OPEN ACCESS
Saudi Journal of Medical and Pharmaceutical Sciences
Abbreviated Key Title: Saudi J Med Pharm Sci
ISSN 2413-4929 (Print) |ISSN 2413-4910 (Online)
Scholars Middle East Publishers, Dubai, United Arab Emirates
Journal homepage: http://scholarsmepub.com/sjmps/

Case Report

Case of Classical Dengue with Pleural Effusion & Ascites

Dr. Gule Tajkia[1*], Dr. Fabia Hannan Mone[2]
[1]Assistant Professor, Department of Pediatrics, Anwer Khan Modern Medical College, House 17, Road 8 Dhanmondi R/A, Dhaka 1205, Bangladesh
[2]Medical Officer, Department of Pediatrics, Anwer Khan Modern Medical College Hospital, House 17, Road 8 Dhanmondi R/A, Dhaka 1205, Bangladesh

***Corresponding author:** Dr. Gule Tajkia
| **Received:** 20.04.2019 | **Accepted:** 26.04.2019 | **Published:** 30.04.2019
DOI:10.21276/sjmps.2019.5.4.12

Abstract

Dengue virus is the most important mosquito-borne viral disease in the world. Co-circulation of the four types of dengue viruses and expansion of dengue epidemic give rise to infection enhancement and a big expansion of clinical aspects of the disease. Infection with dengue virus (DENV) causes diseases ranging widely in severity, from self-limited dengue fever to life-threatening dengue hemorrhagic fever and dengue shock syndrome. But atypical manifestations of dengue fever are rising day by day but they may be under reported. Here in we report a case of 6 years old girl presented with sudden onset of abdominal pain and was diagnosed as classical dengue with ascites and pleural effusion. Pleural effusion and ascites are evidence of plasma leakage which appear in patients of Dengue hemorrhagic fever but not present in Classical Dengue or Dengue fever. Classical Dengue is a common disease in Bangladesh but there have been no reports of Dengue fever or Classical Dengue in association with pleural effusion and ascites.

Keywords: Classical Dengue, Pleural Effusion & Ascites.

INTRODUCTION

Dengue, a mosquito-borne viral illness, an important cause of morbidity and some mortality in many countries, mostly in Asia and Latin America, and is continuing to expand globally. Around 390 million infections occur each year with approximately 500,000 hospital admissions with potentially life-threatening forms of the disease, dengue haemorrhagic fever (DHF) and dengue shock syndrome [1]. Approximately 12,000 deaths, mostly among children, occur worldwide every year [1]. An estimated 50.0% of the global population are at risk of acquiring dengue [1] and over half reside in the World Health Organization's South-East Asia Region (SEAR) [2]. In Bangladesh, dengue is known from its first reporting in1964 [3] followed by its major outbreak on June 2000, when >5,000 hospitalized cases of DF and DHF in Dhaka and other major cities of Bangladesh were recorded [4, 5]. Since then a substantial number of dengue cases are reporting every year [6].

The dengue virus is a RNA virus belonging to the Flaviviridae family and transmitted to humans mainly by Aedis aegypti and Aedis albopictus. There are four dengue viruses (DENV-1, DENV-2, DENV-3, DENV-4) transmitted mainly in tropical countries and virulence seems to be quite variable among them [7]. Classically, infection may be clinically asymptomatic or results in undifferentiated fever, dengue fever (DF), dengue hemorrhagic fever (DHF) or dengue shock syndrome (DSS). However, the co-circulation of the four types of dengue viruses and expansion of dengue epidemic gave rise to infection enhancement and, consequently, not only significant number of severe forms – including dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS) but also a an increasing number of "unusual complications" [8]. The major pathophysiological hallmark that distinguishes DHF/DSS from DF is plasma leakage as a result of increased vascular permeability. That feature is observed instead of hemorrhagic events with secondary hypoalbuminemia or hypoproteinemia, accompanied by thrombocytopenia, altered haemostasis, and, usually, evidence of liver damage [9].

Case Report

A 6 years old girl was admitted to our hospital in October 2017 with 4 days history of fever, myalgia, headache and rashes over the whole body followed by an afebrile period of 3 days. On the seventh day of symptoms she developed abdominal pain and distension, cough and respiratory difficulty. She had no history of bleeding and her bowel, bladder habits were normal. At admission, 7 days after the beginning of her

first symptoms, she was ill-looking, lethargic, afebrile, pulse 85 beats/minute, respiratory rate 34 breaths/minute and blood pressure of 100/60 mmHg. Clinical examination revealed right prominent pleural effusion, ascites, epigastric tenderness with tender hepatomegaly. There were no signs of active bleeding and the tourniquet test was negative.

Table-1: A summary of investigation results of the patient during the illness

Date	**15/10/2017**	**16/10/17**	**19/10/17**
Platelet count ($X10^9$/L)	53	60	185
White Cell Count ($X10^9$/L)	10.70	6.90	7.33
Packed cell volume (%)	46	36	33.20
Haemoglobin(gm/dl)	15.40	11.90	12
ESR	15	10	23
S. Creatinine(mg/dl)	0.5		
S.Total Protein(gm/L)	53		
S.Albumin(gm/L)	28		
S.Globulin(gm/L)	25		
A:G Ratio	1.12:1		
S.Bilirubin(mg/dl)	0.3		
S.Electrolytes(Na+)mmol/L	136		
S.Electrolytes(K+)mmol/L	4.9		
S.Electrolytes(Cl-)mmol/L	103		
S.Electrolytes(TCO2)mmol/L	24		
HBsAg	Negative		
Urine R/E: Pus Cell(/HPF)	4-5		

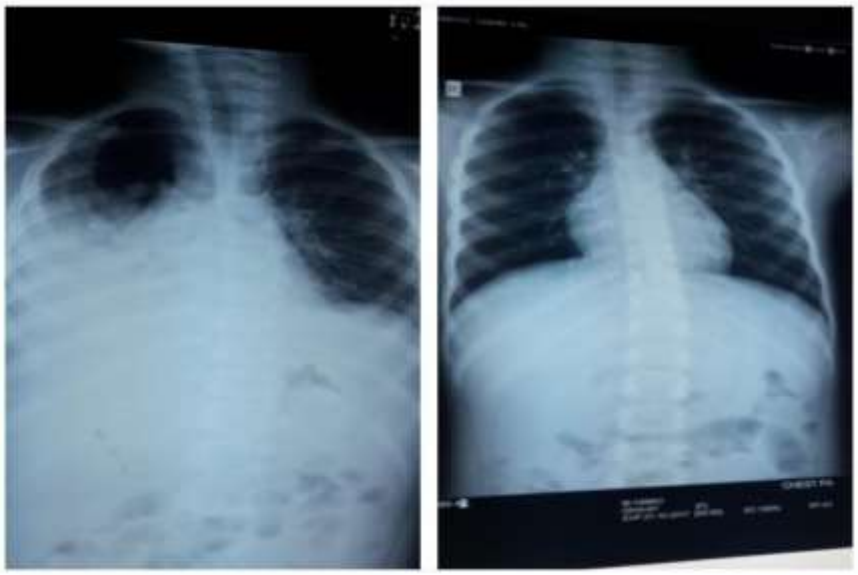

Fig-1: Chest X-ray at admission (A) and 2 weeks after discharge (B)

Laboratory results at admission showed raised hematocrit, thrombocytopenia and hypoproteinemia, hypoalbuminemia. There was no proteinuria or haematuria (Table-1). Extensive right pleural effusion was observed by chest X- ray (Fig-1). Abdominal ultrasound revealed hepatomegaly, and ascites but no lymphadenopathy was seen.

Dengue serological tests were performed and both dengue virus IgM and IgG were positive. These serological findings were strongly suggestive of recent dengue virus infection. Before admission on the 4th day of disease non-structural protein 1 (NS1) was done which was also positive.

Treatment was symptomatic with administration of crystalloids, fresh frozen plasma. Because a secondary bacterial infection could not be ruled out she also received antibiotics. Her clinical condition and laboratory tests improved during the second week and on discharge her clinical symptoms had almost disappeared. At the first follow-up visit, she had complete recovery and laboratory tests had normalized.

DISCUSSION

We have reported the case of a child presenting with classical dengue with features of serositis without any evidence of hemorrhage. Dengue virus infections may be asymptomatic or symptomatic. In symptomatic dengue infection, after an incubation period of 3 to 7 days, symptoms start suddenly and follow three phases — an initial febrile phase, a critical phase around the time of defervescence, and a spontaneous recovery phase. The initial phase is typically characterized by high temperature (≥38.5°C) accompanied by headache, vomiting, myalgia, and joint pain, sometimes with a transient macular rash. This phase lasts for 3 to 7 days, after which most patients recover without complications [10]. In a small proportion of patients, typically in children and young adults, a systemic vascular leak syndrome becomes apparent around the time of defervescence, evidenced by increasing hemoconcentration, hypoproteinemia, pleural effusions, and ascites. Initially, physiological compensatory mechanisms are up-regulated in an attempt to maintain adequate circulation to critical organs, resulting in narrowing of the pulse pressure when loss of plasma volume becomes critical. If the pulse pressure narrows to 20 mm Hg or less, accompanied by signs of peripheral vascular collapse, dengue shock syndrome is diagnosed and urgent, although careful, resuscitation is required. Systolic pressure may remain normal or even elevated at this time, and the patient may appear deceptively well, but once hypotension develops, systolic pressure decreases rapidly and irreversible shock and death may follow despite aggressive attempts at resuscitation. Hemorrhagic manifestations are most common during this critical period. In children, clinically significant bleeding occurs only rarely, usually in association with profound and prolonged shock. However, major skin bleeding, mucosal bleeding (gastrointestinal or vaginal), or both may occur in adults with no obvious precipitating factors and only minor plasma leakage [11].

Dengue may cause mild febrile illness to severe and fatal disease [12]. The severity of dengue infection is usually correlated with the size of the dengue-infected cell mass as shown many times by high titers of circulating virus in early illness blood samples or by persisting high concentrations in blood of dengue viral RNA and dengue non-structural protein 1 (NS1) [13]. This protein also parallels cellular dengue infection. High levels of markers of immune activation and severity of dengue infection have led to the hypothesis of an autoimmune response, mainly as a result of cross-reactivity between anti-NS1 to host proteins, endothelial cells and platelets [14, 15], mediating complement activation [9] and triggering plasma leakage. Although no specific pathway has been identified linking known immunopathogenic events with definitive effects on microvascular permeability, thrombo-regulatory mechanisms, or both, preliminary data suggest that transient disruption in the function of the endothelial glycocalyx layer occurs [16, 17]. This layer functions as a molecular sieve, selectively restricting molecules within plasma according to their size, charge, and shape. Hypoalbuminemia and proteinuria are observed during dengue infection; proteins up to and including the size of albumin are preferentially lost; this is consistent with a small but crucial change in the filtration characteristics of the glycocalyx [18].

Patients were previously classified as having either dengue fever or dengue hemorrhagic fever, with the latter classified as grade 1, 2, 3, or 4. Over a number of years, there was increasing concern regarding the complexity and usefulness of this classification system. In particular, there was concern regarding the requirement that all four specific criteria (fever lasting 2 to 7 days, tendency to hemorrhage evidenced by a positive tourniquet test or spontaneous bleeding, a platelet count of less than 100×109 per liter, and evidence of a plasma leak based on changes in the hematocrit and pleural effusions) be met to support a diagnosis of dengue hemorrhagic fever — such that some patients with clinically severe disease were categorized inappropriately [19-21]. For example, the positive tourniquet test indicative of hemorrhagic manifestation does not significantly distinguish between DHF and DF. In addition, the incidences of major manifestations (hemorrhage, thrombocytopenia, and plasma leakage) observed in DHF patients span a large range [22]. WHO is currently re-evaluating the clinical case definition for dengue fever and DHF. Studies from different countries have reported life-threatening complications from dengue in the absence of one or more of the current criteria for DHF. Despite the name, the critical feature that distinguishes DHF from dengue fever is not hemorrhaging, but rather plasma leakage resulting from increased vascular permeability.

Dengue shock syndrome (DSS) is defined as any case that meets the four criteria for DHF and has evidence of circulatory failure manifested by (1) rapid, weak pulse and narrow pulse pressure (≤20 mmHg) or (2) hypotension for age, restlessness, and cold, clammy skin. Patients with dengue can rapidly progress into DSS. Fatality rates among patients with DSS can be 10% or higher but, with early recognition and treatment, can be less than 1% [1].

Our patient met the WHO criteria for DF but showed features of serositis, although hemorrhagic manifestations were not observed. During the transition from the febrile to the critical phase, between days 4 and 7 of the illness, it is crucial for the clinician to be aware of significant vascular leakage. These signs of impending deterioration include persistent vomiting, increasingly severe abdominal pain, tender hepatomegaly, a high or increasing hematocrit level,

lethargy or restlessness. Whatever may be the classification of patient DF or DHF it is important to focus on any deterioration of the patient in critical phase, as the patients with dengue can rapidly progress into DSS, which, if not treated correctly, can lead to severe complications and death.

CONCLUSION

Our Patient have classical dengue fever with pleural effusion and ascitis, hence we have shared our experience because early diagnosis of capillary leakage is important for adequate patient management with supportive measures and fluid therapy to prevent progression of dengue shock syndrome. As a human being we cannot stop the natural progression of disease but the severity of disease can be modify by early diagnosis and intervention.

REFERENCES

1. World Health Organization. (2012). Dengue and severe dengue. [Updated 2016 July; cited 2016 Augu.st 31]. Available from http://www.who.int/mediacentre/factsheets/fs117/en/
2. World Health Organization. (2011). Comprehensive guidelines for prevention and control of dengue and dengue haemorrhagic fever. Geneva.
3. Aziz, M. A., Gorham, J. R., & Gregg, M. B. (1967). Dacca fever. *an outbreak of dengue. Pak J Med Res*, *6*, 83-92.
4. Amin, M. M. M., Hussain, A. M. Z., Murshed, M., Chowdhury, I. A., Mannan, S., Chowdhury, S. A., & Banu, D. (1999). Searo-Diagnosis of Dengue Infections by Haemagglutination Inhibition Test (HI) in Suspected Cases in Chittagong, Bangladesh. *Dengue Bull*, 23:34-38
5. Yunus, B. (2000). Dengue outbreak 2000: the emerged issues, *Bangladesh Medical Journal*, 33:46-47.
6. Mahmood, B., & Mahmood, S. (2011). Emergence of Dengue in Bangladesh a major international public health concern in recent years. *Journal of Environmental Research and Management*, *2*(3), 35-41.
7. Guzmán, M. G., & Kouri, G. (2002). Dengue: an update. *The Lancet infectious diseases*, *2*(1), 33-42.
8. Halstead, S. B. (2007). Dengue. *Lancet*, 370:1644-52.
9. Green, S., & Rothman, A. (2006). Immunopathological mechanisms in dengue and dengue hemorrhagic fever. *Current opinion in infectious diseases*, *19*(5), 429-436.
10. Government of the People's Republic of Bangladesh. (2013). Ministry of health and Family Welfare. Directorate General of Health Services. National Guidelines on the Management of Dengue.
11. Cameron, P. S., Jeremy, J. F., Nguyen van V. C., & Bridget, W. (2012). Dengue Current concepts. *The New England Journal of Medicine*, 366:1423-32.
12. World Health Organization. (2009). Dengue: Guidelines for treatment, prevention and control. Geneva.
13. Vaughn, D. W., Green, S., Kalayanarooj, S., Innis, B. L., Nimmannitya, S., Suntayakorn, S., ... & Nisalak, A. (2000). Dengue viremia titer, antibody response pattern, and virus serotype correlate with disease severity. *The Journal of infectious diseases*, *181*(1), 2-9.
14. Lin, C. F., Lei, H. Y., Shiau, A. L., Liu, C. C., Liu, H. S., Yeh, T. M., ... & Lin, Y. S. (2003). Antibodies from dengue patient sera cross-react with endothelial cells and induce damage. *Journal of medical virology*, *69*(1), 82-90.
15. Lin, C. F., Wan, S. W., Cheng, H. J., Lei, H. Y., & Lin, Y. S. (2006). Autoimmune pathogenesis in dengue virus infection. *Viral immunology*, *19*(2), 127-132.
16. Michel, C. C., & Curry, F. E. (1999). Microvascular permeability. *Physiological reviews*, *79*(3), 703-761.
17. Levick, J. R., & Michel, C. C. (2010). Microvascular fluid exchange and the revised Starling principle. *Cardiovascular research*, *87*(2), 198-210.
18. Wills, B. A., Oragui, E. E., Dung, N. M., Loan, H. T., Chau, N. V., Farrar, J. J., & Levin, M. (2004). Size and charge characteristics of the protein leak in dengue shock syndrome. *Journal of Infectious Diseases*, *190*(4), 810-818.
19. Phuong, C. X. T., Nhan, N. T., Kneen, R., Thuy, P. T. T., Van Thien, C., Nga, N. T. T., ... & Wills, B. (2004). Clinical diagnosis and assessment of severity of confirmed dengue infections in Vietnamese children: is the World Health Organization classification system helpful?. *The American journal of tropical medicine and hygiene*, *70*(2), 172-179.
20. Deen, J. L., Harris, E., Wills, B., Balmaseda, A., Hammond, S. N., Rocha, C., ... & Farrar, J. J. (2006). The WHO dengue classification and case definitions: time for a reassessment. *The Lancet*, *368*(9530), 170-173.
21. Rigau-Pérez, J. G. (2006). Severe dengue: the need for new case definitions. *The Lancet infectious diseases*, *6*(5), 297-302.
22. Bandyopadhyay, S., Lum, L. C., & Kroeger, A. (2006). Classifying dengue: a review of the difficulties in using the WHO case classification for dengue haemorrhagic fever. *Tropical Medicine & International Health*, *11*(8), 1238-1255.