Value of PLR and Uric Acid in Early Diagnosis of Spinal Tuberculosis and Brucella Spondylitis: A Postprint Study

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Abstract

Background The elderly population accounts for a relatively high proportion of spinal tuberculosis (STB) and brucellar spondylitis (BS) cases, with comorbidities and economic difficulties being relatively common. Traditional methods are difficult for early differentiation between the two diseases, while clinically common serological tests and polymerase chain reaction (PCR) are costly, and bacterial culture is time-consuming. Therefore, under the influence of comorbidities, exploring new laboratory diagnostic indicators holds important clinical significance. Objective To investigate the clinical application value of common laboratory indicators in the differential diagnosis of STB and BS. Methods A total of 232 patients with STB and BS who visited the Second Affiliated Hospital of Inner Mongolia Medical University from January 1, 2015, to October 31, 2024, were collected as study subjects. They were divided into an STB group (132 cases) and a BS group (100 cases) based on disease type, and into a group without comorbidities (132 cases) and a group with comorbidities (100 cases). Laboratory indicators including routine blood tests, blood biochemical tests, and inflammatory markers were collected, and inter-group differences in each indicator were compared. Multivariate Logistic regression analysis was used to screen for significant predictive indicators and construct a prediction model, and the Hosmer-Lemeshow test was used to evaluate model fit. Receiver operating characteristic (ROC) curves were used to assess the sensitivity and specificity of the model, the area under the ROC curve (AUC) was used to evaluate its predictive value, and Delong' s test was used to compare differences in AUC values among indicators. Results Multivariate Logistic regression analysis showed that platelet-to-lymphocyte ratio (PLR) and uric acid (UA) were independent influencing factors for differentiating STB from BS (PLR: OR=0.993, 95%CI=0.990~0.997; UA: OR=0.994, 95%CI=0.991~0.9971). Hosmer-Lemeshow test results indicated good model fit (2=3.721, P=0.881). The AUC value of PLR for diagnosing STB and BS was 0.649, with an optimal

cutoff value of 0.449, sensitivity of 59.1%, and specificity of 64%. The AUC value of UA for diagnosing STB and BS was 0.669, with an optimal cutoff value of 0.339, sensitivity of 34.8%, and specificity of 93%. The AUC value of PLR+UA combined for diagnosing STB and BS was 0.721, with an optimal cutoff value of 0.489, sensitivity of 69.7%, and specificity of 66%. Delong' s test results showed that the AUC value of PLR+UA combined diagnosis was higher than that of PLR alone, with a statistically significant difference (Z=2.167, P=0.030). Compared with UA alone, the difference was not statistically significant (Z=1.884, P=0.065). Conclusion PLR and UA can serve as reference indicators for early differentiation between STB and BS and hold important clinical application value.

Full Text

The Value of Platelet-to-Lymphocyte Ratio and Uric Acid in Early Differentiation of Spinal Tuberculosis from Brucellar Spondylitis

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Abstract

Background: The elderly population constitutes a relatively high proportion of patients with spinal tuberculosis (STB) and brucellar spondylitis (BS), particularly among those with underlying diseases and economic hardships. Traditional methods are inadequate for early differentiation between these two diseases, while serological tests and polymerase chain reaction (PCR) are costly, and bacterial culture is time-consuming. Therefore, exploring new laboratory diagnostic indicators under the influence of underlying diseases holds important clinical significance.

Objective: To investigate the clinical value of common laboratory indicators in distinguishing STB from BS.

Methods: A total of 232 patients diagnosed with STB and BS at the Second Affiliated Hospital of Inner Mongolia Medical University from January 1, 2015, to October 31, 2024, were included in this study. Patients were divided into an STB group (132 cases) and a BS group (100 cases) based on disease type, and further categorized into groups without underlying diseases (132 cases) and with underlying diseases (100 cases). Laboratory indicators including complete blood

count, blood biochemical parameters, and inflammatory markers were collected and compared between groups. Multivariate logistic regression analysis was conducted to identify clinically meaningful predictive markers and construct a predictive model. Model fit was evaluated via the Hosmer-Lemeshow goodness-of-fit test. The sensitivity and specificity of the model were assessed using receiver operating characteristic (ROC) curve analysis, with the area under the curve (AUC) used to evaluate predictive value. Differences in AUC values among various indicators were compared using the DeLong test.

Results: Multivariate logistic regression analysis revealed that platelet-to-lymphocyte ratio (PLR) (OR=0.993, 95%CI=0.990-0.997) and uric acid (UA) (OR=0.994, 95%CI=0.991-0.997) were independent influencing factors for distinguishing STB from BS. The Hosmer-Lemeshow test indicated good model fit (^{2}=3.721, P=0.881). The AUC for PLR in diagnosing STB and BS was 0.649, with an optimal cutoff value of 0.449, sensitivity of 59.1%, and specificity of 64%. The AUC for UA was 0.669, with an optimal cutoff value of 0.339, sensitivity of 34.8%, and specificity of 93%. The AUC for combined PLR+UA diagnosis was 0.721, with an optimal cutoff value of 0.489, sensitivity of 69.7%, and specificity of 66%. DeLong test results showed that the AUC for combined PLR+UA diagnosis was significantly higher than that of PLR alone (Z=2.167, P=0.030), while the difference compared to UA alone was not statistically significant (Z=1.884, P=0.065).

Conclusion: PLR and UA can serve as valuable reference indicators for early differentiation between STB and BS and hold important clinical application value.

Keywords: Spinal tuberculosis; Brucellar spondylitis; Elderly population; Platelet/lymphocyte ratio; Uric acid

Introduction

Spinal tuberculosis (STB) and brucellar spondylitis (BS) are both common spinal infectious diseases in orthopedics. Although their etiologies differ, their clinical manifestations are similar, making early differentiation difficult. STB predominantly affects middle-aged and elderly populations, who often have compromised immune function and multiple chronic underlying diseases, leading to atypical clinical presentations. During the initial infection stage, accurate distinction between STB and BS is challenging before obtaining results from etiological, serological, and imaging examinations, and misdiagnosis may occur even when combined with imaging findings. Bacterial culture requires a long cycle, while serological testing and polymerase chain reaction (PCR) are costly, often causing elderly patients to delay or forgo testing due to economic constraints. In clinical practice, routine examinations such as complete blood count and blood biochemistry have become fundamental assessment tools for patients upon initial admission because of their low cost, wide availability, and short turnaround

time. Previous studies have shown that laboratory test results in STB and BS patients exhibit specific abnormal patterns. Domestic research has found that STB patients have higher monocyte-to-lymphocyte ratios but lower lymphocyte counts, alanine aminotransferase, aspartate aminotransferase, and alkaline phosphatase compared to BS patients. However, these studies did not deeply analyze patient populations with underlying diseases. Given that both diseases predominantly affect elderly patients and that elderly patients with underlying diseases are common in clinical practice, it is necessary to compare laboratory indicators between STB and BS patients while considering underlying diseases to obtain more representative predictive indicators. This study retrospectively analyzed clinical data from STB and BS patients to explore the value of laboratory examination indicators in early differentiation between the two diseases, aiming to provide a reference basis for clinical diagnosis and treatment.

Methods

1.1 Study Population A total of 232 patients hospitalized at the Second Affiliated Hospital of Inner Mongolia Medical University between January 1, 2015, and October 31, 2024, with discharge diagnoses of STB or BS were included in this study. The study was approved by the Ethics Committee of the Second Affiliated Hospital of Inner Mongolia Medical University (approval number: EFY20240057), and all patients provided informed consent.

1.2 Diagnostic Criteria **(1) STB** was diagnosed according to the "Chinese Guidelines for Surgical Treatment of Spinal Tuberculosis (2022 Edition)": History and exposure: tuberculosis history or contact with tuberculosis patients. Clinical manifestations: low-grade fever, night sweats, loss of appetite, weight loss, fatigue, and other tuberculosis toxic symptoms, possibly accompanied by chronic illness appearance, angular kyphosis deformity, tenderness, percussion pain, and limited mobility. Laboratory tests: abnormal C-reactive protein (CRP) (0-8 mg/L) and erythrocyte sedimentation rate (ESR) (0-15 mm/h for males, 0-20 mm/h for females). Etiological examination: positive *Mycobacterium tuberculosis* culture. Imaging examinations: X-ray may show abnormal intervertebral spaces such as narrowing or disappearance; CT may reveal sequestra and calcification; MRI can display early lesions, dural sac and spinal cord involvement, and abscess formation.

(2) BS was diagnosed according to the "Brucellosis Diagnosis and Treatment Plan (2023 Edition)"and "Expert Consensus on Diagnosis and Treatment of Brucellar Spondylitis": History and exposure: history of brucellosis or contact with livestock (contact with cattle and sheep, consumption of unpasteurized animal products, or residence in brucellosis-endemic areas). Clinical manifestations: fever (undulant fever), fatigue, excessive sweating, muscle and joint pain, possibly accompanied by hepatosplenomegaly and lymphadenopathy. Laboratory tests: abnormal CRP and ESR. Etiological examination: positive *Brucella*

culture. Serological tests: positive rose bengal plate test (RBPT), serum agglutination test (SAT), or enzyme-linked immunosorbent assay (ELISA). Imaging examinations: X-ray may show mild marginal vertebral bone destruction; CT may display specific "lace vertebra" and "parrot beak" signs; MRI shows lesions with marked T1-weighted imaging (T1WI) hypointensity, localized bone destruction at the edges of adjacent vertebral bodies, paraspinal soft tissue congestion and edema, and abnormal signals within the paraspinal and psoas muscles.

1.3 Inclusion and Exclusion Criteria **Inclusion criteria:** (1) Met diagnostic criteria for STB or BS; (2) No restrictions on age, sex, region, or occupation; (3) Complete medical records, including admission and discharge records and relevant auxiliary examination results.

Exclusion criteria: (1) Patients with active tuberculosis in other sites or brucellosis in other locations; (2) Patients who had used anti-tuberculosis or anti-brucellosis drugs within the past year; (3) Patients with other spinal infectious diseases (such as purulent spondylitis, fungal spondylitis, syphilitic spondylitis, etc.); (4) Patients with infectious diseases or acute injury diseases in other organs or systems; (5) Patients with missing indicators or incomplete data.

1.4 Grouping Strategy Patients were grouped according to disease status: (1) Based on disease type into Group A (all STB patients) and Group B (all BS patients); (2) Based on presence of underlying diseases into Group C (all patients without underlying diseases) and Group D (all patients with one or more underlying diseases that might affect laboratory results); (3) Combined disease and underlying disease status into Group CA (STB without underlying diseases), Group CB (BS without underlying diseases), Group DA (STB with underlying diseases), and Group DB (BS with underlying diseases).

1.5 General Data Collection General data collected included patient sex, age, ethnicity, residence, smoking and alcohol consumption history, past medical history (including hypertension, coronary heart disease, diabetes, cerebral infarction, chronic obstructive pulmonary disease, etc.), and clinical manifestations (including localized spinal pain, fever, fatigue, night sweats, and weight loss).

1.6 Laboratory Indicator Collection First laboratory examination indicators were collected, including 72 items: white blood cell count, neutrophil count, eosinophil count, basophil count, lymphocyte count, monocyte count, neutrophil percentage, eosinophil percentage, basophil percentage, lymphocyte percentage, monocyte percentage, neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio (PLR), lymphocyte-to-monocyte ratio, red blood cell count, hematocrit, mean corpuscular volume, red cell distribution width-coefficient of variation, red cell distribution width-standard deviation, hemoglobin, mean

corpuscular hemoglobin, mean corpuscular hemoglobin concentration, platelet count, plateletcrit (PCT), mean platelet volume, platelet distribution width, large platelet ratio, large platelet count, prothrombin time, thrombin time, fibrinogen, activated partial thromboplastin time, serum D-dimer, international normalized ratio, prothrombin time activity, prothrombin time ratio, alanine aminotransferase, aspartate aminotransferase, aminotransferase ratio, alkaline phosphatase, γ-glutamyl transferase, total bilirubin, direct bilirubin, indirect bilirubin, total protein, albumin, globulin, albumin-to-globulin ratio, prealbumin, creatinine, urea, uric acid (UA), lactate dehydrogenase, CO_2, total cholesterol, apolipoprotein A1, apolipoprotein B, low-density lipoprotein, high-density lipoprotein, triglycerides, creatine kinase, creatine kinase isoenzyme, α-hydroxybutyrate dehydrogenase, potassium, sodium, chloride, calcium, phosphorus, magnesium, glucose, C-reactive protein, and erythrocyte sedimentation rate.

1.7 Statistical Methods Data were entered and collected using Excel software. Statistical analysis was performed using SPSS 27.0 software, and data visualization was conducted using GraphPad Prism 8.0 software. Categorical data were expressed as constituent ratios and compared between groups using the ^{2} test. Normally distributed continuous data were expressed as mean $\pm$ standard deviation ($\bar{x}\pm s$) and compared between groups using independent samples t-test when variance homogeneity was satisfied. Non-normally distributed continuous data were expressed as median (P25, P75) and compared between groups using Mann-Whitney U test. Laboratory indicators showing statistically significant differences in multiple group comparisons were included as independent variables in multivariate logistic regression analysis to construct a predictive model for differentiating STB from BS. The Hosmer-Lemeshow test was used to evaluate model fit. Receiver operating characteristic (ROC) curves were used to assess the sensitivity and specificity of predictive indicators, with area under the curve (AUC) calculated to evaluate diagnostic efficacy. Differences in AUC values were compared using the DeLong test. A P-value <0.05 was considered statistically significant.

Results

2.1 Comparison of General Data A total of 232 patients were included, comprising 143 males (61.64%) and 89 females (38.36%), with a mean age of 61.0$±12.4*years*.*Elderlypatients*($60 years) accounted for 131 cases (56.47%). Significant differences in sex distribution were observed between Groups A and B, Groups CA and CB, and Groups DA and DB (P<0.001). No statistically significant differences were found in age, ethnicity, residence, smoking history, alcohol consumption history, underlying disease history, or clinical manifestations between these paired groups (P>0.05) ~3.

2.2 Comparison of Laboratory Indicators Among Groups Group A showed significantly higher levels than Group B in white blood cell count, neutrophil count, eosinophil count, basophil count, neutrophil percentage, eosinophil percentage, basophil percentage, neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio, monocyte-to-lymphocyte ratio, red blood cell distribution width-coefficient of variation, mean corpuscular hemoglobin, platelet count, plateletcrit, aminotransferase ratio, total protein, globulin, uric acid, sodium, and erythrocyte sedimentation rate. Group A showed significantly lower levels than Group B in lymphocyte count, lymphocyte percentage, activated partial thromboplastin time, alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, total bilirubin, direct bilirubin, lactate dehydrogenase, α-hydroxybutyrate dehydrogenase, and potassium ($P<0.05$). No statistically significant differences were found in the remaining 41 indicators ($P>0.05$) (see Supplementary Table 1).

In the comparison between Groups CA and CB, Group CA showed significantly higher basophil count, basophil percentage, PLR, platelet count, plateletcrit, aminotransferase ratio, total protein, globulin, UA, and creatine kinase, while showing significantly lower large platelet ratio, alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, total bilirubin, direct bilirubin, albumin-to-globulin ratio, creatinine, urea, and lactate dehydrogenase ($P<0.05$). No statistically significant differences were observed in the remaining 52 indicators ($P>0.05$) (see Supplementary Table 2).

In the comparison between Groups DA and DB, Group DA showed significantly higher white blood cell count, neutrophil count, eosinophil count, neutrophil percentage, eosinophil percentage, neutrophil-to-lymphocyte ratio, PLR, monocyte-to-lymphocyte ratio, mean corpuscular volume, mean corpuscular hemoglobin, plateletcrit, large platelet count, fibrinogen, creatinine, UA, sodium, and erythrocyte sedimentation rate, while showing significantly lower lymphocyte count, lymphocyte percentage, red blood cell count, red cell distribution width-coefficient of variation, and activated partial thromboplastin time ($P<0.05$). No statistically significant differences were found in the remaining 50 indicators ($P>0.05$) .

2.3 Logistic Regression Analysis and Predictive Model Construction Univariate logistic regression analysis was performed using PLR, PCT, and UA as independent variables (all assigned as actual measured values) and disease type as the dependent variable (assigned as brucellar spondylitis=0, spinal tuberculosis=1). The results showed that PLR and UA were predictive factors for differentiating STB from BS ($P<0.05$) .

Subsequent multivariate logistic regression analysis using PLR and UA as independent variables (with the same disease type assignment) confirmed that PLR (OR=0.993, 95%CI=0.990-0.997, $P<0.001$) and UA (OR=0.994, 95%CI=0.991-0.997, $P<0.001$) were independent predictive factors for distinguishing STB from BS. The Hosmer-Lemeshow test yielded $^2=3.721$, $P=0.881$, indicating

good model fit.

2.4 ROC Curves for PLR, UA, and Combined PLR+UA in Diagnosing STB and BS ROC curves were plotted for PLR, UA, and combined PLR+UA in differentiating STB from BS. The results showed that PLR alone had an AUC of 0.649, optimal cutoff value of 0.449, sensitivity of 59.1%, and specificity of 64%. UA alone had an AUC of 0.669, optimal cutoff value of 0.339, sensitivity of 34.8%, and specificity of 93%. The combined PLR+UA approach achieved an AUC of 0.721, optimal cutoff value of 0.489, sensitivity of 69.7%, and specificity of 66% [Figure 1: see original paper]. DeLong test comparisons revealed that the AUC for combined PLR+UA was significantly higher than that of PLR alone (Z=2.167, P=0.030), while the difference compared to UA alone was not statistically significant (Z=1.884, P=0.065).

Discussion

Differentiating STB from BS represents a current hotspot in clinical research. Distinguishing these two diseases based on clinical symptoms and imaging examinations alone is difficult in the early stages, while serological testing, PCR technology, and bacterial culture have limitations related to high cost or time consumption. Therefore, there is an urgent need to explore an economical and efficient method for early differentiation to enable rapid preliminary diagnosis during the initial disease stage.

This study included 232 patients, and across different grouping strategies, the proportion of male patients with BS was consistently higher than that with STB, with statistically significant differences, consistent with findings from Li Junjie et al. and ZHU et al. As both diseases are rare, the gender distribution shows unique patterns, making it difficult to exclude the influence of sex on results. Laboratory indicator comparisons revealed that in Groups A and B, STB patients had lower alanine aminotransferase, aspartate aminotransferase, and alkaline phosphatase levels compared to BS patients, consistent with results from Li Yongai et al. and YASIN et al. STB patients also showed higher monocyte-to-lymphocyte ratios and lower lymphocyte counts compared to BS patients, aligning with findings from YANG Yi et al. However, when considering underlying diseases, Groups CA and CB showed no statistically significant differences in lymphocyte count and monocyte-to-lymphocyte ratio, suggesting that patients' underlying diseases and medications may interfere with lymphocyte and platelet levels. Similarly, Groups DA and DB showed no statistically significant differences in alanine aminotransferase, aspartate aminotransferase, and alkaline phosphatase, indicating that underlying diseases and medications may affect liver function. These results highlight the scientific necessity and importance of detailed grouping design.

This study found that PLR, PCT, and UA showed statistically significant differ-

ences between STB and BS across different grouping strategies. Univariate logistic regression analysis demonstrated that both PLR and UA were significantly associated with disease type, suggesting their potential value in differential diagnosis. Multivariate logistic regression analysis further confirmed that PLR and UA were independent predictive factors for distinguishing STB from BS, consistent with previous research on PLR' s value in inflammatory diseases. Through multiple grouping analyses, this study further verified PLR' s potential in differentiating spinal infectious diseases, providing new perspectives and evidence for diagnosing STB and BS. PLR reflects the inflammatory response, with platelets releasing pro-inflammatory factors to promote inflammatory cell recruitment and activation, while lymphocytes regulate immune responses through cytokine secretion. Differences in PLR levels are related to the degree of inflammation and immune response intensity. Lower PLR levels in BS patients may be associated with the localized and milder inflammatory response caused by *Brucella* infection. UA, as the final product of purine metabolism, is associated with inflammatory responses, oxidative stress, and apoptosis. Changes in UA levels in STB patients may be related to impaired renal excretory function, while changes in BS patients reflect persistent inflammatory responses and metabolic abnormalities.

ROC curve analysis showed that the AUC for combined PLR+UA diagnosis was higher than that of PLR alone, though the difference compared to UA alone was not statistically significant. As an inflammatory marker, PLR' s predictive efficacy may be limited by complex immune responses. Although UA is associated with inflammation in various diseases, its specificity is insufficient to accurately reflect disease status. PLR and UA contain substantially different biological information and pathophysiological mechanisms, potentially providing complementary information. Combined use of PLR and UA integrates multi-angle patient information, improving disease prediction accuracy and increasing the AUC value. UA alone demonstrated diagnostic efficacy approaching that of combined diagnosis in differentiating STB and BS, highlighting its potential in this context. This may be because UA' s high specificity (93%) masked the statistical advantage of combined diagnosis, or due to the relatively small sample size. In clinical practice, preliminary analysis based on routine blood and biochemical data can facilitate early differentiation between these two diseases, providing a reference basis for subsequent diagnosis.

This study has several limitations. First, the sample size was relatively small, and male patients were more common among BS cases, while female patients' medical records were difficult to obtain, preventing adequate exclusion of potential sex differences' influence on laboratory results and affecting the generalizability of findings to some extent. Second, this single-center study included only patients hospitalized at the Second Affiliated Hospital of Inner Mongolia Medical University, without covering cases from other regions or hospitals, limiting the representativeness and external generalizability of the results. Third, due to the extreme rarity of concurrent STB and BS cases, this study could not include patients with both diseases, limiting the applicability of the find-

ings. Future studies should include larger sample sizes with validation groups and healthy control groups to verify the value of PLR and UA as differential diagnostic indicators.

In summary, PLR and UA are independent predictive factors for differentiating STB from BS. Especially when etiological culture results are not yet available or patients cannot afford serological testing due to economic constraints, preliminary diagnosis can be made early based on laboratory data combined with clinical manifestations and imaging features to guide clinical treatment.

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Supplementary Tables

Supplementary Table 1. Comparison of Laboratory Indicators Between Groups A and B

Indicator	Group A (n=132)	Group B (n=100)	P-value
White blood cell count [M(P25,P75), $×10^{9}$/L]	6.635(5.165,8.408)	5.935(4.860,7.423)	<0.001
Neutrophil count [M(P25,P75), $×10^{9}$/L]	4.240(3.325,5.875)	3.735(2.695,4.690)	<0.001

Indicator	Group A (n=132)	Group B (n=100)	P-value
Eosinophil count [M(P25,P75), $×10^{9}$/L]	0.110(0.050,0.198)	0.050(0.020,0.128)	<0.001
Basophil count [M(P25,P75), $×10^{9}$/L]	0.030(0.020,0.040)	0.020(0.010,0.030)	<0.001
Lymphocyte count [M(P25,P75), $×10^{9}$/L]	1.490(1.055,1.938)	1.585(1.323,2.233)	<0.001
Monocyte count [M(P25,P75), $×10^{9}/L$]|0.435(0.320, 0.560)|0.425(0.303, 0.520)|0.772||$Neutrophilpercentage(x±s$,±10.700|62.137±10.211	<0.001		
0.001||$Eosinophilpercentage[M(P25, P75)$,±$s$,±9.297|28.664±$9.519			
Monocyte percentage [M(P25,P75), %]	6.500(5.300,8.250)	6.700(5.600,8.275)	0.913
Neutrophil-to-lymphocyte ratio [M(P25,P75)]	2.930(2.025,4.405)	2.150(1.593,3.088)	<0.001
Platelet-to-lymphocyte ratio [M(P25,P75)]	175.965(131.728,240.848)	138.170(99.528,191.483)	<0.001
Monocyte-to-lymphocyte ratio [M(P25,P75)]	0.296(0.216,0.427)	0.253(0.180,0.324)	0.003
Red blood cell count [M(P25,P75), $×10^{12}$/L]	4.350(3.858,4.680)	4.365(4.025,4.828)	0.772
Hematocrit [M(P25,P75), %]	39.650(35.525,42.775)	38.550(36.525,42.725)	0.913

Indicator	Group A (n=132)	Group B (n=100)	P-value
Mean corpuscular volume [M(P25,P75), fl]	90.600(87.425,95.150)	89.150(86.350,91.500)	0.003
Red cell distribution width-CV [M(P25,P75), %]	13.800(13.000,14.800)	14.100(13.300,15.100)	0.003
Red cell distribution width-SD [M(P25,P75), fl]	44.300(42.000,48.625)	45.000(42.900,47.875)	0.913
Hemoglobin [M(P25,P75), g/L]	132.000(116.000,141.000)	129.500(119.250,141.750)	0.772
Mean corpuscular hemoglobin [M(P25,P75), pg]	30.050(28.900,31.575)	29.600(28.425,30.675)	0.003
Mean corpuscular hemoglobin concentration [M(P25,P75), g/L]	331.000(325.000,337.000)	331.500(328.250,337.000)	0.913
Platelet count [M(P25,P75), $×10^{9}$/L]	258.000(200.250,317.250)	226.500(188.250,268.750)	<0.001
Plateletcrit [M(P25,P75), %]	0.230(0.180,0.280)	0.200(0.170,0.240)	<0.001
Mean platelet volume [M(P25,P75), fl]	8.800(8.100,9.700)	8.900(8.300,9.900)	0.913
Platelet distribution width [M(P25,P75), %]	15.900(15.600,16.100)	15.850(15.600,16.100)	0.913

Indicator	Group A (n=132)	Group B (n=100)	P-value
Large platelet ratio [M(P25,P75), %]	18.050(13.800,24.200)	19.050(15.075,25.850)	0.003
Large platelet count [M(P25,P75), $×10^{9}$/L]	47.000(34.250,62.750)	41.500(31.250,58.000)	0.003
Prothrombin time [M(P25,P75), s]	11.350(10.800,12.100)	11.200(10.700,11.900)	0.003
Thrombin time [M(P25,P75), s]	13.500(12.800,14.375)	13.600(12.800,14.200)	0.913
Fibrinogen [M(P25,P75), g/L]	4.230(3.603,4.630)	3.960(3.453,4.465)	0.003
Activated partial thromboplastin time [M(P25,P75), s]	31.000(28.525,33.300)	31.700(29.500,34.575)	<0.001
D-dimer [M(P25,P75), g/L]	333.500(168.500,655.750)	325.500(206.750,547.250)	0.913
International normalized ratio [M(P25,P75)]	1.030(0.970,1.090)	1.000(0.960,1.070)	0.003
Prothrombin time activity [M(P25,P75), %]	98.000(87.000,106.000)	100.000(91.250,106.000)	0.003
Prothrombin time ratio [M(P25,P75)]	1.030(0.963,1.090)	1.000(0.960,1.070)	0.003
Alanine aminotransferase [M(P25,P75), U/L]	17.750(11.450,29.250)	27.700(15.725,54.750)	<0.001

Indicator	Group A (n=132)	Group B (n=100)	P-value
Aspartate aminotransferase [M(P25,P75), U/L]	18.950(15.125,23.775)	23.100(17.450,36.325)	<0.001
Aminotransferase ratio [M(P25,P75)]	1.040(0.750,1.490)	0.910(0.720,1.188)	<0.001
Alkaline phosphatase [M(P25,P75), U/L]	104.350(81.950,126.175)	115.100(93.900,149.125)	<0.001
γ-glutamyl transferase [M(P25,P75), U/L]	52.500(31.250,102.000)	58.500(32.500,99.000)	0.003
Total bilirubin [M(P25,P75), mol/L]	8.070(6.425,10.365)	9.335(7.563,12.150)	<0.001
Direct bilirubin [M(P25,P75), mol/L]	3.390(2.660,4.768)	4.055(3.088,5.623)	<0.001
Indirect bilirubin [M(P25,P75), mol/L]	4.535(3.795,6.113)	5.005(3.850,6.840)	0.003
Total protein [M(P25,P75), g/L]	68.500(64.250,73.450)	66.000(61.450,70.100)	<0.001
Albumin ($\bar{x}\pm s$, g/L)	38.179$±4.369\|37.736±4.2[illegible]$	[illegible]	[illegible]
Total cholesterol [M(P25,P75), mmol/L]	4.190(3.685,5.138)	4.300(3.613,5.165)	0.913

Indicator	Group A (n=132)	Group B (n=100)	P-value
Apolipoprotein A1 [M(P25,P75), g/L]	1.200(1.043,1.345)	1.215(0.995,1.435)	0.913
Apolipoprotein B [M(P25,P75), g/L]	0.935(0.793,1.108)	0.930(0.760,1.100)	0.913
Low-density lipoprotein [M(P25,P75), mmol/L]	2.770(2.393,3.510)	2.795(2.313,3.423)	0.913
High-density lipoprotein [M(P25,P75), mmol/L]	1.165(0.903,1.310)	1.165(0.903,1.350)	0.913
Triglycerides [M(P25,P75), mmol/L]	1.270(0.913,1.598)	1.370(0.925,1.700)	0.913
Creatine kinase [M(P25,P75), U/L]	37.950(25.500,61.875)	34.650(25.400,50.150)	0.003
Creatine kinase isoenzyme [M(P25,P75), U/L]	9.700(8.000,12.300)	9.700(7.300,11.700)	0.913
α-hydroxybutyrate dehydroge-nase [M(P25,P75), U/L]	133.800(117.050,153.000)	142.050(121.250,163.850)	<0.001
Potassium [M(P25,P75), mmol/L]	4.060(3.863,4.268)	4.155(3.903,4.453)	<0.001
Sodium [M(P25,P75), mmol/L]	141.150(138.900,142.750)	139.850(137.675,141.750)	<0.001
Chloride [M(P25,P75), mmol/L]	103.650(101.525,105.600)	103.000(100.500,105.800)	0.003

Indicator	Group A (n=132)	Group B (n=100)	P-value
Calcium [M(P25,P75), mmol/L]	2.290(2.213,2.360)	2.265(2.190,2.360)	0.003
Phosphorus [M(P25,P75), mmol/L]	1.230(1.090,1.350)	1.235(1.083,1.385)	0.913
Magnesium ($\bar{x}\pm s$, mmol/L)	0.916$±0.082|0.906±$0.07	75	913
Glucose [M(P25,P75), mmol/L]	5.260(4.863,6.348)	5.480(4.933,5.990)	0.913
C-reactive protein [M(P25,P75), mg/L]	28.950(9.950,60.625)	29.600(9.330,66.525)	0.913
Erythrocyte sedimentation rate [M(P25,P75), mm/h]	43.000(21.250,60.525)	35.000(19.000,52.750)	<0.001

Note: "a" indicates Z-value; remaining test statistics are t-values.

Supplementary Table 2. Comparison of Laboratory Indicators Between Groups CA and CB

Indicator	Group CA (n=71)	Group CB (n=61)	P-value
White blood cell count [M(P25,P75), $×10^{9}$/L]	6.590(5.160,8.150)	6.200(5.185,7.470)	0.913
Neutrophil count [M(P25,P75), $×10^{9}$/L]	3.920(3.210,5.590)	4.010(2.940,4.870)	0.913
Eosinophil count [M(P25,P75), $×10^{9}$/L]	0.110(0.050,0.210)	0.060(0.020,0.165)	0.003

<table>
<thead>
<tr><th>Indicator</th><th>Group CA (n=71)</th><th>Group CB (n=61)</th><th>P-value</th></tr>
</thead>
<tbody>
<tr><td>Basophil count [M(P25,P75), $×10^{9}/L]|0.030(0.020, 0.040)|0.020(0.010, 0.030)| < 0.001||Lymphocytecount(x±$s, $×10^{9}/L)|1.610±0.670|1.678±$0.688</td><td>0.574</td><td></td><td></td></tr>
<tr><td>Monocyte count [M(P25,P75), $×10^{9}/L]|0.400(0.300, 0.530)|0.450(0.325, 0.520)|0.913||Neutrophilpercentage(x±s,±11.068|63.746±11.261</td><td>0.120</td><td></td><td></td></tr>
<tr><td>Monocyte percentage [M(P25,P75), %]</td><td>6.300(5.100,8.000)</td><td>6.800(5.800,8.250)</td><td>0.913</td></tr>
<tr><td>Neutrophil-to-lymphocyte ratio [M(P25,P75)]</td><td>2.660(1.890,3.380)</td><td>2.230(1.625,3.995)</td><td>0.003</td></tr>
<tr><td>Platelet-to-lymphocyte ratio [M(P25,P75)]</td><td>169.380(129.660,232.470)</td><td>145.270(102.475,192.515)</td><td>0.003</td></tr>
<tr><td>Monocyte-to-lymphocyte ratio [M(P25,P75)]</td><td>0.272(0.202,0.371)</td><td>0.265(0.186,0.369)</td><td>0.913</td></tr>
<tr><td>Red blood cell count [M(P25,P75), $×10^{12}/L]|4.470(4.070, 4.810)|4.390(3.990, 4.805)|0.913||Hematocrit[M(P25, P75),±$s, $×10^{9}/L)|268.423±75.554|235.853±$80.016</td><td>0.020</td><td></td><td></td></tr>
<tr><td>Plateletcrit [M(P25,P75), %]</td><td>0.230(0.180,0.280)</td><td>0.200(0.170,0.240)</td><td><0.001</td></tr>
<tr><td>Mean platelet volume [M(P25,P75), fl]</td><td>8.800(8.100,9.200)</td><td>8.900(8.250,10.100)</td><td>0.003</td></tr>
<tr><td>Platelet distribution width [M(P25,P75), %]</td><td>15.800(15.600,16.000)</td><td>15.900(15.700,16.100)</td><td>0.003</td></tr>
</tbody>
</table>

Indicator	Group CA (n=71)	Group CB (n=61)	P-value
Large platelet ratio [M(P25,P75), %]	17.400(13.100,21.100)	19.100(15.000,26.500)	<0.001
Large platelet count [M(P25,P75), $\times10^{9}/L$]	44.000(34.000, 58.000)	44.000(31.000, 63.000)	0.913
Prothrombin time $(x\pm s, s)$	11.523±0.961	11.4	
d-dimer $[M(P25, P75), \mu g/L]$	285.000(140.000, 697.000)	329.000(190.500, 535.500)	0.913
International normaliz			
Prothrombin time activity [M(P25,P75), %]	93.000(86.000,105.000)	98.000(90.000,104.000)	0.003
Prothrombin time ratio [M(P25,P75)]	1.040(0.088)	1.031(0.087)	0.585
Alanine aminotransferase [M(P25,P75), U/L]	17.600(11.400,30.100)	35.800(15.700,65.900)	<0.001
Aspartate aminotransferase [M(P25,P75), U/L]	18.200(15.300,22.800)	25.400(18.400,38.250)	<0.001
Aminotransferase ratio [M(P25,P75)]	1.100(0.730,1.610)	0.870(0.635,1.095)	<0.001
Alkaline phosphatase [M(P25,P75), U/L]	100.200(80.600,121.800)	111.200(91.150,155.150)	<0.001
γ-glutamyl transferase [M(P25,P75), U/L]	47.000(28.000,91.000)	65.000(32.950,105.500)	0.003
Total bilirubin [M(P25,P75), mol/L]	7.670(6.340,9.590)	8.440(7.100,12.280)	<0.001

Indicator	Group CA (n=71)	Group CB (n=61)	P-value
Direct bilirubin [M(P25,P75), mol/L]	3.370(2.560,4.320)	4.210(3.095,5.335)	<0.001
Indirect bilirubin [M(P25,P75), mol/L]	4.310(3.650,5.180)	4.690(3.775,6.840)	0.003
Total protein [M(P25,P75), g/L]	68.900(63.700,73.600)	65.300(60.750,68.550)	<0.001
Albumin ($\bar{x}\pm s$, g/L)	38.189$±4.528\|37.900±4.[illegible]$		
Total cholesterol [M(P25,P75), mmol/L]	4.160(3.700,5.020)	4.370(3.705,5.480)	0.913
Apolipoprotein A1 [M(P25,P75), g/L]	1.180(1.040,1.370)	1.240(0.920,1.485)	0.913
Apolipoprotein B [M(P25,P75), g/L]	0.910(0.800,1.130)	0.970(0.810,1.100)	0.913
Low-density lipoprotein [M(P25,P75), mmol/L]	2.770(2.420,3.410)	2.890(2.480,3.675)	0.913
High-density lipoprotein [M(P25,P75), mmol/L]	1.180(0.910,1.320)	1.170(0.885,1.350)	0.913
Triglycerides [M(P25,P75), mmol/L]	1.160(0.900,1.570)	1.370(0.965,1.700)	0.003

Indicator	Group CA (n=71)	Group CB (n=61)	P-value
Creatine kinase [M(P25,P75), U/L]	43.300(29.800,71.100)	33.500(26.650,50.750)	<0.001
Creatine kinase isoenzyme [M(P25,P75), U/L]	9.700(7.700,11.900)	9.300(7.100,11.650)	0.913
α-hydroxybutyrate dehydroge-nase [M(P25,P75), U/L]	132.000(117.000,152.000)	139.000(117.150,167.500)	0.003
Potassium ($\bar{x}\pm s$, mmol/L)	4.046$±0.373\|4.162±0.416\|0.120\|\|$	$Sodium[M(P25, P75), mmol/L]\|140.900(138.900, 142.300)\|$	
Glucose [M(P25,P75), mmol/L]	5.150(4.850,5.800)	5.400(4.895,5.755)	0.913
C-reactive protein [M(P25,P75), mg/L]	28.600(9.900,56.700)	36.800(8.950,81.450)	0.003
Erythrocyte sedimentation rate [M(P25,P75), mm/h]	43.000(20.000,60.000)	38.000(19.000,50.500)	0.003

Note: "a" indicates Z-value; remaining test statistics are t-values.

Supplementary Table 3. Comparison of Laboratory Indicators Between Groups DA and DB

Indicator	Group DA (n=61)	Group DB (n=39)	P-value
White blood cell count [M(P25,P75), $×10^{9}$/L]	6.860(5.185,8.550)	5.760(4.480,6.970)	<0.001

Indicator	Group DA (n=61)	Group DB (n=39)	P-value
Neutrophil count [M(P25,P75), $×10^{9}$/L]	4.830(3.370,6.150)	3.440(2.400,4.430)	<0.001
Eosinophil count [M(P25,P75), $×10^{9}$/L]	0.110(0.070,0.195)	0.050(0.020,0.120)	<0.001
Basophil count [M(P25,P75), $×10^{9}$/L]	0.030(0.020,0.040)	0.030(0.020,0.040)	0.913
Lymphocyte count [M(P25,P75), $×10^{9}/L$|1.370(1.005, 1.815)|1.600(1.370, 2.410)| < 0.001||$Monocytecount$(x±$s, $×10^{9}/L$|0.481±0.176|0.410±0.181|0.020||$Neutrophilpercentage$($x±s$,±10.067|59.621±7.794| < 0.001||$Eosinophilpercentage$[M(P25, P75),±$s, $×10^{12}/L$|4.138±0.715|4.402±0.516|0.020||$Hematocrit$[M(P25, P75),±s, pg)|30.290±1.546|29.115±2.129|			
Platelet count [M(P25,P75), $×10^{9}/L$||253.000(191.000, 315.500)|225.000(184.000, 274.000)|0.003||$Plateletcrit$($x±s$,±0.074|0.198±0.052 $×10^{9}/L$|49.803±17.298|41.846±13.726|0.020||$Prothrombintime$[M(P25, P75), s]|11.200(10.750, 11.900)|1 0.001||$Activatedpartialthromboplastintime$[$M$($P$25, P75), s]|30.100(28.300, 32.800)|32.800(30.800, 35.600)| < 0.001||$D-dimer$[M(P25, P75), $\mu g/L$]|339.000(194.500, 651.500)|323.000(216.000, 630.000)|0.913||$Internationalnormaliz			
Prothrombin time ratio [M(P25,P75)]	1.010(0.960,1.080)	0.980(0.950,1.060)	0.003
Alanine aminotransferase [M(P25,P75), U/L]	18.000(11.650,27.200)	19.500(15.900,42.800)	0.003
Aspartate aminotransferase [M(P25,P75), U/L]	19.400(15.050,25.700)	21.900(14.000,35.700)	0.003
Aminotransferase ratio [M(P25,P75)]	0.990(0.755,1.480)	0.980(0.790,1.290)	0.913

Indicator	Group DA (n=61)	Group DB (n=39)	P-value
Alkaline phosphatase [M(P25,P75), U/L]	111.700(87.650,134.950)	116.600(97.000,144.800)	0.003
γ-glutamyl transferase [M(P25,P75), U/L]	70.000(33.000,116.400)	54.000(32.000,84.600)	0.003
Total bilirubin [M(P25,P75), mol/L]	8.510(6.770,12.190)	9.790(7.760,11.590)	0.003
Direct bilirubin [M(P25,P75), mol/L]	3.450(2.810,5.605)	3.940(3.020,6.230)	0.003
Indirect bilirubin [M(P25,P75), mol/L]	4.920(3.880,6.760)	5.300(3.930,6.850)	0.913
Total protein ($\bar{x}\pm s$, g/L)	68.593$±6.425|66.977±7.2[illegible]	[illegible]	[illegible]
	$to-globulinratio(x\pm s)$|1.281[illegible]	$densitylipoprotein(x\pm s, mmol/L)$|2.961±0.967|2.722±0.776|0.221||[illegible]	
	0.001||$Urea[M(P25,P75), mmol/L]$|6.280(4.495, 7.540)|6.470(4.690, 7.960)|0.913||$Uricacid[M$		
	0.001||$Lactatedehydrogenase[M(P25,P75), U/L]$|196.900(162.300, 214.400)|191.400(174.200,		
	($\bar{x}\pm s$, mmol/L)		
Triglycerides [M(P25,P75), mmol/L]	1.290(1.035,1.740)	1.360(0.830,1.700)	0.913
Creatine kinase [M(P25,P75), U/L]	36.100(21.050,44.550)	39.000(24.100,49.400)	0.003
Creatine kinase isoenzyme [M(P25,P75), U/L]	9.700(8.100,12.650)	10.000(7.600,12.500)	0.913
α-hydroxybutyrate dehydroge-nase [M(P25,P75), U/L]	138.000(116.850,157.200)	145.800(128.000,160.000)	0.003

Indicator	Group DA (n=61)	Group DB (n=39)	P-value
Potassium [M(P25,P75), mmol/L]	4.080(3.870,4.280)	4.170(3.900,4.370)	0.003
Sodium [M(P25,P75), mmol/L]	141.400(138.900,142.900)	139.100(137.200,141.600)	<0.001
Chloride [M(P25,P75), mmol/L]	103.600(101.400,105.400)	103.000(100.100,105.800)	0.913
Calcium [M(P25,P75), mmol/L]	2.300(2.210,2.385)	2.270(2.180,2.410)	0.003
Phosphorus [M(P25,P75), mmol/L]	1.210(1.090,1.345)	1.250(1.120,1.400)	0.913
Magnesium ($\bar{x}\pm s$, mmol/L)	0.909$±0.081	0.886±$0.06	0.358
Glucose [M(P25,P75), mmol/L]	5.660(4.925,6.895)	5.600(4.990,6.900)	0.913
C-reactive protein [M(P25,P75), mg/L]	29.500(10.000,61.750)	24.900(9.420,41.400)	0.003
Erythrocyte sedimentation rate [M(P25,P75), mm/h]	43.000(24.000,61.000)	31.000(20.000,53.000)	<0.001

Note: "a" indicates Z-value; remaining test statistics are t-values.

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Note: Figure translations are in progress. See original paper for figures.