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Exceptional visual impairment complicating viper bite envenomation : Case report

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ABSTRACT

Visual impairment is an exceptional complication of viperin envenomations. It is usually due to a stroke in the occipital lobe, a very rare complication of cerastes cerastes snakes bites. The pathophysiological mechanism is multifactorial. Ophthalmological examination and brain imaging are necessary for the early detection of visual impairment. The treatment is essentially preventive and long-term monitoring of the visual function remains necessary.

Keywords: Envenomation, cerastes cerastes, stroke, visual field.

INTRODUCTION

Snakebite envenomation is a therapeutic emergency. The therapeutic delay engages the vital and functional prognosis.

Visual impairment is exceptional in the course of a viperin envenomation. We report a case of visual deficit complicating a bite of horned viper of cerastes cerastes species.

MATERIALS AND METHODS

Case report:

A 43-year-old patient with no medical history, was admitted to the emergency department of the Laayoune military hospital in March 2014 for the management of a viper bite of the cerastes cerastes species.

On March 31st, at 6 pm, the patient was victim of a viper bite on the left foot, occurred in the Sahara. He was evacuated by ambulance to the Military Hospital of Laayoune.

At admission, on April 1 at 12:30 am, the patient was conscious, well-oriented, agitated, and hemodynamically and respiratorily stable. He showed the marks of the two hooks at the left heel with slight localized edema. He was given probabilistic antibiotics based on amoxicillin-clavulanic acid, titrated morphine analgesia, local bite care, prophylactic-dose calcium heparin and platelet antiaggregant (aspirin 160 mg / d). The patient was injected with anti-tetanus serum; specific anti-venom serum was not available.

The biological balance showed a neutrophilic leucocytosis, a slight thrombocytopenia and a normal hemoglobin level. The patient also had a 3.35 g/l hyperglycaemia and a 11.9 % glycated hemoglobin, which justified a suitable insulotherapy.

In addition, the patient had neither renal insufficiency nor rhabdomyolysis or electromyogram repolarization disorder. The patient was admitted in ICU for clinical and biological monitoring.

On the first of April at 7 pm, the patient noticed a partial loss of the visual field of both eyes, which justified a consultation in ophtalmology.

Ophthalmologic examination of both eyes showed visual acuity at 10/10, positive photomotor reflex, absence of conjunctival haemorrhage, normal intra-ocular pressure and normal anterior and posterior segments.

The Goldmann visual field showed a homonymous lateral quadranopsia evoking retrochiasmatic involvement (Fig 1,2).

Cerebral MRI showed a medial right occipital ischemic stroke and hemorrhagic softening stigma, with no evidence of cerebral thrombophlebitis (Fig 3,4,5).

The patient was kept under aspirin, prophylactic antibiotic therapy for six days, insulin therapy and heparinoprophylaxis with LMWH at a dose of 40 mg / d.

The evolution was marked by the normalization of the biological balance and an improvement of the visual signs.

The six-month screening showed a normalization of the visual field.

RESULT AND DISCUSSION

Discussion:

Vipers are the most commonly encountered snakes in Morocco. The semiology of viper bite envenomation is polymorphic, depending on the species involved and the severity of the envenomation. The haemostasis disorders are observed in case of life-threatening viperin syndrome. The neurovascular involvement is rare, due to several physiopathological mechanisms and can engage by its severity the vital and visual prognosis.

In our case, the visual impairment was due to the stroke of the occipital lobe. Such a complication rarely complicates a snake bite. Indeed, in a series of 309 patients [1], only eight cerebrovascular complications (2.6%) were reported, of which seven haemorrhagic and only one ischemic. This complication was mainly related to vipers and bothrops species, and attributed to various pathophysiological mechanisms: disseminated intravascular coagulation (DIVC), acute hypotension by arterial vasodilation, vasculitis of toxic origin, vasospasm by perivascular bleeding from a parietal lysis caused by the venom hemorrhagin or emboli of cardiac origin [2].

The rare cases of envenomation by cerastes cerastes described, showed its strong capacity to cause marked hypotension, extensive wet necroses, and severe coagulopathies [3,4]. Nevertheless, to our knowledge only two cases of cerebral ischemia have been reported with this species [5,6].

Our patient had thrombocytopenia and hemorrhagic softening stigmata at MRI, which can evoke the mechanism of vasospasm by perivascular bleeding and or vasculitis of toxic origin. On the other hand, our patient neither had obvious signs of DIVC nor hemodynamic instability or myocardial infarction which could be responsible for thrombi formation. These can be explained by the rapid management and the exposure to a small dose of venom.

The treatment of this neurovascular complication is essentially preventive because of its poor prognosis once occurred [7]. It requires the administration of a suitable antivenom (AVS), within the first six hours following the bite when possible, to avoid or alleviate systemic complications [7]. Mosquera et al. Reported five deaths and severe neurological sequelae in three patients despite appropriate treatment [1]

In our patient, we did not administer AVS since we only had FAV-Africa which is not specific to the cerastes genus. It would have been necessary to have the Favirept polyvalent anti-venom. The alternative is the Antirept serum.

CONCLUSION

Although ischemic stroke is an exceptional complication during vipers envenomations, ophthalmologic examination remains essential in order to detect secondary visual damage. Performing cerebral imaging and visual field can help for a more suitable management.

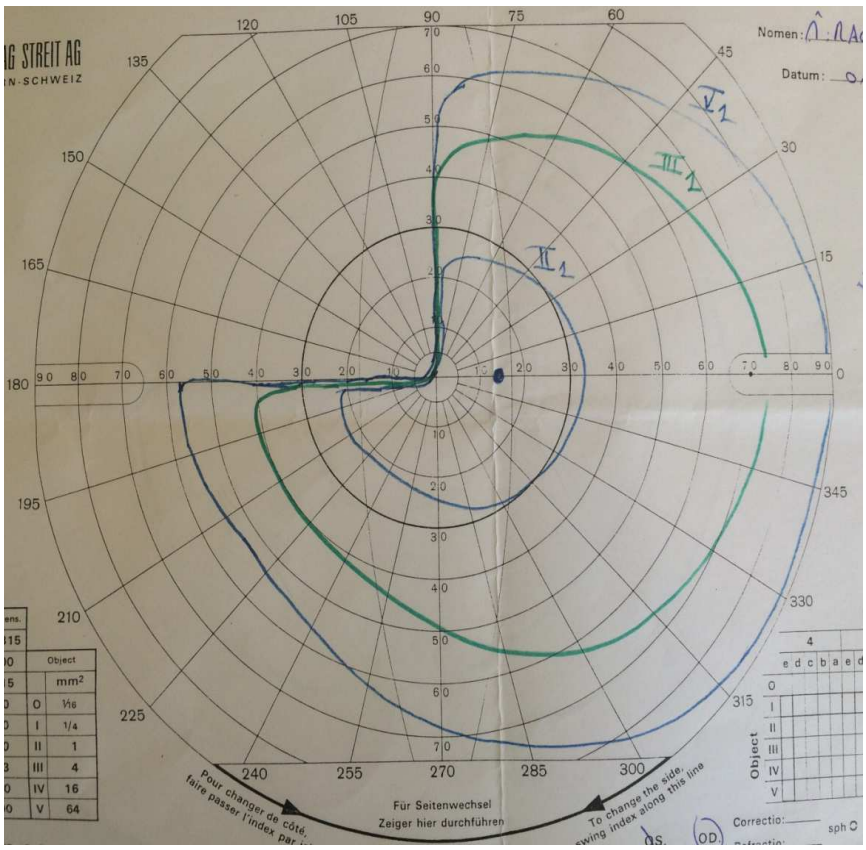

Fig 1: Visual field of the right eye.

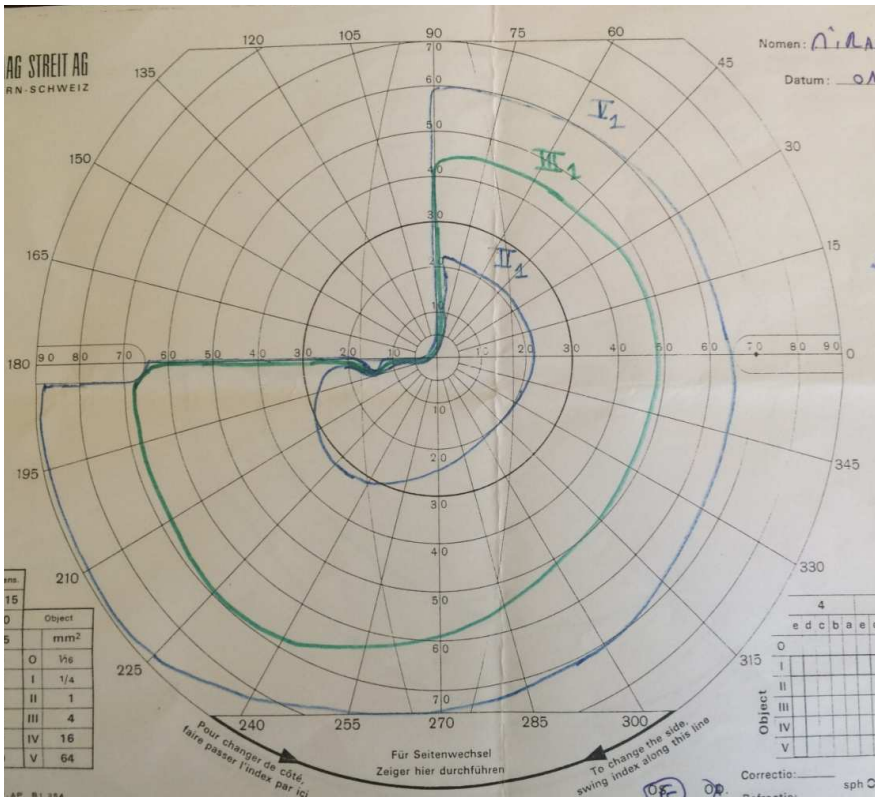

Fig 2: Visual field of the left eye.

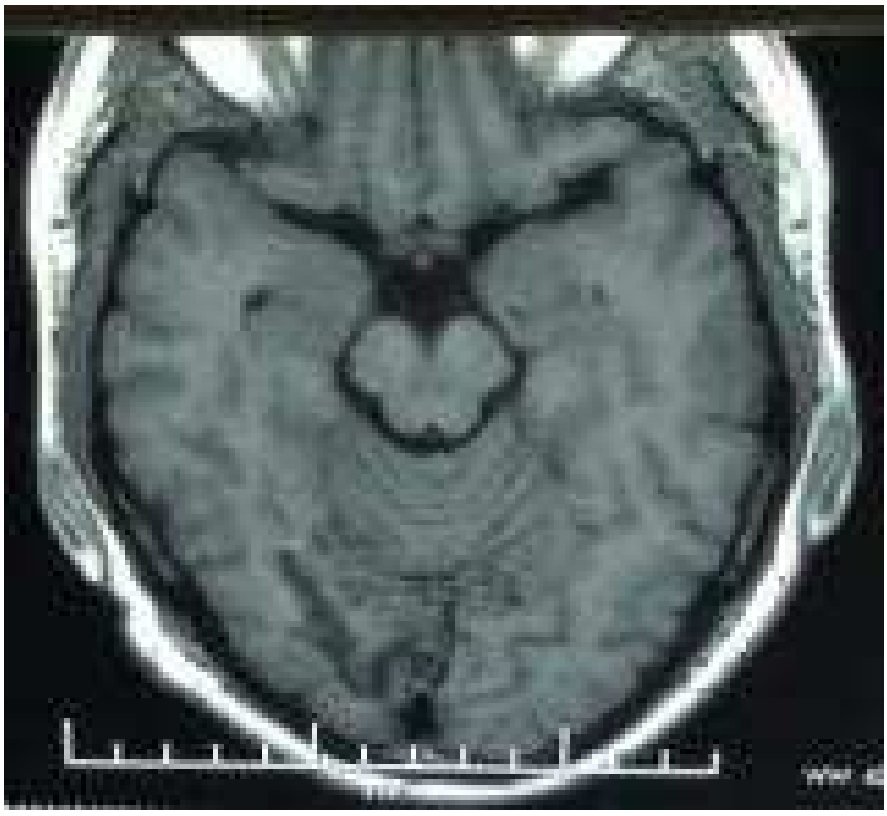

Fig 3 : Axial section T1: Hyposignale poorly limited occipital right.

Fig 4: Axial section in sequence Flair: the hypersignal lesion.

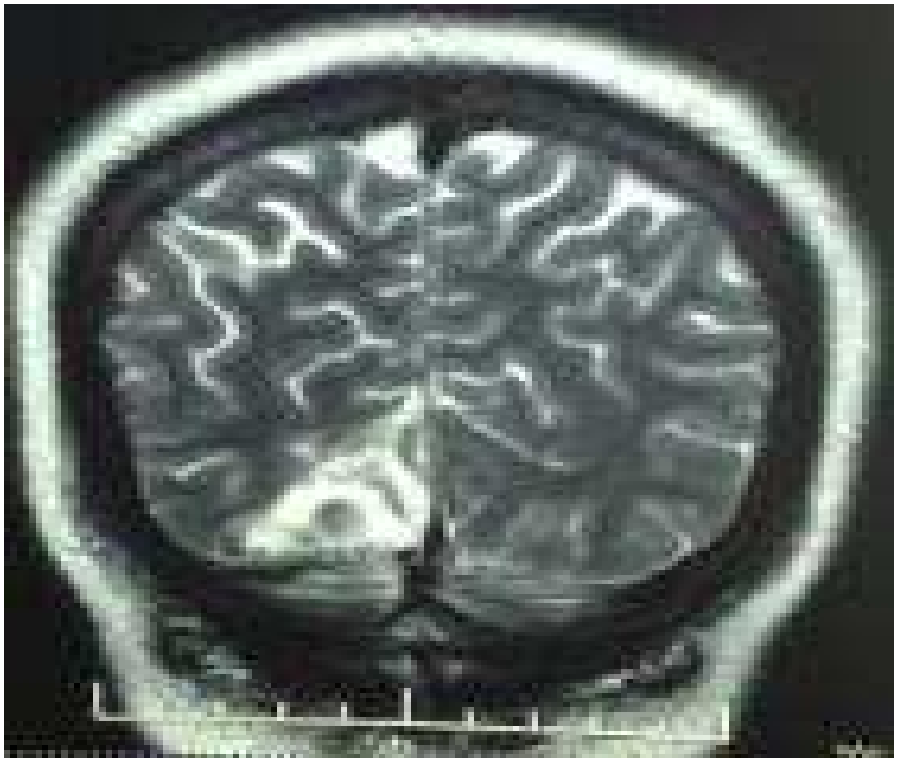

Fig 5 : Sagittal section T2: The hypersignal lesion.

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