

## Report of a bite by the South American colubrid snake *Philodryas olfersii latirostris* (Squamata: Colubridae)

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**Abstract.** Following the bite of *Philodryas olfersii latirostris* Cope, 1862, a 29-year-old male herpetologist developed localized and burning pain, and minimal bleeding from the puncture marks of posterior maxillary teeth, which subsided rapidly. The victim developed no other local signs or symptoms. After few days the victim presented persistent severe rotatory dizziness, nausea, and vomiting. On examination his hearing was normal. Neurological exam was otherwise normal. The patient had acute vertiginous symptoms but had no associated neurological signs. Computed tomography did not show abnormality. A diagnosis of labyrinthine syndrome was made. It was treated conservatively, and the patient recovered uneventfully. It was assumed as an effect of ophitoxemia. This case may be regarded as an unusual presentation of systemic envenoming following a human bite by *Philodryas olfersii latirostris*.

**Keywords.** Green snake, *Philodryas olfersii latirostris*, snakebite, Argentina, vertigo, labyrinthine syndrome.

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### INTRODUCTION

Most snakebites in South America are caused by pit vipers (*Bothrops*, *Crotalus*, *Lachesis* spp.) and coral snakes (*Micrurus* spp.); less serious accidents are caused by colubrids (Prado-Franceschi and Hyslop, 2002). However, some snakes of the family Colubridae, usually regarded as not venomous, are responsible by serious and even so fatal accidents (Santos-Costa and Di-Bernardo, 2000). Rear-fanged species (opisthoglyphous) are respon-

sible for most colubrid envenomations; the principal genera involved being *Clelia*, *Helicops*, *Liophis*, *Philodryas*, *Tachymenis*, and *Thamnodynastes* (Prado-Franceschi and Hyslop, 2002), and also by *Phalotris* (Valls-Moraes and Lema, 1997).

*Philodryas* is a genus of rear-fanged colubrid snakes, which is found in South America, from Amazonas to Patagonia (Assakura et al., 1992, 1994). Bites by species of *Philodryas* have been reported from Argentina (Orduna et al., 1994), Brazil (Martins, 1916; Nickerson and Henderson, 1976; Silva and Buononato, 1983-1984; Silveira and Nishioka, 1992; Bucaretschi et al., 1993; Nishioka and Silveira, 1994; Araújo and Dos Santos, 1997; Ribeiro et al., 1999) and Chile (Schenone et al., 1954; Schenone and Reyes, 1965).

*P. olfersii* deserves greatest attention because it has already caused serious accidents, with the patients showing edema, pain, ecchymosis and enlarged axillary lymph nodes (Martins, 1916; Nickerson and Henderson, 1976; Silva and Buononato, 1983-1984; Silveira and Nishioka, 1992; Bucaretschi et al., 1993; Orduna et al., 1994; Araújo and Dos Santos, 1997; Ribeiro et al., 1999). Systemic envenomation is rare, but Ribeiro and coworkers (Ribeiro et al., 1999) reported one case of envenomation by *P. olfersii* in a 2-year-old child which resulted in nausea and vomiting. Furthermore, Orduna and coworkers (Orduna et al., 1994) reported an altered prothrombin clotting time in a patient bitten by *P. olfersii*. Moreover, there is at least one record of a fatal accident caused by *P. olfersii* (Salomão and Di-Bernardo, 1995). However, none of these authors mention the subspecies involved. On the contrary, Kuch (1999) reported a case of a bite by the South American colubrid snake *P. olfersii latirostris*, which was capable of inflicting even systemic human envenomation with only a very quick defensive bite.

We report here the case of a 29-year-old male herpetologist bitten by *P. olfersii latirostris*, a chacoan subspecies, who presented labyrinthine syndrome few days after the bite. This case may be regarded as an unusual presentation of systemic envenoming by this colubrid snake. Although it is difficult to prove that the presentation of labyrinthine syndrome and the snakebite are actually associated, this case would have to be taken into account to alert professionals of the health area about the necessity of attending carefully accidents involving colubrid snakes since very little is known about their venoms and their effects on human victims.

#### CASE REPORT

On 18 November 1997 at 18:30 hr, a 29-year-old male herpetologist was bitten by a male of *P. olfersii latirostris*, with about 80 cm in total length, while capturing the snake in Corrientes city, northeastern Argentina. The specimen bit him on ventral region of the elbow of his right arm. The snake chewed several times, and it could be withdrawn only after about one minute because it got into the clothes of the victim. Both enlarged rear maxillary teeth deeply penetrated the skin.

The victim of the present bite had no known allergies and no history of venomous snakebite or antivenom administration. He had only experienced minor local reactions to previous colubrid snakebites. His last tetanus immunization was one year prior.

Immediately after the bite, there was a burning pain around the bite marks, which subsided by 22:00 hr. Minimal bleeding from the puncture marks of posterior maxillary

teeth was also presented in the beginning, but stopped very soon. The victim developed no other local signs or symptoms.

During the following two days, any sign or symptom of envenomation could be detected. But on 21 November 1997, the victim presented a transient dizziness, which lasted about 5 min.

On 24 November 1997, the victim presented again dizziness, but this time it had a more persistent course. So, the victim went to a general hospital. On arrival to the emergency department, he was complaining of severe rotatory dizziness, unsteadiness, nausea, and vomiting. After waking up that morning, he noted that the room was spinning, and he had to hold on to keep from falling after getting out of bed. He vomited several times. He was unable to go out unaccompanied.

On examination his hearing was normal. Neurological exam was otherwise normal. There was no spontaneous or gaze-evoked nystagmus. His prior medical history was unremarkable for vertigo, systemic illness, trauma, hearing loss, or ear infections. He was not taking any medication. He did not have a recent viral illness (e.g., cold, flu). The patient definitely had acute vertiginous symptoms but had no associated neurological signs. As magnetic resonance imaging (MRI) was not available, computed tomography (CT) was carried out which showed no abnormality (data not shown). Based on the clinical findings and data from CT, a diagnosis of labyrinthine syndrome was made. Thus, dimenhydrinate (50 mg every 6 hr) and deflazacort (6 mg every 12 hr) were prescribed.

The next day he was still complaining of intense rotatory vertigo. However, nausea and vomits had completely disappeared.

At two weeks follow-up he reported improvement in his symptoms, and his recovery was uneventful.

## DISCUSSION

Some local symptoms described by Kuch (1999) for the envenomation caused by *P. olfersii latirostris* agree with the observations made in this case: burning pain and minimal bleeding. Furthermore, both cases agree in its very quick onset of local symptoms, and the fast subsidence of them. However, the present case differs in that edema, enlargement of axillary lymph nodes and ecchymosis were not observed.

There is only one report about the observation of vertigo after a *Philodryas* species bite. In the case of *Philodryas chamissonis* envenomation described by Schenone and coworkers (1954), the patient suffered from vertigo, mild headache, and fever, besides characteristic local symptoms.

Acute onset of vertigo is mostly related to peripheral vestibular disorders (Magnusson and Karlberg, 2002). The distinction between peripheral and central vertigo usually can be made clinically and guides management decisions (Swartz and Longwell, 2005). In the case of the victim of this work, the central causes of acute prolonged vertigo were excluded by the absence of both cerebral and cerebellar signs/symptoms, and the lack of visible lesions in the computed tomography scan. Thus, labyrinthine syndrome was diagnosed. Lesion of the labyrinth is one of the main causes of prolonged rotatory vertigo (Kerr, 2005). It was assumed as an effect of ophitoxemia.

The venom of *P. olfersii* is highly hemorrhagic, has fibrin(ogen)olytic and edema-forming activities; however, it is devoid of coagulant, procoagulant, phospholipase A<sub>2</sub>, and platelet aggregating enzymes (Assakura et al., 1992). It also has a higher proteolytic activity than that of *Bothrops* spp. (Salomão et al., 1990). In the present case, both local as well as systemic damages might have been caused by proteolytic venom components.

*P. olfersii latirostris* is capable of inflicting local and systemic human envenomation (Kuch, 1999), and this case may be regarded as an unusual presentation of systemic envenoming by this colubrid snake.

Although it is difficult to prove that the presentation of labyrinthine syndrome and the snakebite are actually associated, this case would have to be taken into account to alert professionals of the health area about the necessity of attending carefully accidents involving colubrid snakes since very little is known about their venoms and their effects on human victims.

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