Original Article

Hair Dye Ingestion – An Uncommon Cause of Acute Kidney Injury

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Abstract

Introduction: Dye containing paraphenylene diamine is not an uncommon cause of renal failure in South India. However, there are very few published reports on renal lesions associated with hair dye ingestion.

Material and methods: We studied 30 consecutive cases (24 males and 6 females) of hair dye induced renal failure seen at our department. All the patients were aged between 18 to 40 years (26.9± 4.95 years).

Results: The quantity of dye consumed ranged between 50-100 ml (79.5± 22.45). All patients presented with oliguria and fluid overload. Dyspnea was seen in 24(80%) while 10(33.33%) had hypertension. Encephalopathy and seizures were seen in 10(33.3%). None of the patients had evidence of hemolysis, hematological abnormalities or skin rash. Three patients had elevated SGPT (340 IU/l) which returned to base line after 2 weeks while creatinine phosphokinase (CPK) was elevated in 6(20%) patients. The oliguric phase lasted from 1 to 3 weeks and serum creatinine normalized in 21(70%) patients. Renal biopsy done in 15 patients (done antemortem in 10 and postmortem in 5) showed evidence of acute tubular necrosis (ATN) in 8, acute interstitial nephritis (AIN) in 7 patients. All patients received dialytic support. Eight (26.6%) patients succumbed.

Conclusion: Hair dye is an unusual but important cause of acute kidney injury. The commonest renal lesions are acute tubulointerstitial damage. Respiratory and hemodynamic supportive therapy is essential for recovery.

Introduction

Hair dye containing paraphenylene diamine is commonly used for coloring the hair. The dye is sometimes used to commit suicide in southern India. There is paucity of data on the renal manifestations of hair dye poisoning especially renal histology.

The aim of our study was to analyze the renal manifestations, histology and outcome following consumption of hair dye.

Material & Methods

We studied 30 consecutive patients (24 males and 6 females) admitted in the department with acute kidney injury (AKI) following consumption of hair dye. All the patients were subjected to detailed history, clinical examination and lab evaluation including urine examination, hemogram, liver function tests and ultrasound abdomen. Renal biopsy was performed in 10 of patients who showed delayed recovery from acute kidney injury. The biopsies were done postmortem in 5 patients. Dialysis and supportive treatment was given to all. No specific antidote is available.

Statistical analysis was done using Medcalc.

Results

All the patients (24 males and 6 females) were young aged between 18 to 40 years (26.9± 4.95). The quantity of dye consumed ranged between 50-100 ml (79.5± 22.45). The mean duration before development of symptoms was 3.0 ± 1.31 days. All patients presented with oliguria and fluid overload. High colored urine was seen in 3 patients. Ten (33.3%) patients had hypertension. Encephalopathy and seizures were seen in 30%. Six (20%) patients had respiratory failure and needed tracheostomy, Three needed mechanical ventilation. The mean serum creatinine was 8.57±1.85 mg /dl(5.8-13), blood urea 166.46±62 mg/dl, hemoglobin 12 g/dl, serum Na was 143 ± 6.76 meq/l, serum K 5.1±0.66 meq/l and serum HCO3 18± 2 meq/l. None of the patients had evidence of hemolysis, hematological abnormalities or skin rash. Three patients had elevated SGPT (340 ± 102 IU/l) which returned to base line after 2 weeks while creatinine phosphokinase (CPK) was elevated in 6 patients(20%). All patients received hemodialysis support (mean no. of dialysis 11 ± 4.08).The mean duration of dialysis was 8-45 days (23±10.97). The oliguric phase lasted from 1 week to 3 weeks and serum creatinine normalized in 22(73.3%). Eight (26.6 %) patients succumbed. Renal biopsies were done in 15 (in 10 patients due to delayed recovery of renal function and in 5 patients biopsies were done postmortem), histology revealed acute tubular necrosis (ATN) in 8 and acute interstitial nephritis (AIN) in 7 patients. Regression analysis showed outcome was significantly correlated with volume of hair dye ingested (p=0.003) (Tables 1 & 2).

Discussion

AKI constituted 30 % of all in patient admissions in the Nephrology Department at our centre. The common causes of AKI included hemodynamic mediated renal failure in 52%, drugs in 13 %, glomerulonephritis in 13%, hemolytic uremic syndrome in 0.5%, obstruction in 4.5%, obstetric in 12% and others in 5% (snake envenomation, malaria). More than 60% of cases of AKI had ATN, 2% had cortical necrosis, 16% had AIN and 19% had glomerulonephritis. We had 30 unusual cases of AKI following hair dye ingestion.

Hair dye is a commonly used emulsion (oil) based hair dye

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Table 1 : Patient characteristics

Mean age	26.9 ± 4.95 years
Males : Females	24:6
Mean volume of poison ingested (ml)	79.5 ± 22.45 ml
Interval between ingestion and admission (days)	3.0 ± 1.31 days
Cervicofacial oedema %	6(20%)
Elevated CPK %	6(20%)
Serum creatinine (mg%)	8.57 ± 1.85
Blood urea (mg%)	166.46 ± 22.64
No. of hemodialysis	11.26 ± 4.08

in India. There are reports of hair dye use as a suicidal agent. The composition of dye is- P.P.D, liquid paraffin, cetostearyl alcohol, sodium lauryl sulphate, E.D.T.A., disodium, resorcinol, propylene glycol, herbal extracts, preservatives & perfumes. It is a coal-tar derivative with toxic oxidation product. Though rare in western countries, such poisoning is not uncommon in East Africa, Indian subcontinent and Middle East countries.[1] It is a rare cause of AKI and accounted for 0.6% of all AKI in our study (unpublished data).

All patients consuming PPD may not develop AKI, percentage varies from 47.4-100%.[2] In the study by Kallel et al[2] the frequency of acute kidney injury was 47.4%. Seven patients out of 10 (70%) who consumed PPD developed AKI in another study.[3]

Clinical features are variable. Ram et al[3] showed that clinical manifestations of systemic PPD intoxication are dominated by cervical and upper respiratory tract edema, muscular edema, rhabdomyolysis and oliguric AKI. All patients including those with normal renal function had features of rhabdomyolysis. 40% needed respiratory support-(two required ventilator support and two more required tracheostomy) due to upper airway tract edema. In the study by Kallel et al [2] chocolate brown colored urine was found in 73.3%, cervical edema in 78.9%, upper airway tract edema in 68.4%, muscular edema in 26.3%, oliguria in 36.8% of patients, and anuria in one. In that study oral-tracheal intubation was performed in 68.4% and emergency tracheostomy in 15.8% of patients. Ashar et al suggested that angioedema of the face and neck with difficulty in breathing, secondary to upper respiratory tract edema and chocolate brown color of the urine could be a confirmative evidence of PPD poisoning. [4] Other features are rigidity and tenderness of limbs secondary to rhabdomyolysis, leukocytosis, anemia secondary to hemolysis, hemoglobinemia and hemoglobinuria. Sudden death may occur due to myocarditis and arrhythmias. Myocardial rhabdomyolysis, lysis of other muscles and shock have also been described. [5,6,7,8] Hypovolemia (secondary to the muscular edema) is one of the major complications in systemic PPD intoxication. In a study, 89.5% of patients needed abundant fluid infusion with a median quantity of 7±5 L, [2] while 26.3% of patients developed hemodynamic shock necessitating vasopressors. Hypotensive shock is associated with poor prognosis.[2] In our study rhabdomyolysis was seen in 20%, none had shock while 6 patients had evidence of upper tract oedema / respiratory embarrassment.

Renal failure is probably due to direct tubular or interstitial toxicity. ATN occurs due to rhabdomyolysis as also direct toxic effect of dye on the tubule cell where the dye is concentrated. In a study biopsy showed ATN along with presence of casts in tubules due to myoglobin.[9] In our study, 15 patients were biopsied and showed ATN in 8 and AIN in seven. Apart from ATN other glomerular diseases have been reported in other studies.[10]

Table 2 : Factors predicting outcome

	Recovered (n=22)		Dead (n=8)		
	Mean	SD	Mean	SD	p
Age (yrs)	26.81	4.6	27.11	5.98	0.88
Duration (days)	3.095	1.33	2.77	1.3	0.91
Volume (ml)	71.9	22.38	97.22	8.33	0.003*
Urea (mg%)	167.9	23.19	163.11	22.27	0.69
Creatinine (mg%)	8.29	1.83	7.73	1.69	0.16
Potassium (meq/l)	5.14	0.71	5.1	0.55	0.87
Sodium (meq/l)	143.33	7.05	145.11	6.25	0.51

The mainstay of treatment includes-gastric lavage, fluid infusion (7±5 L), respiratory support and dialysis as needed - mechanical ventilation, oral tracheal intubation and even tracheostomy. Use of alkali, steroids and vasopressor support has been described in various studies.[2,,3] In our study 20% of the patients had respiratory failure. All underwent dialysis.

Death has been attributed to acute respiratory distress and may be so rapid that tracheostomy at presentation is recommended by some authors. In a study from NIMS, renal failure and myocarditis were found to be poor prognostic markers[3] and mortality was 10%. There was a high mortality (26%) in our study and was correlated with the volume of dye consumed.

Conclusion

Hair dye ingestion is an uncommon cause of AKI. Most common biopsy finding is ATN. Supportive therapy is essential for recovery.

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