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Original Article

Could platelet to leucocytic count ratio (PLR) predict sepsis and clinical outcomes in patients with emphysematous pyelonephritis?☆

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ABSTRACT

Objectives: To study risk factors for sepsis and mortality evaluating the role of platelet to leucocytic count ratio (PLR) as a marker for urosepsis and clinical outcomes in cases of emphysematous pyelonephritis (EPN).

Materials: Patients with EPN were retrospectively reviewed. Patients' age, sex, diabetes mellitus (DM), Body Mass Index (BMI), hydronephrosis, types of EPN, air locules volume, serum creatinine, leucocytic count, and platelet count, PLR, albumin, INR and the line of treatment were analyzed as risk factors of sepsis. Correlation between PLR and other variables was done using Pearson correlation coefficient. Univariate and multivariate analyses for sepsis and mortality were performed.

Results: Of fifty four patients, 38 patients had SIRS ≥2 criteria on admission. Twenty patients developed sepsis requiring ICU admission. In univariate analysis, male gender, lower BMI, higher INR, higher WBCs count and lower PLR were associated with sepsis ($P = 0.0001$, 0.009, 0.04, 0.003 and 0.001, respectively). In multivariate analysis, PLR ≤18.4, male sex and BMI ≤24.2 were independent risk factors. Lower PLR directly correlated with serum albumin ($P = 0.01$) and inversely correlated with serum creatinine and random blood glucose level and Klebsiella infection ($P = 0.001$, 0.007 and 0.005, respectively). Also, it was correlated with a higher total score of qSOFA and SOFA ($P = 0.02$ and 0.04). Lower PLR was independent risk factors for death in EPN patients with ($P = 0.003$).

Conclusion: EPN is associated with sepsis development. Lower PLR is an independent simple predictor for sepsis and mortality in patients with EPN.

1. Introduction

Emphysematous pyelonephritis (EPN) is a life-threatening necrotizing infection of the kidney characterized by accumulation of gas in renal parenchyma [1]. It is commonly seen in those with urinary tract obstruction with immune deficiency especially diabetic patients [2]. Bacteremia present in more than 50% of the EPN patients [3] and rapid evolution to sepsis is not uncommon. Even more, it may be the presenting feature in certain cases [4]. Septic shock had become the most common cause of mortality in intensive care units (ICU) ranging from 25% to 80% over the last few decades [5].

Diabetes mellitus (DM) is a common leading metabolic disorder to EPN. It is known to interrelate with the innate immune system with inhibiting effects on the adaptive immune system. Amongst patients with sepsis, around 20% have DM [6]. Without proper control of DM, sepsis occurs along with EPN, complicated by multi-organ dysfunction or even shock [7]. In United States, hospital and ICU admission due to pyelonephritis has high economic influence of nearly $17 billion annually [8].

Early recognition and proper management of EPN is important to avoid its undesirable consequences. Management of EPN varies from antibiotic treatment with or without renal drainage up to

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urgent nephrectomy. Over the past two decades, improved management strategies have resulted in a reduction of mortality among EPN from 78% to 13.5% [9–11].

Using of some markers like Procalcitonin, C-reactive protein (CRP) and lactate dehydrogenase (LDH) as an indicator of bacterial-induced sepsis and septic shock with lower specificity and sensitivity and its higher cost remains a stumbling block for their wide use especially in low-income countries. Lower platelet to leucocytic ratio (PLR) was recently reported to be associated with significantly higher early post-radical nephrectomy infectious complications, in comparison to higher ratio [12]. Also, lower PLR was associated with immunity depleting disorders as DM and dialysis dependence. So, it can be a less-expensive and rapid tool predicting bacteremia, Systemic Inflammatory Response Syndrome (SIRS), septic shock, and consequently, improves outcome and decreases mortality associated with delayed management.

Herein, we evaluated the role of PLR together with other clinical parameters in predicting septic shock in patients with EPN.

2. Material and methods

After approval of institutional review board, patients with EPN admitted between January 2005 and December 2016 were retrospectively analyzed. EPN was diagnosed clinically by fever with a positive urine culture and/or pyuria without other identified infectious foci; and radiologically by gas accumulation in the renal parenchyma, perinephric or pararenal space; in absence of uro-enteric fistula or recent history of trauma or catheterization [10]. EPN classified according to the extent of air accumulation in the kidney and perinephric tissue in CT scan as one of the following grades: type 1, gas in the collecting system only; type 2, gas in the renal parenchyma without extra-renal space involvement; type 3A, perinephric space extension of gas or abscess; type 3B, pararenal space extension of gas or abscess; and type 4, bilateral EPN or a solitary kidney with EPN [13].

In all cases, conservative treatment; including broad spectrum antibiotics (3rd generation cephalosporin) and IV fluids, were started till the availability of urine culture and changed accordingly. Persistence of fever $\geq$3 days and ureteric obstruction with proximal hydronephrosis were an indication for renal drainage by either percutaneous nephrostomy (PCN) or JJ stent. Urgent nephrectomy was performed in case of failure of all previous management. We defined the success of conservative treatment as improvement in clinical condition, decrease in air locules volume in follow-up images, and discharge alive.

Demographic criteria of all patients were retrieved at the time of hospital admission including age, sex, presenting symptoms, past medical history disease as presence of diabetes mellitus, hepatic dysfunction and chronic use of glucocorticoids affecting PLR. Body mass index (BMI), the organism causing infection, serum creatinine, hemoglobin, leucocytic count, platelet count, PLR, International Normalized Ratio (INR), albumin, presence of hydronephrosis, ureteral obstruction, and types of EPN, air locules volume were also retrieved. Air locules volume was estimated in CT by summation of air locules volume using the following formula (Width (W) X Height (H) X Length (L) x 0.52) on 2 dimensions [14].

2.1. Outcomes

The primary outcome was to determine the independent predictors of sepsis and ICU admission including PLR. All variables at the time of hospital admission were used for analysis. We used SIRS criteria as a screening tool for sepsis at the time of hospital admission, SIRS was defined as the presence of two of the following diagnostic criteria: abnormal temperature, heart rate, respiratory rate, and leukocyte count. Presence of $\geq$2 criteria was defined as positive (+ve) SIRS. Also, the correlation between PLR and other clinical variables; SOFA and qSOFA was performed. SOFA scores (range, 0 [best] to 24 [worst] points), and qSOFA scores (range, 0 [best] to 3 [worst] points) were recorded within the first 24 h of ICU admission [15]. Septic shock was defined as life threatening organ dysfunction caused by a dysregulated host response to infection. It is known that qSOFA had poor sensitivity and moderate specificity in comparison of SIRS criteria in mortality prediction and early sepsis detection at ward admission [16,17]. The secondary outcome was to determine the effect of septic shock in patients with EPN including ICU mortality.

2.2. Statistical analysis

Continuous data were expressed as mean ± SD or median and range when appropriate. Univariate analysis of predictors of sepsis was done using chi-square test, Student's sample *t*-test or Mann Whitney *U* test as appropriate. Receiver operating characteristics (ROC) curve was used to identify cut-off values for significant continuous variables in univariate analysis. Multivariate analysis was done using logistic regression analysis. Correlation between PLR and continuous and nominal variables was done by Pearson's correlation coefficient. All statistical tests were carried out using IBM "SPSS" statistics version 21, with a P value < 0.05 was considered significant.

3. Results

This study included 54 patients (12 males and 42 females) with EPN. The mean age ± SD was 48.8 ± 10 years. Only one patient had a past medical history of compensated liver disease and no patients were maintained on glucocorticoids. On hospital admission, only 4 patients had thrombocytopenia (platelet count $\leq$ 120,000) and 5 patients had thrombocytosis (platelet count $>$ 450,000). Thirty eight (70.3%) patients had + ve SIRS at time of admission. Twenty (37%) patients (10 males and 10 females) developed septic shock and required ICU admission. Eighteen patients were admitted to ICU before any intervention and two after PCN drainage of the affected kidney. The onset of sepsis was at a median (range) of 2 (1–6) days after hospital admission. Patients with no SIRS (30.7%) at presentation were managed successfully by conservative treatment and PCN drainage (eight patients each).

At time of hospital admission, in univariate analysis, male gender, lower BMI, higher INR, higher leucocytic count, and lower PLR were associated with a higher risk of sepsis (P = 0.0001, 0.009, 0.04, 0.003 and 0.001, respectively). Neither the types of EPN, air locules volume, positive urine culture, DM nor the lines of treatment were statistically significant predictors of sepsis (Table 1).

Using ROC curve, WBCS $\geq 18 \times 10^3$/cc, INR $\geq$1.3, PLR $\leq$18.4 and BMI $\leq$24.2 kg/m^2 were the cut-off values associated with the higher sensitivity and specificity of prediction of sepsis (Fig. 1a–d). In multivariate analysis, PLR $\leq$18.4 was an independent risk factor for sepsis with 5.1 (1.2–3.7) OR (95% CI) (P = 0.01). Also, male gender and BMI $\leq$24.2 kg/m^2 were associated with 1.9 and 2.6 folds risk of sepsis development (Table 1).

PLR was directly correlated with serum albumin at admission (r = 0.3, P = 0.01) and inversely correlated with serum creatinine (r = − 0.5, P = 0.001), higher random blood glucose level (r = −0.4, P = 0.007) on admission. Urinary tract infection with Klebsiella pneumonia was associated with lower PLR in comparison to other organisms (P = 0.005) (Table 2). On the other hand, there was no correlation between PLR and patients' age, sex, hydronephrosis or the types of EPN.

Table 1
Univariate and multivariate analysis of predictors of septic shock in EPN patients presented with SIRS. (*T-test **Chi-Square test # Mann-Whitney *U* test).

Variables	Univariate analysis			Multivariate analysis	
	Septic shock	No septic shock	P value	OR (95% CI)	P value
Sex. #					
Male	10	0			
Female	10	18	0.0001	1.9 (1.6–6.7)	0.04
BMI. *Mean ± SD**	26.5 ± 8.7	37.9 ± 6.9	0.009	2.6 (1.1–1.8)	0.02
WBCs *(10^3/cc)*Mean ± SD*	22.9 ± 8.5	15.6 ± 4.7	0.003	5 (1–8.3)	0.06
INR**Mean ± SD*	1.5 ± 0.3	1.1 ± 0.1	0.04	4.3 (0.8–8.3)	0.08
PLR *Median (range)*#	10.7 (0.92–18.8)	19.8 (6.5–35)	0.001	5.1 (1.2–3.7)	0.01

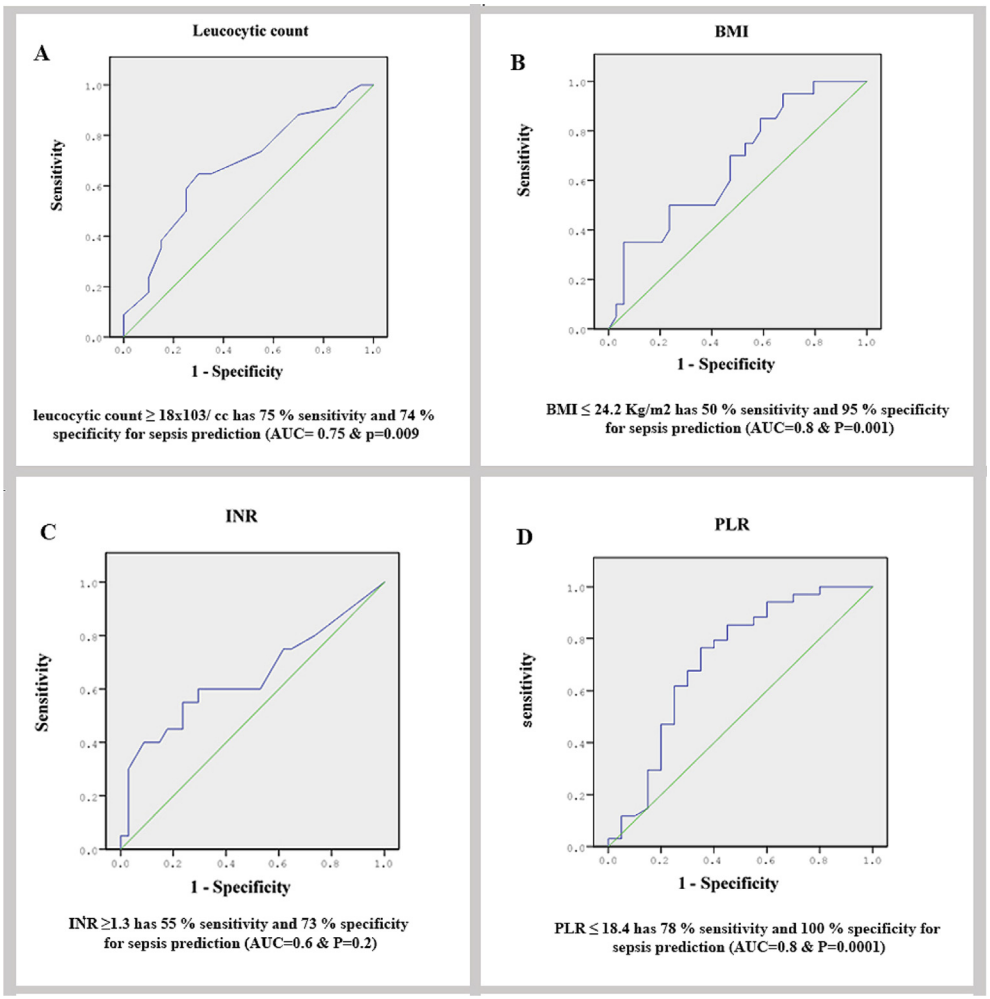

Fig. 1. ROC curves (A) leucocytic count, (B) BMI (C) INR and (D) PLR with cut-off. Values for higher sensitivity and specificity for sepsis prediction.

Among patients with septic shock in the first 24 h in ICU, the mean ± SD SOFA and qSOFA score were 8 ± 2.8 and 2.2 ± 0.6, respectively (Fig. 2). Lower PLR ≤18.4 was correlated significantly to a higher both total scores of qSOFA and SOFA (P = 0.02 and 0.04). Six patients (30%) had Disseminated Intravascular Coagulopathy (DIC) diagnosed by screening tests: prolonged Prothrombin time (PT) and thrombocytopenia. Lower PLR was significantly correlated with presence of DIC (r = 0.45, P = 0.04) (Table 2).

Five (25%) patients (4 male and one female) died in ICU because of irreversible septic shock, DIC with unrecovered multi-organ failure. Presence of renal stone, higher serum creatinine, lower BMI and lower PLR were statistically significant predictor of mortality amongst patients with septic shock in univariate analysis (P = 0.03, 0.02, 0.03 and 0.006, respectively). In multivariate analysis, lower PLR was the only independent risk factor for death in EPN patients [P = 0.003, OR (95% CI) = 1.4 (1.7–7.8)] (Table 3). Assessing the correlation between mortality and PLR, total score of SOFA, qSOFA, strong correlation was found between increased mortality rate and total score of SOFA (r = 0.5, P = 0.02) and PLR (r = −0.6, P = 0.004) (Table 2). No correlation was found between mortality and total score of qSOFA.

4. Discussion

Urosepsis is a life threatening condition of improperly managed EPN. It is a common cause of ICU admission in urology department,

Table 2
Correlation between lower platelet/leucocytic ratio (PLR) & risk factors in septic shock development for EPN patients at hospital admission and lower PLR and DIC, SOFA and qSOFA at ICU admission.

Variable	Lower PLR r (P value)
a. At hospital admission	
a. Serum albumin	0.3 (0.01)
b. Serum creatinine	−0.5 (0.001)
c. Random blood glucose level	−0.4 (0.007)
d. Urinary tract infection with klebsiella pneumonia	0.7 (0.005)
b. At admission in ICU	
a. Presence of DIC	0.45 (0.04)
b. qSOFA score	−0.7 (0.02)
c. SOFA score	−0.6 (0.04)
d. Mortality	−0.6 (0.004)

Correlation was performed by Pearson's correlation coefficient.

which increases the burden on the health authorities, especially in developing countries. With early detection and aggressive management as the accepted core to best practice for this patient population, the use of SIRS plus a known or suspected source of infection as the operational definition of sepsis has led to well documented reductions in both morbidity and mortality [17]. In the current study, 70% of EPN patients presented with +ve SIRS, of which, 52.6% required ICU admission because of urosepsis while 47.4% recovered without progression to sepsis. In a large prospective survey study, 48% of patients with SIRS developed septic shock [18]. Five patients (9.25%) died from unrecovered septic shock. In a large metanalysis discussing risk of mortality in 175 patients with EPN, incidence of mortality was 25%, ranging from 11% to 42% [2].

Leukocyte plays an important role in the systemic inflammatory response to infection. Most of the sepsis scoring systems use either leukocytosis (more than 12.0 × 10^3/L) or leukopenia (lower 4.0 × 10^3/L) as a severity index [19]. Similarly, we found that leukocytosis was a predictor of urosepsis in univariate analysis but did not reach statistical significance on multivariate analysis.

Platelets play an active role in antimicrobial host defense, inflammation and tissue repair induction as well as their contribution in hemostasis. Platelets can bind, internalize pathogens and release microbicidal proteins. By making cell to cell contacts with leukocytes and endothelial cells, platelets support leucocytes in arrest and transmigration. On stimulation by bacteria or thrombin, platelets release its content of α-granules, which include bioactive peptides, such as chemokines and endothelial cells growth factors, smooth muscle cells and fibroblasts. Therefore, platelets may become a defense weapon when irritated by bacteria [20]. Thrombocytopenia usually occurs due to platelet consumption and/or disseminated intravascular coagulation in patients with severe cases of EPN-induced sepsis beside thromboasthenia which lead to hematological and coagulation failure [11,13].

PLR is a new marker that copies patient's baseline health status. Patients with lower ratio were more likely to have more comorbidity [20,21]. Additionally, this ratio is likely an indicator for metabolic syndrome, physiological and biochemical abnormalities causing activation of inflammatory pathways [22]. In recent studies, lower PLR was associated with poor outcomes in many diseases like colon, lung, and testicular germ cell tumors. Also, it is associated with higher mortality in myocardial infarction and post-operative complications for ovarian cancers surgeries [12]. Garbens et al. correlated the rate of UTI and sepsis post radical nephrectomy with the PLR and reported that higher PLR was associated with lower risk of 30-day post-operative infectious complications (P < 0.0001) [12]. In another study, Weng et al. [23]

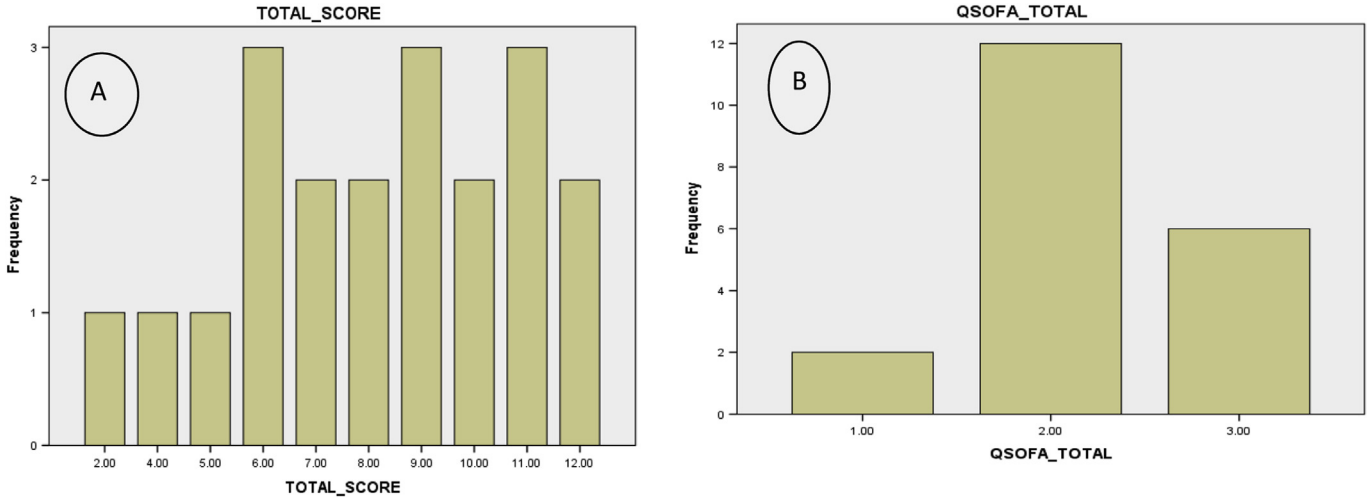

Fig. 2. Distribution of total a) SOFA and b) qSOFA scores in ICU patients.

Table 3
Univariate and multivariate analysis of predictors of mortality among septic EPN patients. (*T-test # Mann-Whitney *U* test).

Variable	Univariate analysis			Multivariate analysis	
	Survivors (n = 15)	Mortality (n = 5)	P value	OR (95%CI)	P value
BMI**Mean* ± *SD*	32.2 ± 9.6	25.6 ± 2.4	0.03	1.4 (0.8–2.3)	0.1
Presence of stone#					
Yes	1	4			
No	14	1	0.03	0.5 (0.04–3.2)	0.6
Serum creatinine# *Median (range) mg/dl*	1.4 (0.9–7.2)	7.3 (3.3–7.3)	0.02	1.3 (0.8–1.1)	0.06
*(PLT/WBC) ratio**	20.9 ± 6.3	11.6 ± 2.5	0.006	1.4 (1.7–7.8)	0.003
Mean ± SD					

proved that PLR less than 20 were associated with higher rate of wound infection and sepsis post splenectomy. In the current study, PLR less than ≤18.4 was an independent risk factor of septic shock development. Also, lower ratio was associated with increased risk of mortality among septic shock patients. Moreover, increased ratio with proper management among ICU admitted patients was associated with improved survival rate. It worth mentions that included patients had no evidence of other medical comorbidities which can affect PLR.

Lower PLR was correlated with lower albumin levels ($r = 0.3$, $P = 0.01$). Serum albumin is the most abundant protein in plasma. Its synthesis is suppressed in response to inflammatory conditions and malnutrition. Hypoalbuminemia (<3 gm/dl) was found to be associated with AKI and increased mortality and morbidity among surgery-exposed and ICU admitted patients [24]. But herein, serum albumin was not a statistically significant predictor of septic shock in EPN patients.

Also, Lower PLR was correlated with higher glucose level. Higher glucose level provides a favorable environment for the growth of gas-producing organisms and impairs the leucocytic function with higher rate of sepsis [25,26]. Yet, DM was not a predictor of septic shock development or the subsequent mortality in the present study.

Klebsiella infection was found as a risk factor for death and poor outcome in previous study [25]. But, here the type of organism was not a risk factor of developing urosepsis. This may be due to the limited number of patients and the different types of organisms encountered in the urine culture. But presence of Klebsiella infection correlates with lower PLR. PLR was inversely correlated with serum creatinine. In the current study, serum creatinine was not associated with increased risk of septic shock development and mortality. Similarly, Falgas et al. in a large meta-analysis documented serum creatinine wasn't independent factor for mortality in EPN patients [2]. DIC is a pathologic overstimulation of the coagulation system. Physiologic hemostasis turns to pathologic DIC when the prothrombotic response exceeds coagulation inhibitors and the fibrinolytic system. Any tissue insult sufficient enough to release tissue products or toxins into the circulation can result in DIC. Degree of coagulation factor consumption and activation can be screened by tests such as PT or platelet count [27]. It was proved that neutrophils in complex with platelets represent a subpopulation of neutrophils with a more activated adhesion molecule profile, and a greater power for phagocytosis [28]. During sepsis, there is an increase in circulating platelet-neutrophil complexes with initially rising during sepsis, and subsequently decrease when multiple organ failure develops, indicating peripheral sequestration and a possible causal relation [29]. This came with we found that there is a strong correlation between lower PLR and DIC in addition to higher SOFA and qSOFA scores.

The risk of sepsis is multi-factorial including age, health status, genetic predisposition and comorbidity. Impaired immunity is one of the important risk factor, because of immunosuppressive drugs, debilitating diseases, malnutrition or stressors such as infections. Khaira et al. reported that patients' age and sex are not risk factors for poor consequence and mortality in patients with EPN [5]. In our study, Male sex is associated with higher risk of evolving urosepsis and ICU admission in cases with EPN ($P = 0.0001$) but it wasn't associated with higher mortality ($P = 0.1$). This may be attributed to the late presentation in male cases and the protective effect of female steroid hormone [30].

Arabi et al. [31] demonstrated that mortality was inversely correlated with BMI. Similarly, overweight (OR 0.43; 95% CI 0.19–0.98; $P = 0.04$) and obese (OR 0.28; 95% CI 0.08–0.93; $P = 0.04$) patients had an independently lower risk of ICU death than those with normal weight [32]. Herein, lower BMI was independent predictor of septic shock development and associated with higher rate of mortality. It might be because the lower weight had more devastating illnesses. Also, we noticed that patients with lower BMI presented later with early onset of fever and having stones.

In addition, we found that renal stones were risk factors of sepsis-related mortality ($P = 0.03$). Stones act as an infectious nidus which causes persistent infection, sepsis and mortality. Previous studies found no correlation between presence of renal stones and increased mortality rate in patients with EPN [2,10]. Sanford et al. documented that EPN patients with stones were younger with lower serum creatinine and lower disease stages and lower mortality rate [33].

We noticed strong correlation between lower PLR ≤18.4 and higher total score of SOFA and qSOFA ($P = 0.04, 0.02$). Also higher total SOFA score in addition with lower PLR was found to be correlated with increased incidence of death. So, lower PLR at time of admission could be an alarm sign for a physician to start aggressive treatment with preparation for early drainage of the affected kidney in order to avoid the prospected adverse consequences.

To the best of our knowledge, this is the first study to assess the risk factors for septic shock development in EPN and assessing the impact of PLR on the clinical outcomes. Yet, it has many limitations. The limited number of patients because of the relative rarity of the disease. It is a retrospective study with inherent nature of selection bias. Lack of available data regarding inflammatory markers like CRP and LDH and differential leucocytic count (DLC) is one of the limitations. But this is because it was not a routine evaluation in our hospital.

5. Conclusion

Despite the advances in treatment strategies, EPN is associated with a significant risk of septic shock development. The available results showed the clinical importance of PLR as a novel marker of septic shock development and mortality in patients with EPN. It can be used as an alarming sign to start early anti-shock measures and drainage of the affected kidney.

Financial disclosure and conflict of interests

None.

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