ACUTE PHENOL POISONING

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Cases of phenol (C_6H_5OH) poisoning were more frequent in the past than they are today. Approximately 50% of those reported have terminated fatally. An oral dose of 1 g. phenol may be lethal to man, although it has been recorded that patients have survived the ingestion of 65 g. (Kobert, 1906). The following cases have been reported:—

Severe acute intoxication occurred in a 3-year-old child, whose scalp was treated accidentally with pure phenol instead of a dilute solution. The mistake was discovered four minutes after application, when the child became unconscious (Brown, 1895).

A nurse, whose thumb and index finger were contaminated with pure phenol, accidentally touched the groin of a 7-day-old infant, leaving two patches, one the size of a sixpence and the other of a half-crown. Severe convulsions were observed within five minutes, ending fatally 10 hours later (Abrahams, 1900).

A 13-year-old girl accidentally poured pure phenol over the scalp and cheek; she died within two hours (Gibson, 1905).

A man accidentally broke a bottle of dilute phenol in his pocket. The contents ran down his thighs, and he showed signs of systemic intoxication within five minutes. The toxæmia was characterized by severe cyanosis, stertorous breathing, vomiting, coma, the abolition of reflexes, pin-point pupils, and lowering of body temperature (Turtle and Dolan, 1922).

Absorption of Phenol and Metabolism

Phenol is readily absorbed by ingestion, inhalation, and through the skin. Deichmann and Witherup (1944) state that the rate of absorption of phenol through a rabbit's skin depends primarily on the area exposed, and not upon the concentration of the substance. According to Jackson (1939) absorption from the stomach, normally slow, is affected by the type and quantity of food it contains.

Phenol is disposed of by oxidation and excretion.

Approximately 72% of a sub-lethal dose in a rabbit is excreted in the urine within 24 hours, either as free phenol or as phenol conjugated with acids (Deichmann, 1944).

Signs and Symptoms of Acute Poisoning

Swallowing causes intense burning in the mouth and throat followed by acute abdominal pain, and the breath has a characteristic odour. Symptoms of shock may occur with dizziness and vomiting. The pupils become contracted or dilated and cyanosis can become marked. The respiratory rate increases at first, but may later decrease in rate and volume. The pulse is usually weak and slow, although it is occasionally rapid. The temperature fluctuates. The urine may be dark-coloured and oliguria may develop.

Reflex activity is lost. General tremor or tonic-clonic convulsions may occur, but are not marked. Weakness, collapse, and loss of consciousness may occur in a few minutes.

Early death from respiratory or cardiac failure is due to shock. Delayed death is due to hepatic or renal damage.

Methods of Treatment

In treating these cases speed is of paramount importance. Contaminated clothing must be quickly removed and affected areas of the skin flooded with warm water. As phenol is only slightly soluble in water, swabbing with solvents such as ethyl alcohol and methylated spirits should be carried out to absorb the surface coagulum. When all phenol has been removed, and this can only be assessed by the absence of its characteristic smell, the areas affected should be covered either with wet dressings of saturated sodium sulphate or with oily dressings.

When the poison has been taken by mouth, emetics should be given, or gastric lavage carried out until the odour of phenol is no longer detectable. This should be followed immediately by the administration of some vegetable oil, such as castor oil,

to avoid intestinal damage. Sollmann, Hanzlik, and Pilcher (1910) have pointed out that oils retard the absorption of phenol and tend to reduce local damage.

Symptomatic treatment consists of allaying shock and giving circulatory stimulants, which counteract stasis.

Case Report

T.M., aged 22, a general worker, was spraying weeds with the effluent from a chemical plant, the organic content of which was predominantly phenolic.

A faulty coupling caused the operative to be sprayed with the fluid. He was wearing goggles, gloves, waterproof coat, and gum-boots, but had omitted to put on his protective trousers.

The following areas of skin were exposed to this fluid:—Left thigh, 7 in. × 4 in.; right thigh, 6 in. × 2 in.; anterior and antero-lateral surfaces of the scrotum and penis.

Copious irrigation with warm water was started immediately and continued for 30 minutes. This was followed with ethyl alcohol swabbing for 10 minutes, and the whole procedure repeated. For at least an hour and a half there was a smell of phenol on the affected areas.

The patient developed symptoms of shock within half an hour. The temperature fell to 97.2° F; the pulse was 62 and of poor volume. The respiratory rate was slightly raised, and the respirations became stertorous. The pupils were contracted and reacted sluggishly to light and accommodation. For a period of half an hour there were convulsive movements of the left leg. All other reflexes were normal.

After two hours' treatment the affected parts of the skin were dressed with a saturated solution of sodium sulphate, and the patient was referred to the local hospital for observation.

Recovery from shock was almost complete when the patient was admitted to hospital, and on the following day there were no clinical signs of intoxication. The burns were superficial. They were treated with spirit dressings and rapidly healed.

There was evidence of minimal liver damage. On the day following admission, the serum bilirubin was slightly raised to 1.7 mg.% (normal range up to 1 mg.%), and the direct Van den Bergh reaction was positive, with a direct/indirect quotient of 40% (Gray, 1947). Other tests for liver function were normal. The urine was not examined for phenol until four days after admission, when tests were negative. One week later the Van den Bergh reaction was negative and readings normal; the serum bilirubin level was 0.7 mg.%.

The patient was discharged from hospital on the seventh day.

The Effluent

At the time of the accident it was assumed that the total phenolic content of the effluent used was less than 3% but analysis showed the following:—

Water	20%	Percentage by Weight
Low boilers (probably aldehydes)	11.5%	
High boilers (resinous materials)	11%	
Phenol	43.5%	
Para cresol	12%	
Meta cresol		
Ortho cresol and others	2%	

Phenol is immiscible with water below 68°C., the lower organic layer consisting of phenol with a little water, and the upper aqueous layer a dilute solution of phenol (Findlay, 1942). The actual concentration of each layer is influenced by the temperature of the water according to the following table (Seidell, 1941):—

SOLUBILITY OF PHENOL IN H₂O AT VARIOUS TEMPERATURES

Temperature	Percentage by Weight	
	Upper Layer	Lower Layer
0°C	6.3	80
10°C	7.5	75
20°C	8.3	72.1
40°C	9.6	66.9
60°C	16.7	55.4
65°C	21.9	49.2
68.3°C	33.4 miscible	33.4

On this occasion the temperature of the fluid was between 10°C. and 20°C., and it seems probable that a high proportion of the lower layer was sucked from the effluent settling tank into the spray container. This was then used undiluted for spraying.

Discussion

The toxicological hazard of phenol and its derivatives must never be under-estimated, and the mishandling of these substances can easily result in serious consequences.

Although unconfirmed by phenol excretion tests, circumstantial evidence would seem to justify a diagnosis, in the case described, of acute phenol poisoning. A direct positive Van den Bergh reaction, confirmed by the direct/indirect quotient finding, together with a raised serum bilirubin content would indicate some degree of liver damage. It is known that phenol is partly detoxicated by the liver. As the renal threshold for bilirubin is above 1.7 mg.% in the serum, one would not, therefore, expect to find anything abnormal in the urine analyses apart from the phenolic content.

If immediate treatment had not been undertaken and continued for some time, or if a larger area of skin had been affected, it is highly probable that the results would have been far more serious.

Summary

The toxicological hazard of phenol has been discussed. The rapidity of onset of toxic symptoms after exposure to this substance has been illustrated, and the necessity for treating phenol and its compounds with due care is stressed.

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