

## LETHAL CASE OF *VIPERA BERSUS* BITE

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### Abstract

*Adder bites are rare events, but they can be fatal. Three adder types live in Romania - Vipera ammodytes, Vipera ursini and Vipera berus. Most adder bites happen during the summer with a peak incidence between July and August.*

*Here we present the case of a 56 years old male patient who was bitten by an adder. The clinical presentation was severe from the beginning with a GCS of 3 points, respiratory and cardiovascular failure; despite of adequate treatment the patient developed multiorgan dysfunction and died 36 hours after the ICU admission.*

*The aim of this report is to raise awareness that snake bites can have a life-threatening course and need immediate attention and medical care.*

**Keywords:** Vipera berus, envenomation, antivenom

### Introduction

Three adder types live in Romania - Vipera ammodytes, Vipera ursini and Vipera berus [1].

Most adder bites happen during summer time, they are possible between May and September, with a peak incidence during July and August. After September adders start hibernating [1].

Adder bite usually consists of two bitten points situated at a 5-12 mm distance. The skin around these lesions usually becomes purplish and sometimes vesicles may appear. In some cases the lesion consists of one single bitten point caused by the retractile teeth [2]. The venom is produced by modified salivary glands situated in the postorbital fossa. It has a white-yellow color; it is odorless and dries at room temperature. The venom volume depends on the size of the snake, the effectiveness of the bite and the existent quantity in the glands at the bite moment [3,4].

The adder venom is a mix of high molecular weight proteins, principally protease, hyaluronidase and phospholipase with cytotoxic and hemorrhagic effects. These affect especially the endothelium causing increased vascular permeability, edema and hypovolemia. Hyaluronidase lysis of the hyaluronic acid in the connective tissue causes hemorrhages and necrosis. Vipera ammodytes

venom contains a particular phospholipase, ammoditoxin A, which produces a pre-synaptic neuromuscular block with ophthalmoplegia and muscle weakness [4].

Clinical findings of adder bites depend on the quantity of the injected venom, the site of the bite, the victim's health condition [4].

### Case Report

A 56 years old male patient, 90 kg, without medical history, was found by the Ambulance in the field, unconscious, with Glasgow Coma Scale 3 points. He was found with warm and wet skin, mydriatic, with oral cyanosis, febrile (41.4 °C), with shortness of breath, peripheral oxygen saturation 60% in atmospheric air, hemodynamically unstable, with hypotension (BP - 70/40 mmHg) and tachycardia (HR-122 bpm), sinus rhythm. At the level of his right thigh he presented two bitten lesions suggesting a snake bite (Figure 1 a). An elastic bandage was applied in the superior third of his right thigh. A rapid sequence induction was performed (Sellick maneuver applied). He was intubated, mechanically ventilated in BIPAP (Bilevel airway pressure mode) and after volemic resuscitation (Cristalloid infusion- 1500 ml of Ringer solution and colloid infusion - Gelofusine 500 ml) the patient remained hemodynamically unstable and vasoactive infusion (Noradrenaline) was started. Corticotherapy (8 mg of Dexamethasone intravenously) and antihistaminic

Manuscript received: 27.09.2015

Accepted: 27.10.2015

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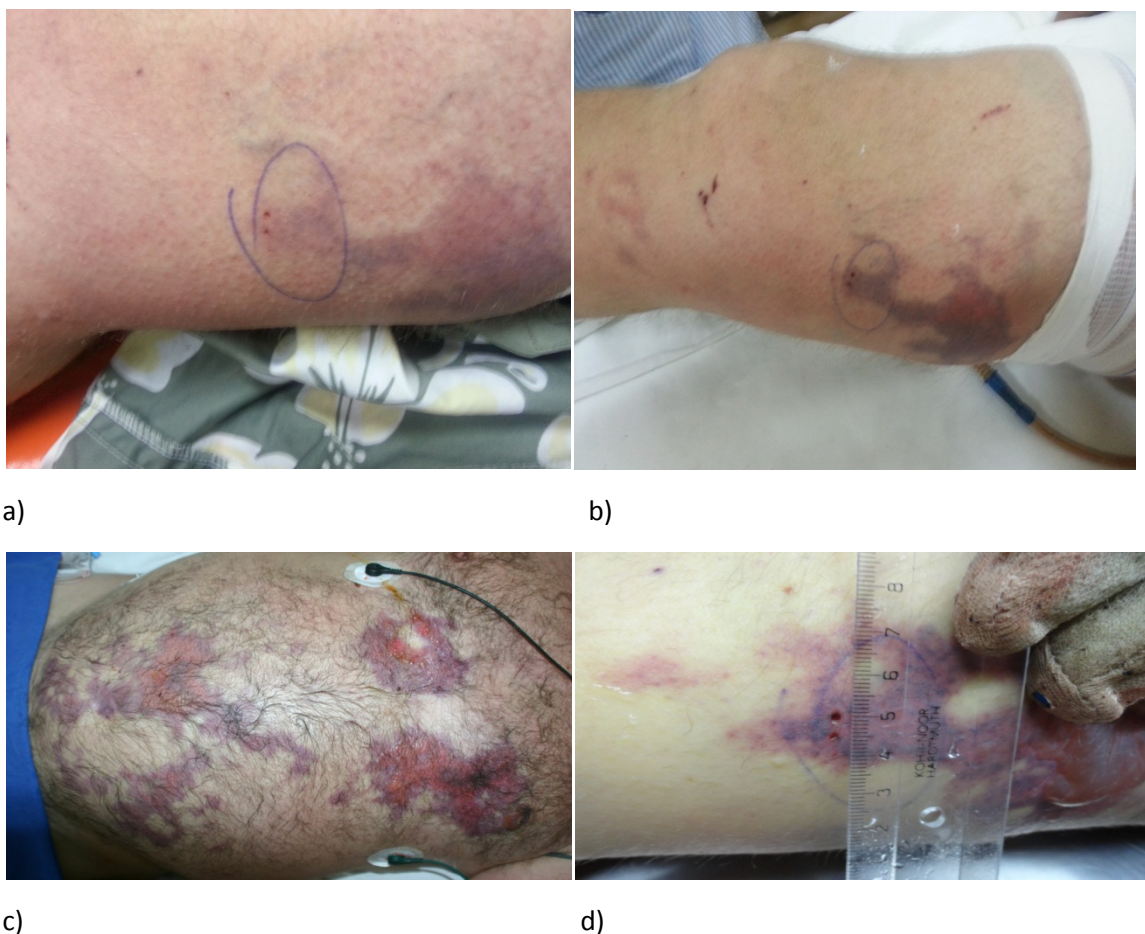
## Case Report

treatment (Clorpheniramine 4 mg sublingual and 50 mg iv of Ranitidine) were administrated. External and internal cooling was applied. The patient was transported to the Emergency Room (ER).

In the Emergency Room the first dose of Zagreb viper antivenom serum was given. The patient remained comatose, intubated and mechanically ventilated, hemodynamically unstable with hypotension and tachycardia despite continuous fluid infusion and vasoactive drug. Adrenaline infusion was associated in this condition. The patient was oligo-anuric (urine analysis revealed intense proteinuria and hematuria). At the level of his right thigh the skin became purple around the two bites (Figure 1b). Biological findings in the ER are shown in Table I. Blood gas analysis revealed metabolic acidosis with increased lactate (pH of 7.25, PaCO<sub>2</sub> of 44 mmHg, PaO<sub>2</sub> of 59.8 mmHg, HCO<sub>2</sub>- of 18 mmol/l, BE of -7 and lactate of 34 mg/dl). Chest x-ray showed accentuated interstitial markings. Head computed tomography was without acute lesions. Second dose of Zagreb viper antivenom serum was given at two hours after the ER presentation.

The patient was transferred to The Intensive Care Unit (ICU). After the ICU admission the patient was maintained sedated, intubated and mechanically ventilated. He presented decreased pulmonary compliance and increased airway resistance, PaO<sub>2</sub>/FiO<sub>2</sub> less than 100; neuro-muscular blocking agents were administrated in order to facilitate ventilation. He continued to be hemodynamically unstable with increased dose of vasoactive and inotrope infusion. He was oligoanuric and proteinuria and hematuria were still present in the urine analysis in the first hours after admission. Biological parameters are shown in table I. Severe coagulopathy was present and needed multiple fresh frozen plasma and cryoprecipitate transfusions. A central venous catheter was placed on the internal jugular vein with measured central venous pressure of 1 cm H<sub>2</sub>O. In this condition volemic resuscitation was continued using colloid and crystalloids.

After the patient's ICU admission he was continuously febrile with a body temperature around 40 °C despite internal and external cooling, purplish petechia with vesicles appeared on his thorax and abdomen (Figure 1 c).



**Figure 1.** Bite aspect a) at admission, b) at 11.30 PM, c) 24 hours after admission, d) postmortem.

**Table I.** Blood analysis of the patient.

	ER	ICU admission	ICU 10 hours after admission	ICU 22 hours after admission	ICU 33 hours after admission
Hb g/dl	14.9	14	13.3	11,7	9.2
Ht (%)	48	43	39	36	28
Leucocyte/dl	3600	10400	7800	4900	5700
Platelets/ dl	46000	70000	49000	25000	28000
GOT U/l	62	86	101	586	2379
GPTU/l	100	238	241	824	3735
Total Bilirubin mg/dl	0.3	0.5	0.7	2.5	4.4
Direct Bilirubin mg/dl	0.21	0.36	0.4	1	2.8
Na mmol/l	146	151	150	153	152
K mmol/l	2.6	3.4	2.4	2.9	5.9
CK u/l	161	456	543	866	990
CK-MB u/l	30	100	199	223	233
Urea mg/dl	35	69	83	99	109
Creatinine mg/dl	1.33	2.5	2.8	3.8	4.4
Glycemia mg/dl	183	67	190	170	88
LDH u/l	255	1104	1474	2654	7200
TQ sec	23	41.8	42	45	39.8
INR	2.22	4.56	4.6	5	4.29

During his ICU admission the patient was comatose; he also developed severe acute respiratory distress syndrome with hypoxemia and hypercapnia; he was continuously hemodynamically unstable; he developed severe renal failure with anuria. Severe coagulopathy and thrombocytopenia were still present despite fresh frozen plasma, cryoprecipitate and thrombocytes transfusion. In this context the patient developed upper gastrointestinal bleeding. Acute hepatic failure developed with increased liver enzymes, increased bilirubin and decreased cholinesterase. A rhabdomyolysis syndrome was present. Disseminated intravascular coagulation was confirmed due to decreased platelet number, altered coagulation, decreased fibrinogen and increased D-dimer (Table I).

In the context of multiple organ failure (neurologic, respiratory, cardiovascular, renal, hepatic and hematologic) the patient deceased 36 hours after ICU admission.

At the postmortem morpho-pathological examination the following lesions were found:

1. Cerebral edema and hemorrhagic lesions in the leptomeninges

2. Atelectasia and pulmonary hemorrhagic edema
3. Myocardial fibrosis, epicardial and interstitial hemorrhages
4. Enteral necrosis and acute mucosal hemorrhages
5. Diffuse hepatic necrosis
6. Acute tubular necrosis
7. Diffuse epidermal necrosis associated with micro-hemorrhages and micro-thrombosis in the dermis and hypodermis.

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