:20 years male R/o Gangapur Bhopal working in battery industry came with history of fever, vomiting and pain in abdomen since 2 to 3 days and anuria since 1day. On admission patient's GCS was full, vitals were normal. Investigations Hb-4.8gm%, TLC = 27500, Urea-161mg%, Creatinine-5.61mg%,LDH-3183 UL, SGPT-74 U/L. CXR NAD. Lead levels were 36 ug/dl. Patient received multiple antibiotics, blood transfusions and also received 16 hemodialysis sessions during his hospital stay of 31 days. On discharge patient was self-voiding with adequate urine output. On discharge Hb was 9.9gm%, Urea – 209mg%, Creatinine -4.65mg%, CRP in normal range. LFT's in normal range. Patient came for follow up and at that time urea was 194mg%, and creatinine 3.38m%.

Case 4: 26 years male R/o Gangapur Bhopal working in battery industry came with history of fever, pain in abdomen and black coloured stools since 2 to 3 days. On admission patient's GCS was full, vitals were normal. Investigations Hb -5.1gm%, TLC-16500, Urea-199mg%, Creatinine-4.94mg%, LDH-3451 U/L, CRP -52.88 mg%, PCT-76.22ng/dl, T.bilirubin 2.3mg%, SGOT-339U/L, SGPT-91 U/L. CXR NAD. Lead levels were 34ug/dl. Patient developed anuria within 2 to 3 days of admission. Patient received multiple antibiotics, blood transfusions and also received 13 hemodialysis sessions during his hospital stay of 31 days. Patient started pouring urine after 20 days of admission. On discharge patient was self-vioding with adequate urine output On discharge Hb was 9.2gm%, Urea-197mg%, Creatinine-3.20 mg%, CRP in normal range. LFT's in normal range. Patient came once for follow up and at that time urea was 186 mg%, and creatinine 3.04mg%.

Case 5:23 years male R/o Gangapur Bhopal working in battery industry came with accidental inhalation of some gas at the industry with anuria since 2 days, redness of eyes, giddiness and vomiting. On admission GCS full, vitals stable. On investigations Hb 4.7gm%, TLC-16300, Urea 155mg%, Creatinine 5mg%, LDH 4842.1U/L, CRP 160.2mg%, PCT 18.6ng%, T. Bilirubin 3.6mg%, SGOT-189 U/L, SGPT-17 U/L, Lead 12.53ug/dl. He underwent 12 cycles of hemodialysis. On discharge patient was self-voiding with adequate urine output. On discharge Hb 9gm%, Urea 124mg%, Creatinine 1.7mg%, SGOT 116 U/L, SGPT 60 U/L, T. Bilirubin 1.30mg%

Case 6: 24 years male R/o Gangapur Bhopal working in battery industry came with history of hematuria and decreased urine output. On admission GCS full, Vitals stable. On investigations Hb 11.2mg%, Sodium 136meq/L, K+ 4.4meq/l, TLC 17200, Urea 45mg%, Creatinine 0.8mg%, T. Bilirubin 6.3mg%, SGOT 76 U/L, SGPT 19 U/L, LDH 1449.4 U/L, Lead 8ug%. No hemodialysis done. Patient responded to IV fluids and injection Furosemide. Patient discharged after 6 days with self-voiding urine and near normal investigations.

Case 7: 24 years male R/o Gangapur Bhopal working in battery industry came with history of hematuria and decreased urine output. On admission GCS full, Vitals stable. On investigations Hb 9.2mg%, Sodium 134meq/L, K+ 4.3meq/l, TLC 14600, Urea 55.2mg%, Creatinine 4.1mg%, T. Bilirubin 15mg%, SGOT 119 U/L, SGPT 46 U/L, LDH 2356 U/L, Lead 13.63ug%. Three cyclesof hemodialysis done along with two PRBC transfusions. Patient discharged after 7 days with self-voiding urine and near normal investigations.

LABORATORY INVESTIGATIONS

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7
Lead (ug/dl)	28	30	36	34	12.53	8.0	13.63
Hb (gm%)	5.7	6.0	4.8	5.8	4.7	11.2	9.2
Na (mmol/l)	135	136	136	138	136	136	134
K (mmol/l)	3.5	3.94	5.6	4.93	5.2	4.4	4.3
CRP (mg%)	111.22	58.22	50.39	52.88	160	50.8	52.6
PCT (ng/dl)	76.88	69.66	88.85	76.22	18.6	18.8	19.3

Urea (mg%)	224	222	161	199	155	45	55
Creatinine (mg%)	9.05	8.10	5.61	4.94	5.0	0.8	1.0
T. Bilirubin (mg%)	2.0	3.6	4.2	2.3	3.6	6.3	15
SGOT (U/L)	185	263	573	339	189	76	119
SGPT (U/L)	69	47	74	91	17	19	46
LDH (U/L)	4196	4210	3183	3451	4842	1449	2356

CLINICAL SYMPTOMS

	Case 1	**Case 2**	**Case 3**	**Case 4**	**Case 5**	**Case 6**	**Case 7**
Anuria	2-3 Days	-	1 Day	-	2 Days	2-3 Days	2-3 Days
Fever	-	-	2-3 days	2-3 Days	-	-	-
Vomiting	2-3 Days	1-2 Days	2-3 Days	-	2 Days	-	-
Pain in abdomen	-	1-2 Days	2-3 Days	2-3 Days	-	-	-
Hematuria	-	1-2 Da ys	-	-	-	-	-
Black colour stool	-	-	-	2-3 Days	-	-	-
Redness of eyes	-	-	-	-	2 Days	-	-
Giddiness	-	-	-	-	2 Days	-	-

DISCUSSION

All the cases in our study were from battery industries which were near Gangapur Bhopal. They presented with anuria, fever, vomiting, pain in abdomen, hematuria, redness of eyes, black colour stools and giddiness.

On lab investigations all of them had severe anemia, deranged liver function test, renal function test, raised CRP, PCT levels. All patients except one underwent multiple cycles of hemodialysis with PRBC transfusions.

Lead poisoning is a seriouscondition with potential multiorgan damage and even death if lead is present in large amounts in the blood stream, representing a major health problem.

The control of lead at work prescribes regulations of safety limits. Blood lead level <1.45ug/dl represents reasonably controlled levels and controlled occupational exposure provided there is 6 monthly monitoring.Levels 1.45 to 2.4 ug/dl requires investigation, action by the employer and levels 2.4 to 2.9 ug/dl calls for suspension of worker from the exposure(1,4)

CONCLUSION

In our case series we studied seven patients there was no mortality and all patients were discharged with near normal investigations.

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