Phenol Marasmus

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CASE REPORT

Phenol Marasmus

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Chronic phenol poisoning was known 100 years ago primarily as a disorder of physicians. Lister first used this chemical to treat the wound and the exposed bone in compound fractures, and reported surgical cures of what had been previously usually a fatal injury. Five years later he recommended a carbolic spray in a concentration of one to forty for antisepsis in the operating room.

The drug found immediate popularity with physicians and patients. It has a warm aromatic odor, and smells as though it would kill bacteria. It masked the odor of gangrene and pus. It was a mild local anesthetic, and when irrigated through a wound decreased pain. Because of these features it was credited with more effectiveness than it possessed. Consequently for several decades carbolic acid was commonly used in sprays, and as a wound irrigant. It was applied externally in the form of jellies, ointments, lotions, soaks, and plasters, and was taken internally in pills and in dilute solution.[1]

During the years in which the spray was used the health of surgeons and their operating room assistants was sometimes affected. The term carbol marasmus was applied to a disorder, recognized as an occupational disturbance of physicians and their assistants, due to the inhalation of phenol. The patient suffered from anorexia, progressive weight loss, headache, vertigo, salivation, and a peculiar dark coloration of the urine. Many more doctors are said to have died from this cause then was generally recognized. An odd form of pigmentation of the sclerae, the skin over the nose and the malar eminences, appeared in some patients, which was thought to represent a form of ochronosis. The urine was generally dark, and on standing became ever darker, taking on a brown or even black coloration. Some early investigators reported the finding of homogentisic acid in the urine, others hydroquinone, and still others were unable to identify the dark material in the urine.[2 3]

By 1890 the abuse of carbolic acid had largely stopped, and, although there were still occasional suicidal attempts or occasional over-use by a patient, the poisonous properties of the drug were sufficiently well recognized that it was used cautiously. Generally it appeared in dilute form for external use. Now, along with other drugs of the past, it has fallen into disuse. In the few pharmaceutical preparations in which it persists, phenol is used largely for its mildly anesthetic effect.

I have encountered one case of classical phenol marasmus, reminiscent of the chronic carbolic acid poisonings that were common in physicians a hundred years or so ago.

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Case Report

The patient, age 44, had worked for 13½ years as a quality control laboratory technician for a company engaged in the processing of phenol, cresol, and xylenol in their pure state. Several times a day he distilled 150 cc's of phenol in a 99% solution. While the distillate was most often trapped in the distillation coils, on occasions, due to inadvertence, the phenol would escape into the laboratory atmosphere, and heavy odors would then be detectable. In addition the patient often spilled phenol on his trousers and clothes, and noticed resultant skin irritation.

Gradually the patient's health deteriorated without specific symptoms. His appetite lessened and he lost weight slowly. He noticed that his urine was dark on occasions, and he was particularly troubled by muscle pain in his legs and his arms. He sought medical care, and, although no diagnosis was reached, it was suggested that the patient discontinue his industrial inhalation of phenol and cresol. The patient stayed away from his job and laboratory for several months, during which time there was a slow gradual improvement. However he still remained quite ill, complaining of weakness, muscle aches and pain. One day he returned to his laboratory and spent 45 minutes in its atmosphere, talking to the man who replaced him. An immediate recurrence

TABLE 1. – Summary of Laboratory Results

Test	Normal	9/29/70	10/6/70	10/9/70	10/13/70	10/27/70	12/28/70	3/10/71
Bilirubin (serum total)	0-1.5	–	–	0.63mg%	–	0.69mg%	–	–
SLDH	200-500	915	668	–	751	644	631	–
SGOT	4-40	850	850	–	850	850	55	42
SGPT	4-40	4200	4200	–	330	900	111	64
SCPK	–	–	–	–	5.3	–	–	–
Hepatitis (associated antigen)	–	–	–	–	–	neg	–	–
Direct Coombs	–	–	–	–	neg	–	–	–
Urine								
Homogentisic acid	neg	neg	neg	–	pos	neg	–	–
glucose	neg	2+	neg	–	trace	–	–	–
urobilinogen	neg	neg	–	–	–	–	–	–
alkaptonuria (Flishberg test)	neg	–	–	–	neg	–	–	–
Blood count (complete)	–	–	–	–	normal	–	–	–
Electrophoresis (serum)	–	–	–	normal	–	–	–	–

* SLDH = serum lactic dehydrogenase; SGOT = Serum glutamic-oxalacetic transaminase; SGPT = serum glutamic-pyruvic tranaminase; SCPK = serum creatine phosphokinase

of symptoms was associated with muscle pain and weakness, and prompt darkening of the urine.

The patient was examined on September 23, 1970. He was emaciated, being 71½ in. tall and weighing 135 lb. The liver was enlarged and slightly tender to the touch. The urine was so dark that its appearance suggested hemoglobinuria. It remained dark for several weeks. Moderately brown in color when excreted, it would take on a darker mahogany color after standing four to six hours. A summary of the laboratory studies on this patient are presented in the accompanying tables.

The first sign of improvement was spontaneous clearing of the patient's urine. Homogentisic acid was found using the silver nitrate test on one occasion, but numerous tests when the urine was light as well as dark were negative as were repeated tests for porphyrins, bilirubin, myoglobin, and urobilinogen. Frustratingly, the dark color of the urine cleared before more sophisticated testing could be carried out. Gradually the patient's appetite improved, and his weight slowly climbed from a low of 135 to 152 lb. However in December 1970 approximately seven months after the first examination, the patient had not yet gained his former state of health. Total protein, albumin, and globulin were normal as was the thymol turbidity. The alpha butyric dehydrogenase was slightly elevated to 155 units. Ig G was 1200 and Ig M 200, compared with average normals of 1200 plus or minus 300 and 100 plus or minus 300 by this author (Pfizer). The cephalin flocculation was 2 plus in 24-hours, 3 plus in 48-hours. The liver was no longer palpable. The patient had gained seventeen pounds.

Comment

This represents a classical example of phenol marasmus. This patient worked in a laboratory boiling solutions of phenol, the vapors of which not infrequently entered his breathing atmosphere. He often soiled his skin with phenol. A dark urine developed and lasted for several years, fluctuating with the way the patient felt. It cleared in two or three months after the patient was completely removed from further exposure to phenol. I was not able to identify the material that caused the darkening. While one test for homogentistic acid was positive, many other tests were negative, and the material was not identified as melanin, bilirubin, porphyrins, myoglobin, or urobilin. Moreover it seems unlikely that this dark material is a metabolite of carbolic acid, or a direct derivative of carbolic acid, since it was present for many months after the patient last worked.

Under all circumstances the underlying disorder is readily identified in this modern case as an anicteric chemical hepatitis, – the probable cause of the occupational death of many physicians in the past.

TABLE 2. – Glucose Tolerance Tests 10/14/70

4/4/71

Specimen	Blood glucose mg/100 ml	Urinary glucose	Blood glucose mg/100 ml	Urinary glucose
Fasting	91	neg	80	neg
½ hour	234	neg	–	–
1 hour	240	trace	128	neg
2 hours	104	neg	76	neg
3 hours	53	neg	69	neg
4 hours	78	neg	–	–

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