# Lysol Poisoning

## PROF. A.J. PSAILA, MD DIH (LOND) MRCP (UK) FRCP

Professor of Medicine The University of Malta Senior Consultant Physician and Head Department of Medicine St. Luke's Hospital

DR. BERNARDETTE SPITERI, MD FROM PROFESSORIAL UNIT DEPARTMENT OF MEDICINE

he term "Lysol" was originally registered by a German firm, but following the First World War this registration was cancelled. It is still a protected trademark in certain countries notably USA.

Lysol or Liquor Cresolis Saponatis is a 50% solution of Cresol (3—Methyl Phenol  $CH_3C_6H_4OH$ ) in a saponified vegetable oil. It replaced carbolic acid as a less toxic disinfectant.<sup>1</sup>

Lysol has an amber or red brown colour and a distinctive phenolic odour. At the beginning of this century, fatalities from carbolic acid or phenol poisoning were exceedingly common. Glaister (1902) stated: "Its use is familiar to the lay public as a popular disinfectant and of late years by reason of its facility of purchase and its common use has given rise suicidally and accidentally to more deaths than any other poisonous substance."<sup>2</sup>

The reason lay with the ease with which it could be purchased, it being sold frequently to the general public without any form of restriction. The large number of phenol induced deaths made it necessary to prevent its misuse by including it as a scheduled poison.

Instances of phenol, cresol and lysol poisoning, although much less infrequent, still occur as a means of suicidal or accidental deaths.

The patient usually presents with the characteristic odour about the body and internal organs, and the pronounced brown staining is present round the mouth, drinking being the most frequent method of ingestion. The following case recorded here is particular in that the mode of administration of lysol was by I.V. injection.

### **Case Report**

This case deals with a 36 year old married man, who had been a heroin addict for about 1 year. He



Fig. 1.



Fig. 2.





had been undergoing medical and psychiatric treatment for his drug addiction and suffered periods of depression. He presented at 3.00 am at the Casualty department after having injected 5cc of lysol I.V. in a suicidal attempt, the lysol having been bought that respective evening from a chemist. His last I.V. injection of heroin had been 3 days previously.

The patient was fully conscious and quieted. On examination the patient was tachypnoeic with a respiratory rate of about 36/min and had a mild tachycardia. He suffered a slight drop in B.P. 100/80 and was running a temperature of 99 F. On auscultation there was a good air entry on both sides; rhonchi and scattered wheezing could be heard in both lung fields. No abnormalities could be detected abdominally or in the CNS.

As emergency treatment an I.V.I. of N. Saline with 250 mg. Aminophilline, (6 hourly) was set up. Boluses of hydrocortisone 100 mg I.V. and Lasix60 mg I.V. were given 6 hourly. Blood for toxicology, including phenol levels were taken.

Within the next few hours the patient developed a more severe tachypnoea, cough and cramping epigastric pain. He was sweating profusely and on auscultation one could hear coarse rhonchi and wheezing. He was transfered to I.T.U.

The patient's urinary output became increasingly impaired and by the 25.7.84 - 1 day post admission the patient was suffering from acute renal failure. On CXR markedly prominent patchy shadows in the

Date	Input	Output
24/7	1800cc	377сс
25/7	693cc	138cc
26/7	922cc	272cc
27/7	1806cc	665cc
28/7	1480cc	1283cc
29/7	2080cc	2450cc
30/7	2950cc	4370cc
31/7	2150cc	5180cc
1/8	4800cc	2950cc
2/8	4000cc	4750cc

Table 1. Daily fluid intake and urinary output (see graphic representation - Fig. 3.)



paracardial basal regions could be seen on both sides indicating interstitial pneumonitis Fig. 1, 2, 6, 7. The patient had his first session of haemodialysis (blood urea 167 mg/dl) on 26.7.84. Subsequent to this the patient had 3 more sessions of haemodialysis but during the third session he developed runs of ventricular ectopics and the haemodialysis was stopped prematurely.

On the 4th day post admission the patient developed continuous abdominal pain with diarrhoea. He was treated with Lomotil 1 tab. t.d.s.

On the 31.7.84 the diuretic phase of acute renal failure set in, the patient passing 5180 cm<sup>3</sup> of urine. His

lung problem was resolving hourly.

Following this the patient made a satisfactory recovery being discharged on 13.8.84 that is 19 days post admission. His general condition was good, his main problem being a residual anaemia (Hb 11.1 g/100 mls) and bouts of depression. He is currently still under medical and psychiatric supervision.

### Discussion

Cresol poisoning, as mentioned previously is almost invariably due to one or other of the coal tar disinfectants e.g. Lysol or Teyes' Fluid. When the mode of ingestion is oral, lysol burns are frequently present on the lips and may extend over the face and neck as corrosive fluid trickles away. The stains are

Date	Hb g/100mls.	WBC per mm <sup>3</sup>	PCV %
24.7.84	17.8		
25.7.84	17.3	22,300	55
26.7.84	13.6	10,200	50
27.7.84	15.0	9,400	30
28.7.84	13.0	8,400	37
29.7.84	12.6	16,100	
30.7.84	11.8	12,800	31
31.7.84	11.6	12,000	
1.8.84	13.0	11,300	35
3.8.84	10.7	12,200	32
8.8.84	10.3	8,900	31
11.8.84	11.1	8,800	
13.8.84		DISCHARGED	

Table 2. Daily haemoglobin concentration, WBC count and PCV (see graphic representation - Fig. 4)



Table 3. Daily electrolyte concentration in the serum (see graphic representation - Fig. 5.)

light brown in colour, but the smell of the breath, even the odour in the mouth of the dead is usually strong and unmistakable. In the case under discussion there were no signs of burns not even at the site of puncture.

Interesting are the findings at post-mortem ---wrinkled greyish leathery hardening of the oesophageal mucous membrane and a softer flaking brown corrosive fixation of the gastric mucosa are present.<sup>3</sup> An interesting finding as recorded by Bruce et al. are changes in the liver and renal cortex. In both there are large accumulations of a brown-orange pigment, most dense in the mid- and centri-lobular g zones in the liver and in the proximal convoluted gtubules and glomerular tufts in the kidneys. the  $\frac{1}{10}$ pigment was found to be lipofuscin.<sup>2</sup>

Peritoneal dialysis and lysol poisoning is mentioned in the literature.<sup>4</sup> It appears that in the presence of adequate renal function peritoneal dialysis appears to be of no value in removing phenol from the body. It may be beneficial in preventing a rise in Serum K<sup>+</sup> possibly an important factor in the early deaths which occur with lysol poisoning. This incidental rise in K<sup>+</sup> was noted in the serum electrolyte estimations of the case under discussion.

#### References

- 2. Bruce et al. (1976) Journal of Medicine Vol. 16 No. 3.
- 3. Simpson K. Forensic Medicine.
- 4. Thomas B.B.L. Peritonial Dialysis and Lysol poisoning (1969) B.M.J. 3:720 (c).



Fig. 8. Graphic representation of Table 4.

Date	Urea mg/100 mls.	(Urea Mean)	Creatinine mg/dl.
24/7	63		5.2
25/7	108	111	8.7
25/7	100		
25/7	126		
26/7	167	160	
26/7	148		
26/7	167		
27/7	234	250	9.2
27/7	265		
28/7	228		10.6
30/7	262		11.2
31/7	248		11.3
1/8	236	227	11.0
1/8	226		
1/8	220		
2/8	210		
3/8	190		
4/8	172		5.0
6/8	101		2.9
11/8	58		

Table 4. Daily blood urea and serum creatinine estimations' (see graphic representation - Fig. 8)

12

<sup>1.</sup> Polson C.J., Green M.A., Dee MR. Clinical Toxicology.