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Case Report

Adrenal Hematoma and Right Hemothorax after *Echis Carinatus* Bite: An Unusual Manifestation

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ABSTRACT

Common bleeding manifestations after viperine bite include bleeding from site of bite, bleeding gums, epistaxis, hemoptysis, hematuria, hematemesis, and intracranial bleed. Bleeding in the adrenal gland is a rare manifestation. We report here a patient of viperine bite who developed right adrenal hematoma and right hemothorax after 3 days of bite. To the best of our knowledge this is the first case report of adrenal hematoma and right hemothorax after *Echis carinatus* bite.

Key words: Adrenal hematoma, *echis carinatus,* hemothorax, viperine bite

INTRODUCTION

Saw scale viper or *Echis carinatus* is primarily a vasculotoxic viper. The snake venom is responsible for the local and systemic manifestations and complications of the snake bite of which bleeding is a major one; which occurs because of venom-induced coagulopathy. The local manifestations includes bleeding from bite site, petechiae, edema, hematoma, purpurae, and gangrenous lesion; whereas systemic are disseminated intravascular coagulation (DIC), intracranial hemorrhage, hemolysis, acute respiratory distress syndrome (ARDS), and acute kidney injury (AKI).[1] Here we report a case who developed adrenal hematoma and right hemothorax after snake bite.

CASE REPORT

A 48-year-old woman presented within 3h after having been bitten on the lateral border of right foot by an Echis carinatus. At the local hospital, management consisted of antibiotics, antitetanus prophylaxis, analgesics, and 10 vials of polyvalent antisnake venom (ASV). ASV was repeated on the next 2 days. On the 3rd day, patient felt dull aching pain in the right flank, hence she was referred to us. She also had breathlessness and heaviness of right chest. She had no neurological deficit. She denied for history of trauma, hematuria, hemoptysis, hematemesis, and bleeding from any other site. Patient was not suffering from any chronic illness and had no addiction. On examination she had swelling of the right foot not extending beyond ankle joint. The blood pressure was 110/70 mmHg, pulse rate was 78 beats/min, and respiratory rate was 20 breaths/min. There was decreased air entry with dull note on percussion on lower zone of right lung. Cardiac auscultation was normal. The whole blood clotting time (WBCT) was > 20 min. CBC revealed hemoglobin (Hb) of 6.4 g/dL, total leukocyte count of 12,000/mm^3, with normal differential count. Platelets were 214,000/mm^3. Renal and liver functions were normal. Serum sodium was 138 meq/L and potassium 3.5 meq/L. Urinalysis was normal. Coagulation profile showed bleeding time (BT) 3.5 min (normal up to 5 min), clotting time (CT) 12.25 min (normal up to 8 min), prothrombin time (PT) 20 s (control 13 s) with international normalized ratio (INR) of 1.6, thromboplastin time with kaolin (PTTK) 40 s (control 35 s), and fibrinogen degradation product (FDP) was 1.3 μg/mL (0–1). Chest X-ray posteroanterior (PA) view showed moderate amount

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of right pleural effusion [Figure 1]. For the evaluation of right flank pain ultrasonography was performed which showed hematoma of right adrenal gland. Echocardiography was normal. One liter of pleural fluid was drained which was hemorrhagic. Contrast-enhanced computed tomographywhole abdomen was done on 6th day of being bitten which showed lobulated altered signal intensity in the right adrenal gland measuring 90 × 53 × 31 mm, suggestive of adrenal hematoma [Figures 2 and 3]. Computed tomography also showed moderate amount of right pleural effusion [Figure 1]. Two unit of packed cell and six bags of fresh frozen plasma (FFP) were transfused. The pain in the right flank started subsiding and by the end of 2nd week the patient was asymptomatic and had normal hematological parameters.

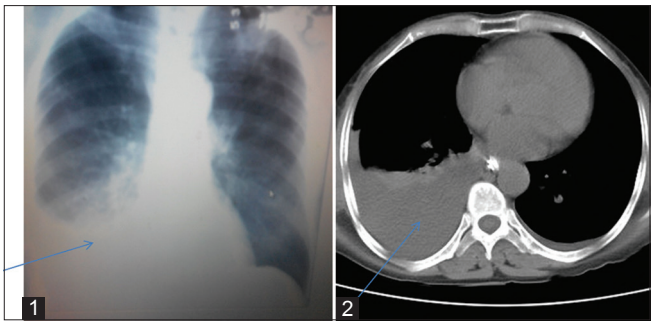


Figure 1: (1) Chest X-ray and (2) computed tomography (CT) showing right pleural effusion

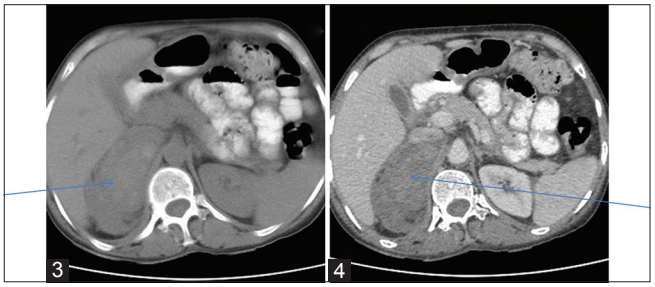


Figure 2: (3) Transverse section plain CT-enlarged right adrenal gland with ill-defined hyperintensities seen within the right adrenal gland. (4) Axial contrast-enhanced (CECT)-heterogeneous enhancement of right adrenal gland

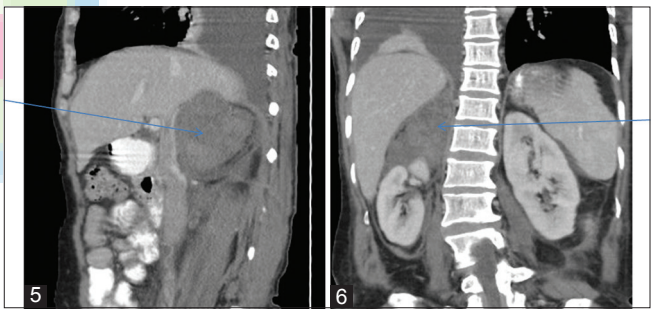


Figure 3: (5) Sagittal section-CECTshowing enlarged right adrenal gland with heterogamous enhancement and right pleural effusion. (6) Coronal section CECT-enlarged right adrenal gland with heterogeneous enhancement and minimal fluid collection in right perirenal space and pleural space

DISCUSSION

Snake bite is a major devastating environmental and occupational disease, especially in rural areas of tropical developing countries. The major cause of mortality and morbidity in snake bite is due to coagulopathy.[2] Snake venom is complex mixture of biologically active proteins and peptides. Many of them affect hemostasis by activating or inhibiting coagulant factors or platelets, or by disrupting endothelium. Some proteins in venom are serine proteases and other procoagulant enzymes that are factor V activators, factor X activators, prothrombin activators, and thrombin-like enzymes (TLEs) which stimulate blood clotting. Whereas anticoagulant factors including factor IX/X binding proteins, protein C activators, thrombin inhibitors, phospholipase A2, and those acting on fibrinolysis, including fibrinolytic enzymes and plasminogen activator in venom predispose to bleeding. The mechanism for platelets dysfunction by venom protein is binding or degradation of vWF or platelets receptor or modulation of ADP release or thromboxane A2 formation. Vascular endothelium is disrupted by the hemorrhagins which are zinc containing metal loproteases.[3]

The common bleeding sites after a viperine bite are skin, gums, nose, gastrointestinal tract, and urinary tract. Bleeding from the site of bite is also frequent. Amongst the closed spaces, bleeding in retroperitoneal space and intracranial bleeding are the most frequent.[4] One case report showed development of hemothorax after snake bite.[5] The exact mechanism for development of adrenal hematoma and hemothorax in our case is not known. Our patient had prolonged WBCT, and marginal increase in BT, CT, PT/INR, activated PTT (aPTT), and fibrin degradation product (FDP), suggests hemostatic dysfunction. We believe that venom-induced coagulopathy along with hemorrhagins-induced direct endothelial injury may be the possible mechanism of hematoma. Thus adrenal hematoma and hemothoraxshould be considered in the list of unusual site of hematoma following *Echis carinatus* bite.

CONCLUSION

Finally we conclude that venom-induced coagulopathy is common after *Echis carinatus* bite. Hematoma in adrenal gland with right hemothorax is a very rare and unusual presentation. Installation of ASV and supportive measures with proper monitoring of coagulation profile can prevent the coagulopathy-induced mortality and morbidity.

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