

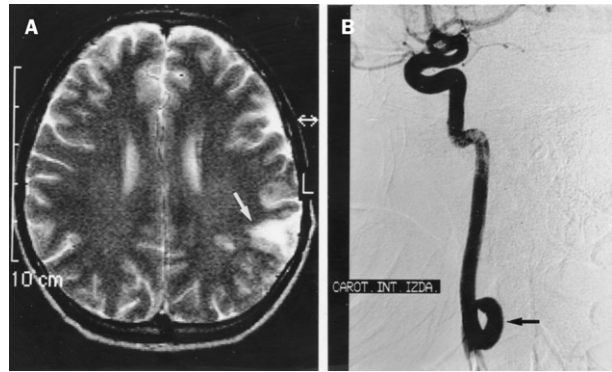
## Case report

## Aphasia in a farmer following viper bite

José M Polo, Amaya Alvarez de Arcaya, Carmen Cid, José Berciano

A 42-year-old right-handed farmer was bitten by a snake on his left thumb at 5 pm on June 29, 1997 while scything. He killed it by crushing its head; its body was recovered shortly afterwards, and easily identified as *Vipera seoanei*. Almost immediately after the bite, he experienced intense local pain and increasing swelling of the thumb and the hand. On admission, 2 h later, two deep fang marks 1 cm apart were seen in the left thumb, with some blister developing; the upper left extremity was markedly swollen and an ecchymotic banner appeared along the inner side of his left arm. The patient looked very anxious, but we found no other abnormalities on general examination. He had no cervical or cardiac bruits. The patient was a non-smoker and had no diabetes, arterial hypertension or particular family history of disease. After topical disinfection of the finger lesions, the patient was transferred to the critical care unit. His blood pressure, pulse rate and temperature remained within normal limits. He was very worried about his painful injury; and a nurse noted in the clinic record that the patient kept his head turned to the left, looking at the arm that he feared would have to be amputated. He had no cervical dystonia or other contractures. 6 h after being bitten, he suddenly developed mild agitation and sensory aphasia. He had no other neurological deficits. We gave him polyvalent antivenom. He had a normal haemoglobin concentration, erythrocyte sedimentation rate, C-reactive protein, full blood count and coagulation screen. His platelet count was  $3.42 \times 10^9/L$ . Cerebral computed tomography on day 2 showed an ill-defined low density left parietal area, consistent with a posterior watershed infarction. Magnetic resonance images done 6 days later confirmed this as an ischaemic area (figure A). An electrocardiogram and two-dimensional echocardiography showed no abnormalities. Arteriography showed elongation of the left internal carotid artery, describing a complete loop (figure B). We treated the patient conservatively and he eventually improved, and was discharged from hospital 10 days later. When last seen in August, 2001, residual expressive aphasia was his only sequela.

Cerebrovascular accidents after viper bites are usually haemorrhagic, and cerebral infarction is a rare complication.<sup>1</sup> In the few published cases, timing of neurological symptoms varies from minutes to hours after the bite. No definite mechanism has



#### Cortical and carotid imaging.

(A) Cerebral T2-weighted magnetic resonance showing left posterior watershed infarction (white arrow). (B) Left internal carotid angiogram showing a complete loop (black arrow).

been established; disseminated intravascular coagulation, arterial hypotension, toxic vasculitis, thrombin-like action from the venom and embolic infarction secondary to toxic cardiopathy have been mentioned as possible causes.<sup>1-3</sup>

Our patient was bitten by a viper and suffered an immediate local and regional reaction, but his general condition was stable and we found no coagulation or cardiac abnormalities. We initially thought that his cerebral infarction was a result of the envenoming, but it seemed unlikely in the absence of other symptoms. When we found the carotid anomaly, we thought again about the contorted posture maintained by the patient, with his head turned to one side. The clinic significance of an elongation of the carotid artery is controversial, particularly in the case of a complete loop.<sup>4</sup> However, unilateral cerebral hypoperfusion has been attributed to the combination of a pre-existing asymptomatic vascular anomaly and head rotation.<sup>5</sup> Our patient may have compromised his left cerebral blood flow simply by anxious fixation on his bitten limb. Reassurance is an essential ingredient in the treatment of snake bite.

We thank Juan Ignacio Pardo de Santayana from the Santillana del Mar Zoo for identifying the viper and John Hawkins for revising the text.

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*Lancet* 2002 **359**: 2164

Service of Neurology, University Hospital Marqués de Valdecilla, 39008 Santander, Spain (J M Polo MD, A Alvarez de Arcaya MD, C Cid MD, J Berciano MD)

Correspondence to: Dr José M Polo (e-mail: polojm@unican.es)