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Case Report

Disseminated Intravascular Coagulation and Ischemic Stroke due to Snake Bites: About One Case

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Abstract

Snake bites are responsible for a high rate of mortality in Africa. There are multiple problems in the early treatment of victims. The overall mortality from these bites is 4%. Viper bites are known to cause coagulopathy leading to organ and/or system failure with muscle paralysis, respiratory failure, circulatory instability, acute kidney injury and local disability, compartment syndrome and necrosis. Disseminated intravascular coagulation and ischaemic stroke is an extremely rare complication of snakebite that can be fatal if treatment is delayed. Here we present A 15-year-old patient with no history of viper envenomation who consulted our emergency department after an accidental viper bite to the index finger of his left hand rapidly developed extensive oedema, necrosis, haemorrhagic shock are presenting in a state of coma and refractory shock. A CT scan of the brain showed meningeal haemorrhages, intracerebral haematomas and diffuse ischaemic lesions. The laboratory results showed abnormalities such as anaemia, thrombocytopenia, hypofibrinogenemia and elevated D-dimer levels. The patient died of disseminated intravascular coagulation (DIC), which developed within 12 hours of being bitten by a snake. Snakebites can cause DIC because the venom activates the coagulation system and causes fibrinolysis, which occurs in less than 24 hours. A nationwide protocol of management, including specific antivenom therapy and prevention strategies, has been implemented to reduce the overall morbidity and mortality of snakebites.

Keywords: ***Disseminated Intravascular Coagulation, Ischemic stroke, Snake Bites.***

Introduction

In Morocco, viper envenomations are second only to scorpion bites [1]. The clinical and biological presentation is often polymorphic. Local signs are dominated by haemorrhagic syndrome, necrosis and compartment syndrome. The development of local and systemic symptoms is directly related to the toxicity of the venom. Local symptoms may present as oedema, ecchymosis, haematomas and gangrenous lesions, while systemic symptoms may include fever, nausea, vomiting, delirium, jaundice, circulatory collapse, convulsions and coma. m Systemic symptoms may manifest as neurotoxicity, disseminated intravascular coagulation (DIC), intracranial haemorrhage and ischaemic stroke, and acute renal failure may occur 6-48 h after the bite [2,3]. Neurological disorders are exceptional. They may be secondary either to circulatory failure following acute hypotension due to arterial and/or haematological vasodilatation following vasospasm due to perivascular bleeding, or to the direct action of the neurotoxic venom of certain populations of vipers [4].

To reduce the possibility of morbidity and mortality, it is critical to administrate the appropriate dose of antivenom as soon as possible after snakebite [5].

Case Report

A 15-year-old patient with no previous history of viper envenomation consulted our emergency department following an accidental viper bite to the index finger of his left hand. Transferred to our emergency department, presented to our hospital in a generally impaired condition following a snake bite and fainting. he had been bitten by a snake while working in a field 8 hours (h) prior to presentation, and had been brought to the emergency department after her condition deteriorated. The species of snake could not be established. The patient was unconscious during the physical examination; his Glasgow Coma Scale score was 3-4, temperature was 37.6°C, arterial blood pressure was 70/40 mmHg, and pulse was 84, the initial examination revealed local edema of the left hand, with ecchymosis and bite marks. 6 hours after admission, an emergency cerebral CT scan revealed meningeal hemorrhage, intracerebral hematomas and diffuse ischemic lesions. A urinary catheter was inserted into the patient in the emergency department; however, he produced no urine. Biological tests revealed: anemia, thrombocytopenia, hypofibrinogenemia, and increased levels of D-dimer prolongation of APTT, PT and INR. there was no doubt that disseminated intravascular coagulopathy had occurred. The patient was intubated and antiserum was administered. The antiserum was used about 8 hours after snakebite. The outcome was rapidly unfavorable, with the patient dying in a state of coma and refractory shock.

Discussion

Snake bites can cause serious complications due to the enzymes, proteins and inorganic components of the venom. These include local induration, ecchymosis and haematoma, although in this case there were only puncture marks and slight bruising at the site of the bite. Enzymes in snake venom, such as serine protease and arginine ester hydrolase, can contribute to the development of DIC by activating the coagulation system. As a result, prolonged PT and aPTT, low levels of fibrinogen, increased levels of fibrin degradation products and low levels of protein C may be observed in coagulation tests. These haematological abnormalities, which are usually mild, can in rare cases lead to serious complications such as intracranial, pulmonary and intra-abdominal bleeding. The thrombocytopenia and PT and aPTT results in the case presented were indicative of severe haematological damage and DIC, as the PT and aPTT values in the case presented were very high. In the case presented, serious systemic complications had already developed by the time he presented to the emergency department [6].

Ischaemic stroke is reported in nine out of 500 victims after snakebite in a report from studies in Sri Lanka [7]. The mechanisms by which cerebral infarction occurs in snakebite may be multifactorial and are as follows: Venom has anticoagulant and procoagulant effects that can lead to small and even large vessel occlusion due to microthrombi, resulting in cerebral infarction [8]. Snake bites can also cause acute tubular necrosis (ATN) associated with renal damage. Cortical necrosis has also been described in severe envenomations [9,10]. In our case, a urinary catheter was inserted into the bladder, but she did not produce urine.

The severity of the poisoning is related to the difficulty of access to health centres, the use of traditional medicine in more than half of the victims, the lack of training of the caregivers and, finally and most importantly, the lack of antivenom serum, which is not available in the appropriate places and at the appropriate times. Despite its cost and the risk of allergy (which is rare with the current serums), immunotherapy, the only effective weapon against the venom, should be part of the essential emergency drugs. [1].

In the majority of snakebite cases, administration of a sufficient dose of the appropriate antiserum will prevent the development of life-threatening complications [11].

Conclusion

Although rare, viperine envenomation can threaten the vital prognosis, essentially through a systemic haemorrhagic syndrome with visceral localization, as demonstrated by this clinical case. A reassessment and protocolization of the management of fatal biological vascular complications in the emergency department are necessary. nevertheless, primary prevention remains the key to reducing snakebites morbimortality.

Ethics approval and consent to participate

Not applicable.

Conflicts of Interest

The authors declare that there is no conflict of interest regarding the publication of this paper.

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