Clinical Toxicology

Hemlock water dropwort poisoning

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Summary: Severe plant poisoning is relatively uncommon in adults. We report two adults who ingested hemlock water dropwort roots, having mistaken them for wild parsnip. One developed prolonged convulsions, severe metabolic acidosis and respiratory distress requiring mechanical ventilation. The toxin – oenantheotoxin – was detected in the gastric aspirate and measured by high performance liquid chromatography.

Introduction

Hemlock water dropwort (*Oenanthe crocata*) is probably the most poisonous plant found in Britain. It belongs to the family Umbelliferae, which has other poisonous members – water hemlock (genus *Cicuta*) and hemlock (genus *Conium*). A 4 to 6 foot perennial which grows in wet river banks, its stem resembles celery and the roots, which are the most poisonous part, resemble small parsnips (Figure 1). Livestock poisoning occurs sporadically but only 14 human poisonings were reported between 1900 and 1978, mostly in children.¹ The mortality rate is, however, about 70%.

Case reports

A 26 year old man ingested a meal of ducks' eggs, nettles, and the boiled leaves and bulbous roots of a plant picked on the Thames riverbank. Forty minutes later, he developed nausea, abdominal pain, tachypnoea, ataxia and had a generalized convulsion. On admission to hospital, he was experiencing major tonic/clonic seizures and was cyanosed with widely dilated pupils. His pulse rate was 145/min, and blood pressure 150/90 mm Hg. 100% oxygen was given. Arterial blood gas analysis revealed a pH of 6.68, P_{aO_2} of 30 kPa, P_{aCO_2} of 5.43 kPa, and a base excess of –36 mmol/l. Sodium bicarbonate, phenytoin and diazepam were administered intravenously, but the convulsions continued and a bradycardia developed.

An intravenous infusion of thiopentone was commenced and the patient was paralysed, intubated and ventilated. Gastric lavage produced a large quantity of plant material. A fresh specimen of plant was identified by botanists as hemlock water dropwort.

The patient received 300 mmol of bicarbonate to assist correction of the acidosis, and thiopentone at 2–6 mg/kg/h for 24 hours to depress cerebral electrical activity. Twelve hours after admission his arterial pH was 7.4, the venous plasma bicarbonate concentration was 23 mmol/l, and the plasma urea was normal. Subsequent analysis of plasma lactate revealed a concentration of 10 mmol/l on admission which fell to 5 mmol/l within 10 hours. After 30 hours in hospital, no further generalized convulsions occurred on phenytoin alone, although the electroencephalogram still showed bilateral slow wave abnormalities. The patient was extubated after 60 hours, and there were no clinically obvious neurological sequelae.

Serial biochemical results revealed markedly raised activities of aspartate transaminase, with a peak value 258 IU/l (reference range 5–35 IU/l), creatine kinase, which reached 5995 IU/l (reference range 30–180 IU/l), and lactate dehydrogenase. These enzyme activities remained elevated for 10 days. Gamma glutamyl transpeptidase activity was also elevated and the plasma bilirubin concentration was slightly raised on day 3, but the alkaline phosphatase activity remained within the reference range.

Case 2

The 20 year old female companion of Case 1 ingested less of the plant and then induced herself to vomit. Gastric lavage was also performed on admission to

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Figure 1 *Oenanthe crocata*.

hospital one hour later. This patient experienced several hours of nausea, mild confusion and paraesthesia. The plasma bicarbonate was 18 mmol/l, the creatine kinase activity was raised at 200 IU/l and the aspartate transaminase activity was greater than 110 IU/l for 3 days, but no other biochemical abnormalities were noted.

Discussion

The main toxic constituent of hemlock water dropwort is oenanthotoxin – an unsaturated higher alcohol ($C_{17}H_{22}O_2$) which was purified by Clarke.² It resembles cicutoxin which is the toxic principle in water hemlock. Oenanthotoxin concentration in the plant roots is highest in winter and spring, and ingestion of very small amounts may prove fatal.

The pharmacological effects of oenanthotoxin have been studied in animals and include an initial increase in respiratory rate and hypotension followed by hypertension.^{2,3} Severe convulsions are common in animals and man and may be due to antagonism of an inhibitory transmitter in the brain stem. In rabbits pentobarbitone reduces the convulsive effects.³ Barbiturates have been used in patients poisoned by cicutoxin⁴ and successful treatment with large doses of thiopentone has been described.⁵ Ten grams of thiopentone was administered in 24 hours to our

patient with oenanthotoxin poisoning and this successfully depressed cerebral electrical activity.

Oenanthotoxin can now be identified by a number of methods,⁶ and measurement has been performed in one previous case – a fatality – where there was 1 mg in 50 ml of stomach contents. In Case 1 ultraviolet absorption spectroscopy revealed the characteristic absorption features of the toxin in the stomach contents although other absorbing materials were also present. High performance liquid chromatography revealed the toxin concentration to be 4 mg/l. Despite the severe toxicity oenanthotoxin was undetectable in plasma or urine (lower detection limit of assay 50 µg/l), and was not detected in the stomach contents of Case 2. It appears that the fatal quantity of oenanthotoxin may be as low as 10 to 20 mg, which is contained in about 20 grams of the *O. crocata* root.

Both patients had a metabolic acidosis. This was extremely severe in the male with a marked base deficit and a high plasma lactate concentration, and it is unusual for such a patient to survive. The plasma chloride concentration was raised but the anion gap was initially 34 mmol/l. The plasma lactate concentration was 10 mmol/l but it would appear that other unmeasured anions were also present. The oenanthotoxin and its metabolites were only present in minute quantities in the plasma and could not have contributed directly, and there was no significant impairment of renal function. The increased lactate concen-

tration was probably due to an increase in anaerobic metabolism as a result of the hypoxia prior to treatment or a direct effect of the toxin on aerobic cellular metabolism.

In this patient the main effective treatment of the acidosis was the mechanical ventilation. This was performed to allow administration of thiopentone and paralysis as treatment for the convulsions and to control correction of the acidosis. The ventilation was adjusted to reduce the arterial P_{CO_2} which resulted in a further rise in the arterial pH. Sodium bicarbonate was also administered initially but the quantity given was much less than that which would be needed to correct promptly such a severe acidosis. The fall in plasma lactate suggested that the tissue production returned towards normal and that the liver was able to metabolize the lactate present in the circulation.

The marked rises in plasma creatine kinase, aspartate transaminase and lactic dehydrogenase activities

in Case 1 may have been caused by the convulsive activity and the anoxia. A direct myotoxic action is also possible, and Case 2, who did not experience hypotension or convulsions, also had a raised creatine kinase and aspartate transaminase.

Poisoning from hemlock water dropwort and related plants is a danger of ingestion of natural vegetation because these plants are widely distributed in Europe and North America and do resemble edible plants. Ingestion of small quantities often proves fatal but prompt identification using an atlas of poisonous plants,^{7,8} and immediate treatment may improve the prognosis.

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