Second Operation (Mr. W. J. M. Brandon).—The abdomen was opened through the previous wound. A second much larger stone was removed from the same area as before. The post-operative course this time, though slow, was uneventful.

The first stone was pyramidal in shape, faceted at its base, and 1 in. (2.5 cm.) in diameter. The second stone was shell-shaped and 1½ in. (3.8 cm.) in diameter (see photograph).

Comment

In this case the stones must have been lying silent for some time in the gall-bladder, the smaller stone ulcerating through first, the larger one taking 10 days longer to force its way through the preformed fistula's track. If the significance of the large facet on the base of the first stone had been realized, an earlier diagnosis of the post-operative symptoms could have been made. Both these stones were radio-opaque, as tested post-operatively. This is unusual. The sex of the patient, the lack of previous history, and the intermittent type of intestinal obstruction are typical features of this condition.

Summary

A case is reported in which 20 days after an operation for gall-stone ileus a second stone was removed from the small intestine. The patient eventually recovered.

I would like to thank Mr. W. J. M. Brandon, consultant surgeon, Law Hospital, for his help and permission to publish this case.

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Medical Memorandum

Mercuric Chloride Poisoning Treated with Dimercaprol

Mercuric chloride poisoning with suicidal intent is fortunately no longer fashionable in this country, although it still seems to be common on the Continent and in America. While Longcope and Luetscher (1949) in America reported on 61 cases of mercuric chloride poisoning treated with dimercaprol (B.A.L.), collected over a period of three years, no reports have, to my knowledge, been published in this country. It therefore seems of interest to make known the following case.

CASE REPORT

A man aged 37 swallowed 2 g. of mercuric chloride with suicidal intention at 1 a.m. on May 6. He vomited repeatedly shortly after taking the tablets, but the vomit is said not to have contained solid particles of the substance, but only to have been stained blue. About one hour later he passed a large amount of pale urine and began to have cramps in his hands and feet. At 10 a.m. he had violent abdominal colic with bloody stools. He then took fright and sent for a doctor. He was admitted to hospital one hour later.

He was seen to be a rather underweight man of slender build, but there was no evidence of shock and no gingivitis; the tongue was coated but not dry. The pulse was regular and the blood pressure 110/80. The bladder was not palpable suprapubically, but no urine had been passed since 2 a.m. He complained of some abdominal colic and of painful cramps in his limbs.

He was given an immediate injection of 100 mg. of dimercaprol, and two hours later a further 200 mg. The injections were repeated at four-hourly intervals for four doses, and then 100 mg. was given four-hourly for a further four doses. The patient reported very shortly after the first injection that the cramps in his limbs were abating and he had no abdominal colic and no bloody stools.

The first specimen of urine was obtained at 10 p.m. in scanty quantity and loaded with protein; the sediment contained casts in most unusual numbers, some hundreds to a field. Next day the patient felt comfortable. His blood pressure was 120/70. The urinary output was still scanty and showed gross proteinuria, and blood urea was 46 mg. per 100 ml. The urinary output increased considerably from the third day, but there was a heavy trace of protein in the urine up to May 11. His blood pressure was stable at 120/70. On May 15 the sediment still contained a few casts. The patient was allowed up, and was discharged home on May 19.

The total dose of dimercaprol given was 1.6 g. over a period of 72 hours.

COMMENT

Although early vomiting may have prevented full absorption of the dose taken, it is likely that the consequences would have been fatal without appropriate treatment. Among a series of 86 cases of mercuric chloride poisoning reported by Longcope and Luetscher (1949), without treatment with dimercaprol, there were 27 deaths: death occurs in most cases of poisoning with 1 g. and more of the compound. Suppression of urine after an early diuresis, and bloody diarrhoea, point to a heavy absorption of the poison.

Following on the original publication by Peters *et al.* (1945) on the value of dimercaprol in the treatment of arsenical poisoning, Waters and Stock (1945) pointed out its possible use in the treatment of poisoning with other heavy metals, including mercury. Further, Gilman *et al.* (1946) investigated experimentally in cats and dogs the protective action of dimercaprol in poisoning with mercury, and reported favourably on it. The first and most important contribution to human therapeutics was by Longcope *et al.* (1946). They treated 23 cases of mercuric chloride poisoning with one death; one patient who recovered had swallowed 20 g. of the poison. Longcope and Luetscher (1949) reported a further 38 cases so treated, with only one other death. The dosage recommended by them is of 300 mg. for the first injection and a further two or three injections of 150 mg. at four-hourly intervals. A very important point is that treatment should be started at the earliest time within two or three hours from taking the poison. The one death reported occurred when treatment was delayed until 13 hours after poisoning.

Dimercaprol is known to produce toxic symptoms itself if given in a dose of 5 mg. and over per kg. body weight. These symptoms comprise nausea, vomiting, headaches, generalized aches and pains, burning sensations in the mouth, nose, and eyes, sweating, restlessness, weakness, and pains in the limbs, jaws, and trunk muscles. The heart rate is often increased, and there may be a rise in both systolic and diastolic blood pressure. These signs and symptoms subside within four hours (Peters *et al.*, 1945). The initial dose of 300 mg. recommended by Longcope may be expected to produce toxic symptoms in a number of patients of rather slender build; these symptoms have not been reported in the treatment of acute arsenical and mercuric poisoning, perhaps because the drug combines very rapidly within the body with heavy metals and is no longer available in free form. In comparison with the extreme danger to life of poisoning with mercuric chloride the risk of toxic reaction from the first large dose of dimercaprol is certainly inconsiderable.

The case described above made a remarkable recovery, as treatment was delayed for 12 hours and the first dose of the antidote was inadequate.

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