POISONING BY CAUSTIC SODA

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(With Special Plate)

Fortunately poisoning by the fixed caustic alkalis is comparatively rare in England, although in Europe fatal cases appear to be not uncommon. Balázs,³ for example, records in three years 754 alkali poisonings, of which 163, or 21.6 per cent., were fatal; and further statistics are given in the works of Popper¹ and Leschke.² In England ammonia is more frequently employed as a fatal poison, and, according to the Registrar-General's returns⁴ for the three years 1930 to 1932, it accounted for ninety-two deaths, of which seventy-eight were suicidal and the remainder accidental.

It is therefore somewhat remarkable that in a period covering little more than one year no fewer than eight cases of poisoning by caustic soda should have occurred in Cyprus, an island with a population of only 348,000. In England poisoning by the fixed alkalis has nearly always been the result of accident or negligence, and exceptionally of suicide, but in the cases in Cyprus, of which the available data are summarized in the table, half were accidental and half suicidal, the former occurring in children and the latter in adults. A number of typical cases of poisoning by caustic soda or caustic potash and their effects are discussed in the well-known works of Taylor,⁵ Glaister,⁶ and Wynter Blyth,⁷ so that little need be said here; while details of certain cases occurring in Central Europe are given in the texts of Popper and Leschke, already referred to. The majority of such cases appear to fall into two distinct groups: (a) those ending fatally within a few days, and (b) those ending fatally some months or years afterwards.⁸

Source of Caustic Soda

The use of caustic soda locally has greatly increased during the last few years, and it would appear that its misuse has increased correspondingly. Caustic soda is used in small quantities by the Cypriot community for domestic washing purposes, and is cheap and freely available. It has always been the practice in washing clothes to add some form of alkali in order to soften the generally prevailing hard waters of the plains of Cyprus. Formerly the alkali used consisted exclusively of wood ashes supplied from domestic sources. It is perhaps noteworthy that plant ashes, according to Mariti,⁹ were at one time produced in certain villages of Cyprus for export for industrial purposes. Within recent years, however, this has given place to washing soda ($\text{Na}_2\text{CO}_3 \cdot 10\text{H}_2\text{O}$), and during the last two or three years, in its turn, to caustic soda. The reasons given for this change are the cheapness and availability of caustic soda, and, it is claimed, the better results in the wash. In all these cases in Cyprus the alkali was obtained in this way, and its ready availability under the conditions prevailing constituted a serious problem. Practically the whole amount of the caustic soda imported from Europe is absorbed in the local soap industry, but, in view of recent experience, it was considered desirable to restrict its general domestic use. Accordingly it was advised that caustic soda and caustic potash should be placed on

the Poisons Schedule, and this recommendation was adopted by Government in February, 1934. A similar situation appears to have existed in Hungary,¹ where poisoning by caustic alkalis was common. There the difficulty was overcome in 1926 by subjecting the sale of caustic alkalis to restrictions.

The caustic soda as sold was in the form of broken fused lumps, and analysis showed an average content of 77 per cent. NaOH with 10 per cent. sodium carbonate. This latter impurity usually accompanies commercial grades. For washing purposes it is usual to dissolve about 50 grams in a little water, and to add this to about 120 litres of water.

General Features of Poisoning

In the table are summarized the available data of known cases of poisoning by caustic soda occurring since December, 1932, although there are possibly others unknown to the authorities. In spite of the difficulties of obtaining reliable data, especially in regard to the amount of alkali taken, it has been possible to study four of this series (Nos. 1, 4, 7, and 8), all affecting adults, in some detail. Starkenstein¹⁰ regards 10 to 20 grams of solid caustic alkali as the fatal dose, but it is clear from our own experience (see table) that a quantity less than 10 grams can be lethal. The fatal dose is unknown, but it is noteworthy that 2.6 grams (40 grains) of caustic potash have proved fatal (Taylor⁵).

Cases of Poisoning by Caustic Soda

Case	Patient	Age	Cause	Dose (grams)	Symptoms	Result
1	Turkish woman	33	Suicide	60	Burning pains in mouth, throat, and stomach; vomiting and exhaustion	Died 29½ hours after ingestion
2	Turkish child	1½	Accident	?	Lips, tongue, and palate burnt	Died 25 hours after ingestion
3	Greek child	3	Accident	Sucked a lump of NaOH	Mouth, lips, and tongue burnt	Recovery in 3 days
4	Turkish woman	20	Suicide	8.5	Burning of mouth and palate; vomiting blood; shock	Died in 11 days
5	Turkish child	3	Accident	?	Oesophageal stricture; unable to swallow any liquid	Gastrostomy; recovering
6	Greek child	5	Accident	?	Emaciation; oesophageal stricture	Gastrostomy; died
7	Turkish girl	16	Suicide	5	Stricture of oesophagus and pylorus	Palliative jejunostomy. Died after 74 days
8	Turkish girl	15	Suicide	?	Burning of lips, tongue, pharynx; vomiting; shock	Died 26 hours after

The immediate effect of swallowing caustic alkali is to produce excoriation and destruction of the mucous membrane, which gives rise to intense pain, often extending from the mouth to the abdomen. The severity of the lesions depends upon the concentration of the alkali, the stomach contents, and the interval elapsing before treatment. Alkali corrosions are usually oedematous and unctuous as compared with those from acids, which are dry and hard. The alkali extracts water from the tissues with which it comes into contact, and attacks the protein, forming alkali-albuminate, which becomes gelatinous, swells, and in the presence of much water commences to dissolve—a process termed "colliquation." Because of this, alkali burns penetrate deeply into the tissues, although cases of perforation are few compared with those which develop stricture later on.

Treatment follows the accepted lines of avoidance of stomach lavage, and the administration of organic acids and liquids in order to reduce the concentration of the alkali. The use of the bougie at the proper time is regarded as being very important, especially in Continental practice, in order to avoid later complications of oesophageal stricture.

Details of some Medical Cases

The first case of this series, which occurred in December, 1932, was one of determined suicide.

First Example.—A Turkish woman of Niccsia, a mother of five children, swallowed apparently no less than 60 grams (925 grains) of caustic soda dissolved in about 120 c.cm. of water. Symptoms of distress came on almost immediately, with vomiting and burning pains in the mouth, throat, and stomach, and with great exhaustion and collapse. It was observed that the buccal cavity, tonsils, and pharynx were soft and oedematous, and of the deep chocolate colour suggestive of recent burning by alkali, while the lips and tongue were red and swollen. The mouth gave a strong reaction to litmus paper, and the vomited matter consisted of a brownish liquid, due to the presence of blood, and contained portions of detached mucous membrane. This liquid gave a strongly alkaline reaction to litmus, which on analysis proved to be due to the presence of 1.6 per cent. of free caustic soda. The patient was allowed lemon juice *ad lib.*, but, although she appeared to have recovered from shock, her condition had deteriorated by next morning, and death took place twenty-nine and a half hours after ingestion of the poison. Unfortunately it was not possible to carry out a post-mortem examination, but it was considered that death had resulted probably from perforation of the stomach. A more detailed account of this case has been published elsewhere.⁸

Second Example.—The second case of interest (No. 4) again concerned a Turkish woman, a domestic servant, who, at 9 p.m. on August 31st, 1933, swallowed about 8.5 grams (123.4 grains) of caustic soda dissolved in a little water, to which had been added some rosewater in order to mask the burning taste. From the woman's statement the alkali had not completely dissolved, and apparently some was swallowed solid, a fact which may account for the oesophageal perforations found after death. The amount of caustic soda taken was more than three times the known fatal dose of caustic potash (40 grains).

The same symptoms as previously observed—that is, burning pains extending from the mouth to the stomach—came on immediately, with, later, vomiting of blood and mucus. On arrival at hospital, one and a half hours later, the patient was conscious, but suffering from shock, with the extremities very cold. Burning of the surface of the buccal cavity, however, was not so severe as that observed in the first case. Vomiting of blood persisted, and the vomitus gave a marked reaction to red litmus. The patient complained of severe pain in the throat and stomach. The temperature was 97° F., and the pulse rate 90, and during the eleven days of observation the temperature varied from 97° to 101°, the pulse rate between 84 and 130, and the respiration rate between 28 and 36. The patient recovered from shock, but swelling of the tissues which had been in contact with the alkali—especially the lips and tongue—was marked. By the fifth day the tongue was healing, though red, but no liquids could be swallowed, and feeding was carried out with liquid glucose per rectum. After the first week it was clear that the patient was not progressing, although the burned parts appeared to have healed, and to be covered with shiny mucous membrane. Later, signs of bronchopneumonia were observed, and the patient died on September 11th—that is, eleven days after swallowing the poison. In view of the amount of destruction of tissue in the oesophagus and stomach the length of survival appears remarkable. The coroner's finding at inquest was that death was due to exhaustion and bronchopneumonia resulting from suicide by swallowing corrosive poison.

Some interesting conditions were seen at necropsy, which was carried out five hours after death. Externally there were no unusual appearances. The lips were covered with smooth and somewhat shining mucous membrane, and the skin and mucous membranes were generally pale. Rigor mortis was present in all parts, and post-mortem staining in the dependent parts. The anterior half of the tongue was covered with smooth, shining mucous membrane, and at the back of the tongue the papillae were well marked. The mucous membrane in the upper part of the oesophagus was again smooth and shining, while the tip of the epiglottis was absent. At a point 9 cm. below the latter the walls of the

oesophagus were very soft and adherent to the vertebral bodies, and at this level an oval perforation, with smooth edges, 1.5 cm. long, was found between the oesophagus and the trachea. One centimetre below this there was a second circular perforation 1.1 cm. in diameter, also leading into the trachea (Fig. 1 on Special Plate). From the site of these perforations to the pylorus the mucous membrane of the oesophagus and stomach was very soft and pulpy, but the muscular wall was firm and intact. From the level of the lower perforation extending to the pylorus, a dark red, lightly adherent slough was found forming a complete cast of the oesophagus and stomach (Fig. 1 on Plate). The stomach was filled with a dark brown fluid, which gave an alkaline reaction. The mucous membrane of the duodenum was deeply injected, but below the pylorus there was no erosion. The trachea was filled with purulent material, and the left lung exhibited patches of aspiration bronchopneumonia. The liver showed cloudy swelling, and the kidneys were injected with pinpoint haemorrhages. Sections from the liver, kidney, and stomach near the cardiac end were prepared and examined. It was found that the stomach wall near the cardiac end exhibited remains of highly oedematous and necrotic mucous membrane, in which remnants of glands were recognizable. The muscular wall was little affected (Fig. 2 on Plate). Degeneration and desquamation of the tubular epithelium were seen in the kidney, with area of complete necrosis containing blood pigment (Fig. 3 on Plate). In the liver there were again areas of slight focal necrosis (Fig. 4 on Plate). The presence of such areas in the liver and kidney does not appear to have been observed in the recorded cases of poisoning by the fixed alkalis.

Third Example.—Case 8 was again one of suicide, in which a Turkish girl, aged 15, bought caustic soda, and made a concentrated solution with water. She swallowed a mouthful at 5.30 p.m. on April 2nd, 1934, and came to hospital an hour later vomiting and in a collapsed condition. She was treated by gastric lavage and lemon juice *ad lib.*, but swallowing was difficult and painful. During the next twenty-four hours there was a purulent exudate from the nose and mouth, increasing pulse rate, pain, and collapse, and death occurred twenty-six hours after taking the poison. A post-mortem examination, performed fifteen hours after death, showed burning of the tip and back of the tongue, the inner surface of the lips, and all over the tonsils and pharynx. The burnt areas were covered with a soft, brown slough. The oesophagus was deeply injected, but the mucous membrane was not eroded. In the stomach a burnt area, 15 cm. by 10 cm., and deep brown in colour, was found extending over the centre of the greater curvature. The mucous membrane was soft, but the wall of the stomach under the burnt area was firmer than the surrounding wall. The stomach contents consisted of a yellow fluid of slightly acid reaction. There was oedema of the lungs, but no consolidation. The liver exhibited cloudy swelling and the kidney congestion. The thymus was large and glandular, and all the lymphatic glands, especially in the neck, were large, soft, and dark red in colour.

Surgical Cases

The remainder consisted of a group of surgical cases under the care of Dr. C. H. Cuff, to whom we are indebted for certain clinical data. Stricture having developed, it was necessary to resort to gastrostomy in two instances and jejunostomy in another. It is interesting to note that stricture of the oesophagus was successfully treated by operation (gastrostomy), for the first time in 1885, by Loreta,¹¹ in the case of a patient of 24, who had swallowed caustic alkali. Since then surgical treatment had been resorted to with considerable benefit. In cases of non-malignant strictures of this nature, Grey Turner¹² has recently recorded striking success with the operation of oesophago-gastrostomy.

In the first of these cases (No. 6) gastrostomy, by Frank's method, was successfully performed, but the patient, aged 5, died the next day from peritonitis.

The second of this group (No. 5) was that of a Turkish girl, aged 3, who accidentally swallowed a little caustic soda solution on July 3rd, 1933, the exact amount being unknown.

The usual symptoms came on, and some burning of the face, lips, and tongue with oedema were observed on arrival at hospital shortly after. This appeared to be a mild case, and after two days the parents removed the child against medical advice. The subsequent history illustrates the necessity for continued observation and treatment in subjects which appear at first sight to be mild cases of alkali poisoning. On December 18th, 1933, the patient re-entered hospital unable to swallow any liquid, and it was impossible to pass any kind of sound through the stricture of the oesophagus which had developed. Radioscopic examination revealed oesophageal stenosis opposite the third costal cartilage, and gastrostomy was performed on December 21st. During three months the patient has made uninterrupted progress on a diet consisting of soup, eggs and milk, dried milk preparations, orange juice, etc., given at four-hourly intervals.

The last case of this group (No. 7) concerned a Turkish girl, aged 16, who attempted suicide on January 19th, 1934, by taking about 5 grams (77.2 grains) of strong caustic soda solution. On arrival at hospital within the hour the patient was suffering from shock, and complained of burning pain in the mouth and stomach. There was also purging and much vomiting of blood. The body temperature was subnormal, varying between 97° and 98° F. It was possible to pass a small sound, and lemon juice was given freely. The patient recovered from shock and appeared to be definitely improving when the relatives removed her after she had been one week in hospital. On February 12th she returned to hospital—this time unable to swallow even liquids—and was, in consequence, much emaciated. Radioscopy revealed a stricture of the oesophagus opposite the fifth costal cartilage. The patient continued to lose strength, and it was suspected that the pylorus also was stenosed. On operation this was found to be the case, and palliative jejunostomy was carried out (March 2nd). The patient was afterwards maintained on a diet of citrated milk, eggs, soup, meat extracts, olive oil, liquid glucose, etc., but the emaciation was progressive, and death took place on April 2nd.

Conclusions

Poisoning by caustic alkali in Cyprus has increased remarkably during the last year, and has justified Government action in restricting its free use for domestic purposes. In all the cases studied the poison was obtained in this way. Of these the suicides were among the adults¹ and the accidental cases among the children. The fatal dose of caustic soda is less than 10 grams, and a quantity of 5 grams, in solution, has produced the most serious complications of stricture. There was perforation of the oesophagus in one fatal case, and possibly perforation of the stomach in another, while in another three cases stricture developed. Among the pathological conditions found post mortem, areas of necrosis in the liver and kidney have been observed (Case 4). It is evident from a study of these cases that the prognosis in alkali poisoning is doubtful, and that even in what appear to be mild cases the need for long-continued observation and treatment is essential.

We are indebted to Dr. C. H. Cuff, acting director of health, for permission to publish the results of this investigation.

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IMPORTANCE OF LOCAL FACTORS IN THE ONSET OF PULMONARY TUBERCULOSIS

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(With Special Plate)

The problem of the origin of recent tuberculous infiltrations of the lung ("Fruhinfiltrate," "Rundherde") is still unsolved. One group of authorities emphasizes exogenous reinfection, whereas other investigators direct their attention to the possibility of an endogenous development of the isolated pulmonary lesion of the adult. The importance of local factors in the location of recent tuberculous foci is perhaps not sufficiently recognized. Thus it may be of interest to publish the two following cases.

History of Case I

The patient, a male aged 30, was in the Army (R.A.S.C.) for seven years, from 1922 to 1929. He was said to have been a heavy drinker until married. There was a family history of tuberculosis. The illness began with a gradual loss of weight and cough. For three months before admission there was blood staining of sputum and also morning vomiting.

POST-MORTEM EXAMINATION

Necropsy revealed carcinoma of the pancreas with metastases in the liver, spleen, kidneys, suprarenals, thyroid, right lung, and lymphatic glands all over the body.

Both upper lobes of the lung showed groups of miliary tubercles with areas of slight fibrosis and emphysema. The left lower lobe was normal, except for small groups of miliary tubercles at the apex and an old calcified focus in one of the lymphatic glands at the bifurcation of the bronchus. The right lower lobe contained a solid mass, of about the size of a tennis ball, close to the hilum: the tissue was firm and yellow, and resembled that of the enlarged mediastinal and abdominal lymphatic glands. The centre of this mass contained some anthracotic lymphatic glands with foci the size of a pea (carcinomatous metastases). Around the large mass was a group of small nodes with distinct walls, some of them liquefied (tuberculous foci) (Plate, Fig. 1). The pleura was thickened and adherent over the right lower lobe.

HISTOLOGICAL EXAMINATION

The tumour in the pancreas, and the secondary nodes in liver, spleen, suprarenals, kidneys, and lymph glands, showed on section solid carcinoma, partially adenocarcinoma.

In the upper lobe of the right lung there was fibrosis, with emphysema; proliferating tuberculous foci in the nature of miliary tubercles, rich in connective tissue and with many giant cells in various places; and caseous endarteritis of smaller vessels.

In the right lower lobe the big node close to the hilum was a solid carcinoma. In the foci surrounding this node caseous tissue with partial liquefaction was found. There were numerous tubercle bacilli in several foci, especially those made up of leucocytes; there were fewer bacilli in the large caseous foci.

Further examination revealed recent, partially liquefying, round tuberculous foci in this lobe. The foci resembled those described as early round infiltrations giving rise to the isolated pulmonary tuberculosis of the adult (so-called "Rundherde"). Such foci in a multiple form were first described by R. von Jaksch-Wartenhorst¹ as pseudo-tuberculous cysticercosis of the lung, later by Lachmann²—as atypical tuberculosis resembling tumour metastasis—and recently by Alb. Fraenkel,³ A. Albert,⁴ and Straub. In this case these foci were found in one part of the lung only (the right lower lobe), where they occurred in the neighbourhood of a large carcinomatous metastasis. The pleura was thickened.

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FIG. 1.—Oesophagus and stomach.

FIG. 2.—Section of cardiac end of stomach.

FIG. 3.—Section of kidney.

FIG. 4.—Section of liver.