

Research Article

Clinical profile of dengue fever infection in patients admitted in tertiary care centre Agroha, Hisar, Haryana, India

Mohd Younus Shah*, Mohd Mubarak Naqash, R. K. Goel, Deepak Galhan, Sunil Kumar, Vivek Chhabra, Abhishek Saini, K. L. Jaggal

Department of Medicine, Maharaja Agrason Medical College and Hospital, Agroha, Hisar, Haryana, India

Received: 01 April 2016

Accepted: 09 May 2016

*Correspondence:

Dr. Mohd Younus Shah,

E-mail: doc_younus@yahoo.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: Dengue infections can result in a wide spectrum of disease severity ranging from an influenza-like illness (dengue fever; DF) to the life-threatening dengue hemorrhagic fever (DHF)/dengue shock syndrome (DSS). The study was aimed to compare the clinical profile of all patients diagnosed with dengue viral infection at MAMC.

Methods: This retrospective study included 188 patients infected with dengue virus, age 6 years to 70 years. Laboratory and haematological data were included.

Results: Peak of infection occurred in October 2015 and least number of cases were recorded in December 2015. Common clinical symptoms were fever, and abdominal pain. Common haematological abnormalities were thrombocytopenia and leucopenia. All patients survived. There was no case of dengue hemorrhagic fever or dengue shock syndrome.

Conclusions: Significant differences in the clinical profile is possibly because of infection with different serotypes of dengue virus (DENV), concurrent/sequential infection of more than one serotype, and differences in host immune responses associated with host genetic variations.

Keywords: Dengue fever, Thrombocytopenia, Leucopenia

INTRODUCTION

Dengue is a mosquito-borne viral illness caused by one of the four serotypes of the dengue virus (DENV; (DENV-1 to DENV-4) belonging to the family Flaviviridae. The virus serotypes are closely related but antigenically distinct. Dengue infections can result in a wide spectrum of disease severity ranging from an influenza-like illness (dengue fever; DF) to the life-threatening dengue hemorrhagic fever (DHF)/dengue shock syndrome (DSS). In recent decades, the incidence of dengue infection has increased around the world and has become a major international public health concern. The disease is now endemic in more than 100 tropical and sub-tropical countries. The World Health Organization (WHO)

estimates that there may be 50 million dengue infections worldwide every year.^{1,2}

Infection with one serotype of DENV provides lifelong immunity to that serotype, but results only in partial and transient protection against subsequent infection by the other three serotypes. It is possible for a person to be infected as many as four times, once with each serotype. It is well documented that sequential infection with different DENV serotypes increases the risk of developing DHF. Ninety percent of DHF infections occur in children less than 15 years of age. There is currently no specific treatment for DENV infection, although several potential vaccines are in development; therefore, the only method of preventing DENV transmission is vector (mosquito) control.^{1,3}

Early clinical features of dengue infection are variable among patients, and initial symptoms are often non-specific; therefore, specific laboratory tests are necessary for an accurate diagnosis.^{7,8}

According to the US Centers for Disease Control and Prevention (CDC) and the WHO dengue guidelines, the clinical features of DF and DHF are sudden onset of fever, severe headache, myalgias and arthralgias, leucopenia, thrombocytopenia, and hemorrhagic manifestations.⁸ It occasionally produces shock and haemorrhage, leading to death. Classic DF symptoms include fever, headache, retro-orbital pain, myalgias and arthralgias nausea, vomiting, and often a rash. Some DF patients develop the more serious form of the disease DHF with symptoms that include a decline in fever and presentation of hemorrhagic manifestations, such as microscopic hematuria, bleeding gums, epistaxis, hematemesis, malena, and ecchymosis. DHF patients develop thrombocytopenia and hemoconcentration; the latter is due to an increase in the concentration of blood cells resulting from the leakage of plasma from the bloodstream.

These patients may progress into DSS, which can lead to profound shock and death if not treated. Advance clinical symptoms of DSS include severe abdominal pain, protracted vomiting, and a notable change in temperature from fever to hypothermia.²

In this study, we analyzed the variation in clinical features of DENV-infected patients at Maharaja Agarsan Medical College and Hospital (MAMC). The clinical presentations were also compared with the US CDC definition.

METHODS

Patients diagnosed with dengue viral infection (n= 188, 63 females and 125 males), aged 6 years to 70 years old at MAMC from September 2015 to December 2015, were included in the study.

All the patients were from the neighbouring catchment area. All patients were admitted and discharged within a period of 3-7 days. All patients received IV fluids and monitored. Few patients required platelet transfusion. All patients survived. No patient went into dengue hemorrhagic fever or dengue shock syndrome.

Laboratory profile

All patients were tested for NS1 ELISA and were positive.

Haematological profile

Haematological parameters evaluated were platelet count, prothrombin time (PT), partial thromboplastin time (PTT), Hb and haematocrit (HCT) levels, complete blood

count (CBC), and white blood cell count (WBC). Blood glucose, urea/creatinine and LFT, X ray chest, ECG were done for all patients as baseline investigations Among the studied patients, 4 were diabetic and 1 showed pleural effusion. USG examination showed acalculary cholecystitis in 2, splenomegaly in 2 and ascitis in 1 patient.

RESULTS

Seasonal distribution

The first case of DENV infection detected in September 2015. Total number of cases seen in September were 47, 63 in October, 59 in November and 19 in December. The peak was seen in October and declined in December.

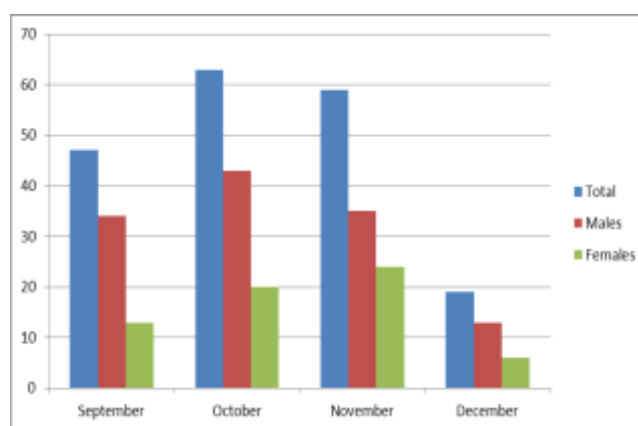


Figure 1: Distribution of patients attending hospital in 4 months with male female ratio.

Table 1: Clinical and laboratory profile of dengue patients admitted at MAMC.

Clinical feature observed	Number of patients	Percentage
Fever, myalgia	188	100
Nose bleeding	2	1.06
Ascitis	1	0.53
Acalculary cholecystitis	2	1.06
Pleural effusion with ascitis	1	0.53
Abdominal pain	2	1.06
Splenomegaly	2	1.06
Lymphadenopathy	2	1.06
LFT (deranged)	34	18.08
Platelets	188	100

Haematological profile

Thrombocytopenia (<100,000 platelets/ μ L) was observed in 100% of patients. The lowest count was recorded as 5000 and highest was 70000.

DISCUSSION

Seasonal distribution

Dengue fever usually commences from mid-June and then there is a surge in September and ends in December. But this time cases were seen from September to December and no cases were seen in June, July and August. Probably this is due to increased temperature (global warming) that cases are seen in December.

The pathogenesis of DENV is poorly understood. A complex interaction between immuno-pathologic, viral, and human genetic factors results in a varied

DENV disease outcome, which may explain the varied range of clinical presentations observed in this retrospective analysis. A possible reason for the significant differences seen in the clinical expression of the disease may be due to infection with different DENV serotypes and the possibility of concurrent infections with more than one serotype. Co-circulation of multiple DENV serotypes has been reported from many parts of the world, including India during an outbreak of DHF/DSS in 2006. Co-circulation of multiple DENV serotypes would result in an increased risk of concurrent infections.^{11,12}

There is, however, limited documentation describing concurrent infections with more than one serotype in the same individual.^{13,14} Furthermore, as already alluded to, sequential infection with more than one serotype is thought to be a major factor for the emergence of DHF.¹ Both primary and secondary infection by any of the four DENV serotypes can cause DF and DHF; however, virus virulence is not the only factor to explain differences in host susceptibility to the disease and disease severity. Host immune response variations have been associated with polymorphism in the human genome, which may help explain why some patients develop end-stage complications in dengue disease and others only experience a mild form of the disease.¹⁷ In another study of children with DENV infection, host genetic differences were shown to affect the immune response and consequently, influence disease outcome.¹⁸

Dengue infection can have potentially fatal consequences, and to date, vector control methods to prevent the spread of the virus have been unsuccessful.¹⁹ Although there are promising vaccine candidates in development, further studies are required for a greater understanding of the humoral immune responses to DENV infection and disease pathogenesis.²⁰

CONCLUSION

It was observed that significant differences in the clinical presentation of DENV infection. Dengue viral infection is a complicated disease and many factors may be attributed to the differences seen, such as infection with different

serotypes or infection with more than one serotype, either sequentially or concurrently. Differences in host genetics and immune responses may also play a role in the severity of infection.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. WHO Fact sheet No 117: Dengue and dengue haemorrhagic fever. (2008). Available: <http://www.who.int/mediacentre/factsheets/fs117/e/>.
2. Dengue and Dengue Hemorrhagic Fever: Information for Health Care Practitioners - CDC Division of Vector-Borne Infectious Diseases. Available: <http://www.cdc.gov/ncidod/dvbid/dengue/dengue-hcp.htm>.
3. Malavige GN, Fernando S, Fernando DJ, Seneviratne SL. Dengue viral infections. *Postgrad Med J.* 2004; 80:588-601.
4. Khan NA, Azhar EI, El-Fiky S, Madani HH, Abuljadial MA, Ashshi AM, Turkistani AM, Hamouh EA. Clinical profile and outcome of hospitalized patients during first outbreak of dengue in Makkah, Saudi Arabia. *Acta Trop.* 2008;105:39-44.
5. Central Department of Statistics & Information, Kingdom of Saudi Arabia. Available: <http://www.cdsi.gov.sa/showproductstandard.aspx?id=26&pid=1005>.
6. Ramos MM, Tomashek KM, Arguello DF, Luxemburger C, Quiñones L, Lang J, Muñoz-Jordan JL. Early clinical features of dengue infection in Puerto Rico. *Trans R Soc Trop Med Hyg.* 2009;103(9):878-84.
7. de Oliveira SA, Bastos Camacho LA, Fernandes Bruno L, de Gusmão RC, de Medeiros Pereira AC, Coca Velarde LG, Mendonça Siqueira M. Acute arthropathy in patients with rash diseases: a comparative study. *Clin Rheumatol.* 2009;28(9):1067-71.
8. WHO (1997) Dengue haemorrhagic fever: diagnosis, treatment, prevention and control, 2nd edition. Geneva: World Health Organization.
9. Griffais R, Andre PM, Thibon M K-tuple. Frequency in the human genome and polymerase chain reaction. *Nucleic Acid Res.* 1991;19:3887-91.
10. Ayyub M, Khazindar AM, Lubbad EH, Barlas S, Alfi AY, Al-Ukayli S. Characteristics of dengue fever in a large public hospital, Jeddah, Saudi Arabia. *J Ayub Med Coll Abbottabad.* 2006;18:9-13.
11. Coffey LL, Mertens E, Brehin AC, Fernandez-Garcia MD, Amara A, Després P, Sakuntabhai A. Human genetic determinants of dengue virus susceptibility. *Microbes Infect.* 2009;11:143-56.

12. Balmaseda A, Hammond SN, Pérez L, Tellez Y, Saborío SI, Mercado JC, Cuadra R, Rocha J, Pérez MA, Silva S, Rocha C, Harris E. Serotype-specific differences in clinical manifestations of dengue. *Am J Trop Med Hyg.* 2006;74:449-56.
13. Bharaj P, Chahar HS, Pandey A, Diddi K, Dar L, Guleria R. Concurrent infections by all four dengue virus serotypes during an outbreak of dengue in 2006 in Delhi, India. *Vírol J.* 2008;5:1.
14. Loroño-Pino MA, Cropp CB, Farfán JA, Vorndam AV, Rodríguez-Angulo EM, Rosado-Paredes EP, Flores-Flores LF, Beaty BJ, Gubler DJ. Common occurrence of concurrent infections by multiple dengue virus serotypes. *Am J Trop Med Hyg.* 1999;61:725-30.
15. Zaki A, Perera D, Jahan SS, Cardoso MJ. Phylogeny of dengue viruses circulating in Jeddah, Saudi Arabia: 1994 to 2006. *Trop Med Int Health.* 2008 13:584-92.
16. Wilder-Smith A, Gubler DJ. Geographic expansion of dengue: the impact of international travel. *Med Clin North Am.* 2008; 92:1377-90.
17. Chaturvedi U, Nagar R, Shrivastava R. Dengue and dengue haemorrhagic fever: implications of host genetics. *FEMS Immunol Med Microbiol.* 2006;47: 155-66.
18. Long HT, Hibberd ML, Hien TT, Dung NM, Van Ngoc T, Farrar J, Wills B, Simmons CP. Patterns of gene transcript abundance in the blood of children with severe or uncomplicated dengue highlight differences in disease evolution and host response to dengue virus infection. *J Infect Dis.* 2009;199:537-46.
19. Swaminathan S, Khanna N. Dengue: recent advances in biology and current status of translational research. *Curr Mol Med.* 2009; 9:152-73.
20. Crill WD, Hughes HR, Delorey MJ, Chang GJ. Humoral immune responses of dengue fever patients using epitope-specific serotype-2 virus-like particle antigens. *PLoS ONE* 2009;4(4):e4991.

Cite this article as: Shah MY, Naqash MM, Goel RK, Galhan D, Kumar S, Chhabra V, et al. Clinical profile of dengue fever infection in patients admitted in tertiary care centre Agroha, Hisar, Haryana, India. *Int J Res Med Sci* 2016;4:2146-9.