CASE REPORT

HAIR DYE POISONING – WALKING ON A TIGHT ROPE

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ABSTRACT
Hair dye poisoning which was uncommon is now raising an alarm in the field of toxicology. It leads to acute angioedema causing airway compromise, thus making it life threatening. It also leads to acute renal failure and multi organ damage. Failure in prompt recognition and timely management may lead to death. We report a 24 year old female who was brought to us six hours after consuming hair dye. She had difficulty in breathing and was unable to talk. Her tongue, submandibular region and neck were oedematous. Resuscitation measures were taken and she was intubated with great effort. Appropriate anaphylaxis treatment along with forced alkaline diuresis to prevent renal damage led to the betterment of her condition.

KEY-WORDS: Angioedema; Forced Alkaline Diuresis; Hair Dye Poisoning

Introduction

Emulsion based hair dyes are commonly used in India. The main ingredients of such products are paraphenylene diamine (PPD), resorcinol, propylene glycol, sodium ethylene diamine tetra acetic acid (EDTA), preservatives, and perfumes. The main compound responsible for the toxicity is paraphenylenediamine.[1] Systemic features of PPD poisoning occur in three phases. Phase 1 is an acute presentation with edema of neck, airway obstruction, gastritis and severe vomiting. Phase 2 is a subacute presentation with acute renal failure, rhabdomyolysis and hemolysis. Phase 3 progresses to multiorgan failure. Here we report a case of hair dye poisoning who was managed with certain difficulties but later recovered completely.

Case Report

A 24 year old female presented to our emergency room six hours after consuming around 50 ml of hair dye. She was initially taken to a nearby health centre, two hours after consumption, following an episode of vomiting. Stomach wash was given and she was then referred to us. On presentation, she had edema of the eyelids, lips, tongue and neck. She had difficulty in breathing and was unable to talk. Her pulse rate was 88 per min, blood pressure was 100/60 mm Hg, her oxygen saturation was 92 % with supplemental oxygen.

Diagnosis of a life threatening anaphylaxis was made and steps were taken to secure a definitive airway. But intubation seemed to be difficult with extensive edema obstructing the upper respiratory tract and also she belonged to class 4 of modified mallampati scoring. Hence surgical airway was planned and was being prepared. Meanwhile, IM adrenaline (1:1000), adrenaline nebulisation (1:10000), hydrocortisone 200 mg and ranitidine 50 mg was given to control the anaphylaxis. It proved to be useful as the edema reduced to a minimal extent and hence IM adrenaline and adrenaline nebulisation was repeated following which a minimal space was created for the laryngoscope blade to pass through.

Intubation was done with only sedation using midazolam and no paralytic agent was used. Tube was secured with great difficulty and controlled mode ventilation was provided (Fig. 1). Her cardiovascular and pulmonary status was normal. Urine was dark brown in colour. She was transferred to intensive care unit for further management. Investigations revealed serum urea of 47mg/dl and creatinine of 1.2 mg/dl. Muscle enzymes levels were elevated. Serum calcium was 6.2 mg/dl and urine myoglobin was positive (Table 1). Arterial blood gas analysis showed metabolic acidosis. She was treated with IV fluids, steroids, antihistamines and calcium gluconate.

Forced alkaline diuresis (FAD) was started with 5 cycles on the first day in order to prevent further renal damage. FAD was continued 3 cycles the second day and 2 cycles the third day. Colour of her urine improved on the 3rd day and renal parameters were normal. On the fourth day, there was complete recovery of the airway with no signs of edema. Urine was clear and CPK/CKMB values were markedly reduced. She was put on support mode ventilation. She was extubated on the sixth day and was observed. After complete recovery of general condition, she was discharged eight days after admission.

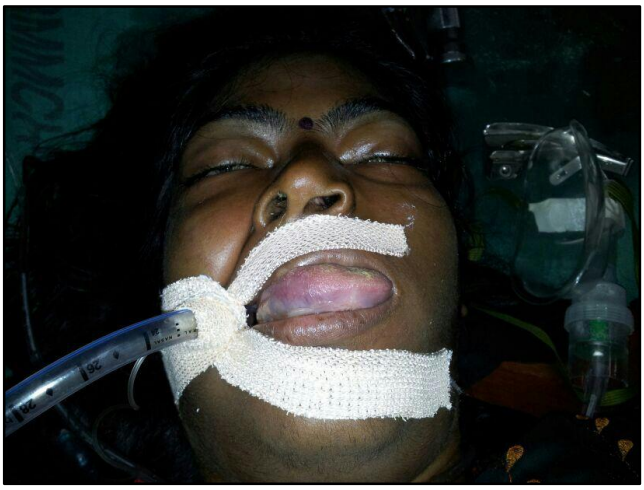

Figure-1: Clinical Picture of Face Showing Edema of Eyelids, Tongue, Face and Neck with Secured ET tube

Table-1: Changes in the Laboratory and Other Parameters

Parameters	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6
Blood Urea (mg/dl)	47	56	50	44	36	38
S. Creatinine (mg/dl)	1.2	1.6	1.4	1.1	1.1	0.9
Calcium (mg/dl)	6.2	8.2	8.5	9	-	-
Sodium (mmol/l)	134	132	132	134	132	133
Potassium (mmol/l)	3.6	3.8	3.2	3.1	3.6	3.6
Chloride (mmol/l)	110	111	108	111	110	106
CPK (U/l)	1280	1062	872	431	216	119
CKMB (U/l)	671	540	382	184	88	62
Ventilator	CMV	CMV	CMV	SIMV	SIMV	Extubated

S. creatinine: serum creatinine; CPK: creatinine phospho kinase; CKMB: creatinine kinase – MB; CMV: controlled mode ventilation; SIMV: synchronized intermittent mandatory ventilation

Discussion

PPD poisoning is not rare but an emerging entity in India. The first ever case reported (1924) was a hairdresser who was continuously exposed to this chemical.[2] PPD, a derivative of paranitro-aniline, on oxidation produces several intermediates, of which Bondrowski's base is most allergenic, mutagenic, and highly toxic.[3] PPD is readily absorbed with mere dermal contact. Dose related symptoms may occur within 4–6 hours of exposure. A three-phase evolution can be seen in PPD intoxication with inflammatory phase (first three days) characterized by a relative immune depression, pro inflammatory phase (3rd to 6th day) due to rhabdomyolysis and immunomodulative action phase (6th day onwards) due to oxidative metabolism.

It is a systemic inflammatory reaction specific to a cytotoxic cell support. The pathophysiologic mechanisms could be the increased free radical formation[4], skeletal and cardiac muscle necrosis (scattered coagulation necrosis)[3], formation of highly nephrotoxic quinonediamine (an oxidation product of PPD metabolites), renal tubular occlusion due to myoglobin casts, and acute tubular necrosis[5]. Methemoglobinemia, hoarseness of voice, cardiac toxicity, hepatitis, hypotension, convulsions, coma, and sudden cardiac death are on the toxic end of the spectrum.[3]

The angioedema of face and neck on initial presentation due to allergic or hypersensitive reaction associated with increased permeability of mast cells along with chocolate colour urine is highly characteristic in most cases.[6] Stigmata of rhabdomyolysis, acute renal failure, leucocytosis, anaemia secondary to haemolysis, are the usual accompaniments. Rarely exophthalmos and blindness may also be seen. Treatment is mainly supportive and depends on clinical presentation. There is no specific antidote for PPD.[2]

Our patient had a severe cervicofacial edema and also belonged to class 4 of modified mallampatti[8] scoring, which necessitates a surgical airway. Antihistamines and steroids are commonly used in the management of airway edema because of the possibility of a hypersensitivity reaction to PPD but there is no evidence to support this mode of treatment.[7] In our case, early and aggressive management with intramuscular adrenaline and adrenaline nebulisation along with steroids and antihistamines favoured intubation or else the condition would have demanded a surgical airway. Alkaline diuresis using isotonic saline, sodium bicarbonate, and diuretics are used in the management of myoglobinuria. Early initiation of forced alkaline diuresis helped in preventing acute

renal failure. Patient responded well to treatment and gained complete recovery without any complications.

Conclusion

Hair dye poisoning is a life threatening emergency which requires emergency resuscitation and aggressive management of anaphylaxis. Early forced alkaline diuresis prevents acute renal failure minimizing the need for dialysis. Though a standard treatment is not available, a complete recovery can be anticipated if adequate intervention is provided.

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