Toxicon 120 (2016) 124–127

Contents lists available at ScienceDirect

Toxicon

journal homepage: www.elsevier.com/locate/toxicon

Case report

Brainstem ischemic stroke after to *Bothrops atrox* snakebite

Carlos A. Cañas

Department of Internal Medicine, Unit of Rheumatology, Fundación Valle del Lili, Universidad Icesi, Avenida Simón Bolívar Cra. 98 No. 18-49, Cali, Colombia

ARTICLE INFO

Article history:
Received 9 April 2016
Received in revised form
2 July 2016
Accepted 11 August 2016
Available online 12 August 2016

Keywords:
Stroke
Snake bite
Venom
Bothrops atrox

ABSTRACT

We report case of a 48 years old woman bitten on her right foot by a *Bothrops atrox* viper. As a result, she developed a severe coagulopathy which improved with application of polyvalent antivenom. Four days after bite she suffered a devastating brainstem ischemic stroke. Possible pathogenetic mechanisms are discussed.

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1. Introduction

Bites by *Bothrops* genus viper snakes are the most frequent venomous snake bite in Colombia. Most patients live in rural areas. Common clinical manifestations of envenomation are local tissue damage (100%), coagulopathy (~40%) and renal failure (~10%) (Cañas, 2016). Bleeding is the usual presentation of coagulopathy due to both/either direct action of antithrombotic properties of some venom components, consequence of consumption of platelets and coagulation factors following to disseminated intravascular coagulation (DIC) (Cañas, 2016). Hemorrhages are the most common manifestation of central nervous system (CNS) involvement, associated with coagulopathy. Ischemic strokes secondary to arterial thrombosis are a rare and potentially devastating complication. The endothelial damage contributes to clinical expression of the coagulopathy (Bustillo et al., 2015).

We report the case of previously healthy female with brainstem cerebral thrombosis associated with *Bothrops atrox* viper bite. Clinical and laboratory findings are compared with similar reported cases, and possible pathophysiology events are discussed.

2. Case report

A 48-year-old healthy woman was referred to our hospital from Colombian Amazonia (Vaupés state) with history of snake bite on her right foot four days before prior admission, and acute progression of neurological symptoms. The accident occurred while working in the fields near her house. The snake was identified as *Bothrops atrox* (analysis conducted by biologist from a photograph of the body of the snake). A few minutes after the bite, progressive oedema and ecchymosis on the posterior aspect of her right foot were noted, and bleeding at the entry points occasioned by snake's fangs. She received initial help from a local witch doctor who performed rituals and applied plant extracts on her right foot. Eight later, she developed bleeding through her gums, hematomas in both upper arms and hemorrhagic blisters on the right leg, and was taken to a local hospital where she was treated with 9 vials of polyvalent antivenom (each vial containing 10 mL of polyvalent antivenom equine lyophilized, which neutralizes at least 25, 10 y 5 mgr. of snake venom of *Bothrops atrox/asper*, *Crotalus durissus* and *Lachesis muta* respectively; manufactured by Laboratorios Probiol SA, Bogota, Colombia). Four days after bite she became progressively drowsy, and had to be transported by helicopter to our hospital.

On admission, pulse was 74 beats per minute, blood pressure 110/54 mmHg and respiratory rate 18 per minute. There was oedema and hemorrhagic blister over the posterior aspect of right foot (Fig. 1). On neurological examination she was comatose (Glasgow coma scale 14/15), both pupils were miotic without reaction to light and there was generalized hypotonia, bilateral extensor plantar response, and lack of response of limbs to painful stimuli. There were no focal neurological signs. Other systems were normal.

Laboratory tests from her local hospital (18 hours after bite) were: hemoglobin 7.1 g.

E-mail address: cacd12@hotmail.com.

http://dx.doi.org/10.1016/j.toxicon.2016.08.005
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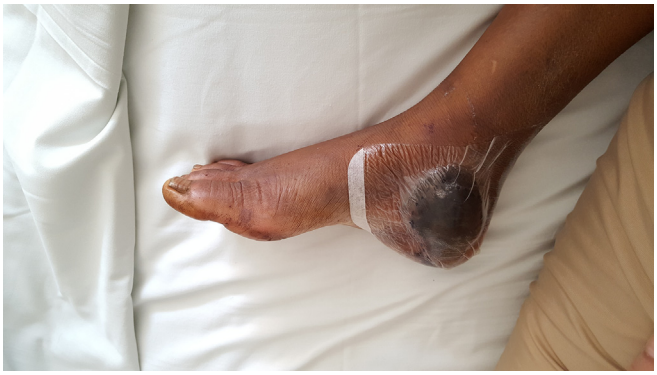

Fig. 1. Site of snake bite. Hemorrhagic blister over the posterior aspect of right foot.

Laboratory tests when she was admitted to our hospital (four days after bite, 12 hour after start of neurological symptoms) were: hemoglobin 8.8 gFig. 2). A diagnosis of severe irreversible cerebral damage was made. Under supportive measures the patient was transferred back to her local hospital for palliative treatment.

3. Discussion

Few cases of cerebral infarction resulting from a viper bite had been reported. A Medline and Scielo data base search using the terms “stroke” and “snake”, from July 1975 to March 2016, yielded 19 articles and 28 patients with cerebral thrombotic complication of snake bite: these were caused one by *Gloydius brevicaudus* (Lee et al., 2001), one by *Crotalus durissus terrificus* (Vale et al., 2013); one by *Hypnale hypnale* (Jeevagan et al., 2012); two by *Echis carinatus* (Bashir and Jinkins, 1985; Murthy et al., 1997); seven by snake of *Bothrops* genus (Numeric et al., 2002; Merle et al., 2005; Angarita and Cárdenas, 2003; Thomas et al., 2006; Mosquera et al., 2003) (see data of seven patient included our case on Table 1); 16 cases were caused by Russell's viper (*Daboia russelli*) (Narang et al., 2009; Ittyachen and Jose, 2012; Subasinghe et al., 2014; Chandrashekar et al., 2014; Kumar, 2015; Deepu et al., 2011; Ameratunga, 1972). In a series of 500 patients of Sri Lankan patients bitten by Russell's viper bitten, 9 (1.8%) had acute ischemic stroke (Gawarammana et al., 2009).

The exact pathophysiology mechanism of thrombosis due to viper bite is still unknown. Snake envenomation produces diverse clinical syndromes due to the in vivo effects of multiple toxic components present in snake venom. Most important among viper snake toxins from a clinical perspective include hemorrhagins, coagulant toxins, nephrotoxins, myotoxins, and necrotoxins (Cañas, 2016). The procoagulant toxins include prothrombin activators, factor X activators, factor V activators and thrombin-like enzymes (Tans and Rosing, 2001; Rosing et al., 2001; Swenson and Markland, 2005). There are large number of thrombin-like enzymes that may have one or several of multiple thrombin-like activities, such as cleaving fibrinogen to release fibrinopeptides A or B, activating protein C, or activating factor V (Serrano and Maroun, 2005; Markland, 1991). In Russell's viper venom several substances and possible mechanism have been identified, among them, the activation of clotting factors, especially factors V, X and prothrombin by procoagulant enzymes such as arginine, esterase, and hydrolase (Mu-Murthy et al., 1997). Moreover, venom containing protease inhibitors, hemorrhagins, several enzymes and phospholipase A 2 cause toxic vasculitis, vasospasm, endothelial injury may also play an important role (Bashir and Jinkins, 1985). The endothelia damage is occasioned by a synergistic effect of PIII metalloproteinase and acidic phospholipase A2 (Bustillo et al., 2015). The hyperviscosity caused by hypovolemic state and hypoperfusion secondary to hypotension may also contribute to vessel occlusion (Narang et al., 2009). A fatal diffuse thrombotic microangiopathy after a bite by *Bothrops lanceolatus* was reported (Malbranque et al., 2008).

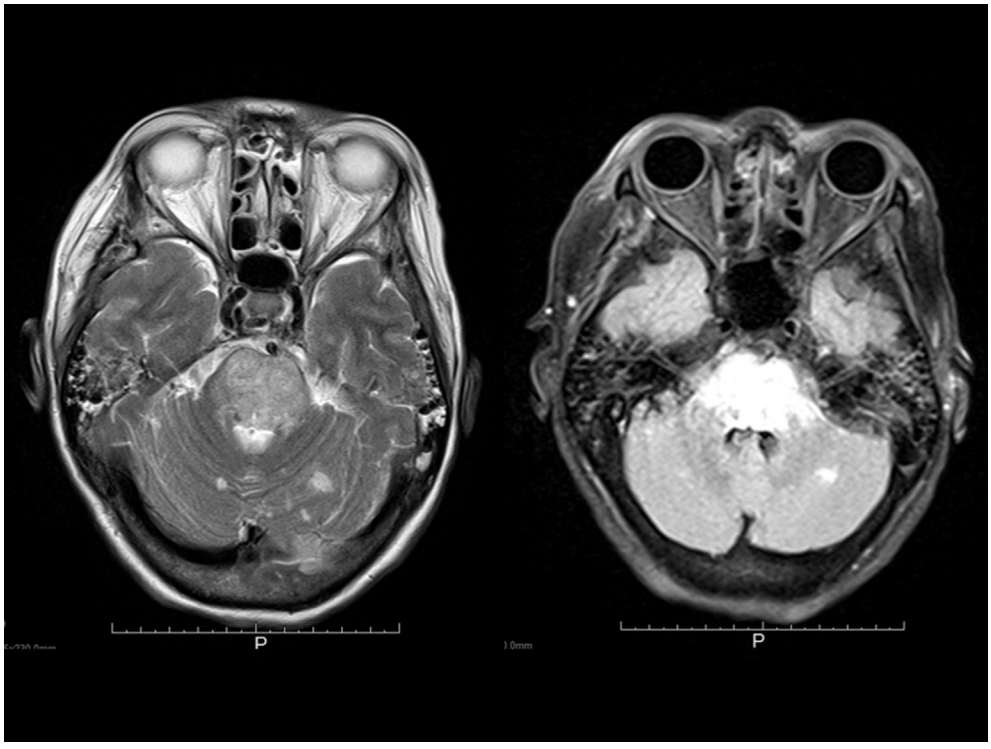

Fig. 2. Magnetic resonance imaging of brain shows severe ischemia in the brainstem. There is an image of a thrombus in the basilar artery.

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