Ricin poisoning: a case of internet-assisted parasuicide

Introduction

A 32-year-old man presented following ingestion of castor oil seeds in an attempt to end his life. Castor oil seeds, when pulped, release ricin. He went on to suffer ricin toxicity, but survived the episode with supportive care. Following ricin ingestion, symptoms mimic that of common gastrointestinal illness. This suicide attempt had been researched on the internet and seeds procured via eBay. Clinicians need to be aware that unusual toxins are recommended and sold online for the purposes of self harm.

Discussion

Ricinus communis (the castor oil plant) is a small wooden tree which grows in South Africa, India, Brazil and Russia (Rana et al, 2012). Ricin toxin is derived from castor bean pulp. Weaponized ricin is most famously associated with the assassination of Bulgarian defector Georgi Markov (shot in the leg with a pellet containing ricin). Ingested castor beans must be mashed or chewed to release toxin. There is no ricin in castor oil (Audi et al, 2005).

Between 1 and 20 mg of ricin per kilogram of body weight constitutes a lethal oral dose in humans. This equates to approximately eight beans (Audi et al, 2005). Case reports of ricin ingestion document effects ranging from mild to lethal with ingestion of between one half to 30 beans. One patient died after ingestion of only three beans (Challoner and McCarron, 1990).

Ricin belongs to a family of related toxins known as the ribosome inactivating proteins. It exists as a two-chain toxin, comprising an A and B protein chain. The B chain binds cell surface glycoproteins and enters the cell by endocytosis. In the cytoplasm the A chain inactivates ribosomes, blocking translation of mRNA to protein and causing cell death.

Following ingestion, clinical symptoms begin within 4–6 hours. Non-specific abdominal pain precedes vomiting, diarrhoea and in some cases gastrointestinal bleeding. Profound fluid loss leads to shock.

Inhalation of ricin can cause cough, fever, dyspnoea, pulmonary oedema and respiratory failure. Animal studies show the lethal dose is lower via inhalation than via the oral route (Audi et al, 2005).

A review of 88 cases of castor bean intoxication (Challoner and McCarron, 1990) listed haemolysis as a clinical manifestation in three cases, in addition to the more frequently occurring symptoms listed above. The authors identify that erythrocyte agglutination and haemolysis does not occur with chemically pure ricin; these properties have been attributed to other toxins present in castor beans.

There is no validated laboratory assay for the detection of ricin in body fluids. Since early clinical features of ingestion mimic common gastrointestinal illnesses, the physician must be alert to this clinical diagnosis in the context of an appropriate history.

There is no antidote, and treatment is supportive. Activated charcoal may be used if presenting within 1 hour of ingestion. The toxin cannot be removed by dialysis.

Figure 1. Ricin beans from the patient's stock. Identity confirmed as Ricinus communis (castor oil plant) by seed morphologists at Kew Gardens.

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Case Report

A 32-year-old man was admitted with deliberate self-poisoning. He ingested, and chewed, 40 castor oil plant seeds (*Figure 1*). Castor oil beans contain ricin. Five hours after ingestion he started vomiting. He presented seeking symptomatic relief from his nausea but fully disclosed his method of self-poisoning and suicidal intent on admission. Knowing ricin toxicity has no cure he expected (and hoped) to die following admission. This planned suicide attempt had been researched on the internet. The seeds were bought from eBay.

Initial observations, blood tests, chest radiograph and electrocardiogram were unremarkable. His abdomen was soft and non-tender. Treatment consisted of intravenous fluid and anti-emetics.

Within hours he developed profuse, brown, watery diarrhoea. This worsened over 2 days, reaching a maximum frequency of 34 stools/day. Blood appeared in the stool 48 hours after the overdose. Prophylactic enoxaparin was withheld because of bloody stools. He developed cramping abdominal pain, acute renal failure (creatinine 188 mmol/litre), and a compensated metabolic acidosis (pH 7·38, pO_2 11·3 KPa, pCO_2 3·85 kPa, base excess -8·1 mmol/litre, bicarbonate 18·9 mmol/litre). The calculated anion gap was normal (4 mEq/litre), reflecting gastrointestinal loss of bicarbonate. He was haemoconcentrated (haemoglobin increased from 18·1 to 21·3 g/dl) and clinically shocked (blood pressure = 98/50 mmHg, pulse=105) on day three. Urine output was monitored hourly and intravenous fluids were sped up.

Within 3 days the diarrhoea and biochemical abnormalities resolved. He developed pleuritic chest pain and a small pulmonary embolism was confirmed on computed tomographic pulmonary angiography. Following anticoagulation with low molecular weight heparin he was discharged to a psychiatric unit (9 days after admission).

Ricinus communis is being studied as a potential therapeutic agent. One study demonstrated in-vitro antibacterial activity against *Staphylococcus aureus* and *Pseudomonas aeruginosa* (Pesaramelli et al, 2012) while other investigators are focussing on ricin as a novel chemotherapeutic agent (De Virgilio et al, 2010; Zhang et al, 2012).

A pilot phase 1B clinical trial of recombinant ricin vaccine (RiVax) showed induction of neutralizing antibodies in healthy human volunteers (Vitetta et al, 2012).

This case highlights how patients can research novel approaches to suicide and procure toxins. Clinicians must be aware that patients may source information on the topic from the internet. **BJHM**

LEARNING POINTS

- Ricin ingestion presents in a similar manner to common viral gastroenteritis.
- Patients attempting suicide may have consulted an online 'manual'. This may facilitate more unusual and effective approaches.
- Ricin has been used as an agent of bioterrorism. Toxic effects vary depending on the route of administration.

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De Virgilio M, Lombardi A, Calindro R, Fabbrini M (2010) Ribosome-inactivating proteins: from plant defence to tumor attack. *Toxins* **2**: 2699–737

Pesaramelli K, Jahnavi V, Keerthi K, Sravanthi C (2012) Evaluation of antibacterial activity of herbs. *International Research Journal of Pharmacy* **3**: 230–2

Rana M, Dhamija H, Prashar B, Sharma S (2012) Ricinus communis L – A review. *International Journal of Pharmtech Research* **4**: 1706–11

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Sudden apparent worsening of diabetic retinopathy leading to a diagnosis of chronic myeloid leukaemia

A 65-year-old woman with well-controlled type 2 diabetes presented with a sudden decrease in visual acuity bilaterally (right eye 6/60, left eye 6/18). The visual acuity had been documented as 6/6 in both eyes just 3 months previously, with stable mild diabetic retinopathy. Fundoscopy revealed extensive bilateral intraretinal haemorrhages (*Figures 1* and *2*).

Such sudden apparent worsening of diabetic retinopathy despite reasonable glycaemic control (glycosylated haemoglobin 8.0%) prompted further investigations to rule out causes of vascular occlusion. Full blood count showed an elevated white cell count (94.8x10^9/litre, normal range 3.6–11.0x10^9/litre) with neutrophilia (48.3 x 10^9/litre, normal range 1.8–7.5x10^9/litre) and myelocyte peak. All stages of myeloid precursors were seen with partially degranulated basophils. She was Philadelphia chromosome (t9:22 translocation) positive. These results were consistent with chronic myeloid leukaemia. Following an urgent haematology review, imatinib (400 mg once daily reduced to 200 mg once daily after a week) was started with good results. The patient's blood count improved dramatically after 5 weeks of treatment, with a white cell count of 6.4x10^9/litre.

Prompt recognition of disease led to early referral and initiation of life-saving systemic treatment with imatinib, a signal transduction inhibitor designed to competitively inhibit BCR-ABL tyrosine kinase activity. According to the National Institute for Health and Clinical Excellence (2012) guidance, imatinib is recommended as first-line treatment in Philadelphia chromosome-positive chronic myeloid leukaemia. Imatinib has transformed this leukaemia with a previously optimistic life expectancy of 4–6 years with interferon-based treatments into a true chronic illness with overall survival rates that appear to be increasing each year (Smith, 2011). **BJHM**

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Figure 1. Image of right fundus.

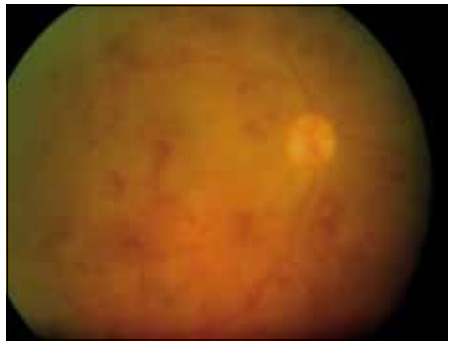

Figure 2. Image of left fundus.