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ABSTRACTS

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Table 1. Snake types and clinical effects in cases of snakebite collected by the Australian Snakebite Project.

Data collected	Number of cases	%
Snake type		
Brown snake (*Pseudonaja* spp.)	274	35%
Tiger snake (*Notechis* spp.)	88	11%
Red-bellied black snake (*Pseudechis porphyriacus*)	104	14%
Rough-scale snake (*Tropidechis carinatus*)	55	7%
Taipan (*Oxyuranus* spp.)	33	4%
Mulga snake (*Pseudechis australis*)	29	4%
Death adder (*Acanthophis* spp.)	24	3%
Clinical syndromes		
Coagulopathy	560	73%
Complete VICC	429	56%
Partial VICC	131	17%
Anticoagulant	86	11%
Major haemorrhage	10	1%
Neurotoxicity	84	11%
Mild	48	6%
Myotoxicity	73	9%
Thrombotic microangiopathy	58	8%
Renal toxicity	89	12%
Acute renal failure	42	6%
Abnormal creatinine	47	6%

VICC = venom-induced consumption coagulopathy.

to 1 vial, decreased repeat dosing from 65% to 24%, associated with a slight decrease in antivenom reactions, over 10 years.
Conclusion: A national multicentre collaboration systematically described clinical syndromes and antivenom effectiveness in snake envenoming. Laboratory support was critical, providing objective evidence based on venom concentrations. The collaborative nature allowed immediate dissemination of research results into clinical practice, rapidly influencing and improving treatment. Reduced antivenom use means decreased cost and less risk of anaphylaxis.

96. Novel ciguatera shellfish poisoning (CSP) cluster after consumption of *Tectus niloticus*, a gastropod, in Nuku-Hiva, French Polynesia

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Objective: Recently, a new pathway of ciguatera-like syndrome, also known as ciguatera shellfish poisoning (CSP), associated with the consumption of marine invertebrates (giant clams, sea-urchins) has been highlighted.[1] This is the first report of a cluster of CSP after consumption of troca (*Tectus niloticus*, a gastropod), although a few unofficial reports are already known among local populations of South Pacific.[2]
Case series: In June 2014, 9 sailor tourists (2 French, 2 Dutch, 5 Italian) were poisoned after the consumption of troca in Nuku-Hiva island (French Polynesia). Seven of them were evaluated at the local hospital for severe gastrointestinal and neurological manifestations. Two patients were evaluated in our centre 1 week after the poisoning. Case A (45-years-old) presented mainly with severe gastrointestinal manifestations (vomiting/diarrhoea), asthenia/myalgia, paresthesias/dysesthesias/thermoalgia and intractable hiccups. Esophagogastroduodenoscopy showed esophagitis. Specific investigations for neuropathological alterations and genetic predisposition for chronic disease were conducted. The patient was treated with mannitol. A 5-month follow-up documented the persistency of mild temperature-related dysesthesias of the upper extremities. Case B (72-years-old) presented mainly with slight gastrointestinal manifestations (vomiting/diarrhoea) associated with asthenia/myalgia during the 1st week. Neuropathological tests (after 1 month) were normal and the 5-month follow-up documented complete clinical resolution. Most of other victims are still symptomatic with gastrointestinal and/or peripheral neurological symptoms. Investigations based on specific clinical questionnaires were submitted to them. Preliminary toxicological analysis of troca specimens confirmed the presence of lipophilic ciguatoxin-like compounds. Moreover, unidentified hydrophilic toxins have also been detected and are currently being analyzed for identification.
Conclusion: Our investigations confirm the implication of *Tectus niloticus* in a CSP cluster. Additional investigations are ongoing in order to specify the toxic source (organisms and toxins involved), to characterize the clinical features of this CSP form, including the occurrence of potential chronic effects and to recommend effective treatments. Moreover, these data would be useful to health authorities to improve the risk management of seafood poisonings, especially in Pacific islands, where *T. niloticus* constitute a significant subsistence and economic resource.

References

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97. Viper envenomation in Italy: Clinical course, laboratory investigations and antivenom treatment in a case series (2002-2012) from Pavia Poison Centre

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Objective: Viper envenomation may be characterized by local and systemic symptoms with an estimated mortality up to 1%. Clinical and laboratory disorders and antivenom administration are often debated[1] Poisoning severity, laboratory alterations and antidote

administration in viper-envenomed patients referred to the Pavia Poison Centre (PPC) are described in order to evaluate predictable clinical and laboratory factors in viper envenomation management.
Methods: All viper bitten patients referred to PPC from 2002-2012 were retrospectively studied. Clinical manifestations and evolution were evaluated according to a Grading Severity Score (GSS)[2] and related to laboratory parameters and antidote treatment.
Results: During the 11-year study period, 482 viper bitten patients were evaluated (44 ± 23 years; male 65%). At hospital admission 43.2% had only fang-marks (GSS0), 39% local edema (GSS1), 15.8% regional edema and/or mild systemic manifestations (GSS2) and 2% severe local and/or systemic manifestations (GSS3). Among GSS0-admitted patients, 38/208 (18%) developed GSS ≥ 1, and 10/208 (5%) required antivenom because they progressed to GSS ≥ 2. Among GSS1-admitted patients, 73/188 (38.8%) developed GSS ≥ 2, and 59/188 (31.3%) needed antivenom. Most GSS2-3 (63-100%) admitted patients received antivenom. Among 482 patients, 170 (35%) had dry bites and 312 (65%) developed envenomation. Systemic symptoms were mainly gastrointestinal (118/312; 38%), hemodynamic (37/312; 11.8%), neurotoxic (36/312; 11.5%) and local thrombosis (24/312; 8%). Seven patients developed hemodynamic shock and three had splenic, myocardial or cerebral ischemia, respectively. No fatal cases occurred. Mean onset time of local manifestations was 11.8 hours and 27.5 hours for mild and extensive edema, respectively; gastrointestinal and hemodynamic disorders developed within 5-7 hours and neurotoxic effects within 10.7 ± 6.2 hours. Increase in leukocytes, D-dimer, INR and decreased thrombocytes and fibrinogen were statistically related with GSS ≥ 2. Antivenom was required in 44% of patients and administered with a mean time of 15.5 hours. Most patients (76%) improved after antivenom. In those (24%) where GSS ≥ 2 was present within a few hours edema worsened despite antivenom administration.
Conclusion: Viper bite is potentially serious and requires immediate hospital care. GSS0-patients at hospital admission may worsen and require antivenom within 12-24 hours. Leukocytosis and increased D-dimer occur with severe envenomation. Prompt antivenom administration is important and further administration may be evaluated in patients that develop severe envenomation.

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98. A novel strategy for identifying *Naja atra* species-specific venom antigen C3 for developing cobra snakebite confirmation test in Taiwan

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Objective: Taiwan, a subtropical island has more than 40 snake species, and 6 of them play a clinically significant role in snakebites. They are *Deinagkistrodon acutus, Viridovipera stejnegeri, Protobothrops mucrosquamatus, Daboia russelii siamensis, Bungarus multicinctus* and *Naja atra*. Due to lack of specific tests misdiagnosis of the culprit snake is common (nearly 10%). This is especially true when patients are bitten by *Viridovipera stejnegeri*, Protobothrops mucrosquamatus or *Naja atra*, because they have similar clinical presentations in the early phase. As a consequence administration of the wrong antivenin is frequently seen. We combined immunologic and mass spectrometry methods to develop a new strategy to determine snakebite biomarkers. In this study, we tried to identify *Naja atra* species-specific antigen by a novel strategy. As a model, the identified proteins can be further used as candidates for developing venomous snakebites detection kits.
Methods: *Naja atra*-specific antibodies (NA-SSAbs) were purified by affinity chromatography. *Naja atra* venom immunized horse plasma was allowed to flow through the 5 columns which contained beads coating with the other 5 venomous snakes, respectively.[1] After this step, we could produce NA-SSAbs. Secondly, the specificity of NA-SSAbs was analyzed by immunoblotting and ELISA. Then, the NA-SSAbs corresponding antigens of *Naja atra* venom were obtained by immunoprecipitation. After trypsin in-gel digestion, liquid chromatography-mass spectrometry (LC-MS/MS) analysis and Swiss-Prot database searching the target protein identifications were obtained. Lastly, species-specific antigen C3 was verified.
Results: A *Naja atra* species-specific antigen C3 was identified (Table 1).

Table 1. The list of target antigens of *Naja atra* species-specific antibody with high confidence.

Bands	Name	Score	Coverage	Unique Peptides	Peptides	PSMs	MW [kDa]
A	NA species-specific antigen A1	391.12	58.9	3	6	11	16
	NA species-specific antigen A2	132.24	28.57	1	3	4	14
	NA species-specific antigen A3	277.33	28.08	1	3	5	16.1
B	NA species-specific antigen A1	792.74	74.66	5	8	20	16
	NA species-specific antigen A3	505.17	52.05	2	5	10	16.1
C	NA species-specific antigen 3	198.41	22.22	1	2	9	9
	NA species-specific antigen C5	169.45	28.4	2	3	8	9
	NA species-specific antigen C6	135.92	34.57	2	3	6	9
	NA species-specific antigen C9	194.49	50	2	4	7	7
	NA species-specific antigen C8	123.72	34.94	1	3	5	9.3
	NA species-specific antigen C7	160.17	30.49	1	2	4	9.1
D	NA species-specific antigen C3	427.07	61.73	1	8	28	9
	NA species-specific antigen C4	681.74	67.9	2	9	36	9.1
	NA species-specific antigen C6	379.87	65.43	3	7	25	9
	NA species-specific antigen C1	654.64	70	2	6	32	6.7

Viper envenomation in Italy
clinical course, laboratory investigations and antidote treatment in a 11 years case series (2002-2012) from Pavia Poison Centre

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INTRODUCTION

Viper envenomation may be characterized by severe local/systemic symptoms with an estimated mortality up to 1%. Clinical and laboratory disorders and antidote administration are often debated (1). Poisoning severity, laboratory alterations and antidote administration in viper-envenomed patients referred to Pavia-Poison-Centre (PPC) are described in order to evaluate predictable clinical and laboratory factors in viper envenomation management.

METHODS AND MATERIALS

All viper bitten patients referred to PPC from 2002-2012 were retrospectively studied among those clinically followed until conclusive outcome. Clinical manifestations and evolution were evaluated according to a Grading-Severity-Score (GSS) (2). Laboratory parameters and antidote treatment were evaluated and related to GSS at acme.

GRADING SEVERITY SCORE (GSS)

0	Fang marks only, no other signs (dry bites)
1	Edema around the bite wound, no systemic signs.
2	Extensive swelling. Presence of moderate systemic symptoms like diarrhea, vomiting, hipotension, mild neurological signs, ptosis
3	Giant swelling (extended to the whole extremity/trunk) and/or presence of severe systemic manifestations (ie. shock, hypotension, bleeding, DIC…).

RESULTS

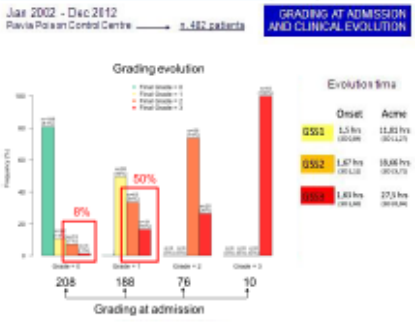
GEOGRAPHICAL AND AGE DISTRIBUTION

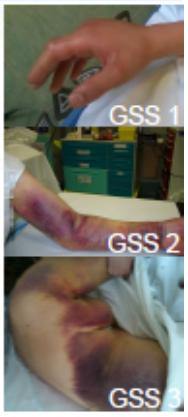
482 patients were included in the study, selected over the 11 years study period. Average age was 44±23 yrs and 65% were males. 170 (35%) never showed signs of envenomation (dry bites), while 312 (65%) manifested clinical signs and symptoms. In figure 1 and 2 geographic, seasonal and age distribution are represented.

Figure 1

Figure 2

GRADE SEVERITY SCORE (GSS) AND CLINICAL EVOLUTION



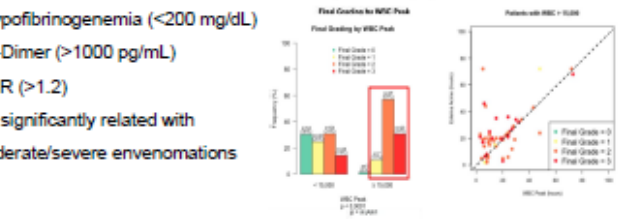


Local and extensive edema have mean time of 1,6 hrs. Acme was reached at the 11,8 hours and 27,5 hrs for mild and severe edema respectively

LABORATORY ANALYSIS

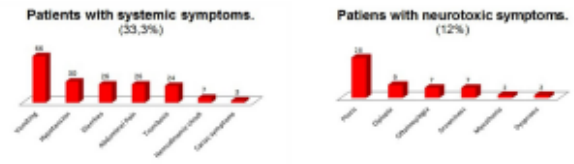
- Leukocytosis (>15000/mm3)
- thrombocytopenia (<150000/mm3)
- hypofibrinogenemia (<200 mg/dL)
- D-Dimer (>1000 pg/mL)
- INR (>1.2)

are significantly related with moderate/severe envenomations



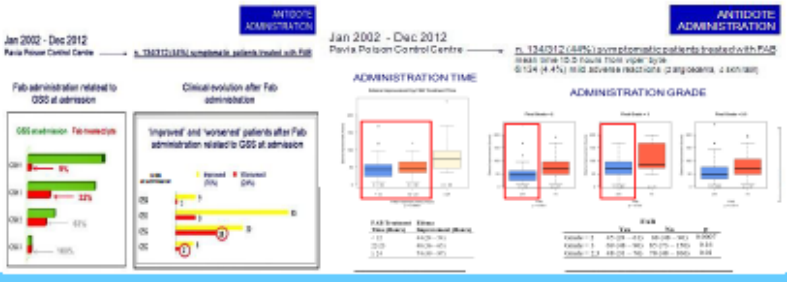
SYSTEMIC SYMPTOMS

Systemic symptoms are mainly gastrointestinal (118/312; 38%), hemodinamic (37/312; 11,8%), neurotoxic (36/312; 11,5%) and local thrombosis (24/312; 8%). Seven patients presented hemodinamic shock and 3 distrectual ischemia. No fatal cases were registered.



ANTIDOTE TREATMENT

137/312 (44%) envenomed patients (63-100% GSS2-3) were treated with the antidote which was injected at an average time of 15.5 hrs after the bite. Most treated patients (76%) improved their clinical picture; however 24% manifested worsening of local edema after antidote administration (most of these presented GSS≥2 at ED admission within the first 6 hrs from the byte).



CONCLUSION

Viper bite is a potentially serious event that requires immediate hospital care. GSS0-patients at hospital admission may worsen and require antidote within 12-24 hours after the byte. Leukocytosis and increased d-dimer are related with severe envenomation. Prompt antidote prescription is important and further administration may be evaluated in patients that present severe envenomation already at hospital admission.

Reference
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