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Unilateral massive hemothorax in Dengue hemorrhagic fever: A unique presentation

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ABSTRACT

Dengue hemorrhagic fever is a more serious form of disease characterised by plasma leakage syndrome, thrombocytopenia and disseminated intravascular coagulation. We present a 51 year old male who presented with fever, petechiae and acute onset of breathlessness. Emergency chest rhoentogram showed a massive right sided pleural effusion. On insertion of intercostal drain, there was a sudden gush of blood tinged fluid suggestive of hemothorax. There was no history of trauma or bleeding tendencies. Laboratory investigations revealed a raised hematocrit and severe thrombocytopenia. Dengue IgM was surprisingly positive. After aggressive supportive management the patient gradually improved and was discharged. While bilateral pleural effusion is a known occurrence in dengue hemorrhagic fever, massive hemothorax is unheard of. We report the first case in literature of dengue hemorrhagic fever presenting as unilateral massive hemothorax. A suspicion of dengue must also be borne in mind in cases of non- traumatic hemothorax especially in endemic areas.

1. Introduction

Dengue fever can vary from an asymptomatic or self-limiting illness to a life threatening disease characterised by haemorrhage and shock known as Dengue hemorrhagic fever (DHF) and Dengue shock syndrome (DSS) respectively[1]. DHF is manifested by high grade fever associated with bleeding diathesis, circulatory disturbance and in severe cases, shock[2]. We present a rare case of massive hemorrhagic pleural effusion requiring urgent intercostal drain (ICD) drainage. To our best knowledge, this is the first case of dengue presenting as unilateral massive hemothorax.

2. Case report

A 51 yr old gentleman was admitted to the emergency department with a two day history of fever and breathlessness. At presentation, he was febrile with diffuse erythematous rash, petechiae and bilateral pitting pedal edema. Tachycardia and tachypnoea were present with no hypotension. Respiratory examination revealed dull note on percussion and decreased breath sounds in the right lower interscapular, infrascapular and infraaxillary areas. There was abdominal distension with shifting dullness. Other systems were unremarkable.

Blood investigations were suggestive of hemoconcentration (haemoglobin: 19 g/dL, hematocrit: 57.7%) and severe thrombocytopenia (platelets: 9×10^9/L) and pre-renal failure (urea: 51 mg/dL; creatinine: 1.7 mg/dL). Albumin levels were low (2.5 g/dL). Transaminases were grossly deranged with SGOT of 3 526 U/L and SGPT of 1 771 U/L. Workup for disseminated intravascular coagulation revealed a prolonged prothrombin time (24 s, control: 15.4 s), activated thromboplastin time (81.5 s, control: 34 s), INR of 2 and positive D-Dimer assay. Amylase and lipase were mildly elevated. Chest radiograph showed evidence of right sided massive pleural effusion (> 4 intercostal spaces were involved) with USG thorax demonstrating an additional underlying right lower lobe collapse. Hypoxia was evident on arterial blood gas analysis with oxygen saturation of 85% with arterial partial pressure of oxygen being 56 mmHg). There was no metabolic acidosis. We ruled out a possible pulmonary embolism by a negative Doppler venogram of the lower limbs and computed tomography pulmonary angiogram. Echocardiogram was normal. An emergency thoracocenthesis was done after correction of the platelet count to permissible levels. Blood tinged serous pleural fluid was aspirated which on analysis showed increased RBC (16.5×10^3/L) and WBC (0.15×10^9/L) of which 75% were lymphocytes. The ratio of pleural to serum hematocrit was more than 0.5, suggestive of hemothorax. Pleural fluid and blood cultures were sterile. An ICD was inserted in view of severe breathlessness, evidence of hypoxia and

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presence of massive hemothorax. On insertion, there was an immediate gush of 1.1 L of blood tinged serous fluid from the pleural cavity. We voluntarily clamped the ICD within five minutes in fear of precipitating hemodynamic compromise. Meanwhile, serology for dengue IgM by E/M specific Capture ELISA was positive. Serotyping of virus could not be performed. USG abdomen showed hepatomegaly with moderate ascites. He was continued on aggressive fluid replacement therapy with transfusion of platelet concentrates.

The next day, ICD drain reduced to 600 mL with clearing of the blood tinged fluid gradually. There was a gradual drop of haemoglobin (19 g/dL at admission versus 11.2 g/dL on day 4). There was no evidence of gastro-intestinal bleed. Packed red cell transfusions were given at this stage. Aggressive supportive care was continued. From then on, serial chest radiographs confirmed re-expansion of the previously collapsed right lung. ICD drain gradually reduced to nil over the next 4 days and was removed. There was improvement in all of the previously deranged laboratory parameters. There was complete resolution after 14 days of hospital stay. At two subsequent follow ups chest radiographs were normal with persistence of Dengue IgG antibodies.

3. Discussion

Dengue fever is a transmitted by the bite of *Aedes aegypti* mosquito and by *Aedes albopictus* which are efficient epidemic vectors. Dengue virus belongs to the family Flaviviridae which has four serotypes 1 to 4[1]. There has been a dramatic increase in prevalence of dengue globally over the recent years with over 100 million people affected annually[3]. Some of the postulated reasons are increased international travel, rapid urbanisation and failed measures to control the vector population[4-6]. Dengue fever typically presents with fever, arthralgia, myalgia and retro-orbital pain[7]. DHF is a more severe form of dengue. WHO defines DHF by the presence of continuous high fever, marked thrombocytopenia (<100 000/μL), a plasma leakage syndrome, hemorrhagic manifestations with increased hematocrit>20% above baseline[2]. Plasma leakage syndrome is life threatening in DHF which can present as ascites, pleural effusion and hypoproteinemia. The respiratory manifestations such as pneumonitis, acute respiratory distress syndrome and pulmonary hemorrhage are rare. The pathogenesis of lung involvement by dengue is incompletely understood[8]. Histological findings include interstitial edema and hemorrhage. Dengue virus has been detected in the pulmonary endothelial cells and macrophages[9,10]. In a series by Wang *et al*, 4 patients were reported to have bloody pleural effusion[11]. However there have been no reports of such a massive hemorrhagic effusion as a presentation in DHF. The causation of bleeding in dengue is multifactorial including defects in the coagulation cascade, platelet dysfunction, disseminated intravascular coagulation and thrombocytopenia. Histamine release has also been postulated to cause a vascular leak[12]. Usually blood and platelet transfusions are given in the settings of coagulopathy leading to severe bleeding[2]. In our patient, there was a significant drop in haemoglobin levels due to continuous bloody leakage into the right pleural cavity requiring blood transfusion. We were perplexed by the immediate gush of 1.1 L of blood tinged fluid within 5 min of ICD insertion. We refrained ourselves from allowing further flow fearing a hemodynamic collapse. We subsequently released the ICD drain for short intervals each time till the drainage gradually stopped. We avoided thorocotomy in view of improvement. He gradually became less tachypnoeic followed by re-expansion of the right lung. In addition, he also had markedly raised liver enzymes which is a more common phenomenon in dengue. This results from the direct damage to the hepatocytes by the dengue virus or due to an unregulated immune response[13]. Our patient did not develop hepatic encephalopathy though he had an increased bleeding tendency. Liver enzymes gradually improved without any undue complications.

We would like to highlight this unusual case of massive right sided hemothorax as a presenting feature of dengue. Physicians must be aware of DHF as a cause of non-traumatic hemothorax especially in an endemic area. Prompt diagnosis and aggressive supportive therapy with blood and platelet transfusions are imperative to maintain hemodynamic stability and prevent mortality. To our best knowledge, this is the first case of DHF presenting as unilateral massive hemothorax.

Conflict of interest statement

We declare that we have no Conflict of interest.

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