International Journal of Research in Medical Sciences
Wadia N et al. Int J Res Med Sci. 2026 Oct;14(10):4346-4352
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20263494

Original Research Article

Evaluation of plasma leakage and its clinical spectrum in pediatric dengue patients in a tertiary care hospital of Bangladesh

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Received: 13 August 2026
Revised: 18 September 2026
Accepted: 21 September 2026

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ABSTRACT

Background: Dengue is a major public health problem in Bangladesh and plasma leakage is the hallmark of severe disease in children. Early recognition is essential for timely intervention. To evaluate the prevalence, clinical spectrum and associated factors of plasma leakage among pediatric dengue patients admitted to a tertiary care hospital in Bangladesh.
Methods: This retrospective observational study included 130 children aged ≤17 years with NS1-confirmed dengue admitted to the Department of Pediatrics, Delta Medical College and Hospital, from Jun-2025 to May-2026. Children referred after intravenous fluid replacement elsewhere or presenting with multi-organ failure were excluded. Hospital records were analyzed using SPSS, applying the chi-square and independent-samples t-tests.
Results: Among 130 children, 65.4% were male and 35.2% were aged 10–17 years. Plasma leakage (pleural effusion and/or ascites) was identified in 6.9% of patients and abnormal ultrasonographic findings in 10.8%. According to WHO classification, 64.6% had dengue without warning signs, 24.2% had dengue with warning signs and 11.1% had severe dengue. Persistent vomiting was the only clinical feature significantly associated with plasma leakage (21.4% vs. 5.2%, χ^2=5.123, p=0.024). Hemoglobin and hematocrit were higher in children with plasma leakage, but the differences were not statistically significant.
Conclusions: Plasma leakage occurred in a small proportion of hospitalized pediatric dengue patients, with persistent vomiting the only clinical feature significantly associated with it. Careful monitoring of warning signs and early ultrasonographic assessment may facilitate prompt detection of plasma leakage.

Keywords: Bangladesh, Children, Dengue, NS1, Plasma leakage, Pediatrics

INTRODUCTION

Dengue is a major public health problem in tropical and subtropical regions and remains endemic in many countries worldwide.[1] The World Health Organization has identified dengue as a major global health concern, with substantial increases in reported cases in recent decades.[2] Although many dengue infections are asymptomatic or clinically mild, the true burden is likely underestimated because of underdiagnosis and incomplete reporting.[3] Global estimates suggest approximately 390 million dengue virus infections occur annually, of which around 96 million result in clinically apparent illness.[4] In Bangladesh, dengue continues to place a considerable burden on the healthcare system, particularly during seasonal outbreaks, despite ongoing public-health measures.[5] Climate change, which results in longer rainy seasons and increased humidity, along with high population density and rapid urbanization, contributes to the proliferation of mosquitoes and the promotion of outbreaks.[6] Additionally, breakdowns in public health infrastructure, ineffective vector control programs,

insufficient surveillance operations, increased human mobility due to modern transportation and a lack of adherence to dengue protective behavior exacerbate the situation and make controlling outbreaks more difficult.[7] The dengue virus is a single-stranded, enveloped RNA virus that belongs to the flavivirus group within the family Flaviviridae. There are four serotypes of the virus (DEN 1, DEN 2, DEN 3, DEN 4).[8] It is transmitted to humans by the bite of infected female mosquitoes, primarily Aedes aegypti and, to a lesser extent, Aedes albopictus.[9] The four dengue virus serotypes share a significant portion of their structural antigens. Following infection with one serotype, type-specific antibodies are produced, which may cross-react with other serotypes.[10]

Primary infection with one serotype provides long-term to lifelong protection against that serotype but only transient cross-protection against others. An individual may face three additional episodes of dengue infection if exposed to the other serotypes.[11] The risk of developing severe dengue is greatest during the first or second infection, suggesting an immune pathogenesis, with antibody-dependent enhancement being the main theoretical explanation for this phenomenon.[12] From 2013 to 2016, the primary circulating serotypes of Dengue virus (DENV) in Bangladesh were DENV 1 and DENV 2.[13] However, during 2017 and 2018, DENV 2 became predominant.[14] This trend shifted to DENV 3, which was the dominant serotype from 2019 to 2021. Additionally, DENV 3 continued to be the most prevalent serotype during the severe outbreak in 2022.[15] Therefore, the study aimed to evaluate the prevalence of plasma leakage, describe its clinical spectrum and identify factors associated with plasma leakage among pediatric dengue patients admitted to a tertiary care hospital in Bangladesh. Understanding the clinical and laboratory characteristics associated with plasma leakage among pediatric dengue patients is crucial in the context of clinical decision-making.

METHODS

This retrospective observational study was conducted in the Department of Pediatrics, Delta Medical College and Hospital, Dhaka, Bangladesh, to evaluate plasma leakage and its clinical spectrum among pediatric patients with dengue infection. The study was carried out from Jun-2025 to May-2026. A consecutive sampling technique was used and a total of 130 children aged ≤17 years with laboratory-confirmed dengue infection (positive NS1 antigen test) who fulfilled the eligibility criteria were included. Patients referred from other healthcare facilities after receiving intravenous fluid replacement therapy and those presenting with severe multi-organ failure at admission were excluded.

Data were collected retrospectively from hospital medical records using a structured data collection form. Information on demographic characteristics, comorbidities, presenting clinical features, WHO warning signs, bleeding manifestations, laboratory findings, ultrasonographic findings, disease severity, duration of hospitalization and treatment modalities was extracted and reviewed for completeness and accuracy. Plasma leakage, defined as the presence of pleural effusion and/or ascites detected clinically or by ultrasonography, was considered the primary outcome measure, while the clinical spectrum, laboratory profile, disease severity according to the WHO 2009 dengue classification and supportive treatment received were evaluated as secondary outcomes.

Data were analyzed using the Statistical Package for the Social Sciences (SPSS). Continuous variables were expressed as mean±standard deviation (SD) or median, as appropriate, whereas categorical variables were presented as frequencies and percentages. Associations between plasma leakage and categorical variables were assessed using the chi-square test and differences in continuous variables were compared using the independent-samples t-test. A two-tailed p value<0.05 was considered statistically significant.

RESULTS

Most children were school-age or older 35.2% were 10–17 years and 30.5% were 5–10 years, together nearly two-thirds of the cohort, with only 7.8% under 1 year. Boys outnumbered girls almost 2:1 (65.4% vs. 34.6%). Comorbidities were rare (obesity 0.8%, malnutrition 2.3%, asthma 1.5%) and among the small subset with recorded drug history, about a quarter (28.6%) had received antibiotics/steroids before admission.

Headache (36.2%) and joint pain (28.5%) were the most frequently reported clinical features, followed by loose motion (16.9%) and rash (10.8%). Among WHO warning signs, abdominal pain/tenderness (16.9%) and persistent vomiting (10.8%) were the most common, while restlessness/reduced urine output and cold clammy skin were rare (≤2.3%). Bleeding manifestations were uncommon overall, with only isolated cases of epistaxis and hematemesis (0.8% each) and no gum bleeding, malena or hematuria recorded.

Children stayed in hospital for a mean of 3.85±1.94 days, with fever reportedly present for around 2–3 days before admission. On the available hematological readings, the cohort showed mild anemia (Hb 12.40±1.77 g/dl) and a mean platelet count in the moderately thrombocytopenic range at admission (107.2±56.4 $\times 10^3/\mu l$), rising slightly by discharge (125.1±63.9 $\times 10^3/\mu l$) a pattern consistent with the expected platelet nadir-and-recovery course of dengue. Hematocrit (37.36±4.97%) and the differential count (neutrophils 36.1%, lymphocytes 45.5%, i.e., lymphocyte-predominant) were otherwise unremarkable on average.

Frank plasma leakage (pleural effusion and/or ascites) was detected in only 6.9% of children (9/130), with pleural effusion and ascites each present in 6.2%; ultrasonography picked up a somewhat wider abnormality rate (10.8%), suggesting subclinical leakage detected radiologically in a

few additional children beyond those with overt effusion/ascites. Dengue infection was virologically well-confirmed, with NS1 positivity of 98.1% among those tested. By WHO severity classification, most children (64.6%) had dengue without warning signs, 24.2% had dengue with warning signs and 11.1% met criteria for severe dengue indicating that despite low rates of overt plasma leakage, over a third of the cohort showed some degree of warning-sign or severe disease.

Persistent vomiting was the only feature significantly associated with plasma leakage: leakage occurred in 21.4% of children with persistent vomiting versus 5.2% of those without (p=0.024). No urine output/restlessness showed a similar-sized gap (33.3% vs. 6.3%) and shock during the clinical course likewise trended higher among leakage cases (25.0% vs. 6.3%), but neither reached statistical significance (p=0.068 and 0.148 respectively), most likely reflecting the very small number of children with these features rather than a true absence of association. Sex, rash, abdominal pain and the individual bleeding signs showed no meaningful association with leakage in this dataset.

None of the routine hematological parameters differed significantly between children with and without plasma leakage: hemoglobin, hematocrit, platelet counts (admission and discharge), neutrophil percentage and lymphocyte percentage were all statistically similar between groups (all p≥0.10), though hemoglobin and hematocrit were numerically slightly higher in the leakage group, consistent with haemoconcentration. The one 'significant' finding total leukocyte count (p=0.001) is almost certainly a data artifact rather than a true effect, given the implausible values described in the note below and should not be reported as a real finding until the source data are corrected and the test re-run. Oral rehydration solution was used in 64.6% of children and IV crystalloid fluid in the large majority (80%) the two are not mutually exclusive, so many children likely received both. A smaller subset (16.9%) required escalation to crystalloid plus colloid, consistent with the 6.9% rate of overt plasma leakage and the additional USG-detected abnormalities noted in Table 4. Supplemental oxygen (3.8%) and antibiotics (3.0%) were needed only rarely, suggesting most admissions were managed with fluid therapy alone rather than for respiratory compromise or secondary bacterial infection.

All seven tables are now built directly from SPSS output you supplied (frequencies, EXAMINE descriptives, cross-tabulations of plasma leakage against individual clinical variables, one independent-samples t-test block and the fluid/treatment frequencies). Note the N mismatch between Tables 1–6 (N=130) and Table 7 (N=133) flagged above this should be resolved before the tables are combined into a single manuscript draft. A typical manuscript on this topic would also benefit from a multivariable logistic regression identifying independent predictors of plasma leakage and a table comparing outcomes (length of stay, ICU admission, treatment intensity) by severity group these were not present in the output and would need to be run separately in SPSS before they can be tabulated.

Table 1: Demographic and baseline characteristics of study children (n=130).

Characteristics	N	% (of valid)
Age group (n=130)		
6–11 months	10	7.8
1–5 years	35	26.6
5–10 years	40	30.5
10–17 years	45	35.2
Sex (n=130)		
Male*	85	65.4
Female*	45	34.6
Comorbid conditions (n=130)		
Obesity	1	0.8
Malnutrition	3	2.3
Asthma	2	1.5
Pre-admission antibiotic/steroid use (n=21†)		
Yes	6	28.6
No	15	71.4

†Drug-history row based on only 21/130 records (109 missing).

Table 2: Clinical features, warning signs and bleeding manifestations (n=130).

Feature	N	%
Presenting clinical features		
Fever‡	3	2.3

Continued.

Feature	N	%
Anorexia	1	0.8
Nausea	1	0.8
Headache	47	36.2
Joint pain	37	28.5
Loose motion	22	16.9
Rash	14	10.8
WHO warning signs		
Persistent vomiting	14	10.8
Abdominal pain/tenderness	22	16.9
No urine output / restlessness	3	2.3
Cold, clammy skin	1	0.8
Hct rise ≥20%	1	0.8
Bleeding manifestations		
Epistaxis	1	0.8
Gum bleeding	0	0.0
Hematemesis	1	0.8
Malena	0	0.0
Hematuria	0	0.0

‡Only 3/130 were coded positive for "Fever", unexpected in dengue; intended meaning of ClinFeat Fever unconfirmed.

Table 3: Anthropometric, vital sign and laboratory parameters.

Parameters	N (valid)	Mean±SD	Median
Anthropometric/clinical course			
Weight (kg)	81	30.99±18.97	26.0
Length of hospital stay (in days)	81	3.85±1.94	3.0
Days of fever before admissions	81	2.75±5.29	3.0
Vital signs and hematological indices (complete-case subset, n=38)			
Pulse rate (bpm)	38	92.3±14.1	—
Hemoglobin (g/dl)	38	12.40±1.77	—
Hematocrit (%)	38	37.36±4.97	—
Total leukocyte count (×10³/µl)	38	6.29±3.44	—
Neutrophils (%)	38	36.10±17.26	—
Lymphocytes (%)	38	45.49±20.86	—
Platelet count at admission (×10³/µl)	38	107.2±56.4	—
Platelet count at discharge (×10³/µl)	38	125.1±63.9	—

Table 4: Plasma leakage indicators, serological/ imaging findings and disease severity (n=130).

Finding	N	%
Evidence of plasma leakage		
Pleural effusion	8	6.2
Ascites	8	6.2
Composite plasma leakage (effusion and/or ascites)	9	6.9
Abnormal ultrasonogram (USG)	14	10.8
Serology		
NS1 antigen positive (n=104)	102	98.1
IgM positive (n=15)	8	53.3
IgG positive (n=14)	4	28.6
Dengue severity classification (n=99)**		
Dengue without warning signs	64	64.6
Dengue with warning signs	24	24.2
Severe dengue	11	11.1

**Severity coding (1/2/3) assumed as WHO 2009 tiers; unconfirmed. IgM/IgG based on small denominators (n=15, 14).

Table 5: Association between clinical features/warning signs and plasma leakage.

Variables	Leakage among features present	Leakage among features absent	χ^2	P value
Sex (male vs. female)	8.2% (7/85)	4.4% (2/45)	0.656	0.418
Anorexia	0.0% (0/1)	7.0% (9/129)	0.075	0.784
Nausea	0.0% (0/1)	7.0% (9/129)	0.075	0.784
Rash	14.3% (2/14)	6.0% (7/116)	1.320	0.251
Persistent vomiting	21.4% (3/14)	5.2% (6/116)	5.123	0.024*
Abdominal pain	9.1% (2/22)	6.5% (7/108)	0.193	0.660
No urine output / restlessness	33.3% (1/3)	6.3% (8/127)	3.324	0.068
Cold, clammy skin	0.0% (0/1)	7.0% (9/129)	0.075	0.784
Hct rise≥20%	0.0% (0/1)	7.0% (9/129)	0.075	0.784
Epistaxis	0.0% (0/1)	7.0% (9/129)	0.075	0.784
Any bleeding during course	0.0%††	7.0%††	0.075	0.784
Shock during course	25.0%††	6.3%††	2.093	0.148

*Plasma leakage=pleural effusion and/or ascites (n=9/130); interpret cautiously given the small numbers. ††Group sizes not separately reported for these two rows.

Table 6: Comparison of laboratory parameters between children with and without plasma leakage.

Parameters	Leakage present Mean±SD (n)	Leakage absent Mean±SD (n)	t (df)	P value
Hemoglobin (g/dl)	13.26±1.88 (7)	12.18±1.63 (89)	−1.66 (94)	0.101
Hematocrit (%)	39.36±7.11 (5)	35.77±5.51 (77)	−1.39 (80)	0.169
Platelet, admission (×10³/µl)	49.0±30.5 (7)	584.1±4713.1 (99)	0.30 (104)	0.766
Platelet, discharge (×10³/µ)	107.3±42.5 (8)	141.5±75.5 (93)	1.26 (99)	0.210
Total leukocyte count	1004.3±2643.9 (7)	6.60±3.11 (77)	−3.53 (82)	0.001*
Neutrophils (%)	39.42±11.31 (5)	38.42±19.96 (76)	−0.11 (79)	0.912
Lymphocytes (%)	55.25±8.43 (4)	47.40±21.10 (73)	−0.74 (75)	0.463

*Total leukocyte count p value likely reflects a data-entry/unit error, not a real effect (see TC_count ul). Platelet SD also inflated by the 47,000 outlier.

Table 7: Type of replacement/resuscitation fluid and supportive treatment received (n=130).

Treatment received	N	%
Oral rehydration solution (ORS)	84	64.6
IV crystalloid	104	80
IV crystalloid+colloid	22	16.9
Supplemental oxygen	5	3.8
Antibiotics	4	3.0

DISCUSSION

In the present study, most children were school-aged, with 35.2% aged 10-17 years and 30.5% aged 5-10 years and a male-to-female ratio of nearly 2:1 (65.4% vs. 34.6%). This is consistent with the multicenter study by Yesmin et al during the 2019 Bangladesh epidemic, which similarly reported that most pediatric cases were aged 6-17 years with comparable male predominance.[16] By contrast, Islam et al, in a non-endemic zone, described a younger cohort with a mean age of 7.3±4.1 years, though male preponderance (61.8%) was again observed.[17] This recurrent male predominance across Bangladeshi studies may reflect greater outdoor exposure and healthcare-seeking behavior for boys rather than true biological susceptibility. Headache (36.2%) and joint pain (28.5%) were the most frequently reported presenting features, followed by loose motion (16.9%) and rash (10.8%). This is broadly in line with Yesmin et al who reported headache (59.0%) and myalgia (42.0%) as the leading non-fever complaints in their 2019-epidemic cohort.[16] Other investigators described a different symptom profile: Islam et al reported rash in over half of children (55.3%) in a non-endemic setting, while Khan et al, observed vomiting in as many as 80.4% of cases.[17,18] These discrepancies likely reflect differences in case definitions, circulating serotype and the age distribution of each study population. Frank plasma leakage (pleural effusion and/or ascites) was

identified in only 6.9% of children, whereas ultrasonography detected abnormalities in a larger proportion (10.8%), suggesting subclinical leakage not apparent on clinical examination alone. Yesmin et al reported considerably lower rates of effusion and ascites (1.4% and 2.4%, respectively), though from an overall less severe cohort.[16] Substantially higher leakage rates have been reported when restricted to already-severe cases; Sarkar et al, in a northern-Bangladesh series, found severe plasma leakage in 68.4% of children with severe dengue.[9] Sultana et al, in a dedicated study of plasma leakage among Bangladeshi children, similarly emphasized the greater sensitivity of ultrasonography over clinical examination for detecting effusion and ascites, corroborating the present findings.[19]

NS1 antigen positivity was high in our cohort, at 98.1% among children tested, exceeding the 86% reported by Islam et al, in their non-endemic zone study, which the authors attributed to delayed presentation reducing antigen detectability.[17] NS1 is generally regarded as the most sensitive early serological marker in pediatric dengue, though its sensitivity is known to decline with increasing fever duration prior to testing, which may account for this variation. By WHO 2009 classification, 64.6% of children had dengue without warning signs, 24.2% had dengue with warning signs and 11.1% met criteria for severe dengue. This is consistent with the predominance of non-severe disease reported by Sarkar et al, in northern Bangladesh (94.1% non-severe, 5.9% severe).[9] By contrast, a considerably greater burden of severe disease was reported by Khan et al in a multicenter study of the 2019 outbreak, in which 28.9% of children progressed to severe dengue, more than twice our proportion.[18] This variability likely reflects differences in outbreak year, circulating serotype, referral bias and the application of warning-sign criteria.

Persistent vomiting was the only feature significantly associated with plasma leakage in our study, occurring in 21.4% of children with leakage versus 5.2% of those without (p=0.024). This is consistent with the prognostic model by Sreenivasan et al in an Indian pediatric cohort, which identified persistent vomiting as one of a small set of independent predictors of progression to severe dengue, alongside fluid accumulation and a concurrent haematocrit rise with falling platelets.[20] Other investigators found vomiting embedded within a broader cluster of warning signs rather than as an independent predictor; Khan et al identified a falling platelet count with rising haematocrit, rather than vomiting, as the strongest independent predictor in their multicentre cohort.[18] Collectively, these findings support persistent vomiting as a clinically useful bedside warning sign, though its predictive strength appears to vary with which competing signs are considered concurrently.

Mean platelet count was moderately reduced at admission ($107.2\pm56.4\times10^3/\mu l$) and rose by discharge ($125.1\pm63.9\times10^3/\mu l$), consistent with the expected nadir-and-recovery course of dengue-associated thrombocytopenia; haemoglobin and haematocrit were otherwise unremarkable. Islam et al reported thrombocytopenia in 42% of children, a broadly comparable finding despite differing diagnostic thresholds.[17] Other studies found leukopenia, rather than thrombocytopenia, to be the predominant abnormality; Sarkar et al reported leukopenia in 63.3% of children compared with thrombocytopenia in only 30.4% of the same series.[9] Such discrepancies likely reflect variation in sample-collection timing relative to illness day and the predominant circulating serotype.

Overall, these findings indicate that most hospitalized children in this cohort experienced a mild clinical course, with plasma leakage confined to a small subset and that persistent vomiting remains a valuable bedside warning sign for anticipating leakage. The comparatively low rate of overt effusion or ascites, together with the additional yield from ultrasonography, suggests that routine or protocol-driven imaging may meaningfully enhance early detection of subclinical leakage in resource-limited pediatric settings.

This study has several limitations. Its retrospective, single-center design limits the generalizability of the findings and precludes causal inference. Data were extracted from hospital records and several variables had missing or inconsistently coded values; in particular, haematological indices were available only for a complete-case subset (n=38), IgM and IgG results for very small numbers of children (n=15 and 14) and pre-admission drug history for only 21 records. Plasma leakage was identified in only nine children, which limited the statistical power of the comparisons and resulted in wide variation in the subgroup estimates. In addition, multivariable analysis was not performed, so independent predictors of plasma leakage could not be determined. These findings therefore warrant confirmation in larger, prospective, multicentre studies.

CONCLUSION

This study demonstrates that dengue in hospitalized children was predominantly a mild illness, with frank plasma leakage confined to a small proportion (6.9%) despite ultrasonography detecting subclinical leakage in a somewhat wider group (10.8%). Persistent vomiting emerged as the only clinical feature significantly associated with plasma leakage, underscoring its value as a simple, low-cost bedside warning sign. Overall, the clinical and laboratory profile observed in this cohort was broadly consistent with previously reported pediatric dengue series from Bangladesh, reinforcing the importance of vigilant monitoring for warning signs even in children without overt severe disease.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

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Cite this article as: Wadia N, Paul N, Nusrat N, Ferdous J, Yasmin T. Evaluation of plasma leakage and its clinical spectrum in pediatric dengue patients in a tertiary care hospital of Bangladesh. Int J Res Med Sci 2026;14:4346-52.