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Hair dye poisoning and rhabdomyolysis

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Abstract

Hair dye ingestion is a rare cause of toxicity in Pakistan. We are presenting the case report of a 55 year old male who presented with accidental hair dye ingestion and developed laryngeal oedema requiring emergent tracheostomy. He had also developed aspiration pneumonitis and chemical oesophagitis. However, the most alarming manifestation was rhabdomyolysis. Hair dye toxicity can be fatal if not recognized early. There is no antidote available. Rhabdomyolysis is a complication and needs to be managed aggressively in order to prevent long term morbidity.

Keywords: Hair Dye, Rhabdomyolysis, morbidity.

Introduction

Hair dye poisoning has been reported from the developing world particularly from Africa and Asia. Most of the incidents reported were those of deliberate ingestion with suicidal intent. In a case series from India 23.92 % died during treatment.¹

Although there have been several case reports and case series from India, indicating it to be a major emerging problem in that part of the world, there has been only one case report from Pakistan to date which was a case of a young man with suicidal attempt.² As there is widespread usage of hair dyes in South East Asia, it is imperative to understand the clinical implication of accidental and suicidal ingestion of hair dyes. Since it is an under recognized and perhaps under reported problem in Pakistan, we feel that our case report will help to create awareness on the potential life threatening properties of hair dye.

Case Report

This is the case of a 55 years male who presented with accidental hair dye ingestion in July 2011. Around 2 hours after the ingestion, he developed acute respiratory distress with facial oedema and plethora along with stridor. He underwent emergent tracheostomy in a nearby hospital after which he was transferred to a

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Table-1:

	Day 1	Day 3	Day 5	Day 7	Day 15	Day 21
Blood urea (mg/dl)	47	86	95	75	62	49
Creatinine (mg/dl)	4.4	6.3	7.4	7.5	10.1	7.9
Sodium (mEq/L)	149	134	142	140	133	146
Potassium (mEq/L)	5.3	3.6	4.3	3.8	3.5	4.6
CPK	38837	7948	3398	1603	233	

CPK- Creatine phosphokinase.

Table-2:

	3 weeks + 3 days	4 weeks + 5 days	5 weeks	5 months	1 year
Creatinine (mg/dl)	6.1	4.5	3.1	2	1.6

tertiary care hospital. He was haemodynamically stable requiring only 2 litres oxygen via the tracheostomy mask. On examination, he had oral ulcers and a tender right hypochondrium. His initial workup in ER showed BUN 17 mg/dl Cr 1.5 mg/dl TLC 19.4 SGOT 1982 SGPT 303 and chest xray showed right perihilar ill defined shadowing. In the ER he was managed for aspiration pneumonitis and chemical oesophagitis. He was transferred to the special care unit where he was found to have decreased urine output. Upon catheterization he was noticed to have red coloured urine. Repeat laboratory workup revealed BUN 67 mg/dl Cr 4.4 mg/dl. Electrolytes were K 5.3 mmol/L BIC 18.5mmol/L. CPK was 39863 IU/L and Phosphate 7.8 mg/dl suggesting acute kidney injury and rhabdomyolysis.

Patient was aggressively hydrated and urine alkalinization with IV bicarbonate infusion was started. However, his renal function continued to deteriorate, nephrologist was consulted and haemodialysis was done. Gradually his urine output increased and renal function stabilized. On discharge his condition had improved significantly. He was followed up by the Nephrology Department and in one year his renal function improved to the extent that there was no requirement for long term haemodialysis.

Discussion

Hair dye contains a of chemical compound paraphenyldiamine PPD. Most of the cases of hair dye

poisoning have been in the context of suicide from different parts of India and Africa.³ The reported mortality rates from India are as high as 23.92%.¹

The clinical manifestations after hair dye ingestion are quite variable. The two major clinical effects that have been reported are of angioneurotic oedema and renal failure. There have been cases in which the initial presentation was that of anaphylaxis followed by severe kidney injury.⁴ Other notable manifestations have been those of rhabdomyolysis, haemolysis, myocarditis, and liver failure.^{5,6} Hence in patients coming to emergency with above clinical features, the index of suspicion for hair dye poisoning should be high. The management of hair dye poisoning is largely supportive as there is no antidote to PPD.

Our case had all the three clinical manifestations that is angioneurotic oedema, rhabdomyolysis and anuric acute renal failure which have all been associated with very high mortality. The largest study from India on hair dye poisoning done over a period of five years showed rhabdomyolysis in 54%, decreased urine output in 12.7 % and anuria in 4.5% .Mortality was much higher in anuric cases (63.04%).¹

Our patient underwent haemodialysis as his acute kidney injury was not improving despite aggressive fluid resuscitation. He was fortunate to recover his renal function after five sessions of haemodialysis. In a case series study from India, all patients had severe acute

kidney injury, requiring haemodialysis support and mortality was reported in 27%.⁷

Conclusion

Hair dye use is quite prevalent in our part of the world and though it is a well recognized cause of suicide in the developing world, there has been only one case report from Pakistan. In view of easy accessibility and common usage of hair dye, there is a greater possibility for accidental ingestions. Our case therefore highlights the need for keeping a strong index of suspicion for hair dye poisoning in patients presenting to emergency medicine with the described clinical manifestations. Moreover it emphasizes the importance of creating awareness among people of hair dye as a hazardous household item.

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