

Hemothorax Following Snakebite

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We report a 12 year old girl with snakebite, who developed hemothorax 5 days after admission. One liter of blood was aspirated. The bite was presumed to be that of saw scaled viper (*Echis carinatus*) that resulted in DIC and direct endothelial injury leading to bleed. Selective bleed into the pleural cavity is a rarity.

Key words: Saw scaled viper, Pleural effusion, Hemothorax.

Saw scaled viper (*Echis carinatus*) is considered to be the world's most dangerous snake because of its highly virulent hemotoxic venom [1]. In Jammu, saw scaled vipers account for about 95% of the total snake bites [2]. Envenomation results in local symptoms as well as severe systemic manifestations that may prove fatal. Hematemesis, malena, hemoptysis, hematuria, acute renal failure, epistaxis and hypovolemic shock have been reported as complications [2, 3].

CASE REPORT

We report a 12-years old female child referred from a rural health centre to our hospital for the management of snake bite on her right foot. She received first aid and 100 units of ASV (anti snake venom) at the referring hospital. At the time of admission, bite marks were found over the right foot with swelling, bluish discoloration, tenderness over the surrounding area and slight oozing of the blood from the bite site. On examination, she was well oriented. Her pulse was 76/minute and blood pressure 100/70 mm Hg, with no symptoms or signs of bleeding from any other site. There were no signs of neurotoxicity. Despite repeated doses of ASV and supportive management, her 20 WBCT (20 minutes whole blood clotting time) remained prolonged.

On 5th day of admission, she developed pallor, tachycardia and hypotension. On general physical examination, there was decreased air entry on right side of the chest with dull note on percussion. Chest X-ray and ultrasonography of the chest revealed fluid in the pleural cavity. The laboratory findings showed anemia (hemoglobin 4.5 g/dL), leukocytosis (16500/mm³), and low platelet count (90,000/mm³). The patient's prothrombin time (15 seconds) and activated partial thromboplastin time (45 seconds) were prolonged. In addition, the fibrin degradation product levels were raised and D-dimers were positive.

On thoracostomy, about 1000 mL of blood was

aspirated and intercostal tube was kept for drainage of any further collection. During her stay in the hospital she received 800 units of ASV, 8 units of blood transfusion, and 7 units of FFP (fresh frozen plasma) transfusion besides antibiotics. The general condition of the patient improved gradually and was discharged after 25 days of stay in the hospital. Her coagulogram was within normal limits at the time of discharge.

DISCUSSION

Snake bite is an environmental hazard, particularly in rural areas, causing significant morbidity and mortality. The vipers (saw scaled viper and russell's viper) are primarily vasculotoxic. The toxins affecting haemostasis have been classified as per their overall effect and include the following: (a) procoagulants due to prothrombin activating toxins and thrombin like enzymes; (b) anticoagulants acting by activating Protein C etc.; (c) platelet activating and anti-platelet function; (d) fibrinolytic activators; and (e) hemorrhagins [5].

Several reports showing bleeding manifestations in the various parts of the body following snake bite have been reported in the past. One report showed hemoperitoneum developing after a Russell's viper bite [5]. Intracranial bleeding and ischemic stroke following snake bites have also been reported [6]. Another report showed ischemic colitis after a viperine snake bite, which resulted in an emergency laparotomy revealing a necrotic ileum and caecum with the occlusion of the superior mesenteric artery [7].

As in the present case, hemothorax developing after saw scaled viper has not been reported in the literature to the best of our knowledge. In this patient, the levels of FDP (fibrin degradation product) and D-dimer suggested that DIC had occurred. The exact pathophysiology for the development of hemothorax after snake bite is not known. We believe that DIC along with direct endothelial injury as a result of a component in the venom itself such

as hemorrhagin is the possible mechanism of hemothorax in this patient. DIC causes fibrin deposits in the micro-circulation, platelets and coagulation factors consumption with secondary fibrinolysis leading to bleeding.

Systemic envenomation by snakes can affect various organs of the body due to disturbances in the coagulation pathways. Hemothorax developing as a complication of snake bite has not been reported in the past and should be considered as a possible complication following snake bite.

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Lane-Hamilton Syndrome: Association or Coincidence?

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The combination of idiopathic pulmonary hemosiderosis (IPH) and celiac disease (CD) is rare. The clinical importance of this association is that a significant improvement can be obtained with gluten free diet not only in intestinal but also in pulmonary symptoms. A four and half-years old girl was admitted with complaints of cough, difficulty in breathing and paleness. She had intermittent episodes of abdominal pain and diarrhea. She had dyspnea and tachycardia, and oxygen saturation 88%. The patient was diagnosed with CD and concomitant IPH. With gluten-free diet and corticosteroid treatment, both intestinal and pulmonary symptoms were controlled.

Key words: *Celiac disease, Gluten-free diet, Pulmonary hemosiderosis.*

Idiopathic pulmonary hemosiderosis is a rare disease characterized by recurrent episodes of hemoptysis, pulmonary infiltration and iron deficiency anemia [1-4]. Celiac disease is an enteropathy characterized by life-long intolerance to ingested gluten in genetically susceptible people [5]. The simultaneous occurrence of these conditions is rare and is called Lane-Hamilton syndrome [6]. Significant improvement can be obtained with gluten-free diet, not only in intestinal but also in pulmonary symptoms [1,4,7].

CASE REPORT

This four and half years old girl was admitted with complaints of cough, difficulty in breathing and pallor. She had intermittent episodes of abdominal pain and

diarrhea. The body weight and height were 16 kg and 105 cm (both at 3rd percentile). The heart rate, respiratory rate, blood pressure and oxygen saturation were 168 per min, 54 per min, 80/40 mm Hg and 88% respectively. The patient showed severe pallor. Fine crepitations were heard and liver was palpable 2 cm below the right subcostal margin. Investigations showed hemoglobin level of 2.5 g/dL, leukocytes 8300/cu mm and platelets 296000/cu mm. The mean corpuscular volume was 59 fl, reticulocyte count 10%, plasma iron 13 µg/dL, iron binding capacity 296 µg/dL and ferritine 74 µg/L. The peripheral smear examination showed hypochromic microcytic anemia. The chest radiograph showed bilateral infiltrates; echocardiography was normal. The findings improved after blood transfusion and the