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CORRELATION BETWEEN MEAN PLATELET VOLUME AND CURB-65 SCORE IN PREDICTING PROGNOSIS IN LOWER RESPIRATORY TRACT INFECTIONS: A PROSPECTIVE OBSERVATIONAL STUDY

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ABSTRACT

Background: Lower respiratory tract infections are a major cause of hospitalization and mortality worldwide. CURB-65 is a widely used bedside severity score, while mean platelet volume (MPV), a routine haemogram parameter reflecting platelet activation, has been proposed as an inexpensive adjunct prognostic marker. The Objective is to assess severity and prognosis using MPV and CURB-65 individually, and to study their correlation in predicting outcome.

Materials and Methods: This prospective observational study enrolled 142 adults hospitalized with community-acquired pneumonia or acute exacerbation of COPD over 18 months. CURB-65 was calculated at admission, and MPV was measured serially on Days 1, 3, 5, 7 and 9. Patients were followed until discharge for ICU admission, mechanical ventilation, pleural effusion, hospital stay, and in-hospital mortality. Chi-square, t-test, and one-way ANOVA were used, with $p<0.05$ considered significant.

Results: The cohort was predominantly elderly (61% aged 51–69 years) with a male predominance (54.93%); 66.90% had CAP. ICU admission, mechanical ventilation, and mortality occurred in 26.06%, 21.83%, and 7.04% of patients respectively. High CURB-65 risk was significantly associated with mechanical ventilation (61.54% vs 14.74%, $p<0.001$), prolonged stay ($p=0.006$), and mortality ($p=0.047$). Mean MPV fell significantly from 9.07±0.85 fL (Day 1) to 8.83±0.84 fL (Day 3, $p<0.001$), and was significantly higher among patients requiring ventilation and prolonged stay. MPV rose significantly across CURB-65 risk strata on both Day 1 and Day 3 ($p<0.001$ for both).

Conclusion: Both CURB-65 and serial MPV correlated significantly with adverse outcomes in LRTI, and MPV tracked closely with CURB-65 severity. Combining this inexpensive, routinely available marker with CURB-65 may improve early risk stratification, particularly in resource-limited settings.

Keywords: Mean platelet volume, CURB-65, lower respiratory tract infection, community-acquired pneumonia, prognosis, mechanical ventilation.

INTRODUCTION

Lower respiratory tract infections (LRTIs), encompassing community-acquired pneumonia (CAP) and acute exacerbations of chronic obstructive pulmonary disease (AECOPD), remain among the leading causes of hospitalization and preventable mortality worldwide, contributing disproportionately to years of life lost in low- and middle-income countries.[1]

India bears a substantial share of this global burden, with CAP accounting for a large proportion of adult hospital admissions and case fatality that rises sharply with advancing age and comorbidity.[2]

Timely and accurate severity assessment at the point of admission is therefore central to rational triage, resource allocation, and treatment intensification.

Among the several validated bedside tools available, the CURB-65 score—derived from Confusion, blood Urea, Respiratory rate, Blood pressure, and age ≥65 years—remains one of the most widely used because of its simplicity and reproducibility across settings,[3] alongside other tools such as the Pneumonia Severity Index.[4]

However, CURB-65 is a static, largely clinical composite that may not fully capture the underlying inflammatory and prothrombotic activity that drives deterioration, nor does it account for how a patient's physiological trajectory evolves after admission.[5]

Mean platelet volume (MPV), a routinely reported parameter on the complete blood count, reflects platelet size and, indirectly, platelet activation. Larger, more metabolically active platelets release greater quantities of pro-coagulant and pro-inflammatory mediators, and several studies have linked elevated or rising MPV with greater severity and worse prognosis in pneumonia.[6,7]

Combining MPV with CURB-65 has been reported to improve mortality prediction beyond either marker alone,[8] but most existing evidence is drawn from single admission values in Western cohorts of CAP, with limited data examining serial MPV trends, and still fewer studies from Indian LRTI populations that include both CAP and AECOPD together.

This gap is clinically relevant: MPV is inexpensive, already captured on a routine haemogram, and requires no additional cost or infrastructure, making it an attractive adjunct in resource-constrained settings where advanced biomarkers are not always feasible.

Establishing how serial MPV behaves in relation to CURB-65 severity strata and hard clinical outcomes could therefore refine bedside risk stratification without added expense. The present study was designed to prospectively evaluate MPV and CURB-65, individually and in relation to each other, as predictors of severity and outcome in hospitalized adults with LRTI.

Aims and Objectives

1. To assess severity and prognosis in lower respiratory tract infections using mean platelet volume.
2. To assess severity and prognosis in lower respiratory tract infections using the CURB-65 score.
3. To study the correlation between MPV and CURB-65 in assessing severity and predicting prognosis in lower respiratory tract infections.

MATERIALS AND METHODS

Study Design and Setting: This was a prospective observational study conducted in the Department of General Medicine at P.E.S. Institute of Medical Sciences and Research (PESIMSR), Kuppam, Andhra Pradesh, over an 18-month period from April 2024 to October 2025.

Study population and eligibility: Adults aged 18–80 years admitted with a clinical and radiological diagnosis of CAP, or with acute exacerbation of COPD, and presenting with fever, cough, sputum production, or dyspnoea, were considered eligible after written informed consent. Patients who were moribund at presentation, or who had destroyed lung, pulmonary tuberculosis, aspiration pneumonia, lung or pleural malignancy, haematological disorders affecting platelet indices, hospital-acquired or ventilator-associated pneumonia, interstitial lung disease, or known immunocompromised status were excluded.

Sample size: Using a reported in-hospital mortality of 34% among ICU-admitted pneumonia patients as the reference proportion,[9] and applying the standard formula for estimating a proportion ($Z = 1.96$, $p = 0.34$, absolute precision $d = 7.8\%$), a minimum sample size of 142 was calculated and achieved through consecutive purposive enrolment of all eligible admissions.

Study procedure: Following institutional ethics committee approval, each enrolled patient underwent structured evaluation comprising demographic and comorbidity profiling, clinical examination, and confirmatory radiological (chest radiograph, HRCT thorax where indicated) and, where available, microbiological assessment. Baseline investigations included complete blood counts, renal and liver function tests, serum electrolytes, arterial blood gases, C-reactive protein, D-dimer, and red cell distribution width. The CURB-65 score was computed at admission, and MPV was measured serially on hospital days 1, 3, 5, 7, and 9. Patients were followed daily until discharge for clinical progress, complications, need for intensive care or mechanical ventilation, and in-hospital mortality.

Variables: Independent variables included age, sex, comorbidities, CURB-65 score, MPV, and other laboratory parameters. Outcome variables were ICU admission, duration of hospital stay, mechanical

ventilation, pleural effusion, and in-hospital mortality.

Statistical analysis

Data were entered in Microsoft Excel and analysed using SPSS version 23.0. Categorical variables were expressed as frequencies and percentages and compared using the chi-square or Fisher's exact test as appropriate; continuous variables were expressed as mean ± standard deviation and compared using the independent-samples t-test or one-way ANOVA across CURB-65 strata. Paired comparison of serial MPV values used the paired t-test. A two-sided p value <0.05 was considered statistically significant.

RESULTS

A total of 142 patients admitted with lower respiratory tract infection were enrolled and followed to discharge. The demographic, clinical, comorbidity, and severity-score profile of the cohort is summarized in [Table 1]. Older adults predominated: participants aged 51–59 years and 60–69 years each constituted nearly one-third of the cohort (30.99% each), with a further 18.31% aged ≥70 years, while patients below 40 years formed a small minority. A modest male predominance was observed (54.93%). Community-acquired pneumonia accounted for two-thirds of admissions (66.90%), with the remainder attributable to AECOPD. Hypertension (50.00%), underlying COPD (45.07%), smoking (33.10%), and diabetes mellitus (32.39%) were the most frequent comorbid and risk-factor associations. Among the individual CURB-65 components, elevated urea (30.28%), tachypnea (27.46%), and age ≥65 years (27.46%) were the most commonly met criteria, and consequently the majority of the cohort was stratified as low CURB-65 risk (66.90%), with 23.94% moderate and 9.15% high risk.

Table 1: Baseline demographic, clinical and severity profile of study participants (N=142)

Parameter	Frequency (n)	Percentage (%)
Age category (years)		
20–29	1	0.70
30–39	6	4.23
41–49	21	14.79
51–59	44	30.99
60–69	44	30.99
≥70	26	18.31
Sex		
Male	78	54.93
Female	64	45.07
Diagnosis		
Community-acquired pneumonia (CAP)	95	66.90
Acute exacerbation of COPD (AECOPD)	47	33.10
Comorbidities		
Hypertension	71	50.00
Underlying COPD	64	45.07
Smoking	47	33.10
Diabetes mellitus	46	32.39
Alcohol use	31	21.83
Chronic kidney disease	17	11.97
Ischemic heart disease	16	11.27
CURB-65 individual components		
Urea >40 mg/dL	43	30.28
Respiratory rate ≥30/min	39	27.46
Age ≥65 years	39	27.46
Low blood pressure	25	17.61
Confusion	21	14.79
CURB-65 risk group		
Low (0–1)	95	66.90
Moderate (2)	34	23.94
High (3–5)	13	9.15
Total	142	100.00

Baseline laboratory and vital parameters are summarized in [Table 2]. The cohort showed a picture of systemic inflammation with relatively preserved renal function on average: mean urea was 29.27 ± 11.13 mg/dL and creatinine 0.92 ± 0.22 mg/dL, while CRP (57.92 ± 22.56 mg/L) and total leukocyte count (10,090 ± 2575/µL) were both elevated. Mean SpO_2 at presentation was 90.41 ± 3.78%, reflecting significant hypoxemia in the admitted cohort despite relatively preserved systolic (118.39 ± 16.36 mmHg) and diastolic (74.46 ± 9.21 mmHg) blood pressures.

Table 2: Baseline laboratory and vital parameters (Mean ± SD)

Parameter	Mean ± SD
Laboratory parameters	
Urea (mg/dL)	29.27 ± 11.13

Creatinine (mg/dL)	0.92 ± 0.22
Sodium (mEq/L)	137.91 ± 3.93
Potassium (mEq/L)	4.01 ± 0.47
Hemoglobin (g/dL)	12.13 ± 1.90
Total leukocyte count (/µL)	10090 ± 2575
Neutrophils (%)	69.65 ± 8.28
Lymphocytes (%)	26.19 ± 6.30
Platelets (×10³/µL)	233.54 ± 60.72
Neutrophil–lymphocyte ratio (NLR)	2.89 ± 1.10
Platelet–lymphocyte ratio (PLR)	102.42 ± 54.37
Monocyte–lymphocyte ratio (MLR)	0.23 ± 0.50
ALT (U/L)	25.50 ± 10.59
AST (U/L)	31.01 ± 11.00
AST/ALT ratio	1.33 ± 0.50
Albumin (g/dL)	3.92 ± 0.40
C-reactive protein (mg/L)	57.92 ± 22.56
D-dimer (mg/L)	0.68 ± 0.32
D-dimer–lymphocyte ratio (DLR)	0.30 ± 0.23
Vital parameters	
Systolic blood pressure (mmHg)	118.39 ± 16.36
Diastolic blood pressure (mmHg)	74.46 ± 9.21
Respiratory rate (/min)	21.06 ± 5.03
SpO□ (%)	90.41 ± 3.78

The overall clinical outcome burden and its association with CURB-65 risk category are presented in [Table 3]. Of the 142 patients, 26.06% required ICU admission, 21.83% required mechanical ventilation, 28.87% developed pleural effusion, and in-hospital mortality was 7.04%. When stratified by CURB-65 risk group, the high-risk category showed a consistently higher proportional burden of adverse outcomes than the low-risk category — most strikingly for mechanical ventilation (61.54% vs 14.74%, p<0.001), prolonged hospital stay ≥7 days (76.92% vs 33.68%, p=0.006), and in-hospital mortality (15.38% vs 6.32%, p=0.047). The gradient for ICU admission across risk groups (46.15% high vs 21.05% low) did not reach statistical significance (p=0.097), and the association between CURB-65 category and pleural effusion was likewise non-significant (p=0.581).

Table 3: Clinical outcomes and their association with CURB-65 risk category

Outcome	Overall n (%)	Low (0–1) n (%)	Moderate (2) n (%)	High (3–5) n (%)	p value
ICU admission	37 (26.06)	20 (21.05)	11 (32.35)	6 (46.15)	0.097
Hospital stay ≥7 days	59 (41.55)	32 (33.68)	17 (50.00)	10 (76.92)	0.006
Mechanical ventilation	31 (21.83)	14 (14.74)	9 (26.47)	8 (61.54)	<0.001
Pleural effusion	41 (28.87)	25 (26.32)	11 (32.35)	5 (38.46)	0.581
In-hospital mortality	10 (7.04)	6 (6.32)	2 (5.88)	2 (15.38)	0.047

Serial MPV values and their relationship with clinical outcomes and CURB-65 risk category are detailed in [Table 4]. Mean MPV declined significantly from a baseline of 9.07 ± 0.85 fL on Day 1 to 8.83 ± 0.84 fL on Day 3 (paired t=9.88, p<0.001), consistent with a downward trajectory during clinical recovery. On Day 1, baseline MPV did not differ significantly by ICU admission (p=0.522), pleural effusion (p=0.808), or in-hospital mortality (p=0.575), but was significantly higher among patients who ultimately required mechanical ventilation (9.41 vs 8.94 fL, p=0.005) and among those with hospital stay ≥7 days (9.26 vs 8.90 fL, p=0.011). By Day 3, the same pattern persisted for mechanical ventilation (9.14 vs 8.73 fL, p=0.019) and prolonged stay (8.98 vs 8.71 fL, p=0.047), and in addition, MPV on Day 3 was now significantly associated with in-hospital mortality (9.18 vs 8.80 fL, p=0.017), whereas the ICU admission and pleural effusion associations remained non-significant. Notably, MPV on both Day 1 and Day 3 showed a strong, statistically significant graded increase across CURB-65 risk strata (Day 1: 8.73 fL low, 9.51 fL moderate, 10.11 fL high, p<0.001; Day 3: 8.52, 9.21, and 9.82 fL respectively, p<0.001), directly demonstrating that higher platelet activation, as reflected by MPV, tracked with increasing CURB-65-defined severity.

Table 4: Mean platelet volume (MPV): serial trend and association with clinical outcomes and CURB-65 risk category

Comparison	Group A Mean (SD)	Group B Mean (SD)	Mean difference	t value	p value
Serial MPV (fL): Day 1 vs Day 3 (paired)					
Day 1 (baseline) vs Day 3	9.07 (0.85)	8.83 (0.84)	0.24	9.88	<0.001
MPV on Day 1 (fL) by outcome					
ICU admission: Yes vs No	9.12 (0.86)	9.02 (0.83)	-0.10	-0.64	0.522
Hospital stay ≥7d vs <7d	9.26 (0.87)	8.90 (0.78)	-0.36	-2.59	0.011

Mech. ventilation: Yes vs No	9.41 (0.83)	8.94 (0.81)	-0.47	-2.83	0.005
Pleural effusion: Yes vs No	9.07 (0.69)	9.04 (0.89)	-0.04	-0.24	0.808
In-hosp. mortality: Yes vs No	9.19 (1.06)	9.04 (0.82)	-0.15	-0.56	0.575
MPV on Day 3 (fL) by outcome					
ICU admission: Yes vs No	8.88 (0.89)	8.81 (0.82)	-0.07	-0.42	0.673
Hospital stay ≥7d vs <7d	8.98 (0.86)	8.71 (0.81)	-0.27	-1.85	0.047
Mech. ventilation: Yes vs No	9.14 (0.82)	8.73 (0.83)	-0.40	-2.37	0.019
Pleural effusion: Yes vs No	8.91 (0.72)	8.79 (0.89)	-0.11	-0.70	0.486
In-hosp. mortality: Yes vs No	9.18 (0.90)	8.80 (0.83)	-0.38	-1.38	0.017
MPV by CURB-65 risk group (Low / Moderate / High) — one-way ANOVA					
Day 1	8.73 (0.73)	9.51 (0.61)	10.11 (0.67)*	—	<0.001
Day 3	8.52 (0.76)	9.21 (0.59)	9.82 (0.72)*	—	<0.001

*Mean (SD) shown for the High-risk CURB-65 group in the ANOVA rows; overall one-way ANOVA p value across Low/Moderate/High groups is reported in the p-value column.

DISCUSSION

This prospective study of 142 hospitalized adults with LRTI demonstrates that both CURB-65 and serial MPV carry independent and complementary prognostic information, and that the two parameters move together across severity strata, supporting their combined use at the bedside.

The demographic and comorbidity profile observed here—older age, male predominance, and a high burden of hypertension, COPD, diabetes, and smoking—mirrors the well-described epidemiology of hospitalized LRTI, in which immunosenescence, multimorbidity, and reduced physiological reserve increase susceptibility to severe pneumonia and complicated COPD exacerbation. Torres et al. similarly note that host vulnerability, including age, often shapes pneumonia trajectory as much as pathogen factors.[10] Almirall et al. similarly identified chronic cardiopulmonary and metabolic comorbidity as key risk factors for CAP in a systematic review of observational studies,[11] consistent with the pattern seen in the present cohort; the modest male predominance and substantial smoking and alcohol-use history are also in keeping with recognized sex-linked differences in infection susceptibility from impaired mucociliary clearance and airway inflammation.[12] The coexistence of CAP and AECOPD in a single inpatient population, as emphasized by Woodhead et al. in the joint European LRTI guidelines,[13] requires careful clinical phenotyping, since exacerbation physiology can mimic or compound pneumonia severity and may partly explain the hypoxemia and ventilatory requirement observed across the cohort.

CURB-65, originally derived and validated by Lim et al,[3] retained clear discriminative utility in this local setting. High-risk patients had markedly greater requirements for mechanical ventilation and prolonged stay, and a statistically significant excess of in-hospital mortality, findings that align with Yamazaki et al.'s comparative evaluation of CURB-65 against other severity tools for mortality prediction in hospitalized pneumonia.[14] The lack of a statistically significant association between CURB-65 category and ICU admission is noteworthy but not unexpected: as Metlay et al. discuss in the ATS/IDSA CAP guideline,[15] ICU triage is shaped by bed availability, institutional policy, and non-respiratory considerations in addition to illness severity, so ventilation requirement and mortality may be more objective endpoints for validating a severity score than ICU admission alone, a distinction also reflected in comparative ward-versus-ICU outcome data.[16] This interpretation is reinforced by the strong CURB-65–ventilation association seen in [Table 3], echoing the rationale behind purpose-built escalation tools such as SMART-COP described by Charles et al.[17]

The behaviour of MPV in this cohort adds a dynamic, laboratory-based dimension to CURB-65-based risk stratification. Korniluk et al. have described MPV as reflecting platelet activation, inflammatory signalling, and endothelial interaction across diverse inflammatory states,[6] and the significant decline in MPV from Day 1 to Day 3 observed here is consistent with resolving platelet activation during clinical stabilization. Importantly, baseline MPV alone did not distinguish patients by ICU admission or mortality, but it was already higher among those who would go on to need mechanical ventilation or prolonged hospitalization—suggesting that MPV may better flag evolving respiratory failure risk than immediate triage status. This is broadly consistent with Gorelik et al., who reported that a rising MPV trajectory during hospitalization for CAP predicted long-term mortality,[7] although the present cohort showed an overall declining trend; differences in directionality likely reflect differences in measurement timing, the mixed CAP–AECOPD case-mix, pathogen heterogeneity,[18] and baseline platelet activation state across study populations.

The emergence of a significant MPV–mortality association specifically at Day 3, rather than at admission, is clinically important. It implies that a single admission value may under-recognize risk, and that early in-hospital trends could refine prognosis beyond a one-time severity score and may also inform longer-term recovery trajectories after discharge,[19] an idea supported by Lee et al., who found that changes in MPV during ICU admission predicted prognosis in pneumonia patients.[9] Golcuk et al. previously proposed that combining MPV with

CURB-65 improved 28-day mortality prediction in CAP;[8] the present findings extend this concept by showing a graded, statistically significant increase in both Day 1 and Day 3 MPV across CURB-65 risk categories, offering direct evidence that the two markers track the same underlying severity gradient rather than capturing unrelated information. Other haematological ratios such as the neutrophil–lymphocyte and platelet–lymphocyte ratios have similarly been reported to add prognostic value in CAP,[20] suggesting that MPV fits within a broader family of accessible, CBC-derived severity markers.

From a practical standpoint, these findings support a two-step bedside strategy: apply CURB-65 at presentation for rapid initial triage, and reassess platelet indices—alongside standard clinical stability markers—at 48–72 hours to refine prognosis and flag patients who may be trending toward respiratory failure. Because MPV is generated automatically with every complete blood count, this strategy adds no cost or additional testing burden, a particularly relevant consideration in resource-constrained hospital settings as highlighted in the broader CAP economic-burden literature reviewed by Welte et al.[21]

The overall in-hospital mortality of 7.04% and ICU admission rate of 26.06% in this cohort fall within ranges reported in comparable hospitalized CAP series, such as that described by Jain et al,[22] and reflect a genuine spectrum of illness severity rather than a uniformly mild case-mix, consistent with descriptions of CAP as a systemic illness with inflammatory responses extending beyond the lungs.[23] The absence of a significant CURB-65–pleural effusion association suggests that effusion may represent a partially distinct pathway of severity, potentially influenced by comorbid cardiac or renal disease rather than purely infective load, a possibility also raised in prior work on radiological complications of CAP. Similarly, the modest but real burden of thrombo-inflammatory markers such as D-dimer in this cohort is in keeping with literature describing coagulation activation as a contributor to adverse outcomes in severe respiratory infection,[24] reinforcing the biological plausibility of platelet-based markers such as MPV in this context. The diagnostic split between CAP and AECOPD also carries antimicrobial stewardship implications, since antibiotic overuse in mixed inflammatory-exacerbation cohorts contributes to resistance and adverse effects.[25]

Taken together, this study reinforces that CURB-65 remains a pragmatic, guideline-consistent first-line triage tool, as emphasized in the consensus statements of Mandell et al. and the subsequent ATS/IDSA update,[26] while serial MPV offers a low-cost, readily available adjunct that captures the dynamic, evolving component of illness severity that a single admission-based score cannot. Identifying patients with a rising or persistently elevated MPV trend, particularly in the context of a moderate-to-high CURB-65 category, may help clinicians anticipate ventilatory failure and prolonged hospitalization, and could inform proactive staffing, bed-allocation, and escalation decisions in settings with limited ICU capacity, consistent with the practical implications discussed by Aksu et al. and Khalifa et al. in their respective work on mortality risk factors and bedside severity correlates in CAP.[27,28] Given that pneumonia hospitalization has also been linked to subsequent cardiovascular risk, discharge planning for high-severity patients identified by this combined approach should extend beyond the index admission.[29]

Strengths and Limitations

- This study was conducted at a single tertiary care centre, which may limit generalizability of the findings to other institutional or geographic settings.
- The number of non-survivors (n=10) was relatively small, which may have reduced the statistical power available for mortality-related comparisons and could account for the borderline significance observed for some outcome associations.
- Purposive, non-randomized consecutive sampling, while pragmatic and reflective of real-world case-mix, carries an inherent potential for selection bias common to observational designs of this nature.
- Etiological confirmation by microbiology was not uniformly available for all patients, and pathogen heterogeneity (bacterial, viral, or atypical) could not be fully accounted for in the interpretation of inflammatory marker behaviour.

CONCLUSION

In this prospective cohort of 142 hospitalized adults with lower respiratory tract infection, both CURB-65 and mean platelet volume demonstrated significant associations with important markers of clinical severity, including prolonged hospital stay, requirement for mechanical ventilation, and in-hospital mortality. CURB-65 risk stratification performed well for outcomes reflecting respiratory decompensation and death, while serial MPV added a dynamic dimension, rising in step with CURB-65 severity and identifying risk that was not always apparent from a single admission value alone. Because MPV is inexpensive, routinely available, and requires no additional testing infrastructure, its use alongside CURB-65 offers a practical, low-cost strategy for early identification of high-risk patients and for guiding escalation of care in the management of lower respiratory tract infections, particularly in resource-limited settings.

REFERENCES

1. Ferreira-Coimbra J, Madureira Lima J, Veras TN, Silva DR, Guslandi M, Santos-Silva FM, et al. The global burden of

community-acquired pneumonia and unmet clinical needs. J Clin Epidemiol. 2020;122:1-12.
2. Niederman MS. Community-acquired pneumonia: epidemiology and treatment. Chest. 1998;113(3 Suppl):S116-24.
3. Lim WS, van der Eerden MM, Laing R, Boersma WG, Karalus N, Town GI, et al. Defining community acquired pneumonia severity on presentation to hospital: an international derivation and validation study. Thorax. 2003;58(5):377-82.
4. Fine MJ, Auble TE, Yealy DM, Hanusa BH, Weissfeld LA, Singer DE, et al. A prediction rule to identify low-risk patients with community-acquired pneumonia. N Engl J Med. 1997;336(4):243-50.
5. Sintes H, Sibila O, Waterer GW, Chalmers JD. Severity assessment tools in CAP. Eur Respir Monogr. 2014;63:88-104.
6. Korniluk A, Koper-Lenkiewicz OM, Kamińska J, Kemona H, Dymicka-Piekarska V. Mean platelet volume (MPV): new perspectives for an old marker in the course and prognosis of inflammatory conditions. Mediators Inflamm. 2019;2019:9213074.
7. Gorelik O, Otero RM, Madanes S, et al. A rise in mean platelet volume during hospitalization for community-acquired pneumonia predicts long-term mortality. BMC Pulm Med. 2017;17:48.
8. Golcuk Y, Golcuk B, Bilge A, Irik M, Dikmen OO. Combination of mean platelet volume and the CURB-65 score better predicts 28-day mortality in patients with community-acquired pneumonia. Am J Emerg Med. 2015;33(5):648-52.
9. Lee JH, Park M, Han S, Hwang JJ, Park SH, Park SY. An increase in mean platelet volume during admission can predict the prognoses of patients with pneumonia in the intensive care unit: a retrospective study. PLoS One. 2018;13(12):e0208715.
10. Torres A, Cilloniz C, Niederman MS, Menéndez R, Chalmers JD, Wunderink RG, et al. Pneumonia. Nat Rev Dis Primers. 2021;7(1):25.
11. Almirall J, Serra-Prat M, Bolíbar I, Balasso V. Risk factors for community-acquired pneumonia in adults: a systematic review of observational studies. Respiration. 2017;94(3):299-311.
12. Arcavi L, Benowitz NL. Cigarette smoking and infection. Arch Intern Med. 2004;164(20):2206-16.
13. Woodhead M, Blasi F, Ewig S, Garau J, Huchon G, Ieven M, et al. Guidelines for the management of adult lower respiratory tract infections. Clin Microbiol Infect. 2011;17 Suppl 6:E1-59.
14. Yamazaki R, Ito A, Kobayashi T, Ishida T, et al. Comparison of CURB-65, PSI, and qSOFA for predicting mortality from pneumonia in hospitalized patients. Sci Rep. 2021;11(1):83381.
15. Metlay JP, Waterer GW, Long AC, Anzueto A, Brozek J, Crothers K, et al. Diagnosis and treatment of adults with community-acquired pneumonia. An official clinical practice guideline of the American Thoracic Society and Infectious Diseases Society of America. Am J Respir Crit Care Med. 2019;200(7):e45-67.
16. Restrepo MI, Mortensen EM, Velez JA, Frei CR, Anzueto A. A comparative study of community-acquired pneumonia patients admitted to the ward and the ICU. Chest. 2008;133(3):610-7.
17. Charles PG, Wolfe R, Whitby M, Fine MJ, Fuller AJ, Stirling R, et al. SMART-COP: a tool for predicting the need for intensive respiratory or vasopressor support in community-acquired pneumonia. Clin Infect Dis. 2008;47(3):375-84.
18. Cilloniz C, Ewig S, Polverino E, Marcos MA, Esquinas C, Gabarrus A, et al. Microbial aetiology of community-acquired pneumonia and its relation to severity. Thorax. 2011;66(4):340-6.
19. Restrepo MI, Faverio P, Anzueto A. Long-term prognosis in community-acquired pneumonia. Curr Opin Infect Dis. 2013;26(2):151-8.
20. Enersen CC, Egelund GB, Petersen PT, Jensen JS, Østergaard C, Michelsen AE, et al. The ratio of neutrophil-to-lymphocyte and platelet-to-lymphocyte and association with mortality in community-acquired pneumonia: a derivation-validation cohort study. Infection. 2023;51(5):1339-47.
21. Welte T, Torres A, Nathwani D. Clinical and economic burden of community-acquired pneumonia among adults in Europe. Thorax. 2012;67(1):71-9.
22. Jain S, Self WH, Wunderink RG, Fakhran S, Balk R, Bramley AM, et al. Community-acquired pneumonia requiring hospitalization among U.S. adults. N Engl J Med. 2015;373(5):415-27.
23. File TM Jr, Ramirez JA. Community-acquired pneumonia. N Engl J Med. 2023;389(7):632-41.
24. Zhang L, Yan X, Fan Q, Liu H, Liu X, Liu Z, et al. D-dimer levels on admission to predict in-hospital mortality in patients with COVID-19. J Thromb Haemost. 2020;18(6):1324-9.
25. Llor C, Bjerrum L. Antimicrobial resistance: risk associated with antibiotic overuse and initiatives to reduce the problem. Ther Adv Drug Saf. 2014;5(6):229-41.
26. Mandell LA, Wunderink RG, Anzueto A, Bartlett JG, Campbell GD, Dean NC, et al. Infectious Diseases Society of America/American Thoracic Society consensus guidelines on the management of community-acquired pneumonia in adults. Clin Infect Dis. 2007;44 Suppl 2:S27-72.
27. Aksu NM, Doğanay F, Çelik B, Önal O. Mortality-associated risk factors in hospitalized patients with community-acquired pneumonia: a retrospective study. J Surg Med. 2021;5(6):534-8.
28. Khalifa M, Fekry M, Ragab A. Correlation between lung ultrasound findings and CURB-65 score in community-acquired pneumonia: a prospective study using wireless probes. Egypt J Bronchol. 2022;16:8.
29. Corrales-Medina VF, Alvarez KN, Weissfeld LA, Angus DC, Chirinos JA, Chang CC, et al. Association between hospitalization for pneumonia and subsequent risk of cardiovascular disease. JAMA. 2015;313(3):264-74.