A NOTE ON EPIDEMIC DROPSY

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Several articles have appeared in the medical literature of the last few years referring to epidemic dropsy in India and other countries of the East, and associating outbreaks of the disease with the use of mustard oil for cooking purposes. The most recent we have seen is that by Lal and Roy (1937), who give a description of the disease and the various theories that have been put forward as to its causation, and who have come definitely to the conclusion that the ingestion of mustard oil is the cause. They admit that the majority of people who use mustard oil for cooking do not get the disease, and conclude that only certain forms of the oil sold commercially are toxic. In spite of several experiments they were unable to distinguish between harmless and harmful mustard oil. The Annual Report of Fiji for 1927, quoted by J. N. Leitch (1930), describes an outbreak there in which mustard oil certainly appears to have been the cause, and gives a detailed account of the symptomatology.

Three Recent Cases

Three cases which have been under our care recently at the General Hospital, Singapore, would seem to add corroborative evidence to these conclusions. The syndromes present suggested to us a common toxic origin, such as from some drug or plant taken internally. We consulted Professor R. B. Hawes of the Singapore College of Medicine on the matter, and he informed us that he had come across a similar syndrome in a few people some years previously, and that it had corresponded closely with mustard-oil poisoning or epidemic dropsy as described in the Fiji Annual Report mentioned above.

Case 1.—A male Bengali was admitted to hospital on June 17, 1937, complaining of general malaise. There was cardiac irregularity due to premature beats, microcytic anaemia, and oedema of the legs with the overlying skin of a dusky purplish colour. There was no fever while in hospital. He improved gradually with rest in bed and iron internally, and was discharged comparatively well on July 29, though still with slight oedema of the legs.

Case 2.—The wife of Case 1, also a Bengali, was admitted on June 24, 1937, complaining of pain and swelling in both legs and of one month's continued fever. Her temperature was 101° F. on admission, but this rapidly subsided in hospital. Her legs were oedematous from knee to ankle, tender, and painful, and were discoloured a peculiar mottled purplish red, rather more strikingly so than her husband's. There was a loud systolic cardiac murmur, heard best over the pulmonary area, as well as tick-tack rhythm. She was treated with iron for the anaemia, and with methylene blue (on account of a possible toxic element in mustard oil being a cyanide derivative.) She was discharged much improved on August 1, but with considerable anaemia still present.

Case 3.—The sister of Case 2 was admitted on June 27, 1937, seriously ill. Her legs were oedematous and intensely painful and tender; she also had had fever for one month, and this took some time to subside in hospital. The patient's legs were still more strikingly discoloured with purplish-red mottling. There was a dry irritating cough, and dry coarse crepitations all over both lungs. The heart was rapid and

slightly irregular, with tick-tack rhythm, a loud mitral systolic murmur, and an accentuated second sound at the pulmonary area. The urine was normal and the blood urea 36 mg. per 100 c.cm. Teleroentgenography of the heart showed enlargement, but when repeated at the end of treatment there was a considerable reduction in size. Microcytic anaemia was present, a little more severe than in the other two cases; the red blood cell count was 2,600,000 per c.mm., and the haemoglobin 44 per cent.; there was slight leucopenia. On July 15 she complained of blindness in one eye. On examination small scattered retinal haemorrhages were seen, one on the macula; these all cleared up before the patient was discharged on August 15, when the sight of both eyes was good. She was treated on the same lines as Case 2, with additional symptomatic treatment for the cough. On discharge there was still some anaemia and a pulse of rather poor volume, but the cardiac murmur had practically disappeared.

Conclusions and Commentary

These three patients had lived in the same house and shared the same meals; a fourth member of the family, a brother of the two girls who had previously lived with them, stated that he had also been affected in a mild form, and that he had suspected the food and had accordingly changed his abode. Mustard oil was used daily in the household cooking, and had been so used constantly in the past without ill effect. Some of the oil from the household was subjected to chemical analysis and also used for animal-feeding experiments; these were kindly undertaken by the Government analyst, Mr. M. Jamieson. The results were entirely negative, but it is doubtful whether the oil we obtained was from the same tin as that which had caused the illness; all the contents of that tin had probably been consumed before the patients came to hospital. On inquiry we found that a few other cases had occurred in the town, and we obtained other specimens of oil for examination, but none was found to be toxic to animals.

The syndromes of these patients fit in well with the usual description of epidemic dropsy, and differ from beriberi by the presence of the purplish-red mottling of the skin of the legs, the retinal haemorrhages, the fever, and the lung signs. The disease throughout the world appears only to have been observed among people who use mustard oil for cooking their food, and then merely in sporadic outbreaks; it is strange that the toxic element present has so far defied analysis.

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REFERENCES

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G. de Flora and A. Crocetta (*Minerva med.*, December 2, 1937) state that though the hypotensive action of acetylcholine was demonstrated by Hunt and Taveau in 1915, the introduction of this drug into clinical medicine did not take place until 1928, when Villaret and Besançon were among the first to use it in various vascular disturbances of spastic origin, in scleroderma, and in lead poisoning with encouraging results. Their findings were confirmed by numerous Italian workers, but later investigators showed the extremely transient action of the drug, a dangerous diphasic stage, and an injurious influence on the heart. Subsequently, however, it was shown that the action of acetylcholine was counterbalanced by eserine, and the present authors found that the combination of acetylcholine with eserine made the hypotensive action of the former much more persistent without causing any dangerous diphasic stage.