

Postcards from Beijing: Annual Meeting Abstracts

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The following are highlights from the scientific presentations of the 8th Annual Congress of the Asia-Pacific Association of Medical Toxicology, which was held in Beijing, China, October 2009. Clinicians and researchers from over a dozen countries attended this meeting, where more than 100 abstracts were showcased as either oral platform or poster presentations.

Although it is challenging to distill a whole meeting into a collection of abstracts, we feel that these selections share qualities common to all innovative research in our field—they each compel us to think differently about how best to care for the poisoned patient.

Collectively, these brief reports also provide a window into an exciting current development: the emergence of medical toxicology as a vital subspecialty in many countries where the burden of poisoning is tremendous. We hope that these abstracts will encourage *Journal of Medical Toxicology* readers to contribute to future international toxicology meetings and research collaborations worldwide.

For those who are interested, the next congress of the Asia-Pacific Association of Medical Toxicology will be held in Hanoi, Vietnam, on November 17 to 19, 2010. (see <http://www.apamt2010.vn/> for details). All presentation from recent APAMT meetings, and other useful information, can also be found at <http://www.asiattox.org>.

1. SYMPATHETIC SKIN RESPONSE (SSR) AND HEART RATE VARIABILITY IN PATIENTS WITH ACUTE ORGANOPHOSPHORUS (OP) POISONING

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Introduction: Some well-defined neurological syndromes are seen following acute organophosphorus (OP) poisoning. However, it is not clear whether there is medium to long-term autonomic nervous system dysfunction. Therefore, we aimed to examine the autonomic nervous system function in patients with acute OP poisoning.

Method: A case-control follow-up study was conducted. Sympathetic skin response (SSR) latency and amplitude of dominant hand, R-R (heart rate) interval variation during standing, deep breathing and Valsalva maneuver were measured in 21 patients with acute OP poisoning around the time of discharge (participants were otherwise well) and

1–2 months later. Assessments were performed a mean of 8 ± 8 days (first assessment) and 46 ± 9 days (second assessment) from exposure. First assessment was done mean 3 ± 2 days following cessation of atropine therapy. Twenty-one controls matched for age and gender were also examined. ANOVA and Post Hoc comparison were used for the analysis.

Results: The mean ages of cases (and controls) was 31 ± 13 years and there were 16 males in each group. The mean HbA_{1c} of cases and controls were $5.2 \pm 0.32\%$ and $5.4 \pm 0.51\%$, respectively. Atropine was commenced on six patients at peripheral hospital and transferred. All others had cholinergic features before the commencement of atropine therapy. Three patients were admitted to the intensive care unit (ICU) during the hospital stay and two were ventilated. All patients were treated with atropine. Nineteen patients received pralidoxime. The mean latency of SSR in controls, first, and second assessments of cases was $1,527 \pm 125$ ms, $1,634 \pm 123$ ms, and $1,532 \pm 123$ ms, respectively ($F=4.08$ ($p<0.05$)). Mean amplitude of SSR in controls, first, and second assessments of cases were 1.48 ± 1.0 mV, 0.33 ± 0.30 mV, and 1.05 ± 0.81 mV, respectively ($F=12.25$, ($p<0.01$)). Post Hoc comparison showed statistically significant differences in amplitude between the controls and the first assessment ($p<0.01$), and between the first and the second assessments ($p=0.01$). The first assessment latency was also significantly different from the controls ($p<0.05$). Heart rate variability analysis did not show any statistical significant difference between cases and controls.

Conclusion: Statistically significant amplitude reduction and prolongation of latency was observed in sympathetic skin responses at the time of discharge (mean 8 days following acute exposure to OP) which was not present 1 to 2 months later.

2. ACUTE FORMIC ACID POISONING IN SOUTH INDIA

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Introduction: Complications of ingestion of formic acid, the diluted form of which is used in coagulation of rubber latex, are not described in literature. Kerala, a state in south-western India, is well known for its rubber plantations. Easy accessibility to formic acid makes it susceptible to be used for deliberate self-harm in this region. This retrospective study was conducted to study the patterns of presentation and identify the predictors of morbidity and mortality of acute formic acid poisoning.

Methods: Data regarding patients admitted to the medical wards from January 2007 to December 2008 (2 years) with formic acid ingestion were retrieved and analyzed for symptoms at presentation, clinical parameters, and complications.

Results: Of the 302 patients (181 males), with a mean age of 42.78 years (13–85 years), accidental ingestion was reported in 23 patients (7.6%). The mean time taken for presentation to our center after consumption was 2.5 h. Formic acid was mixed in alcohol for consumption by 24.2% patients. Common symptoms at presentation were vomiting (78.1%), respiratory distress (44%), hematemesis (42.1%), and hematuria (30.1%). Complications of the poisoning were oral cavity burns (87.7%), metabolic acidosis (70.2%), septicemia (51.3%), dysphagia (51%), esophageal stricture (ES; 32.5%), gastro-intestinal perforation (GIP; 12.9%), aspiration pneumonia (47.4%), ARDS (33.8%), acute renal failure (38.7%), chemical pneumonitis (25.5%), and shock (24.2%). Rare complications were tracheo-esophageal fistula (four), pneumomediastinum (two), and chemical injury to the cornea (one). Of the 33 patients who underwent hemodialysis, nine developed deep vein thrombosis. Logistic regression was employed to predict morbidity (ES). Metabolic acidosis with pH <7.3 (OR 27.78, 95% CI 3.5–223.2), hematemesis (OR 5.5, 95% CI 2.7–11.1), and age >40 years (OR 0.976, 95% CI 0.95–0.99) were independent predictors of morbidity. Hematemesis ($p=0.000$) and melena ($p=0.000$) had significant associations with ES. Hematuria ($p<0.001$), respiratory distress ($p<0.001$), hematemesis ($p<0.001$), and GIP ($p=0.000$) at presentation were significantly associated with mortality.

Conclusion: Easy availability of formic acid should be curtailed by enforcing statutory limitations in its distribution. Metabolic acidosis, if taken care of by administration of sodium bicarbonate intravenously at the local medical centers, before referring the patient to a tertiary setup, may reduce mortality and morbidity in acute formic acid poisoning. Patients with hematemesis or melena, if they survive, should be followed-up with serial esophageogastroduodenal endoscopy for diagnosis and early treatment of strictures.

3. FRUCTOSE-1, 6-DIPHOSPHATE (FDP) AS A NOVEL ANTIDOTE IN YELLOW OLEAN- DER-INDUCED CARDIAC TOXICITY; PHASE II AND PHASE III CLINICAL TRIALS

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Introduction: Fructose-1, 6-diphosphate (FDP) was shown to be effective in oleander-induced cardiac toxicity in dogs. It is widely used for other indications and regarded as safe. We wished to explore its potential as an antidote in humans. The Phase II study was to choose the optimal dose, the phase III study to examine for effectiveness.

Methods: Phase II: We conducted a double-blind phase II placebo controlled dose ranging study of four doses levels of FDP in two rural hospitals. Patients received one of the four doses of FDP (30, 60, 125, 250 mg/kg) or placebo (normal saline). At each dose tested, six subjects received FDP and two placebo. **Phase III:** This study is a randomised double blind clinical trial in 240 patients of FDP (250mg/kg loading dose of FDP over 20 minutes followed by 6mg/kg/hr for 24 hours) vs. placebo in acute yellow oleander poisonings. All patients admitted to Kurunegala Teaching Hospital are initially resuscitated following the national guidelines. Consenting patients with AV block are randomised to receive FDP or placebo. The primary outcome is the sustained reversion to sinus rhythm with a heart rate greater than 50/min within 2 hours of the FDP/placebo bolus. Secondary outcomes include death, reversal of hyperkalaemia on the 6, 12, 18 and 24 hour samples and maintenance of sinus rhythm on the holter monitor. Analysis will be on intention-to-treat.

Results: Phase II: The FDP was well tolerated and there were no adverse reactions observed at any dose level. Our primary outcome measure: the reversion of atrio-ventricular block to sinus rhythm within 2 hours proved impractical as most (28/32) patients were transferred for cardiac pacing in a tertiary hospital within this time and there was frequent electrical interference with Holter readings. Favorable dose-related falls were seen in the serum calcium and potassium within 30 minutes of the infusion ($p=0.09$ & $p=0.03$, ANOVA). These supported the following in the Phase III study design: use of the highest bolus dose plus an infusion, conduct study only in tertiary hospital with pacing facilities, use normal ECG machine as well as Holter monitors, use serum potassium as secondary outcome measure. **Phase III:** This study has randomised 41 patients since February 2009 out of 319 oleander self-poisoning admissions. There have been 5 deaths and no adverse reactions to FDP. The study remains blinded. An interim analysis will be conducted after 120 patients.

Conclusions: Our findings from the phase II study suggested FDP is well tolerated and could have favorable effects on electrolytes. This study greatly helped to design a Phase III study well placed to determine the effectiveness of FDP in oleander induced cardiac toxicity, which is now progressing well and should be completed in 2011.

4. MUSHROOM POISONING FOLLOWING CONSUMPTION OF *INOCYBE* SPECIES

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Introduction: Mushroom poisoning is quite frequent in Nepal during rainy season. Approximately 40 species of poisonous mushroom are found in Nepal. There are currently eight recognized classes of mushroom poisonings, seven of which are caused by specific known toxins. The mushroom of *Inocybe* species (*Inocybe patouillardii*) is the only muscarine-containing mushroom found in Nepal. The clinical course of patients with typical muscarinic presentations after consumption of muscarine-containing mushrooms is reported.

Methods: A retrospective analysis was done for all calls to a poison center related to muscarinic symptoms following mushroom ingestion during the period of July 1998 to June 2008. A total of 77 consecutive cases were reported to Nepal Drug and Poison Information Center.

Results: Fifty-eight percentage of cases involved female ($n=45$) and remaining were male (42%, $n=32$). Ages ranged from 4 to 67 years, mean 24.70 (± 17.96). Combinations of nausea, vomiting, diarrhea, abdominal pain, urination, hypersalivation, bradycardia, hypotension, lacrimation, blurred vision, and miosis were initial presenting symptoms. Time to onset of toxicity ranged from 30 min to 2 h after consumption of mushroom. Treatment was symptomatic and supportive including intravenous fluids and atropine intravenously; maximum of 1.8 mg of atropine was needed for reversal of muscarinic symptoms. In all cases, full recovery occurred within 10 h post exposure.

Conclusion: Supportive and symptomatic treatment along with atropine for patients with ingestion of muscarine-containing mushrooms resulted in a favorable outcome.

5. QRS AND QTc ELECTROCARDIOGRAM (ECG) DURATIONS IN PATIENTS PRESENTING WITH ACUTE COCAINE AND COCAETHYLENE TOXICITY

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Introduction: QRS and QTc prolongation, and associated cardiac arrhythmias, following cocaine use are due to cocaine-related cardiac ion channel dysfunction. The simultaneous use

of cocaine and ethanol leads to an increased production of the cocaethylene metabolite, which has greater binding to cardiac ion channels and potentially therefore greater risk of cardiac arrhythmias. The effects of simultaneous cocaine–ethanol use on the QRS and QTc duration compared to those seen with lone cocaine use have not been reported.

Methods: A 24-month retrospective review of patients with acute toxicity related to self-reported lone cocaine or simultaneous cocaine–ethanol use was undertaken. Data on the sex, presenting symptoms/signs and physiological parameters were extracted on these presentations. ECGs were reviewed for all presentations, where available, and QRS duration and QTc calculated using Bazett's formula were extracted. Comparison of the QRS and QTc durations was undertaken between the two groups.

Results: There were 48 and 31 presentations with acute toxicity related to self-reported simultaneous cocaine–ethanol use and self-reported lone cocaine use, respectively. There was no significant difference in the mean (SD) age of those with simultaneous cocaine–ethanol use (29.8 ± 10.2 years) compared to those with lone cocaine use (29.3 ± 7.7 years; $p=0.80$). There were no significant differences between the mean (SD) heart rate ($p=0.90$), systolic blood pressure ($p=0.81$), and temperature ($p=0.61$) in the simultaneous cocaine–ethanol and lone cocaine use groups. The mean (SD) QRS and QTc durations were 87.3 ± 10.8 ms (range 60–108) and 397.4 ± 32.0 ms (range 323–484) for the simultaneous cocaine–ethanol use and 86.9 ± 12.5 ms (range 67–126) and 396.2 ± 34.6 ms (317–488) for lone cocaine use groups ($p=0.87$ and $p=0.88$, respectively). There were no QTc or QRS related cardiac arrhythmias in either group.

Conclusions: In this study, we have not detected a significant difference in the QRS and QTc durations between those self-reported simultaneous cocaine–ethanol use compared to those with lone cocaine use. Further studies are needed correlate the concentrations of cocaine and its metabolites, including cocaethylene, to confirm the findings seen in vitro and animal models of cardiac ion channel dysfunction.

6. PHENYLPROPANOLAMINE POISONING DUE TO THE USE OF COLD PREPARATIONS IN VIETNAM

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Introduction. Phenylpropanolamine (PPA) which is often combined with acetaminophen in cold preparations has been known to cause many adverse effects including cardiovascular involvement and is an independent risk factor for hemorrhagic stroke in women. This study described prospectively the features of its poisoning.

Method. This is a prospective and descriptive study performed in patients who get sick after the use of cold preparation containing phenylpropanolamine and is treated at our poison control center from September 2002 to September 2005. The poisoning or adverse effects are considered if the drugs were used very recently (within 6 h after the last dose) in a previously healthy person. The clinical and laboratory parameters were evaluated and monitored if exist. PPA-poisoned patients were treated symptomatically and discharged when all the signs and symptoms resolve.

Results. Forty-six patients including 21 males (45.7%) and 25 females (54.3%) were enrolled. The age of the patients was 32.89 ± 10.13 (range, 17–55 years). Rhumenol® (Tenamyd, Canada, 30 mg phenylpropanolamin per tablet) and Decolgen Forte® (United Pharma, 25 mg phenylpropanolamin per tablet) were two most common proprietary products leading to PPA poisoning, 95.65%. The reason for taking medicine was self-treatment of cold: 43/46 cases (93.5%), 37 patients (80.4%) presented symptoms of PPA poisoning at the first dose of PPA. Thirty-three cases (71.7%) got poisoning at doses of PPA equal or less than 60 mg. The symptoms of PPA poisoning were: headache (93.5%), nausea (50%), dizziness (37%), and elevated systolic blood pressure (higher than 140 mmHg; 95.7%); bradycardia (39.1%); hypokalemia was presented in 11 patients (23.3%). Adalat (oral liquid nifedipin) was given with doses of 3–10 mg. All the patients recovered from hypertension after 7.6 ± 5.03 h, 43 cases (93.5%) were discharged on the admitted day. No complications of hypertension or death were observed.

Conclusions. The manifestations of PPA poisoning include acute hypertension, which happens even if patients take the recommended dose of PPA for cold management. Poisoned patients recover quickly after PPA discontinuation and the use of a simple and rapid acting antihypertensive agent. The result from this study contributed to the decision by the ministry of health of Vietnam to remove all the pharmaceutical agents containing phenylpropanolamine from the domestic market since 2003.

7. PUFFERFISH POISONING—A LARGE OUTBREAK IN BANGLADESH

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Introduction: The study was carried out in the Medicine and Pediatrics department of Rajshahi Medical College Hospital, and Natore Sadar Hospital, both are located in the northern territory of Bangladesh.

Methods: On 8th June 2008, 83 patients (male 50, female 33) of Singra Upazila Natore were admitted in Rajshahi Medical College Hospital and Natore Sadar Hospital with the history of consumption of Puffer fish. A presumptive diagnosis of Puffer fish poisoning was made on the basis of classical clinical presentations followed by Puffer fish ingestion. Blood and urine sample were taken from 38 patients and sent for toxicological analysis to Frankfurt, Germany.

Results: Important symptoms observed were peri-oral paresthesia (71), tingling over entire body (50), nausea and vomiting (43), dizziness (35), headache (20), and abdominal pain (13). Muscular paralysis of the limbs was noted in 13 patients, of which seven patients developed respiratory involvement. All the patients who developed respiratory involvement died. Out of 83 patients, 76 patients were improved with conservative management and seven patients died. Out of 38 blood samples sent for toxicological analysis, 27 patients had detectable levels of Tetrodotoxin (TTX) in their blood, and in 11 patients blood TTX level was not detectable (<1.6 ng/ml). Blood TTX level seems to have a strong correlation with development of neuromuscular paralysis. Average TTX concentration in patients who developed neuromuscular paralysis was 8.1 ng/ml.

Conclusion: Early diagnosis and supportive management could ensure a safe and favorable outcome. Although Puffer fish poisoning is uncommonly encountered in our daily practice, physicians should be familiar with the clinical presentations and management and get prepared to handle such potentially life-threatening intoxications.

8. LOW MOLECULAR WEIGHT HEPARIN OVERDOSE

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Introduction

Low molecular weight heparin (LMWH) has been used for the treatment and prevention of several disorders including deep vein thrombosis, pulmonary embolism, unstable angina, and myocardial infarction. Its anticoagulant effect creates the potential for bleeding. There are several studies showing the risk of major bleeding from therapeutic anticoagulant use but there have been no reports in the literature on acute overdose in adults to date.

As the California Poison Control Centre (PCC) has been consulted on several cases of LMWH overdose, this series

aimed to provide data that may help poison center practice. We also believe this to be the first case series of patients reported with an acute overdose on LMWH.

Method

A retrospective chart review of PCC database: Visual Dot Lab between 1997 and 2007 was obtained. Inclusion criteria included all patients with a reported overdose on LMWH. The route of exposure is subcutaneous. Cases were excluded if therapeutic doses of LMWH were administered.

Results

There were 21 patients (mean 42.4 years).

The reasons for overdose include medical miscalculation (three cases, all infants), intentional misuse (two patients), accidental overdose (seven cases), suicidal attempt (seven cases), and unknown in two patients.

Seven cases were documented to have overdosed more than two times the therapeutic dose. The overdose ranged from 0.1 to 80 times the therapeutic range. No patients were documented to have bleeding or thrombocytopenia. Six patients were documented to have no bleed and were well after at least 36 h.

Reassurance was given to patients with less than 0.14 times the therapeutic dose. Two patients in the series received protamine because they received more than 2.5 times the therapeutic dose of LMWH.

Conclusion

Given the rarity of overdose, there is no clear consensus on its management. Most patients had no complications and were not treated with protamine. This series suggests that a large dosage of LMWH is unlikely to result in any life-threatening complications.

9. ROTUNDIN POISONING IN VIETNAM

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Introduction: Rotundin (L-tetrahydropalmatin) was extracted from the plant *Stephania rotunda* which has been known for the sedative and analgesic effects and used widely in Vietnam. However, this agent has been increasingly used without prescription and the incidence of poisoning ensued as a result.

Methods: This is a prospective descriptive study. All the patients overdosed with rotundin (by history and sample of medications from the patients), with the urine or gastric fluid positive with rotundin by thin layer chromatography were admitted to our poison control center from December 2003 to January 2005.

Results: 122 patients (27 males, 22.1% and 95 females, 77.9%) were included. Age of the patient, 23.6±6.3 (12–

52 years), reasons for poisoning was suicide in 120 patient (98.4%). The mean dose was $1,258.7 \pm 1,082.81$ mg (range, 300–6,000 mg). Severity of poisoning: mild, 69.7%; moderate, 25.4%; severe, 2.5% and no death occurred. The most common symptoms were CNS depressant (32.8%), nausea (22.1%), vomiting (20.5%), sinus bradycardia (3.3%), and hypotension (0.8%). ECG abnormalities accounted for 74.6% of the patients including prolonged QT (27%), sinus extrasystole (3.3%), and first degree AV block (1.6%). Sinus bradycardia, sinus tachycardia, elevated ST segment, and T wave inversion were also observed. Very mild elevation in AST and ALT were seen in 7.4% of the patients. The dose of rotundin was 77.8 ± 474.09 mg in patients with normal ECG and $1,432.0 \pm 1,185.8$ mg in patients with ECG abnormalities ($p=0.004$). The dose of rotundin was $1,172.0 \pm 1,026.67$ mg in totally conscious patients and was $1,346.9 \pm 1,139.23$ mg in patients with CNS depressant ($p=0.385$).

Conclusion: Rotundin only causes mild CNS inhibition (if any). However, in our patients, it also causes cardiac abnormalities which associate with higher doses.

10. DIAGNOSTIC IMAGING OF OPIUM BODY PACKING

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Objective: Opium body packing is a common cause of admission to our Medical Toxicology ward. Since the body packers are drug smugglers, they are mostly brought to the hospital by police. Those who are alert usually deny body packing. Ultrasonography, plain X-ray, and CT scan are recommended for the diagnosis of body packing and stuffing. It was aimed to compare the diagnostic values of the three techniques in opium body packing.

Methods: A questionnaire was designed to record all clinical and paraclinical findings of all body packers admitted to the ward between 10 October 2000 and 11 October 2008. Ultrasonography, plane X-ray, and CT scan were performed for all body packers on admission and at certain intervals as clinically indicated. Magnesium sulfate was used as a cathartic in all admitted body packers; naloxone was administered in symptomatic patients. The asymptomatic cases were under close observation both medically and forensically. The packets recovered in the feces were counted, weighted, and collected by the police. The comatose patients with many packets who did not respond to the medical treatment or revealed bowel obstruction consulted surgically. The surgically removed packets were also counted, weighted, and collected by the police. The results of the three techniques were compared with

the clinical findings and the number of recovered packets. Statistical analysis (Chi square test) was made using SPSS.

Results: Out of 3,281 poisoned patients admitted to the ward over the years, 490 patients (15%) had narcotic poisoning, of which 50 patients (5%) were opium body packers. There were two female body packers (a 16-year-old girl and a 35-year-old woman), one of whom had severe opium poisoning; she underwent emergent surgery and died a day later in ICU. Out of 48 male patients, two (30 and 69 years old) also had surgery and died. The other 47 patients aged 17 to 58 (mean 31) years were treated medically and all survived despite the severe intoxication in 18 of them. The body packers were either illiterate (28%), primary educated (32%), or secondary educated (40%). More than 44% of them were drug addicts. The number of packets varied between 1 and 48 (mean of 21) with weights of 6 to 102 g (mean of 46). Ultrasonography did not show any clear countable packets, whereas plain abdominal X-ray revealed the packets in 24 patients (48%) and abdominal CT scan were positive in 48 (96%) patients ($p<0.001$).

Conclusion: (1) Ultrasonography is of no value in diagnosis of opium body packing. (2) Plane abdominal X-ray is simple but not efficient. (3) CT scan is the best diagnostic technique in opium body packing.

11. CENTIPEDE ENVENOMATION IN THAILAND

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Introduction: Centipede envenomation occurs commonly in tropical countries. At the present time, however, there are no epidemiologic or clinical studies of centipede envenomations in Thailand. This study is the first of its kind studying epidemiology, clinical manifestations, and various treatments of envenomations by centipedes in patients registered at the Department of Emergency Medicine, Bangkok Metropolitan Administration Medical College and Vajira Hospital, Bangkok, Thailand.

Method: We retrospectively analyzed 104 cases of definite envenomations by centipedes among patients that presented to the hospital, between 1 January, 2004 and 30 June, 2009. Demographic data, data on local and systemic effects, and treatments after centipede envenomations were collected.

Results: There were 104 cases included in this study. Fifty-two percent were female. Mean age was 27.8 ± 17.8 years old (ranging from 1 month to 76 years). The time from envenomation to presentation at the Emergency Department ranged between 15 min and 48 h (median=40 min). Most

of the envenomations (85.9%) occurred at night time between 6 pm and 6 am. The incidence of envenomations was highest in the summer months; April and May, and winter months; October through December. Envenomation sites were recorded in 96% of patients, and 91 out of 100 cases were stung only once. Feet (32%) and hands (25%) were the parts of the body most often envenomated. Local effects were common. Ninety-six percent of patients had localized pain and 78% had swelling at the site of envenomation. Systemic effects consisted of nausea (7.7%), vomiting (5.8%), rash (2.9%), fever (1.9%), systemic swelling (1.9%), abdominal pain (1%), palpitations (1%), and wheezing (1%). Anaphylaxis was diagnosed in three patients with two or more systemic effects, but neither wheezing lungs nor shock was found. For pain control, 98.1% received analgesic drugs, while 33.7% were injected with local anesthesia. Antibiotics, antihistamines, and steroids were prescribed in 73.1%, 24%, and 9.6%, respectively. Fortunately, no deaths occurred in this study.

Conclusion: Most of the centipede envenomations we see in Thailand occur in two clusters each year. The first cluster is in April and May and the second is in October through December. Patients were the most vulnerable to centipede envenomations during the night time hours. Nearly all patients had local effects, in contrast to systemic effects which rarely presented. Most of the patients had favorable outcome.

12. A CASE OF OPHTHALMIC INJURY BY SPITTING CHINESE COBRA TREATED WITH LOCAL ANTIVENOM

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Introduction: Ophthalmic injury by the venom of Chinese Cobra (*Naja atra*) is an uncommon but well-recognized mode of injury. The clinical value of local antivenom for this type of injury is uncertain although animal studies showed possible benefits. We report a case of ocular injury by spitting Chinese Cobra with rapid relief of symptoms after local antivenom irrigation.

Case Report: A 50-year-old man had left eye injury by a spitting Chinese Cobra at a 3 ft distance and attended the Accident and Emergency Department 30 min later. He had persistent symptoms with left eye pain and blurred vision after local irrigation with copious amount of normal saline solution. Examination showed bilateral eye congestion, visual acuity was 6/21 on the left eye compared with 6/9 on right side. There was no corneal abrasion. Naja Antivenin® (Shanghai Institute of Biological Products, Ministry of Health, China) diluted to 500-mL normal saline irrigation was performed for persistent

symptoms. The patient had relief of his symptoms almost immediately after irrigation with the diluted antivenom. Topical antibiotic solutions containing polymyxin, neomycin, and gramicidin (tetracycline eyedrop was not available in our center) was also given and his left eye congestion and visual acuity improved overnight. He remained asymptomatic on assessment by ophthalmologist 3 days after the injury.

Discussion: It is generally believed that the cardiotoxin component of the venom of the Cobra family causes ocular injury. Local toxicities reported for other species of Cobra ranges from pain, redness, corneal injury to blindness. Animal model and case reports had showed possible beneficial effect from the use of tetracycline eyedrops and local administration of antivenom. Although the role of local administration of antivenom is controversial for other species of Cobra, the antivenom used was not specific in other parts of the world. On the contrary, the Naja Antivenin® is specific to Chinese Cobra and it is possible that treatment with the antivenom is more specific. In fact, local antivenom eyedrop has been used for ocular injury by Chinese Cobra in some centers in China with good outcome.

Conclusion: Apart from irrigation by water or normal saline solution, and application of local antibiotics, the administration of local antivenom, either as eyedrops or in diluted irrigation can be considered in cases of ocular injury by spitting Chinese Cobra with persistent symptoms or severe injury.



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