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Association of Biomarkers Related to Preoperative Inflammatory and Coagulation with Postoperative In-Hospital Deaths in Patients with Type A Acute Aortic Dissection

Ming Li
Xi'an Jiaotong University
Haifeng Sun
Xi'an Jiaotong University
Suochun Xu
Xi'an Jiaotong University
Yang Yan
Xi'an Jiaotong University
Haichen Wang
Xi'an Jiaotong University
Jianjie Zheng
Xi'an Jiaotong University
Yongxin Li
Xi'an Jiaotong University
Yongjian Zhang
Xi'an Jiaotong University
Junjun Hao
Xi'an Jiaotong University
Chao Deng
Xi'an Jiaotong University
Xinglong Zheng
Xi'an Jiaotong University
Miaomiao Liu
Xi'an Jiaotong University
Yang Gao
Xi'an Jiaotong University
Xue Wang
Xi'an Jiaotong University
Li Xue (xuelially@163.com)
Xi'an Jiaotong University

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Abstract

Background: The aim of this study was to analyze the predictive value of biomarkers related to preoperative inflammatory and coagulation in the prognosis of patients with type A acute aortic dissection (AAD).

Methods: A total of 206 patients with type A AAD who had received surgical treatment were enrolled. Patients were divided into two groups according to whether they died during hospitalization. Peripheral blood samples were collected before anesthesia induction. Preoperative levels of D-dimer, fibrinogen (FIB), platelet (PLT), white blood cells (WBC) and neutrophil (NEU) between the two groups were compared. Univariate and multivariate logistic regression analysis were utilized to identify the independent risk factors for postoperative in-hospital deaths of patients with type A AAD. Receiver operating characteristic (ROC) curve were used to analyze the predictive value of D-dimer, FIB, PLT, WBC, NEU and CRP in the prognosis of the patients.

Results: Univariate logistic regression analysis showed that the *P* values of the five parameters including D-dimer, FIB, PLT, WBC and NEU were all less than 0.1, which may be risk factors for postoperative in-hospital deaths of patients with type A AAD. Further multivariate logistic regression analysis indicated that higher preoperative D-dimer and WBC levels were independent risk factors for in-hospital deaths of patients with type A AAD. ROC curve analysis indicated that FIB+PLT combination is provided with the highest predictive value for in-hospital deaths.

Conclusion: Both preoperative D-dimer and WBC in patients with type A AAD may be used as independent risk factors for the prognosis of such patients. Combined use of FIB and PLT may improve the accuracy and accessibility of clinical prognostic assessment.

1. Introduction

Type A AAD may lead to sudden deaths of patients if it was not diagnosed early and treated appropriately [1]. Currently, both inflammation and blood coagulation process have been found to play important roles in the pathogenesis of type A AAD. As prognostic indicators of patients with type A AAD, various biomarkers of inflammation and coagulation function have attracted more and more attention, since the predictive factors determining the in-hospital mortality of patients with type A AAD may help guide clinical treatment and reduce the in-hospital mortality [2–4].

In the acute phase of AAD, white blood cells (WBC), C-reactive protein (CRP), fibrinogen (FIB), platelet (PLT) and D-dimer are produced or consumed in large quantities. Previous studies were mainly focused on evaluating the prognosis of type AAAD patients using an individual biomarker, which was always difficult to achieve an ideal prediction effect. At present, a new study showed that AAD is a disease of inflammation and coagulation disorders caused by a combination of multi-factors [5].

We retrospectively analyzed clinical data of 206 patients with type A AAD to investigate the relation between biomarkers related to preoperative inflammatory and coagulation and post-operative in-hospital mortality of patients with type A AAD. We found that preoperative WBC and D-dimer levels were independent risk factors for the prognosis of patients with type A AAD, and the combined analysis of FIB and PLT could be better used to predict the prognosis of the patients, which could provide reference for clinical diagnosis and treatment.

2. Method

2.1 Patients

Patients with Stanford type A AAD who had been admitted to the First Affiliated Hospital of Xi'an Jiaotong University from January 2018 to October 2020 were enrolled. These patients were diagnosed with type A AAD by the computer tomography. The primary inclusion criteria were patients with Stanford type A AAD within 2 weeks after symptom onset, aged 18 to 75 years. The exclusion criteria were as follows: 1) History of cardiogenic shock or pericardial tamponade; 2) Traumatic aortic dissection; 3) Iatrogenic aortic dissection; 4) Severe valvular diseases; 5) Congenital heart diseases; 6) Severe organ dysfunctions such as liver and kidney failure; 7) Malignant tumors; 8) Suspected subclinical myocardial involvement (e.g., history of chronic inflammation or acute infections). 231 patients were screened and 206 were enrolled in the study. Patients were divided into two groups: the death group (28 patients who died during hospitalization) and the survival group (178 patients).

This study was approved by the Research Ethics Committee of the First Affiliated Hospital of Xi'an Jiaotong University.
All data was analyzed in the blinded manner.

2.2 Surgical method

All the patients with type A AAD were treated with surgeries within 1 day after admission. After successful general anesthesia, a patient was placed in supine position, and the right axillary artery or the right axillary artery and the femoral artery were dislocated for use. The patient had a midthoracic incision, and the chest was opened layer by layer to dislocate the innominate vein and brachiocephalic vessels. The patient was treated with pericardial incision, and intubation was implanted in right axillary artery or right axillary artery and femoral artery with right atrial to establish extracorporeal circulation. Left cardiac drainage was conducted, and the ascending aorta was occluded; and ascending aortic replacement or Bentall was performed depending on the condition of the aortic valve. Nasopharyngeal and anal temperatures were reduced to 23–25°C. After occlusion of brachiocephalic vessels, the ascending aorta was opened, and cerebral perfusion of 5–10mL·Kgmin was selected. Artificial stent implantation of the descending aorta was performed, followed by classic SUN's Procedure or arch top-preserving surgery. Right heart bypass was performed with residual aortic wall and pericardium after open circulation.

2.3 Data Collection

Clinical information of the enrolled patients was obtained by consulting their medical records. Demographic data, medical history, vital signs, laboratory test and clinical results of the patients were recorded.

2.4 Endpoints

The study endpoint was defined as all-cause deaths during hospitalization.

2.5 Statistical Analysis

IBM SPSS Statistical Software 19.0 (SPSS Inc., Chicago, IL, USA) was used for statistical analysis. Continuous variables with the normal distribution were presented as mean values ± standard deviation. Non-normally distributed data were presented as median (interquartile range). Qualitative variables were expressed as frequencies or percentage. The differences between the two groups were analyzed by independent sample t-test, and chi-square test was used for the qualitative variables. The prognostic factors of patients with type A AAD were identified using univariate and multivariate logistic regression analysis. Receiver operating characteristic (ROC) curve was used to analyze the predictive value of D-dimer, FIB, PLT, WBC, neutrophil (NEU) and CRP on prognosis of the patients. $P < 0.05$ was considered to be statistically significant.

3. Results

3.1. Baseline Characteristics of the Patients

206 patients were included in this study. Each patient received a surgical operation. Clinical features of survivors and the dead patients were summarized in Table 1. In our study, the indicators including D-dimer, FIB, WBC, NEU, PLT and CRP in the death group were significantly different from those in the survival group. Furthermore, there were no significant differences regarding gender, hypertension, dyslipidemia, diabetes, smoking, alcohol abuse, hemoglobin, ALT, AST, EF, and Cr between the two groups. Additionally, no significant difference in surgical time, CPB time, cross-clamp time and HCA time was observed between the two groups.

Table 1

Baseline characteristics between the death group and the survival group.

Variable	Non-survivor (n = 28)	Survivor(n = 178)	*P* Value
Age (years)	52.3 ± 12.38	51.84 ± 10.88	0.842
Female/Male	5/23	32/146	0.770
Hypertension, n (%)	11 (39.3)	87 (48.9)	0.123
History of smoking, n (%)	10 (35.7)	65 (36.5)	0.590
Alcohol consumption, n (%)	6 (21.4)	35 (19.7)	0.671
Diabetes mellitus, n (%)	1 (3.6)	5 (2.8)	0.911
ALT (U/L)	32.31 ± 27.11	30.76 ± 20.23	0.130
AST (U/L)	39.58 ± 25.42	38.97 ± 21.35	0.074
BUN (mmol/L)	7.4 ± 2.96	6.78 ± 2.52	0.242
Cr (umol/L)	69 ~ 139	55 ~ 95	0.081
Mb (ng/mL)	67 ~ 950	45 ~ 152.25	0.071
EF (%)	51.25 ± 3.47	51.82 ± 2.75	0.788
D-Dimer (mg/L)	23.04 ± 17.69	12.66 ± 11.38	0.040
INR	1.23 ± 0.54	1.15 ± 0.21	0.444
FDP (mg/L)	69.08 ± 26.74	42.84 ± 26.59	0.069
FIB (g/L)	2.12 ± 1.51	2.93 ± 1.72	0.041
Cys-C (mg/L)	1.14 ± 0.66	0.91 ± 0.35	0.085
Hb (g/L)	130.12 ± 30.24	133.91 ± 14.65	0.508
White blood cell ($\times 10^9$/L)	13.96 ± 4.76	11.53 ± 4.32	0.046
Neutrophil ($\times 10^9$/L)	11.93 ± 3.70	9.98 ± 4.17	0.024
Platelet ($\times 10^9$/L)	136.25 ± 64.31	174.31 ± 61.75	0.003
CRP (mg/L)	34.64 ± 23.96	29.18 ± 24.31	0.041
Data are mean ± SD, or median (interquartile range), n (%).			
ALT, alamine aminotransferase; AST, aspartate transaminase; BUN, blood urea nitrogen; Cr, creatinine; Mb, myoglobin; EF, ejection fraction; INR, international normalized ratio; FDP, fibrinogen degradation product; FIB, fibrinogen; Hb, hemoglobin; Cys-C, Cystatin C; CRP, C reactive protein.			

3.2 ROC Analysis

The predictive value of D-dimer, FIB, PLT, CRP, WBC and NEU for in-hospital deaths was evaluated by using the receiver operating characteristic (ROC) method, as shown in Table 2. The area under the ROC curve (AUC) of D-Dimer was 0.647 [95% (0.573, 0.716)], and the sensitivity and specificity was 63.0% and 65.2%, respectively (P = 0.0128). The AUC of FIB was 0.634 [95% (0.562, 0.705)], and the sensitivity and specificity was 59.3% and 69.6%, respectively (P = 0.0221).The AUC of PLT was 0.684 [95% (0.611, 0.750)], and the sensitivity and specificity was 48.2% and 84.2%, respectively (P = 0.0028).The AUC of CRP was 0.542 [95% (0.468, 0.616)], and the sensitivity and specificity was 70.4% and 41.1%, respectively (P = 0.4560).The AUC of WBC was 0.641[95% (0.567, 0.710)], and the sensitivity and specificity was 55.6% and 72.8%, respectively (P = 0.0109). The AUC of NEU was 0.653 [95% (0.580, 0.721)], and the sensitivity and specificity was 55.6% and 75.3%, respectively (P = 0.0128).

Table 2

Diagnostic value of D-dimer, FIB, PLT, CRP, WBC and NEU for in-hospital mortality.

Variable	AUC	Cut-off value	SE	95% CI	Sensitivity	Specificity	P value
D-dimer	0.647	> 10.3	0.0590	0.573–0.716	0.630	0.652	0.0128
FIB	0.636	≤ 2.12	0.0593	0.562–0.705	0.593	0.696	0.0221
PLT	0.684	≤ 122	0.0615	0.611–0.750	0.482	0.842	0.0028
CRP	0.542	> 11	0.0569	0.468–0.616	0.704	0.411	0.4560
WBC	0.641	> 13.17	0.0554	0.567–0.710	0.556	0.728	0.0109
NEU	0.653	> 11.94	0.0532	0.580–0.721	0.556	0.753	0.0041
FIB, fibrinogen; PLT: platelet; CRP, C reactive protein; WBC, white blood cells; NEU, neutrophil.							

It can be seen from the ROC analysis results that PLT had the highest specificity, while CRP was provided with the highest sensitivity. Therefore, as shown in Table 3, Fig. 1 and Fig. 2, using ROC method, we further evaluated the predictive value for in-hospital deaths by combining PLT or CRP with other indicators in pairs, respectively. As can be seen from the results in Table 4, FIB + PLT combination is provided with the highest predictive value for in-hospital deaths due to its greatest value of AUC among all the kinds of these combinations. The AUC of FIB + PLT combination was 0.722 [95% (0.651, 0.785)], and the sensitivity and specificity was 59.26% and 80.38%, respectively (P = 0.0001). The AUC of D-dimer + CRP combination was 0.686 [95% (0.614, 0.752)], and the sensitivity and specificity was 51.85% and 78.48%, respectively (P = 0.0008). The AUC of D-dimer + PLT combination was 0.656 [95% (0.582, 0.724)], and the sensitivity and specificity was 62.82% and 65.82%, respectively (P = 0.0086).

Table 3
Diagnostic value of combination of the single index (D-dimer, FIB, WBC, NEU) and PLT or CRP for in-hospital mortality.

Variable	AUC	SE	95% CI	Sensitivity	Specificity	*P* value
D-dimer + PLT	0.656	0.0592	0.582 to 0.724	62.96	65.82	0.0086
FIB + PLT	0.722	0.0565	0.651 to 0.785	59.26	80.38	0.0001
WBC + PLT	0.584	0.0612	0.509 to 0.656	85.19	32.91	0.1716
NEU + PLT	0.571	0.0612	0.496 to 0.644	85.19	32.28	0.2449
D-dimer + CRP	0.686	0.0556	0.614 to 0.752	51.85	78.48	0.0008
FIB + CRP	0.613	0.0625	0.539 to 0.684	55.56	74.68	0.0699
PLT + CRP	0.680	0.0622	0.608 to 0.747	51.85	80.38	0.0037
WBC + CRP	0.631	0.0533	0.557 to 0.701	96.30	32.28	0.0139
NEU + CRP	0.647	0.0517	0.573 to 0.715	59.26	70.89	0.0046

PLT, platelet; FIB, fibrinogen; WBC, white blood cells; NEU, neutrophil; CRP, C reactive protein.

Table 4
Predictors of in-hospital mortality in patients with type A AAD by logistic regression.

Variable	Univariable			Multivariable		
	OR	95% CI	*P* value	OR	95% CI	*P* value
Age	1.106	0.752–1.625	0.610			
Sex	0.895	0.315–2.546	0.835			
BMI	1.180	0.95–1.466	0.135			
WBC	1.742	1.095–2.772	0.019	1.645	1.017–2.659	0.042
Hb	1.013	0.601–1.708	0.961			
CRP	1.069	0.837–1.364	0.593			
D-dimer	1.530	1.123–2.085	0.007	1.471	1.075–2.014	0.016
FIB	0.615	0.388–0.975	0.039			
PLT	0.988	0.978–0.997	0.013			
NEU	1.108	1.011–1.213	0.028			

BMI, body mass index; WBC, white blood cells; Hb, hemoglobin; CRP, C reactive protein; FIB, fibrinogen; PLT, platelet; NEU, neutrophil.

As shown in Fig. 3, Patients were divided into two groups according to the optimal critical value of D-dimer (> 10.3 g/L). Of the 132 patients in the low D-dimer group, 10 (7.6%) died during hospitalization. In contrast, of the 74 patients in the high D-dimer group, 18 (24.3%) died during the hospital stays. Chi-square test showed a significant difference in mortality between the two groups ($P = 0.002$). For FIB, patients were divided into two groups according to the optimal critical value (< 2.12 g/L). Of the 64 patients in the low FIB group, 16 (25.0%) died during hospitalization. In contrast, of 142 patients in the high FIB group, only 12 (8.5%) died during the hospital stays. Chi-square test showed a significant difference in mortality between the two groups ($P = 0.002$). For PLT, patients were divided into two groups according to the optimal cut-off value (< 122×10^9/L). Of the 38 patients in the low PLT group, 13 (34.2%) died during hospitalization. In contrast, of the 168 patients in the high PLT group, only 15 (8.9%) died during the hospital stays. Chi-square test showed a significant difference in mortality between the two groups ($P = 0.001$). For WBC, patients were divided into two groups according to the optimal critical value (> 13.17×10^9/L). Of the 148 patients in the low WBC group, 13 (8.8%) died during the hospital stays. In contrast, of the 58 patients in the high WBC group, 15 (25.9%) died during hospitalization. Chi-square test showed a significant difference in mortality between the two groups ($P = 0.002$). For NEU, patients were divided into two groups according to the optimal critical value (> 11.94×10^9/L). Of the 152 patients in the low NEU group, 13 (8.6%) died during the hospital stays. In contrast, of the 54 patients in the high NEU

group, 15 (27.8%) died during hospitalization. Chi-square test showed a significant difference in mortality between the two groups ($P = 0.001$).

3.3 Logistic Regression Methods

Logistic regression methods were further used to analyze the independent risk factors for postoperative in-hospital deaths of patients with type A AAD. Univariate Logistic regression analysis showed that the P values of D-dimer, FIB, PLT, WBC and NEU were all less than 0.1, which may be risk factors for postoperative in-hospital deaths of patients with type A AAD (Table 4). In order to exclude the influence of possible confounding factors, multivariate logistic stepwise regression analysis was conducted to identify the independent risk factors. It was found that WBC and D-dimmer were independent risk factors for postoperative in-hospital deaths of patients with type A AAD.

Discussion

Type A AAD is one of the common diseases in the emergency treatment for cardiovascular surgery. Early identification of risk factors for deaths of patients with type A AAD may reduce the death risk of the patients. At present, several risk factors including older age, cardiac tamponade, hypotension, myocardial ischemia, acute renal failure, limb ischemia, neurological deficits pre-operation, and mesenteric ischemia have been recognized as independent predictors of in-hospital death [1], but these risk factors still cannot meet the needs of clinical practice. We found that WBC and D-dimer on admission were closely associated with increased risks of in-hospital deaths. Both preoperative D-dimer and WBC were independent risk factors for in-hospital deaths of patients with type A AAD after correction of possible confounding factors.

D-dimer is a specific end-product belonging to the degradation fragment of cross-linked fibrin. When the coagulation system is activated, there is a large number of thrombus formed in the body, and thus leading to hyperactivity of the fibrinolytic system and concurrent increase of D-dimer. Previous studies have confirmed that D-dimer levels may be useful in risk stratifying patients with suspected aortic dissection to rule out aortic dissection if used within the first 24 hours after symptom onset[6–8]. After injury of the aortic wall occurred in AAD, a large number of tissue factors are released into the blood system, which activates the coagulation system, and then the fibrinolytic system becomes hyperreactive, resulting in the elevation of D-dimer. In recent years, many studies have also confirmed that D-dimer is associated with poor clinical prognosis of patients with dissection. Ohlmann *et al*. observed that D-dimer levels were correlated with the number of segment of dissected aorta and tended to be higher in AAD patients of DeBakey I type than those in both DeBakey II and DeBakey III types [9]. The mechanism by which elevated D-dimer may be used to predict the risk of deaths of the patients with AAD is unclear. However, there is evidence that in patients with acute aortic syndrome, D-dimer levels are significantly higher in patients with AAD than in patients with intramural hematoma, and the D-dimer of patients with type-A dissection was significantly higher than that of patients with type-B dissection [10]. These evidences above all suggest that D-dimer may be related to the size of thrombosis and the contact area

between thrombosis and blood of the patients with AAD. Elevated D-dimer may indicate larger dissection tear area and thrombosis of the patients. Moreover, the elevated D-dimer may be the result of systemic inflammatory reaction, which often interacts with coagulation function. Inflammation may activate coagulation function, and activate coagulation function in turn may also modulate the inflammatory activity.

A large number of studies have shown that inflammation is closely related to the occurrence and development of aortic dissection. And inflammation reaction plays an important role in the pathological formation of aortic dissection. C reactive protein (CRP) is increased in acute aortic dissection (AAD) and correlates with markers of ischemia [11, 12]. Likewise, white cell blood count (WBC) is higher in AAD compared to aortic aneurysms and normal subjects and is associated with higher mortality [13, 14]. Inflammation may destroy the medial layer of the aortic wall, eventually leading to dilation, dissection, or rupture of the aortic wall, since inflammation is an important pathogenetic mechanism of AAD, and white blood cells are the most sensitive and direct inflammatory indicators. After the acute onset of AAD, mechanical injury in the lesion of aorta wall can stimulate the expression of neutrophil chemotactic factor and granulocyte colony stimulating factor, thus promoting neutrophils migration, and accumulation of a large number of neutrophils in the dissection vessel wall. The release of inflammatory factors enhanced the inflammatory reaction of the adventitia following the onset of AAD, leading to further dissection of AD and rupture of the dissection [15, 16]. The higher the WBC count on admission was, the more increased mortality risk would be, which was consistent with the conclusion of this study. In-hospital infection is also an independent risk factor for increased in-hospital deaths of patients with AD. Infection of patients during the hospital stays will aggravate the original AD condition and cause systemic metabolic disorder. Severe infection may even lead to septic shock, respiratory failure, heart failure or multiple organ failure, which dramatically increases the risk of deaths.

Our study indicated that FIB + PLT combination is provided with the highest predictive value for in-hospital deaths in patients with type A AAD. The reason why the combination of FIB + PLT has the highest predictive value for deaths of patients during hospitalization may be that the intimal tearing of aortic dissection gives rise to the exposure of tissues under endothelial cells, and thus leading to the release of tissue factors and initiation of blood coagulation cascade, which lead to tissue factor release and initiates coagulation cascade. This process consumes large amounts of fibrinogen and results in decreased level of the later in bloodstream. So lower fibrinogen level would indicate greater extent injury of aorta and poorer prognosis [17]. Large amounts of FIB and PLT are consumed during this process, resulting in FIB and PLT levels decreased in the blood in the later phase. Therefore, the lower FIB and PLT levels are, the more severe the aortic wall injury and the worse the prognosis will be. Considering that FIB and PLT levels have powerful predictive value for prognosis of the patients with type-A AAD, we recommend that FIB and PLT levels be detected immediately after admission of patients with AAD. Large prospective cohort studies should be conducted to investigate the role of FIB and PLT in guiding emergency treatment of type-A AAD.

Limitations

There are some limitations in the current study. First, blood pressure and heart rate at onset of AAD have been confirmed as factors that may affect the prognosis of patients with AAD, but they were not included in our data analysis. The reason is that most patients with AAD who were taken to our hospital for surgical treatment were transported by ambulance after receiving symptomatic treatment of sedation, analgesia, hypotension and heart rate control in local hospitals. On admission, the blood pressure and heart rate of these patients were all controlled within the normal range. Many patients' family members accidentally lost the original medical record data from the local hospital, resulting in the difficulty in collecting heart rate and blood pressure data during the onset of disease. Therefore, we decided to exclude the analysis of blood pressure and heart rate at the time of onset from this study. Second, we evaluated only the relation between blood biomarkers and in-hospital mortality, but the long-term effects of blood biomarkers on patients are still unknown. Third, since the enrolled patients were of Chinese Han ancestry, the results of this study need to be confirmed with other ethnic backgrounds. Finally, the sample size of our study is relatively small. And it is a retrospective rather than a prospective study, so large-scale, multi-center clinical studies are required to further confirm the application value of plasma biomarkers including D-dimer, FIB, PLT, CRP, WBC, and NEU in aortic dissection.

Conclusions

Both preoperative D-dimer and WBC in patients with type A AAD may be used as independent risk factors for the prognosis of such patients. Combined use of FIB and PLT may improve the accuracy and accessibility of clinical prognostic assessment. Biochemical examination technology has the advantages such as being quick, simple, non-invasive, and cheap. Therefore, it has a great prospect of clinical application to determine whether the biochemical monitoring of peripheral blood markers in aortic dissection has the value of early diagnosis and prognosis judgment, so as to better guide the future clinical research and treatment strategy.

Abbreviations

AAD
acute aortic dissection; CRP:C-reactive protein; FIB:fibrinogen; NEU:neutrophil; PLT:platelet; ROC:Receiver operating characteristic; WBC:white blood cells.

Declarations

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Authors’ contributions

ML, LX and SCX designed the experiment strategy and wrote the manuscript. YJZ, JJH, CD, XLZ, MML and YG assessed patients and collect data. HFS, YY, HCW, YXL and XW conducted statistical analyses and interpreted the data. The authors read and approved the final manuscript.

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Availability of data and materials

All data generated or analyzed during this study are included in this published article.

Ethics approval and consent to participate

In this retrospective analysis, no patient confidentiality was involved; therefore, no ethics approval was needed. The authors have no ethical conflicts to disclose. The authors declare that there is no conflict of interest regarding the publication of this paper. All patients included in this study were admitted as emergency cases. No consent was obtained.

Consent for publication

Our study does not contain any individual person’s data in any form. All authors signed a consent form for publication in case of acceptance.

Competing interests

We do not have any competing interests with respect to the study or any part of it.

Author details

[1] Department of Cardiovascular Surgery, The First Affiliated Hospital of Medical College of Xi'an Jiaotong University, Xi'an, Shaanxi 710061, China. [2] Third Department of Medical Oncology, Shaanxi Provincial Cancer Hospital Affiliated to Medical College of Xi’an Jiaotong University, Xi'an, 710061, China. [3] Department of Clinical Laboratory, The Second Affiliated Hospital of Xi'an Jiaotong University, Xi'an, Shaanxi 710004, China.

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Figures

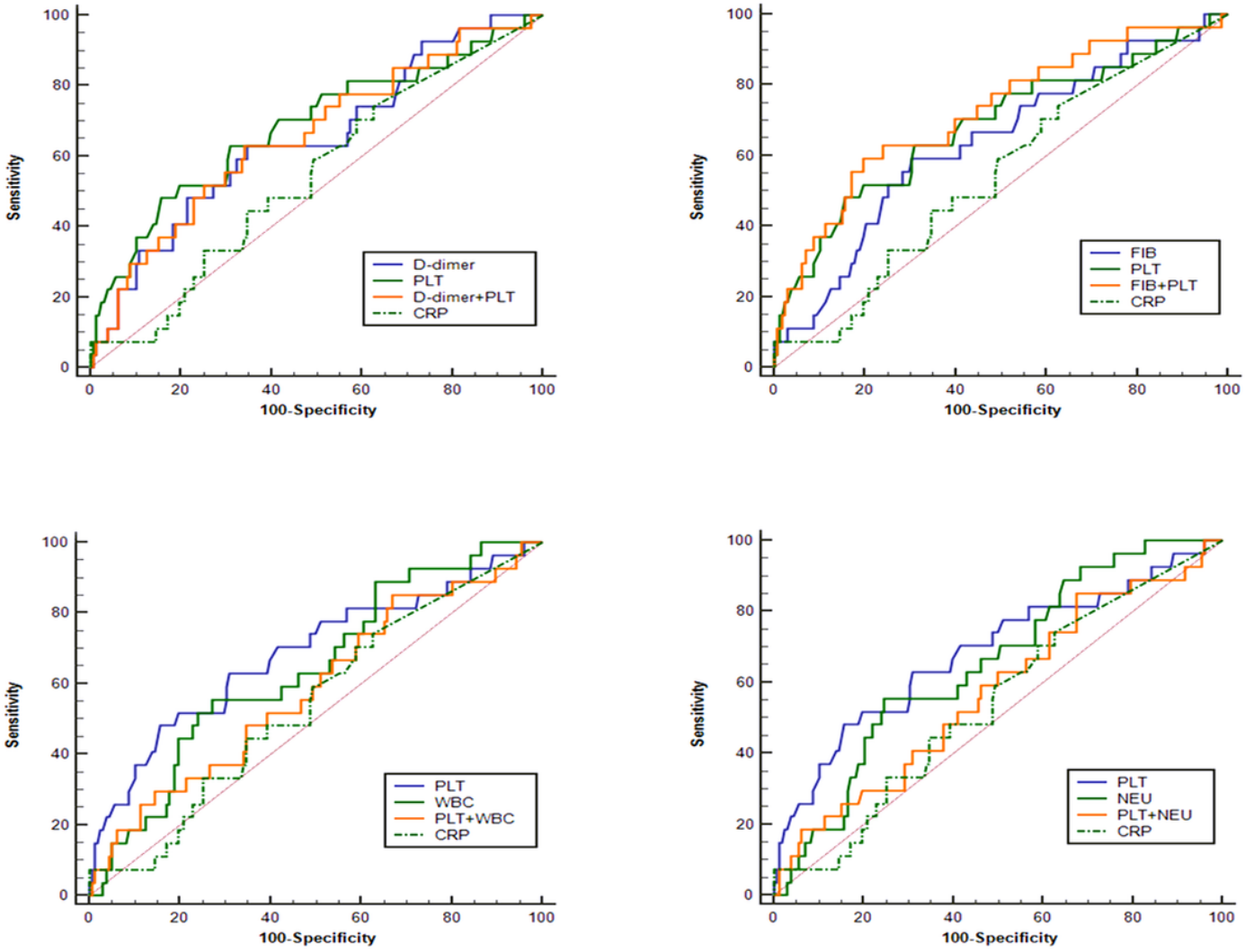

Figure 1

ROC curve of a single index of D-dimer, FIB, PLT, CRP, WBC, or NEU, and the combination of the single index and PLT for predicting in-hospital deaths in patients with type A AAD. ROC, receiver operating characteristic; FIB, fibrinogen; PLT, platelet; CRP, C-reactive protein; WBC, white blood cells; NEU, neutrophil; AAD, acute aortic dissection.

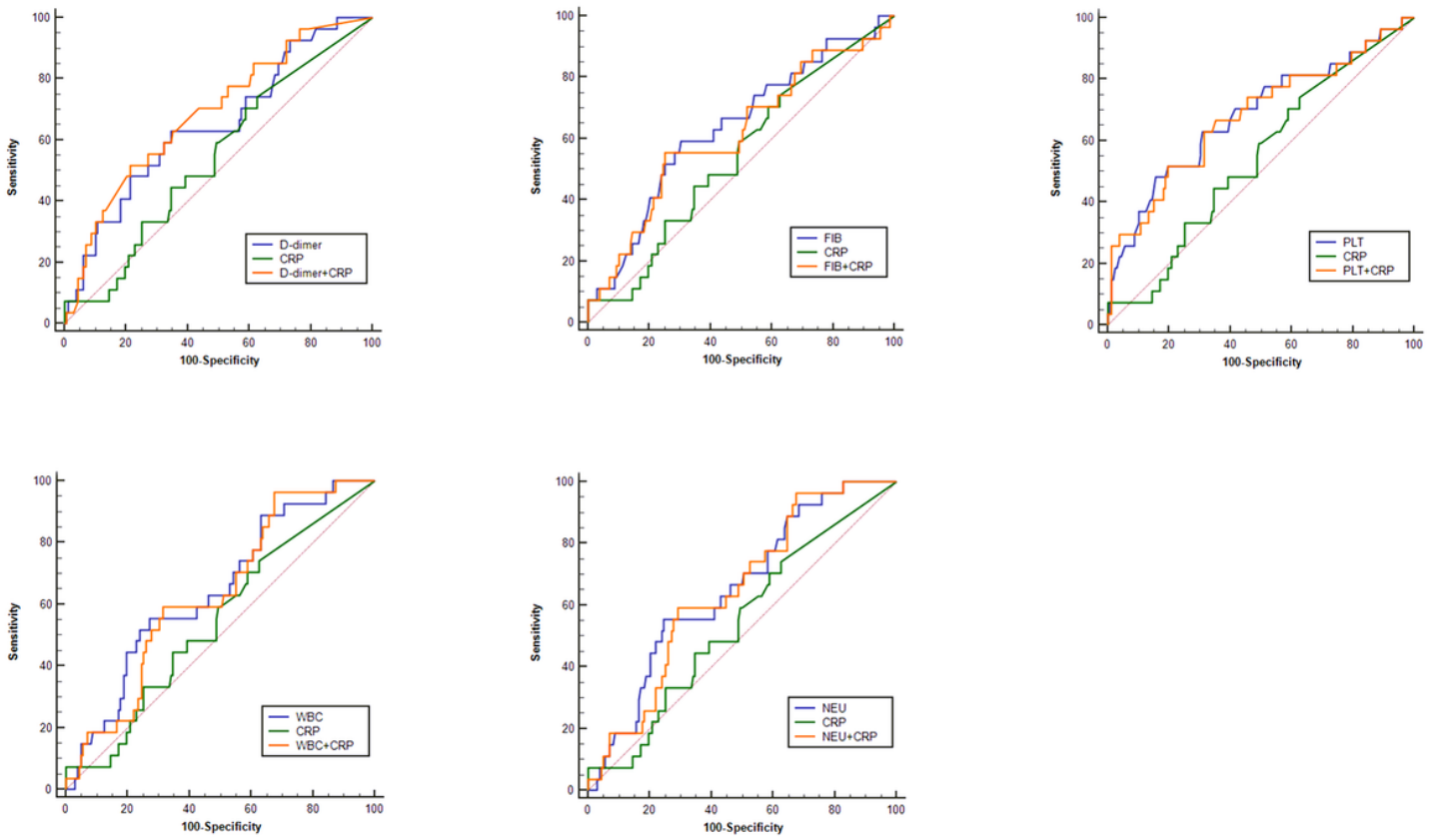

Figure 2

ROC curve of a single index of D-dimer, FIB, PLT, CRP, WBC, or NEU, and the combination of the single index and CRP for predicting in-hospital deaths in patients with type A AAD. ROC, receiver operating characteristic; FIB, fibrinogen; PLT, platelet; CRP, C-reactive protein; WBC, white blood cells; NEU, neutrophil; AAD, acute aortic dissection.

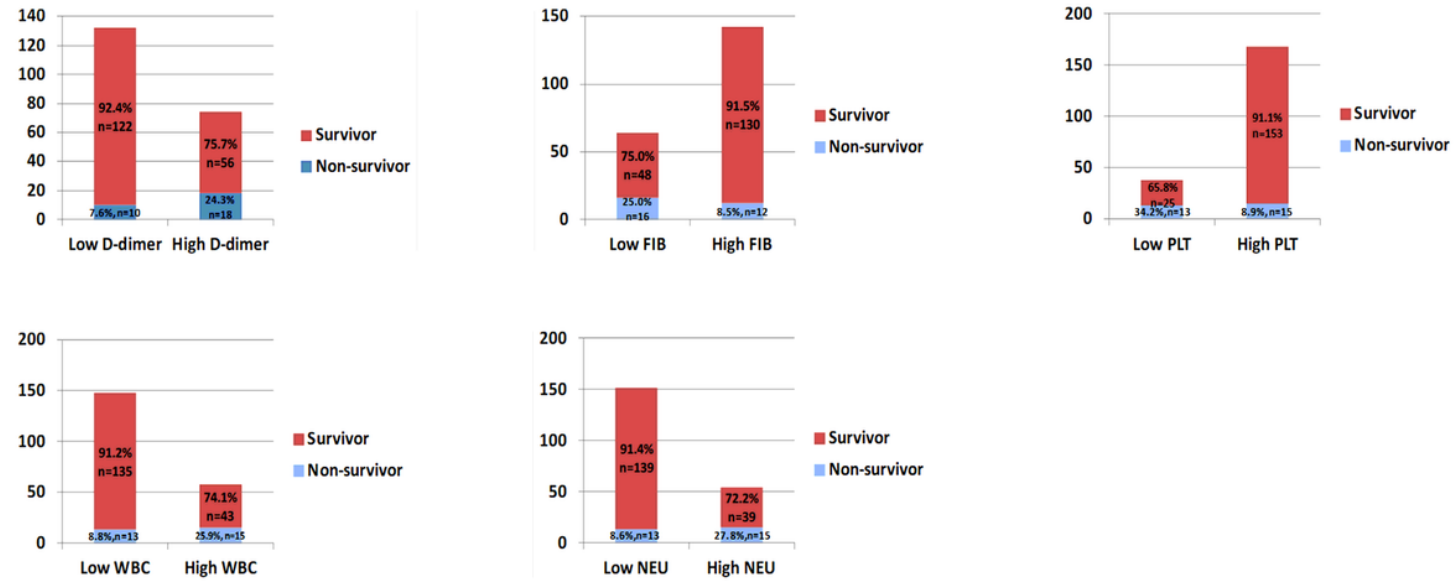

Figure 3

Distribution of the in-hospital mortality rate in patients with type A AAD according to categories of the indices including D-dimer, FIB, PLT, WBC and NEU. FIB, fibrinogen; PLT, platelet; WBC, white blood cells; NEU, neutrophil; AAD, acute aortic dissection.