CLINICAL CASE

SNAKE BITE OF A DIAMONDBACK RATTLESNAKE - CLINICAL CASE AND MANAGEMENT

Anca Colcigeanu¹, Nicoleta Ferariu¹, Nicoleta Unc², I. Lascăr^{1,2}, Roxana Gabriela Chiș², Ioana Teona Sebe^{1,2}

¹The Emergency Clinical Hospital Bucharest

²The University of Medicine and Pharmacy "Carol Davila", Bucharest, Romania

Corresponding author: Ioana Teona Sebe

Phone no. 0040744330967

E-mail: sebeioanateona@yahoo.com

Abstract

Rattlesnake envenomation incidence and its severity remain largely misunderstood in Europe. The evolution of cases reported in the few countries, where these accidents are correctly reported, proves to be unpredictable. Rattlesnake venom is mainly hemotoxic, affecting mainly the blood vessels, blood cells and the heart. The venom contains zinc metalloproteinases, cytotoxins and myotoxins. As an additional effect, the necrosis of skeleton muscles is produced by the venom through secondary ischemia and reduced perfusion. We will present the case of a 30-year-old lady, snake and tarantula breeder from Bucharest, presented at the emergency department on the 15Th of March 2015 after one of her pets, a diamondback rattlesnake had bitten her wrist.

Keywords: snake bite, diamondback rattlesnake, venom, cytotoxins, myotoxins

Introduction

Rattlesnake envenomation incidence and its severity remain largely misunderstood in Europe. The evolution of cases reported in the few countries, where these accidents are correctly reported, proves to be unpredictable. Crotalus Atrox, the western diamondback rattlesnake, is responsible for the majority of snake bites fatalities in South America and Mexico [1,2].

Rattlesnake venom is mainly hemotoxic, affecting mainly the blood vessels, blood cells and the heart. The venom contains zinc metalloproteinases, cytotoxins and myotoxins. As an additional effect, the necrosis of skeleton muscles is produced through secondary ischemia and reduced perfusion [3,4].

Case presentation

We will present the case of a 30-year-old lady, snake and tarantula breeder, from Bucharest, who came at the emergency department on the 15Th of March 2015, after she was bitten by one of her pets, a diamondback rattlesnake. The patient presents herself in the emergency room within the first 3 hours from the snake bite. First clinical exam points out a small bitten wound at the radio carpal articulation of the left arm and moderate pain in the lower third of her left arm. At the hospital admission, the patient was conscious, cooperative, normal with neurological respiratory and cardio-vascular exam. Local examination showed two fang marks with significant erythema and edema of the subcutaneous tissue, tender tissues surrounding the marks, both active and passive movements of the arm were painful (Figure 1).



Figure 1 - Hospital admission

During the first 6 hours the clinical course was not modified. The biological tests reveal a slight decrease of the fibrinogen (184.000 mh/dl) and an increase of the creatine kinase (65000 UI/L).

We initiated the replete fluid therapy, electrolyte rebalancing, anticoagulation with unfractionated heparin, corticosteroids in high doses, antihistamine, prophylactic broad spectrum antibiotic and pentoxifylline. Pentoxifylline acts in particular by increasing red blood cell deformability, by reducing blood viscosity and by decreasing the potential of platelet aggregation and thrombus formation [5].

In the first 12 hours, the aspect of the left superior member modified as following: important erythema, marked edema, translucent skin which highlights the subcutaneous thrombosis, functio laesa, flexing and extensing along with active and passive mobility abolished in the first joins (RCA, MFA, IFA) and reduced at elbow joint, numbness of the fingers. All these facts added pointed to compartment syndrome therefore requiring the surgical indication (Figure 2).



Figure 2 - Compartment syndrome

Anesthetic and surgical risk was assed as ASA III.

The surgical intervention was acomplished as seen below:

The 1st step surgery was performed on the third of March: the "S" decompression incisions on the dorsal face of the hand starting with the 3rd metacarpophalangeal articulation and extended over the 3rd digital projection of the left hand up to the elbow joint (Figures 3,4).



Figure 3 - Decompression, posterior incision, thrombosis



Figure 4 - Thrombosis

On the anterior face of the hand we started the incision of the carpal tunnel extending it to one third of the length of the left arm. After dissection we detected multiple microthrombosis at the dermal level, with thrombotic suffusions of the extensor muscles of the forearm flexors (Figure 5).



Figure 5 - Microthrombosis of flexors fascia

Fasciotomy of the left forearm and arm was performed, strong hemostasis and an abundend lavage with antispectic solutions. Bandages soaked in revulsive solutions were applied followed by the immobilization of the affected limb

Postoperatively, the general status of the patient remained stable. With normal body temperature, conscious, semi-cooperative, having a cardio-pulmonary status within 1 normal limits, a slender abdomen, normal diuresis, bowel movement. The hematological modified probes (leuc 12670/mmc, hb 9,9, Ht 26,9%, TH 108x1000/l) required the administration of 2 units of CEF.

The local evolution was slow favorable with the partial remission of the inflammatory edema of the affected limb in within days. A second intervention is decided due to the local evolution.

The 2nd Surgery was performed on the 18th of March and was practiced for the separate suture threads and onhold threads. Bandages with revulsive and antiseptic solutions were applied (Figures 6,7,8).

We noted the remission of the inflammatory process, partial regaining of the mobility and normal sensitivity.



Figure 6 - Fasciotomy, fasciectomy



Figure 7 – Fasciotomy, fasciectomy



Figure 8 – Aspect of posterior incision



Figure 9 – Final aspect



Figure 10 – Final aspect

On the 24 the of March, our patient was discharged against medical advice; this was the climax of a poor doctor-patient communication. The patient recovered full function of the limb with 2 well healed anterior and posterior scars (Figure 9,10).

Conclusion

Snake bite envenomation produces local and systemic effects due to the presence of proteases, hyaluronidases, and peptides in the venom. 2 Forty-eight percent of bites occur on the feet and 52% on the hands [1]. Death can occur between six and 60 hours after a snake bite [2]. Systemic effects appear within minutes and include hypotension; victims may faint and children, in particular, become drowsy or semiconscious. Locally, there is increased vascular permeability fluid allowing accumulation and increased fluid pressure within the limited space, which if not treated can compromise vascularity leading to ischemic contracture and the worst outcome, amputation. Snake bites on the hand lead to early systemic manifestations due to its rich vascularity and the skin over the dorsum of hand being very thin [3].

References

[1]JM Gutiérrez, T Escalante, A Rucavado, C Herrera, Hemorrhage Caused by Snake Venom Metalloproteinases: A Journey of Discovery and Understanding - Toxins, 2016.

[2]C Yañez-Arenas, AT Peterson, P Mokondoko et al., The use of ecological niche modeling to infer

potential risk areas of snakebite in the Mexican state of Veracruz, PloS one, 2014.

[3]RY Lefkowitz, J Taylor, D Balfe, Reality Bites A Case of Severe Rattlesnake Envenomation, Journal of intensive care medicine, 2013.

[4]CW Hwang, FE Flach, Recurrent Coagulopathy after Rattlesnake Bite Requiring Continuous Intravenous Dosing of Antivenom, - Case reports in emergency medicine, 2015.

[5]DM Aviado, JM Porter, Pentoxifylline: a new drug for the treatment of intermittent claudication; mechanism of action, pharmacokinetics, clinical efficacy and adverse effects, Pharmacotherapy: The Journal of Human Pharmacology and Drug Therap, 1984 - Wiley Online Library.