

Abnormal platelet count as a prognostic indicator in community-acquired pneumonia in children

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ABSTRACT

Background: Platelets play an important role in the host defense mechanism during bacterial infection, besides their hemostatic action. **Objective:** The objective of the study was to find out whether abnormal platelet count can be used as a prognostic indicator for the severity of community-acquired pneumonia (CAP) in children by determining the relationship of abnormal platelet count (high/low) with complications of pneumonia. **Materials and Methods:** An observational study was conducted with a total of 298 children of ages 2 months–5 years admitted with a complaint of pneumonia, between June 2015 and May 2016 into a tertiary care teaching hospital. Complications of pneumonia were identified. Platelet count, total leukocyte count (TLC), and C-reactive protein (CRP) were estimated. Time taken for recovery was calculated. Relationship of platelet count with different types of complications, time taken for recovery, TLC, and CRP were analyzed. **Results:** The male to female ratio was 1.6:1 and 85.9% of children belonged to 2–12 months of age. Complications of pneumonia were empyema (7.4%), septicemia/meningitis (11.4%), and shock (14.8%). Platelet count was normal in 68.1%, high in 8.05%, and low in 23.8% of cases. There was a significant relationship between abnormal platelet count and complications ($p < 0.001$). Thrombocytosis was related to respiratory complications ($p < 0.001$) and thrombocytopenia was related to systemic complications and mortality ($p < 0.001$). Platelet count had a significant positive correlation with the time taken for recovery ($r = 0.14$, $p = 0.03$). **Conclusion:** Abnormal platelet count can be used as a bad prognostic indicator in CAP in children, as abnormal platelet count was found to be associated with adverse outcomes.


Key words: Complications, Pneumonia, Thrombocytopenia, Thrombocytosis

Pneumonia is the single largest infectious cause of death in children worldwide. In India, around 1.7 million children died before reaching the age of 5 years in 2010 and more than half of them (52%) died in the 1st month of life. The major cause of death was pneumonia (24%), prematurity (20%), and diarrhea (13%) [1]. According to the revised World Health Organization (WHO), pneumonia is defined as the presence of cough with fast breathing (≥ 60 breaths/min in < 2 months of age, ≥ 50 breaths/min in 2–12 months of age, and ≥ 40 breaths/min in 1–5 years of age) and/or lower chest in drawing, which requires home therapy with oral amoxicillin; and severe pneumonia is defined as pneumonia with any general danger sign (presence of refusal to feed, persistent vomiting, convulsions, lethargy or unconsciousness, cyanosis, and stridor in a calm child with chest indrawing), which requires a referral and injectable therapy [2].

Pneumonia has been found to be associated with various complications. Respiratory failure and sepsis are considered

as the most important a direct cause of death among patients with pneumonia [3,4]. Pulmonary complications of pneumonia include pleural effusion, empyema, and lung abscess. Sepsis and pulmonary complications are found to be associated with longer duration of hospital stay and shock [5]. There have been previous studies on the risk factors of acquiring pneumonia, but studies that investigate the predictors of poor outcomes of pneumonia in children are lacking [6].

Platelets are known to be an important part of the hemostatic process. In addition to this, platelets have been increasingly recognized as an important component of an immune response to infections [7-9]. Platelet response in antimicrobial host defense is similar to the leukocyte response in many ways [10]. During bacterial infection, platelets play a role in host defense. Leukocytes and platelets, both, contain antimicrobial peptides that act against a broad range of pathogens that contribute to limit the infection. Platelets undergo chemotaxis and are able to release numerous pro-inflammatory molecules [11]. Thrombocytosis favors an exaggerated systemic inflammatory response [10]. A positive association has been found between levels of inflammatory

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cytokines and severity of the disease in patients with community-acquired pneumonia (CAP) [12].

Thrombocytopenia is a recognized marker of poor outcome in patients with pneumonia due to its association with disseminated intravascular coagulation and severe sepsis [13]. It is a recognized severity criterion and a predictor of mortality in hospitalized patients with CAP [14,15]. Thrombocytopenia is included in the minor severity criteria defined by the Infectious Disease Society of America/American Thoracic Society guidelines to predict intensive care unit (ICU) admission [16].

Various previous studies conducted in India regarding the predictive value of abnormal platelet count in deciding the prognosis in CAP in children showed controversial results. Hence, the present study was designed to study the relationship between pneumonia in children and abnormal platelet count (high/low) in determining the outcome, with the hypothesis that abnormal platelet count (high/low) is an indicator of poor prognosis in children with CAP. We also tried to compare the role of abnormal leukocyte count as a prognostic indicator in CAP in children with that of abnormal platelet count.

MATERIALS AND METHODS

A hospital-based observational study was done in the Department of Paediatrics of a tertiary care teaching hospital in North East India. Ethical clearance was obtained from Institutional Ethics Committee (H) to conduct the study.

The study subjects were children of ages 2 months–5 years, admitted into the Paediatrics ward during June 2015–May 2016, fulfilling the clinical criteria of “Pneumonia” and “Severe Pneumonia” according to the revised WHO classification and treatment of childhood pneumonia at health facilities [2]. Exclusion criteria were children suffering from congenital heart disease, respiratory tract diseases such as tuberculosis, cystic fibrosis, bronchial asthma, foreign body inhalation, laryngomalacia, aspiration and chemical pneumonitis, trauma causing pneumothorax, leukemia and other malignancies, known cases of idiopathic thrombocytopenic purpura, known cases of HIV, history of intake of antibiotics before admission, and parents unwilling to give consent.

The study objectives were to study the relationship (i) between abnormal platelet count (high/low) and complications of CAP in children, (ii) between platelet count and the time taken for recovery of pneumonia, and (iii) the relationship between platelet count and C-reactive protein (CRP)/total leukocyte count (TLC). Outcome measures were (i) to estimate the platelet count primarily, TLC, and CRP, (ii) to find out the complications of pneumonia, that is, empyema, lung abscess, sepsis, or shock, and (iii) calculation of time taken for recovery from pneumonia. Recovery was considered as the resolution of fever, tachypnea, chest retractions, and improvement of sensorium.

For sample size calculation, it was decided that all consecutive “pneumonia” and “severe pneumonia” cases coming to the department of pediatrics during the study period, fulfilling the inclusion criteria will be taken as cases. A total of 578 cases of

pneumonia and severe pneumonia were admitted during the study period. Out of them, 159 cases were excluded on the basis of exclusion criteria. Hence, 453 cases were enrolled as cases after taking written consents from their parents/guardians, after explaining properly about the study. However in 121 children, platelet count could not be done due to the short time period between admission and death. Hence, ultimately 298 children were included as cases.

A detailed history regarding the presence of fever, cough, difficulty in respiration, refusal to feed, etc., were taken. In general examination, special emphasis was given to the sensorium, respiratory rate, pulse, chest indrawing, temperature, blood pressure, etc. All the systems were examined giving special emphasis on the respiratory system. The findings were recorded in a predesigned structured pro forma.

Routine blood examination (Hb%, TLC, differential leukocyte count, and erythrocyte sedimentation rate), CRP, and throat swab were sent as soon as possible following admission. Platelet counts were done in fresh samples and hence the patients who were admitted after 9 pm and expired before 8 am the next morning were excluded from the study. Chest X-ray was done once the patient was stabilized. Normal leukocyte and platelet count were determined as per Dacie and Lewis hematological values in normal children [17] (Table 1). Values above the reference range for age were considered as leukocytosis/thrombocytosis and those below the reference range for age were considered as leukopenia/thrombocytopenia. CRP was estimated by Particle enhanced turbidometry immunoassay where the reference range was 0.00–0.5 mg/dl. Other investigations such as pleural fluid analysis, CSF analysis, and computed tomography scan were done as and when required. Patients were monitored for the presence of fever, respiratory rate, chest indrawing, and signs of complications 12 hourly, and more frequently as needed, till discharge.

Categorical variables were described as frequencies and percentages and compared with the Chi-square test. Pearson's correlation coefficient (r) was calculated to examine the correlation between quantitative variables to compare strength and direction of association between the two variables. $p < 0.05$ was taken as significant.

RESULTS

We enrolled a total of 298 children after fulfilling the inclusion and exclusion criteria. The majority of the children (85.9%) belonged to the age group of 2–12 months. Male to female ratio was 1.6:1. There were no complications in the pneumonia group ($n=170$) and 78.1% of cases developed complications in the severe pneumonia group ($n=128$). The most common complications were septicemia/meningitis, followed by shock and empyema (Table 2).

Table 1: Dacie and Lewis hematological values in normal children

Parameters	2 mo	3–6 mo	1 year	2–6 years
Platelet count ($\times 10^9/l$)	210–650	200–550	200–550	200–490
Leukocyte count ($\times 10^9/l$)	5–15	6–18	7–16	5–15

All the cases had fever, cough, and fast breathing at the time of admission. Chest indrawing was present in 98.99% of cases, followed by the refusal of feeding in 122 (40.9%) cases. Other complaints were grunting, cyanosis, convulsion, altered sensorium, and coma. *Streptococcus pneumoniae* was the most common (39.2%) isolate from the throat, followed by *Klebsiella pneumoniae* in 20.4% of cases. The mean platelet count was 331,000/ μ L (ranging from 40,000 to 800,000/ μ L), mean TLC was 16,150.33/ μ L (range – 2400 to 88,300/ μ L), and the mean CRP was 9.72 mg/L (range –0.14 to 136.25 mg/L). Platelet count was high in 8.1% and low in 23.8% children. TLC was high in 38.3% and low in 7.7% of cases. CRP was increased in 95.6% cases (Table 3).

The majority of children in both pneumonia and severe pneumonia group recovered, and 34 (11.4%) cases died (all in the severe pneumonia group). Out of 34 cases, 31 children had thrombocytopenia, two cases had thrombocytosis, and one had normal platelet count. Approximately, 50% of cases recovered in <5 days (Table 4).

Out of 95 children with abnormal platelet counts, 76.84% of cases developed complications. Thrombocytopenia was significantly associated with mortality but not thrombocytosis.

Table 2: Baseline variables of the cases

Variables	Pneumonia n (%)	Severe pneumonia n (%)	Total n (%)
Age			
2 mo–12 mo	150 (88.2)	106 (82.8)	256 (85.9)
13 mo–60 mo	20 (11.8)	22 (17.2)	42 (14.1)
Sex			
Male	101 (59.4)	82 (64)	183 (61.4)
Female	69 (40.6)	46 (36)	115 (38.6)
Complications			
Septicemia/Meningitis	0 (0)	34 (26.6)	34 (11.4)
Shock	0 (0)	44 (34.4)	44 (14.8)
Empyema	0 (0)	22 (17.2)	22 (7.4)
Lung abscess	0 (0)	0 (0)	0 (0)
No complications	170 (100)	28 (21.9)	198 (66.4)

Table 3: Hematological Parameters of the cases

Parameters n (%)	Pneumonia	Severe Pneumonia	Total
Platelet count			
High	3 (1.8)	21 (16.4)	24 (8.1)
Low	11 (6.5)	60 (46.9)	71 (23.8)
Normal	156 (91.8)	47 (36.7)	203 (68.1)
Leukocyte count			
High	38 (22.4)	76 (59.4)	114 (38.3)
Low	6 (3.5)	17 (13.3)	23 (7.7)
Normal	126 (74.1)	35 (27.3)	161 (54)
C-reactive protein			
High	158 (92.9)	127 (99.2)	285 (95.6)
Normal	12 (7.1)	1 (0.8)	13 (4.4)

Out of 138 children with abnormal TLC, 52.17% had developed complications. Both leukopenia and leukocytosis were significantly associated with mortality (Table 5). Thrombocytosis was associated with respiratory complications, whereas, thrombocytopenia was associated with systemic complications. On the other hand, leukocytosis was associated with both respiratory and systemic complications but leukopenia was associated with only systemic complications (Table 6). CRP was invariably raised in almost all cases (95.6%). No association was found between CRP and either systemic or pulmonary complications ($p=0.262$ and 0.050 , respectively).

On analysis, platelet count was found to have no correlation with TLC ($r=0.001791$, $p=0.78$), but had a significant negative correlation with CRP ($r=-0.119$, $p=0.04$) (Fig. 1). Platelet count was also found to have a weak but significant positive correlation with the time taken for recovery ($r=0.13$, $p=0.029$) (Fig. 2).

DISCUSSION

In India, one of the major causes of death in under-five children is pneumonia (24%) [1]. Delay in diagnosis is one of the reasons for this morbidity and mortality. The future of health of the children

Table 4: Status of patients and time taken for recovery

Status of patients, n (%)	Pneumonia	Severe Pneumonia	Total
Recovered	163 (94.1)	87 (68)	250 (83.9)
Discharged against medical advice	7 (4.1)	7 (5.5)	14 (4.7)
Died	0 (0)	34 (26.6)	34 (11.4)
Total	170 (100)	128 (100)	298 (100)
Time taken for recovery			
<5 days	123 (76)	26 (29.5)	149 (50)
5–10 days	38 (23.4)	52 (59.1)	90 (30.2)
>10 days	1 (0.6)	10 (11.4)	11 (3.7)
Total	162 (100)	88 (100)	250 (100)

Table 5: Relationship of platelet and leukocyte count with complications and mortality

Parameters, n (%)	Complications	No complications	p-value
Abnormal platelet count (n=95)	73 (76.84)	22 (23.15)	<0.001
Normal platelet count (n=203)	7 (3.45)	196 (96.55)	
Abnormal leukocyte count (n=138)	72 (52.17)	66 (47.82)	<0.001
Normal leukocyte count (n=160)	8 (5)	152 (95)	
Mortality, n (%)			
Thrombocytopenia (n=71)	31 (43.66)		<0.001
Thrombocytosis (n=24)	2 (8.33)		0.613
Leukopenia (n=19)	10 (52.63)		<0.001
Leukocytosis (n=119)	21 (17.64)		0.00452

Table 6: Relationship of platelet and leukocyte count with type of complications

Parameters, n (%)	Respiratory (n=22)	Systemic (n=58)	p-value
Thrombocytosis	15 (68.18)	3 (5.17%)	<0.01
No thrombocytosis	7 (31.81)	55 (94.83)	0.368
Thrombocytopenia	2 (9)	53 (91.34)	0.0918
No thrombocytopenia	20 (90.9)	5 (8.62)	<0.001
Leukocytosis	20 (90.9)	37 (63.79)	<0.001
No leukocytosis	2 (9)	21 (36.2)	<0.001
Leukopenia	0	15 (25.86)	0.203
No leukopenia	22 (100)	43 (74.14)	<0.001

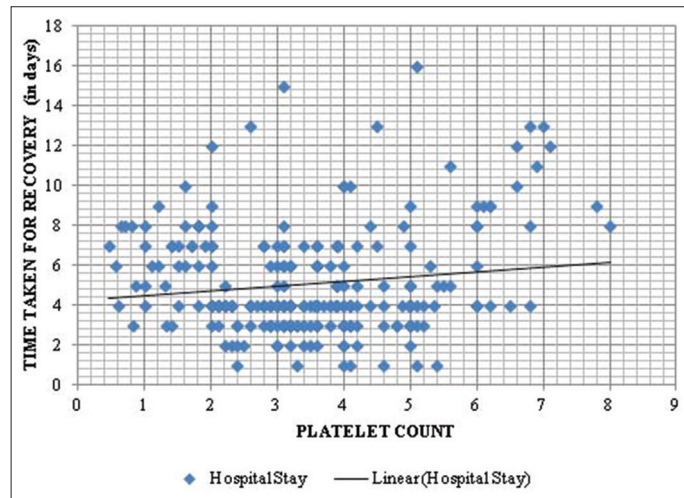


Figure 1: Scatter plot of platelet count and time taken for recovery showing a weak positive correlation

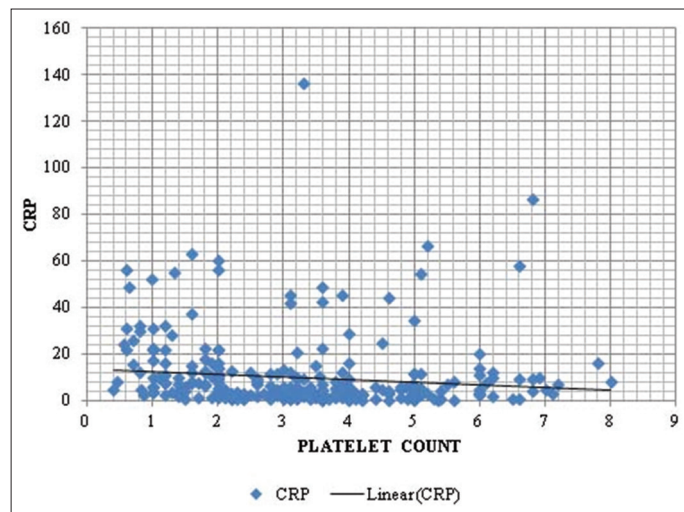


Figure 2: Scatter plot of platelet count and C-reactive protein (showing a weak negative correlation)

of our country depends on preventing, diagnosing, treating, and limiting this disease. Better predictive markers are required to aid us in evaluating and treating these children over and above those that are already used, that is, clinical parameters, leukocyte count, and CRP. In our study, 85.91% of children were between the age group of 2–12 months which is similar to the demographic profile

of the study conducted by Usha and Rudrappa [18]. The male to female ratio was 1.6:1 in our study which was in accordance with the study done by Atwa [6].

In our study, 7.38% of children had pulmonary complications in the form of empyema and 26.2% developed systemic complications in the form of septicemia/meningitis and shock. We found a significant association between thrombocytosis and pulmonary complications but not with systemic complications. This finding was in agreement with the study done in children by Atwa [6]. Prina *et al.* also found that thrombocytosis was a strong indicator for the occurrence of respiratory complications of pneumonia [19]. Usha and Rudrappa also showed a significant association between thrombocytosis and pleural effusion in children with CAP [18]. However, in contrast to the above findings, thrombocytosis was not found to be an indicator for the occurrence of empyema in children with CAP by Wolach B *et al.* [20]. We found no significant association of thrombocytosis with mortality similar to that shown by Usha and Rudrappa. [18].

In our study, thrombocytopenia was present in 71 children and out of them, 53 (74.6%) developed severe sepsis. Later on, 31 (43.66%) children expired. Thrombocytopenia had a significant association with the development of systemic complications and mortality but not with pulmonary complications. This is in agreement with Atwa and Prina *et al.* who found that thrombocytopenia in CAP patients was strongly associated with severe sepsis ($p=0.038$ and $p<0.0001$, respectively) [6,19]. However, Prina *et al.* found no significant association between thrombocytopenia and mortality [19]. On the other hand, Mirsaedi *et al.* indicated that both thrombocytopenia and thrombocytosis are significantly associated with mortality in patients with CAP [21].

In our study, leukocytosis had a significant association with pulmonary complications, severe sepsis, and mortality while leukopenia was significantly associated with sepsis and mortality but not with pulmonary complications. In contrast to our study, Atwa had found that leukocytosis does not affect the prognosis but leukopenia was associated with a fatal course of the disease [6]. Hesham and Heba found that TLC had no significant effect ($p>0.05$) on the severity of pneumonia but thrombocytopenia and thrombocytosis both were significantly associated with mortality in patients with CAP [10]. Thus, they suggested that at the time of hospitalization, abnormalities in platelet count are better predictors of clinical outcomes in patients with CAP when compared with abnormal TLC. Mirsaedi *et al.* also found abnormal platelet count as a better predictor of adverse clinical outcomes than the abnormal TLC [21].

In our study, we have found that abnormal platelet count and TLC were both associated with adverse outcomes in patients with CAP, but abnormal platelet count was able to predict the type of complication a patient is going to develop. Thrombocytopenia predicted the development of severe sepsis and fatal outcome; whereas, thrombocytosis was associated with pulmonary complications but not with systemic complications and mortality. We found no correlation between platelet count and TLC in our study. This can be explained by the fact that the platelet count increases in local complications and decreases in

systemic complications, that is, as the severity increases, platelet count decreases but TLC increases in both local and systemic complications and decreases in only systemic complications.

CRP was raised in almost all cases irrespective of systemic or pulmonary complications. A weak but significant negative correlation was found between CRP and platelet count ($r=-0.119$, $p=0.04$). The negative correlation may be explained by the fact that as the severity of sepsis increases, CRP increases, and platelet count decreases.

Platelet count was found to have a weak but significant positive correlation with the time taken for recovery. In determining the time taken for recovery, the patients who were discharged against medical advice before full recovery were excluded from the study. Furthermore, 43.66% of the patients who had thrombocytopenia had died. This might have a bearing on the positive correlation. Prina *et al.* found that the length of hospital stay was significantly longer in patients with thrombocytosis and thrombocytopenia compared with those with normal platelet count [19]. No statistical difference in the length of stay was found between thrombocytosis and thrombocytopenia. Usha and Rudrappa also found a significant association of thrombocytosis with the duration of hospital stay ($p=0.010$) [18]. In contrast, Hesham and Heba found that platelet count has no significant effect in the length of stay in ICU, while TLC was one of the most powerful predictors [10]. Wolach *et al.* also found no correlation between thrombocytosis, neutrophilia, fever, the clinical course, complications, prognosis, or treatment in patients with CAP [20].

Although there is a wide variation in normal range of platelets, we decided abnormal (high or low) platelet count, in each patient according to the “Dacie and Lewis” hematological values. We excluded the hematological and other diseases that might affect platelet count and thereby tried as far as possible to avoid confounders which might have affected the results. As all the sepsis, meningitis, and shock cases had an initial history of fever, cough, fast breathing and/or chest indrawing, they were included as complicated pneumonia cases.

Our study has some limitations. First, we evaluated platelet count only on admission. Serial measurements to see the effect of therapy on platelet count were not done. Second, in the patients presenting with pulmonary complications, we were not able to evaluate whether platelet counts were abnormal before the development of empyema or not. Third, biomarkers and cytokines were not analyzed in the patients, thus limiting our analysis.

CONCLUSION

It can be concluded that abnormal platelet count can be used as a marker of poor prognosis in patients with CAP. Thrombocytosis is associated with pulmonary complications and thrombocytopenia with sepsis and mortality. Platelet count also correlates with the time taken for recovery. Abnormal platelet count is a better predictor for severity and mortality than abnormal leukocyte count. CRP cannot predict severity and had a significant negative correlation with platelet count in our study.

REFERENCES

1. World Health Organization. Causes of Child Mortality, by Country, 2000-2010, Global Health Observatory (GHO) Data. Geneva: World Health Organization; 2010. Available from: <http://www.who.int/gho/child-health/mortality/mortality-causes-text/en>. [Last assessed on 2020 Nov 20].
2. Park K. Acute respiratory infections. In: Park's Text Book of Preventive and Social Medicine. 20th ed. Jabalpur: M/s Banarasidas Bhanot Publisher's; 2009. p. 151-9.
3. Mortensen EM, Coley SM, Snger DE, Marrie TJ, Obrosky DJ, Kapoor WN, *et al.* Causes of death for patients with community acquired pneumonia: Results from the pneumonia patients outcomes research team cohort study. *Arch Intern Med* 2002;162:1059-64.
4. Ruhnke GW, Perraillon MC, Cutler DM. Mortality reduction among pneumonia patients still substantial despite the impact of coding changes. *Am J Med* 2013;126:266-9.
5. Cilloniz C, Ewig S, Polverino E, Almagro CM, Marco F, Gabarrus A, *et al.* Pulmonary complications of pneumococcal community acquired pneumonia: Incidence, predictors, and outcomes. *Clin Microbiol Infect* 2012;18:1134-42.
6. Atwa ZT. Usefulness of gender and abnormal blood count for predicting pneumonia outcome in children. *Egypt J Chest Dis* 2015;64:169-74.
7. Yeaman MR. The role of platelets in antimicrobial host defense. *Clin Infect Dis* 1997;25:951-68.
8. Smyth SS, Mc Ever RP, Weyrich AS, Morrell CN, Hoffman MR, Arepally GM, *et al.* Platelet colloquim participants. platelet functions beyond hemostasis. *J Thromb Haemost* 2009;7:1759-66.
9. Katz JN, Kolappa KP, Becker RC. Beyond thrombosis: The versatile platelet in critical illness. *Chest* 2011;139:658-68.
10. Hesham AA, Heba HA. Thrombocytosis at time of hospitalization is a reliable indicator for severity of CAP patients in ICU. *Egypt J Chest Dis Tuberc* 2012;61:145-9.
11. Espan PP, Capelastegui A, Quintana JM, Soto A, Gorordo I, Garcia-Urbaneja M, *et al.* A prediction rule to identify allocation of inpatient care in community acquired pneumonia. *Eur Respir J* 2003;21:695-701.
12. Siegel RE. Clinical opinion prevails over the pneumonia severity index. *Am J Med* 2005;118:1312-13.
13. Yeaman MR, Bayer AS. Antimicrobial peptides versus invasive infections. *Curr Top Microbiol Immunol* 2006;306:111-52.
14. Brogly N, Devos P, Boussekey N, Georges H, Cliche A, Leroy O, *et al.* Impact of thrombocytopenia on outcome of patients admitted to ICU for severe community acquired pneumonia. *J Infect* 2007;55:136-40.
15. Feldman C, Kallenbach JM, Levy H, Reinach SG, Hurwitz MD, Thorburn JR, *et al.* Community acquired pneumonia of diverse aetiology: Prognostic features in patients admitted to an intensive care unit and a severity of illness score. *Intensive Care Med* 1989;15:302-7.
16. 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;44:27-72.
17. Brain BJ, Bates I, Laffan M, Lewis S. Reference ranges and normal values. In: Dacie and Lewis Practical Hematology. 11th ed. London, United Kingdom: Churchill Livingstone; 2011. p. 16-7.
18. Usha D, Rudrappa S. Significance of thrombocytosis in lower respiratory tract infections. *Med Pulse Int Med J* 2014;1:470-5.
19. Prina E, Ferrer M, Ranzani OT, Polverino E, Cilloniz C, Moreno E, *et al.* Thrombocytosis is a marker of poor outcome in community-acquired pneumonia. *Chest* 2013;143:767-75.
20. Wolach B, Morag H, Drucker M, Sadan N. Thrombocytosis after pneumonia with empyema and other bacterial infections in children. *Pediatr Infect Dis J* 1990;9:718-21.
21. Mirsaedi M, Peyrani P, Aliberti S, Filardo G, Bordon J, Blasi F, *et al.* Thrombocytopenia and thrombocytosis at time of hospitalization predict mortality in patients with community-acquired pneumonia. *Chest* 2010;137:416-20.

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