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Inapparent Dengue Death

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Introduction

Dengue is a viral fever symptoms like disease, especially with severe joints and muscle pain, hence also previously known as break bone fever. This Dengue fever is primarily known for > 200 years, to health experts. Dengue is fast emerging pandemic prone viral disease affecting primarily Asian and Latin American countries.

Over the past 20-25 years, next to disease related to diarrhea and respiratory infection, dengue has become one of the leading causes for hospitalization and deaths among children. Today, dengue is one of the most important arthropods borne viral diseases in humans, as far as mortality and morbidity is concerned. It is also the number one killer worldwide amongst viral infection. Among travelers dengue is the second most diagnosed cause of fever after malaria. In 2015, Delhi [1] India, recorded its worst outbreak since 2006 with over 15,000 cases (1996, which was the first major outbreak in Delhi). Globally the number of cases increased from 2.2million in 2010 to over 3.34 million in 2016[2].

Dengue is found where Aedes mosquitoes are present. Aedes mosquitoes are found in tropical and subtropical regions. These mosquitoes cannot fly more than 200 meters horizontally and 1000 meters vertically. But now Dengue has become a global problem and is common in > 110 countries. Each year 10,000 to 25,000 die and between 50 and 528 million people get infected and 2.5 Billion people (2/5th world population) at risk[3] of infection.

Approximately 1 in every 13 patients of dengue reports to doctor. Mortality in Dengue fever is 1-2 %, in DHF (Dengue Hemorrhagic fever) is 10% and in DSS (Dengue Shock Syndrome) is 12-44% in various studies.

Death is because of low blood pressure, major bleeding or multi- organ failure.

The progress towards DHF/DSS occurs after 3 to 5 days of fever. When fever is coming down one may get mislead that the patient is heading towards recovery. In fact this is the most dangerous time that requires high vigilance and care[4].

Case presentation of Inapparent dengue death

History: 41 years female presented with fever for 6 days, loose stools and vomiting for 2 days.

On Examination: Patient conscious, oriented
Pulse; 110/min, BP- 100/60mmHg, Temp- 101.4 ^{0} F, SPO2- 95% on RA,
PA- Soft, mild tender
Chest – Clear, CVS- S_1 S_2 normal

Diagnosis: Secondary dengue with DSS with Multi – organ Failure with Lactic acidosis with ARDS.

Course in Hospital: Patient was brought to emergency department and the diagnosis thought was acute febrile illness ? Typhoid ? Dengue. Hence in ward was resuscitated with IV antibiotic, IV Fluids (bolus & maintenance), Antiemetics, Antacids and supportive measures. All fever panel investigations sent which revealed Dengue serology (IgG, IgM) positive, hence secondary dengue was the diagnosis. USG abdomen showed liver 1cm enlarged with no pleural effusion or ascites. There was no bleed from any site. Platelet count and PCV were within normal limits. Hence patient was maintained with, oral fluids, soft diet, and supportive measures, keeping in mind her urine output which was also adequate. Gradually, loose motion, vomiting were settle down, but fever persisted intermittently. On 4th day of hospitalisation, repeat PCV was 28.98, platelets being normal; at 7:20pm patient developed breathlessness, her SPO2 on room air dropped (with 4litres/min O2 support was 99%), pulse- 90/min, BP- 110/70mmHg, chest examination revealed bilateral rhonchi, immediately patient was shifted to ICU, under the care of intensivist team.

In ICU patient's pulse– 92/min, BP- 132/100mmHg, Temp- 102^0F, RR- 32/min, SPO2- 99% (on O_2). ABG implied PH- 7.364, PaO_2 - 88.5(low), PCO_2 – 34 (being normal), Lactate- 5.9 (high), HCO_3 – 18.9 (low), Chest x- ray showed bronchovascular markings prominent with hilar haze. Gradually lactic acid increased, with gradually lowering of BP, tachycardia and hypoxia, probably because of myocardial depression. Also patient developed bilateral crepitations/rhonchi.

On 5th day of hospitalisation, she was intubated and ventilated in view of double acidosis, (Metabolic & Respiratory). IV inotropes support were gradually increased, IV antibiotics were escalated also. She remained in intractable shock. In Spite of team containing Cardiologist, Nephrologist Pulmonologist and intensivist care, she progressively deteriorated became anuric and developed ARDS.

At 11:50pm, she developed cardiac arrest, CPR as per ACLS protocol done but could not be survived and was declared dead at 12:30am.

Probable Cause of Death: Lactic acidosis, ARDS, female and secondary dengue fever.

Bad Prognosis factors: Female[5]; secondary dengue.

Discussion

Severe dengue as per WHO consists of (1) severe plasma leakage (leading to shock, fluid accumulation with respiratory distress), (2) severe bleeding and (3) severe organ involvement (impaired consciousness ALT/AST more than 1000, heart involvement etc.).

Warning signs as per WHO are abdominal pain/tenderness, persistent vomiting, clinical fluid accumulation, mucosal bleeding, lethargy/ restlessness, liver enlargement more than 2 centimetres, increase in haematocrit and rapid decrease in platelet count.

But in our case neither severe dengue nor warning signs/symptoms were present. PCV, platelets, coagulation profile and organs (well perfused) were within normal limits.

Patient admitted with fever, loose motion, vomiting but stable initially, admitted in ward, suddenly deteriorated in the form of hypoxia, breathlessness; platelets & PCV being normally stabilized.

Patient usually dies because of bleeding (with low platelets or deranged coagulation) or capillary leakage (with PCV high and shock). But this patient both platelets, coagulation profile) and PCV were normal. Patient deteriorated after a few days, in spite of PCV/ platelets being normal, in the form of breathlessness/hypoxia leading to acidosis, lactic acidosis, ARDS.

Hence Onset of Shock in dengue can be dramatic and its progression relentless[6].

Why DSS (dengue shock syndrome) patients die:
Patient who dies from severe Dengue with DSS and MOF is not fully understood, hypothesis may be as below:

1. May be due to more virulent strains of dengue virus.
2. May result from abnormal and exaggerated host immune response- in particular dengue virus cross reactive antibodies, production; which augment the reaction (known as antibody dependent enhancement leading to shock).
3. Malfunction of vascular endothelium.
4. In spite of best care :
   i) Case fatality of severe dengue in Asian Countries is 0.5 to 3.5% [7].
   ii) Mortality rate is 2 to 5% when appropriately treated [8].

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