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RESEARCH ARTICLE

FATAL BRAINSTEMISCHEMIC STROKE FOLLOWING A CERASTES CERASTESVIPER BITE: ABOUT A CASE REPORT

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Abstract

We report a case of 25 year-old men bitten on herleft foot by *Cerastescerastes* snake in the MoroccanDesert. This case evolved3 daysafter bite withbrainstemischemicstroke resulting in deathdespiteapplication of polyvalent antivenomtherapy.

Introduction:-

Ischemic strokefollowingsnake biteenvenomationsisveryuncommon and canbe life threatening. Reportedcases of this complication are extremely few [1]. Cerastescerastesis a venomousspecies of viper native to the deserts of northernAfrica and parts of the Middle East [2].Herewedescribe a case of brainstemischemic strokeoccuringin a 25 year-old men threedaysafterenvenomation by Cerastescerastessnake in the Moroccandesertwhodiedafterwardsfollowingthis complication.

Case presentation:

A 25-year-old soldierwithoutanyparticularpathologicantecedentswasadmitted toourhospital four hoursafterbeingbitten by aCerastescerastessnake type on hisleft foot.The snakewasidentified as a Cerastescerastesviper by its distinctive supraorbital horns. Beforearrivingat the hospital, patient immediatelyreceived first aidfrom the unit doctor by receiving a first dose of polyvalent antiveninwith local care and paracetamolanalgesia. On admissionathospital, hewasfullyconscious and her vital signsshowed a blood pressure and heart rate of 135/70 mmHg and 90 beats per minute, respectively. No neurologicalsignwasevidentduringexamination. On local examination, therewere fang marksclearly visible, milderythema and swellingat the site of bite [Figure 1]. He had a hemoglobinlevel of 13,7 g/L, 7900cells/mm3 of leukocytosis, and 91,000 platelets/mm3 wereobserved. Shealsoshowedsigns of disseminatedintravascular coagulation (prothrombin 27%, activatedcephalin time 46 seconds).Liverfunction and renalfunctionwere normal. He wasimmediatelytreatedwitha second dose of equine polyvalent antisnakevenom[(Inoserp serum (BIOPHARMA)], followed by a third dose after 6 h. On the 3^{d} day, the patient developedleft-sidedhemiplegia, aphasia, visualdisturbances (diplopia) and her GCS furtherreduced to 8, at which time hewaselectivelyintubated and ventilated.Computedtomography (CT) brainshowed lacunar hypodense lesions of the two thalamic nuclei more marked on the left[Figure 2]. Magneticresonanceimaging of brainshowedischemia in the brainstem and thalamic nuclei[Figure 3].Doppler studysuggested no evidence of arterial or venousthrombosis. Furthermore,

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echocardiographic and electrocardiographicfindingswereunremarkable.The evolutionwasmarked by the death of the patient 4 dayslaterfollowingthis complication.

Discussion:-

Stroke secondary to snakeenvenomationis a rare but serious complication. As reported, the mostcommonspecieswereRussell'sviperswithhigher incidence of ischemic stroke thanintracranialhemorrhage (ICH) [3].Nevertheless, it should be noted that this dogma of thrombotic accidents occurring only on this type of viper has been broken up because although rare cases of ischemic stroke have been reported in vipers of the genus Cerastescerastes [4]. Our clinical case illustrates this condition very well.

The viper Cerastescerastes is a small poisonous snake (size 30 to 60 cm) whichis found in the deserts of North Africa and the Middle East. The twoscales, or "horns", erect above its eyes have earned it this nickname [5].

The mechanism pathophysiological is not clearly established, but appears to be multifactorial [6]. Snakeenvenomationproduces diverse clinical syndromes due to the in vivo effects of multiple toxic componentspresent in snakevenom. Most important amongvipersnaketoxinsfrom a clinical perspective includehemorrhagins, coagulant toxins, nephrotoxins, myotoxins, and necrotoxins[5, 6].

Mortalitywas more commonamongthosewhoeitherarrived in coma or required intubation due to AMS during the course of hospitalization. Deathhappenedwithin the first 4.2 daysafter the exposure. Risk of mortalitywasamplified by ICH, bilateral extensive cerebral, cerebellarinfarction, mass effect, or post circulation occlusion [7].

Furthermore, someauthorsreportedthat no thrombotic complications wereobserved in patients whoreceivedantivenomtreatmentwithin 6 hours of beingbitten.In thisstudy by Thomas *et al.*, of the 33 patients withenvenomation by *Bothropslanceolatus* whohad not received ASV or received ASV after 8 hours of envenomation, 14% developedthrombotic complications and 4 of the 14 patients whohad not received ASV died[8]. Of the 70 patients whoreceived ASV within 6 hours of envenomation, none thrombotic complications, which not often the case as in our patient, despitetreatmentwith ASV within 1 hour of envenomationand whoreceived a total of 3 doses, developeddelayedcerebralinfarction on the thirdday.

Figure 1:- Fang marks clearly visible (twoarrows in green), milderythema and swelling at the site of snake bite.

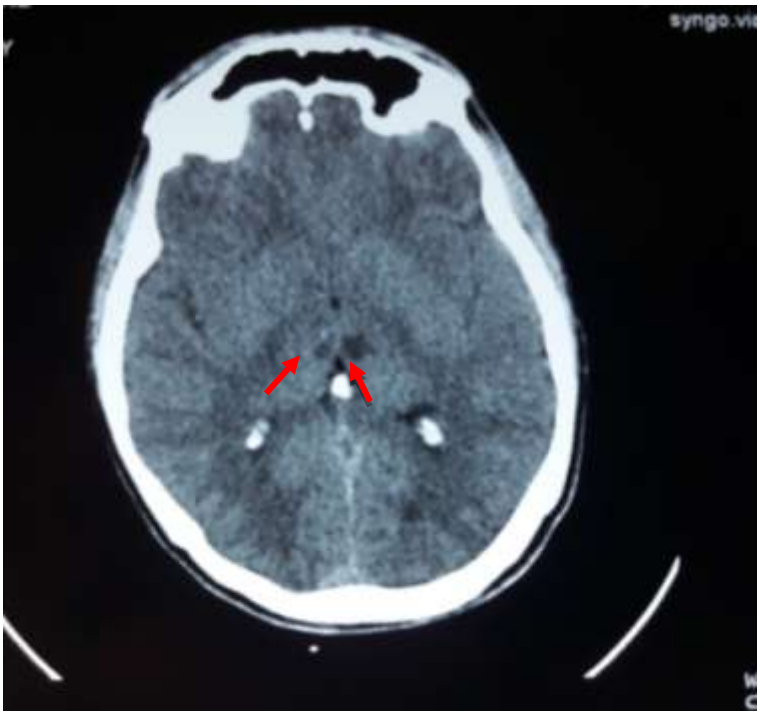

Figure 2:- Brain CT in axial section without injection of contrast product showing lacunar hypodense lesions of the two thalamic nuclei more marked on the left (twoarrows in red).

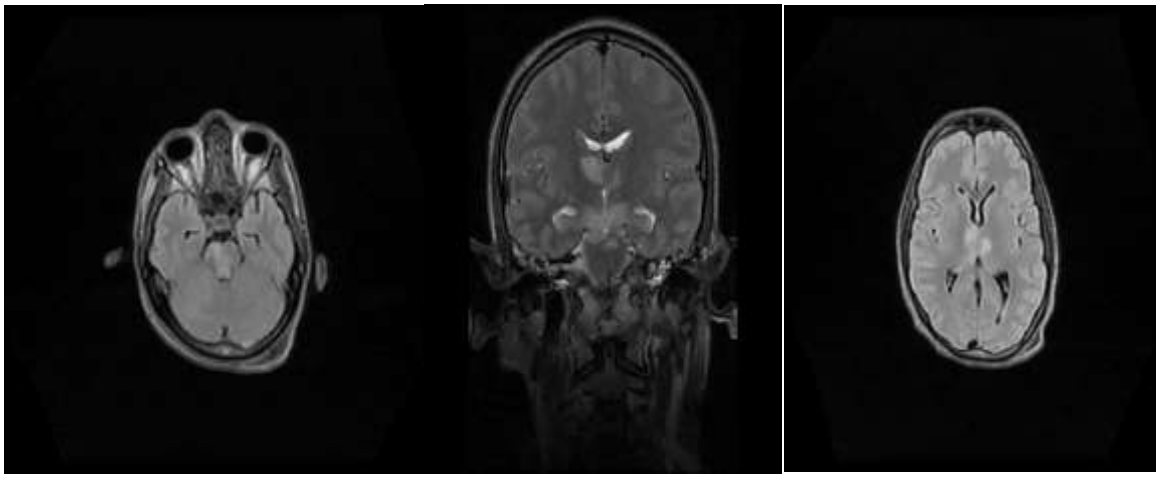

Figure 3:- Magneticresonanceimaging of brainin axial and coronal FLAIR sections showing hypersignal of the thalamic nuclei and brainstem (midbrain).

Conclusion:-

Ischemic stroke is a complication exceptional viperin envenomation, physiopathological mechanism probably multifactorial. This case highlights also that such as other vipers, Cerastescerastes can also cause a cerebrovascular thrombotic accident with a fatal outcome.

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