Original Research Article

Creatine phosphokinase, lactate dehydrogenase and total leukocyte count during treatment for oral hair dye poisoning

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International Archives of Integrated Medicine, Vol. 3, Issue 7, July, 2016.

Available online at http://iaimjournal.com/
ISSN: 2394-0026 (P) **ISSN: 2394-0034 (O)**
Received on: 08-06-2016 **Accepted on:** 21-06-2016
Source of support: Nil **Conflict of interest:** None declared.
How to cite this article: Pallapothula N, Reddy BS. Creatine phosphokinase, lactate dehydrogenase and total leukocyte count during treatment for oral hair dye poisoning. IAIM, 2016; 3(7): 72-76.

Abstract

Introduction: Paraphynylenediamine (PPD) in hair dye causes angioneurotic edema leading to acute respiratory distress, rhabdomyolysis, i.e. necrosis of skeletal muscle resulting in acute renal failure and liver injury upon ingestion. We studied the time course of treatment on organ damage markers such as plasma Creatine phosphokinase (CPK), Lactate dehydrogenase (LDH) and Total leukocyte count (TLC) of these patients till discharge.

Materials and methods: We reviewed the case records of 10 Patients of suicidal hair dye poisoning. Data was collected in pre-specified data collection forms regarding WBC count, CPK levels, age, gender, time to index admission, ventilator support, the volume of ingestion and outcome. We followed the values of CPK and TLC of these patients till discharge.

Results: The average age was 25.8±10.5, the mean volume of consumption of hair die was 102.5±17.5, and time to reach emergency room was 7.6±3.2 hours. On arrival, all patients were intubated and received gastric lavage, antihistamines, parenteral steroids, and sodium bicarbonate. Duration of ventilator support was 2.9±0.7 days and length of hospital stay was 10.1±2.7 days. At admission, CPK, TLC, and LDH were elevated, during hospital stay values CPK, LDH and TLC gradually decreased by day-5 after that they showed a marginal increase by day-7.

Conclusion: We observed significantly elevated levels of CPK, LDH and TLC at index admission after oral ingestion of hair dye suggesting muscle, hepatic and hematological injury. The rate of decline of these values (recovery) is parallel.

Key words

Creatine phosphokinase (CPK), Lactate dehydrogenase, Total leukocyte count (TLC), Hair dye poisoning, Paraphenylenediamine (PPD), Rhabdomyolysis.

Introduction

Hair dyes and their ingredients have moderate to low acute toxicity. Human poisoning accidents are following oral ingestion. Oral consumption of hair dye has become one of the leading causes of poisoning in South India, next to organophosphorus poisoning. It is usually consumed by young females of the poorer class, with suicide intention, due to easy availability in the market [1]. The principal adverse effect of high acute doses of its most toxic ingredient para-phenylene diamine (PPD) in man is angioneurotic oedema leading to acute respiratory distress; also, rhabdomyolysis, i.e. necrosis of skeletal muscle resulting in acute renal failure and liver injury [2]. Creatine phosphokinase (CPK) is a marker signifying muscle injury arising from myofibril disruption in rhabdomyolysis [3]. Various studies [4-6] have shown an elevated total leukocyte count, lactate dehydrogenase (LDH) and CPK count from the blood samples taken at index admission. We studied the time course of treatment on organ damage markers such as plasma CPK, Lactate dehydrogenase (LDH) and Total leukocyte count (TLC) of these patients till discharge.

Materials and methods

We reviewed the case records of 10 patients of suicidal hair dye poisoning. The present study was done at ACSR Government Medical College, Nellore for the period of two years from May 2014 to May 2016. IEC has approved the study protocol. Data entered in pre-specified data collection forms regarding WBC count, CPK levels, age, gender, time to index admission, ventilator support, the volume of ingestion and outcome. We followed the values of CPK and TLC of these patients till discharge.

Statistical analysis

Data was entered into excel sheet and tabulated using pivotal tables. Data was described as a mean and standard deviation. ANOVA was used for significance. Pearson correlation analysis verified the correlation. A two tailed p value less than 0.05 was considered significant.

Results

A total of 10 patients' case records with oral hair dye poisoning were followed until their discharge. The classical features of hair dye poisoning such as cervicofacial edema with the hard protruding tongue was observed in all patients at admission. There were eight females and two males. All of them belonged to a rural area and consumed oral hair dye with suicidal intention. The average age was 25.8±10.5 years, the Mean volume of consumption of hair die was 102.5±17.5 mL, and time to reach emergency room was 7.6±3.2 hours. On arrival, all patients were intubated and received gastric lavage, antihistamines, parenteral steroids, and sodium bicarbonate. Duration of ventilator support was 2.9±0.7 days and length of hospital stay was 10.1±2.7 days. At admission, CPK, TLC, and LDH were elevated, during hospital stay values CPK, LDH and TLC gradually decreased by day-5 after that they showed a marginal increase by day-7 (**Table – 1**, **Table – 2**, **Figure - 1**).

Discussion

In India, hair dyes contain PPD along with other ingredients [7]. PPD commonly mixed with henna and traditionally applied to colour palms of hands & to dye the hairs [8]. Paraphenylene diamine and a mixture of other chemicals can damage the respiratory, muscular, renal and hepatic systems and cause death. In this study, we observed increased CPK, LDH, and TLC suggesting that PPD might have resulted in muscle, hepatic and haematological injury. Creatine phosphokinase (CPK) is an enzyme in the body. Elevated CPK often means there has been injury or stress to muscle tissue, the heart, or the brain. Muscle tissue injury is most likely.

When a muscle is damaged, CPK leaks into the bloodstream [9, 10]. CPK concentration was a marker of muscle damage, but may not have necessarily reflected the amount of structural damage [11]. Leukocytosis during oral hair dye poisoning could be due to multiple mechanisms such as oxidative stress [12], repair of damaged tissues [13] and/or activation by cytokines [13]. Change in CPK has the strongest relationship seen with IL-6 and G-CSF [14]. IL-6 inhibits the release of the pro-inflammatory cytokines IL-1beta and TNF-alpha and stimulates other anti-inflammatory cytokines such as IL-1ra and IL-10, which mobilises and augments neutrophil function. G-CSF may support the anti-inflammatory actions of IL-6 by inducing its release and inhibiting the release of IL-1beta and TNF-alpha [13]. In our study we found a significant correlation between CPK and TLC. IL-8 is a potent neutrophil chemotactic carries neutrophil to metabolically active muscle tissue after injury to aid in repair [13]. In our study, LDH raise was in parallel with CPK. It is possible that hepatic damage [6] occurs during oral hair dye poisoning. Additionally, hepaticmarker enzymes, such as LDH, ALT, and AST, are found in both liver and muscle cell [15].

Table – 1: Clinical profile and outcomes of oral hair dye poisoning.

Clinical profile and outcomes of oral hair dye poisoning	**Mean**	**SD**
Age	25.8	10.5
Gender	Female=8	Male=2
Economic status	Mddle-5	Low-5
Consumption of oral hair dye (ml)	102.5	17.5
Time to ER (hours)	7.6	3.2
Length of hospital stay (days)	10.1	2.7
Duration of ventilator (Days)	2.9	0.7

Table – 2: Time-course effects of treatment on CPK, TLC, and LDH.

Days	**CPK (U/L)**	**SD**	**TLC (cells/mm^3)**	**SD**	**LDH (U/L)**	**SD**
1	20940.00	14277.06	21640.00	5655.35	4383.60	1936.94
3	9445.00	9546.41	15400.00	565.69	1750.67	979.57
5	734.50	331.63	12200.00	1131.37	800.00	0.00
7	1587.00	1961.51	14800.00	4489.99	831.00	1033.79
P value	P<0.0001		P<0.0001		P<0.0001	

Conclusion

We observed significantly elevated levels of CPK, LDH and TLC at index admission after oral ingestion of hair dye suggesting muscle, hepatic and haematological injury. The rate of decline of these values (recovery) is parallel. Early treatment restores organ functions to normalcy and prevents complications. Additionally, antioxidants such as N-acetyl cysteine may be tried to reduce the morbidity.

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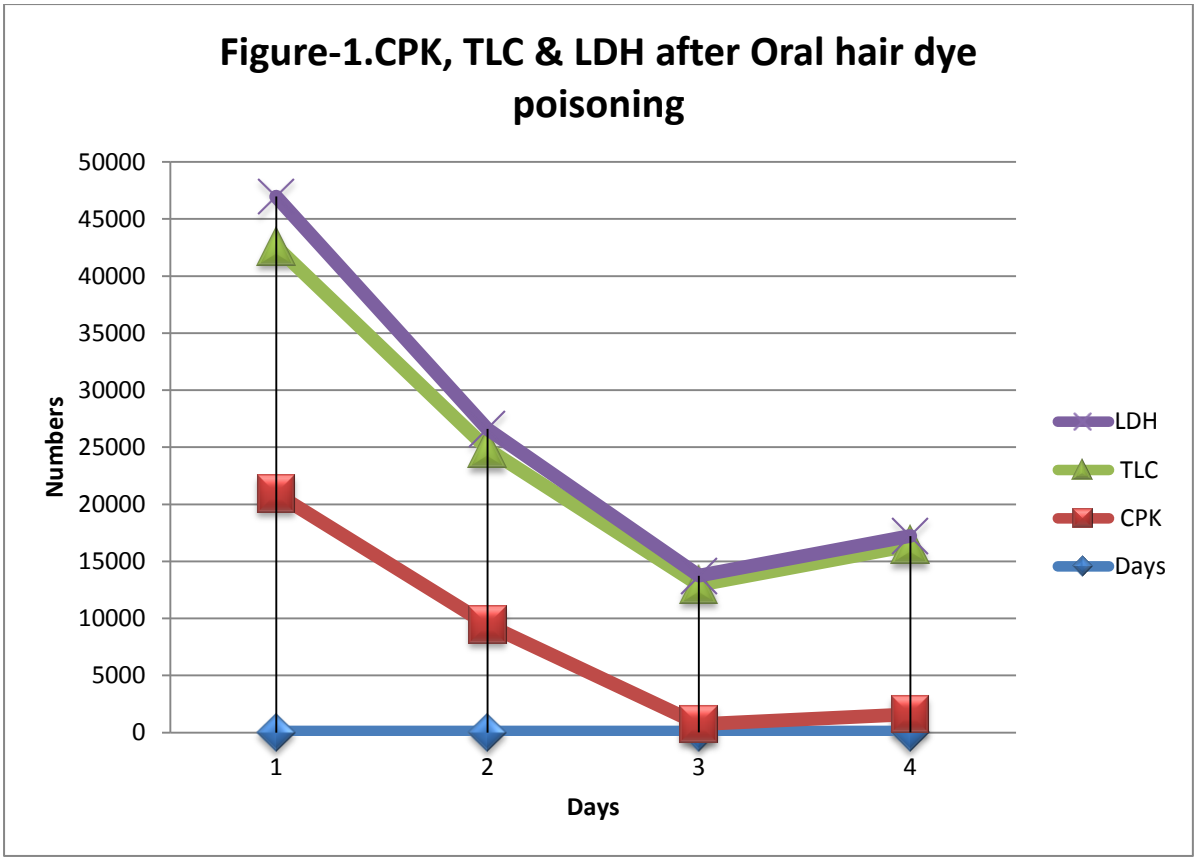
Figure-1.CPK, TLC & LDH after Oral hair dye poisoning
50000
45000
40000
35000
30000
25000
20000
15000
10000
5000
0
Numbers
1
2
3
4
Days
LDH
TLC
CPK
Days