Clinical aspects and consequences of envenoming by a captive Rhinoceros viper (Bitis nasicornis) in Hungary

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Abstract

A case of Rhinoceros viper (Bitis nasicornis) bite is reported. The bitten hand distended to the wrist, which was tense and painful, with only mild local livid discoloration manifested around the fang mark. Slight hypertension with moderate tachycardia and temporary coagulopathy were observed. The patient received analgesic and intravenous fluids, antibiotics and anti-tetanus therapy. Use of antiserum was not necessary. The bitten person was treated in the main centre for snake-bite first aid: in the Toxicological Ward of Erzsébet Hospital of Budapest. We attach importance to the implications of this case because envenoming by B. nasicornis being relatively rare in captivity all over the world (particularly in Europe and the USA), as well as in the wilderness in Africa.

Key words: snake; first aid; venom; coagulopathy; oedema

Introduction

Snake-bite accidents caused by different venomous species deserve much more attention worldwide [1], especially since there is limited experience and a paucity of information available for bites (and their consequences) from certain species, e.g., B. gabonica or this case of B. nasicornis. These are medically important species, mainly due to their highly toxic venom and large venom yield. The Rhinoceros viper (Bitis nasicornis) is one of the largest vipers in Africa with its total length 100–150 cm, however the adult size of this species differs across its range [2]. Its distribution spreads from West Africa through Central Africa to western Kenya, thus its geographical range partly overlaps with that of the closely related B. gabonica [2, 3].

In Africa, the Puff adder (Bitis arietans) is responsible for the majority of bites from the Bitis genera [3] due to its large distribution and diverse habitats, while the other two puff adder species mentioned above have hardly ever inflicted accidents in their original habitats [3, 4]. Unfortunately, there is a lack of experience in B. nasicornis envenomations and the symptoms caused, as accidents are rare in its natural environment due to its sedentary and nocturnal lifestyle [2, 3]. Data of B. nasicornis bites in their natural habitats among the local human population are often unreliable, as positive identification of the snake seldom occurs. Envenomings are not frequent in captivity either, probably due to the snake’s calm behaviour [5]. Only four bites of B. nasicornis have been recorded, all in the USA [6–8]: one in Minnesota in 1965 [6], later another victim suffered two bites in St. Louis, Missouri in April then in May in 2002 [7], and one fatal accident occurred in Dayton, Ohio in 2003 [7, 8].

Nowadays the keeping of tropical venomous snakes is in fashion in Hungary [9]. The species of the genus Bitis are beloved and all three big-bodied Bitis species (B. arietans, B. gabonica, B. nasicornis) are present in private collections. Bitis arietans has already been involved in several severe or even life-threatening accidents in the last decades in Hungary [9, 10]. On account of the similar effects of the toxin of B. nasicornis compared to the venom of B. gabonica, toxicologists rely on experiences gathered during accidents caused by this species when regarding the consequences of B. nasicornis intoxication [11]. For this reason we attach particular importance to reporting the circumstances of a single B. nasicornis accident and its clinical outcomes, discovered during a nationwide survey of snake-bite incidents in Hungary.
Case report

A 30-year-old healthy male was transported by car to the Toxicological Ward of Erzsébet Hospital of Budapest, the main centre for snake-bite first aid, on 9th November 2001. The person was bitten by an adult (90–100 cm long) male Rhinoceros viper (B. nasicornis) (fig. 1). He had never been bitten previously (patient’s personal comment). The snake’s last feeding date was unknown. The captive bred specimen’s ancestral origin was Ghana, West Africa. The specimen was estimated to be 2–3 years old and its owner bought it, when it was still juvenile (patient’s personal comment). According to the patient, the snake had always been calm and handled easily and special caution was only necessary during the snake’s feeding time. The accident happened when the owner pulled the viper from its cage with a snake-hook.

After being bitten the victim was transported to hospital within an hour and did not receive any first aid until admission. The bite occurred with one fang only and was clearly visible on the pulp of the patient’s right index-finger (fig. 2). At admission he had clear consciousness, was wide-awake and in a good mood considering the circumstances. Local symptoms were moderate: the bitten hand was oedematous (oedema extended to the wrist), the fingers were moderately swollen and tense, and slight livid discolouration developed around the fang mark (fig. 2). No other local symptoms occurred. He experienced slight hypertension, with systolic pressure of 155 mmHg, and diastolic of 80 mmHg. The ECG did not show any abnormal changes despite the fact that the venom of B. nasicornis can affect heart function [11, 12]. Only moderate tachycardia (105/min) was observed at the time of admission. Of the laboratory findings some rates, especially those of the cardiovascular system, diverged from the normal ones (table 1). The envenomed person had mild temporary coagulopathy (table 1), represented by the following laboratory results: INR 1.30; PTT 32 sec; and IVY 1.15 min.

Infusion (lactated Ringer solution 500 ml) and strong antiphlogistic (Tramadolium chloratum 50 mg i.v.) were administered at admission. Infusion is administrated routinely following mild to moderate snake-bite envenomation in Hungary [9]. Furthermore the patient received tetanus antitoxin injection (1 ampoule − 5 ml) and antibiotic therapy (Clindamicin 2×300 mg). The analgesic was necessary due to the tension and intense pain of the hand. Antivenom therapy was not considered. Hospital observation lasted for 4 days only. The patient complained of pain – presumably due to arthralgia of the bitten hand – for two weeks after the incident.

Discussion

There are differences of opinion regarding routine antibiotic use in cases of snake-bite. On the one hand the risk of routine antibiotic therapy leading to increases in resistant bacterial phyla [13] is well known, on the other, antibiotic use is not essential in many snake-bite incidents [14, 15]. Many authors [16–18] assume that routine antibiotic therapy is necessary due to the bacterial flora of snakes’ saliva, especially in cases of accidents caused by a species with haemo-cytotoxic venom such as the family of Viperidae [18].

Justifying the use and explaining the benefit of anti-tetanus therapy is problematic, in particular since tetanus after snake-bite has not been documented [19]. Necrosis, blistering and abscess formation are often observed following Bittis bites [4, 8, 20, 21] but are caused in part by actions of the haemo- and cytotoxicity of the venom [18]. Local necrosis evolves on account of the properties of the venom (i.e., haemo- cytotox- and myotox- icity) [1, 18], possibly interacting with the bacterial flora of snake’s saliva, and the natural flora of human skin [18].

A similar case was recorded in 2002, when severe necrosis necessitated amputation of the patient’s finger following snake-bite [7]. In our case...
Table 1

<table>
<thead>
<tr>
<th>Laboratory analysis</th>
<th>Patient’s rates 09.11.2001 (Unit)</th>
<th>Patient’s rates 10.11.2001 (Unit)</th>
<th>Normal rates 26, 27, (Unit)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Blood</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WBC</td>
<td>9.2 (M/μL)</td>
<td>12.3 (M/μL)</td>
<td>4.5–11.0 (M/μL)*</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>22.9 (%)</td>
<td>12.6 (%)</td>
<td>17–45 (%)</td>
</tr>
<tr>
<td>Granulocytes</td>
<td>73.4 (%)</td>
<td>83.0 (%)</td>
<td>42–74 (%)</td>
</tr>
<tr>
<td>Lymphocytes abs.</td>
<td>2.1 (G/l)</td>
<td>1.6 (G/l)</td>
<td>1.5–4.0 (G/l)</td>
</tr>
<tr>
<td>Granulocytes abs.</td>
<td>6.8 (G/l)</td>
<td>10.4 (G/l)</td>
<td>2.0–7.5 (G/l)</td>
</tr>
<tr>
<td>RBC</td>
<td>5.38 (M/μL)</td>
<td>4.90 (M/μL)</td>
<td>4.5–5.8 (M/μL)*</td>
</tr>
<tr>
<td>Hæmatoglobin (Hb)</td>
<td>163 (g/l)</td>
<td>150 (g/l)</td>
<td>130–180 (g/l)*</td>
</tr>
<tr>
<td>Hæmatocrit</td>
<td>0.51 (l/l)</td>
<td>0.473 (l/l)</td>
<td>40–50 (%)</td>
</tr>
<tr>
<td>Mean Cell Volume (MCV)</td>
<td>96 (fl)</td>
<td>97 (fl)</td>
<td>80–100 (fl)</td>
</tr>
<tr>
<td>Mean Cell Hæmatoglobin (MCH)</td>
<td>30.3 (pg)</td>
<td>30.6 (pg)</td>
<td>27–32 (pg)</td>
</tr>
<tr>
<td>Mean Cell Hæmatoglobin Concentration (MCHC)</td>
<td>318.9 (g/l)</td>
<td>315.6 (g/l)</td>
<td>300–350 (g/l)</td>
</tr>
<tr>
<td>Thrombocyte count</td>
<td>271 (G/l)</td>
<td>250 (G/l)</td>
<td>150–400 (G/l)</td>
</tr>
<tr>
<td>Thrombin time</td>
<td>20 (sec)</td>
<td>19 (sec)</td>
<td>16–21 (sec)</td>
</tr>
<tr>
<td>Fibrinogen</td>
<td>2.0 (g/l)</td>
<td>1.8 (g/l)</td>
<td>1.5–3.0 (g/l)</td>
</tr>
<tr>
<td>Activated Partial Thromboplastin Time (PTT)</td>
<td>32 (sec)</td>
<td>33 (sec)</td>
<td>&lt;29 (sec)</td>
</tr>
<tr>
<td>International Normalised Ratio (INR)</td>
<td>1.30</td>
<td>1.70</td>
<td>–</td>
</tr>
<tr>
<td>Coagulation time</td>
<td>4.10 (min.)</td>
<td>4.25 (min)</td>
<td>&lt;9 (min)</td>
</tr>
<tr>
<td>SCOT</td>
<td>43 (U/l)</td>
<td>–</td>
<td>&lt;10 (U/L)*</td>
</tr>
<tr>
<td>Alkaline phosphatase</td>
<td>143 (U/l)</td>
<td>–</td>
<td>25–100 (U/l)</td>
</tr>
<tr>
<td>Carbanid</td>
<td>8.0 (mmol/l)</td>
<td>–</td>
<td>3.0–8.0 mmol/l</td>
</tr>
<tr>
<td>IVY</td>
<td>1.15 (min.)</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td><strong>Urine</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RBC</td>
<td>3–4 (C/visual area)</td>
<td>–</td>
<td>&gt;2*</td>
</tr>
<tr>
<td>WBC</td>
<td>5–6 (C/visual area)</td>
<td>–</td>
<td>&gt;2*</td>
</tr>
</tbody>
</table>

**anti-tetanus prophylaxis and antibiotic (clindamycin 2x300 mg/d) administration were deemed necessary to prevent secondary infections. Antibiotics and tetanus antitoxin are often applied for snake-bite therapy in Hungary [9]. The venom of *B. nasicornis* is haemo-cytotoxic, potently destruc-
tive to cellular elements of blood, degrading tis-
sues and causing coagulation disorders [3, 12, 22, 23]. It also contains myocardotoxins [11] affect-
ing cardiac function and blood pressure, thus it can readily provoke arrhythmia, cardiac-mediated hypo-
tension and myocardial damage.**

Local and systemic bleeding can occur owing to the haemotoxic components of the venom. The venom yield of an adult specimen is high (200 mg – dry weight) [2], similar to the same-
sized *B. arietans* (150–250 mg – dry weight) [24]. With its long fangs (250–300 mm) the species usually injects the venom directly into the mus-
cles. The lethal dose of venom is only 8.6 mg/kg i.m., thus *B. nasicornis* produces the most toxic venom intramuscularly of the big-bodied *Bitis* species (i.e., *B. gabonica* and *B. arietans*) [20]. An envenoming is potentially as dangerous as one arising from one of the two taxa mentioned above and can be fatal [7]. In Europe, two polyvalent antivenins (Ipsen Africa® Pasteur Vaccins, France; and Anti-Bitis-Echis-Naja Africa®, Institut Pasteur, France) were produced against the bite of *B. nasicornis*, while another polyvalent serum (Saimr Polyvalent Snake Antivenom®, South African Vaccine Producers, Johannesburg, South Africa) is made in Africa. The gender of the snake is important as sexual venom variation is present in this species [12]. The female’s venom contains one component absent from the venom of males [12], which might influence the outcome of a poiso-
ning. Geographical venom variation might also influence the development of symptoms, a fact already proved in many other species [25].

The body size and age of the species affect the ophidism as well. Juveniles of variant species of Viperidae have more toxic venom, which has higher coagulant activity and lower proteolytic effects [25]. Experience shows that *B. nasicornis* bites cause intense pain and moderate to severe oedema of the bitten extremity. Compartment syndrome may also develop in severe cases, as has been observed among victims of *B. arietans* and/or *B. gabonica*. In our case, the local effects were similar to one of the US cases, where also the finger was bitten. Following this latter event the hand and the wrist became oedematous and intensive pain developed after injury [7]. Local necrosis also occurred [7], although this is often less serious and extensive than in the case of *B. arietans* bites.

In this case the patient was lucky not to apply a tourniquet as first aid; since the application of pressure dressings contributes to necrosis at the various *Bitis* bites [17]. Besides the typical local symptoms of puffadder-like bites, systemic symptoms also developed in our patient involving moderate tachycardia, slight hypertension and temporary coagulopathy. Temporary coagulo-
pathy included shortened prothrombin time (table 1), and disappeared spontaneously without any medical intervention. It is likely that different coagulopathies will develop in moderate and severe poisonings by *B. nasicornis* owing to the haemostatic activity of their venom [23]; however these may also appear in mild cases. Fortunately, *B. nasicornis* are seldom encountered in Hun-
garian private collections, probably owing to diffi-
culties in their procurement. The accident in a tourniquet case may have been contributed to by human negligence, as occurs in almost every snake-bite incident, mainly with specimens in captivity [15]. Most probably this was “only” a warning bite, which occurred with one fang only and the snake injected a small amount of venom. Thus our pa-
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