A Retrospective Analysis of Clinical Observations on the Development of Ascites in Dengue Patients in an Urban Dhaka Setting

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Abstract

Introduction The World Health Organization (WHO) has reported a substantial increase in the occurrence and severity of dengue outbreaks worldwide, estimating ~390 million cases each year.

Materials and Methods This retrospective research examined clinical data from 48 dengue patients with ascites from a single urban health care center in Dhaka, Bangladesh. Preexisting comorbidities such as hepatitis, diabetes, hypertension, and renal impairment were examined in the research to determine ascites-onset risk factors. Descriptive statistics summarized demographic and clinical data, and a *t*-test compared ascites onset in patients with and without comorbidities. Statistical significance was defined as p-value <0.05.

Results The study included patients aged 19 to 77 years, with a mean age of 38.5 years. Males constituted 60% of the sample, and females 40%. The average onset of ascites was 6.9 days after the initial dengue symptoms, with females experiencing slightly earlier onset (6.4 days) compared with males (6.7 days). Comorbidities were present in 58% of patients, with hepatitis being the most common (33%), followed by diabetes (12.5%). Patients with comorbidities had a significantly earlier onset of ascites (mean: 6.4 days) compared with those without (mean: 7.2 days; $p < 0.05$). The findings suggest that preexisting conditions, particularly hepatic and metabolic disorders, contribute to accelerated disease progression by exacerbating vascular permeability and immune dysfunction.

Keywords
- dengue
- ascites
- hepatomegaly
- organ failure

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Conclusion The 31 to 40 age group had the largest mean start time for ascites, and females developed it sooner. Previous investigations have linked severe dengue to liver dysfunction and vascular consequences. Hepatitis-related liver damage may limit protein synthesis, causing abdominal fluid leaks. Diabetes reduces immunological and vascular function, which may accelerate ascites. These results urge early detection and attentive monitoring of at-risk dengue patients with comorbidities. Early fluid and electrolyte control may avert organ failure or shock. Health care practitioners may improve dengue patient outcomes by early comorbidity detection.

Introduction

Dengue is a mosquito-borne viral infection caused by the dengue virus (DENV) which has become a major public health problem, especially in urban areas.[1] The situation now affects millions in both endemic and nonendemic regions, with a significant escalation attributed to increased population density, inadequate vector control measures, and rapid urbanization. The World Health Organization (WHO) has reported a substantial increase in the occurrence and severity of dengue outbreaks worldwide, estimating ~390 million cases each year.[2] In 2008, Southeast Asia and the Western Pacific represented 70% of the global dengue fever cases. The highest incidence rates were observed in India, Bangladesh, Sri Lanka, Myanmar, and Indonesia. The four distinct serotypes of the DENV have combined to cause dengue fever. Infection with any of these serotypes can lead to the disease, and the risk of severe symptoms increases when multiple serotypes are involved. Dengue fever may range from a minor case of the flu to a life-threatening case with multiple organ failure, shock syndrome, and hemorrhagic fever. In the early stages of dengue infection, patients typically experience symptoms such as fever, headache, retro-orbital pain, muscle and joint pain, and rash. Most cases resolve within a week with supportive care.[3] However, in a subset of patients, the disease progresses to severe dengue is ascites, where more serious manifestations such as bleeding, shock, and organ involvement occur.

Ascites, the accumulation of fluid in the peritoneal cavity, is a serious complication that can occur in severe cases of dengue fever.[4] The pathophysiology of ascites in dengue is closely related to the alterations in vascular permeability, fluid dynamics, and immune response that are characteristic of the disease. One of the key pathophysiological mechanisms underlying ascites in dengue is the phenomenon known as capillary leak syndrome. DENV causes damage to endothelial cells lining blood vessels, leading to increased vascular permeability.[5] The virus induces inflammatory responses that release various cytokines (such as tumor necrosis factor-α, interleukin-6, and interleukin-8), which in turn increase the permeability of the vascular endothelium.[6] As a result, plasma proteins (especially albumin), along with electrolytes and water, leak out of the blood vessels into the interstitial space and body cavities, including the peritoneal cavity, causing fluid accumulation. This leakage is more pronounced in severe dengue, characterized by increased vascular permeability, hypovolemia, and the development of shock. A significant consequence of the increased vascular permeability is hypoalbuminemia, a condition where the concentration of albumin in the blood decreases.[7] Albumin is a major protein responsible for maintaining osmotic pressure within the vascular system. As the endothelial cells become more permeable, albumin leaks into the interstitial space, reducing the oncotic pressure that normally helps retain fluid within the blood vessels.[8] This leads to a shift of fluid from the intravascular space to the extravascular space, including the peritoneal cavity, resulting in ascites. Hypoalbuminemia also exacerbates edema in other parts of the body, contributing to the overall fluid imbalance in dengue patients.[9] As plasma proteins and fluid leak out of the vasculature, there is a reduction in the circulating blood volume, leading to hypovolemia. This fluid imbalance is one of the hallmark features of severe dengue, where a significant amount of fluid shifts from the blood vessels into the interstitial and peritoneal spaces.[10] The body attempts to compensate for the loss of circulating volume by activating the renin–angiotensin–aldosterone system and the sympathetic nervous system, which increase fluid retention and vasoconstriction. However, these compensatory mechanisms may not be sufficient to prevent the development of hypovolemic shock and ascites, particularly in cases with severe vascular leakage.[11]

The DENV triggers a complex immune response that plays a significant role in the pathogenesis of ascites. The virus induces the activation of the immune system, leading to the release of proinflammatory cytokines, which increase vascular permeability and promote fluid leakage.[12] The immune response also results in the production of immune complexes and the activation of endothelial cells, further contributing to vascular damage and fluid extravasation. In severe cases, a phenomenon called cytokine storm can occur,[13] where an excessive release of cytokines contributes to widespread endothelial injury, increased vascular permeability, and the development of complications such as ascites. The liver plays a central role in fluid balance by producing proteins such as albumin and clotting factors. Hepatic involvement is common in severe dengue infections, as the virus can directly infect liver cells, causing hepatocellular injury and inflammation. Liver dysfunction further exacerbates the development of ascites, as the impaired synthesis of albumin worsens hypoalbuminemia. Additionally, liver injury may lead to impaired production of

clotting factors, which can result in bleeding complications and further contribute to vascular leakage. Patients with preexisting liver conditions, such as hepatitis or cirrhosis, are particularly vulnerable to developing ascites in dengue.[14] In some cases, dengue infection leads to renal impairment, which may further complicate fluid balance and contribute to the development of ascites. Kidney dysfunction can reduce the ability to excrete excess fluid and electrolytes, exacerbating the accumulation of fluid in the peritoneal cavity.[15] Renal impairment may occur due to direct viral invasion of renal tissue or as a consequence of hypovolemia and ischemia resulting from severe fluid loss.

In severe dengue, hypovolemia and the inability to maintain adequate circulating volume can lead to shock, which may further worsen vascular permeability and ascites development. The reduction in blood pressure and impaired tissue perfusion during shock can cause endothelial dysfunction, promoting the leakage of fluid from blood vessels into the abdominal cavity. The combination of shock, capillary leak, and hypoalbuminemia can lead to massive ascites, which is often a marker of poor prognosis in dengue patients. According to the WHO, plasma leakage is defined as an increase in hematocrit of 20% or more, accompanied by hypoalbuminemia and fluid accumulation in the pleural cavity or peritoneal space.[16] Recent research, including a study by Leowattana and Leowattana, highlights key diagnostic signs of plasma leakage, including hemoconcentration of 15.1% or higher, albumin levels of 3.49 mg/dL or lower, platelet counts of 49,500/µL or lower, and an aspartate aminotransferase ratio of 2.51 or above.[17] This research seeks to offer insights into the patterns and determinants of ascites in dengue patients, thereby contributing to enhanced clinical outcomes and management strategies.

Methodology

Study Design

This study employed a retrospective design to analyze clinical data from patients who were diagnosed with dengue and developed ascites. The data were collected from a single urban health care facility in Dhaka, Bangladesh, which treats a diverse patient population. The primary aim was to assess the clinical characteristics and potential risk factors associated with ascites onset in dengue patients, particularly focusing on the role of preexisting comorbidities.

Data Collection

The dataset comprised 48 patients who were confirmed to have dengue and subsequently developed ascites. The inclusion criteria were patients with a confirmed diagnosis of dengue, as evidenced by laboratory tests, who showed signs of ascites during their hospital stay. The data collected included demographic variables such as age and sex, as well as clinical information such as the day on which ascites developed following the initial suspicion of dengue. Furthermore, comorbidities that may have influenced disease progression were also recorded, including hepatitis, diabetes, hypertension, and renal impairment. These variables were selected based on their potential to exacerbate the pathophysiological effects of dengue infection and contribute to the development of severe complications such as ascites.

Statistical Analysis

Descriptive statistics were used to summarize the patient demographics and clinical features. Continuous variables were presented as means with standard deviations, while categorical variables were presented as frequencies and percentages. A *t*-test was performed to compare the means of ascites development days between patients with and without comorbidities. A *p*-value of less than 0.05 was considered statistically significant.

Results

Patient Demographics

The study focused on 48 patients diagnosed with dengue who developed ascites during their illness. The ages ranged from 19 to 77 years, with an average age of 38.5 years. Males made up 60% of the sample, while females accounted for 40%. Age-specific patterns showed variability in ascites development, with the largest group being those aged 41 to 50 years (17 individuals), followed by the 21 to 30 age group (14 individuals) (►**Table 1**).

Timeline for the Onset of Ascites

On average, ascites appeared 6.9 days after the first signs of dengue fever. Most patients (73%) experienced ascites onset between the fifth and seventh days, coinciding with dengue's critical phase (►**Fig. 1**). Gender analysis revealed that females had a slightly earlier mean onset of 6.4 days compared with 6.7 days for males. However, there was notable variability: male patients experienced onset within 4 to 12 days, while for females, it ranged from 5 to 9 days (►**Fig. 2**).

Comorbidities

A significant 58% of patients had one or more comorbidities, which appeared to influence ascites development. Hepatitis was the most common, affecting 33% of patients. Diabetes was present in 12.5% of cases, including both controlled and uncontrolled instances. Other notable conditions included proteinuria, urinary tract infections, peripheral edema, febrile

Table 1 Summary of ascites development by age group

Age group	Number of patients	Mean age	Mean day of ascites development
0–20	1	19	6
21–30	14	25.9	6.2
31–40	8	35	8.1
41–50	17	45	6.9
51–60	3	50.7	7.7
61–70	3	63.3	6
71–80	2	74.5	8.5

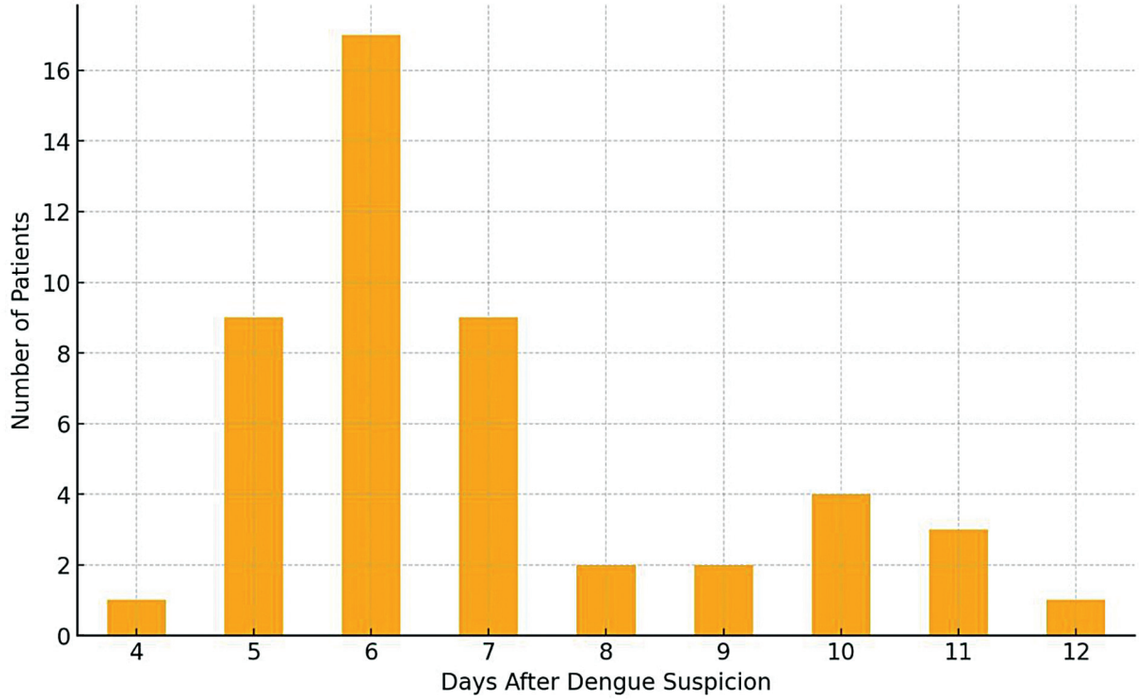

Fig. 1 Frequency of ascites onset days among dengue patients.

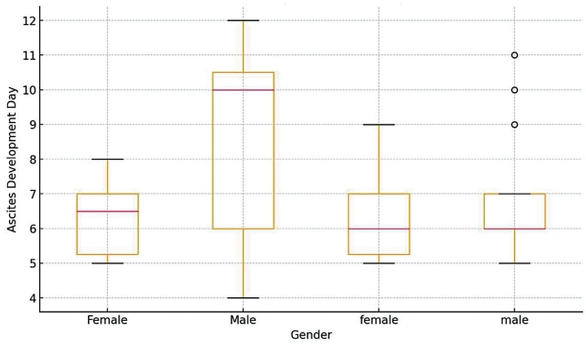

Fig. 2 Ascites onset day distribution by gender.

seizures, and hypertension. These conditions likely contributed to increased vascular permeability and disrupted fluid balance, exacerbating ascites severity (►**Table 2**).

Noncomorbidities

Case-specific data offered insights into the connection between comorbidities and ascites onset. For instance, a 38-year-old man with hepatitis developed ascites on day 6, highlighting the role of liver damage. Similarly, a 25-year-old woman with a urinary tract infection developed ascites on day 8, suggesting a potential link to renal complications. Some patients without significant comorbidities also developed ascites, pointing to its multifactorial nature (►**Table 3**).

The statistical analysis comparing the onset of ascites between patients with and without comorbidities revealed significant differences. The mean onset of ascites in patients with comorbidities was 6.4 days, compared with 7.2 days in those without comorbidities. This difference was statistically significant, with a p-value of <0.05 (►**Fig. 3**). The earlier onset in patients with comorbidities underscores the impact of preexisting conditions, such as hepatitis and diabetes, in exacerbating the vascular and immune disruptions caused by dengue. These findings align with the pathophysiological mechanisms observed in severe dengue cases, where comorbidities amplify fluid leakage and endothelial dysfunction, accelerating the progression of ascites.

Discussion

Analysis of age groups showed little variation in the average onset of ascites. The 31 to 40 age group had the highest mean onset at 8.1 days, while patients aged 21 to 30 and 41 to 50 years experienced earlier onset, with averages of 6.2 and 6.9 days, respectively. Gender trends consistently showed earlier onset in females, while male cases spanned a wider range. From another study of Chaudhary et al, the chances of a severe dengue patient having ascites, gallbladder wall edema, or any ultrasonography finding were 2.74, 2.04, and 2.619 times, respectively.[2] These findings underscore the importance of early identification and close monitoring of dengue patients, especially those with preexisting conditions such as hepatitis and diabetes, to reduce the impact of ascites.

Correlation with Comorbidities

The association between preexisting conditions such as hepatitis and diabetes and the onset of ascites in dengue patients suggests that these comorbidities may intensify the

Table 2 Ascites development with comorbidities

Age	*Sex*	*Ascites development day*	*Other findings*
38	Male	6	Hepatitis
29	Female	6	Increase diabetes
50	Male	6	Hepatitis
23	Female	5	Peripheral edema
23	Male	5	Proteinuria
40	Male	7	Hepatitis
29	Male	5	Febrile convulsion
31	Male	7	Hepatitis
46	Male	10	Hepatitis
26	Female	9	Diabetes uncontrolled
48	Male	6	Hepatitis
43	Female	7	Hepatitis
42	Male	10	Hepatitis
42	Male	6	Hepatitis
33	Male	12	Hepatitis
49	Male	6	Hepatitis
51	Male	6	High blood pressure
38	Female	8	Hepatitis
29	Male	9	Hepatitis

Table 3 Ascites development with noncomorbidities

Age	Sex	Ascites development day	Other findings
41	Female	5	None
47	Female	6	None
46	Male	6	None
22	Male	6	None
25	Female	8	UTI
25	Female	6	None
68	Female	5	None
24	Male	5	UTI
26	Female	5	None
61	Male	7	None
38	Female	6	None
29	Male	6	None
19	Female	6	None
61	Male	6	None
42	Female	7	None
51	Male	11	None
47	Female	7	None
72	Male	10	None
29	Female	5	None
33	Male	11	Pressure headache
77	Female	7	None
42	Male	6	None
47	Male	10	None
49	Female	7	None
23	Female	7	None
37	Male	11	None
32	Male	4	None
48	Male	6	None
46	Female	5	None

Abbreviation: UTI, urinary tract infection.

effects of dengue on vascular permeability and fluid balance. Hepatitis, which often leads to liver dysfunction, can impair the liver's ability to produce proteins such as albumin, which are crucial in maintaining normal blood osmotic pressure. As a result, patients with liver conditions may be more prone to fluid leakage into the abdominal cavity, contributing to ascites. Similarly, diabetes, which can cause vascular damage and affect immune responses, may further compromise the body's ability to regulate fluid balance during dengue infection. These findings align with other studies that have identified liver involvement as a significant risk factor for the development of severe dengue complications, such as bleeding, shock, and ascites. From a study of Razi et al, 3 out of 32 patients underwent ultrasonography in 2019, and all 3 showed good results that were corroborated by blood and immune system tests. Ascites, pleural effusion, a thick gallbladder wall, and other symptoms of plasma leakage are significant consequences of dengue fever.[18] The presence of these preexisting conditions seems to exacerbate the pathophysiological mechanisms of dengue, underscoring the need for heightened awareness and early intervention in patients with hepatic or metabolic disorders who contract the virus.

Clinical Implications

The clinical implications of this study emphasize the importance of early identification and monitoring of at-risk patients, particularly those with preexisting liver or renal conditions, in the management of dengue. These patients are more vulnerable to developing severe complications, including ascites, due to their compromised organ function. Hepatic or renal dysfunction can impair the body's ability to maintain fluid balance and immune response, making it essential to closely monitor these individuals during the critical phase of dengue infection, typically around days 3 to 7 when the risk of progression to severe disease is highest. A study by Leowattana and Leowattana found that 60 to 90% of dengue hemorrhagic fever patients had hepatocellular damage, which showed up as enlarged liver tissue, yellowing of the skin and eyes, increased levels of certain enzymes, and in the worst cases, acute liver failure (ALF).[19] Early detection of signs of fluid accumulation or worsening symptoms allows for timely intervention, such as fluid management, electrolyte correction, and appropriate medications, which can help prevent the progression to more severe manifestations

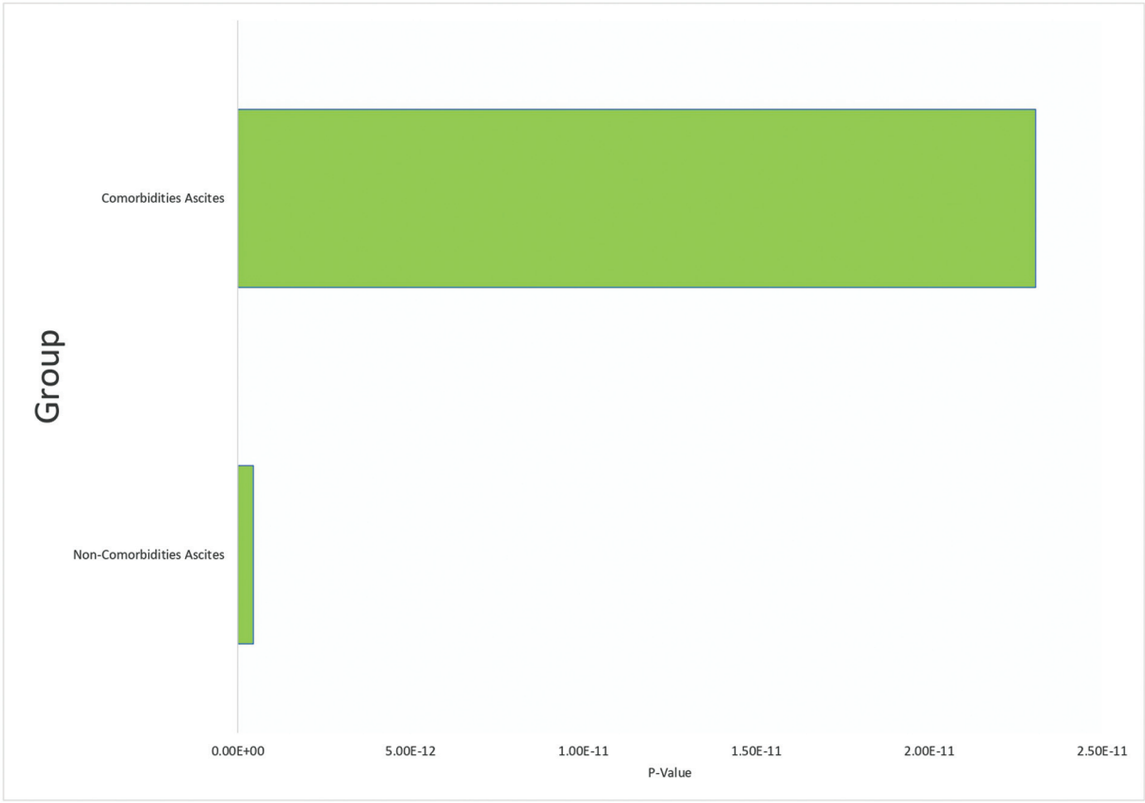

Fig. 3 Comparison of *p*-values for ascites in patients with and without comorbidities.

such as ascites, organ failure, or shock. Another study by Prajapati et al[20] told that there were 166 survivors (83%) of the dengue hepatitis patients who received conventional medical treatment, including crucial organ support as required. Thirty-three patients (17%) died, with 24 patients succumbing to multiorgan failure and 9 patients falling victim to septic shock. A mortality rate of 6.4 (95% confidence interval: 1.2–34) was independently predicted by the presence of shock. ALF (38%), severe dengue hepatitis (24%), dengue shock syndrome (47%), and severe dengue overall were associated with a greater death rate among dengue hepatitis patients. Additionally, this proactive approach can reduce the risk of mortality and long-term complications, improving patient outcomes. By recognizing and managing the risks associated with comorbidities early on, health care providers can significantly reduce the severity of dengue infections in vulnerable populations.

Limitations

This study has several limitations that should be acknowledged. First, it was conducted in a single urban health care facility in Dhaka, Bangladesh, which may not fully represent other regions of the country, particularly rural or economically disadvantaged areas where health care access and disease prevalence can differ significantly. Additionally, the urban setting may introduce socioeconomic biases, as patients in urban centers are likely to have better health care access and different socioeconomic conditions than those in lower-income or remote areas. The retrospective nature of the study limits the ability to establish causal relationships, as it relies on previously collected data from patient records. This design also means the study could not control for confounding variables, and inaccuracies in medical documentation or missing records could affect the reliability of the findings. Moreover, the scope of observations was restricted to what was available in the patient records, which may not have captured important factors such as social determinants of health or patient behaviors. Finally, due to the retrospective approach, the study lacked long-term follow-up data, which would have been beneficial to assess the progression of diseases and confirm the findings. Future prospective studies with more extensive follow-up are necessary to validate these results and explore the long-term effects.

Conclusion

This study provides valuable insights into the prevalence and onset of ascites among dengue patients in an urban setting in Dhaka, Bangladesh. Ascites, a common complication associated with severe dengue infection, was observed to develop predominantly around the 6th day following the suspicion of dengue infection. This timing highlights the critical window for monitoring and management of fluid accumulation in affected individuals. Notably, the prevalence of ascites was found to be higher in males, which aligns with existing literature suggesting that gender may influence the clinical

outcomes of dengue infection. Furthermore, patients with liver-related comorbidities, such as hepatitis, were more likely to experience ascites. This could be attributed to the added stress on the liver from both the underlying liver condition and the viral infection, which could impair the body's ability to regulate fluid balance.

Given the findings, it is crucial for health care providers to closely monitor dengue patients, especially those with pre-existing comorbid conditions such as hepatitis and diabetes. These conditions can exacerbate the development of complications such as ascites. Early detection and management of ascites through appropriate fluid management strategies can significantly improve patient outcomes by reducing the risk of severe complications and organ failure. Moreover, this study underscores the need for heightened awareness and timely intervention in at-risk populations, particularly those in urban environments such as Dhaka, where dengue outbreaks are frequent. By prioritizing early monitoring and intervention, health care systems can enhance the care provided to dengue patients and reduce the morbidity and mortality associated with severe cases of the disease.

Authors' Contribution

S.M.S.I. and N.L. supervised the data collection process, checked the writing and questionnaire design, and approved the methodology; S.A., M.R.A.M., and N.S. wrote the first draft of the manuscript, contributed to the data analysis and final paper revision; A.M.W.H., A.H., and S.N.T. contributed to final editing, manuscript reviewing, and checked the writing.

Conflict of Interest

None declared.

References

1 Afrin S, Hasan MS, Ahmed Mahedi MR, et al. Eltrombopag Olamine in dengue fever: systematic review of clinical trials and beyond. Res J Pharm Technol 2024;17(06):2778–2782
2 Chaudhary S, Manrai K, Dhagat P, et al. Abdominal and chest ultrasonography: a predictor for disease progression in nonsevere dengue. Med J Armed Forces India 2023;79(04):386–391
3 Htun TP, Xiong Z, Pang J. Clinical signs and symptoms associated with WHO severe dengue classification: a systematic review and meta-analysis. Emerg Microbes Infect 2021;10(01):1116–1128
4 Higgins JPT, Thomas J, Chandler J. Cochrane Handbook for Systematic Reviews of Interventions. 2nd ed. 2020
5 Prasad D, Kumar C, Jain A, Kumar R. Accuracy and applicability of the revised WHO classification (2009) of dengue in children seen at a tertiary healthcare facility in northern India. Infection 2013; 41(04):775–782
6 Gowri Sankar S, Alwin Prem Anand A. Cytokine IP-10 and GM-CSF are prognostic biomarkers for severity in secondary dengue infection. Hum Immunol 2021;82(06):438–445
7 Cavalcanti LP, Mota LA, Lustosa GP, et al. Evaluation of the WHO classification of dengue disease severity during an epidemic in 2011 in the state of Ceará, Brazil. Mem Inst Oswaldo Cruz 2014; 109(01):93–98
8 Zhang H, Xie Z, Xie X, Ou Y, Zeng W, Zhou Y. A novel predictor of severe dengue: the aspartate aminotransferase/platelet count ratio index (APRI). J Med Virol 2018;90(05):803–809
9 Agrawal VK, Prusty BSK, Reddy CS, Mohan Reddy GK, Agrawal RK, Sekher Srinivasarao Bandaru VC. Clinical profile and predictors of severe dengue disease: a study from South India. Caspian J Intern Med 2018;9(04):334–340
10 Jayaratne SD, Atukorale V, Gomes L, et al. Evaluation of the WHO revised criteria for classification of clinical disease severity in acute adult dengue infection. BMC Res Notes 2012;5(01):645
11 Singh S, Meena JK, Verma CR, et al. A hospital-based study of hepatic dysfunction in children with dengue fever. Asian Pac J Trop Dis 2015;5(12):964–967
12 Riaz M, Harun SNB, Mallhi TH, et al. Evaluation of clinical and laboratory characteristics of dengue viral infection and risk factors of dengue hemorrhagic fever: a multi-center retrospective analysis. BMC Infect Dis 2024;24(01):500
13 Aroor AR, Saya RP, Sharma A, Venkatesh A, Alva R. Clinical manifestations and predictors of thrombocytopenia in hospitalized adults with dengue fever. N Am J Med Sci 2015;7(12): 547–552
14 Weerakkody RM, Patrick JA, Sheriff MH. Dengue fever in renal transplant patients: a systematic review of literature. BMC Nephrol 2017;18(01):15
15 Giraldo D, Sant'Anna C, Périssé AR, et al. Characteristics of children hospitalized with dengue fever in an outbreak in Rio de Janeiro, Brazil. Trans R Soc Trop Med Hyg 2011;105(10):601–603
16 Talukdar S, Thanachartwet V, Desakorn V, et al. Predictors of plasma leakage among dengue patients in Thailand: a plasma-leak score analysis. PLoS One 2021;16(07):e0255358
17 Vidanapathirana M, Atukorala I. Dengue hemorrhagic fever with bleeding and fluid overload in a patient with active lupus nephritis: a case report of diagnostic and therapeutic challenges. BMC Infect Dis 2023;23(01):433
18 Razi RZ, Ahmed AM, Sultana S, et al. Clinical and haematological findings in dengue fever. Mymensingh Med J 2022;31(02): 344–349
19 Leowattana W, Leowattana T. Dengue hemorrhagic fever and the liver. World J Hepatol 2021;13(12):1968–1976
20 Prajapati R, Mehta R, Kabrawala M, et al. Dengue hepatitis: incidence, spectrum and outcome. Indian J Gastroenterol 2023; 42(03):355–360

