OCCIPITAL INFARCTION REVEALED BY QUADRANOPSIA FOLLOWING SNAKEBITE BY BOTHROPS LANCEOLATUS

HAROLD MERLE,* ANGÉLIQUE DONNIO, LUCAS AYEBOUA, YVES PLUMELLE, DIDIER SMADJA, AND LAURENT THOMAS

Department of Ophthalmology, Hematology Laboratory, Department of Neurology, and Emergency Department, University Hospital Center of Fort de France, Fort de France, Martinique

Abstract. We report a case of snakebite in which envenomation was manifested through impairment of the visual field. The patient, a 46-year-old man, was bitten on the right thumb by Bothrops lanceolatus. Treatment with a specific equine antivenom (Bothrofav®) was administered one hour after the bite. With the exception of fang marks, the results of a clinical examination, particularly the neurologic component, were normal. The day after the bite, the patient developed an inferior left lateral homonymous quadranopsia with macular epargne. T2 magnetic resonance imaging showed a right occipital infarction. His condition improved clinically and biologically. This observation of snakebite is the first in which envenomation was manifested exclusively by an impairment of the visual field. Envenomation by B. lanceolatus is distinct in its incidence of significant thrombotic complications at a distance from the site of the bite.

INTRODUCTION

Approximately 2.5 million people worldwide are bitten by snakes every year, resulting in one death per 120,000 cases.1 In Malaysia, 1,000 deaths from snakebites are reported every year. In Europe, the annual incidence is much lower, with 50 deaths per 20,000 bites. Viper species are most often involved, the victims are primarily men, and the most common site of the bite is the upper limbs.2 However, there are few reports of the clinical or epidemiologic aspects of snakebites. The impairment of visual function by snakebites is rare. The clinical presentation caused by snakebite varies with the species involved and the severity of envenomation.3

Martinique is an island in the French West Indies in the middle of the arch of the Lesser Antilles (latitude 14°36′N, longitude of 62°34′W). Twenty to thirty cases of snakebites are observed each year in Martinique.4 The primary snake responsible is Bothrops lanceolatus (family Crotalidae). It is the only snake present on this island and is not found elsewhere in the world.5 We report one case of bite by B. lanceolatus, in which envenomation manifested through an impairment of the visual field.

CASE REPORT

The patient, a 46-year-old man, was bitten by a snake on the right thumb. He was immediately transported to the emergency department of the University Hospital Center of Fort de France, Martinique, where he arrived 45 minutes after being bitten. The snake was captured by a member of his family. It was identified as Bothrops lanceolatus and was brown with approximately 40 cm long. The patient had no medical or surgical history. Characteristic fang marks were noted at the site of the bite, as well as pain, erythema, and edema limited to the right hand. The severity of envenomation was estimated to be a 2 on a scale of 1 to 4.6 The results of a clinical examination, especially the neurologic component, were normal.

Treatment was instituted immediately (one hour after being bitten) with intravenous administration by electric syringe of 20 mL of a specific equine antivenom fragment (Bothrofav®; Pasteur Mérieux Sérum et Vaccins, Lyon, France) at a rate of 10 mL/hour. A subcutaneous injection of adsorbed tetanus vaccine (Vaccin Tétanique®; Pasteur Mérieux Sérum et Vaccins) and an intramuscular injection of human tetanus immunoglobulin (Gammaténo®; Laboratoire Français du Fractionnement et des Biotechnologies, Courtaboeuf, France) were also administered. A dressing containing alcohol was applied to the hand wound.

He had a normal blood count, a hemoglobin level of 13.3 g/L, and a hematocrit of 38% (normal = 40–54%). Renal and hepatic functions were normal. There was no elevation of levels of cardiac enzymes or C-reactive protein, but thrombocytopenia (52,000 cells/mm³) was observed. He also showed signs of disseminated intravascular coagulation: prothrombin level = 14% (normal = 70–100%), activated cephalin time = 51 seconds (normal = 32 seconds), fibrinogen level < 0.5 g/L (normal = 2–4 g/L), factor II level = 66% (normal = 70–120%), factor V level = 17% (normal = 70–120%), fibrin degradation product = 2,560 μg/mL (normal < 5 μg/mL). The finding of disseminated intravascular coagulation led to additional administration of 40 mL (10 mL/hour) of Bothrofav®. His clinical and biologic therapeutic tolerance was excellent, and no undesirable anaphylactic reaction, immediate or delayed, attributable to the heterologous antivenom was observed.

The day after the bite, he reported blurred vision in the left part of his visual field. The visual acuity of his eyes was 20/20. The results of a biomicroscopic examination of the anterior and posterior segments of the eyes were normal, and the ocular tonicity was 10 mm of Hg. There was no afferent pupillary deficiency, and the photomotor reflexes and ocular motility were normal. A visual field test with Goldmann’s perimeter showed an inferior, left, lateral, homonymous quadranopsia with macular epargne (Figure 1). T2 magnetic resonance imaging (MRI) showed a posterior hypersignal, consistent with an occipital infarction (Figure 2). The patient’s overall condition remained stable and no cardiovascular or neurologic anomalies were noted. The coagulopathies regressed in 24 hours, and platelet level increased after 48 hours. The injury to the right hand healed in two weeks, without necrosis or secondary infection. His ocular progression was favorable and results of a visual field test three weeks after the bite were normal. An MRI conducted one

* Address correspondence to Harold Merle, Department of Ophthalmology, Pierre Zobda-Quitman Hospital, BP 632, 97261 Fort de France Cedex, Martinique. E-mail: harold.merle@chu-fortdefrance.fr

Copyright © 2005 by The American Society of Tropical Medicine and Hygiene

583
year later showed a sequellary hypersignal, indicating occipital ischemia.

**DISCUSSION**

In this patient, envenomation resulted in an infarction in the posterior cerebral artery, with thrombocytopenia and disseminated intravascular coagulation. The most important symptom was inferior, left, lateral, homonymous quadransia. The clinical and biologic progressions were favorable. To our knowledge, this snakebite was the first in which envenomation was exclusively accompanied by an impairment of visual field.

All venoms of snakes of the *Viperidae* and *Crotalidae* families are hemotoxic. However, certain species of vipers possess neurotoxins in their venom capable of causing oculomotor paralysis by their effects on neuromuscular junctions. Among the *Crotalidae*, envenomation by *B. lanceolatus* is unique because of the incidence of thrombotic complications, particularly cerebral, in 40% of patients not treated with specific antivenom serum. The fang marks, and pain, edema, and erythema at the site of the bite and adjacent tissues are characteristic of envenomation.

Cerebral vascular impairment after snakebite is rare. In a series of 309 snakebite patients, Mosquera and others reported cerebral vascular complications in eight (2.6%) patients, seven of a hemorrhagic nature and one of an ischemic nature. The prognosis was unfavorable in all eight patients: five died and three had serious neurologic sequelae. Bashir and Jinkins reported a 13-year-old girl bitten on the hand by a viper, with complications of hemiplegia and aphasia, which was consistent with an infarction in the middle cerebral artery. They suggested that the absence of coagulopathy indicates a direct action of venom in the region of vascular endothelium cells. Other reports have been published, but these involved only anecdotal cases. Boviatsis and others reported multiple hemorrhagic cerebral infarctions in a 65-year-old woman bitten by a viper. Numeric and others reported a 32-year-old patient in St. Lucia bitten by *B. caribbaeus*, a species closely related to *Bothrops lanceolatus*, who had several cerebral infarctions, particularly in the anterior cerebral artery. However, reports of cerebral infarction are rare and amount to only a few observations. For the most part, they involve multiple or massive cerebral lesions. There are no published cases limited to ocular symptoms.

Half of the patients envenomed by *B. lanceolatus* have thrombocytopenia and half have disseminated intravascular coagulation. However, hemorrhagic manifestations are ob-

![Figure 1](image1.png)  
**Figure 1.** Visual field test of the patient with Goldmann’s perimeter, showing inferior, left, lateral, homonymous quadransia with macular epargne.

![Figure 2](image2.png)  
**Figure 2.** T2 magnetic resonance image (fluid-attenuated inversion recovery) of the patient, showing a right posterior hypersignal consistent with an occipital infarction.
served only as occasional ecchymoses around the site of the bite. The exact mechanism(s) of these thromboses is unknown. When added to human plasma plus citrate, the venom of *B. lanceolatus* does not have coagulant activity, even at a concentration as high as 100 µg/mL. This suggests that other factors, such as alteration of the vascular wall, which causes adhesion and aggregation of platelets, contribute to the incidence of these thromboses.14

The prognosis for envenomation by *B. lanceolatus* has improved considerably, mainly because of specific antivenom serum. This serum is obtained from horses hyperimmunized with venoms of *B. lanceolatus*. It is composed of 97% antivenom F(ab’)2 (bivalent antigen-binding fragment) and 3% Fab (antigen-binding fragment), and can neutralize more than 25 50% lethal doses per millilitre. Its dosage can be adapted to the severity of the envenomation,6 and its effectiveness is a function of how rapidly it is administered.

Among 33 patients reported by Thomas and others who did not receive antivenom serum or who received it eight hours after being bitten, 14 (42.4%) developed severe thrombotic complications: seven cerebral infarctions, two myocardial infarctions, two myocaridal and cerebral infarctions, and three pulmonary embolisms.9 Of 14 patients who were not treated with antivenom, 4 died. Thrombotic complications occurred an average of 36 hours after being bitten and their proportion increases with the degree of envenomation.9 Thomas and others also reported that in 70 patients who received antivenom treatment within six hours of being bitten, no thrombotic complications were observed.9 Six (8.6%) patients had an immediate, but temporary, undesirable reaction attributable to the antivenom. However, antivenom is not effective in preventing necrosis or secondary infection at the site of the bite.9 In our patient, the incidence of occipital infarction, despite treatment one hour after being bitten, is surprising, and suggests that antivenom may have been less effective in this patient.

Received March 11, 2005. Accepted for publication April 14, 2005.

Authors’ addresses: Harold Merle, Angélique Donnio, and Lucas Ayebooua, Department of Ophthalmology, Pierre Zobda-Quitman Hospital, BP 632, 97261 Fort de France Cedex, Martinique. Telephone: 596-55-22-51, Fax: 596-75-84-47, E-mail: harold.merle@chu-fortdefrance.fr. Yves Plumelle, Hematology Laboratory, University Hospital Center of Fort de France, Fort de France, Martinique. Didier Smadja, Department of Neurology, University Hospital Center of Fort de France, Fort de France, Martinique. Laurent Thomas, Emergency Department, University Hospital Center of Fort de France, Fort de France, Martinique. Reprint requests: Harold Merle, Department of Ophthalmology, Pierre Zobda-Quitman Hospital BP 632, 97261 Fort de France Cedex, Martinique.

REFERENCES


