nucleotides, amino acids, oligopeptides, cardiotoxins, nephrotoxins, haemolytic toxins, phosphodiesterase, phospholipase A, hyaluronidase, acetylcholine-esterase, glycosaminoglycans, histamine, serotonin, 5-hydroxytryptamine and proteins that inhibit protease, angiotensinase, succinate-dehydrogenase, ribonuclease and 5-nucleotidase. Multiple toxins may be present in the venom of a single species which can produce a synergic effect in the victim [3]. Many other ion channels are also involved, and the term ‘autonomic storm’ reflects the stimulation of the parasympathetic system.

Although the toxins in Sri Lankan white scorpion venom have not been definitely identified, two mechanisms are thought to contribute to its cardiotoxic properties. Firstly the direct cardiotoxic effect of the venom causing toxic myocarditis by reduction of Na-K-ATPase and adrenergic myocarditis by releasing adrenaline and noradrenaline from neurons, ganglia, and adrenals. The second mechanism is myocardial ischaemia caused by coronary spasm due to release of vasoactive, inflammatory and thrombogenic peptides and amine constituents (histamine, serotonin, bradykinin, leukotrienes and thromboxane) [4]. This acts on the coronary vasculature and induces coronary artery spasm and facilitates platelet aggregation, leading to thrombosis [5]. Prompt treatment with prazosin hydrochloride and supportive care has reduced the morbidity and mortality from scorpion stings in hospitalised patients.

**Ethics**

Patient has given informed, written consent for this publication.

**Conflicts of interests**

There are no conflicts of interest.

**References**


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**A foreign body in liver mimicking an intrahepatic cholangiocarcinoma**

**A A Pathirana**, **U G D S Manawasinghe**, **N P N Karunaratne**, **V Thusyanthan**


**Introduction**

The presence of foreign bodies in the liver is rare. Usually foreign bodies migrate to liver after perforating the upper gastro intestinal tract and cause an abscess or a granuloma. We report a foreign body in the liver mimicking a malignant neoplasm on imaging.

**Case report**

A 58-year old woman was seen for vague episodic right upper abdominal pain and loss of appetite. She had lost 2-3 kg of weight over two months. Physical examination was unremarkable. She has had a single episode of high fever with chills and rigors, three months previously. The

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fever was associated with vague right hypochondrial pain. She had not noted any features of obstructive jaundice and her bowel habits had been normal. As the symptoms settled within a day she had not sought medical advice.

Blood tests including liver profile were normal except for an elevated CRP of 30 mg/l. Ultrasound scan of abdomen showed a suspicious irregular lesion in the left lobe of the liver. Plain X-ray of abdomen showed no abnormality. Contrast enhanced computed tomography (CECT) of abdomen showed an irregular, heterogeneous lesion of intermediate density occupying segments II and III of the liver. The appearance was similar to that of an intrahepatic cholangiocarcinoma. There were prominent para-aortic lymph nodes (Figure 1).

Discussion

Presence of a foreign body in the liver after upper gut perforation is rare [1]. Such perforation occurs mainly around the stomach and duodenum and can be induced by sharp foreign bodies like fish bones, chicken bones, needles and toothpicks [2]. Most of these perforations do not cause significant symptoms. Probable time of perforation may be assumed by retrospective speculation. Most patients do not recall a particular incident out of the ordinary and may remain silent until an abscess is formed [2].

According to a Swiss study that reviewed 59 cases of foreign body migration, computed tomography demonstration of a thickened gastrointestinal wall in continuity with the abscess was suggestive of foreign body migration [3]. But CECT of our patient showed a prominent liver lesion similar to an intrahepatic cholangiocarcinoma, with misleadingly prominent paraaortic lymph nodes. An unknown foreign body mimicking colorectal liver metastases has been reported [4]. According to two previous case reports, a foreign body could simulate carcinoma in the head of pancreas [5, 6]. Except for those instances most patients had features of a liver abscess. As pre-operative diagnosis by biopsy is not favoured by many hepatobiliary surgeons, foreign body in liver mimicking a neoplasm could remain a diagnostic dilemma.

Ethics

The patient gave informed, written consent for publication of this article including pictures.

Conflicts of interests

There are no conflicts of interest.
References


Fatal poisoning with plant growth regulator – chlormequat

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Introduction

Globally, 30% of suicidal deaths are caused by self-poisoning with pesticides [1]. Deaths due to suicidal or intentional ingestion of plant growth regulators are rare. We report a case of suicide after consuming chlormequat chloride [(2-chloroethyl) tri-methyl-ammonium chloride; C₅H₁₃Cl₂N] a quaternary ammonium compound and a plant growth regulator. It is used widely in agriculture to reduce unwanted longitudinal shoot growth without lowering plant productivity [2]. Chlormequat (Cycocel®) is not approved for plants consumed by humans and animals in USA, but is approved in Europe [2, 3]. In Sri Lanka it is used during cultivation of vegetables, fruits and floriculture. There are four case reports and a case series of seven patients with acute poisoning reported previously [4-9]. Ten out of these eleven cases have been fatal. One was after inhalation and the others were after ingestion of chlormequat chloride. Clinical features of acute poisoning are cholinergic crisis, cardiac arrest, acute pulmonary oedema, respiratory failure and death mostly within an hour of ingestion. Atropine has been used in four cases. Death occurred within a day of exposure in all fatal cases [4-9].

Case report

A 50-year old male farmer was admitted to the emergency treatment unit of Teaching Hospital, Anuradhapura following ingestion of 200 ml of "Cycocel®" under the influence of alcohol. Thirty minutes later he developed abdominal pain, vomiting and dyspnoea. On the way to hospital he became unconscious.

On admission the Glasgow coma scale was three, pupils were small (less than 2mm) and reactive to light, heart rate was 21 beats per minute, blood pressure was not recordable, respiratory rate was eight per minute and peripheral arterial oxygen saturation was undetectable by pulse oximetry. Atropine was given and cardiopulmonary resuscitation initiated. Heart rate increased to 130 beats per minute and blood pressure increased to 120/90 mm Hg. He had bilateral crackles heard over the chest and oxygen saturation was 95% with FiO₂ of 100%. After endotracheal intubation, gastric lavage was performed. Activated charcoal and intravenous cefuroxime were administered.

The chest X-ray showed pulmonary congestion with right lower zone opacities. There was neutrophil leukocytosis and sinus bradycardia. He had metabolic acidosis. The pH was 7.28 (normal range 7.35-7.45), HCO₃⁻ was 18 mmol/l (normal range 22-26) with hypoxia and PaO₂ was 60 mmHg (normal range 75-100). Serum creatinine was 126 μmol/l (normal range 70-120), and serum potassium was 3.2 mmol/l (normal range 3.5-4.5). Clotting tests were normal: PT 12.5s (control 13s), APTT 36s

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