Environ Health Prev Med (2016) 21:231–236
DOI 10.1007/s12199-016-0516-4

REGULAR ARTICLE

Acute interstitial nephritis, toxic hepatitis and toxic myocarditis following multiple Asian giant hornet stings in Shaanxi Province, China

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Received: 2 January 2016 / Accepted: 3 February 2016 / Published online: 24 February 2016

Abstract
Objectives During July to October 2013, the Asian giant hornet has killed 42 and injured 1,675 people in the southern part of Shaanxi Province, China. This study investigated this unusual and frequent public health event.
Methods During the 3 months, 103 patients with severe Asian hornet stings were hospitalized in our hospital. Clinical data were collected using a standardized data collection form which included sex, age, length of hospital stay and medical recorder.
Results After physical examination and laboratory investigation, 25.2, 46.6 and 44.7 % of the patients were found with varying degrees of acute interstitial nephritis, acute toxic hepatitis and acute toxic myocarditis, respectively. After timely and appropriate treatment including removal of the stings and the use of intravenous methylprednisolone and antihistamines, the kidney function, liver function and heart function of 99 patients recovered within 1 month, while four patients died.
Conclusions This study provided a good opportunity for recognizing the effect of Asian giant hornet stings and evaluating this serious public health event.

Keywords Asian giant hornet stings · Acute interstitial nephritis · Acute toxic hepatitis · Acute toxic myocarditis · Heart function protection

Introduction

Wasps are found almost in all parts of the world. They play an important role in the balance of natural ecosystems through pollination, natural pest control and biodiversity [1, 2]. So far, over 30,000 species have been identified [3]. The most common types of wasps are hornets, yellow jackets and paper wasps. The Asian giant hornet (*Vespa mandarinia*) belongs to hornets and is the world's largest eusocial wasps (Fig. 1). They are mainly found throughout Eastern Asia, but have appeared in France, Spain, Portugal, Belgium, Italy, and Germany [4]. Usually, the Asian giant hornet stings cause local reactions and anaphylaxis. In recent years, some cases from Japan and Thailand have caused severe clinical complications such as acute interstitial nephritis (AIN), acute toxic hepatitis (ATH), acute toxic myocarditis (ATM) and multiple organ dysfunction syndrome (MODS) [5–7].

From July to October 2013, the Asian giant hornets have killed 42 and injured 1,675 people with their powerful venomous stings in Hanzhong, Ankang and Shangluo. These three cities have humid subtropical climate and are located in Qin Mountains which provide a natural boundary between the north and south of China. Although wasp stings have received much attention in recent years, it is rare to see so many cases of stings during a short time

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Fig. 1 The Asian giant hornet (*Vespa mandarinia*). The Asian giant hornet is the world's largest hornet, native to temperate and tropical Eastern Asia. This Asian giant hornet was involved in this sting event. *Scale bar* 10 mm

frame. Therefore, it is significant to know the clinical features, treatment strategies and outcomes of patients in this event.

Materials and methods

Patients

Between July to October 2013, a total of 103 patients with Asian hornet stings were admitted to our hospital. The diagnosis of wasp stings was based on the patient's clinical history, physical examination and laboratory test results. Detailed clinical history was recorded and a complete physical examination was performed for each patient. The following information has been recorded: the presence of hematuria or proteinuria, number of stings, time between sting and hospitalization and other clinical manifestations including vomiting, numbness, consciousness and complications. We also recorded the duration of hospital stay, in-hospital case fatality rate and cause of death. Additional informed consent was obtained from all individual participants for whom identifying information has been included in this article. These studies were carried out in accordance with the approved guidelines. All the experimental protocols were approved by the Ethics Committee of Medical College, Xi'an Jiao Tong University, and were in accordance with the Helsinki Declaration.

Clinical data collection

The related laboratory examinations were obtained soon after the patients were admitted. Using these laboratory findings combined with clinical manifestations, the following diagnoses could be made—AIN: abnormally high levels of blood urea nitrogen (BUN) and serum creatinine (SCr), along with anuria (<100 ml/day) or oliguria (100–400 ml/day) and abnormal urine tests (proteinuria and hematuria); ATH: abnormal levels of total bilirubin (TBIL), indirect bilirubin (IBIL), total protein (TP), alanine transaminase (ALT) and aspartate aminotransferase (AST); ATM: elevated troponin I (TnI), myoglobin (Mb) and creatine kinase MB (CKMB). All patients were treated with intravenous methylprednisolone and antihistamines. Those with serious toxic reactions received additional epinephrine treatment. Continuous renal replacement therapy (CRRT), hemoperfusion (HP) and therapeutic plasma exchange (TPE) were given to patients with AIN, ATH and ATM. The rest of the treatment included intravenous fluids, prophylactic antibiotics, oxygen and symptomatic measures.

Statistical analysis

The one-way analysis of variance (ANOVA) was used for group comparisons. All analyses were implemented using SPSS 13.0 for Windows. A P value of <0.05 for 95 % confidence intervals was set.

Results

Clinical data

All patients were adults (>18 years old) with a mean age of 50.6 years (range 18.0–78.0 years). Most patients were stung in the head and/or upper limbs and many had multiple (up to 112) stings (Fig. 2). Female patients accounted for 53 of the cases (51.5 %). The mean length of time

Fig. 2 The patient after wasp sting. The area that has been stung became red, painful and swollen. The patient was stung 72 times in the head and upper limbs

between wasp stings and hospitalization was 10.4 h (range 1–144 h; Table 1). All patients showed local inflammatory reaction (redness, swelling, heat and pain). Dizziness, nausea and vomiting were the most common symptoms. As shown in Table 1, hematuria was detected in 38 cases (36.9 %). Thirty-five patients had proteinuria (34.0 %).

Table 1 Clinical characteristics of patients

Age (years)	50.6 (18.0–78.0)
Sex (M:F)	50:53
Time elapse from bite to hospital (hours)	10.4 (1–144)
Duration of hospital admission (days)	10.7 (0.1–288.0)
AIN (%)	26 (25.2)
ATH (%)	48 (46.6)
ATM (%)	46 (44.7)
Hematuria (%)	38 (36.9)
Proteinuria (%)	35 (34.0)
Death (%)	4 (3.9)

AIN acute interstitial nephritis, *ATH* acute toxic hepatitis, *ATM* acute toxic myocarditis

Altogether, 42 patients (40.8 %) suffered from two or more organ dysfunction. The length of stay in the hospital was 10.7 days (0.1–288.0). Four patients died and the case fatality rate was 3.9 % (Table 1). Three patients died with ATM, ATH and AIN. One patient died with ATM and ATH. As shown in Table 2, the white blood cell (WBC), hemoglobin (HB), platelet (PLT), TBIL, IBIL, TP, albumin (ALB), ALT, AST, BUN, SCr, TnI, Mb and CKMB had been measured and averaged.

Incidence of acute interstitial nephritis

2 % of patients (26/103) exhibited varying degrees of AIN (Table 1). The average BUN and SCr levels were higher in AIN patients than non-AIN patients (11.1 vs. 8.7 mmol/L and 141.7 vs. 80.4 μmol/L), whereas the average TP level was lower in AIN patients than in non-AIN patients (57.7 vs. 66.0 g/L) (Table 3). Four AIN patients had BUN levels >20 mmol/L and seven had SCr levels >200 μmol/L. All survivors had complete recovery of renal function within 1 month.

Table 2 Laboratory data of patients

Laboratory test (normal)	No. of cases with abnormal data (%)	Mean of abnormal data (±SD)	Range of abnormal data
Hematology			
WBC (4–10 × 10^9/L)	91 (88.3 %)	20.4 (±10.5)	10.4–78.25
HB men <120 g/L (120–160 g/L)	14 (28 %)	107.1 (±4.3)	102.9–115.6
HB men >160 g/L (120–160 g/L)	12 (24 %)	168.6 (±4.9)	164.8–175.2
HB women <110 g/L (110–150 g/L)	14 (26.4 %)	99.3 (±11.0)	79.0–109.4
HB women >150 g/L (110–150 g/L)	8 (15.1 %)	160.6 (±6.6)	155.9–165.2
PLT (150–400 × 10^9/L)	46 (44.7 %)	106.5 (±30.7)	14–145
Blood chemistry			
TBIL (3.4–20 μmol/L)	67 (65 %)	57.8 (±18.1)	20.5–165.5
IBIL (3–15 μmol/L)	62 (60.2 %)	44. 2 (±10.8)	15.0–158.1
TP < 60 (60–80 g/L)	30 (29.1 %)	54.6 (±4.4)	40.0–59.7
TP > 80 (60–80 g/L)	9 (8.7 %)	82.7 (±1.9)	80.9–84.9
ALB (35–55 g/L)	31 (30.1 %)	31.4 (±3.2)	23.6–34.9
ALT (0.00–42.00 U/L)	34 (33.0 %)	264.8 (±45.3)	42.2–576.1
AST (0–40 U/L)	61 (59.2 %)	620. 0 (±13.7)	42.2–2034.0
BUN (2.1–7.1 mmol/L)	40 (38.3 %)	10.5 (±3.4)	7.2–26.9
SCr (39–113 μmol/L)	20 (19.4 %)	176.8 (±10.4)	114.7–288.0
TnI (0.00–0.80 ng/ml)	8 (7.8 %)	1.5 (±0.3)	0.82–2.03
Mb (0–85 ng/mL)	64 (62.1 %)	2004.2 (±414.4)	89.9–4009.0
CKMB (7–25 U/L)	64 (62.1 %)	165.9 (±50.4)	26–310
Urine test			
Qualitative proteinuria	35 (34.0 %)		
Qualitative hematuria	38 (36.9 %)		

AIN acute interstitial nephritis, *ATH* acute toxic hepatitis, *ATM* acute toxic myocarditis, *WBC* white blood cell, *HB* hemoglobin, PLT platelet; *TBIL* total bilirubin, *IBIL* indirect bilirubin, *TP* total protein, *ALB* albumin, *ALT* alanine transaminase, *AST* aspartate aminotransferase, *BUN* blood urea nitrogen, *SCr* serum creatinine, *TnI* elevated troponin I, *Mb* myoglobin, *CKMB* creatine kinase MB

Table 3 Laboratory data of patients

	AIN patients	Non-AIN patients	*P* value
TP (g/L)	57.7 ± 7.1	66.0 ± 7.5[a]	0.021
BUN (mmol/L)	11.1 ± 5.2	8.7 ± 2.0[a]	0.019
SCR (umol/L)	141.7 ± 10.2	80.4 ± 10.7[a]	0.008
	ATH patients	Non-ATH patients	
TBIL (umol/L)	55.5 ± 12.1	20.4 ± 7.8[b]	0.011
IBIL (umol/L)	45.6 ± 11.1	17.8 ± 6.3[b]	0.002
TP (g/L)	60.8 ± 6.8	68.0 ± 5.7	0.096
ALB (g/L)	35.8 ± 5.4	40.7 ± 5.2[b]	0.047
ALT (U/L)	188.1 ± 40.5	30.3 ± 4.9[b]	<0.001
AST (U/L)	749.6 ± 135.6	80.3 ± 18.2[b]	<0.001
	ATM patients	Non-ATM patients	
TnI (ng/ml)	1.37 ± 0.1	0.03 ± 0.01[c]	<0.001
Mb (ng/ml)	215.9 ± 40.9	26.7 ± 19.3[c]	0.015
CKMB (U/L)	192.7 ± 11.2	25.0 ± 4.7[c]	<0.001

WBC white blood cell, *HB* hemoglobin, PLT platelet; *TBIL* total bilirubin, *IBIL* indirect bilirubin, *TP* total protein, *ALB* albumin, *ALT* alanine transaminase, *AST* aspartate aminotransferase, *BUN* blood urea nitrogen, *SCr* serum creatinine, *TnI* elevated troponin I, *Mb* myoglobin, *CKMB* creatine kinase MB

[a] Significant differences when compared with the AIN group

[b] Significant differences when compared with the ATH group

[c] Significant differences when compared with the ATM group

Incidence of acute toxic hepatitis

46.6 % of patients (48/103) exhibited varying degrees of ATH (Table 1). Viral markers for hepatitis B, hepatitis C and hepatitis A were negative in these patients. The average TBIL, IBIL, ALT and AST levels were much higher in ATH patients than in non-ATH patients (55.5 vs. 20.4 μmol/L, 45.6 vs. 17.8 μmol/L, 188.1 vs. 30.3 U/L, and 749.6 vs. 80.3 U/L, respectively). The average TP and ALB levels in AHT patients were slightly lower than in non-AHT patients (60.8 vs. 68.0 g/L and 35.8 vs. 40.7 g/L) (Table 3). Eight AHT patients' TBIL levels were over 100 μmol/L. Four AHT patients' IBIL levels were over 100 μmol/L. Thirteen AHT patients' ALT levels were over 100 U/L and four were over 500 U/L. Sixteen AHT patients' AST were over 100 U/L and eight were over 500 U/L. All survivors had complete recovery from AHT within one month.

Incidence of acute toxic myocarditis

44.7 % of patients (46/103) had ATM (Table 1). The average TnI, Mb and CKMB levels were higher in ATM patients than in non-ATM patients (1.37 vs. 0.03 ng/ml, 215.9 vs. 26.7 ng/ml and 192.7 vs. 25.0 U/L) (Table 3). Two AHM patients' TnI levels were over 2 ng/ml. Sixteen ATM patients' Mb were over 500 ng/ml. Five ATM patients' CKMB were over 300 U/L.

Table 4 Relationship between number of wasp stings and laboratory data

No. of envenomations	<10		>10		*P* value
	No.	%	No.	%	
No. of patients	24		79		
Deaths (%)	0	0	4	5.1	N/A
WBC (%)	17	70.8	74	93.7*	0.018
Elevated HB (%)	7	29.2	13	16.5	0.767
Decreased HB (%)	3	12.5	25	31.6*	0.047
Decreased PLT (%)	15	62.5	31	39.2	0.120
Elevated TBIL (%)	11	45.8	56	70.1*	0.0003
Elevated IBIL (%)	9	37.5	53	67.1*	0.0001
Decreased TP (%)	1	4.2	29	36.7*	0.036
Elevated TP (%)	4	16.7	5	6.3	0.050
Decreased ALB (%)	4	16.7	27	34.2*	0.001
Elevated ALT (%)	4	16.7	30	38.0*	0.049
Elevated AST (%)	8	33.3	53	67.1*	0.045
Elevated BUN (%)	2	8.3	38	48.1*	0.025
Elevated SCr (%)	0	0	20	25.3	N/A
Elevated Tnl (%)	0	0	8	10.1	N/A
Elevated Mb (%)	8	33.3	56	70.9*	0.049
Elevated CKMB (%)	9	37.5	55	69.6*	0.028

N/A not applicable, *WBC* white blood cell, *HB* hemoglobin, PLT platelet; *TBIL* total bilirubin, *IBIL* indirect bilirubin, *TP* total protein, *ALB* albumin, *ALT* alanine transaminase, *AST* aspartate aminotransferase, *BUN* blood urea nitrogen, *SCr* serum creatinine, *TnI* elevated troponin I, *Mb* myoglobin, *CKMB* creatine kinase MB

* Significant differences when compared with the patients group with <10 stings

Correlation of the number of stings with the severity of outcome

As shown in Table 4, 76.7 % of cases (79/103) suffered from more than 10 stings. Elevated WBC, TBIL, IBIL, ALT, AST, BUN, SCr, TnI, Mb and CKMB were observed in the >10 stings group. However, the HB, TP and ALB levels were decreased in the >10 stings group.

Discussion

Our study investigated the severe Asian hornet sting events which happened between July and October 2013 in the southern part of Shaanxi Province, China. As our hospital is one of the three regional central hospitals in the southern

part of Shaanxi Province, 103 sting patients with severe clinical symptoms were hospitalized. Complications in our patients included AIN, ATH and ATM. Fatal stings usually occur in the head or neck, and death typically results from MODS. In Australia, wasp sting mortality rates were 0.02 % per million population per year [8]. In Nepal, the case fatality rate could go up to 25 % after wasp stings. The case fatality rate of hospitalized patients was 3.9 % in our patients, which was similar to the report from another Chinese province (5.1 %) [9, 10]. The cause of patients' death was AIN, ATH and ATM, which were caused by toxic reaction to wasp venom [11–13]. Compared to the other wasp species, the Asian giant hornet has an especially neurotoxin, mandaratoxin. High doses of mandaratoxin have been demonstrated to induce violent convulsions, constriction of airways, muscle weakness and death, but the mechanism remains unclear [14].

AIN is the most common complications of wasp stings [15]. It was observed in 25.2 % of our patients. To confirm AIN, histopathology examination was performed in four patients [16]. The kidney function of AIN patients who were treated with CRRT, HP and TPE within 24 h after being stung returned to normal within 7 days. It suggested that timely CRRT, HP and TPE treatment is critical for kidney function recovery. In cases with over 48 h between wasp sting and hospitalization, kidney function was much more difficult to recover. ATH is another common complication of wasp stings. ATH was observed in 46.6 % of our patients. Some studies reported ATH due to centrilobular necrosis, portal triaditis and pericholangitis, as seen on histopathology [17–20]. For the liver to recover its function, Solu-Medrol, NaHCO3 administration and fluid infusion were given to the ATH patients until the serum ALT and AST levels decreased to the normal range. According to the reports by Tsai et al. [21], ATH was usually short and lasted several hours after wasp stings. However, most of our ATH patients experienced at least a week of treatment for their liver function to return to normal.

ATM was rarely reported after wasp stings. Also, there were very few systematic analyses on ATM patients of large sample sizes. In our patients, up to 44.7 % ATM was observed. In 1974, Brasher [22] first reported that the wasp sting patients had electrocardiographic changes. In 1976, two patients were first diagnosed with acute myocardial infarction after wasp stings [23]. To date, almost twenty cases of acute myocardial infarction have been reported. After reviewing the electrocardiogram, laboratory results and cardiac magnetic resonance imaging, these patients are more likely to be diagnosed with ATM. Some reports believe that acute kidney injury and accumulation of toxic metabolites would have contributed to myocardial dysfunction [24, 25]. The four dead patients all had ATM, suggesting that ATM was the main reason of death. Early treatment is vital in protecting the heart functions and reducing the case fatality rate. Since the severity of clinical features depended on toxic reactions, the severity of clinical manifestation is related to the number of stings. Xie et al. [10] believed that blood purification therapies could remove not only the free myohemoglobin and hemoglobin due to rhabdomyolysis and hemolysis, but also venom toxins and inflammatory factors [26, 27]. It has also been proven by our study that CRRT, HP and TPE could significantly improve the patients' condition.

Conclusions

Our study provided a comprehensive insight into the clinical characteristics of Asian giant hornet sting. Life-threatening reactions including AIN, ATH and ATM could develop after multiple stings. Timely and appropriate supportive managements such as CRRT, TPE and HP are necessary for the patients. Furthermore, we suggest that the family doctors in rural areas should bring the cases of Asian giant hornet stings to the hospital as early as possible. The doctors must not only consider and exclude an allergic reaction, but also monitor the injury to the kidneys, liver and heart.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Human and animal rights statement All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors.

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