

Changing Patterns of Low Platelet Counts among Patients Presenting with Fever in Western IndiaSingh Shailja¹, Babariya Bharti², Jitendrakumar Parmar³, Singh Pragati⁴, Arun T.⁵¹Lab Incharge, Microbiology, DMHS, Silvassa²Tutor, Department of Physiology, NAMO Medical Education and Research Institute (NAMO MERI), Silvassa, India³Associate Professor, Department of Pathology, NAMO Medical Education and Research Institute (NAMO MERI), Silvassa, India⁴Research Scientist, ICMR-AMR Programme, NAMO Medical Education and Research Institute (NAMO MERI), Silvassa, India⁵Dermatologist, Silvassa, DNH, India

Received: 20-07-2026 / Revised: 24-08-2026 / Accepted: 27-09-2026

Corresponding Author: Dr. Jitendrakumar Parmar

Conflict of interest: Nil

Abstract:**Background:** Routine complete blood count records can describe the burden of low platelet counts among patients presenting with fever at a hospital. This study evaluated annual proportions at two platelet thresholds and their temporal variation.**Methods:** CBCs were performed for all fever cases, and peripheral blood smears confirmed decreased platelets in all cases with low automated platelet counts. Annual aggregate counts were examined for 2022–2025 and an incomplete 2026 reporting period. Outcomes were platelet counts $\leq 150,000/\mu\text{L}$ and $\leq 80,000/\mu\text{L}$. Mutually exclusive categories were derived by subtraction. Proportions, Wilson 95% confidence intervals and unadjusted prevalence ratios were calculated. Trend testing was restricted to the four complete years.**Results:** Across 50,410 CBC records, 20,600 (40.86%) had platelet counts $\leq 150,000/\mu\text{L}$ and 3,091 (6.13%) had counts $\leq 80,000/\mu\text{L}$. The proportion $\leq 150,000/\mu\text{L}$ increased from 28.75% in 2022 to 49.56% in 2025, an absolute increase of 20.81 percentage points and a prevalence ratio of 1.72 (1.67–1.78). Counts $\leq 80,000/\mu\text{L}$ remained approximately 5.53–6.11% during 2022–2025. In the incomplete 2026 period, the respective proportions were 38.13% and 9.69%. All cases with counts $< 80,000/\mu\text{L}$ were reported CRP-positive.**Conclusion:** Low platelet counts became more frequent among patients presenting with fever during 2022–2025, primarily in the $> 80,000$ to $\leq 150,000/\mu\text{L}$ interval. The incomplete 2026 period showed a higher proportion at the $\leq 80,000/\mu\text{L}$ threshold. These hospital findings do not establish population-wide platelet depletion or a sustained decline in individual platelet counts.**Keywords:** Platelet Count; Thrombocytopenia; Complete Blood Count; Hospital Surveillance; Western India; Retrospective Study.**DOI:** 10.25258/ijcpr.18.9.199

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

Platelet counts are frequently measured during evaluation of acute and chronic illness. A greater proportion of low results in routine laboratory records may reflect changes in disease burden, patient characteristics, referral patterns or the indications for testing. Repeated annual summaries can identify patterns that warrant closer clinical and laboratory review, but the population represented by these records is the population tested at the hospital.

Platelet counts differ by age and sex, as demonstrated in population studies [1,2]. Consequently, annual comparisons without

demographic information cannot distinguish changes in platelet status from changes in the patients attending for testing. Aggregate counts also describe how many results fall below thresholds; they cannot establish a reduction in the mean platelet count or within-person platelet depletion.

Low platelet counts can arise through impaired production, increased destruction or consumption, and splenic sequestration. Evaluation requires clinical history and review of the peripheral blood smear. [3,4] A laboratory threshold describes the measured count; it does not by itself establish

bleeding risk or a specific cause. [5] EDTA-related platelet clumping can produce spuriously low automated counts and should be considered during smear review. [6]

C-reactive protein (CRP) is an acute-phase marker that can accompany infectious and noninfectious inflammation. [7,8] Studies in sepsis and dengue illustrate clinically distinct settings in which thrombocytopenia occurs. [9,10] These findings provide context for laboratory surveillance but do not establish the diagnoses responsible for the present hospital pattern.

This study aimed to quantify the annual frequency of platelet counts $\leq 150,000/\mu\text{L}$ and $\leq 80,000/\mu\text{L}$ at a hospital in Western India, derive nonoverlapping platelet categories and examine changes during complete calendar years. An incomplete 2026 period was evaluated separately.

Materials and Methods

Reporting framework: STROBE and its RECORD extension provide a framework for transparent reporting of observational research using routine health records. [11,12]

Study design and setting: This retrospective descriptive laboratory study examined annual CBC aggregates from PHC Kachigam Under Director of Medical and Health Services DNH, Daman and Diu. Complete calendar years 2022–2025 were included; 2026 was treated as an incomplete reporting period. The exact start and end dates of the 2026 extract must be recorded before submission.

Laboratory records: CBCs were performed for all patients presenting with fever. Peripheral blood smear examination confirmed decreased platelets in all cases with low automated platelet counts. CBC denominators were 11,200, 11,000, 12,500, 12,510 and 3,200 for 2022, 2023, 2024, 2025 and the incomplete 2026 period, respectively. The investigator described EDTA samples of 2 mL,

measurement on a Sysmex analyzer and exclusion of repeated CBCs. Until that verification, results are expressed per CBC record. CRP was reported positive in all cases with platelet counts $< 80,000/\mu\text{L}$. The CRP assay, units and positivity cutoff require specification; results at exactly $80,000/\mu\text{L}$ require separate confirmation because the tabulated category uses an inclusive threshold.

Outcome Definitions: The primary operational threshold was $\leq 150,000$ platelets/ μL and the secondary threshold was $\leq 80,000/\mu\text{L}$. The latter was a nested subset of the former. Three mutually exclusive categories were calculated: $\leq 80,000/\mu\text{L}$; $> 80,000$ to $\leq 150,000/\mu\text{L}$; and $> 150,000/\mu\text{L}$. The $> 150,000$ category was not labeled normal because it also includes possible thrombocytosis. Exact-boundary records at $150,000/\mu\text{L}$ are included in the operational definition.

Statistical Analysis: Counts were divided by the corresponding annual denominator. Wilson intervals quantified uncertainty in proportions. Absolute differences were reported in percentage points. Unadjusted prevalence ratios used log-scale 95% confidence intervals. A two-sided Cochran–Armitage trend test with equally spaced annual scores assessed 2022–2025. Tests assume independent records; they are exploratory until cross-year patient independence is verified. No annualization or imputation was performed for 2026.

Results

All cases with platelet counts $< 80,000/\mu\text{L}$ were reported CRP-positive. Peripheral blood smears confirmed decreased platelets in all 20,600 cases meeting the $\leq 150,000/\mu\text{L}$ threshold. Table 1 shows annual proportions and the remaining platelet categories. All counts were arithmetically consistent: each $\leq 80,000/\mu\text{L}$ count was smaller than the corresponding $\leq 150,000/\mu\text{L}$ count, and the three nonoverlapping categories sum to the annual denominator.

Year	Total CBCs	$\leq 150,000$ n (%)	$\leq 80,000$ n (%)	$> 80,000$ to $\leq 150,000$ n (%)	$> 150,000$ n (%)
2022	11,200	3,220 (28.75%)	668 (5.96%)	2,552 (22.79%)	7,980 (71.25%)
2023	11,000	4,450 (40.45%)	672 (6.11%)	3,778 (34.35%)	6,550 (59.55%)
2024	12,500	5,510 (44.08%)	691 (5.53%)	4,819 (38.55%)	6,990 (55.92%)
2025	12,510	6,200 (49.56%)	750 (6.00%)	5,450 (43.57%)	6,310 (50.44%)
2026†	3,200	1,220 (38.13%)	310 (9.69%)	910 (28.44%)	1,980 (61.88%)
Total	50,410	20,600 (40.86%)	3,091 (6.13%)	17,509 (34.73%)	29,810 (59.14%)

Table 1. Platelet categories by reporting year. The $\leq 80,000$ group is included within the $\leq 150,000$ group and must not be added to it. † Incomplete

2026 period; exact dates require confirmation. Pooled totals combine reporting periods of different durations and are descriptive.

Year	≤150,000 proportion 95% CI	≤80,000 proportion 95% CI	≤80,000 among ≤150,000
2022	27.92–29.60%	5.54–6.42%	20.75%
2023	39.54–41.37%	5.68–6.57%	15.10%
2024	43.21–44.95%	5.14–5.94%	12.54%
2025	48.68–50.44%	5.59–6.42%	12.10%
2026†	36.46–39.82%	8.71–10.76%	25.41%

Table 2. Wilson confidence intervals and the proportion meeting the lower threshold among low platelet records.

The ≤150,000/μL proportion increased in each complete year: 28.75%, 40.45%, 44.08% and 49.56%. Successive changes were +11.70, +3.63 and +5.48 percentage points. From 2022 to 2025, the relative increase was 72.39%. The derived >80,000 to ≤150,000 category increased from 22.79% to 43.57%.

By comparison, the ≤80,000/μL proportion was 5.96%, 6.11%, 5.53% and 6.00% in 2022–2025.

The proportion at this threshold among all ≤150,000 results decreased from 20.75% to 12.10%. Thus, the complete-year rise in low platelet results was concentrated in the intermediate interval.

Comparisons Across Years: The trend test for ≤150,000/μL during 2022–2025 was significant ($Z=32.50$; $p<0.001$). The corresponding test for ≤80,000/μL did not support a linear increase ($Z=-0.48$; $p=0.632$). These unadjusted findings do not account for case mix or possible patients recurring across years.

Comparison	Outcome	Absolute difference pp	Prevalence ratio 95% CI
2025 vs 2022	≤150,000/μL	+20.81	1.72 (1.67–1.78)
2025 vs 2022	≤80,000/μL	+0.03	1.01 (0.91–1.11)
2026† vs 2022–2025 pooled	≤150,000/μL	-2.93	0.93 (0.89–0.97)
2026† vs 2022–2025 pooled	≤80,000/μL	+3.80	1.64 (1.47–1.84)

Table 3. Exploratory unadjusted comparisons. pp denotes percentage points. The 2026 comparisons are not matched for calendar months and cannot establish an annual trend.

The four complete years comprised 47,210 CBCs, of which 19,380 (41.05%) met the primary

threshold and 2,781 (5.89%) met the secondary threshold. In 2026, 1,220/3,200 (38.13%) and 310/3,200 (9.69%) met the respective thresholds. The lower-threshold group accounted for 25.41% of low platelet results in 2026.

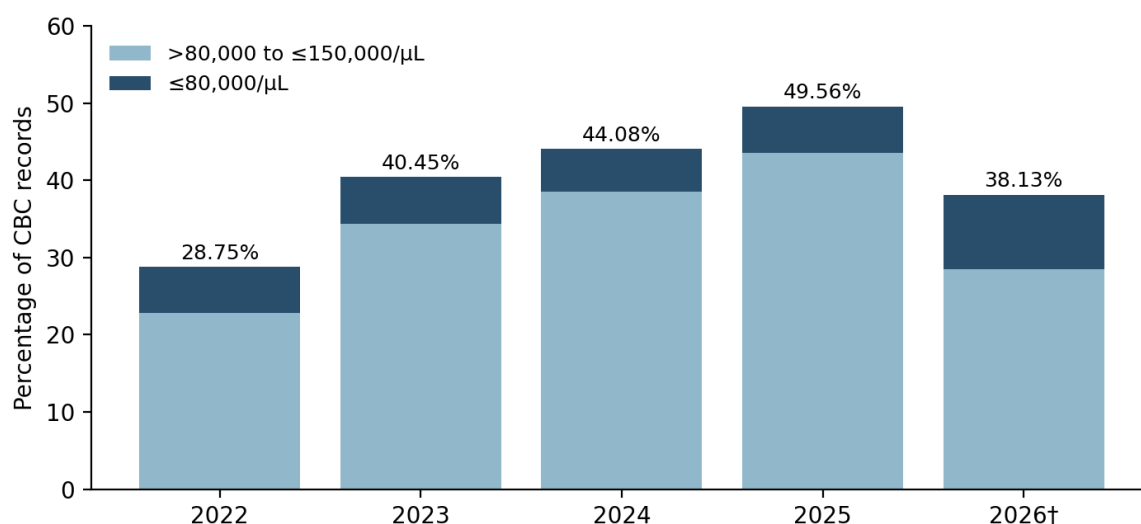


Figure 1: Nonoverlapping low platelet categories as a percentage of CBCs. The total bar height represents ≤150,000/μL. The incomplete 2026 period is displayed separately and does not constitute a full-year estimate.

Discussion

The principal finding was an increase in the frequency of platelet counts ≤150,000/μL among

patients presenting with fever during 2022–2025. The change from 28.75% to 49.56% corresponds to approximately 21 additional low results per 100

CBCs. Because the $\leq 80,000/\mu\text{L}$ proportion remained near 6%, the increase primarily involved counts above 80,000 and at or below $150,000/\mu\text{L}$. This distinction matters: a greater frequency below the primary threshold does not necessarily imply a parallel rise at the lower threshold.

The incomplete 2026 period showed a different pattern: fewer results met the primary threshold than in 2025, while more met the lower threshold. The rise to 9.69% at $\leq 80,000/\mu\text{L}$ is a signal for review, but its interpretation requires comparisons with the same calendar months in earlier years. A change in the seasonal mix of patients, referral activity or the proportion of inpatients could influence this result. These possibilities cannot be tested using annual aggregate counts. CRP positivity in all cases below $80,000/\mu\text{L}$ provides an accompanying inflammatory finding. Without CRP results in higher-count groups or a specified positivity cutoff, an association between CRP and platelet category cannot be quantified. CRP reactivity is nonspecific and cannot identify an infectious agent or prove that inflammation caused the lower platelet count. [7,8]

Population studies show that platelet counts vary with age and sex [1,2]. A hospital series can therefore change when its patient profile changes, even in the absence of a shift in the surrounding community. Future analysis should retain age, sex, location of care, clinical diagnosis and month of testing. Individual counts would enable examination of medians and the complete platelet distribution, rather than only two cutoffs.

The appropriate interpretation is an increasing frequency of smear-confirmed low platelet counts among patients presenting with fever during the four complete years. The term population platelet depletion would imply wider representativeness or longitudinal evidence that is unavailable here. Neither the causes of low counts nor clinical bleeding, mortality or treatment requirements can be inferred from these aggregates. Disease-specific studies in sepsis and dengue demonstrate why diagnostic classification and patient-level follow-up are needed before mechanisms or outcomes are inferred. [9,10]

CRP reactivity should be interpreted within the systemic acute-phase response, which accompanies infectious and noninfectious inflammatory conditions. [13] Platelets also participate in inflammatory and immune pathways beyond their role in haemostasis. [14] These biological links do not identify the cause of thrombocytopenia in this series. Clinical assessment and exclusion of alternative causes are necessary when considering immune thrombocytopenia; the aggregate counts cannot establish that diagnosis. [15]

Strengths and limitations: Strengths include CBC testing of all fever cases, smear confirmation of decreased platelets in all low-count cases, explicit annual denominators, two nested platelet thresholds and internally consistent category counts. The analysis uses proportions rather than absolute counts alone, making differences in testing volume visible. The derived categories clarify which part of the low platelet range contributed to the observed pattern.

Limitations include the hospital-selected sample, absence of individual platelet values and demographic adjustment, unconfirmed independence across years and an incomplete 2026 period. Fever was the indication for CBC testing, and smear review confirmed decreased platelets in every low-count case. Detailed fever diagnoses, medication exposures, smear methods, platelet clumping assessment and clinical outcomes were unavailable in the aggregate dataset. The inclusive $150,000/\mu\text{L}$ threshold may differ from studies using a strict less-than cutoff. Mean platelet count, median, standard deviation, MPV, PDW, plateletcrit and proportions below 50,000 or $20,000/\mu\text{L}$ cannot be calculated from these aggregates. Quantitative CRP values and CRP results in comparator platelet categories were unavailable; CRP positivity was reported only for the group below $80,000/\mu\text{L}$.

Conclusion

Among patients presenting with fever at a hospital in Western India, the proportion with platelet counts $\leq 150,000/\mu\text{L}$ increased substantially from 2022 to 2025, mainly within the $>80,000$ to $\leq 150,000/\mu\text{L}$ interval. The incomplete 2026 period had a higher proportion of counts $\leq 80,000/\mu\text{L}$. These patterns support further patient-level and seasonally matched investigation; they do not prove population-wide platelet depletion.

Acknowledgements: The authors acknowledge the laboratory staff involved in routine CBC testing and peripheral blood smear examination.

References

1. Segal JB, Moliterno AR. Platelet counts differ by sex, ethnicity, and age in the United States. *Ann Epidemiol.* 2006;16(2):123-130. doi:10.1016/j.annepidem.2005.06.052.
2. Biino G, Santimone I, Minelli C, et al. Age- and sex-related variations in platelet count in Italy: a proposal of reference ranges based on 40987 subjects' data. *PLoS One.* 2013;8(1):e54289. doi:10.1371/journal.pone.0054289.
3. Stasi R. How to approach thrombocytopenia. *Hematology Am Soc Hematol Educ Program.* 2012;2012:191-197. doi:10.1182/asheducation-2012.1.191.

4. Izak M, Bussel JB. Management of thrombocytopenia. *F1000Prime Rep.* 2014;6:45. doi:10.12703/P6-45.
5. Gauer RL, Whitaker DJ. Thrombocytopenia: evaluation and management. *Am Fam Physician.* 2022;106(3):288-298.
6. Lardinois B, Favresse J, Chatelain B, Lippi G, Mullier F. Pseudothrombocytopenia—a review on causes, occurrence and clinical implications. *J Clin Med.* 2021;10(4):594. doi:10.3390/jcm10040594.
7. Pepys MB, Hirschfield GM. C-reactive protein: a critical update. *J Clin Invest.* 2003;111(12):1805-1812. doi:10.1172/JCI18921.
8. Sproston NR, Ashworth JJ. Role of C-reactive protein at sites of inflammation and infection. *Front Immunol.* 2018;9:754. doi:10.3389/fimmu.2018.00754.
9. Venkata C, Kashyap R, Farmer JC, Afessa B. Thrombocytopenia in adult patients with sepsis: incidence, risk factors, and its association with clinical outcome. *J Intensive Care.* 2013;1:9. doi:10.1186/2052-0492-1-9.
10. Ojha A, Nandi D, Batra H, et al. Platelet activation determines the severity of thrombocytopenia in dengue infection. *Sci Rep.* 2017;7:41697. doi:10.1038/srep41697.
11. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. *PLoS Med.* 2007;4(10):e296. doi:10.1371/journal.pmed.0040296.
12. Benchimol EI, Smeeth L, Guttmann A, et al. The REporting of studies Conducted using Observational Routinely collected health Data (RECORD) statement. *PLoS Med.* 2015;12(10):e1001885. doi:10.1371/journal.pmed.1001885.
13. Gabay C, Kushner I. Acute-phase proteins and other systemic responses to inflammation. *N Engl J Med.* 1999;340(6):448-454. doi:10.1056/NEJM199902113400607.
14. Vieira-de-Abreu A, Campbell RA, Weyrich AS, Zimmerman GA. Platelets: versatile effector cells in hemostasis, inflammation, and the immune continuum. *Semin Immunopathol.* 2012;34(1):5-30. doi:10.1007/s00281-011-0286-4.
15. Provan D, Arnold DM, Bussel JB, et al. Updated international consensus report on the investigation and management of primary immune thrombocytopenia. *Blood Adv.* 2019;3(22):3780-3817. doi:10.1182/bloodadvances.2019000812.