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IMAGES IN CLINICAL NEUROLOGY

Intracranial subarachnoid hemorrhage in snakebite

Dillibabu Ethiraj, Saggana Sree Victory Senthilkumar, Neha Bagri

Department of Radio-Diagnosis,Vardhman Mahavir Medical College and Safdarjung Hospital, New Delhi, India

A 28-year-old male farmer was admitted to the hospital after being bitten by a viper snake while working in his fields. The patient had been initially taken to a nearby primary health care center in an unconscious state where a snakebite mark was noticed on the right leg and was transfused eight vials of polyvalent antiserum venom before being transferred to our hospital. On laboratory investigation with the 20-min whole blood clotting time (20WBCT) test, the patient's blood was found to be incoagulable even after 20 min. On examination, the vitals were as follows: heart rate, 120 beats per min; blood pressure, 150/80 mmHg; temperature, 37.2°C. On local examination, the bite mark was oozing out blood with surrounding tissues, showing local inflammatory signs such as edematous swelling and redness. The patient remained unconscious and hence a plain computed tomography of the brain was taken. Computed tomography showed subarachnoid hemorrhage involving basal cisterns, suprasellar cisterns, bilateral sylvian fissures, and sulcal spaces of bilateral frontoparietal regions (Figures 1a-d), and a subsequent brain computed tomography angiography was unremarkable. The patient was admitted to the intensive care unit and was started on blood components and symptomatic management. The patient was intubated, and the vitals were monitored. Eventually, the patient regained consciousness and was discharged without any neurological sequelae.

Snakebite is a common occupational and health hazard across the globe. Snakes are classified into four types: common krait, Russell's viper, Indian cobra, and saw-scaled viper. These four snakes are termed as the "big four." Of these, the Viperidae (vipers) are vascularly toxic and the Elapidae (cobra and krait) are neurotoxic. The Viperidae group involves local symptoms and systemic manifestations due to vasculotoxic and neurotoxic components in the snake.[1] The mechanism of snake envenomation is due to the variety of components in the snake venom and its interaction with multiple organ systems in the human body. A coagulation abnormality caused by snake venom, called venom-induced consumptive coagulopathy is the most common presentation. Different enzymes such as proteases, phospholipases A2, hyaluronidase, and arginine ester hydrolase present in vipers' venom are responsible for venom-induced consumptive coagulopathy. The hyaluronidase break downs tissue mucopolysaccharides, while phospholipase A2 results in hemolysis, a consequence of red blood cell membrane disruption, also causing myonecrosis.[2]

The coagulation cascade is activated by the viper's venom, leading to feeble fibrin clot formation, which in turn activates the fibrinolytic pathway by plasmin activation. This continuous cycle of fibrin formation and lysis leads to excessive coagulation factor consumption and internal systemic bleeding manifestations.[2] Cerebral manifestations of a viper bite include stroke, acute demyelinating encephalomyelitis, and acute hemorrhagic leukoencephalitis. A stroke can be either ischemic or hemorrhagic. Ischemic stroke is the most common central nervous system presentation due to inappropriate thrombocyte activation and disturbed blood supply to brain tissue.

Correspondence: Neha Bagri, MBBS, MD. Department of Radio-Diagnosis,Vardhman Mahavir Medical College and Safdarjung Hospital, 110029 New Delhi, India
E-mail: drnehabagri@gmail.com
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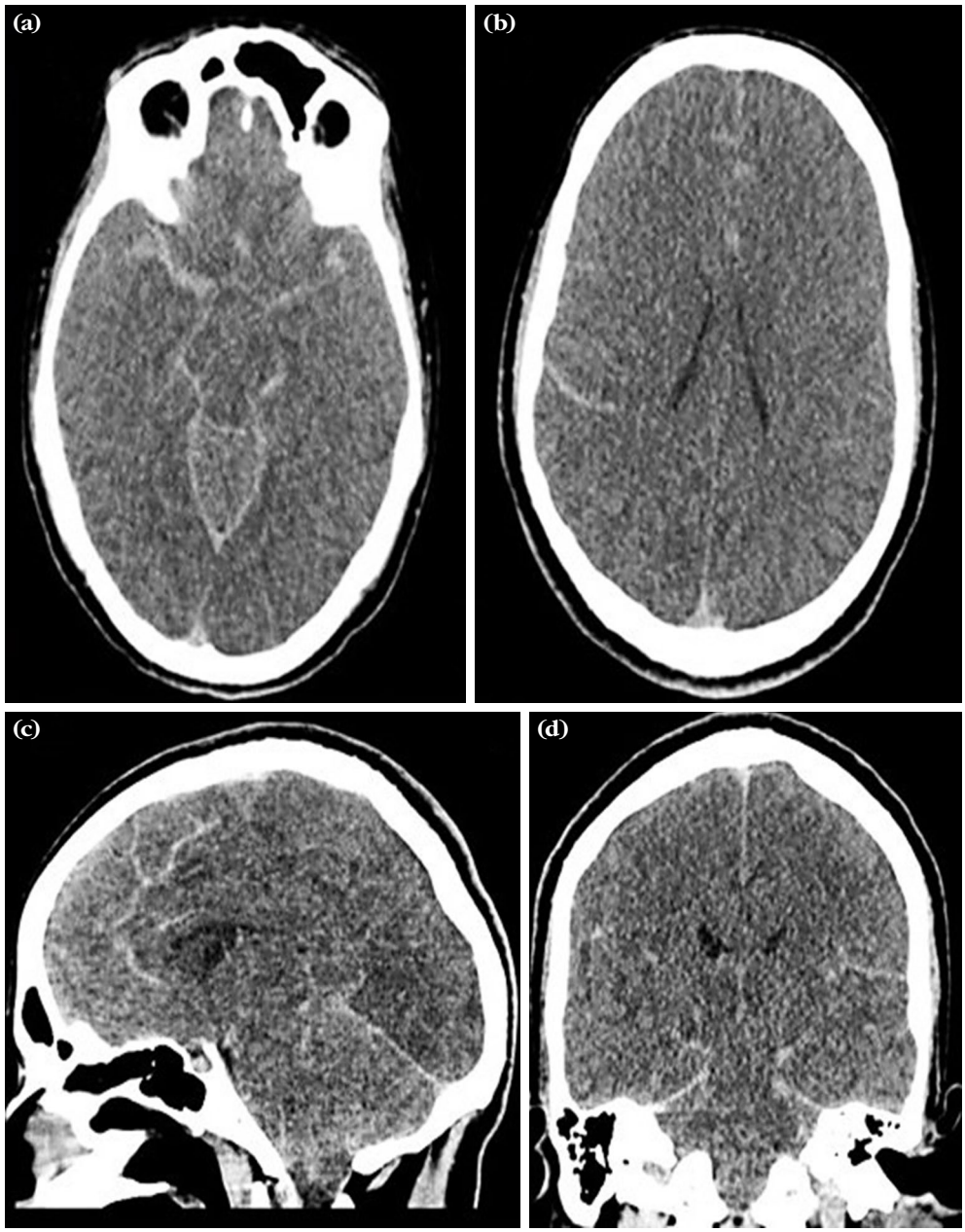

Figure 1. Plain computed tomography of the brain; **(a, b)** axial, **(c)** sagittal, and **(d)** coronal sections show diffuse subarachnoid hemorrhage in basal cisterns and sulcal spaces of bilateral frontal regions.

Hemorrhagic stroke, on the other hand, is triggered by hemorrhagins, a toxic viper venom component. Hemorrhagins damage the endothelium and increase blood vessel permeability, leading to spontaneous intracerebral bleeding.[3] As in our case, a derailed coagulation profile with elevated D-dimer levels suggested that the subarachnoid hemorrhage was spontaneous due to venom-induced consumptive coagulopathy. Subarachnoid hemorrhage is a rare cerebral manifestation of a snake bite.[2] Early diagnosis and appropriate management of such cases are essential to improving morbidity and mortality.

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