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Atypical respiratory complications of dengue fever

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ABSTRACT

In last decade, dengue has emerged as one of the most important vector born disease. With increasing cases, uncommon presentations and complications are now commonly recognized. Here, we report two cases of rare pattern of respiratory involvement in dengue: acute respiratory distress syndrome and bronchiolitis with respiratory failure.

1. Introduction

The incidence of dengue fever has increased 30 times in the last four decades^[1]. It is an upcoming important viral febrile illness of tropical countries. Most of the cases are asymptomatic. It has spectrum of manifestation from mild fever to life threatening hemorrhagic manifestation to shock syndrome. Lung involvement is common in form of pleural effusion as a part of poly-serositis. Less common pulmonary complications include pneumonitis, non-cardiogenic pulmonary edema (Acute respiratory distress syndrome, ARDS), and hemorrhage-hemoptysis. These complications usually coincide with plasma leakage syndrome and thrombocytopenia.

2. Case report

2.1. Case 1

A 60 year old male, a known case of diabetes and hypertension for 15 years, was admitted with one episode of syncope at work place. He gave history of fever for 3 days, which was low grade, and resolved 2 days before

admission. Since then, he was having weakness, generalized body ache, decreased appetite and nausea. On examination, there was generalized flushing; BP 100/70 mmHg, PR 110/min, temperature 38.3 °C; right hypochondrial tenderness was present. There was postural drop of blood pressure. On investigation, ECG had LVH with strain pattern, Trop I was negative and 2D Echo was normal. NCCT head was within normal limits. His total leucocyte count was 4 000/mL, platelet count 80 000/min, PCV 45%, bilirubin 0.6 mg/dL and SGOT/SGPT was 167/113 IU/L respectively. Ns1 antigen was positive. On the second day of admission, the patient complained of breathlessness, which rapidly progressed in severity over 12 hours to breathlessness at rest. On examination, he was tachypnic (RR 26/min), had crepts in bilateral inter- and infra-scapular area and SpO₂ was only 78%. Chest X ray showed bilateral infiltrate in middle and lower zone of lung fields. ABG was done, which had respiratory alkalosis (pH 7.48, pCO₂ 23.8 mmHg), type 1 respiratory failure (pO₂ 53.9 mmHg) and PaO₂/FiO₂ ratio of 134.75. H1N1 serology was negative, and pro-BNP and D dimer were normal. A diagnosis of non-cardiogenic pulmonary edema/ARDS due to dengue was kept. He was started on BiPAP respiratory support, broad spectrum antibiotics and diuretics. Ultrasound had bilateral pleural effusion, hepatomegaly and mild ascites. Platelet count fell up to 30 000/mL; however, there was no bleeding from any site, and transfusion was not required. He was able to maintain saturation, without BiPAP by Day 7 and without oxygen support by Day 10. Changes in chest radiograph, ultrasound abdomen and LFT were improved, and he was discharged by the 12th day.

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2.2. Case 2

A 26 year old female was admitted with complain of running nose, dry cough for 2 days and shortness of breath for 1 day. One week back, she had high grade fever for 3 days. There was no past history of asthma or any other chronic disease. On examination, she was conscious, not cyanosed, but very breathless and unable to talk in full sentences. Blood pressure was 112/64 mmHg, pulse 132/min, respiratory rate 32/min, temperature 37.0 °C and SpO₂ was 77% without oxygen, increasing up to 98% with 2 L O₂/min. Chest had extensive polyphonic rhonchi in all area; rest systemic examination was normal. Chest radiograph had prominent broncho–vascular marking and haziness in bilateral lower zone. Total leucocyte count was 11 900/mL, platelet count 1.15×10³/mL, bilirubin 0.7 mg/dL, SGOT 141 IU/L, SGPT 174 IU/L. ABG had type 1 respiratory failure (pH 7.41, pCO₂, 35.7 mmHg, pO₂ 58.1 mmHg, SpO₂, 90.8%) with PaO₂/FiO₂ ratio of 290.10. Levels of proclacionin, pro–BNP and D dimer were not raised. She was positive for dengue IgM serology. A diagnosis of dengue pneumonia was kept and she was started on oxygen supplementation, nebulization with bronchodilators, broad spectrum antibiotic and IV steroids. There was gradual improvement: breathlessness was reduced, tachypnoea was settled, rhonchi in chest gradually became better and was able to maintain saturation on room air by Day 7 of admission. At 2 weeks of follow up, all changes had reversed and pulmonary function tests were normal.

3. Discussion

ARDS is an uncommon and under–recognized complication of dengue fever but has high mortality[2]. In a study involving 606 patients in China, Wang *et al.* reported an incidence of acute respiratory failure in only 1.8% of patients; mortality rate was 72.7% in this group of patient[2]. All these patients had concomitant DHF. In another large retrospective study on 3 488 pediatric dengue patients from Thailand, Laoprasopwattana *et al.* found acute respiratory failure in only 36 patients (1.03%); mortality in this group of patient was 61.1%[3]. Cases have also been reported from various parts of India[4,5]. The exact pathophysiology of ARDS in dengue is unknown. The main difference between severe dengue hemorrhagic fever and simple dengue fever is the increase in vascular permeability due to endothelial injury, leading to plasma leakage into the extravascular compartment. The same process leads to increased permeability of the alveolar–capillary membrane resulting in the edema of the alveoli and interstitial spaces and thus leading to pulmonary dysfunction. In predisposed patients, it can lead to severe form of lung involvement like pneumonia and ARDS. Early restoration of adequate tissue perfusion is critical to prevent progression to ARDS, but equal care must be taken to avoid excess fluid infusion as it can also lead to ARDS[6]. However, in study by Wang *et al.*, aggressive fluid replacement was not a factor contributing to acute respiratory failure[2]. Wang *et al.* concluded from their study that sepsis and upper gastrointestinal bleeding were two most important risk factor for progression to ARDS in dengue patients[2]. Other risk factors they recognized include old age, abnormal investigations (Prothrombin time, activated partial thromboplastin time, aspartate aminotransferase, alanine aminotransferase, blood urea nitrogen, creatinine,

and albumin), and complications (acute renal failure, acute hepatic failure, UGI bleeding, and sepsis). Similarly, Laoprasopwattana *et al.* found acute respiratory failure in dengue to be associated with severe disease and obesity[3]. In their study, risk of mortality was associated with severe dengue, acute kidney injury, active bleeding and severe liver disease[3].

Management of ARDS in dengue fever is similar to ARDS of any other etiology. Study by Cam *et al.* proved the role of noninvasive ventilation in improving the morbidity and mortality of dengue associated ARDS[7]. The supportive management for dengue also influences the outcome of the condition. Apart from respiratory support, minimizing nosocomial complications and preventing risk factors as mentioned above in previous studies play prominent roles in its management. The good prognosis of our patient could be due to mild form of the disease, prompt institution of supportive therapy and use of broad spectrum antibiotic.

In our second case, the patient had bronchiolitis type of presentation, leading to type 1 respiratory failure. She neither had typical history of dengue nor developed other typical complication of this condition. The patient improved rapidly as the primary process was not very severe and adequate treatment was instituted promptly. This type of respiratory involvement is also quite uncommon in dengue fever and has been reported to add to the list of atypical respiratory complications.

In conclusion, clinicians in endemic areas should be aware of these unusual complications. Crux to their management is an appropriate therapy without delay, as in both of our cases.

Conflict of interest statement

The authors declare that they have no competing interests.

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