

Medical Memoranda

Cantharides Intoxication

Brit. med. J., 1967, 4, 33

Preparations containing cantharides, commonly known as "Spanish fly," have been used since the time of Hippocrates for conditions such as dropsy and amenorrhoea, and for the aphrodisiac properties that it is wrongly thought to possess. Cantharides is the active component contained in the ovaries, soft tissues, and blood of the blister beetle *Cantharis vesicatoria*. It contains not less than 0.6% of cantharidin (3:6-epoxy-1:2-dimethylcyclohexane-1:2-dicarboxylic anhydride), which is the active principle. The fatal dose of cantharidin is described as being less than 60 mg. (Nickolls and Teare, 1954).

Only four cases (*Pharm. J.*, 1963; Nickolls and Teare, 1954; Craven and Polak, 1954; Lécuyer, 1954), all terminating in death, have been reported in the English medical literature in the past 25 years. In the world literature 30 cases have been described since 1900.

CASE HISTORY

A man aged 42 was admitted to hospital at 2.15 a.m. on 9 August 1966. He had previously been in good health, except for two episodes of "nephritis" when aged 10 and 22 years.

At 5.30 p.m. he had taken a teaspoonful of liquor epispasticus, which he knew contained cantharidin (approximately 20 mg.) to increase his libido, even though he had read of the man who caused the death of two typists by concealing cantharides in coconut ice (Nickolls and Teare, 1954; Craven and Polak, 1954).

One hour after taking the mixture symptoms started with the onset of frequency of micturition and severe dysuria. Approximately two hours later he felt nauseated and vomited. He micturated five times between 9 and 10 p.m., each specimen becoming progressively more blood-stained. Half an hour later he was passing pure blood, and had abdominal discomfort accompanied by bouts of diarrhoea. He continued to have haematuria and developed priapism; a few hours later he became worried and decided to seek medical advice.

On examination he was pale and extremely anxious. There was no pyrexia and his blood pressure was 120/80. The mucous membranes and throat were reddened, but blistering was not evident. Generalized tenderness was present over the abdomen and the bladder was palpable. The penis was erect and blood was present at the tip of the urethra. The central nervous, respiratory, and cardiovascular systems were normal.

The urine was loaded with protein (300 mg./100 ml.) and blood was visible macroscopically; pus, red blood, and scanty epithelial cells were seen on microscopy. No casts were present.

Laboratory Investigations.—Serum urea was 41 mg./100 ml., serum sodium 133 mEq/l., potassium 4.4 mEq/l., and bicarbonate 22 mEq/l. Total protein 6.7 g./100 ml. His haemoglobin was 13.8 g./100 ml., and the white blood cell count was 16,300/cu. mm. E.C.G., chest, and straight abdominal x-ray films were normal.

It was thought that a stomach wash-out performed nine hours after ingestion would be of no value. He was therefore treated with a high fluid intake of milk, a magnesium sulphate enema, analgesics, and sedation.

The next day ulceration and blistering of the mucous membranes of both cheeks, the tongue, and palate were observed. These responded well to hydrocortisone pellets. His blood pressure was maintained, and the daily urinary output remained at approximately 2 litres per 24 hours. The priapism subsided spontaneously and within four days the urinary abnormalities had resolved.

When seen as an outpatient one month later he was extremely fit. His haemoglobin was 13.4 g./100 ml. and serum urea 35 mg./100 ml., and his urine showed no abnormality.

DISCUSSION

Liquor epispasticus contains: cantharidin 400 mg., castor oil 2.5 ml., colophony 1.2 g., plus acetone to 100 ml.

Acute renal damage after cantharides poisoning was documented as long ago as 1913 (Pearce). Dérobert and Le Breton (1957) described the case of a 77-year-old man who died from cantharidin poisoning, and at necropsy the most significant findings were those of acute tubular necrosis. Nickolls and Teare (1954) showed that microscopically there was haemorrhage into the tubules, producing tubular epithelial damage. Lécuyer (1954) stated that the acute tubular necrosis is due to the direct toxic action of the drug. It is possible, however, that the toxic action is aggravated by shock, which results in diminished renal blood flow. Powell (1907) reported chronic renal disease with associated contracted kidneys after cantharides ingestion.

No specific treatment is available. Csiky (1958) described four cases of pure cantharides poisoning; all the patients lived. His treatment was similar to ours, but he stated that oil, milk, and alcohol should not be taken as they aid absorption of the drug.

Management of the renal disorder and the associated fluid and electrolyte imbalance is probably the most important aspect of cantharides ingestion. As diminished renal blood flow and a reduced glomerular filtration rate are well described, acute tubular necrosis must be anticipated. Treatment will depend on urinary output, specific gravity, and the signs and symptoms of dehydration.

In none of the reported cases was there any evidence of an increase in libido. The myth that cantharides is an aphrodisiac should be destroyed, as its use for this purpose is fraught with danger and it is this property alone that perpetuates its illegal sale.

My thanks are due to Professor M. D. Milne for permission to publish this case, and to Drs. J. D. Swales and D. G. Gibson for their helpful advice.

R. D. ROSIN, M.B., B.S.,

Formerly House-physician, Professorial Medical Unit,
Westminster Hospital, London S.W.1.

BIBLIOGRAPHY

- Andrewes, C. H. (1921). *Lancet*, 2, 654.
Craven, J. D., and Polak, A. (1954). *Brit. med. J.*, 2, 1386.
Csiky, P. (1958). *Arch. Toxicol.*, 17, 27.
Dérobert, L., and Le Breton, R. (1957). *Ann. Méd. lég.*, 37, 41.
Goodman, L. S., and Gilman, A. (1965). *The Pharmacological Basis of Therapeutics*, 3rd ed., p. 983. New York.
Lécuyer, M. A. (1954). *Brit. med. J.*, 2, 1399.
Lipsitz, S. T., and Cross, A. J. (1917). *Arch. intern. Med.*, 20, 889.
Morgulis, S., and Muirhead, A. L. (1919). *Ibid.*, 23, 190.
Nickolls, L. C., and Teare, D. (1954). *Brit. med. J.*, 2, 1384.
Oaks, W. W., Ditunno, J. F., Magnani, T., Levy, H. A., and Mills, L. C. (1960). *Arch. intern. Med.*, 105, 574.
Pearce, R. M. (1913). *J. exp. Med.*, 17, 542.
Pharm. J., 1953, 171, 467.
Powell, R. D. (1907). *Lancet*, 2, 1296.