Case Report

An Electromyographic and Enzymatically Confirmed Case of Inflammatory Myopathy in Hairdye Poisoning

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ABSTRACT

Reports on electromyographic changes in poisoning with hairdye containing paraphenylenediamine are scant. In view of the number of cases of hairdye ingestion presenting with severe pain in the limbs, it was planned to record the electromyogram and observe the pattern. This is a report on electromyographically and enzymatically proved case of systemic poisoning with hairdye (paraphenylenediamine) ingestion, resulting in quadriparesis and acute renal failure.

Key Words: Hairdye; Paraphenylenediamine; Electromyogram (EMG)

Introduction

Paraphenylenediamine (hairdye) poisoning is usually associated with the typical syndrome of cervico-facial oedema, rhabdomyolysis and renal failure.[1] Hairdyes are inexpensive and easily available cosmetic preparations. Suicidal ingestion of these cosmetics now has become an emerging trend. Paraphenylenediamine (PPD) is the most toxic component in these preparations. Fatal dose is unpredictable due to variable concentration of PPD in different commercial brands. Apart from ingestion, topical application may sometimes produce acute or chronic toxicity.[2]

The Case: A 48-year-old male presented ten hours after ingestion of a hairdye (paraphenylenediamine) preparation with complaints of pain in the extremities, palpitations, vomiting and chocolate-brown urine. On examination, he was conscious, oriented, and haemodynamically stable. General and systemic examination were unremarkable, except for tenderness in the limb muscles. The usual manifestations of paraphenylenediamine ingestion such as swelling of lips, mouth, face and tongue, and breathlessness were absent.

Investigations undertaken at the time of admission revealed marked elevation of CPK, CPK-MB, SGOT, SGPT and leucocytosis (**Table 1**). ECG showed evidence of sinus tachycardia. Pain in the extremities gradually worsened. Deep tendon reflexes were diminished, and muscle power was reduced to grade one. Electromyogram (EMG) was performed and revealed evidence of early active inflammatory myopathic pathology (**Fig 1**). Nerve conduction study was normal. Serial renal function tests showed progressive rise in urea and creatinine levels for which dialytic support was given. The patient continued to fare well on supportive measures and initial short-term steroid therapy. He was discharged on the 19th day.

Discussion

Paraphenylenediamine (PPD) poisoning presents with features of angioneurotic oedema, rhabdomyolysis and intravascular haemolysis with haemoglobinuria leading to acute renal failure.[3]

Reports on EMG-confirmed inflammatory myopathy due to PPD poisoning are very rare. The case being reported here is a bit unusual from other reported cases as there were mild features of cervico-facial oedema. Pain in the extremities, tender muscles, and inability to move the

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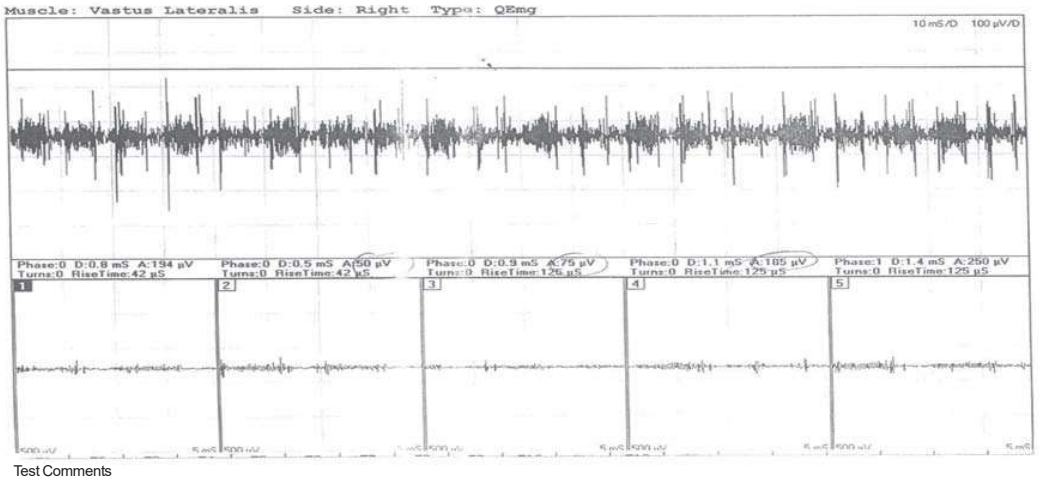
Table 1

Investigations	Day 1	Day 2	Day 4	Day 5	Day 8	Day 10	Day 12	Day 15	Day 18
Hb (gm%)	17.3	-	-	-	-	-	13.2	-	-
TLC (/mm^3)	22,400	-	-	-	-	-	17,000	-	-
DLC	N-92, L-8	-	-	-	-	-	N-80, L-18	-	-
Blood urea (mg/dl)	47	95.1	121	135	186	155	182	167	28
Serum creatinine (mg/dl)	1.49	2.1	4.4	6.0	6.9	9.7	8.6	8.2	1.5
Serum bilirubin (mg%)	2.52	-	0.7	-	-	1.9	1.12	1.3	-
SGOT (IU/L)	819	-	-	-	-	96	107	47	-
SGPT (IU/L)	126	-	794	792	-	147	145	71	-
Alkaline phosphatase (IU/L)	81	-	88	-	-	141	136	143	-
Serum protein (gm%)	7.6	-	5.9	-	5.2	7.2	6.2	6.3	7.5
CPK (IU/L)	4250	-	9125	-	-	-	960.1	-	-
CPK-MB (U/L)	1883	-	-	-	-	-	-	-	-
Platelets (lacs/cu mm)	2.2	-	-	-	-	-	2.8	-	-
Blood sugar (mg%)	-	138	-	-	-	-	-	-	-
Serum Na^+ (mmol/L)	-	138	-	-	-	-	-	-	-
Serum K^+	-	4.1	-	-	-	-	-	-	-
Urine R+M	-	Alb ++, PC8-10 /HPF.G Cast+	-	-	-	-	-	-	-



Test Comments

Note: The result may be clinically correlated
RSM, RMG, EP Mark II

Fig 1 Reduced muscle unit action potential: small amplitude & decreased duration in vastus lateralis

limbs were the cardinal manifestations in this case. Elevated CPK, and active inflammatory myopathic pattern in EMG confirmed inflammatory myopathy in the extremities, which resulted in quadriparesis. The severity was more marked in the lower limbs, probably due to greater bulk of muscles. The pain gradually improved, and muscle power returned to normal over 7-10 days. Chugh reported acute tubular necrosis of kidney due to paraphenylenediamine ingestion.[4] Acute renal failure in this case responded well to dialytic support.

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