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A study of oxalic-acid poisoning.

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The earliest case of oxalic-acid poisoning, reported by Royston, occurred in England, in 1814. Since then, the number of deaths due to oxalic acid and its soluble salts has so increased that today it ranks among the first three poisons in the number of fatalities. A. W. Blyth states that in the five years between 1912 and 1916, there were 448 deaths in England and Wales due to oxalic acid.

The *duration* of a case of oxalic-acid poisoning is usually between 2 and 14 days. There is one case on record by Ogilvie (*Lancet*, 1845) however where death occurred within 3 minutes.

Oxalic acid acts locally as a corrosive and also as a systemic poison. Locally it is more or less destructive to the mucous membrane with which it comes in contact. The lips, tongue, pharynx and esophagus are discolored yellowish white, sometimes marked with patches of a reddish hue. The mucous surface of the stomach is coarsely corrugated and presents a bright red color both in the elevations and depressions; this may change to brown or even black by postmortem action. In some cases the mucous surface is in part or in whole pale, opaque or translucent, and marked by a coarse ramiform vascularity of the submucous tissue. The mucous membrane is soft, pulpy, eroded in patches, thrown into folds, and is easily detached. Perforation is rare.

The systematic effects are attested by falling of the blood pressure, arhythmia and retardation of the pulse, slow breathing, paralytic symptoms and fibrillary muscular contractions. Some consider it a poison acting on the extracardiac ganglia. The red blood corpuscles are destroyed, with consequent fatty degeneration of the tissues. The activity of the muscles is diminished consequent upon loss of irritability. The respiratory muscles are paralyzed.

Symptoms.—Although more than 1,000 cases of oxalic-acid poisoning have occurred since Christison wrote his treatise, his description still holds good. "If a person immediately after swallowing a solution of a crystalline salt, which tasted strongly acid, is attacked with burning in the throat, then with a burning in the stomach, vomiting, particularly of bloody matter, imperceptible pulse, and excessive languor, and dies in half an hour, or still more, in twenty, fifteen, or ten minutes, I do not know of any fallacy which can interfere with the conclusion that oxalic acid was the cause of death. No parallel disease begins so abruptly, and terminates so soon; and no other crystalline poison has the same effect." There may also occur headache, cold extremities, numbness and tingling, loss of voice, cramps, convulsions, delirium, coma, etc.

Prognosis.—Out of 242 reported cases, there were 132 deaths, a mortality of 54.5 per cent. This ratio, of course, depends upon the manner and speed of treatment. Nor is the outlook for complete recovery favorable if the initial degree of poisoning was severe. There have been a few cases reported in which patients returned after some months suffering from gastric irritability, dyspepsia and symptoms of constriction of the esophagus, the latter due apparently to destruction and subsequent repair of the mucous membrane.

Postmortem.—Aside from the local corrosive action, there are no typical pathological lesions with one exception, namely, in the kidneys, where the cortical substance may present a definite whitish appearance due to the presence in the tissues of crystals of calcium oxalate. None is deposited in the glomeruli. Calcium-oxalate crystals have also been found in the blood, bile, aqueous humor, and pleural and pericardial fluids.

Elimination.—Is mainly through the urine. The reported analyses show that from 80 to 90 per cent. is excreted through this channel. The urine also contains albumin, a reducing substance and hyaline casts.

Treatment.—The oxalic acid should be neutralized and precipitated as quickly as possible, by giving plenty of syrup of lime or a suspension of calcium carbonate. After a few minutes the stomach should be washed out with lime water and, lastly, with

plenty of plain water. Stimulants and warmth should be administered to avoid collapse. Diuretics and an abundance of liquid should be given to combat nephritic conditions, and alkalies should be administered to prevent the tendency toward acidosis.

I purpose to present to you to-night a case that came for treatment on the Third Medical Division of Bellevue Hospital, under the direction of Dr. S. A. Brown. This case is of interest because the patient recovered, although antidotal treatment was delayed, and also because of the complete blood study throughout the patient's stay in the hospital.

J. M., a building superintendent, age 44.

Present History.—The patient had no bowel movement for 2 days and decided to take what he thought to be a dose of epsom salts. He immediately noticed a peculiar sour taste, with a burning sensation along the esophageal tract, followed presently by severe pains in the epigastrium, which gradually increased. An ambulance was summoned and when the surgeon attempted to examine the patient's throat he vomited. The surgeon, diagnosing it as a case of indigestion, administered sedatives and left. Shortly thereafter the patient developed pain in the lumbar region, the ambulance was summoned again and he was taken to the hospital on October 1, 1921, at 4:30 A.M.

In the course of the succeeding hours he complained of burning pains in the stomach, first localized, then radiating through the abdomen, and of pain and tenderness in the lumbar region. The next morning, after taking a glass of milk, he again vomited.

On physical examination, he was found to be well developed and well nourished. The pupils were equal and regular, reacting readily to light and accommodation. The tongue was clean, the teeth poor, the throat congested. There were no adventitious sounds in the lungs. The heart sounds were of fair quality, no murmurs, rhythm regular, rate 90. The abdomen showed no rigidity, no masses, no tenderness. The spleen was palpable just below the costal margin. The liver edge was also palpable. On deep pressure in the epigastrium, there was pain. There was no edema. Reflexes—no Babinski, no clonus, abdominal cremasteric and knee jerk active.

Treatment consisted of fluid diet, with hot packs, colon irriga-

tions and the following medication: At first bromides and chloral and magnesium sulphate, followed by luminal; later Tr. nux vomica, sodium bicarbonate and veronal. Toward the latter part of his stay in the hospital he was given bismuth subgallate and triple phosphates. It should be noted that no antidotes for oxalic acid were given, as the nature of the poison, if any, was unknown. Upon analysis the salts of which patient had partaken were found to contain 73 per cent. Mg. SO_4 and 27 per cent. oxalic acid. The patient stated that he took what is estimated to be 15 to 18 grams of the salt. This means that he obtained 4 to 4.9 grams of oxalic acid. The most common fatal doses are 7 to 15 grams, although there is one case on record in which 3.88 grams proved fatal.

The urine was analyzed for oxalates and found to contain 9.2 mg. of oxalic acid in 100 c.c. of urine. It also contained albumin, white blood cells, red blood cells, hyaline and granular casts.

The blood was analyzed throughout the patient's stay in the hospital, at intervals of a few days, with the following results:

BLOOD ANALYSIS.

	Time Interval in Days.							
	4	10	14	19	25	28	31	33
	mg.	mg.	mg.	mg.	mg.	mg.	mg.	mg.
Non-protein nitrogen.	85	270	200	60	73	37	40	37
Urea nitrogen.....	59	211	149	39	51	16	17	17
Creatinine.....	4.3	5.1	2.3	1.6	1.2	1.0	1.5	1.6
Uric acid.....	4.1	6.3	3.2	1.5	1.0	—	1.2	1.5
Sugar.....	174	98	—	75	70	—	85	—
Alkaline reserve, per cent.....	48	41	46	55	—	59	—	55

The study of the chemistry of the blood shows a gradual increase in the excretory products. This finding may be explained by the mechanical effects of the calcium-oxalate deposition in the kidneys, seen at autopsy in similar cases. The highest point was reached on the tenth day. In connection with the accepted view that cases in which creatinine is higher than 5 gm. do not recover, it is interesting to notice that the creatinine in this case was 5.1 mg. on the tenth day. A possible explanation for recovery

in cases of oxalic-acid poisoning with creatinine over 5 mg. may be that the kidney changes are temporary. The alkaline reserve was below normal throughout the retention period. This evidently indicates that in poisoning by oxalic acid the oxidation processes are subnormal. On the twenty-eighth day all values were back to normal.

The urine output during the first few days was much suppressed; the amount voided per day was 30 to 50 c.c. On about the twelfth day, the urine output suddenly increased to 1,900 to 2,200 c.c. It contained little albumin, few hyaline and granular casts, a reducing substance which did not ferment, together with red blood cells, white blood cells and an increased amount of oxalates.

In view of the fact that over 4 grams of oxalic acid had been taken and that treatment was delayed (in fact, no antidote was given at all), it is interesting to note that recovery occurred. The most plausible explanation for this is the simultaneous taking of magnesium sulphate, which probably hastened the elimination of the poison.

About one month later the patient returned to the hospital complaining of gnawing pain in the epigastric region. This pain usually started a half hour after meals and continued until the next meal. It was aggravated by any kind of food, more so by hot food. He had eructations that were sour in character, was constipated, but not anureous. Fluoroscopic examination showed no ulcer or new growth. The x-ray plates showed a calcareous area in the region of the gall-bladder. This condition is evidently a direct result of the corrosive action of the oxalic acid on the mucous membrane of the stomach.