



ATYPICAL MANIFESTATION OF DENGUE FEVER.

Internal Medicine

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ABSTRACT

Dengue fever with different spectrum of presentation can affect any age group. Along with typical presentations of fluid leak and thrombocytopenia it may present with many Atypical manifestations. Here we are discussing a case who presented to PESIMSR, kuppam, Andhrapradesh with dengue IgG antibodies positive he had manifestations of Disseminated Intravascular Coagulation.

KEYWORDS

INTRODUCTION:

Dengue virus is an Arbo virus belonging to genus flaviviridae. It is a RNA virus transmitted by the bite of infected female Aedes mosquito, which feeds on humans during day time. It has four serotypes DENV1, DENV2, DENV3, DENV4¹. Infection of one serotype confers life time immunity against that serotype and partial immunity to other serotypes. The dengue virus after entering the body enters the mononuclear cells and releases inflammatory mediators which are responsible for the manifestations². It also causes increased vascular permeability causing not only shifting of fluid, but also proteins like albumin³. The burden of the disease will be more in tropical countries like India. The incidence of dengue in our country increased from 28292 in 2010 to 157220 in 2017 and the dengue related death increased from 110 in 2010 to 250 in 2017⁴.

CASE REPORT:

A 34 yr male patient, farmer from gudupalle(Andhrapradesh) brought to ER with C/O Fever from 8 days which was high grade associated with chills, rigors. Headache, body pains, abdominal pain from 4days, vomiting from 4days which was non projectile, non bilious, not associated with blood, black coloured stools from 4 days, no h/o blood in stools, altered sensorium from 2hours, no h/o cough, breathlessness, chest pain, palpitation, loose stools. He was a diabetic from 1yr on INJ. INSUGEN -N 15u-0-15u, INJ. H.ACTRAPID 15u-15u-15u, Smoker and Alcoholic from 15 yrs. No h/o drug intake, intoxication, no h/o prior surgeries / hospital admissions.

On General physical examination Patient was drowsy, disoriented, Pulse rate of 124bpm, Blood pressure was 100/60 mmHg, Resp rate of 30/min, temperature was 99.6° F, SpO₂ was 88% on room air and with 6lit of o₂-94%, he had Icterus and sub conjunctival haemorrhages, no pallor or dependent oedema or lymphadenopathy or eschar. On Systemic examination there were crackles over bilateral basal lung areas, his GCS was low, abdomen was soft, no hepatosplenomegaly, cardiovascular system was normal. Patient was intubated in view of respiratory failure later inotropes were started as he had hypotension.

Based on the above clinical history we kept the possibilities of viral fever with meningoencephalitis, malaria, leptospirosis, scrub typhus, enteric fever. Investigations done revealed Hb-10.6g/dl, TLC-10.1(N-64.5, N-34.4), platelets-26,000, HCT-42.1, deranged liver parameters (total bilirubin-5.2mg/dl, direct-2.83, AST-353, ALT-80, ALKP-625) and coagulation profile (PT-20.2, Aptt-41.7, INR-1.45). serology for malaria, leptospira were negative, titres of widal test were not suggestive of enteric fever. Dengue serology showed IgG positive, IgM, NS1 antigen negative. Blood gas analysis showed metabolic acidosis. His chest X ray showed features of ARDS. ECG, 2DECHO were normal, ultrasound abdomen showed features of polyserositis. CT scan brain was normal.

He was diagnosed as viral fever with MODS (ARDS, acute liver failure, shock, coagulation defects). Patient was treated with meropenem, metrogyl (suspecting sepsis secondary to ?UTI,

aspiration pneumonia). He was also given artesunate empirically for malaria and doxycycline for atypical coverage, I.V fluids, platelets and FFP were transfused.

Patient condition improved improved, inotropes tapered on stopped. we weaned from ventilator and extubated. On 8th day of his admission he suddenly became unresponsive and collapsed, during resuscitation about 400ml blood gushed out of his ET tube. lab values showed deranged coagulation profile [(raised PT24.1, Aptt68.4, INR-3.67. D-dimer was elevated (>0.5mcg/mlFEU)]. Ultrasound abdomen showed hemoperitoneum which was confirmed by tapping, Venous Doppler of both lower limbs showed no deep vein thrombosis. He was given platelets, FFP, packed cells. In spite of our best efforts patient could not be revived, death declared after 6 hours of first cardiac arrest.

DISCUSSION: Dengue is one of the common viral infection affecting the tropical countries, its spectrum may range from simple febrile illness to severe dengue hemorrhagic fever/dengue shock syndrome also many atypical manifestations can occur which may be life threatening⁵. Some of the atypical manifestations are Severe hepatitis, ARDS, Myocarditis, pericardial effusion, Acute Kidney Injury, Disseminated Intravascular Coagulation, Encephalitis⁶. Patient reported above had dengue hemorrhagic fever but his IgG antibody positive. He presented with multiple atypical manifestations (ARDS, Liver failure, bleeding manifestations) after early improvement there is a dreadful bleeding manifestations. We suspected pulmonary embolism but his 2D ECHO showed no evidence of pulmonary artery hypertension or Right ventricular strain. So we attributed the cause of his bleed most probably due to DISSEMINATED INTRAVASCULAR COAGULATION as there are elevated D-dimer. Dengue hemorrhagic fever may not present only with bleeding manifestations due to thrombocytopenia secondary to capillary leak, but also there may mechanisms by which dengue causes derangement in the clotting factors. There is decrease in anticoagulant proteins C, S, antithrombin III, this occurs due to the leakage of the above proteins due to increased permeability. Tissue injury causes release of tissue factor which initiated extrinsic pathway of coagulation. Plasminogen will cleave this and forms Fibrin Degradation Products (FDP). Also some study showed plasminogen gets directly activated by dengue virus extracts⁷. Infected with dengue virus are known to activate endothelial cells. Dengue virus can directly activate plasminogen dengue virus infected patients had cross reacting antibodies against plasminogen⁸. Thus in dengue infection there may be thrombosis development because of the loss of anticoagulants, activation of extrinsic pathway. Bleeding secondary to platelets loss, dysfunctioning platelets, and direct activation of fibrinolytic system.

The pathogenesis of dengue hemorrhagic fever is unclear yet, the proposed mechanism is Antibody Dependent Enhancement⁹ in which the antibodies formed during prior dengue infection with a particular serotype of dengue virus cannot completely neutralise the new serotype but they partially block them and increase the virus uptake in

to mononuclear cells, hence the disease will be severe because of increased released of inflammatory mediators by the cells.

CONCLUSION: There are many atypical manifestations of dengue hemorrhagic fever, some may present with multiple Atypical presentations. DIC is one among the rare manifestations, it's occurrence have been reported in many primary dengue infection. But sometimes DIC may improve initially and reoccur. Hence one should be careful in treating patients who are positive for IgG antibodies to prevent dreadful complications.

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