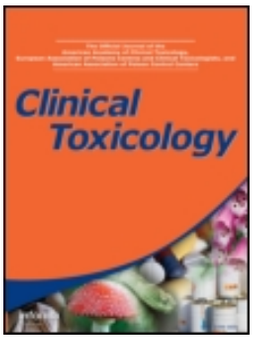

Clinical Toxicology

ISSN: 1556-3650 (Print) 1556-9519 (Online) Journal homepage: informahealthcare.com/journals/ictx20

Sinusoidal obstruction syndrome associated with the ingestion of gynura root

Chen Li, Xue-Song Liang & Cheng-Zhong Li

To cite this article: Chen Li, Xue-Song Liang & Cheng-Zhong Li (2010) Sinusoidal obstruction syndrome associated with the ingestion of gynura root, Clinical Toxicology, 48:9, 962-964, DOI: 10.3109/15563650.2010.527851

To link to this article: https://doi.org/10.3109/15563650.2010.527851

Published online: 20 Dec 2010.

addressed this in our original discussion, and noted that continuing absorption would have made the half-life appear longer. Thus, our patient's true elimination half-life was probably shorter. As the writers pointed out, using this shorter half-life in the equation would yield a larger total body clearance value. This would bolster the argument that CVVH had a smaller impact on the total clearance of acetaminophen.

We did not mean to suggest that we endorse CVVH as a preferred procedure for extracorporeal removal of acetaminophen. It may help remove some of the ingested drug, and assist in restoration of a normal blood pH. However, if rapid removal of large amounts of acetaminophen is necessary, conventional high-flux, high-flow hemodialysis would be superior.

Timothy J. Wiegand
Northern New England Poison Center
Department of Medical Toxicology
Portland 04102
USA

Mary Margaretten
Department of Rheumatology
UCSF
UCSF Box 1369
San Francisco 94143
USA

Kent R. Olson
California Poison Control System and
The University of California,
San Francisco, San Francisco Division
San Francisco General Hospital
San Francisco 94143
USA

Nosocomial scorpion envenomation: An unusual mode of scorpion sting

To the Editor:

Scorpion envenomation is common in tropical and subtropical regions. In Tunisia, almost 40,000 patients are stung per year, around one thousand of them have systemic features requiring hospitalisation with about 10 patients eventually die.[1] Severe scorpion envenomation requiring hospitalisation in the intensive care unit (ICU) usually results from a sting by one of the two species: *Androctonus australis* or *Buthus occitanus*.[1,2] Scorpion envenomation is more often observed in the middle and south of Tunisia which are rural endemic areas. Scorpion envenomation occurring in hospital (nosocomial) has not been previously reported.

A 33-year-old male presented in August 2010 to the department of surgery for suspected appendicitis. After biological and radiological explorations, the diagnosis of likely appendicitis was made. A few hours after hospital admission and during changes of his cloths, the patient was envenomed by a scorpion (*Androctonus australis*) requiring his admission in our ICU. Clinical examination on admission showed local pain without inflammatory signs. However, the patient exhibited systemic manifestations with nausea, vomiting, agitation and sweating. Blood pressure on ICU admission was 130/80 mmHg, and heart rate 85/min. There were no signs of respiratory distress, oxygen saturation measured by pulse oximetry [SpO2] was 96% on air room. Electrocardiogram and chest radiograph performed on admission were normal. A diagnosis of scorpion envenomation grade II (with systemic manifestations) was made, and the patient received scorpion antivenom. Evolution was favourable and the patient quickly improved.

We have previously shown that intoxications caused by scorpions in south Tunisia region are mostly seen in hot summer months especially in July and August.[2–5] Moreover, we have postulated that in many of these cases, the patients were stung because of careless behaviour such as walking bare foot. We have never previously encountered scorpion envenomation during hospital stay. This patient came from an endemic region, and we postulate that the scorpion was carried in the patient bag and cloths into hospital.

Our observation shows that scorpion envenomation can occur in hospital as an unusual nosocomial event.

Mabrouk Bahloul, Anis Chaari, Hassen Dammak,
Najla Ben Algia, and Mounir Bouaziz
CHU Habib Bourguiba, ICU, Sfax, 3029 Tunisia

References

1. Goyffon M, Vachon N, Broglio N. Epidemiological and clinical characteristics of the scorpion envenomation in Tunisia. Toxicon 1982; 20:337–344.
2. Bouaziz M, Bahloul M, Kallel H, Samet M, Ksibi H, Dammak H, et al. Epidemiological, clinical characteristics and outcome of severe scorpion envenomation in South Tunisia: multivariate analysis of 951 cases. Toxicon 2008; 52:918–926.
3. Bahloul M, Ben Hamida C, Chtourou K, Ksibi H, Dammak H, Kallel H, et al. Evidence of myocardial ischemia in severe scorpion envenomation: ''Myocardial perfusion Scintigraphy study''. Intensive Care Med 2004; 30:461–467.
4. Bouaziz M, Bahloul M, Hergafi L, Kallel H, Chaari L, Hamida CB, et al. Factors associated with pulmonary edema in severe scorpion sting patients – a multivariate analysis of 428 cases. Clin Toxicol (Phila) 2006; 44:293–300.
5. Bahloul M, Bouaziz M, Dammak H, Ben Hamida C, Ksibi H, Rekik N, et al. Value of the plasma protein and hemoglobin concentration in the diagnosis of pulmonary edema in scorpion sting patients. Intensive Care Med 2002; 28:1600–1605.

Sinusoidal obstruction syndrome associated with the ingestion of gynura root

To the Editor:

Gynura root (*Gynura segefum*) (Lour.) Merr, is a traditional Chinese herbal medicine used for the treatment of bleeding injuries in the rural areas of China. But the root is one of more than 6000 kinds of plants around the globe that contains pyrrolizidine alkaloids (PAs),[1] which

Received 14 October 2010; accepted 18 October 2010.

Address correspondence to Dr. Mabrouk Bahloul, CHU Habib Bourguiba, ICU, Sfax, 3029 Tunisia. E-mail: bahloulmab@yahoo.fr

Received 11 August 2010; accepted 23 September 2010.

Address correspondence to Cheng-Zhong Li, The Department of Infectious Diseases, Changhai Hospital, Second Military Medical University, Shanghai, China. E-mail: leo_lee666@126.com

can cause hemorrhagic necrosis of liver cells, hepatic giant cell disease, and veno-occlusive disease. When PAs reach the liver, they undergo a catalytic reaction of cytochrome P450, due to which dehydrogenation of PAs and dehydrogenation split base called metabolic pyrroles are formed. When glutathione (GSH) is decreased, the metabolic pyrroles may combine with nucleophilic enzymes, proteins, DNA, and RNA, causing a variety of damages. Because the concentration of GSH in sinusoidal endothelial cells is less than that in the hepatocytes, in zone 3 of the liver acinus, sinusoidal endothelial cells are more sensitive to damage than hepatocytes.[2,3] If this condition progresses, it will affect the hepatic vein, and the small vein wall will gradually be hardened. When occlusion occurs, the surrounding liver cells will undergo necrosis, leading to sinusoidal obstruction syndrome (SOS) which is also referred to as hepatic veno-occlusive disease (HVOD).

We report here a case of a patient who was afflicted with hepatomegaly, ascites, jaundice and weight gain after ingesting gynura root. A 37-year-old Asian woman (standard weight was 55 kg) ingested gynura root to correct irregular menstruation, consuming 100 g per day for 1 week. A month later, she started to experience upper abdominal pain, accompanied by fatigue and poor appetite. Since then, the symptoms described above gradually aggravated with abdominal distention. Three months later, the patient was hospitalized. Physical examination showed that jaundiced scleras, hepatomegaly, and shifting dullness were positive. Laboratory tests showed hypoproteinemia (ALB 28 g/L; normal: 35–55 g/L), serum total bilirubin was 41.1 μmol/L (normal: 3.4–17.1 μmol/L), and AST was 54 U/L (normal: 0–40 U/L). She gained 5 kg during the course of her illness. Her abdominal CT scan showed, in non-contrast scan phase, hepatomegaly, uneven density, patchy and low-density changes, and abdominal fluid; in the arterial enhancement scan phase, hepatomegaly was seen and the liver had heterogeneous enhancement in portal venous scan phase and lag phase, there were characteristics of map-like strengthening areas and low-perfusion areas, the two areas were mixed, hepatic veins were not clearly visible, hepatic segments of inferior vena cava were flat, and there was no lateral expansion and collateral circulation of the remote inferior vena cava. A laparoscopic abdominal exploration and liver biopsy operation result showed a congestive liver without nodules. Postoperative pathology showed that the central venous hepatic lobule and hepatic sinusoid were significantly congestive and extensive. The majority of the liver cells at the central areas of hepatic lobule had an obvious atrophy and necrosis, and the individual vein wall had a thickening and hyaline degeneration. The surrounding liver cells of the portal area had a fatty degeneration. Her liver pathology demonstrated that she had a SOS. The patient was treated with ursodeoxycholic acid capsules (750 mg/day), low-molecular weight dextran (500 ml/day), and prostaglandin E1 (10 μg/day). After therapy, her clinical symptoms finally improved and her ascites have subsided. After discharge from the hospital, the patient continued to take ursodeoxycholic acid capsules. Eight months later, her liver function test showed normal results (ALB 39 g/L, serum total bilirubin 15.2 μmol/L, AST 25 U/L), and abdominal B-mode examination showed normal liver size without ascites.

SOS is characterized by a post-sinusoidal portal hypertension; it is a rare disease and its early symptoms are atypical. The diagnosis of SOS depends on its histopathology. The disease is mainly caused by the hematopoietic stem cell transplantation and the usage of high-dose chemotherapy drugs during the liver transplantation phase, as well as the consumption of certain herbs.[4] Defibrotide, tissue plasminogen activator, antithrombin III, prostaglandin E1, low-dose heparin, low-molecular weight heparins and ursodeoxycholic acid are administered to treat SOS. The treatment of liver cirrhosis involves liver transplantation and transjugular intrahepatic porto-systemic shunts (TIPS) on patients who show portal hypertension signs.[5] At present, there are a few reports on the occurrence of SOS after the intake of gynura root[6,7] and almost all SOS patients presented with poor prognosis.[8] The differences between the patient in this case and those in previous cases are: (1) SOS diagnosis is difficult. We used a laparoscopic abdominal exploration and liver biopsy, combined abdominal CT with pathologic examination, and finally obtained a clear diagnosis; (2) A study using an *in vitro* technique indicated the dose of PAs-related toxicity, with necrosis at high concentrations and apoptosis and abnormalities of the cytoskeleton at lower concentrations.[9] WHO has indicated that the lowest intake causing disease may be 1 mg total PAs per day for a 70 kg adult.[10] The patient took a large dose of gynura root, suggesting that although the dose of intake of gynura root with PAs, and duration of medication is to some degree correlated with SOS severity and patient prognosis. Individual factors also played an important role; (3) Illness was obviously relieved through active treatment, and re-examination showed excellent conditions. In most areas of China, people can be easily confused by *panax notoginseng* and gynura root because the Chinese names of panax notoginseng (san qi in Chinese) and gynura root (tu san qi in Chinese) are very similar. Owing to this, people select gynura root for disease treatment over panax notoginseng because it is cheaper. *Panax notoginseng* is a precious Chinese herbal and thus commands a higher price. Such incidences, as the case presented in this study, are common occurrences. The patient in this case took gynura root because of these reasons. Thus, in China, the hepatic toxicity of the gynura root should be given enough attention. The Chinese traditional herbal medicine should meet specific and appropriate standards of safety and quality, and the government should provide risk management actions and public education to the consumers of gynura root.

Dr. Chen Li
The Department of Infectious Diseases
Changhai Hospital
Second Military Medical University
Shanghai, China
The Department of Liver Failure Treatment and Research Center
The 302ed Hospital of PLA
The Beijing, China

Dr. Xue-Song Liang and Dr. Cheng-Zhong Li
E-mail: leo_lee666@126.com)
The Department of Infectious Diseases
Changhai Hospital, Second Military Medical University
Shanghai, China

References

1. Stegelmeier BL, Edgar JA, Colegate SM, Gardner DR, Schoch TK, Coulombe RA, Molyneux RJ. Pyrrolizidine alkaloid plants, metabolism and toxicity. J Nat Toxins 1999; 8:95–116.
2. Coppell JA, Brown SA, Perry DJ. Veno-occlusive disease: cytokines, genetics, and haemostasis. Blood Rev 2003; 17:63–70.
3. Deleve LD. Dacarbazine toxicity in murine liver cells: a model of hepatic endothelial injury and glutathione defense. J Pharmacol Exp Ther 1994; 268:1261–1270.
4. Eisenberg S. Hepatic sinusoidal obstruction syndrome in patients undergoing hematopoietic stem cell transplant. Oncol Nurs Forum 2008; 35:385–397.
5. VT Ho, C Revta, Richardson PG. Hepatic veno-occlusive disease after hematopoietic stem cell transplantation: update on defibrotide and other current investigational therapies. Bone Marrow Transpl 2008; 41:229–237.
6. Dai N, Yu YC, Ren TH, Wu JG, Jiang Y, Shen LG, Zhang J. Gynura root induces hepatic veno-occlusive disease: a case report and review of the literature. World J Gastroenterol 2007; 13:1628–1631.

7. Dai HF, Gao Y, Yang M, Yu CH, Gu ZY, Chen WX. Hepatic veno-occlusive disease induced by *Gymura segetum*: report of two cases. Hepatobiliary Pancreat Dis Int 2006; 5:406–408.
8. Coppell JA, Richardson PG, Soiffer R, Martin PL, Kernan NA, Chen A, et al. Hepatic veno-occlusive disease following stem cell transplantation: incidence, clinical course, and outcome. Biol Blood Marrow Transplant 2010; 16:157–168.
9. Zuckerman M, V Steenkamp, Stewart MJ. Hepatic veno-occlusive disease as a result of a traditional remedy: confirmation of toxic pyrrolizidine alkaloids as the cause, using an in vitro technique. J Clin Pathol 2002; 55:676–679.
10. World Health Organization 1988. Pyrrolizidine alkaloids, environmental health criteria no 80, Geneva, Switzerland: World Health Organization. http://www.inchem.org/documents/ehc/ehc/ehc080. htm. Accessed 16 October 2010.