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Acute renal failure following a viper bite

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ABSTRACT

Snake bites kill around 100,000 people worldwide each year. The management of the envenomations implies rigorous monitoring in order to detect, rather, the first signs of serious evolution (hemorrhage, neurotoxic syndrome, collapse) which indicate the administration of immunotherapy and hospitalization in intensive care. We present the case of an adult patient who was the victim of a snake bite. Despite the administration of antivenom and adequate hydration, she developed acute renal failure secondary to disseminated intravascular coagulation and thrombotic microangiopathy. Recognition of symptoms, close monitoring, prompt diagnosis and treatment are imperative to prevent acute complications and long-term morbidities.

INTRODUCTION

Snake bite poisoning is a major public health problem, and can lead to death from neurological and / or hematological disorders. This problem is compounded by the high cost and availability of antivenom (AVS). Approximately 5.5 million / year of snakebites worldwide have been described, including 125,000 cases of death [1]. Kidney damage is rare. There are 3500 known species of snakes, only one tenth is poisonous and dangerous. These species belong to five groups: elapids (Najas, Mamba); vipers (Echis, Bitis); hydrophilic (sea snake); colubrids and crotalids. In the Maghreb, the most frequent are the poisonings by Lebetine viper (Viperalebetina), horned viper or sand viper (Cerastescerastes), and sometimes Bitis or Echissp [2].

MATERIALS AND METHOD

Medical observation:

We report the case of a 53-year-old patient, with no particular pathological history, housewife, living in the South of Morocco (Guelmim region), victim of an envenomation by viper bite (BitisArietans) (Figure 1) in level of the right hand, the evolution was marked by local and general complications.

Figure 1: Bitisarietans viper

The local signs started half an hour after the bite, characterized by severe edema of the hand and then of the whole upper limb, cutaneous hyperesthesia of the right hand associated with erythema and pain on pressure from the muscle compartments. Lodge syndrome has been reported in this patient. (Figure 2).

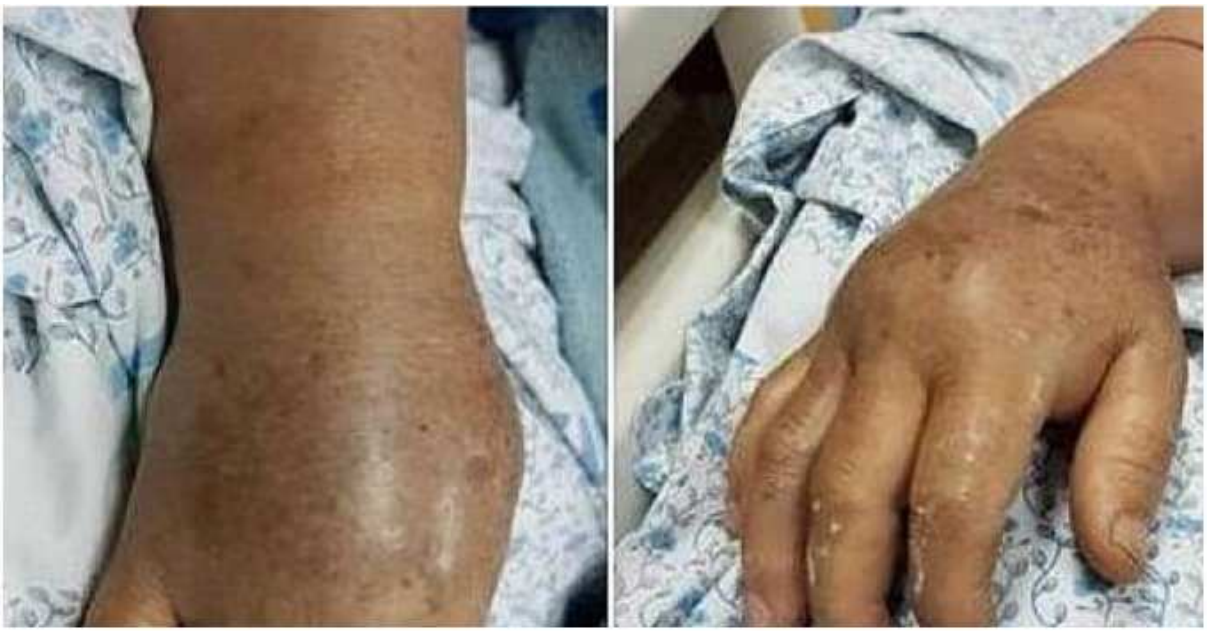

Figure 2: Edema on the back of the right hand extended to the wrist.

At the systemic level, the patient presented with bruises, thrombocytopenia at 50,000 / mm3 and blood crase disorders five hours after admission (Quick time at 32%, Fibrinogen at 1.8 g / l).

She was admitted urgently to the Peripheral Hospital Center of the region in a DIC table. After stabilization of the patient on the hemodynamic and respiratory plan, she benefited from symptomatic measures including a vascular filling with physiological serum, blood transfusions in

pellet Globular (CG) and fresh frozen plasma (PFC), an antibiotic therapy based on amoxicillin 1 gram every 08 hours and metronidazole 500 milligrams every 08 hours, and an analgesia associating paracetamol, morphine and icing with immobilization of the member. Faced with the unavailability of after-sales service, she was transferred to another peripheral hospital center (AGADIR) where she received the anti-venom at H15 from the bite.

The patient was then referred to us (25 days after the viper bite) at the Center Hospitalier Universitaire Mohamed VI in Marrakech for additional specialist care in front of the worsening of her picture by the installation of severe anuric renal failure. requiring emergency dialysis, every other day, with multiple blood transfusions in CG and PFC.

A picture of secondary thrombotic micro-angiopathy has been suspected in thrombocytopenia, hemolytic anemia and malignant hypertension.

The results of the biological balance are shown in the following table. (Table I).

Table I: Results of the patient's biological assessment upon admission

Biological markers	Values
Haptoglobin (g / l)	0,10
LDH (IU / l)	346
CPK (IU / l)	18
Hemoglobin (g / dl)	5
Platelets / mm 3	50 000
TP (%)	32
Fibrinogen (g / l)	1,8
CRP (mg / l)	32
Schizocytes (%)	5
Urea (g / l)	2,1
Creatinine (mg / l)	95,9

LDH = Lactate Dehydrogenase, CPK = Creatinine Kinase, TP = Prothrombin level.

A renal biopsy was performed (03 weeks after the viper bite) showing a thrombotic micro-angiopathy associated with acute tubular necrosis with subacute interstitial nephritis.

The evolution towards chronic end-stage renal disease was the outcome of this patient despite the revival of a good diuresis calculated at 1L500 / 24 hours but without improvement in renal function.

RESULTS AND DISCUSSION

Kidney sequelae represent 1 to 10% of viperine poisoning. Acute renal failure (ARI) is one of the main causes of death in patients bitten by snakes of the Viperidae and Hydrophidae class [3,4]. snakes are concentrated in the kidney tissue in a few minutes and appear in the urine a few hours after inoculation [5, 6,7]. Unless preventive measures are implemented quickly, early renal dysfunction frequently occurs [8, 9, 10]. IRA may be severe, oliguric and dependent on dialysis [9, 11, 12]. Associated clinical results could include, according to the accused snake, those linked to a local injury, rhabdomyolysis, intravascular hemolysis, myocardial injury, neurotoxicity,

hypopituitarism and a systemic inflammatory response syndrome [13, 14, 15, 16 Hematuria and proteinuria are the most common kidney abnormalities in 20 to 70% of reported cases. Oliguria or anuria can develop from a few hours to 96 hours after the bite. Acute tubular necrosis is the most common histological renal abnormality found in ARI due to snake poisoning. A small but large minority of cases develop acute cortical necrosis or acute interstitial nephritis [11, 17, 18, 19, 20]. Cortical necrosis has been described mainly after viper bites, and predicts a poor renal prognosis. Bilateral and diffuse involvement is associated with irreversible renal failure [21,22, 23]. Cortical necrosis should be suspected when the oliguria persists beyond 2 weeks and can be confirmed by renal biopsy or CT scan with improved contrast. Glomerular lesions have also been described and manifest histologically as swelling of the capillaries, division of the glomerular basement membrane, swelling of endothelial cells and focal proliferation of mesangial cells [24].

Several pathogenetic factors contribute to ARI, including hemodynamics, hemolysis, rhabdomyolysis, direct tubular toxicity, systemic inflammation, activation of the coagulation pathway leading to glomerular deposition of microthrombi, stress oxidant, hyperuricemia, release of cytochrome C and induction of apoptosis in the tubular epithelium [12, 18, 19, 25, 26, 27, 28, 29].

The most effective way to prevent ARI is to give adequate antivenom [30].

A prospective study on ARI after a snake bite done in Brazil showed that an interval of more than 2 hours between the bite and the administration of antivenom increased the risk of ARI by 10 times [9].

The basic treatment of viperin envenomations is first based on antivenom immunotherapy with highly purified and species-specific immunoglobulins, and allows healing in three to five days with complete tissue repair [31, 32, 33]. Administration is intravenous and must be early, as soon as signs of severity appear. Since 2012, an antivenom serum has been available in our country: the FAV Africa, which remains accessible at the Pasteur Institute in Casablanca and distributed in several regions of the country.
The morbidity and mortality of snake bites vary by country. In France, there are 2 to 5 deaths per year [34] but in Morocco the epidemiological data concerning the eye bites are insufficient [35]. Of the 217 snakebites reported between 2001 and 2003 by the Poison Control Center in Rabat, 13 people died of poisoning, a death rate of 6%. Among the 05 regions most affected in Morocco, the Marrakech-Tensift-Elhaouz region occupies the second position, surpassed by the Souss-Massa-Daraa region, further south. [36]

In contrast, acute kidney failure, when it occurs, is responsible for 5% of deaths.

The prognosis for poisoning is influenced by the patient's characteristics (age, weight, history, pregnancy) and bite (the type of snake, the amount of venom inoculated essentially, but also the location).

CONCLUSION:

Viperid bite poisoning is the most frequent complication of kidney damage secondary to DIC, rhabdomyolysis, hemolysis or shock, or even the direct toxicity of the venom on the basal membrane of the glomerulus, responsible for " extra-capillary glomerulonephritis. Kidney failure can be reversible if early and effective management is started.

Rapid and immediate transport of the injured victim to a hospital environment with antivenoms and hemodynamic and ventilatory assistance reduces mortality.

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