e-ISSN: 0975-1556, p-ISSN:2820-2643

Available online on www.ijpcr.com

International Journal of Pharmaceutical and Clinical Research 2023; 15(2); 954-960

Original Research Article

Mean Platelet Volume (MPV) as a Prognostic Marker of Severe Pneumonia

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Received: 29-12-2022 / Revised: 10-01-2023 / Accepted: 10-02-2023

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Conflict of interest: Nil

Abstract

Background: Platelets have historically been thought of as hemostatic and thrombotic factors, but their relevance in inflammation and immunity is rapidly growing. It has been established that platelet form and function may change significantly in response to pathological and physiological signals. The commonly used metric of platelet size and activity known as mean platelet volume (MPV) is easily accessible and precise. In patients with severe pneumonia (SP), association among mean platelet volume (MPV) and in-hospital mortality is unclear, despite the fact that MPV looks to be related with a bad prognosis of pneumonia.

Methods: In this prospective hospital based observational study, 288 severe pneumonia patients from, 1st July 2021 to 30th June 2022, were included and cut off MPV was taken as 10.5 fl.

Results: 34 incidences of fatality were noted while patients were hospitalized. 28 from high MPV group and 6 patients from low/ normal MPV group. Statistical analysis suggests that High MPV >10.5fl and normal or low MPV </=10.5fl related with poor outcome in severe pneumonia cases comparing with normal or low MPV (</= 10 fl) with P<0:001.

Conclusion: High MPV level is an independent risk factor for in-hospital mortality in patients with severe pneumonia.

Keywords: Severe Pneumonia, Mortality, MPV

Introduction

Lung parenchyma inflammation is known as pneumonia. Children across the world experience a high rate of morbidity and death due to pneumonia [1]. 7,40,180 children die from pneumonia in 2019, which is 14% of all deaths in children under 5 years old. With up to 0.26 episodes per child-year, Southeast Asia has highest incidence of pediatric pneumonia in world (the global incidence is 0.19 episodes per child-year) [2]. According to the WHO recommendations, "very severe pneumonia" is defined as children who have Tachypnea and danger sings (such as chest indrawing, cyanosis, refusal to feed) [3]. Many biochemical markers, including cytokines such tumour necrosis factor and interleukin 6 (IL-6), procalcitonin, and D-dimer C-reactive protein (CRP), have been studied in relation to clinical prognosis in patients with

severe pneumonia. The key significance of cytokines as mediators in lung's defensive systems against infections and inflammation is well documented. Cytokines may have pro- or anti-inflammatory effects depending on range of interconnected microbiologic, environmental, and genetic factors that are hypothesized to influence host response to respiratory infections. An increasing collection of clinical data points to the importance of platelets in the inflammatory response.

Apart from hemostatic and thrombotic functions, platelets are gaining more acceptance now a days because of their role in inflammation [4]. Platelet shape and function have been shown to significantly change in response to physiological and pathological indications [5-8]. Increased platelet activity and hence more acute inflammation are indicated by a higher MPV value [9]. To our knowledge, these alterations have never been investigated in relation to the prognosis of severe pneumonia in children. MPV changes have been investigated in number of chronic inflammatory disorders [10].

Determine prognostic value of MPV in patients with severe pneumonia who were admitted to PICU at Silchar Medical College & Hospital was aim of our study.

Materials and Methods

In compliance with the Helsinki Declaration, the local ethics committee accepted this study. Each participant's parent or legal guardian gave their informed permission. The PICU at Silchar Medical College & Hospital, which serves as referral tertiary care facility for Assam's Barak Valley, was the site of this prospective research. Study period was 1st July 2021 to 30th June 2022.

Inclusion criteria: All patients admitted to PICU who have a history of severe pneumonia and are between ages of one month and five years.

Exclusion criteria: Individuals with established haematological disorders and those with congenital cardiac disease.

All patients were diagnosed with pneumonia using WHO case definition; any child presenting with coughing or breathing issues was considered eligible if they also had abnormal chest radiograph (consolidation or perihilar infiltrates) and tachypnea (>60/min age 1-2 months, >50/min age 2-12 months, >40 age 12-59 months).

Patients with severe pneumonia were hospitalized and detail history and clinical examination was done during admission. Patients were categorised into severe pneumonia according to WHO criteria. Chest x ray was done for all patients. Under all aseptic and antiseptic precaution 2ml blood sample were taken in EDTA vial and send to Pathology department for Complete blood count and differential counts. Samples were analysed by using auto analyser. MPV of patients were obtained from CBC reports and plotted in Excel a she*et al*ong with other demographic and clinical details. MPV of 10.5 fl was taken as a cut off [7]. Outcome was determined by in-hospital mortality.

Statistical analysis: Analyses were performed using Statistical Package for Social Sciences, version 17.0. (SPSS for Windows 17.0, Inc., Chicago, IL, USA). Categorical variables were represented as numbers and percentages whereas numerical data were given as mean standard deviation. Using T-test, group comparisons were done. Using ROC analysis, cut-off MPV value that best differentiates between the surviving and expired group was identified. Sensitivity and specificity values were then derived. Statistical significance was defined as a p-value<0.05.

Results

Based on inclusion and exclusion criteria, 288 of the 392 patients who were admitted to the PICU between June 2021 and May 2022 had baseline clinical features that qualified them for research participation. The male to female ratio was 1.3, and average patient age was 13.5 +/- 0.5 months. According to in-hospital mortality, Table 1 presents the baseline demographic, clinical, and biochemical data for each group. The survival and non-survival groups' entrance levels of total count, hemoglobin, and platelet count did not substantially differ from one another.

However, a substantial difference between the groups of survivors and non-survivors was seen in mean platelet volume (MPV).

Table 1: Comparison of various parameters and laboratory values between survived and non-survived patients

Variables	Total (Mean+/-SD)	Survived (Mean+/-SD)	Non-survived (Mean+/-SD)	p value
Age (months)	13.5(+/-0.5)	15.2(+/-0.45)	6.8 (+/-0.55)	<0.05
Sex M:F	1.3	1.24	1.34	
Respiratory rate	56(21)	56(18)	62(23)	>0.05
Heart rate	146(15.5)	138(15)	142(14)	>0.05
Total leukocyte count (cumm)	12500(3211)	12380(3200)	13455(3252)	>0.05
Neutrophils (%)	54.2(3.8)	52.2(4.8)	55.5(3.2)	>0.5
Lymphocytes (%)	40(3)	41(7)	39(6)	>0.5
Haemoglobin gm/dl	10.3(0.6)	10.3(0.5)	10.4(0.6)	>0.5
Platelet count (lakh/cumm)	2.8(0.2)	3(0.3)	2.5(0.4)	>0.5

Out of 288 patients in the study group, 34 patients expired during the course of treatment, 11.8% mortality.

MPV in survived and non-survived group: Mean MPV of 288 patient was 9.39 (0.98). But among survivors and non-survivors MPV was 8.93 fl and 10.78 fl respectively which is statistically significant (Table 2).

Table 2: Mean MPV among survivors and non-survivors

Group	Mean MPV (SD)	p value
Survived	8.93 (0.94)	<0.05
Non-survived	10.78(1.06)	

Need for mechanical ventilation: Out of 288 patients, 42 patients (14.5%) required Invasive mechanical ventilation. Mean MPV among patients required ventilation was significantly higher as compared with patients received non-invasive ventilation (p <0.05) (Table 3).

Table 3: Comparison of MPV among patients received invasive and non-invasive ventilation

Need for ventilation	Number (%)	MPV (SD)	p value
Yes	42 (14.5)	10.28(1.01)	<0.05
No	246(85.5)	9.03 (0.92)	

Inotropes support and MPV: Total 38 patients received inotropes support during the course of treatment. Mean MPV was significantly higher in patients received inotropes (p <0.05) (Table 4).

Table 4: Mean MPV among patients received inotropes

Inotropes	Number (%)	MPV (SD)	p value
Yes	38(13.2)	10.58(1.02)	<0.05
No	250(86.8)	8.99 (0.94)	

MPV and in-hospital mortality: Performance of baseline MPV ROC Curve Analysis as predictor of in-hospital mortality AUC = 0:855, 95% ROC analysis revealed that MPV had limited capacity to predict in-hospital mortality, with sensitivity of 87.4% and specificity of 77.4% at cut-off of 10.5 (CI: 0.789-0.920, P <0:001) (Figure 1).

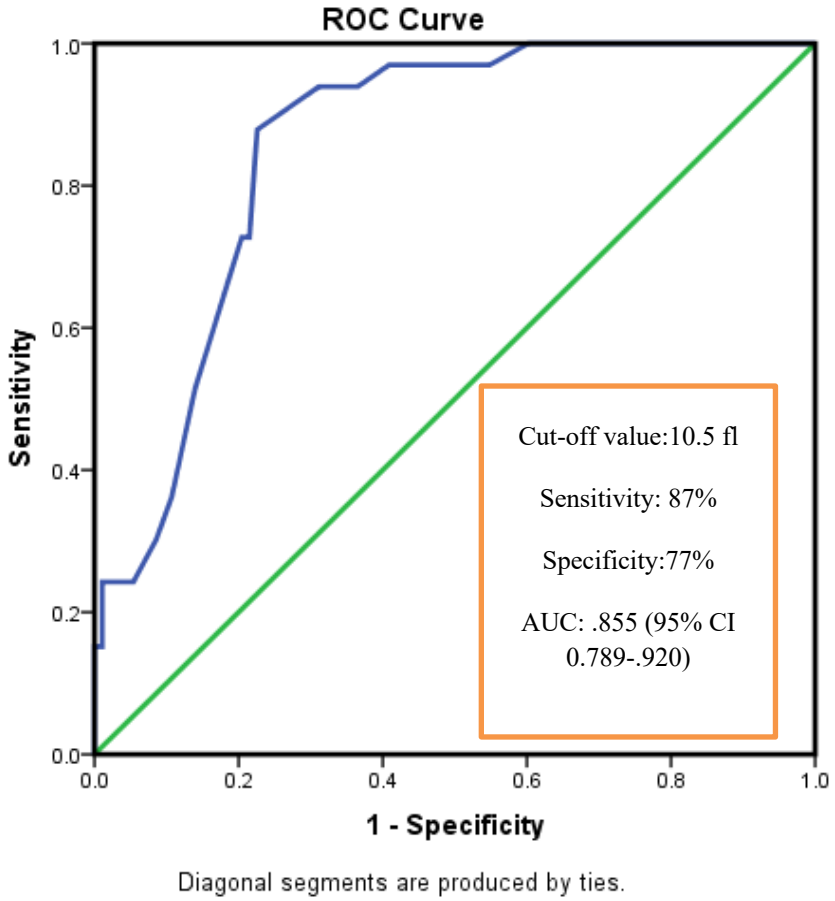

Figure 1: ROC curves of MVP for predicting in-hospital mortality.

Discussion

In this study, we found that in-hospital mortality rate for patients with severe pneumonia was significantly linked with MPV > 10.5 fl. With AUC of ROC 855 (95% CI 0.789-.920), MPV> 10.5 fl was independent predictor for in-hospital mortality and had respectable predictive ability for it.

In cases of severe infection, MPV has been reported to have prognostic significance for death. According to Gao *et al* research, MPV showed the best predictive potential for in-hospital mortality in septic shock patients among platelet indices [11]. The fact that they also found that 10.5 was optimal MPV cutoff level for in-hospital mortality is noteworthy to note. Aydemir *et al*. [12] discovered significant correlation between MPV and fungal sepsis.

Kim *et al*. found that individuals with severe sepsis or septic shock had elevated MPV as an independent risk factor for poor outcomes [13], which is consistent with other investigations. Only a small number of studies were done on children with severe pneumonia; the rest were all done on adults. Few studies, meanwhile, have shown a connection between MPV and SP patients' outcomes. Our research revealed a possible link between MPV and the prognosis for severe pneumonia.

In contrast to our study, where we discovered that the mean MPV of all pneumonia patients was 9.39 fl, The MPV level cut-off point for diagnosing CAP was found to be 8.1 fL in research on children done by Oncel K E *et al* [14], with sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) of 91%, 51%, 80.8%, and 70.5%, respectively. However, no studies have been done to determine cut-off point for in-hospital mortality. According to research by Chen J. *et al*., high MPV levels are standalone risk factor for in-hospital death in patients with SP [15]. This opinion is consistent with our findings. Their study's MPV (cut-off 10.5 fl) sensitivity and specificity for predicting mortality were 73.02 & and 73.08%, respectively, which is less than our study's sensitivity and specificity.

The underlying causes of increased MPV and a poor prognosis in individuals with severe pneumonia are yet unknown. It could be as result of the fact that platelet activation was crucial to development of severe pneumonia. After being exposed to platelet agonists, platelets are released from previously activated injured cells, platelets, or other inflammatory factors.

Following activation, platelets contribute to immune system in several ways, including direct interaction with pathogens, regulation of inflammatory responses by innate and complement immune systems, contact with leukocytes, and involvement in specific immune system [16]. A proven biomarker of platelet activation, MPV measures platelet size. Higher MPV platelets are bigger and more active, which suggests a thrombotic and inflammatory status [13].

Due to enhanced thrombopoietin release and significant amount of inflammatory elements present in severe infection, platelet activation will be boosted and more large platelets will be released into blood [17]. Because bigger platelets generate more procoagulant surface proteins and internal thromboxane A2, they exhibit greater prothrombotic potency (TXA2). The pulmonary vascular endothelium, which is well known to play key role in development of acute lung injury brought on by severe infection, can also be activated by TXA2 [16].

Additionally, it was discovered in this study that higher MPV level can linked to a greater in-hospital death rate in children. The development of big platelets is associated with the progression of inflammatory state, maybe as outcome of intracellular production of proinflammatory and procoagulatory substances and activation of the platelet pool.

Conclusion

Limitations of the current research must undoubtedly be taken into account. First off, this study only involved one location and one race of people. Results may be impacted by possible bias and lingering confounders. It has to be determined if these results might be generalized to other racial populations. Second, only MPV upon admission was included; whether more prognostic information would have been conveyed by repeated assessments while hospitalized is questionable. Third, it was argued that MPV alone was insufficient to predict death due to the very low AUC of 0.855. Further research is required to control predictive significance of the MPV combined with other mortality prediction models.

Fourth, there is insufficient information on other inflammatory indices, such as cytokines and procalcitonin, thus it is unclear how well MPV predicts outcomes when compared to these indices.

To summarize, primary flaw of this study is very small sample size. Making a firm conclusion in this study is challenging because of the tiny sample size.

For the association between MPV and severe pneumonia to be confirmed, there is a need for more extensive multicenter prospective research. According to results of our study, higher MPV at PICU admission was associated with higher incidence of pediatric in-hospital mortality.

MPV, as a straightforward, affordable, and generally accessible biomarker, may be a possibility for risk classification in patients with severe pneumonia.

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