

Dengue Fever Related Autoimmune Response - A Case Report

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Abstract: *Dengue is an infectious, mosquito-borne disease caused by four distinct serotypes of an arbovirus, i.e. dengue virus. Dengue is seen in tropical and sub-tropical countries with a recent trend showing rise in infection in urban and semi-urban areas. Dengue virus produces auto-antibodies against human cells, triggering events causing an abnormal immune response. We hereby report a case of Dengue virus infection; who developed an auto-immune response, and want to increase awareness of this condition amongst physicians, as some patients may erroneously be diagnosed as rheumatological disorder.*

Keywords: Dengue, Dengue Fever, Auto-immune response

1. Introduction

Dengue is one of the most common viral and probably also the most important arbovirus infections in the world. Dengue is a mosquito-borne infection caused by four distinct serotypes of an arbovirus, i.e. DEN-1, DEN-2, DEN-3, DEN-4. Dengue virus produces auto-antibodies against human cells, triggering events causing an abnormal immune response. Autoimmunity is characterized by auto-antibody production and activation of auto reactive lymphocytes, which have been demonstrated to be associated to a number of viral pathogens. In dengue viral infection there is an enhanced immune response in a host ; which results in formation of circulating immune complexes, complement activation; increased histamine release and increase cytokine release. We report a case of Dengue fever related Autoimmune responses.

2. Case Report

A 22 year old female presented to our hospital with fever, high grade associated with chills and rigors for the last 10 days, along with history of abdominal pain, vomiting and generalised body aches for last 5 days. On examination, patients pulse was 112/min, blood pressure was 98/68 mmHg, temperature was 38.8 degree celsius and bilateral pedal oedema. On respiratory system examination there was decreased air entry in right infra scapular area. Investigations revealed CBC: Hb:10.9 g/dl, TLC: 9,900/cumm , platelets: 1,26,000/cumm, ESR was 26mm/1sthr, SGOT: 3662 ,SGPT: 1772 . Patients Dengue IgM was positive. Other infective causes of fever (malaria, scrub typhus, leptospira, brucella) were negative. USG abdomen showed Moderate ascites and Bilateral pleural effusion. Tapping of ascitic fluid, 1600 ml was done which was transudative.AFB, KOH, Gram stain was negative. Ascitic fluid culture was sterile. In view of persistent fever, HLH markers were sent which showed Ferritin 2797 ng/ml, Triglyceride 187 mg/dl, Fibrinogen level was 3.47g/l. Tapping of pleural fluid of 800 ml from right pleural cavity and 200 ml from left pleural cavity was done which showed cell count of 873/dl, fluid protein of 2.78gm/dl, fluid albumin of 1.25gm/dl, glucose of 100 mg/dl, ADA of 8.3u/L, AFB and KOH staining was negative, Pleural fluid culture was sterile. As fever spikes persisted ,CECT Thorax was done showed fatty infiltration of liver, moderate right

and minimal left pleural effusion, consolidation and collapse in the right lower lobe, mild pericardial effusion. High grade fever spikes persisted for 25 days. Autoimmune profile was done which showed, ANA was negative. ANA profile showed anti-histone antibody positive. Anti c-ANCA level was positive (34AU/ml). C-ANCA immunofluorescence - negative. Anti p-ANCA was normal (<3 AU/ml). Serum C3 level was 1057 mg/dl. Serum C4 level was 152mg/L. DAT was positive 2+ reaction , IDAT negative. Bronchoscopy was done which was normal. BAL was taken which was negative for AFB, gram stain, KOH preparation, and was sterile. 2D ECHO showed no RWMA, trace TR, PASP 35mmHg, LVEF 65%. In our patient the dengue fever lasted for 25 days and Autoimmune markers were positive which were due to immune complex. Over the period of time the patients fever subsided with supportive measures and the autoimmune markers were normalised.

3. Discussion

Dengue virus produces auto-antibodies against human cells; triggering events causing an abnormal immune response; mainly as a result of cross-reactivity between anti-nonstructural protein and the host proteins; endothelial cells and platelets; mediating complement activation and triggering plasma leakage. Molecular mimicry could also be involved in the pathogenesis of the autoimmune activation. Auto-immunity is implicated in the pathogenesis of many viral infections such as; HIV, Hepatitis-C, CMV, Herpes simplex virus, EB virus and Dengue virus. In our patient the probable immunological mechanism was suggested by the wide spectrum of laboratories abnormalities: anti-histone antibodies positive, anti-c-ANCA levels positive, DAT 2+ positive, raised ferritin levels.

Prolonged symptoms of Dengue fever, such as we observed; are not common and could lead to diagnostic difficulties and so the patient could be easily confounded with a rheumatological disorder. Therefore, the physicians should be aware and vigilant before erroneously diagnosing a Dengue patient as a rheumatological disorder.

4. Conclusion

Dengue virus produces auto-antibodies against human cells, triggering events causing an abnormal immune response. We the physicians should have awareness of this condition, as some patients may erroneously be diagnosed as rheumatological disorder.

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