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General Medicine

CLINICAL PROFILE OF DENGUE INFECTION IN A TERTIARY CARE TEACHING HOSPITAL OF WESTERN UP (NORTH INDIA)

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ABSTRACT

Introduction: Dengue is a rapidly emerging global health scourge. It has variable clinical presentation with many atypical presentations, thus posing a diagnostic challenge for the physician.

Objective: To study the clinical profile of dengue infection during an outbreak in August-November, 2017 in a tertiary care teaching hospital of western UP.

Material and methods: A prospective, observational, single centre study was carried out on patients of dengue fever who were admitted to the medicine ward of Sharda School of Medical Sciences and Research Hospital.

Results: 110 patients were diagnosed as dengue fever based on the presence of NS1 antigen, dengue IgM in blood samples. The common signs and symptoms were fever, headache, bodyache, backache, retro-orbital pain and abdominal pain in 100%, 96.3%, 91.8%, 65.4%, 50%, 45.4% respectively. Final categorization of these patients was DF(71.8%), DHF(25.4%), DSS(1.8%), EDS(3.6%). Atypical presentations included encephalopathy, ARDS and acalculous cholecystitis.

KEYWORDS : Dengue, acalculous cholecystitis

INTRODUCTION

Dengue is the most rapidly spreading arboviral disease in the tropical countries. Incidence of dengue fever has increased 30 fold in the last 50 years.[1] It is caused by one of the four serotypes called DENV 1,2,3 & 4.[2] The principal vector mosquito for the disease is *Aedes aegypti.* A majority of regions in India such as Delhi, Haryana, Rajasthan, Gujarat, Karnataka, Tamil Nadu & West Bengal are endemic for Dengue.[3] Dengue fever begins typically after an incubation period of 4-7 days.[4] The patients experience sudden onset fever, frontal headache, retro-orbital pain & severe myalgia – "break bone fever".[5] Additional signs & symptoms include anorexia, nausea or vomiting and marked cutaneous hypersensitivity.[5] Near the time of defervescence, a maculopapular rash begins on the trunk and spreads to the extremities and the face. Epistaxis and scattered petechiae are often noted in uncomplicated dengue,[5] and preexisting lesions like peptic ulcers may bleed during the acute illness. Laboratory findings include leukopenia, thrombocytopenia, and in many cases, elevations of liver enzymes.

An increasing number of cases are being reported with atypical presentations. Rare manifestations of the disease are being reported as the awareness about the disease is increasing. This study was conducted during a dengue outbreak in 2017 in a tertiary care hospital in western U.P to study the clinical profile of the disease.

Table 1.

DF/DHF	Grade	Signs & Symptoms	Laboratory
DF	With or without hemorrhagic manifestations	Fever with two of the following: (i) Headache. (ii) Retro-orbital pain. (iii) Myalgia. (iv) Arthralgia/bone pain. (v) Rash. (vi) Hemorrhagic manifestations. (vii) No evidence of plasma leakage.	Leucopenia (WBC ≤5000cells/mm 3). (i) Thrombocytopenia (platelet count <150 000cells/mm 3). (ii) Rising hematocrit (5%–10%). (iii) No evidence of plasma loss.
DHF	I	Fever and hemorrhagic manifestation (positive tourniquet test) and evidence of plasma leakage.	Thrombocytopenia <100 000cells/mm 3 ; HCT rise ≥20%.
DHF	II	As in Grade I plus spontaneous bleeding.	Thrombocytopenia <100 000cells/mm 3 ; HCT rise ≥20%.
DHF	III	As in Grade I or II plus circulatory failure (weak pulse, narrow pulse pressure (≤20 mmHg), hypotension, restlessness).	Thrombocytopenia <100 000cells/mm 3 ; HCT rise ≥20%.
DHF	IV	As in Grade III plus profound shock with undetectable BP and pulse.	Thrombocytopenia < 100 000cells/mm 3 ; HCT rise ≥20%

MATERIALS AND METHODS

This study was a prospective, hospital-based observational study conducted at Sharda School of Medical Sciences and Research Hospital, from August 2017 to November 2017. 110 patients suffering from dengue fever during this period were enrolled. All patients who were admitted with complaint of fever and were found positive for either NS1 antigen or dengue IgM antibodies were included in the study. Informed consent and detailed history were taken and careful clinical examination was performed. Besides routine biochemical and hematological investigations [hemoglobin, total leucocyte count (TLC) and differential leucocyte count (DLC), platelet count, hematocrit (HCT), liver function tests (LFT), blood urea, and serum creatinine], malarial antigen, Slide test for malarial parasite, IgM antibodies and Widal test for typhoid, and X-ray chest and ultrasonography (USG) of abdomen were also done in all patients. All patients were classified according to WHO guidelines (Table 1). Patients having gastrointestinal, cardiac, neurological, renal and other nonspecific manifestations were grouped in expanded dengue syndrome (EDS) category.

Table 2 : Distribution of the patient according to age & gender

Age (Years)	Male	Female	Total
18-30	42	26	68(61.81%)
31-40	12	6	18(16.36%)
41-50	8	6	14(12.72%)
51-60	8	2	10(9.09%)
Total	70(64.54%)	40(35.45%)	

Table 3 : Distribution of the patients according to presenting symptoms

Symptoms	N	%
Fever	110	100.00
Headache	106	96.36
Myalgia	101	91.81
Retro – orbital Pain	55	50.00
Abdominal pain	50	45.45
Skin rash	48	43.63
Nausea / Vomiting	46	41.81
Bleed(Any hemorrhagic manifestation)	28	25.45
Breathlessness	20	18.18
Diarrhoea	6	5.45
Itching	22	20
Other	23	15.23

Table 4 : Distribution of the patients according to positive findings

Findings	Positive cases	%
Pleural effusion	25	22.72
Gall Bladder Edema	40	36.36
Hepatomegaly	32	29.09
Splenomegaly	30	27.27
Ascites	12	10.90

Table 5 : Laboratory Parameters

Laboratory Parameters	N	%
Thrombocytopenia (<50000/cumm)	58	52.72
Leucopenia (<4000/cumm)	32	29.09
SGPT (>55IU/L)	68	61.81
SGOT (>45IU/L)	65	59.09
S. Total Billirubin (>2mg/dl)	4	3.63
Raised Hematocrit (>45%)	28	25.45
Malarial Antigen	1	0.09
Slide test for Malaria	1	0.09
Widal Test	3	2.72

Table 6: Categorization of patients

Categorisation	Frequency	%
DF	79	71.8%
DHF	28	25.45%
DSS	2	1.80%
EDS	4	3.6%
Total	110	100%

RESULTS

Out of the 186 patients admitted with clinical suspicion of dengue in Sharda School of Medical Sciences and Research Hospital from August 2017 to November 2017, 110 patients had confirmed laboratory diagnosis of dengue. Among these 110 patients, 71 were male and 39 were female. Maximum patients were in 18–30 years age group (61.81%).

All the patient of dengue fever had fever as one of the presenting symptoms. Headache was also found as presenting symptom in majority of patients (96.36%), followed by myalgia(91.81%), retro-orbital pain(50%), pain abdomen(45.45%) and skin rash(43.63%)(Table2) .On physical examination right hypochondrium/ epigastric tenderness was present in 44(40%), Murphy's sign was positive in 20(12.72%). Hepatomegaly and splenomegaly was noted in 15(13.63%) and 12(10.90%) respectively.

On abdominal ultrasound 40 patients (36.36%) with abdominal pain had edematous gall bladder without gall stones. Diagnosis of acute acalculous cholecystitis was made and all patients responded well to supportive therapy.

On laboratory investigations, thrombocytopenia (platelet count <50000/cumm) was present in 58(52.72%) and leucopenia in 32(29.09%) patients. Serum SGOT and SGPT were found to be raised in 65(61.81%) and 65(59.09%) respectively. S. Total bilirubin > 2 mg/dL was found only in 4 patients, all other causes of jaundice were ruled out in these and all patients recovered without any hepatic sequelae.

28 patients (25.5%) had hemorrhagic manifestations in form of petechiae, epistaxis, hematuria, melena and menorrhagia. 15 patients required platelet transfusion an d 4 both blood and platelet transfusion. Intravenous fluid therapy was given according to WHO guidelines.

Three patients (2.7%) required ICU care because of DSS, ARDS, and encephalopathy. One patient of DSS had come with acute respiratory distress in shock , required inotropic support and mechanical ventilation. She had pleural effusion as well as ascites. Unfortunately she failed to respond and died on the 3rd day of admission.

One patient was admitted with history of fever and altered sensorium. All his investigations were normal except positive NS1 antigen, very low platelet and raised SGOT(x3 times) and SGPT(x4 times). CSF was positive for mild lymphocytosis and raised protein. MRI brain was inconclusive. Diagnosis of dengue encephalopathy was made and patient recieved 5 platelet transfusions and supportive care in ICU for 4 days. He recovered in the next 2 weeks without any neurological sequelae.

Out of 110 patients, two had coinfection with malaria (confirmed by Slide test as well as malaria antigen positive for *Plasmodium vivax* malarial parasite) and three had typhoid (confirmed with Widal test) as shown in Table 3.

DISCUSSION

The present study was done with the aim to describe the clinical manifestations, laboratory features and outcomes of dengue infection in adult patients with atypical presentations if any. Majority of the cases were in the age group of 18-30 years with male preponderance (64.54%).These findings coincide well with the previous studies as well as current literature.[6,7]

Typically, DF presents as a self limiting disease characterized by fever associated with symptoms such as headache, nausea, vomiting, arthralgia, myalgia, retro-orbital pain and/or rash. The atypical manifestations include acalculous cholecystitis, encephalopathy, myocarditis, isolated cranial nerve palsies. In our study the most common presenting symptom was fever (100%) followed by headache (96.3%), myalgia(91.8%) and retro orbital pain(50%). These findings are similar to a study conducted by Itoda et al[8] where 90% patients presented with headache whereas in a study conducted by Awasthi et al it was reported that only 9% of the patients had headache as their complaint.[9]

Haemorrhagic manifestations is one of the common complications of DF due to throbocytopenia. In our study we found that 25.45% of the patients had bleeding manifestations in the form of bleeding gums, haematemesis, haemoptysis, malena, subconjunctival haemorrhage while 52.72% of the patients had a platelet count below 50,000/cumm. In a study by Khan et al only 5% patients had bleeding episodes while 40% had thrombocytopenia.[10] In a study by Mandal et al 13.51% patients had haemorrhagic manifestations with 37.8% of the patients with a platelet count of <50,000/cumm.[11] A study conducted in Sudan by Ageep AK et al haemorrhagic manifestations were there in 93% cases with thrombocytopenia in 88%.[12]

Ascites and pleural effusion are being increasingly reported in DF with the help of imaging modalities like ultrasound. Third space fluid collection in the form of ascites and pleural effusion was noted in 10.9% and 22.72% of the patients respectively. Mandal et al. reported that ascites was present in 8.1% and pleural effusion in 18.9% of the cases. In a study conucted in Bangladesh by Mia et al. 42% had pleural effusion and 41% patients had developed ascites.[13]

Dengue fever can cause hepatic injury and transaminase elevation similar to viral hepatitis. It was found that 59.09% patients had raised SGOT while 61.81% patients had raised SGPT. A study done by Silva EM et al in Brazil found an interacting partner between NS1 proteins and liver proteins in the causation of hepatic dysfunction in dengue fever.[14] In a study in Taiwan by Kuo CH et al raised SGOT & SGPT was found in 93.3% and 82.2% of the cases respectively.[15]

45.4% of the patients presented with pain abdomen alongwith fever. Amongst these a significantly high number (36.3%) were diagnosed with acute acalculous cholecystitis. This correlates well with the findings of Prasad A et al and Chandey M et al who found the incidence 37.5% and 38.8% respectively.[16,17] The exact mechanism of acalculous cholecystitis is unknown. It could be due to viral invasion of the walls of gall bladder causing microangiopathic injuries and increased vascular permeability leading to protein rich plasma leakage resulting in the edema.[2] The course of acalculous cholecystitis is self limiting but sometimes it progresses to ischemic gangrene & perforation .

Leucopenia seen in DF may be due to viral induced inhibition or destruction of the myeloid progenitor cells. In our study, 29.09% of the patients had leucocyte count below 4000/cumm which is similar to the study by Mandal et al with leucopenia in 29.73%. However 71% cases were detected with leucopenia in a study by Itoda et al while Ageep AK et al reported it in 90% of the cases.

CONCLUSION

Dengue has become a challenging disease with multisystemic, varied and atypical presentations. While some manifestations are known several atypical features have also been noted. Awareness of these atypical presentations may go a long way in early recognition, diagnosis, prompt intervention & accurate treatment. Therefore a continuous epidemiological surveillance and timely interventions are necessary to identify the cases so that outbreak, complications and mortality can be minimized.

Competing interests

The authors declare that there is no conflict of interests regarding publication of this paper.

REFERENCES

1. Dengue-treatment, prevention and control. WHO. 2009
2. S. Gulati and A. Maheshwari, "Atypical manifestations of dengue," Tropical Medicine and International Health, vol. 12, no. 9, pp. 1087–95, 2007.
3. National Vector Borne Disease Control Programme. Annual Report 2014-15. Ministry of Health and Family Welfare. Government ofIndia.
4. WHO, "Comprehensive guidelines for prevention and control of dengue and dengue hemorrhagic fever," World Health Organization, Regional Office for South-East Asia, http://apps .searo.who.int/pds docs/B4751.pdfua=1.
5. Kuhn JH, Peters CJ. Arthropod-borne and rodent-borne virusinfections. In: Kasper DL, Fauci AS, Hauser SL, Longo DL,Jameson JL, Loscalzo J, editors. Harrison's Principles of InternalMedicine. 19th ed. Volume 2. McGraw-Hill Education; Columbus,OH, USA: 2015. pp. 1304–23.
6. Chandralekha, Gupta P, Trikha A. The north Indian dengueoutbreak 2006: a retrospective analysis of intensive care unitsadmissions in a tertiary care hospital. Trans R Soc Trop Med Hyg2008;102:143–7.
7. Kashinkunti MD, Shiddappa, Dhananjaya M. A study of clinical profile of dengue fever in a tertiary care teaching hospital. Sch J App Med Sci 2013; 1(4):280–2.
8. I. Itoda, G. Masuda, A. Suganuma et al., "Clinical features of 62 imported cases of dengue fever in Japan," American Journal of Tropical Medicine and Hygiene, vol. 75, no. 3, pp. 470–4, 2006.
9. S. Awasthi, V. K. Singh, S. Kumar, A. Kumar, and S. Dutta, "The changing clinical spectrum of Dengue fever in the 2009 epidemic in north India: a tertiary teaching hospital based study," Journal of Clinical and Diagnostic Research, vol. 6, no. 6, pp. 999–1002, 2012.
10. A. H. Khan, A. S. Hayat, N. Masood, N. M. Solangi, and T. Z. Shaikh, "Frequency and clinical presentation of dengue fever at tertiary care hospital of Hyderabad/Jamshoro," Journal of the Liaquat University of Medical and Health Sciences, vol. 9, no. 2, pp. 88–94, 2010.
11. S. K. Mandal, J. Ganguly, K. Sil et al., "Clinical profiles of dengue fever in a teaching hospital of eastern India," National Journal of Medical Research, vol. 3, no. 2, pp. 173–6, 2013.
12. Ageep AK, Malik AA, Elkarsani MS. Clinical presentations andlaboratory findings in suspected cases of dengue virus. SaudiMed J 2006 Nov;27(11):1711-3.
13. Clinical and Sonographic Evaluation of Dengue Fever in Bangladesh: A Study of 100 Cases. Mia MW, NurullahAM, Hossain A, Haque MM Dinajpur Med Col J 2010 Jan; 3(1)
14. Silva EM, Conde JN, Allonso D, Nogueira ML, Mohana-BorgesR. Mapping the Interactions of Dengue Virus NS1 Protein withHuman Liver Proteins Using a Yeast Two-Hybrid System:Identification of C1q as an Interacting Partner. PLoS One.2013;8(3):e57514
15. Kou CH, Tai DI, Chang-Chien CS, Lan CK, Chiou SS, LiawYF. The liver biochemical tests and Dengue fever. Am J TropMed Hyg 1992; 47: 265-70.
16. Prasad A, Yasmeen M, Prasad SG, Taj MR, Saxena G. Study of acute acalculous cholecystitis as an atypical manifestation in dengue fever. Int J Med Sci Public Health 2015;4:1231-1234.
17. Chandey M, Kaur H, Kaur S. Acute acalculous cholecystitis in dengue fever patients. Int J Adv Med 2017;4:375-7.