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Can Mean Platelet Volume Predicts Renal Outcome in Acute on Chronic Kidney Disease?

Running title: Mean platelet volume and renal outcome

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The study protocol complied with the Declaration of Helsinki and was approved by the local ethics committee. (Afyonkarahisar Health Sciences University Clinical Research Ethics Committee, date: 04/03/2022, meeting number: 2022-3, decision no: 139).

The study is retrospective and archival records have been examined. For this reason, the patient consent statement is not available.

Abstract

Introduction

This study aimed to investigate the role of mean platelet volume (MPV) in predicting renal outcome in acute kidney injury (AKI) developing on pre-existing chronic kidney disease (CKD).

Methods

The patients whose first hemodialysis program was initiated in our center were divided into two groups as those who were taken to the scheduled dialysis program after discharge and those who were not dialysis-dependent. Groups were compared in terms of demographic characteristics, and laboratory parameters including MPV.

Results

A total of 288 patients were included in the study (scheduled dialysis=162 patients, non-dialysis dependent=126 patients). High MPV was found to be an independent risk factor for scheduled dialysis programs in multivariable analyses [OR (95%CI): 90.9(6.3-1313.6), p: 0.001].

Conclusion

CKD patients with high MPV were more likely to be included in scheduled dialysis programs after an AKI attack. MPV is found to be an independent risk factor and a reliable predictor for a scheduled dialysis program.

Keywords: Acute Kidney Injury; Chronic Kidney Disease; End-Stage Renal Disease; Mean Platelet Volume; Renal Replacement Therapy.

Introduction

Mean platelet volume (MPV), evaluated by hematological analyzers, is a parameter that provides information about platelet function and activation. It is thought to be associated with proinflammatory and prothrombotic diseases (1). Large platelets are more easily activated than small ones and secrete platelet factor-4, b-thromboglobulin, and thromboxane A₂ (1,2). There are studies that MPV may be associated with diseases such as non-alcoholic hepatosteatosis (NASH) (3), chronic obstructive pulmonary disease (COPD) (4), chronic viral hepatitis (5), and pulmonary embolism (PE) (6). There are also studies showing that high MPV levels are associated with acute myocardial infarction (AMI) (7), restenosis after coronary angioplasty (1), and ischemic stroke (8).

We know that chronic kidney disease (CKD) is associated with high cardiovascular morbidity and mortality. In addition, approximately 50% of deaths in patients receiving hemodialysis treatment are due to cardiovascular events (9). MPV has rarely been investigated in individuals with CKD. It is stated that increased MPV levels in patients with CKD may reflect the chronic inflammatory level (1). In addition, some studies that associate cardiovascular risk factors such as smoking, diabetes, hypertension (HT), hyperlipidemia, and metabolic syndrome with increased MPV levels (10). The combination of one or more of these risk factors in CKD patients may explain the elevated MPV levels (1).

Studies evaluating the relationship between acute kidney injury (AKI) and MPV are very limited. The mechanisms by which AKI causes CKD are not clear. Various mechanisms such as inflammation, hypoxia, nephron loss, endothelial damage, epigenetic changes in epithelial

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cells, problems in cellular cycles, and vascular sparseness have been suggested by researchers (11). Considering the aforementioned reasons, it is thought that acute damage may result in CKD and accelerate the existing chronic disease and pave the way for the progression to end-stage renal disease (ESRD) (12). In addition, some studies have shown that endothelial damage triggers hemostatic and inflammatory processes and activates platelets (13,14). It is stated that microvascular circulation problems and acute kidney injury may develop after triggered mechanisms (13).

It is known that cases of ESRD requiring permanent renal replacement therapy (RRT) are increasing worldwide. We think that there is a need for new parameters that will enable us to predict the current situation and guide our treatment strategies. In our study, we aimed to investigate the answer to the question of whether MPV is a predictor of renal outcome in acute injury in patients with CKD.

Material-Methods

Patients

Electronic medical records of patients with a baseline diagnosis of CKD and hospitalized for AKI on CKD between 01.01.2017 and 01.01.2022 were evaluated retrospectively. Our study was designed as a single-center study containing clinical data from the nephrology department. Patients having a history of malignancy, using immunosuppressive therapy, congestive heart failure, chronic hepatic disease, acute or chronic infection, sepsis, shock, major surgery, hematological disease, chronic obstructive pulmonary disease, and autoimmune disease were not included in the study. Aged between 18 years and 85 years, cases of acute kidney injury due to all causes other than exclusion criteria were included in the study. A hundred and forty-nine out of a total of 437 patients were excluded. The study was conducted with 288 patients. eGFR values were calculated with the 2021 CKD-EPI creatinine formula;

$$\text{eGFR} = 142 * \min(\text{standardized } S_{cr}/K, 1)^{\alpha} * \max(\text{standardized } S_{cr}/K, 1)^{-1.200} * 0.9938^{\text{Age}} * 1.012$$
 [if female].

Patients were divided into two groups scheduled dialysis programs and non-dialysis dependent patients according to the continuation of their dialysis needs 3 months after discharge.

All complete blood counts of patients were analyzed with an automatic analyzer (Cobas 6000, Roche, Switzerland). MPV measured at the beginning of hospitalization was evaluated. The normal reference range of MPV was; 9.4-12.4 Fl.

The study protocol complied with the Declaration of Helsinki and was approved by the local ethics committee. (date: 04/03/2022, meeting number: 2022-3, decision no: 139).

Definitions

Chronic kidney disease= CKD was defined as a GFR of less than 60 ml/min/1.73m² for at least 3 months.

Acute kidney injury on chronic kidney failure= Acute kidney injury on chronic kidney injury was defined as at least ≥ 0.3 mg/dl increase in creatinine or at least ≥ 1.5 times increase in creatinine from the baseline or < 0.5 ml/kg/h decrease in urine output for 6-12 hours.

Baseline CKD stage= Patients having an eGFR between 30-59 ml/min/1.73m² were considered stage 3 and patients having an eGFR between 29-15 ml/min/1.73m² were considered stage 4 CKD.

RRT at admission= Dialysis treatment was applied to patients with eGFR < 7 ml/min/1.73m² at the time of admission and severe metabolic acidosis, hypervolemia and hyperkalemia at the time of admission.

Scheduled dialysis program= In our country, clinical and laboratory evaluations for the need for dialysis after discharge are routinely performed by experienced nephrologists at least once a month. Patients who still needed dialysis 3 months after discharge and were still registered in the national dialysis registry system were identified as scheduled dialysis patients.

Non-dialysis dependent patients= Patients who do not need dialysis during hospitalization or whose dialysis requirement ends in the first 3 months after discharge.

Statistical analysis

Categorical variables were presented as percentages and frequency. Numerical variables were checked for normality with visual histograms and, analytical methods like Kolmogorov-Smirnov and Shapiro Wilk tests. Normally distributed numerical variables were presented as mean \pm standart deviation (SD), and non-normally distributed numerical variables were presented as median and IQR(interquartile range). Categorical variables were compared with

a chi-square test between groups. Numerical variables were compared with independent samples t-test if there is a normal distribution and Mann-Whitney U test if there is not. The ROC curve was constructed to determine the predictive power of MPV for the scheduled dialysis program. Youden index was used to determine the best cutoff value of MPV. Parameters found to be different ($p < 0.05$) between groups were taken into logistic regression analysis. The forward stepwise-Wald method was used and the model having the highest nagelkerke R^2 was presented. Conditions below 5% of the type 1 error level were interpreted as statistically significant. Analyzes were performed using SPSS 26.0 (IBM Corp. 2019 IBM SPSS Statistics for Windows, version 26.0. Armonk, NY: IBM Corp).

Results

Of 288 patients 52.1% ($n = 150$) were male and 47.9% ($n = 138$) female. The mean age was 64.1 ± 16.3 years. Of the patients, 56.3% ($n = 162$) were discharged with a scheduled dialysis program. Compared to non-dialysis dependent patients, patients discharged with a scheduled dialysis program were significantly younger and had higher systolic blood pressure, lower diastolic blood pressure, higher baseline CKD stage, higher history of stroke, higher rate of starting RRT at admission, and lower diuretic use ($p < 0.05$). Characteristic features of the patients were shown in Table I.

Serum urea, creatinine, potassium, phosphorus, PTH, MPV, CRP, and sedimentation were significantly higher in patients discharged with scheduled dialysis programs than non-dialysis dependent patients ($p < 0.05$). eGFR, calcium, hemoglobin, pH and HCO_3^- were significantly lower in patients discharged with scheduled dialysis programs ($p < 0.05$). Laboratory measurements were shown in Table II.

ROC analysis showed that MPV had a significant predictive power for renal outcome in our patients, with $\text{AUC} = 0.907$, 95% CI= 0.875-0.939, and $p < 0.001$ (Figure 1). An MPV value of 10.25 was found to have 78.4% sensitivity and 87.3% specificity for the renal outcome.

MPV was also found to have a significant predictive power of dialysis at admission, with $\text{AUC} = 0.811$, 95%CI= 0.725-0.896, and $p < 0.001$ (Figure 2). An MPV value of 9.95 was found to have 82.3% sensitivity and 66.7% specificity for dialysis at admission.

All of the 19 candidate parameters that were significantly different between groups were entered into a regression model as candidate predictors of renal outcome. A model constructed by age, history of stroke, baseline CKD stage, RRT at admission, eGFR, calcium,

MPV, and PTH had the highest R^2 value with 95.4%. It was found that younger age, lower eGFR, and lower calcium, stroke history, higher baseline CKD stage, RRT at admission, higher MPV, higher PTH are independent risk factors for scheduled dialysis programs (Table III).

Discussion

Mean platelet volume is a parameter that is automatically calculated and easily found in every blood count, as an important indicator of platelet activity. MPV is inherited at the locus on chromosome 7q22.3 (15). There are studies indicating that increased MPV levels may be a risk factor for general vascular mortality (16). In addition, it is stated by the authors that MPV not only provides information about coagulative processes but also reflects the inflammatory load (16). Considering the hypotheses put forward in terms of the importance of MPV, it can be said that there is a general consensus on inflammation and platelet stimulation (1). In some studies, it has been shown to increase megakaryocyte proliferation via interleukin-6 (16,17). Therefore, it is stated that increased MPV levels in critically ill patients may indicate the development of severe inflammation (16). It is known that more than 13 million people worldwide are affected by acute kidney injury and approximately 2 million people die annually (12). In a study conducted in cases with advanced AKI, patients receiving continuous RRT were followed up for 28 days and it was reported that MPV may be both a prognostic marker and a risk factor for mortality (16). However, studies are showing that MPV also reflects inflammatory load and disease activity in chronic inflammatory conditions (18). In our literature review, we did not find any study investigating the relationship between MPV and renal outcome in acute injury in patients with CKD. In our study, we found that the patient group discharged with scheduled dialysis treatment had higher MPV levels compared to the other patient group. We think that the inflammatory burden present in both acute and chronic processes is an important mechanism responsible for increased MPV levels. In the comparison between groups, we found significantly higher CRP and sedimentation levels in patients discharged with scheduled dialysis treatment. These data support our hypothesis. In a study comparing CKD stage 3-4 and renal transplanted cases in terms of CRP, it was reported that CRP levels were close to normal in the transplantation group (9). Another result from the same study was that the transplanted patients had the lowest sedimentation value among the compared groups (9)(3). In another study, it was observed that MPV levels decreased after kidney transplantation and this was associated with the disappearance of the chronic inflammatory state (19). Estimated glomerular filtration rate (eGFR) and CKD stage are

important parameters in our study. There are studies stating that there is a correlation between MPV and eGFR (20) and that MPV levels increase according to the CKD stage (1). In this respect, our results are similar to the literature. When we evaluate the data in terms of stroke; We observed that it was significantly higher in patients discharged with scheduled dialysis. This may be associated with an increased risk of stroke in advanced CKD cases. Many studies are showing the association of increased MPV levels with stroke (8,21). It is known that MPV can provide information about coagulative processes as well as inflammatory processes.

If we evaluate our data in terms of age; This can be explained by the loss of patients before they reach the ESRD stage (22) and the increased mortality rates in individuals over 65 years of age undergoing hemodialysis (23). Because the rate of development of thromboembolic events is high, including patients receiving RRT therapy in all stages of CKD (9). We know that cardiovascular events are the most important cause of mortality and morbidity in individuals with CKD (24). In addition, the data we obtained in terms of calcium and parathormone (PTH) in our study were similar to the previous studies. As is known, secondary hyperparathyroidism (sHPT) is an important complication of CKD. sHPT develops with the involvement of complex pathophysiological mechanisms such as an increase in serum phosphorus, fibroblast growth factor 23 (FGF-23) levels, and a decrease in serum calcium and active vitamin D levels in individuals with CKD (25). Although the prevalence of sHPT in CKD varies at stages 3-5, it has been defined as 20%-80% (26). In a study, it was reported that the incidence of sHPT was observed at the highest level in cases with advanced CKD (26). In this regard, we think that the presence of hypocalcemia and sHPT accompanying CKD in patients hospitalized for AKI may guide our treatment strategies.

We observed in our study that there is a significant relationship between the need for RRT at the time of hospitalization and scheduled dialysis after discharge. In a study by Chi-yuan Hsu et al; The patients were evaluated considering parameters such as basal eGFR, diabetes, HT, known proteinuria, coronary artery disease (CAD), stroke, and congestive heart failure (CHF) (27). At the end of the study, an acute episode of CKD was associated with a 30% increase in long-term risk for ESRD (27). In addition, some studies have reported that hypoxia, inflammation, fibrosis, and nephron loss that develop during an AKI attack may result in decreased GFR (11). It is stated by researchers that vascular sparseness, especially in the peritubular area, can predict interstitial damage and decreased GFR. However, it has been reported that vascular sparseness in the peritubular area is also associated with the severity of fibrosis (28). In addition, in a study investigating the relationship between CKD and MPV;

Higher MPV was encountered in progressive CKD cases and a negative correlation was shown between MPV and eGFR (1). Considering the aforementioned studies and our study data, CKD progression and scheduled dialysis requirements may be associated with increased MPV. As a result, we think that the more dramatic the severity and extent of inflammation, the greater the loss of renal functions.

It should be noted that there are some limitations to our study. MPV is routinely measured in our hospital within 2 hours following venipuncture using EDTA-containing tubes on automated analyzers. The potential effect of anticoagulants on MPV is unclear as the design of our study was retrospective. Because time-dependent swelling of platelets can be observed in EDTA-induced coagulation and may increase MPV (29). MPV measured with citrate tube is smaller than tube containing EDTA (30). In another limitation of our study, non-consecutive one-time MPV measurement during hospitalization may have increased the possibility of analytical disadvantages. Finally, the lack of a prospective study may have caused bias as well as affecting the cause-effect relationship.

Conclusions

In our study, we found higher MPV levels at the time of hospitalization in CKD patients discharged with scheduled dialysis. This situation can guide treatment strategies from the moment of hospitalization. In addition, inflammatory markers such as CRP and sedimentation were higher in the group discharged with scheduled dialysis compared to the other group at the time of the first admission to the hospital. These data may indicate a possible relationship between the severity of inflammation and renal survival. The third important finding in our study was the negative correlation between eGFR and MPV. Our fourth finding was that the relationship between stroke and MPV was consistent with the literature. Therefore, we think that patients with high MPV levels should be followed more carefully in terms of cerebrovascular events during the treatment period. Finally, we would like to point out that prospective studies with large numbers of patients are needed to more clearly establish the relationship between MPV and renal outcome in acute injury in CKD patients.

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Table I. Characteristics of the patients

Characteristics	Total (n= 288)	Scheduled dialysis (n= 162)	Non-dialysis dependent (n= 126)	p
Age, mean±SD	64.1±16.3	61.13±17.4	67.81±13.7	<0.001*
BMI, mean±SD	25.14±2.5	25.02±2.5	25.29±2.5	0.368
Sys-BP, mean±SD	136.01±20.5	140.9±23.6	129.69±13.4	<0.001*
Dia-BP, mean±SD	80.1±12.9	76.3±14.1	83.1±11.1	<0.001*
Male gender, %-n	52.1-150	50-81	54.8-69	0.476
DM, %-n	52.4-151	56.2-91	47.6-60	0.156
HTN, %-n	92.4-266	94.4-153	89.7-113	0.179
Basal CKD stage				
Stage 3, %-n	37.5-108	13-21	69-87	<0.001**
Stage 4, %-n	62.5-180	87-141	31-39	
CAD, %-n	37.5-108	35.2-57	40.5-51	0.391
Stroke, %-n	10.1-29	14.2-23	4.8-6	0.01**
RRT at admission, %-n	66.7-192	96.3-156	28.6-36	<0.001**
ACE inh., %-n	19.8-57	20.4-33	19-24	0.882
ARB, %-n	22.9-66	20.4-33	26.2-33	0.261
CCB, %-n	40.6-117	44.4-72	35.7-45	0.148
BB, %-n	34.4-99	33.3-54	35.7-45	0.708
Diuretics, %-n	28.1-81	22.2-36	35.7-45	0.012**
αB, %-n	16.7-48	16.6-27	16.7-21	1
Anti-Platelet, %-n	39.9-113	38.3-62	40.5-51	0.717
Anti-Coagulant, %-n	12.5-36	11.1-18	14.3-18	0.474

ACE= angiotensin-converting enzyme, ARB= angiotensin receptor blocker, BB= beta blocker, BMI= body mass index, CAD= coronary artery disease, CCB= calcium channel blocker, CKD= chronic kidney disease, dia-BP= diastolic blood pressure, DM= diabetes mellitus, HTN= hypertension, RRT= renal replacement treatment, sys-BP= systolic blood pressure, αB= alpha-blocker, *independent samples t-test,

**Fisher's exact test

Table II. Comparison of the patients in terms of laboratory tests

Laboratory measurement (median-IQR)	Total (n= 288)	Scheduled dialysis (n= 162)	Non-dialysis dependent (n= 126)	p
Urea (mg/dL)	133-78.9	141.7-71.5	121.6-76.4	< 0.001
Creatinine (mg/dL)	4.48-3.2	5.6-2.8	2.67-3.2	< 0.001
eGFR (mL/min/1.73m ²)	12-8.8	8.6-5	18-14	< 0.001
Sodium (mEq/L)	136-7	136-8	137-38	0.563
Potassium (mEq/L)	4.65-1.5	4.8-1.3	4.4-1.7	0.002
ALT (U/L)	14-10	14-8	14-12	0.654
Albumin (gr/dL)	3.48-1.01	3.61-0.9	3.43-1.2	0.204
Calcium (mg/dL)	8.5-1.3	8.39-1.2	8.68-1.3	0.003
Phosphorus (mg/dL)	4.88-1.4	5.3-1.2	4.6-1.2	< 0.001
PTH (ng/L)	128.9-229.5	148.9-254	105-144	0.029
Leukocyte (10 ³ /uL)	9465-3880	9360-3020	9515-4450	0.190
Hemoglobin (g/dL)	10.4-2.9	10-2.3	10.85-4	0.017
MPV (fL)	10.2-1.4	10.9-1.1	9.4-1.1	< 0.001
PLT (10 ³ /uL)	216.5-106.5	210-101	228.5-115	0.093
Glucose (mg/dL)	122-65.3	122.45-54.7	121.6-67.9	0.729
pH	7.33-0.13	7.32-0.12	7.36-0.12	< 0.001
HCO ₃ (mEq/L)	18.7-7.5	17.8-6.9	19.65-8.2	0.016
pCO ₂ (mmHg)	32.9-11.3	32.75-11.2	33.55-10.1	0.888
Lactate (mmol/L)	12-7	11.5-6	12-7	0.345
CRP (mg/dL)	4-7.65	5-7.6	3.5-5.6	< 0.001
Sedimentation (mm/h)	32-44.3	35-46	24-32	< 0.001

ALT= alanine aminotransferase, CRP= C-reactive protein, GFR= estimate glomerular filtration rate, MPV= mean platelet volume, PLT=

platelet PTH= parathormone

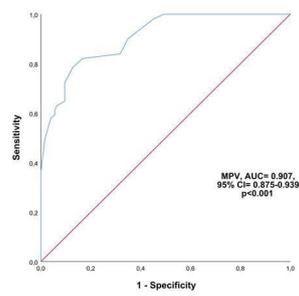
Table III. Univariate and multivariate regression analysis of predictive factors for scheduled dialysis program

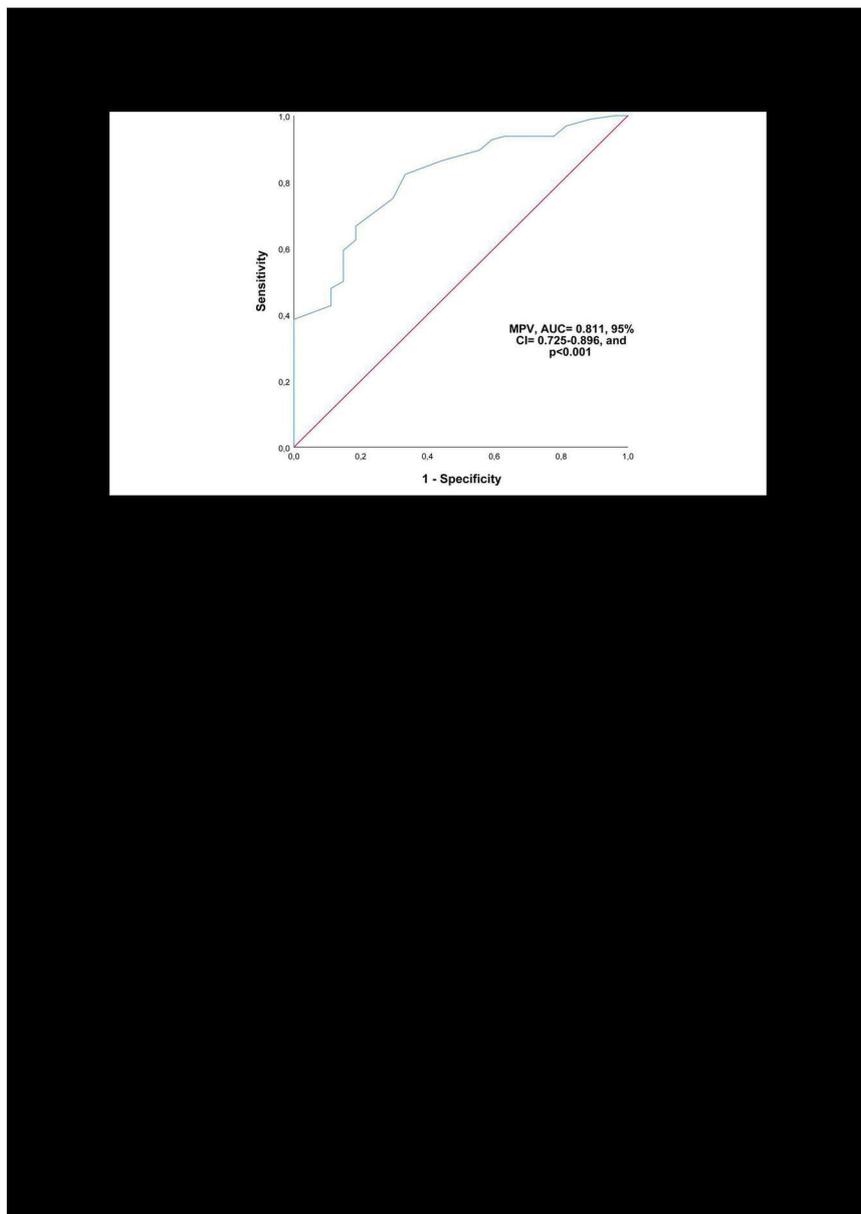
Parameters	Univariate		Multivariate	
	OR(95%CI)	p	OR(95%CI)	p
Age	0.973(0.958-0.988)	0.001	0.871(0.780-0.973)	0.015
Stroke	3.309(1.304-8.397)	0.012	59.1(1.93-1815.4)	0.019
Baseline CKD stage	14.9(8.3-27.1)	<0.001	93.7(4.03-2181.3)	0.005
RRT at admission	65(26.4-160.3)	<0.001	1141(19.9-65343.9)	0.001
eGFR	0.562(0.485-0.652)	<0.001	0.371(0.201-0.687)	0.002
Calcium	0.739(0.602-0.907)	0.004	0.167(0.038-0.715)	0.016
MPV	15.7(8.26-30.1)	<0.001	90.9(6.3-1313.6)	0.001
PTH	1.002(1.001-1.004)	0.002	1.008(1.001-1.016)	0.031

Figure legends

Figure 1. ROC curve of MPV for renal outcome

Figure 2. ROC curve of MPV for dialysis at admission





TAP_13935_Figure 2.jpg