International Journal of Contemporary Pediatrics
Singh K et al. Int J Contemp Pediatr. 2025 Sep;12(9):1492-1499
http://www.ijpediatrics.com
pISSN 2349-3283 | eISSN 2349-3291

DOI: https://dx.doi.org/10.18203/2349-3291.ijcp20252598

Original Research Article

Clinical profile of infectious causes of thrombocytopenia in children admitted in tertiary care hospital in Eastern Uttar Pradesh: a prospective study

Kushagra Singh, Priyanka Aggarwal, Vineeta Gupta, Chandradeep Srivastava*, Minketan Sidar

Department of Pediatrics, Institute of Medical Sciences, BHU, Varanasi, Uttar, Pradesh, India

Received: 26 June 2025
Revised: 01 August 2025
Accepted: 11 August 2025

***Correspondence:**
Dr. Chandradeep Srivastava,
E-mail: chandradeep27051995@gmail.com

ABSTRACT

Background: Thrombocytopenia, a common hematological abnormality in pediatric patients, can result from diverse infectious and non-infectious causes. Identifying the clinical and etiological profile of thrombocytopenia is crucial for early diagnosis and effective management. To assess the clinical, laboratory and etiological characteristics of thrombocytopenia due to infectious etiology in hospitalized pediatric patients and evaluate their outcomes.
Methods: This prospective observational study was conducted in the Department of Pediatrics, Institute of Medical Sciences, Banaras Hindu University, Varanasi, from August 2023 to July 2024. Pediatric patients aged 1–16 years with thrombocytopenia due to infectious causes were included. Demographic details, clinical features, laboratory parameters, complications and outcomes were recorded.
Results: Among 125 children, majority were 1–5 years old (37.6%), with a slight male predominance (53.6%). Fever was the most common presentation (100%), followed by bleeding manifestations (25.6%). Dengue (34.4%) and Scrub Typhus (21.6%) were the leading etiologies. Severe thrombocytopenia (<10,000/ul) was significantly associated with increased PICU admissions (p=0.0052) and mechanical ventilation (p=0.0001). The mortality rate was 16.8%. Significant predictors of adverse outcomes were ARDS (p=0.0274), vasopressor requirement (p=0.0002) and coagulation abnormalities (p=0.0000).
Conclusions: Infectious diseases, particularly vector-borne illnesses such as dengue and scrub typhus, are predominant causes of thrombocytopenia in pediatric patients. Severe thrombocytopenia correlates with higher PICU admissions and mortality, emphasizing the need for early identification and targeted management strategies.

Keywords: Dengue, Intensive care, Infectious diseases, Pediatric, Scrub typhus, Thrombocytopenia

INTRODUCTION

Thrombocytopenia, defined as a platelet count below 150,000/µl, is a common hematological abnormality in pediatric patients. It can result from decreased platelet production or increased destruction, with causes ranging from infections and autoimmune disorders to malignancies and coagulation disorders.[1] Additionally, immune-mediated conditions such as immune thrombocytopenic purpura (ITP) and drug-induced thrombocytopenia play a crucial role in its pathogenesis.[2] While some cases are asymptomatic and detected incidentally, severe thrombocytopenia (<50,000/µl) can lead to life-threatening bleeding, including intracerebral and intra-abdominal hemorrhage. The condition can be classified as mild (100,000–150,000/µl), moderate (50,000–100,000/µl) or severe (<50,000/µl).[3] Diagnosis and treatment depend on the underlying cause, with

management strategies ranging from observation to targeted interventions such as platelet transfusions in critical cases. The clinical significance of thrombocytopenia varies widely, ranging from asymptomatic cases detected incidentally to severe bleeding manifestations that necessitate urgent medical intervention.[4]

The prevalence and etiology of thrombocytopenia in critically ill patients have been extensively studied in adult populations. Studies by Vanderschueren et al and Strauss et al demonstrated a strong association between thrombocytopenia and adverse clinical outcomes in intensive care unit (ICU) patients, with increased risks of bleeding, transfusion requirements and mortality.[5,6] Levi et al and Löwenberg et al further emphasized that thrombocytopenia in critically ill patients contributes to microvascular failure and organ dysfunction, making it a crucial prognostic marker.[7]

Given the wide spectrum of underlying causes and clinical implications, a systematic evaluation of thrombocytopenia in pediatric patients is essential. This prospective observational study aims to characterize the clinical, etiological and microbiological profile of thrombocytopenia in hospitalized children, particularly in low-resource settings where diagnostic challenges arise due to overlapping presentations and limited laboratory facilities. By analyzing the patterns and infectious causes of thrombocytopenia in children admitted to a tertiary care hospital in Eastern Uttar Pradesh, this study seeks to provide insights into early diagnosis, risk stratification and appropriate management strategies for this common and potentially life-threatening condition.

METHODS

This prospective observational study was conducted at the Department of Pediatrics, Institute of Medical Sciences, Banaras Hindu University over a period of one year (from August 2023 to July 2024). The study was approved by the institutional ethical committee. The study included children aged 1–16 years with thrombocytopenia secondary to infectious causes, while cases of non-infectious thrombocytopenia, such as immune thrombocytopenic purpura, malignancies and children receiving chemotherapy, were excluded. Clinical history, including demographic details, presenting symptoms, duration of illness and comorbidities, was documented. A thorough physical examination was conducted, focusing on signs of bleeding, hepatosplenomegaly and systemic involvement.

All patients underwent laboratory investigations, including complete blood count (CBC) with platelet count, liver function tests (LFT), renal function tests (RFT), coagulation profile (PT, INR, APTT) and serological tests for common infections such as Dengue IgM, Scrub typhus IgM, Leptospira IgM, Malaria rapid antigen test and blood culture for bacterial sepsis. Additionally, inflammatory markers (CRP and Procalcitonin) were assessed. In cases with unexplained thrombocytopenia, bone marrow examination was performed.

Management strategies were recorded, including supportive care, oxygen therapy, blood product support and specific antimicrobial therapy, such as doxycycline for scrub typhus, antimalarials for malaria and intravenous antibiotics for bacterial sepsis. The need for ICU admission, mechanical ventilation, vasopressor support and blood product transfusions were also documented. Patients were followed until discharge or death, with primary outcome variables including improvement (discharge after recovery), adverse outcomes (death or discharge against medical advice) and duration of hospitalization.

Descriptive statistics were used to present the data under different headings. Categorical variables were mainly presented as proportions. Data entry was performed in Microsoft Excel and the analysis was conducted using appropriate statistical methods. Chi-square test was applied for the comparison of proportions and a p-value of less than 0.05 was considered statistically significant.

RESULTS

The study included total 125 cases of non-infectious thrombocytopenia, majority were aged 1–5 years (37.6%), followed by 6–10 years (30.4%) and 11–15 years (30.4%), with a slight male predominance (53.6%) and a median age 7 (3,12) years. In terms of thrombocytopenia severity, 16% had severe thrombocytopenia (<10,000 platelets), while 32.8% had platelet counts between 10,000–50,000, 28.8% between 50,000–100,000 and 22.4% between 100,000–150,000. Fever was universally present (100%), with other common sign/symptoms include respiratory distress (52.8%), abdominal pain (30.4%), vomiting (30.4%), rash (24.8%), hepatomegaly (60.8%) and splenomegaly (33.6%).

Laboratory abnormalities were frequent, with anemia in 58.4% of cases, deranged liver function in 77.6%, hypoalbuminemia in 64.8%, electrolyte abnormality in 57.6% and coagulation abnormalities in 39.2%. Complications were significant, with ARDS in 48.8%, encephalitis in 32% and myocarditis in 16%, necessitating PICU admission in 76.8% of cases with a median PICU stay of 4 (2,5) days. Dengue (34.4%) and scrub typhus (21.6%) were the most common etiologies, followed by leptospirosis (21.6%), malaria (10.4%) and septicemia (11.2%). Management strategies included oxygen support (92.8%), platelet transfusions (32%) and vasopressor support (28.8%). Among patients receiving oxygen therapy, 33.6% required non-invasive ventilation (NIV) and 31% needed invasive mechanical ventilation (IMV). While 81.6% of patients showed improvement 16.8% expired and 1.6% left against medical advice

(LAMA) (Table 1). The association between the severity of thrombocytopenia and various clinical, etiological and laboratory parameters is analyzed in Table 2. Severe thrombocytopenia (<10,000 platelets) was significantly associated with higher PICU admissions (p=0.0052) as well as higher PICU stay (p=0.0445) and increased need for mechanical ventilation (p=0.0001), indicating greater disease severity. Etiology varied significantly with platelet count (p=0.0017), with Dengue, Scrub Typhus and Leptospirosis showing distinct thrombocytopenia patterns. However, complications such as bleeding manifestation, ARDS, myocarditis and deranged kidney function did not show significant differences across platelet count categories, suggesting they may occur independently of thrombocytopenia severity.

Clinical, etiological and laboratory factors in relation to patient outcomes are analyzed in table 3. PICU admission (p=0.0177), mechanical ventilation (p=0.0052), vasopressor support (p=0.0002), ARDS (p=0.0274) and electrolyte (p=0.0016) and coagulation abnormalities (p=0.0000) were significantly associated with higher mortality. Thrombocytopenia severity played a crucial role in predicting outcomes, with patients having a platelet count <50,000 showing significantly worse prognosis (p=0.0274). Patients with deranged kidney function (p=0.0107) also had worse outcomes. However, bleeding manifestation, etiology, encephalitis and deranged liver function showed no significant impact on mortality, suggesting they may not be direct predictors of adverse outcomes in thrombocytopenia patients.

The distribution of the clinical profile, complications and management based on etiology is analyzed in table 4. ARDS (p=0.0339) and anemia (p=0.0051) were significantly associated with specific infections, suggesting that conditions like Dengue and Scrub Typhus may increase the risk of respiratory failure and red blood cell depletion. However, other complications, including encephalitis, myocarditis, vasopressor use and kidney dysfunction, showed no significant differences across etiologies, indicating they occur independently of the underlying cause. Similarly, electrolyte imbalances (p=0.9952) were not linked to specific infections, reflecting a more generalized systemic effect rather than an etiology-driven disturbance.

Table 1: Comprehensive analysis of demographics, clinical presentation, laboratory findings, complications, etiology, management and outcomes in patients with thrombocytopenia.

Parameter	n=125	%
Age group (in years)		
1-5	47	37.6
6-10	38	30.4
11-15	38	30.4
>15	2	1.6
Sex		
Male	67	53.6
Female	58	46.4
Thrombocytopenia severity		
<10000	20	16
10000-50000	41	32.8
50000-100000	36	28.8
100000-150000	28	22.4
Clinical profile		
Fever	125	100
Rash	31	24.8
Abdominal pain	37	29.6
Vomiting	38	30.4
Swelling	27	21.6
Jaundice	8	6.4
Seizure	36	28.8
Altered sensorium	39	31.2
Headache	16	12.8
Hepatomegaly	76	60.8
Splenomegaly	42	33.6
Meningeal signs	34	27.2
Respiratory distress	66	52.8
Bleeding manifestation	32	25.6
Laboratory parameters		
Anemia	73	58.4

Continued.

Parameter	n=125	%
Leucocytosis	48	38.4
Leukopenia	19	15.2
Coagulation abnormality	49	39.2
Deranged liver function	97	77.6
Deranged kidney function	29	23.2
Hypoalbuminemia	81	64.8
Hypernatremia	6	4.8
Hyponatremia	53	42.4
Hyperkalaemia	14	11.2
Hypokalaemia	14	11.2
Complications		
ARDS	61	48.8
Encephalitis	40	32
Myocarditis	20	16
Coagulation abnormality	49	39.2
2 HLH	7	5.6
PICU admission	96	76.8
Etiology		
Dengue	43	34.4
Enteric	1	0.8
Leptospirosis	27	21.6
Malaria	13	10.4
Scrub	27	21.6
Septicaemia	14	11.2
Management		
Doxycycline	90	72
Antimalarial	47	37.6
Vasopressor support	36	28.8
Oxygen support	116	92.8
PRBC transfusion	50	40
FFP transfusion	33	26.4
Platelet transfusion	40	32
Type of oxygen support	**n=116**	
O_2 mask	27	23.3
NRBM	14	12.1
NIV	39	33.6
IMV	36	31
Outcome		
Improved	102	81.6
Death	21	16.8
LAMA	2	1.6

Table 2: Association between thrombocytopenia severity and clinical, etiological and laboratory profile.

Parameters	Platelet count <10000 (n=20)	Platelet count 10000–50000 (n=41)	Platelet count 50000-100000 (n=36)	Platelet count 100000–150000 (n=28)	P value
Duration of PICU stay median (IQR)	4.5 (4.0-6.0)	4.0 (3.0-5.0)	3.0 (0.0-5.25)	3.0 (0.0-5.0)	0.0445*
Bleeding manifestation (n=32)	9	12	8	3	0.5228
Etiology					
Dengue (n=43)	12	12	11	8	0.0017
Scrub (n=27)	0	17	8	2	

Continued.

Parameters	Platelet count <10000 (n=20)	Platelet count 10000–50000 (n=41)	Platelet count 50000-100000 (n=36)	Platelet count 100000–150000 (n=28)	P value
Leptospira (n=27)	4	6	11	6	
Malaria (n=13)	1	4	4	4	
Septicemia (n=14)	3	2	1	8	
PICU admission (n=96)	19	36	22	19	0.0052
IMV+NIV (n=65)	17	25	17	6	0.0001
Encephalitis (n=40)	9	12	10	9	0.4676
ARDS (n=61)	11	22	16	12	0.7139
Myocarditis (n=20)	2	6	9	3	0.3415
Vasopressor requirement (n=36)	6	16	10	4	0.1718
2 HLH (n=7)	2	3	1	1	0.6321
Coagulation abnormality (n=49)	10	17	14	8	0.4944
Deranged LFT (n=97)	17	31	30	19	0.402
Deranged KFT (n=29)	7	13	5	4	0.1009

*Kruskal-Wallis Test.

Table 3: Comparison of outcomes based on clinical, etiological and laboratory profile.

Parameters	Improved (n=102)	Adverse event (Death+LAMA) (n=23)	P value
Bleeding manifestation (n=32)	23	9	0.0997
Etiology			0.6696
Dengue (n=43)	36	7	
Scrub (n=27)	20	7	
Leptospira (n=27)	24	3	
Malaria (n=13)	10	3	
Septicemia (n=14)	11	3	
PICU admission (n=96)	74	22	0.0177
IMV+NIV (n=65)	47	18	0.0052
Encephalitis (n=40)	32	8	0.7514
ARDS (n=61)	45	16	0.0274
Myocarditis (n=20)	14	6	0.144
Vasopressor requirement (n=36)	22	14	0.0002
2 HLH (n=7)	4	3	0.0856
Coagulation abnormality (n=49)	31	18	0
Deranged LFT (n=97)	79	18	0.9329
Deranged KFT (n=29)	19	10	0.0107
Electrolyte abnormality (n=72)	52	20	0.0016
Hypoalbuminemia (n=81)	65	16	0.5963
Anemia (n=73)	56	17	0.0947
Thrombocytopenia severity			0.0274
Platelet count <50000 (n=61)	45	16	
Platelet count >50000 (n=64)	57	7	

Table 4: Etiology-based distribution of laboratory profile, complications and management strategy.

Parameters	Dengue (n=43)	Scrub (n=27)	Leptospira (n=27)	Malaria (n=13)	Septicemia (n=14)	P value
Bleeding manifestation (n=32)	10	5	7	5	4	0.7286
PICU admission (n=96)	32	24	22	9	9	0.356
IMV+NIV (n=65)	23	17	14	4	7	0.0849
Encephalitis (n=40)	14	7	7	5	7	0.5094
ARDS (n=61)	18	18	17	4	4	0.0339

Continued.

Parameters	Dengue (n=43)	Scrub (n=27)	Leptospira (n=27)	Malaria (n=13)	Septicemia (n=14)	P value
Myocarditis (n=20)	4	5	5	4	2	0.4331
Vasopressor requirement (n=36)	11	11	7	4	3	0.6107
2 HLH (n=7)	4	1	0	1	1	0.8538
Coagulation abnormality (n=49)	14	16	10	3	5	0.1404
Deranged LFT (n=97)	34	21	19	10	12	0.846
Deranged KFT (n=29)	13	6	6	2	2	0.6848
Electrolyte abnormality (n=72)	24	17	13	10	8	0.5011
Hypoalbuminemia (n=81)	24	20	14	11	11	0.0955
WBC						0.6139
Leucocytosis (n=48)	15	11	10	9	3	
Leucopenia (n=19)	7	5	5	0	2	
Anemia (n=73)	18	23	16	9	6	0.0051

DISCUSSION

Thrombocytopenia is a significant clinical condition with varying etiologies, presentations and outcomes, particularly in pediatric populations. This study comprehensively analyzes the demographics, clinical profile, laboratory findings, complications, etiology, management and outcomes of pediatric thrombocytopenia cases. Similar findings were reported in studies by Nair et al, Agarwal et al, Naik et al and Dhunputh et al, emphasizing the high incidence of thrombocytopenia in younger children in tropical regions.[8-11] Laboratory findings revealed that anemia (58.4%) and deranged liver function (77.6%) were common. Severe thrombocytopenia (<20,000 platelets) was significantly associated with increased PICU admissions and the need for mechanical ventilation, highlighting greater disease severity. These findings align with studies by Pal et al, Jain et al and Gutthi et al, which reported an increased risk of complications such as acute kidney injury, encephalopathy and hepatic dysfunction in patients with lower platelet counts.[12-14]

Dengue (34.4%), Scrub Typhus (21.6%) and leptospirosis (21.6%) were the leading causes of thrombocytopenia, consistent with findings from Parasher et al, Jain et al and Agarwal et al, where vector-borne diseases were predominant in pediatric thrombocytopenia cases.[9,13,15] Malaria (10.4%) and Septicemia (11.2%) also contributed significantly to the disease burden. As per Erkurt et al, infections, including viral (e.g., dengue, Epstein-Barr virus, cytomegalovirus), bacterial (e.g., sepsis, meningococcemia) and parasitic (e.g., malaria) etiologies, are leading contributors to thrombocytopenia in children, particularly in endemic regions.[3]

Complications such as ARDS (48.8%) and PICU admission (76.8%) were frequently observed, with vasopressor support (28.8%) and mechanical ventilation being critical determinants of prognosis. The study's mortality rate (16.8%) is comparable to findings from Pal et al and Naik et al, which reported mortality rates of 18% and a strong correlation between multi-organ dysfunction and mortality.[10,12] Significant predictors of adverse outcomes included ARDS, vasopressor requirement, electrolyte and coagulation abnormalities and deranged kidney function. Studies by Nair et al, Jain et al and Gutthi et al, confirm the impact of multi-organ dysfunction on mortality.[8,13,14] A previous study from our institution on scrub typhus profiling identified platelet count <50,000/µl as an independent predictor of PICU admission, shock and acute kidney injury (AKI).[17] Similarly, a study from our institution on dengue profiling further corroborates that low platelet counts serve as a crucial marker for severe outcomes in vector-borne illnesses.[18]

Several studies have demonstrated the impact of thrombocytopenia on critically ill patients. Vanderschueren et al and Strauss et al, highlighted the association of thrombocytopenia with poor prognosis, increased transfusion requirements and higher mortality rates in intensive care settings.[5,6] Our study aligns with these findings, demonstrating that severe thrombocytopenia is significantly associated with PICU admissions, mechanical ventilation and poor outcome, reflecting increased disease severity. Levi and Lowenberg emphasized the contribution of thrombocytopenia to microvascular failure and organ dysfunction, particularly in critically ill patients.[7] Our findings corroborate this observation, as complications such as ARDS (48.8%), deranged liver function (77.6%) and coagulation abnormalities (39.2%) were prevalent in our cohort. These factors were significantly associated with adverse outcomes, reinforcing the role of thrombocytopenia as a prognostic marker in pediatric intensive care settings.

The association between thrombocytopenia and specific infections has been widely studied. Studies such as those by Dilshad et al, Parasher et al, Naik et al and Dhunputh et al, emphasize the role of sepsis, dengue, malaria and enteric fever as primary infectious causes of thrombocytopenia in children.[10,11,17,18] The study by Jain et al, highlighted that scrub typhus was a major etiology

in pediatric patients, contributing to thrombocytopenia-related complications.[13] Furthermore, Pal et al and Gutthi et al, noted that acute encephalitis syndrome (AES) frequently presented with thrombocytopenia, though the condition itself did not significantly impact mortality.[12,14] Studies by Agarwal et al and Dhunputh et al also found that sepsis, even in cases with moderate thrombocytopenia, was linked to an increased risk of bleeding due to multi-organ failure.[9,11] The findings from two previous studies from our institution further reinforce our observations, highlighting that dengue and scrub typhus are major contributor to pediatric thrombocytopenia in our setting.[15,16]

Management strategies included oxygen support (92.8%) and platelet transfusions (32%), though transfusions did not always correlate with bleeding manifestations. Empiric therapy for vector-borne infections, such as doxycycline for scrub typhus and supportive care for dengue, remains the mainstay of treatment, as confirmed by studies like Parasher et al, Jain et al, and Gutthi et al.[13,14,17] Additional studies, including those by Agarwal et al and Dhunputh et al, indicate that judicious use of platelet transfusions should be guided by clinical presentation rather than platelet count alone, as unnecessary transfusions may increase complications.[9,11]

The findings emphasize the importance of early identification, aggressive supportive management and targeted therapy for vector-borne illnesses to improve patient outcomes. Future research should focus on long-term prognostic factors and improved treatment algorithms to reduce mortality in pediatric thrombocytopenia cases.

The single-center design of this study restricts the generalizability of findings to other regions with different epidemiological patterns. The short follow-up duration limited long-term outcome assessments, while reliance on available serological and microbiological tests may have led to undiagnosed infections. Selection bias was present as only hospitalized cases were included, potentially overlooking milder outpatient cases. Additionally, variability in platelet transfusion decisions, being clinician-dependent, could have influenced management outcomes.

CONCLUSION

Infectious diseases, particularly vector-borne illnesses such as dengue and scrub typhus, are the predominant causes of thrombocytopenia in pediatric patients. Severe thrombocytopenia is significantly associated with higher PICU admissions and increased mortality. Common complications include ARDS, anemia and organ dysfunction, underscoring the need for early identification and targeted management. Supportive care, including oxygen therapy and appropriate antimicrobial treatment, plays a crucial role in patient recovery. Future studies should focus on long-term outcomes and refined treatment protocols to optimize patient care.

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

REFERENCES

1. Greenberg EM, Kaled ES. Thrombocytopenia. Crit Care Nurs Clin North Am. 2013;25(4):427-34.
2. Fang Y, Shen S, Zhang J, Xu L, Wang T, Fan L, et al. Thrombocytopenia in severe fever with thrombocytopenia syndrome due to platelets with altered function undergoing cell death pathways. J Infect Dis. 2025;231(1):183-94.
3. Erkurt MA, Kaya E, Berber I, Koroglu M, Kuku I. Thrombocytopenia in adults. J Hematol. 2012;1(3):44-53.
4. Gauer RL, Braun MM. Thrombocytopenia. Am Fam Physician. 2012;85(6):612-22.
5. Vanderschueren S, De Weerdt A, Malbrain M, Vankersschaever D, Frans E, Wilmer A, et al. Thrombocytopenia and prognosis in intensive care. Crit Care Med. 2000;28(6):1871-6.
6. Strauss R, Wehler M, Mehler K, Kreutzer D, Koebnick C, Hahn EG. Thrombocytopenia in patients in the medical intensive care unit: bleeding prevalence, transfusion requirements and outcome. Crit Care Med. 2002;30(8):1765-71.
7. Levi M, Löwenberg EC. Thrombocytopenia in critically ill patients. Semin Thromb Hemost. 2008;34(5):417-24.
8. Nair BT, Sharma K, Paimode SD. A study of clinical and laboratory profile of febrile children presenting with thrombocytopenia. Int J Contemp Pediatr. 2017;4(6):2114-9.
9. Agarwal P, Dash DK, Sinha M, Kumari M, Mohanty MD, Srikant AS. Clinical profile and laboratory findings of patients with thrombocytopenia in hospital setup: a hospital-based prospective study. Sri Lanka J Child Health. 2021;50(1):58.
10. Naik IB, Gokula Kumari NJ, Sailaja A, Devireddy NR, Aishwarya VB. A study on the etiological, investigative profile and outcome of fever with thrombocytopenia in children between the age group of two months to twelve years. Eur J Cardiovasc Med. 2024;14(3):54.
11. Dhunputh P, Acharya R, Umakanth S, Shetty SM, Mohammed AP, Saraswat PP. Clinical profile of thrombocytopenia in acute febrile illnesses: a hospital-based study. Kathmandu Univ Med J. 2021;19(2):248-52.
12. Pal M, Verma M, Kumar C, Kumar R, Jain A, Kushwaha R, et al. Thrombocytopenia in children with acute encephalitis syndrome: a prospective observational study. Int J Contemp Pediatr. 2024;11(6):782.

13. Jain R, Kaur P, Kumar P, Guglani V, Randev S. Clinico-etiological profile and outcome of children with acute febrile thrombocytopenia caused by various infections. Pediatr Oncall J. 2023;21(1):7-12.
14. Gutthi LP, Vegesna S, Pundarikaksha V, Kolla S, Gundapaneni M, Karimi PK. A study of clinical and lab profile of fever with thrombocytopenia. Int J Contemp Med Res. 2017;4(5):1057-61.
15. Prakash P, Prasad R, Abhinay A, Singh A. Burden of pediatric scrub typhus at a tertiary care hospital: Clinical, biochemical, complications and predictors of mortality and morbidity. Indian J Pediatr. 2024;91(4):415-9.
16. Singh A, Abhinay A, Prasad R, Mishra OP. Clinical, biochemical and outcome profile of dengue fever in hospitalized children in Eastern Uttar Pradesh, India. Sudanese J Paediatr. 2023;23(2):171.
17. Parasher V, Haeems AA, Dutta S, Panchal AJ. Incidence, severity & therapeutic response of thrombocytopenia in vector-borne diseases among the paediatric age group in Southern Rajasthan. Int J Pharm Clin Res. 2024;16(6):88.
18. Dilshad D, Jamal A, Firoz M, Gul M, Raza M, Yasir A, et al. Fever with thrombocytopenia in the pediatric age group. Pak J Med Health Sci. 2023;31;17(5):545.

Cite this article as: Singh K, Aggrawal P, Gupta V, Srivastava C, Sidar M. Clinical profile of infectious causes of thrombocytopenia in children admitted in tertiary care hospital in Eastern Uttar Pradesh: a prospective study. Int J Contemp Pediatr 2025;12:1492-9.