

Case Report

PULMONARY – RENAL-HAEMORRHAGE SYNDROME IN DENGUE - A RARE CASE REPORT

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ABSTRACT

Dengue is a vector borne viral disease characterized by high fever, joint pains, and rashes. In its severest of forms, it presents as hemorrhagic fever (DHF), sepsis and shock syndromes (DSS) causing widespread multi-organ failure. Invariably every organ is involved in dengue. We present a case of a 28-year-old female, who presented to us with dengue fever with catastrophic hemoptysis and hematuria and succumbed to illness.

Keywords: DHF, DSS, Shock, Sepsis

INTRODUCTION

Dengue is a mosquito-borne viral infection causing a severe flu-like illness and, sometimes causing a potentially lethal complications called severe dengue (previously known as dengue hemorrhagic fever). Dengue fever is caused by different serotypes of dengue virus DEN-1, DEN-2, DEN-3, DEN-4 dengue virus. Infection by any of the four serotypes of virus can cause this fever. It is transmitted by female mosquito, mainly of the genus *Aedes*. It is an important cause of serious illness and death amongst tropic and sub-tropic regions. Patient usually presents with symptoms like high fever, headache, vomiting, muscle and joint pain and a characteristic skin rash. The disease may result into serious life-threatening complications like dengue hemorrhagic fever (DHF), Dengue Shock Syndrome (DSS) leading to circulatory failure. Pulmonary haemorrhages and kidney failure with haematuria in dengue is a rare occurrence (Sharma *et al.*, 2007).

Dengue fever, in its more serious forms- DHF and DSS may lead to kidney injury, of nephritic type leading to glomerular inflammation, hematuria, proteinuria, and failure in rare cases, the mechanism of which is unclear but suggests immune complex deposition leading the Type- III hypersensitivity reaction in the glomerulus. The circulating IgG antibodies combine with viral antigens to form a complex which is deposited in the glomerulus (Khalil Muhammad *et al.*, 2012). In some cases, due to the association of AKI with DSS, the cause of nephritis has also been believed to be due to septic shock. Also, in many cases with hematuria in dengue fever, involvement of other organs has also been seen like those of the viscera, as well as lung which also points towards Goodpasture syndrome-like picture leading to rapidly progressive glomerulonephritis (RPGN) (Lizarraga and Nayer 2014). AKI can also be caused by septic shock, hypotension, myositis leading to rhabdomyolysis and myoglobinuria. Pulmonary symptoms like effusion and hemoptysis are even rarer. Clinically, it may only be evident as tachypnea, which initially may be associated with dry cough and later bloody cough or hemoptysis. The mechanism of such events is unknown but is believed to be multifactorial with abnormalities in coagulation system, thrombocytopenia, platelet dysfunction, DIC, vascular defects and increased vascular permeability thought to be mediated by histamine during the process of inflammation. Imaging studies like X-Rays, showed bilateral fluffy condensations, CT imaging showed ground glass appearance and high resolution CT scan revealed large areas of consolidation along with ground glass appearance (Marchiori *et al.*, 2009).

CASE

A 28-year-old female presented to us with history of fever, headache, and myalgia of 2 days duration. There was no history of cough, expectoration, breathlessness, palpitations, burning micturition. On

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examination; she was alert, responding to questions. Pulse was 108/ minutes, regular. Blood pressure was 110/70 mm of Hg. RR was 16/ min, Spo2 was 100% while breathing ambient air. Pallor, icterus, cyanosis were absent. JVP was normal. All the other system examination was normal.



Figure 1: CXR showing Bilateral pulmonary infiltrates

Investigations revealed, Hb -11 gram%, TLC 11,500/mm³ with lymphocytic leukocytosis. Absolute platelet count- 66,000/mm³. PS was negative for malarial parasites. PARACHEK was negative. Urine examination showed 12 RBCs/HPF. Dengue IgM/ NS1 was positive. KFT, LFT was normal. She was treated with IV fluids, prophylactic antibiotics, antipyretics.

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Twelve hours after hospitalization she started complaining of cough and dyspnea. On examination; pulse was 145/min. RR was 35/ min with use of accessory muscles. BP was 98 systolic. Spo2 was 74% while breathing ambient air. Auscultation revealed scattered crepitations in bilateral lung fields. ABG revealed hypoxia. Pao2/FIO2 ratio was 188. CXR showed pulmonary infiltrates (Figure 1). ECG showed sinus tachycardia. 2D Echo was normal. Repeat absolute platelet count was 40,000/mm³, APTT was 92 secs, PT was 16 secs.

She was immediately started with NIPPV, vasopressors, diuretics, fresh frozen plasma and platelet transfusions. In view of increased cough and one episode of hemoptysis, she was intubated and mechanically ventilated in ARDS protocol. But her condition continued to deteriorate with ongoing bleeding from lungs with frequent ET tube suction revealing bloody fluid. Her bladder catheter showed frank hematuria. Her repeat counts were increased at 20,000/mm³. Platelet count was 18,000/mm³. Repeated platelet transfusions were given to maintain platelet count above 50,000/mm³. Her pulmonary hemorrhage didn't stop and she succumbed to illness in next 12 hours.

DISCUSSION

The clinical spectrum of dengue virus infection can range from mild febrile illness like any other viral fever followed by recovery to catastrophic presentation like DHF and DSS along with involvement of any organ in human body.

Encephalitis, meningoencephalitis, (Acharya *et al.*, 2013) ARDS, pulmonary haemorrhage, myositis, (Acharya *et al.*, 2010) AKI, myocarditis are known to occur. Pulmonary haemorrhage is a catastrophic presentation and is usually rarely seen according to reports. Hemoptysis is seen in only 1.4% of dengue infections (Hayes *et al.*, 1988; Liam *et al.*, 1993; Setlik *et al.*, 2004). The process and the pathophysiology of Dengue Pulmonary Haemorrhage is multifactorial and is attributed to abnormalities in coagulation cascade, thrombocytopenia, functional platelet disorders, DIC, Endothelial Injury, Vasculitis, capillary leakage. Specially the capillary leakage and vasculitis has been attributed to histamine mediated phenomenon (Sharma *et al.*, 2007; Setlik *et al.*, 2004). Our patient had catastrophic pulmonary haemorrhage and ARDS possibly due to severe thrombocytopenia and DIC. Dengue is also attributed to induce kidney in various forms. AKI in the setting of dengue fever typically occurs due to hypotension, rhabdomyolysis (Acharya *et al.*, 2010) myoglobinuria, haemolysis leading to haemoglobinuria, dehydration and glomerulonephritis.

Glomerulonephritis in dengue fever is usually of mesangioproliferative type ³ (Lizarraga and Nayer, 2014) and sometimes kidneys are involved due to systemic immune mediated inflammatory response. Proteinuria is seen in 74% of patients with DHF, (Lima and Nogueira, 2008; Horvath *et al.*, 1999) sometimes even to nephrotic range proteinuria (Vasanwala *et al.*, 2009). In another study the prevalence of proteinuria in dengue was up to 30% (Gracia *et al.*, 1995).

Our patient had haematuria and proteinuria suggesting a possible glomerular inflammation. Hypotension due massive pulmonary haemorrhage may have contributed to further renal deterioration. In this rare case report, our patient manifested with sudden onset of pulmonary haemorrhage and haematuria. She had thrombocytopenia, DIC and immunologic glomerulonephritis suggesting multifactorial features features suggesting for this catastrophe.

This case report also highlights that once the pathophysiological processes are set into action, then within no time life threatening complications can occur in dengue fever. From a seemingly well-preserved dengue virus infection, our patient succumbed to the complications within 24 hours time.

To conclude, physicians dealing with dengue fever should keep in mind all these possibilities and remain in high alert while treating dengue patients with sub-clinical laboratory abnormalities.

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