International Journal of Research in Medical Sciences
Egboh SMC et al. Int J Res Med Sci. 2018 Aug;6(8):2827-2830
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20183278

Case Report

A case of dengue fever in a health worker in university of Port-Harcourt teaching hospital

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Received: 02 June 2018
Accepted: 28 June 2018

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ABSTRACT

Dengue fever is a mosquito borne disease which can easily be missed or misdiagnosed in early stages as a result of poor surveillance, similarity with other febrile illness and unavailability of PCR necessary for its diagnosis. We hereby report a 44year old female nurse living around a bushy and swampy environment, who presented to university of Port-Harcourt teaching hospital (UPTH) with high grade fever of 1week duration, also developed joint pains, passage of coke colored urine, sensor neural deafness, hyperglycemia and petechial hemorrhage while on admission. Patient continued to have persistent fever despite having completed her anti-malaria and antibiotics, viral studies were subsequently done for her and she was found to have dengue virus. Patient was conservatively managed and discharged for follow-up in medical outpatient and Ear nose and throat clinic.

Keywords: Dengue fever, Health worker, Port-Harcourt

INTRODUCTION

Dengue fever is a mosquito-borne disease caused by any one of four closely related dengue viruses (DENV1, 2, 3, and 4). Dengue virus is a positive-stranded encapsulated RNA virus, which is approximately 11 kb in length and is composed of three structural protein genes that encode the nucleocapsid or core protein (C), a membrane-associated protein (M), an envelope protein (E), and seven nonstructural (NS) protein genes.[1] Infection with one serotype of dengue virus provides immunity to that serotype for life but provides no long-term immunity to other serotypes. Dengue viruses are transmitted from person to person by Aedes mosquitoes (most often Aedes aegypti) in the domestic environment, although anthropophilic transmission can be by Aedes albopictus, Aedes africanus, and Aedes. luteocephalus.[2,3]

CASE REPORT

The patient was a 44year old nursing staff, who presented to the accident and emergency unit of UPTH, with high grade fever of 1-week duration associated with chills and rigors, generalized body pains and weakness. There was also a history of non-bilious, non-projectile vomiting of recently ingested food and no history of contact with any person with high fever. Patient has been on quinine for 2 days before presentation, but fever persisted. On examination she was febrile with a temperature of 390C. Investigations revealed that she had + of *Plasmodium Falciparum*, urinalysis showed >500mg of protein, 14mmol of glucose and 80cells/ul of blood, random blood glucose done was 24.3mmol/l, serum electrolyte, urea and creatinine were within normal range. Urine culture and blood culture yielded no growth. Patient had a

normal WBC count of 6.9 X 109/l with relative lymphoctosis (61%) and neutropenia of 31%, left ward shift of the neutrophils to the myelocytes and few toxic granulation. Platelet count was within normal range (227 X 109/L).

Based on this, an initial diagnosis of unresolved malaria and type II diabetes mellitus to keep in view sepsis was made and patient was commenced on intramuscular arthemeter, oral sulfadoxine-pyrimethamine, intra-muscular paracetamol, subcutaneous humulin and intravenous antibiotics, but however fever persisted with temperature of 390C to 400C. Patient then developed joint pains, passage of coke coloured urine, frothiness of urine, petechial rashes and sensorineural deafness, while on admission. A diagnosis of a viral hemorrhagic fever was made and blood sample was sent by the state epidemiologist for viral screening and she was found to have dengue fever virus using ELISA and PCR. A final diagnosis of dengue hemorrhagic fever was made. Patient was placed on conservative management which includes intravenous fluids, essential amino acids (B-histidine and nicotinic acid) and prednisolone was prescribed by the ENT surgeons for the deafness. Patient made marked clinical improvement. Environmental health assessment done at her residence and reading area showed defective drainage system with retained stagnant water and bushy environment.

Figure 1: Environmental health assessment. A) At the church where she does private reading revealed pool of stagnant water, unkempt bushes and open septic tank. B) At the entrance of the patient's house.

DISCUSSION

Dengue fever which originated in Africa is the most rapidly spreading mosquito-borne viral disease in the world with increasing geographic extension.[4] An estimated 50 million dengue infections occur annually and approximately 2.5 billion people live in dengue endemic countries.[3]

Although dengue virus exists in the WHO African Region, it is poorly reported because of low awareness by health care providers, similarity with other febrile illnesses, lack of diagnostic testing and systematic surveillance.[3]

The virus is transmitted by aedes aegypti, a mosquito that breeds around human dwellings in water filled habitat, this was confirmed by environmental health assessment done in the index patients home, which revealed that there were pools of stagnant water as well as bushes around her house, which may have served as a breeding space for aedes aegypti mosquito.[3]

After infection, there is usually an incubation period of 3-14 days, after which an individual may experience acute onset of fever associated with non-specific symptoms and signs like headache, arthralgias, myalgias and sometimes vomiting, which is consistent with the symptoms our index patient had.[5] During this period of acute illness, the virus may circulate in the blood stream so that if an aedes mosquito bites another person, there may be transfer of the virus leading to transmission of dengue fever.[6]

The patient also had hyperglycemia which could be stress hyperglycemia resulting from the release of counter-regulatory hormones. This was confirmed by HBA1C result of 5.9mmol/l, which shows that the patient, s blood glucose control 3 months prior to the onset of the illness was good. However, Muhammed et al have reported a significant association between dengue fever and development of diabetes mellitus, as 33% of 100 previously normoglycemic patients diagnosed with dengue fever in Lahore, were found to develop diabetes mellitus and this association was found to be higher among middle aged and elderly patients.[7]

Dengue virus infection can be grouped into undifferentiated dengue fever, dengue hemorrhagic fever and dengue shock syndrome.[8] The patient presented with features of dengue hemorrhagic fever which is characterized by vascular endothelial dysfunction and thrombocytopenia leading to plasma leakage and hemorrhage.[3]

The passage of coke coloured urine, presence of petechial rashes and blood cells in urine were suggestive of a possible endothelial dysfunction. Thrombocytopenia is common in dengue patients and is one of the implicated mechanisms for the development of hemorrhage in such patients, although the index case had a normal platelet count.[5] It could be because the full blood count of the patient was done at the early phase of the infection, this is supported by Joob et al who reported that 38.9% of patients with early dengue fever diagnosed by non-structural protein 1 had normal platelet count.[9]

More severely ill patients have gastrointestinal hemorrhage, hematemesis and passage of melena stool, these symptoms may subsequently lead to hypovolemic shock.[6] The pathogenesis of dengue hemorrhagic fever and dengue shock syndrome is not clear, but the

mechanisms that have been widely accepted are the expression of genetic changes in the virus and immune enhancement hypothesis.[6]

The immune enhancement hypothesis postulates that previous infection, through a process known as antibody-dependent enhancement, increases the replication of dengue virus in cells of the mononuclear cell lineage, with subsequent secretion of vasoactive mediators, leading to increased vascular permeability, hypovolemia and shock. Genetic changes in the virus genome may be associated with some phenotypic expression which include increased virus replication, virulence and epidemic potential.[6]

Neutropenia and lymphocytosis are also typical laboratory findings that may be associated with dengue fever, this was supported by the findings in our patient who had relative lymphocytosis (61%) and neutropenia of 31%.[3]

The index patient also had sensori-neural deafness, although the relation between dengue virus and sudden hearing loss, still remains unknown, Bruna et al, has reported a case of dengue fever who developed sensorineural hearing loss after the fifth day of onset of symptoms.[10] This could be explained by the viral theory of sudden hearing loss, where sensorineural deafness in viral infections could be attributed to the occlusion of an end artery that could lead to a reduction in blood supply to the cochlea.

Early notification of dengue cases seen in primary and secondary care is crucial for identifying outbreaks and initiating an early response.[3] There has to be a high index of suspicion for a physician to be able to make the diagnosis, however diagnosis on the basis of clinical syndromes alone is not reliable, and therefore should be confirmed by laboratory studies, because more than half of infected individuals either are asymptomatic or have a mild undifferentiated fever.[3]

The laboratory criteria for the confirmation of dengue fever include, isolation of dengue virus in the serum or organ fragments collected post mortem, detection of viral RNA using the reverse transcription-polymerase chain reaction (RT-PCR) technique, detection of non-structural 1 (NS1) protein in serum of infected patients, presence of IgM and IgG antibodies specific for dengue and presence of viral antigen in necropsied tissues usually detected by immunohistochemistry.[11]

The diagnosis of viral hemorrhagic fever in Nigeria is faced with a lot of challenge as there are only few hospitals with facilities for establishing this diagnosis. The index patients sample was sent to Lagos before a diagnosis could be made with Enzyme linked immunosorbent assay and polymerase chain reaction. This is a contributing factor to an increased mortality that is associated with viral hemorrhagic fever in developing countries, as early diagnosis, enhances prompt initiation of treatment. The management of dengue fever is conservative.

Intravenous fluids like normal saline should be used for immediate rehydration, however, there is increased risk of hyperchloremic acidosis with prolonged use. Therefore, ringers lactate is more appropriate for long term rehydration. Colloid can also be used for rehydration.[3]

The index patient was also placed on essential amino acid whose deficiency has been linked to hearing loss, steroid (prednisolone) was also given to reduce the inflammation that may be associated with sensorineural deafness. Fayad et al, has reported that nicotinic acid and steroid may be beneficial in the management of sensori-neural deafness.[12] Bouthinah et al, however, didn't find any deficiency of essential amino acid among his study subjects who are students aged 18-21years, hard of hearing.[13]

Oral-antidiabetic was also given to control patient's blood glucose after initial use of subcutaneous humulin. Environmental assessment was conducted by representatives of world health organization, federal ministry of health and state ministry of health, by visiting the patient's house and community. Bushy environment and defective drainage system with retained stagnant water, that served as a possible breeding place for mosquitoes was seen at the entrance of the index patients home, the church environment where she does her private reading, and the hospital where she works.

Patient is being seen regularly at the medical out patients and ENT clinics for follow-up. She currently uses hearing aids. Vector control was promptly initiated with the help of state ministry of health by indoor and outdoor spraying of the patient's environment.

Further measures are frequent emptying and cleaning of water-storage vessels, flower vases, gutters, recycling or proper disposal of discarded containers and tyres.[3] Human vector contact should be limited by installation of mosquito shield on doors, windows and other entry point for mosquito and use of mosquito nets while sleeping.[3]

CONCLUSION

Dengue fever is a viral hemorrhagic fever transmitted by aedes species of mosquito which breeds around bushes as well as stagnant water, it has a similar presentation with other viral illnesses, therefore, a high index of suspicion is necessary for its diagnosis, the index patient presented with diabetes mellitus and sensorineural deafness which is an uncommon presentation of dengue fever, confirmation may be challenging in our environment because of non-availability and non-affordability of polymerase chain reaction. We felt this case should be

documented as it is the first case of dengue fever in this establishment.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Egboh SMC, Otike-Odibi BI, Altraide DD, Awopeju ATO. A case of dengue fever in a health worker in university of Port-Harcourt teaching hospital. Int J Res Med Sci 2018;6:2827-30.