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Volume 16 Number 8 August 2024

1605 HSD17B6

Figure 1605 HSD17B6 involved in sex hormone metabolism

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Monthly Volume 16 Number 8 August 2024

COMMENTS

Advances in laboratory diagnostic studies of brucellosis

ORIGINAL ARTICLES

Predictive value of Hsp90 CER and AFP for the curative effect of TACE in patients with primary liver cancer

The role of PMS2 MTA1 and VCAM 1 in the evaluation of colon cancer and prognosis

Construction of a nomogram model for predicting the risk of pancreatic fistula after pancreatic2MQ

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Effect of self shaping catheter intubation on levels of Cor E and NA in infants with premature closure of cranial suture

Changes of serum NSE IL 1 and 5 HT levels in patients with chronic insomnia disorder and relationship with cognitive function and sleep

Effects of exercise rehabilitation intervention on cardiac function autophagy and mTOR pathway of patients with chronic heart failure

Effect of semaglutide on elderly diabetic nephropathy and its effect on NLRP3 inflammasome pathway

Effect of remifentanyl combined anaesthesia on serum IL 6 IL 2 and T lymphocyte subsets in patients undergoing laparoscopic cholecystectomy

Relationship between OPN CRP and RANKL levels in gingival crevicular fluid and peri implant inflammation

Efficacy of preoperative lymphocyte/C reactive protein ratio combined with HALP score in the prognosis of cases with persistent atrial fibrillation undergoing radiofrequency ablation

Study on the predictive value of serum markers sTREM 1 and AQP5 for infectious endophthalmitis after cataract surgery

Analysis of HPV infection and TCT results in women with health examination in a tertiary hospital in Beijing

Characterization of gene mutations in deafness patients of Han and Mongolian ethnicities in Inner Mongolia

The value of TCCD and ONSD on blood biochemical indexes APACHE score and prognosis of cerebral resuscitation patients

Effects of Jianpi Runfei Pills on levels of CAR VEGF IFN and IL 17 in patients with pulmonary tuberculosis

Analyze the influencing factors of autologous hematopoietic stem cell mobilization in patients with multiple myeloma

The value of PCT SAA and Treg factor assays in the antimicrobial treatment of multidrug resistant bacterial infections

Changes of sLOX 1 sST2 and SREBP 1 in patients with coronary heart disease and their relationship with the severity of coronary lesions

Analysis of IL 6 CRP Hcy ACA and prognosis of patients with acute cerebral infarction

Effect of neoadjuvant chemotherapy combined with immunotherapy on locally advanced esophageal cancer and its influence on Sil 2R IFN and TSGF levels

The significance of serum IL 1 sE cad E2 combined with breast ultrasound in the differential diagnosis of plasma cell mastitis and breast cancer

Expression of SALL4 GS and HSP70 in hepatocellular carcinoma tissues and its clinical value in early screening

Risk factors of cognitive impairment and predictive value of serological indexes in patients with schizophrenia

Correlation between *TNFSF15* gene polymorphism and its related protein and primary biliary cholangitis

Relationship between serum SAA HMGB1 TNF expression and disease activity in patients with ankylosing spondylitis

Efficacy of butylphthaloin combined with E daravone dextrocamphorol in the treatment of arteriolar occlusion type stroke

Correlation between serum FIB urine RBP NAG TRF and pathological grade of kidney in children with purpura nephritis

Correlation between AMPK/mTOR activation and epithelial cell injury in chronic nasosinusitis

Influencing factors of intracranial infection and predictive value of NF B PCT and IL 1 in cerebrospinal fluid after external ventricular drainage

Effect of Huangqi Yishen Granules combined with sevelamer on TLR4 and NF B in patients with maintenance hemodialysis

Expression and prognostic value of tumor markers cytokeratin 19 fragment antigen 21 1 ALI and PLR in non small cell lung adenocarcinoma

Efficacy of Zhenwu decoction on patients with chronic pulmonary heart disease and influence on levels of IL 8 PCT and hs CRP

Clinical efficacy of NPWT combined with silver ion dressing in elderly male diabetic toe osteomyelitis and its effect on MMP 2 TMP 1 and VEGF

The relationship between 1q21 amplification serum IL 21 lactate dehydrogenase and efficacy and prognosis of patients with multiple myeloma

The levels and clinical significance of serum lncRNA PVT1 and HIF 1 in patients with endometriosis

Correlation between PDK1 expression in peripheral blood and inflammatory response in patients with chronic heart failure and its clinical significance

REVIEWS

The introduction of methods of bioinformatics analysis and Cut off value determination on pathogen metagenome Sequencing reagent

The main function of HSD17B6 and its role in endocrine metabolism and tumors



DING Haitao CHEN Yuetong WANG Bo HE Juan SHI Yue LI Xiacong WANG Rui WANG Zhanguo
Clinical Laboratory Inner Mongolia Peoples Hospital Hohhot Inner Mongolia China 010017

Brucellosis is a zoonotic disease with multiple clinical presentations. Due to the variety of clinical presentations and the lack of specific symptoms laboratory diagnosis is essential to confirm the diagnosis and treatment of the disease. The traditional laboratory tests for Brucella can be divided into culture identification and serological tests. Nucleic acid amplification tests are a new development that has emerged in recent years and a variety of molecular diagnostic techniques have been applied to the detection of Brucella. In this paper we will describe the most common and updated laboratory testing methods for Brucella and compare their advantages and disadvantages to provide a more comprehensive guide for the diagnosis and treatment of brucellosis.

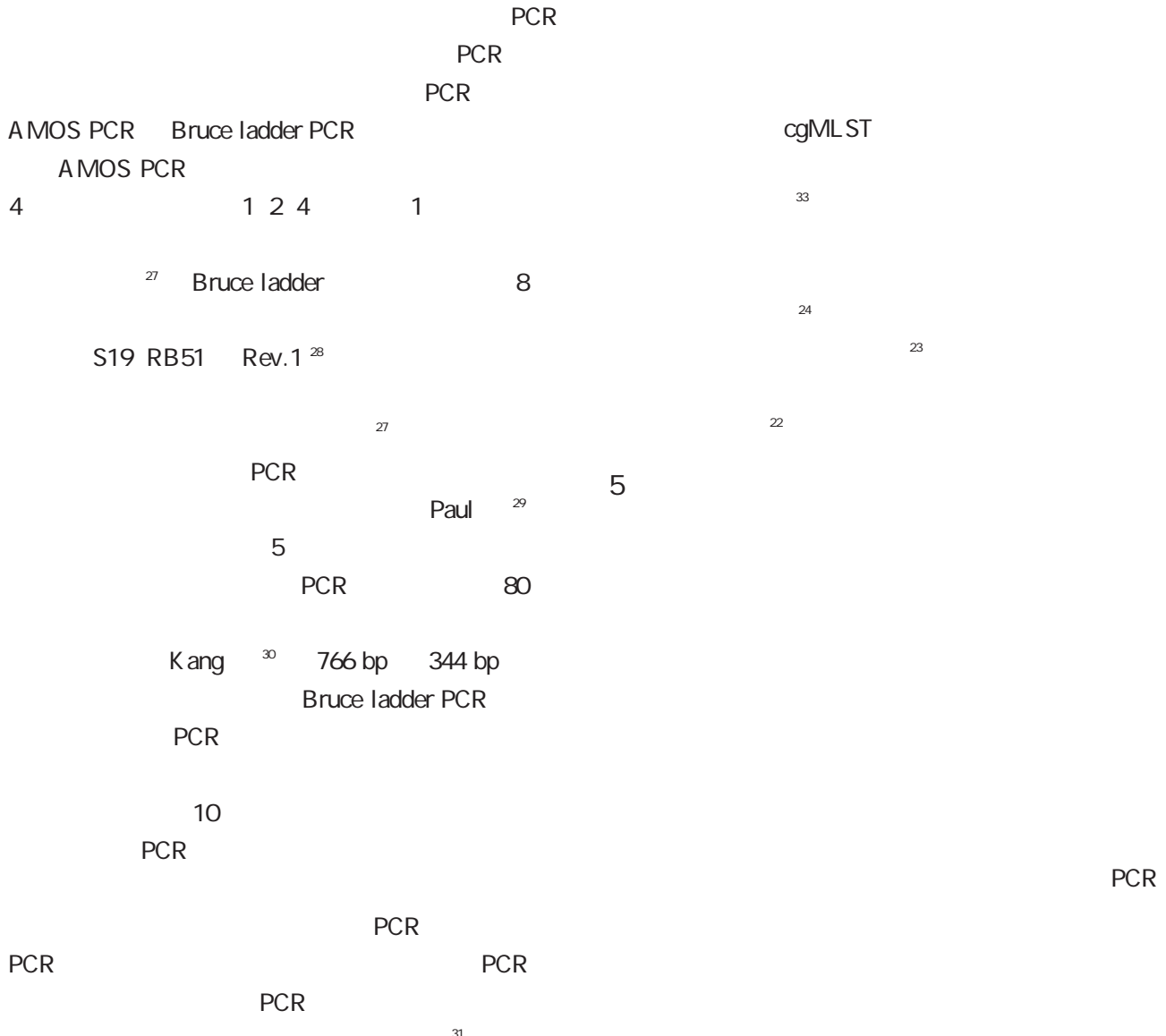
Brucellosis Laboratory diagnosis Polymerase chain reaction Metagenomic next generation sequencing

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Hsp90 CER AFP TACE

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90 Hsp90 CER AFP

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70 TACE 2 mRECIST

Hsp90 CER AFP

ROC TACE TACE TACE

72.86% $t^2=5.440$

10.928 $P<0.05$ TACE Hsp90 CER AFP

$t=2.501$ 2.082 2.964 $P<0.05$ TACE Hsp90 CER AFP

$t=7.196$ 6.866 8.687 $P<0.05$ TACE Hsp90 CER

AFP $F=4.966$ 4.817 7.878 $P<0.05$ Hsp90 CER AFP

$t=8.169$ 3.434 10.276 $P<0.05$ ROC TACE

Hsp90 CER AFP TACE AUC $P<0.05$ Hsp90

CER AFP TACE

90

YAO Dongmei¹ TIAN Tian²

1. Department of Oncology Linquan County People s Hospital Fuyang Anhui China 236400 2 Department of Oncology Fuyang People s Hospital Fuyang Anhui China 236000

To analyze the predictive value of serum Hsp90 CER and AFP for the curative effect of TACE in patients with primary liver cancer. A total of 70 patients with primary liver cancer undergoing TACE at Linquan County People s Hospital were enrolled as research subjects between January 2020 and January 2023. The postoperative curative effect was evaluated using mRECIST at 2 months after surgery and the treatment response rate was calculated. Based on the curative effect patients were divided into a good group and a poor group. The baseline data levels of serum Hsp90 CER and AFP were compared between the two groups. The predictive value of the serum indicators mentioned above for the curative effect of TACE was analyzed using ROC curves. In the 70 patients with primary liver cancer the response rate of TACE was 72.86%. There were significant differences in clinical staging and tumor boundary between the good group and poor group $t^2=5.440$ 10.928 $P<0.05$. Before TACE levels of serum Hsp90 CER and AFP in the poor group were higher than those in the good group $t=2.501$ 2.082 2.964 $P<0.05$. After TACE levels of serum Hsp90 CER and AFP in the poor group were higher than those in the good group $t=7.196$ 6.866 8.687 $P<0.05$. Before TACE levels of serum Hsp90 CER and AFP increased with the increase of clinical staging

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F=4.966 4.817 7.878 P<0.05 . The levels of serum Hsp90 CER and AFP in patients with irregular tu
 mor boundaries were higher than those with regular tumor boundaries t=8.169 3.434 10.276 P<0.05 . ROC
 curve analysis showed that the AUC of serum Hsp90 combined with CER and AFP before TACE for predicting
 the curative effect was the greatest P<0.05 Serum levels of Hsp90 CER and AFP can help
 predict the clinical effectiveness of TACE. These markers can provide a basis for clinical intervention.

. Primary liver cancer TACE Hsp90 CER AFP

transcatheter arterial chemo
 embolization TACE TACE 2
 immune modified Response Evaluation Criteria In
 Solid Tumors mRECIST 8
 TACE 3 30%
 TACE 4 Hsp90 90 heat shock protein Hsp90 20%
 Ceruloplasmin CER 5 TACE 2
 5 mL 10 min 3 500 r/min
 Hsp90 AFP Roche 2010
 CER DADERHERING
 alpha fetoprotein AFP 6 Hsp90 CER 1.4
 SPSS 22.0
 n % 2
 1
 1.1 t t
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 TACE P<0.05
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 TACE TACE 2
 2.1 TACE TACE 2 72.86%
 2.2 70 TACE
 n=51 n=19
 1.2 TACE 4 mg 40 P<0.05 1
 mg 10-20 mL 2.3 TACE TACE

	AUC	95% CI			
Hsp90	0.697	241.28 ng/mL	0.566-0.827		
CER	0.739	44.32 mg/dL	0.615-0.863		
AFP	0.669	156.34 ng/mL	0.5	6CER	
	0.826		A	CER	1
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Table 2 Comparison of serum indexes between the two groups

	n	Hsp90 ng/mL		CER mg/dL		AFP ng/mL	
	19	250.17±25.36	135.45±15.17 ^a	46.35±4.72	32.47±4.14 ^a	167.80±19.34	123.48±13.62 ^a
	51	232.36±26.89	106.62±14.81 ^a	43.58±5.03	25.60±3.56 ^a	152.73±18.76	96.25±10.87 ^a
t		2.501	7.196	2.082	6.866	2.964	8.687
P		0.015	<0.001	0.041	<0.001	0.004	<0.001

^aP<0.05

3 TACE

Table 3 Comparison of serum indexes in patients with different clinical characteristics before TACE

	n	Hsp90 ng/mL	t	P	CER mg/dL	t	P	AFP ng/mL	t	P
	48	225.16±29.85	4.966	<0.001	42.60±4.41	4.817	<0.001	146.05±15.76	7.878	<0.001
	22	263.45±30.16			48.14±4.59			180.33±19.21		
	44	213.59±30.65	8.169	<0.001	42.55±5.89	3.434	0.001	140.78±15.12	10.276	<0.001
	26	277.14±32.77			47.35±5.21			183.95±19.78		

<p>11</p> <p style="padding-left: 40px;">TACE</p> <p style="padding-left: 80px;">TACE</p> <p style="padding-left: 120px;">Hsp90 CER AFP</p> <p>TACE Hsp90 CER AFP</p> <p>Hsp90 CER AFP</p> <p style="padding-left: 40px;">Hsp90 CER AFP</p> <p>12 TACE AFP</p> <p style="padding-left: 80px;">TACE</p> <p style="padding-left: 120px;">Hsp90</p> <p>13</p> <p style="padding-left: 40px;">CER</p> <p style="padding-left: 80px;">Hsp90 CER</p> <p>AFP</p> <p>AFP</p> <p>14 AFP</p> <p style="padding-left: 80px;">ROC</p> <p>TACE Hsp90 CER AFP</p> <p>TACE AUC</p> <p>15</p> <p style="padding-left: 40px;">0.550 0.600</p> <p>Hsp90 CER AFP</p> <p>TACE</p> <p style="padding-left: 80px;">Hsp90 CER AFP</p> <p style="padding-left: 120px;">TACE</p>	<p>1 Xie Y Tian H Xiang H. Is transcatheter arterial chemoembolization plus sorafenib better than chemoembolization plus placebo in the treatment of hepatocellular carcinoma J . Tumori 2021 107 4 292 303.</p> <p>2 Han T Yang X Zhang Y et al. The clinical safety and efficacy of conventional transcatheter arterial chemoembolization and drug eluting beads transcatheter arterial chemoembolization for unresectable hepatocellular carcinoma A meta analysis J . Biosci Trends 2019 13 5 374 381.</p> <p>3 v TACE J . 2018</p> <p>25 23 1658 1663.</p> <p>4 90 J .</p> <p>2022 38 3 577 581.</p> <p>5 . . 1 3826 58 \$ 1 . \$ " \$66 166</p>
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PMS2 MTA1 VCAM 1

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 PMS2 MTA1 VCAM 1
 PMS2 MTA1 VCAM 1
 MTA1 + VCAM 1 + ~
 P<0.05
 P>0.05 COX
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²=9.527 P<0.05
 PMS2 MTA1 VCAM 1

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 PMS2 MTA1 VCAM 1
 COX
 PMS2 MTA1 VCAM 1
 P<0.05 TNM ~ PMS2 +
 PMS2 + MTA1 + VCAM 1 +
 PMS2 + MTA1 + VCAM 1 +
 G3 TNM ~ PMS2 + MTA1 +
 P<0.05 COX G3
 PMS2 + MTA1 + VCAM 1 +
²=7.161 P<0.05 VCAM 1
 PMS2 MTA1 VCAM 1

1 MTA1
 2019 1 2021 3
 81
 PMS2 MTA1 VCAM 1
 COX
 PMS2 MTA1 VCAM 1
 PMS2 + MTA1 + VCAM 1 +
²=6.688 P<0.05 MTA1

XI Lefeng ZHANG Dechen WANG Liqin LI Fengying ZHAO Zhi
 Department of Pathology Zhengzhou Yihe Hospital Zhengzhou Henan China 450000

To investigate the role of postmeiotic protein 2 (PMS2) tumor metastasis associated gene 1 (MTA1) and vascular cell adhesion molecule 1 (VCAM 1) in the evaluation of colon cancer and prognosis. A total of 81 patients with colon cancer who were admitted to Zhengzhou Yihe Hospital from January 2019 to March 2021 were selected as the study subjects. All patients underwent surgical treatment. The expression of PMS2, MTA1 and VCAM 1 in colon cancer tissues and adjacent tissues of these patients was compared. The expression levels of PMS2, MTA1 and VCAM 1 in different pathological features were analyzed. The COX univariate and multivariate factors affecting the survival of patients with colon cancer were examined. The prognostic survival rates of patients with different PMS2, MTA1 and VCAM 1 expression were compared. The positive expression rates of PMS2, MTA1 and VCAM 1 in colon cancer tissues were significantly higher than those in adjacent tissues and the differences were statistically significant (P<

0.05. PMS2 + MTA1 + and VCAM 1 + in TNM stage ~ were higher than those in TNM stage ~ PMS2 + MTA1 + and VCAM 1 + in lymph node metastasis were higher than those in no lymph node metastasis. PMS2 + MTA1 + and VCAM 1 + in low differentiation were higher than those in high and medium differentiation and the differences were statistically significant $P < 0.05$. However, there was no significant difference in PMS2 + MTA1 + and VCAM 1 + between different tumor sizes $P > 0.05$. COX univariate analysis showed that tissue differentiation G3 TNM stage ~ distant yes PMS2 + MTA1 + VCAM 1 + were the poor prognostic factors affecting the death of colon cancer patients $P < 0.05$. COX multivariate analysis showed that tissue differentiation G3 TNM stage ~ distant metastasis yes PMS2 + MTA1 + VCAM 1 + were independent prognostic risk factors for death in patients with colon cancer $P < 0.05$. The survival rate of the PMS2 positive group was lower than that of the negative group, the difference was statistically significant $\chi^2 = 6.688$ $P < 0.05$. The survival rate of the MTA1 negative group was lower than that of the positive group and the difference was statistically significant $\chi^2 = 7.161$ $P < 0.05$. The survival rate of the VCAM 1 negative group was lower than that of the positive group and the difference was statistically significant $\chi^2 = 9.527$ $P < 0.05$ PMS2, MTA1 and VCAM 1 are highly expressed in colon cancer tissues. By detecting the expression of the three indicators, the severity and prognostic risk of the patients can be more accurately judged.

. PMS2, MTA1, VCAM 1, Colon cancer

1

Metastasis associated gene

family MTA 2 Post

meiotic Segregation Increased 2 PMS2

² PMS2

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1 Metastasis Associated

7 PMS2 MTA1 0
 <5 1
 6%~25% 2
 26%~50% 3
 51% 0
 1~3 VCAM 1
 0 1
 <30% 2 + VCAM 1 +
 31%~70% 3 P<0.05
 71% 0 1~3 PMS2 + MTA1 + VCAM 1 +
 1.2.2 P>0.05 2
 3 2.3 COX

2024 3 81 61
 1.3 75.31% 20 24.69% COX
 SPSS 22.0 G3 TNM ~
 n % 2 Kaplan Meier PMS2 + MTA1 + VCAM 1 +
 Log rank Cox P<0.05
 P<0.05 G3
 TNM PMS2 +
 MTA1 + VCAM 1 +
 P<0.05 3
 2.1 PMS2 MTA1 VCAM 1 2.4 PMS2 MTA1 VCAM 1
 PMS2 MTA1 VCAM 1
 P< 81.25% 39/
 0.05 1 48 54.55% 18/ 5 %
 2.2 PMS2 MTA1 VCAM 1 5 %
 TNM ~ PMS2 + MTA1 +
 VCAM 1 + ~
 PMS2 + MTA1 + VCAM 1 +
 PMS2 + MTA1

3 COX
 Table 3 COX univariate and multivariate analysis of survival in patients with colon cancer

		HR		95% CI		P	
TNM	60 =0 >60 =1	0.813	0.428-1.189			0.211	
	=0 =1	0.750	0.336-1.059			0.118	
	G1/2=0 G3=1	1.835	1.137-2.315			0.015	
	=0 =1	0.713	0.321-1.076			0.102	
PMS2	~ =0 ~ =1	2.436	1.680-3.428			<0.001	
	=0 =1	3.308	2.188-5.027			<0.001	
MTA1	=0 + =1	3.483	2.014-5.147			<0.001	
VCAM 1	=0 + =1	4.112	2.371-6.811			<0.001	
	=0 + =1	4.414	1.192-14.376			<0.001	

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8 PMS2

PCT

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POPF PCT PD

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PD POPF POPF 35 POPF POPF 26 9

P<0.05 logistic Back wald

AUC

Hosmer Lemeshow

POPF POPF FRS

PCT PCT P<0.05 Logistic

0.25 cm FRS 5 240 cm 98 cm

PD POPF POPF

P<0.05 PD POPF

ROC AUC 0.889 95%CI 0.740-

Hosmer Lemeshow P=0.793 0.688

0.966

PCT PD POPF

POPF POPF

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MA Ying¹ WEI Jihong¹ HU Haiyan¹ ZENG Huijuan¹ YANG Huan¹ CHEN Xi²

1. Hepatobiliary Pancreatic Spleen Breast Otolaryngology day care unit Mianyang Central Hospital Mianyang Sichuan China 621000 2 Hepatobiliary Pancreatic and Splenic Surgery Mianyang Central Hospital Mianyang Sichuan China 621000

To investigate a column line graphical model of procalcitonin (PCT), albumin and bilirubin levels to predict the risk of postoperative pancreatic fistula (POPF) following pancreaticoduodenectomy (PD)...

A total of 120 patients with PD who underwent pancreatic surgery at Mianyang Central Hospital from January 2020 to January 2023 were retrospectively selected. 7 cases were excluded, leaving a total of 113 cases included in the study. According to a 3:1 ratio, 35 cases developed POPF based on the diagnostic criteria proposed by ISGPF in 2016. Of these, 26 cases were from the modeling group and 9 cases from the validation group. We analyzed the single factors contributing to POPF after PD in the modeling group. We included variables with P<0.05 in the binary logistic regression analysis and screened them using the Backward method. Based on this analysis, we constructed a prediction model for the risk of postoperative POPF after

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PD in a column line diagram. The AUC was used to evaluate the discrimination of the column line graph model the calibration curve to assess the relationship between the model's predicted odds and the actual probability and the Hosmer Lemeshow goodness of fit test to evaluate the fit of the column line graph model. Comparison of the levels of pancreatic duct diameter pancreatic texture FRS score total abdominal fat abdominal wall fat preoperative bilirubin preoperative albumin and preoperative PCT between those who developed POPF and those who did not in the modeling group showed statistically significant differences $P < 0.05$. The results of multifactorial logistic regression analysis showed that pancreatic duct diameter > 0.25 cm pancreatic texture as hard FRS score ≥ 5 total abdominal fat ≥ 240 cm abdominal wall fat ≥ 98 cm preoperative bilirubin and preoperative increase in PCT level were the risk factors affecting the occurrence of postoperative POPF in PD and the preoperative decrease in albumin level was the protective factor affecting the occurrence of POPF $P < 0.05$. Each factor corresponded to a corresponding score and an increase in the risk factor score increased the risk of POPF after PD surgery if the protective factor score increased the risk of POPF after PD surgery decreased. Model validation the AUC curve in the ROC of the modeling group was 0.889 with a 95% CI of 0.740-0.966 the calibration curve of the model in the modeling group and the validation group was close to the standard curve Hosmer Lemeshow goodness of fit test $P = 0.793$ 0.688. PCT albumin and bilirubin levels play an important role in predicting POPF after PD surgery.

Keywords: pancreatic duct cancer; POPF; CT; abdominal wall fat; abdominal wall thickness; bilirubin; albumin; PCT; FRS score; pancreatic texture; pancreatic duct diameter; logistic regression analysis; ROC curve; calibration curve; Hosmer Lemeshow goodness of fit test

PCT

1.2.3

		3 1			POPF	n=26
		n=84	n=29			
2016	ISGPF	POPF	6	35	14	53.85
POPF		POPF	26	9	12	46.15
1.3					59.36±8.62	
	SPSS 26.0 R 4.1.0				12	46.15
	-				15	57.69
t	n %		2		10	38.46
	Logistic		Back wald		13	50.00
				ASA	362.88±100.06	
					200.14±48.06	
	AUC				3	11.54
					23	88.46
				cm		0.39
	Hosmer Lemeshow					
	P<0.05					

2

2.1	PD	POPF		FRS	20%	5-(4
		POPF	POPF	cm	%	z	5.36
				cm	15	57.69	2.06 (z ' " & ") z
		ASA		μmol/L	200	200E	
		P>0.05		g/L	14.57.		
	FRS			PCT ng/mL			
		PCT		95% CI	0.740-0.966	57.368±	
		P<0.05	1			69	
2.2	PD	POPF	Logistic	Lemeshow			
		POPF			P=0.793	0.688	
					3		

Logistic

		FRS	5	0.25 cm	3	
		98 cm		240 cm	POPF	
	PD	POPF		PCT		
		POPF	P<0.05	2		
2.3	PD	POPF				
		FRS				
		PCT				
PD	POPF			ASA		11
		PD	POPF	Gallery	FRS	
		ROC	AUC		POPF	12
			0.889			

Table 2 Multivariate logistic regression analysis for predicting postoperative POPF in PD patients

		SE	Wald	OR	95% CI	P
FRS	0=<0.25 cm 1= 0.25 cm	1.660	0.633	6.833	5.259 1.520-18.186	0.010
	0= 1=	1.473	0.637	5.424	4.362 1.251-15.203	0.018
	0=<5 1= 5	1.570	0.589	9.640	4.806 1.515-15.247	0.008
	0=<240 cm 1= 240 cm	1.426	0.650	5.375	4.162 1.164-14.879	0.024
	0=<98 cm 1= 98 cm	1.583	0.565	7.632	4.869 1.608-14.737	0.001
PCT		1.674	0.611	7.857	5.333 1.610-17.664	<0.001
		-1.522	0.498	10.340	0.218 0.082-0.579	<0.001
		1.620	0.596	7.863	5.053 1.571-16.251	<0.001

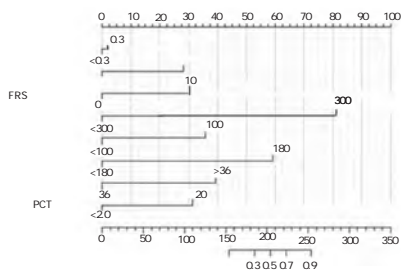


Figure 1 Prediction column line graph model

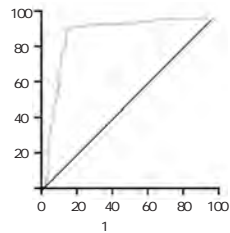


Figure 2 ROC curve of the prediction model in the modeling group

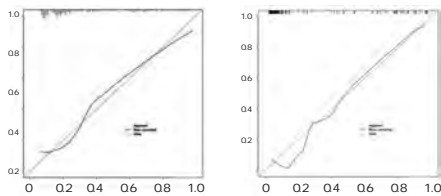


Figure 3 Calibration curves of the predictive model in the modeling and validation groups

Logistic
FRS
PCT PD POPF
POPF
POPF

PD

PCT

PCT

POPF

13.14

PO

Back wald

PD POPF

U• ç 50e! #rAE 0 1'

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PCT

PCT

POPF

POPF

0.889 0.863 $P < 0.05$. Logistic analysis showed that the increase of dialysis age blood phosphorus ALP CRP iPTH OPN and FGF23 in MHD patients were risk factors for vascular calcification $P < 0.05$. Receiver operating characteristic curve ROC curve analysis showed that the area under the curve AUC of serum OPN and FGF23 were 0.845 0.891 and . ~~U~~

wer

U DSF PPI

2

2.1 MHD

P>0.05

ALP CRP iPTH OPN FGF23

P<0.05 1

1 MHD n % ±s

Table 1 Univariate analysis of vascular calcification in MHD patients n % ±s

	n=42	n=74	t/	P
			0.185	0.667
	25 59.52	41 55.41		
	17 40.48	33 44.59		
	51.24±8.87	52.30±8.47	0.637	0.526
			0.556	0.906
	17 40.48	27 36.49		
	12 28.57	19 25.68		
	7 16.67	15 20.27		
	6 14.29	13 17.57		
	3.14±0.68	4.27±1.02	6.410	<0.001
/	2.35±0.43	2.50±0.47	1.703	0.091
	1.62±0.35	1.57±0.41	0.664	0.508
mmol/L	2.31±0.61	2.48±0.75	1.252	0.213
mmol/L	1.41±0.32	2.25±0.48	10.126	<0.001
ALP U/L	94.37±14.28	76.59±13.34	6.725	<0.001
μmol/L	813.48±134.20	806.35±136.52	0.272	0.786
mmol/L	18.12±3.26	17.94±3.34	0.281	0.779
CRP mg/L	7.46±2.28	9.74±2.36	5.062	<0.001
g/L	103.67±10.25	106.47±10.48	1.394	0.166
g/L	36.29±4.37	36.85±4.29	0.671	0.504
iPTH pg/mL	278.55±36.14	426.37±54.80	15.642	<0.001
OPN ng/mL	148.75±26.43	289.56±40.59	26.427	<0.001
FGF23mg/L	84.76±11.28	247.56±35.41	28.926	<0.001

2.2

MHD OPN FGF23

MHD OPN FGF23

> > P<

0.05 2

2.3 MHD

Spearman

MHD OPN FGF23

r=0.889 0.863 P<0.05

2.4 MHD

MHD MHD

ALP CRP iPTH OPN FGF23

P<0.05 3

2

MHD OPN FGF23

Table 2 Comparison of serum OPN and FGF 23 levels in MHD patients with different degrees of calcification

	n	OPN ng/mL	FGF23 mg/L
	21	192.82±27.10	122.54±14.60
	29	294.31±30.05 ^a	254.27±20.73 ^a
	24	368.46±35.14 ^{ab}	348.84±32.25 ^{ab}
F		148.683	231.201
P		<0.001	<0.001

^aP<0.05 ^bP<0.05

3 MHD

Table 3 Multi factor analysis of vascular calcification in MHD patients

	SE	Wald/	OR	95% CI	P
	0.624	0.312	4.000	1.866 1.012-3.440	0.046
	0.816	0.297	7.549	2.261 1.263-4.048	0.006
ALP	0.779	0.273	8.142	2.179 1.276-3.721	0.004
CRP	0.673	0.334	4.060	1.960 1.018-3.772	0.044
iPTH	0.886	0.286	9.597	2.425 1.385-4.248	0.002
OPN	0.975	0.309	9.956	2.651 1.447-4.858	0.002
FGF23	1.143	0.311	13.507	3.136 1.705-5.769	<0.001

2.5

OPN FGF23 MHD

OPN FGF23 MHD

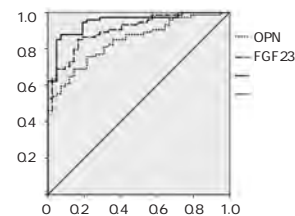
AUC 0.948

P<0.05 4 1

4 OPN FGF23 MHD

Table 4 The predictive value of serum OPN and FGF 23 levels on vascular calcification in MHD patients

	AUC	95% CI	P
OPN	0.845	0.777-0.913	0.546 0.857 0.689 <0.001
FGF23	0.891	0.853-0.958	0.684 0.833 0.851 <0.001
	0.948	0.909-0.987	0.807 0.929 0.878 <0.001



1 ROC

Figure 1 ROC curve

3

MHD

20-30

MHD

MHD

8

MHD

MHD

9

MHD

MHD

OPN

OPN

OPN

¹⁰¹¹

Fletcher 10UÀ

7 345 HPV

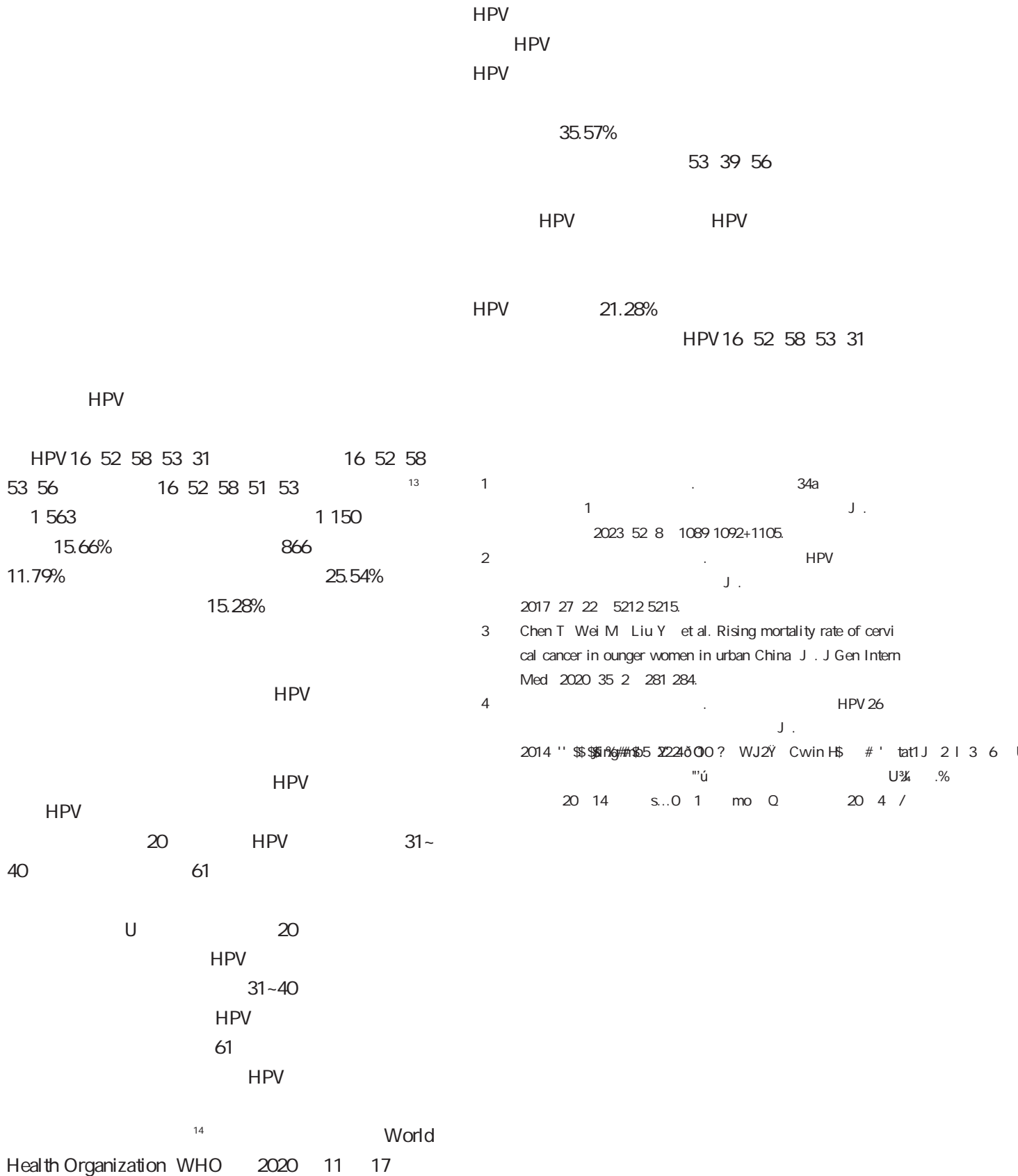
HPV 7 345 HPV 7 345 HPV 1 563
2020 1 2022 12
21.28% 25.54% 1 097/4 295
15.28% 466/3 050 $\chi^2=112.13$ P<
0.05 11.79% 866/7 345 5.62% 413/7 345
5 HPV 16 3.89% HPV 52 3.05% HPV 58 2.06% HPV 53 1.97% HPV 31
1.54% HPV 20 39.06% 61 27.24%
HPV 64.43% HPV
21.28%

ZHANG Feng Wq q tc# o O Wq NGNGG Wq NGG

51 52 56 58 66 68 82 26 53 72 10
 HPV 6 11 40 42 43 44 54 61 81 83
¹² HPV 99.88%
³ 95.94% 5×10³
 human pap /mL
 illoma virus HPV 1.4
 200 HPV 7 345 6
 HPV 20 64 21-30 1 394 31-40
⁴ HPV 2 915 41-50 1 899 51-60
 HPV ⁵ HPV 918 61 155
 HPV 1.5
 SPSS 23.0
 n % ² P<0.05
 1
 1.1 2
 2020 1 2022 12 HPV 2.1 HPV 7 345 1 563
 HPV 7 345 21.28%
 4 295 3 050
²=11213 P<0.05
 38.68±5.50 HPV 22.71% 342/1 563
 HPV
 1.2 2023 082
 24 h 3 d
 5
 HPV
 1.3 HPV DNA
 PCR PCR RDB
 28
 18 HPV 16 18 31 33 35 39 45
 1 2020-2023 HPV n %
 Table 1 HPV infection in 2020-2023 n %

	n	%	%				%			
							+			
2020	1 994	416 20.86	252 12.63	59 2.96	311 15.60	37 1.86	5 0.25	63 3.16	105 5.27	
2021	2 447	516 21.08	298 12.18	80 3.27	378 15.45	65 2.66	8 0.33	65 2.66	138 5.64	
2022	2 904	631 21.72	316 10.88	145 4.99	461 15.87	70 3.34	12 0.41	88 3.03	170 5.85	
	7 345	1 563 21.28	866 11.79	284 3.87	1 150 15.66	172 2.34	25 0.34	216 2.94	413 5.62	

HPV	2020		0.95 100	z # + Uc
	n=1 994			
16	78	3.91		
52	66	3.31		
58	57	2.86		
53	49	2.46		
31	21	1.01		
39	21	1.05		
56	25	1.25		
66	23	1.15		
51	16	0.80		
18	24	0.12		
68	11	0.55		
33	19	0.95		
35	10	0.50		
59	4	0.20		
82	4	0.20		
45	2	0.10		
26	2	0.10		
73	1	0.05		
61	37	1.86		
54	23	1.15		
81	11	0.55		
42	15	0.75		
6	15	0.75		
40	11	0.55		
44	8	0.40		
11	6	0.30		
43	8	0.40		
83	2	0.10		



• •

FT3 FT4 RC

RC ACI FT3 FT4 8 AC =μμx 78%50%x5#x ' ö ` @ Õβ%
“ f ! ñ %

significantly higher than those in the good prognosis group $P < 0.05$. Serum FT3 and FT4 levels were also significantly lower in the poor prognosis group. High NIHSS score high hs CRP high D D low FT3 low FT4 high RC high maximum diameter of infarct and moderate and severe arterial stenosis were identified as independent risk factors for poor prognosis in ACI patients $P < 0.05$. The area under curve AUC of serum FT3 FT4 RC and their combined diagnosis of poor prognosis in ACI patients were 0.869 0.832 0.840 and 0.951 respectively. The AUC of their combined diagnosis was significantly higher than that of their single diagnosis $P < 0.05$. The abnormal levels of serum FT3 FT4 and RC are closely related to the occurrence and development of ACI. The combined detection of these levels has a high diagnostic value for predicting the prognosis of ACI patients.

Arteriosclerotic cerebral infarction Free triiodothyronine Remnant cholesterol

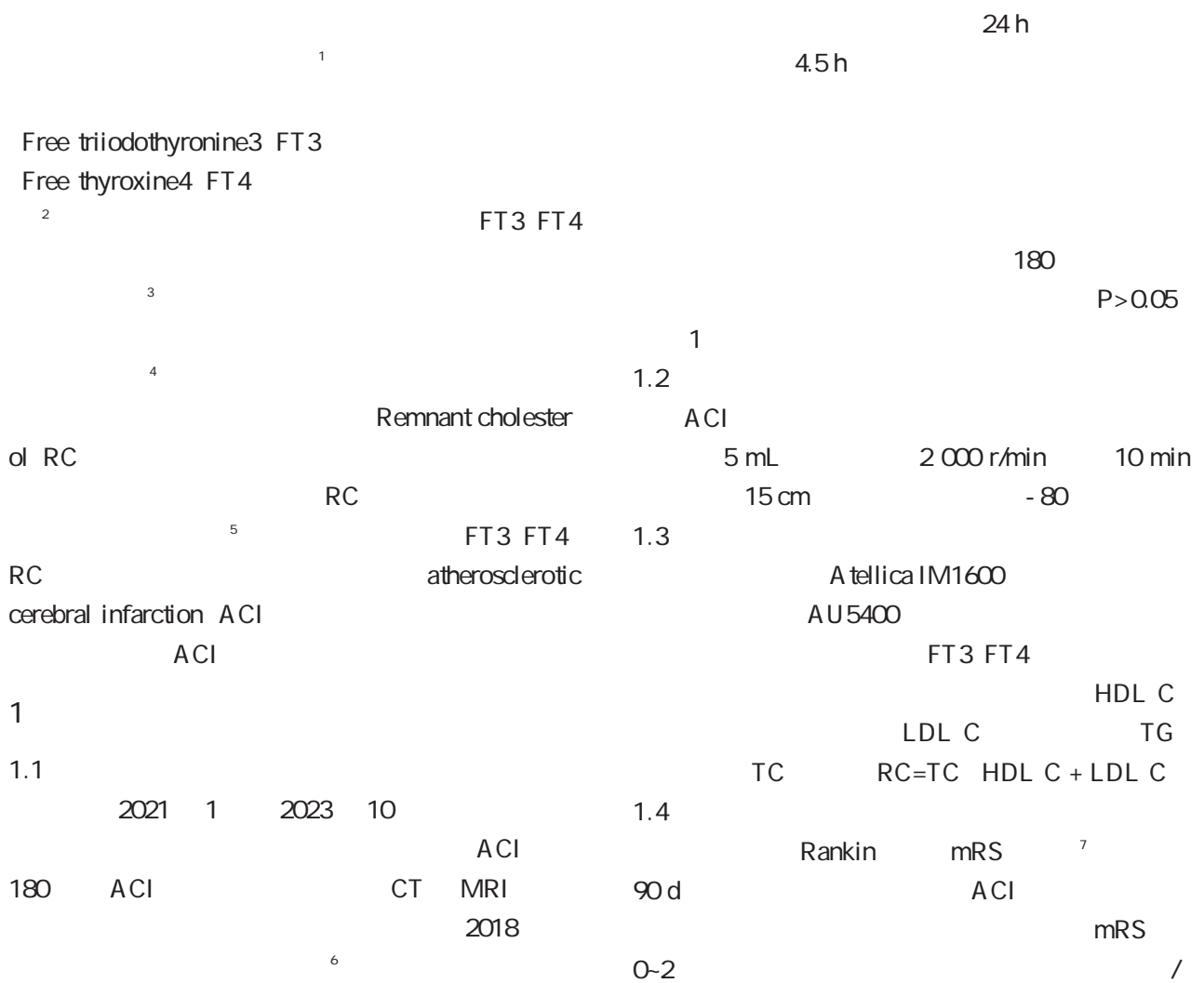


Table 1 Comparison of general data between ACI group and control group

	n											
ACI	180	55.11±6.20	105	75	86	94	91	89	59	121	62	118
t/	180	55.47±6.19	103	77	85	95	88	92	76	104	75	105
P		0.551		0.046		0.011		0.100		3.425		1.991
		0.582		0.831		0.916		0.752		0.064		0.158

		mRS		3-6		90 d			
/	5	3	2	175	59				
		116							
1.5								n=116	n=59
		SPSS 22.0						54.93±6.24	55.86±6.11
		-		t		/		51/65	23/36
n %		2		Logistic		/		59/57	26/33
ACI				ROC		/		53/63	32/27
FT3 FT4 RC				ACI		/		32/84	43/16
		P<0.05				TIA /		25/91	50/9
						/		18/98	10/49
2						/		11/105	6/53
						/		15/101	8/7
2.1	ACI			FT3 FT4 RC		/		9/107	1
	ACI			FT3 FT4		/		6/110	59±0
RC						HDL C mmol/L		1.17±0.36	29±0/69
						LDL C mmol/L		3.09±0.69	4
						TC mmol/L		4.59±0.39	
	2					TG mmol/L		1.62±0.53	
						NIHSS		9.81±0.86	
						hs CRP mg/L		11.02±2.31	
						D D mg/L		3.15 1.85±0.24	
						/		41/75	
						cm		3 ±2/3±20/26	z z " z z z z " z) z " ! %
								29/87	h (" ! ! % z z " ! !
								# # #	z "
								#% ! z " z "#!	
2.2									
		NIHSS		hs CRP D D					
		LDL C							
		P<0.05		3					
2.3						FT3 FT4 RC			
						FT3			
FT4				RC					
		P<0.05		4					
2.4		Logistic		ACI					
		ACI				=0 =1			
				NIHSS					
FT3 FT4 RC						Logistic			
				NIHSS		hs CRP			
		D D		FT3		FT4			
						RC			
						ACI			
				P<0.05		5			

2.5 FT3 FT4 RC ACI

14 15

FT3 FT4 RC ACI
 AUC 0.951
 P<0.05 6 1

ACI RC RC
 RC RC

RC

ACI

ACI

FT3 FT4

RC ACI

FT3 FT4 RC ACI

FT3

FT4 RC

ACI

FT3 FT4 RC ACI

AUC

0.869 0.832

0.842

ACI

ROC

ACI

AUC 0.951

ACI

FT3 FT4 RC

ACI

3

FT3 FT4 RC

ACI

ACI

ACI

⁸ FT3 FT4

⁹ ¹⁰

ACI

1

CD4~+CD25~+Treg TLR4

FT3 FT4

FT3 FT4

hCMV IgM

J .

2024 52 1 123

127.

ACI

ACI

2

FT3 FT4

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FT3 FT4

ACI

3

FT3 FT4

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FT3 FT4

ACI

4

FT3 FT4

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ACI

¹¹

¹² RC

5

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¹³ RC

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17

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h

7

ProGRP CA 242 PCT Hp

		2020 6		2022 6		Hp	
86		Hp A		Hp 26 B		Hp 60 B	
B		Hp 45		Hp 15		A B	
ProGRP CA 242 PCT		ProGRP CA 242 PCT		ProGRP CA 242 PCT		Hp	
ProGRP CA 242 PCT		ProGRP CA 242 PCT		ProGRP CA 242 PCT		Hp	
ProGRP	CA 242	PCT	A	t=7.927	8.288	5.419	P<0.05
ProGRP	CA 242	PCT		t=11.092	6.897	3.788	P<0.05
ProGRP	CA 242	PCT	Hp	r=0.723	0.711	0.688	0.725 0.722 0.701
P<0.05	ROC		AUC 0.912	ProGRP	CA 242	PCT	0.899
0.871	0.886	P<0.05	ProGRP	CA 242	PCT	Hp	Hp
		ProGRP		CA 242		PCT Hp	

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To analyze the correlation between serum levels of ProGRP CA 242 PCT Hp infection and prognosis in patients with intestinal metaplasia of chronic atrophic gastritis. 86 patients with chronic atrophic gastritis and intestinal metaplasia admitted to the First People s Hospital of Mengcheng County from June 2020 to June 2022 were selected as the research subjects. According to whether they were infected with Hp they were divided into group A 26 cases without Hp infection and group B 60 cases with Hp infection. Based on the curative effect of Hp group B patients were divided into a group with good prognosis 45 cases with curative Hp and a group with poor prognosis 15 cases without curative Hp. The study aimed to compare the ProGRP CA 242 and PCT levels between group A and group B as well as between the poor prognosis group and the good prognosis group also to analyze the correlation between ProGRP CA 242 and PCT levels and Hp infection and prognosis. This study also aimed to evaluate the value of ProGRP CA 242 and PCT alone and in combination for detecting Hp infection in intestinal metaplasia of chronic atrophic gastritis. The levels of ProGRP CA 242 and PCT in group B were higher than those in group A and the differences were statistically significant t=7.927 8.288 5.419 P<0.05. ProGRP CA 242 and PCT levels in the poor prognosis group were higher than those in the good prognosis group t=11.092 6.897 3.788 P<

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0.05 . ProGRP CA 242 and PCT levels were positively correlated with HP infection and poor prognosis $r=0.723$ 0.711 0.688 0.725 0.722 0.701 $P<0.05$. The ROC curve showed that the AUC of combined diagnosis was 0.912 which was higher than 0.899 0.871 and 0.886 of ProGRP CA 242 and PCT alone $P<0.05$ The levels of ProGRP CA 242 and PCT are closely related to Hp infection and prognosis in patients with chronic atrophic gastritis and intestinal metaplasia. The combined detection of the three markers has high diagnostic value for Hp infection in patients with chronic atrophic gastritis and intestinal metaplasia.

Chronic atrophic gastritis Intestinal metaplasia ProGRP CA 242 PCT Hp

	Hp 26	B	Hp 60	A
	15	11	25-81	58.11±5.11
	B	35	25	25-81
helicobacter pylori Hp	58.15±5.13		Hp	B
			Hp 45	
	Hp 15			25 20
	25-81		58.17±5.18	
pro gastrin releasing peptide ProGRP	10	5	25-81	58.09±5.21
	A	B		
			P>0.05	
carbohydrate antigen 242 CA 242		3		Hp
	5		RUT	
procalcitonin PCT	3		13C	14C UBT
	HpSA		Hp	
				3
ProGRP CA 242 PCT			Hp	5 13C 14CUBT
Hp		HpSA		
		RUT		
1				
1.1	1.2			
	ProGRP CA 242 PCT			
2020 6	2022 6		5 mL	
86			8 cm 3 500 r/min 10-15	
		min		
>18	4			- 20
ProGRP CA 242 PCT		Hp		
		LA 2000		
	18			
ProGRP CA 242 PCT				
Hp				
	Hp	A	ProGRP CA 242 PCT	6

1.3

SPSS 22.0
 Spearman - t
 Hp ProGRP CA 242 PCT
 ProGRP CA 242 PCT
 Hp P<0.05

2

2.1 A B ProGRP CA 242 PCT
 B ProGRP CA 242 PCT A
 P<0.05 1

2.2 ProGRP
 CA 242 PCT
 ProGRP CA 242 PCT
 P<0.05 2

3

2.3 Hp
 Hp ProGRP
 CA 242 PCT
 ProGRP CA 242 PCT Hp
 Spearman ProGRP CA 242 ProGRP
 PCT Hp CA 242 PCT Hp
 P<0.05 3 7
 2.4 ProGRP CA 242 PCT B ProGRP CA 242 PCT
 Hp A ProGRP CA 242
 ROC AUC PCT ProGRP CA 242 PCT
 0.912 P<0.05 4 1 Hp

34 20215
AUC 0.912 ProGRP CA 242
PCT 0.899 0.871 0.886⁸

Hp ProGRP CA 242 PCT
Hp

ProGRP CA 242 PCT
Hp

ProGRP CA 242 PCT

9

ProGRP

10

CA 242

CA 242

11 12

PCT

13 ProGRP CA 242 PCT

Hp

Hp

ProGRP CA 242 PCT

CA 242¹⁴ Hp

PCT

15 16

Hp

ProGRP

Hp ProGRP

ProGRP Hp

ProGRP CA 242 PCT

ProGRP CA 242 PCT

Hp Hp

Hp

- 1 Yin J Yi J Yang C et al. Chronic atrophic gastritis and intestinal metaplasia induced by high salt and N methyl N nitro N nitrosoguanidine intake in rats J . Exp Ther Med 2021 21 4 315.
- 2 Lerch JM Pai RK Brown I et al. Interobserver agreement of estimating the extent of intestinal metaplasia in patients with chronic atrophic gastritis J . Virchows Arch 2022 480 6 1277 1281.
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2020 19 5 397 402
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J . 2023 52 6 771 774.
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J .
2023 46 6 521 525.
- 9 IL 8 PCT TNF Hp J .
- 10 2021 21 6 731 734.
hs CRP Cys C PCT
J . 2020 17 22 4.
- 11 sTREM 1 Ang 2 PCT
J . 2020 32 1 60 63.
- 12 Chitapanarux T Kongkamka S Wannasai K et al. Prevalence and factors associated with atrophic gastritis and intestinal metaplasia A multivariate hospital based statistical analysis J . Cancer Epidemiol 2023 82 102309.
- 13 D . 2023
- 14 Liu Y Ma YJ Huang CQ. Evaluation of the Gastric Microbiome in Patients with Chronic Superficial Gastritis and Intestinal Metaplasia J . Chin Med Sci J 2022 37 1 44 51.
- 15 PG /PG
J .
2021 21 12 1589 1593

NLR PLR

NLR / PLR BP / 2021 4 2022 2
 84 BP 42 +
 42 NLR PLR
 IL 6 PCT ADR + 95.24%
 80.95% P<0.05 +
 P<0.05 1 NLR PLR IL 6 PCT
 P<0.05 + ADR
 9.52% 4.76% P>0.05 BP
 NLR PLR

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To explore the efficacy of reduning combined with antibiotics on neutrophil/lymphocyte ratio (NLR), platelet/lymphocyte ratio (PLR) in children with bronchopneumonia (BP).

A retrospective analysis was conducted on the data of 84 pediatric patients with BP who were admitted to our hospital from April 2021 to February 2022. According to different treatment methods, they were divided into an antibiotic group (42 cases) treated with cefoperazone and an antibiotic + reduning group (42 cases) treated with reduning in addition to the antibiotic group. The efficacy (disappearance of clinical manifestations), NLR, PLR, serum inflammatory indicators (interleukin-6 (IL-6), procalcitonin (PCT) levels, and adverse drug reactions (ADRs) were compared between the two groups. The total effective rate was 95.24% in the antibiotic + reduning group, which was higher than the 80.95% in the antibiotic group, and the difference was statistically significant ($P < 0.05$). The disappearance time of cough, body temperature, lung rales, and lung shadows in the antibiotic + reduning group was shorter than that in the antibiotic group, and the difference was statistically significant ($P < 0.05$). After one week of treatment, the levels of NLR, PLR, serum IL-6, and PCT in both groups were lower than before treatment ($P < 0.05$), with the antibiotic + reduning group being lower than the antibiotic group, and the difference was statistically significant ($P < 0.05$). There was no difference in ADR between the antibiotic + reduning group (9.52%) and the antibiotic group (4.76%) ($P > 0.05$). The application of reduning combined with antibiotics for BP children has a positive effect. It can significantly

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reduce the levels of NLR PLR and serum inflammatory indicators effectively promoting symptom resolution.

Reducing Cefthiamidine Bronchopneumonia Neutrophils Lymphocytes Platelet

	84	84		n=42	+	n=42	
pneumonia BP	broncho						P>0.05
	1			1			
				1.2			
	2			1.2.1			
			BP				
	3			20200121		H20030648	3
BP	BP	4		5 mL	7-8	4 mL 4~6	8 mL
	5			6 h	1	4 /d	
/			neutrophil to lymphocyte ratio				2 mL 1 mg
NLR	/		platelet to lymphocyte			H20140475 1 mg	
ratio PLR	BP		6				2 mL
			BP	5 mg		H20140108 2.5 mg	
	BP						
	NLR PLR					50-100mg/kg	
1				20201113		H20103656 250 mL	2-3
1.1				+			
		2021	4	2022		0.4-0.6 mL/kg	
2		BP	103				
		BP		20200511		Z 20050217 150 mL	
2017	7	BP		5%			1 /d
2-8	1						
				1.2.2			
			19	1.2.3	NLR PLR		
	7		4			1	
2		6		5 mL	2		GRT 6001
		1		n %	-		

Table 1 Comparison of general data between the two groups n % -

	n					d					
+	42	22	52.38	20	47.62	3.60±1.18	4.37±1.21	12	28.57	21	50.00
	42	19	45.24	23	54.76	3.69±1.25	4.51±1.39	13	30.95	18	42.86
t/Z			0.429			0.339	0.492				0.130
P			0.513			0.735	0.624				0.896

NE
 NLR NE/
 10 cm 10 min
 BY B10140 hz 1780
 IL 6
 1.2.4
 1.2.5
 X
 90%
 75%~90%
 50%~74%
 50%
 = - / ×100%
 1.3
 SPSS 22.0
 - t n %
 2 P<0.05
 2
 2.1
 + P<0.05 2
 2 n %
 Table 2 Comparison of efficacy between the two groups

n	n %	
+	42 23 54.76	11 26.19 6 14.29 2 4.76 40 95.24
	42 16 38.10	13 30.95 5 11.90 8 19.05 34 80.95
P		4.086 0.043

2.2
 + P<0.05 3
 2.3
 NLR PLR
 NLR PLR P>
 0.05 1 NLR PLR

3
 - d
 Table 3 Comparison of vanishing time of various manifestations between the two groups - d

+	42	4.81±1.33	3.32±0.94	5.46±1.57	5.27±1.52
	42	5.73±1.69	4.56±1.34	6.85±1.81	6.63±1.75
		2.772	4.910	3.760	3.802
		0.007	<0.001	<0.001	<0.001

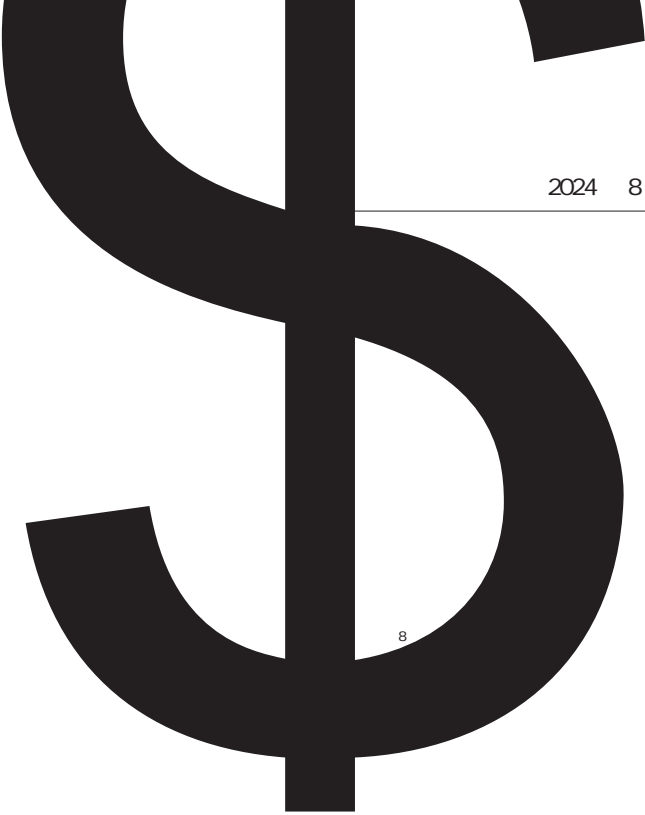
+ P<0.05 4
 4 NLR PLR -
 Table 4 Comparison of NLR and PLR between the two groups -

n	NLR		PLR		
	1	1	1	1	
+	42	1.62±0.47	1.17±0.33 ^a	123.41±34.62	98.17±23.75 ^a
	42	1.70±0.53	1.39±0.41 ^a	125.07±36.78	109.95±26.36 ^a
t		0.732	2.709	0.213	2.152
P		0.466	0.008	0.832	0.034

^aP<0.05
 2.4
 IL 6 PCT
 P>0.05 1 3
 + P<0.05 5
 5 -
 Table 5 Comparison of serum inflammation indexes between the two groups -

n	IL 6 pg/mL		PCT μg/L		
	1	1	1	1	
+	42	146.49±16.27	23.47±6.28 ^a	3.74±0.61	0.61±0.18 ^a
	42	148.31±18.56	35.14±8.49 ^a	3.87±0.74	0.98±0.32 ^a
t		0.478	7.162	0.879	6.531
P		0.634	<0.001	0.382	<0.001

^aP<0.05
 2.5
 ADR
 + ADR
 P>0.05 6
 3



8

+

9

+

10

ADR 9.52% 4.76%

IL 6 PCT
BP

+

1

11

IL

4 /

1PCT

NT proBNP Ang NE

1 2 1

NT proBNP ICM HF N

2020 10 2023 2 86 ICM HF

43

2 2 2 NT proBNP Ang NE

2 2 74.42% 93.02%

$\chi^2=5.460$ $P<0.05$ 2 NT proBNP Ang NE TNF IL 6

IL 8 LVTWP PWS IVSS $t=5.672$ 20016 62% $\delta=0.0001205-0.672$

AHWJ2021b103

1.

246501

2

246052

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4BW 4B • 7T 1 XhÅ + 4F ' 4F ' 4B% 4J% 4F øIE

cross factor TNF 8 Interleukin 8

L 8

1.5

SPSS 26.0

n %

2

t

P<0.05

2

2.1

2

P<0.05

1

1

n %

Table 1 Comparison of the efficacy of the two groups

		n %			
n					
	43	22	51.16	18	41.86
	43	17	39.53	15	34.88
				3	6.98
				40	93.02
				11	25.58
				32	74.42
P					5.460
					0.019

2.2

2

NT proBNP Ang NE

P<0.05

2

2

ng/L

Table 2 Comparison of neuroendocrine molecular levels between the two groups before and after treatment

	n	NT proBNP	Ang	NE
	43	5.01	105.44±17.26	306.22±32.41
	43	4.99	105.96±16.87	305.25±33.33
t		0.095	0.141	0.137
P		0.925	0.888	0.892
2				
	43	2.13	41.43±5.13 ^a	101.26±6.23 ^a
	43	2.79	69.04±7.45 ^a	187.55±8.09 ^a
t		5.672	20.016	55.416
P		<0.001	<0.001	<0.001

^aP<0.05

2.3

2

LVTWP PWS IVSS

P<0.05

3

2.4

2

P<0.05

4

3 - mm

Table 3 Comparison of indices of ventricular remodeling before and after treatment in the two groups - mm

	n	LVTWP	PWS	IVSS
	43	13.56±4.14	14.56±2.06	13.56±1.12
	43	13.42±4.24	13.93±2.09	13.71±1.09
t		0.155	1.408	0.629
P		0.877	0.163	0.531
2				
	43	8.41±1.22 ^a	9.21±1.09 ^a	10.12±1.06 ^a
	43	10.35±1.76 ^a	11.05±1.44 ^a	11.95±1.75 ^a
t		5.940	6.681	5.865
P		<0.001	<0.001	<0.001

^aP<0.05

4 - ng/L

Table 4 Comparison of inflammatory factor levels before and after treatment in the two groups - ng/L

	n	IL 6	IL 8	TNF
	43	16.98±3.10	12.24±2.12	62.44±5.12
	43	17.05±3.09	12.31±2.03	61.98±5.54
t		0.105	0.156	0.400
P		0.917	0.876	0.690
2				
	43	9.01±1.03 ^a	7.10±1.06 ^a	42.69±3.65 ^a
	43	11.21±2.24 ^a	9.27±2.49 ^a	48.28±4.03 ^a
t		5.851	5.258	6.742
P		<0.001	<0.001	<0.001

^aP<0.05

3

ICM HF

8

9

HF

1

50%

10

11

12

ICM HF

13

ICM

HF

ICM HF
¹⁴ ICM HF
NT proBNP Ang NE
NT proBNP Ang NE

with the levels in the experimental group being lower than those in the control group and the differences were statistically significant ($P < 0.05$). After treatment the cycle cancellation rate and abortion rate in the experimental group were lower than those in the control group and the clinical pregnancy rate and live birth rate were higher than those in the control group but the differences were not statistically significant ($P > 0.05$). There was no significant difference in the incidence of adverse reactions between the experimental group and the control group ($P > 0.05$). Compared to the GnRH α regimen alone the GnRH α + rhGH adjuvant regimen can significantly regulate the levels of FSH, LH, and E_2 in women with advanced infertility. It also improves the number of eggs obtained, the rate of eugenic embryos, and the rate of clinical pregnancy.

Recombinant human growth hormone

1.3.2

3 d
5 mL 3 500 r/min
10 min 8 cm
FSH E2 LH

1.3.3



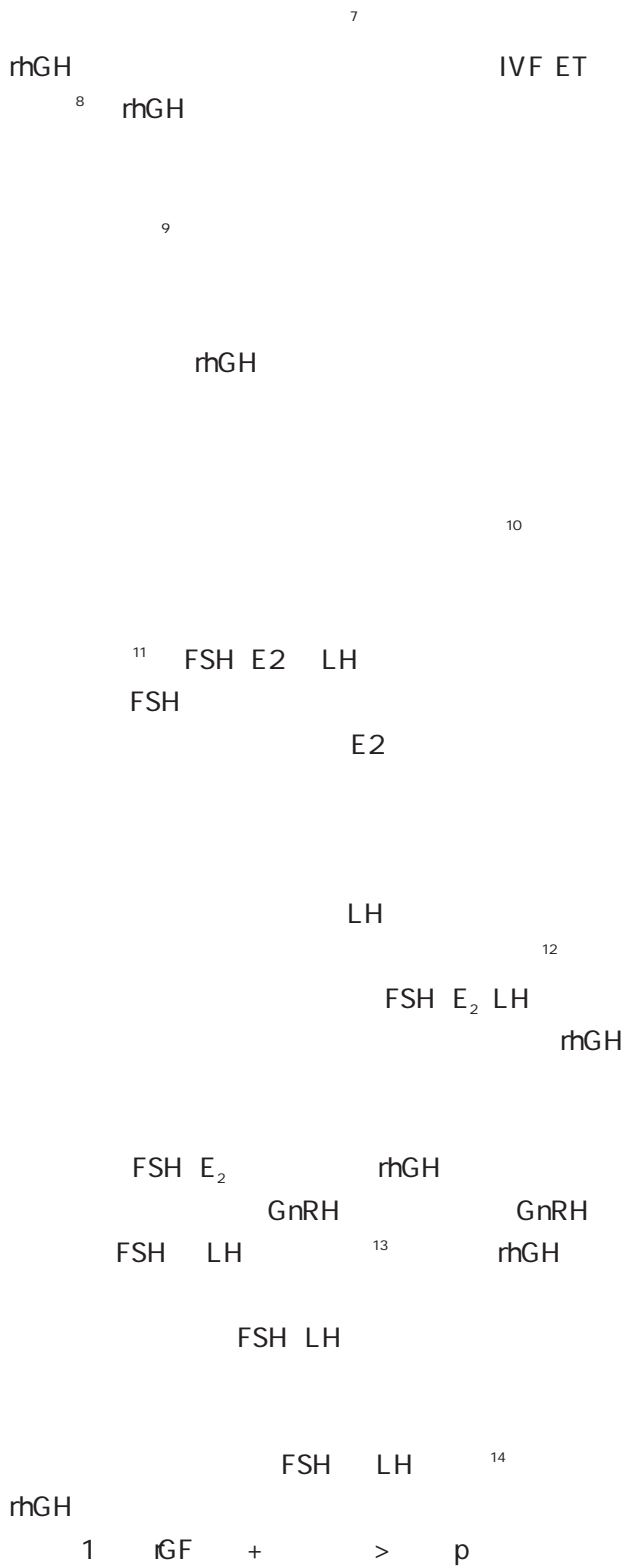
1.3.4

û• p

1.4

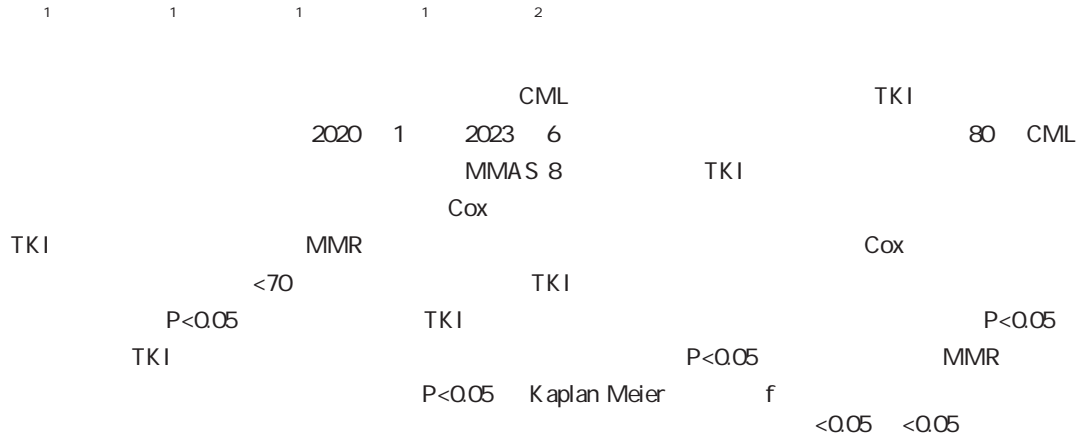
SPSS 22.0

y





TKI



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the low adherence group with a statistically significant difference $P < 0.05$. The MMR response rate in the high adherence group was significantly higher than that in the low adherence group also showing a statistically significant difference $P < 0.05$. Kaplan Meier survival analysis revealed that the progression free survival in the high adherence group 62.35 ± 12.39 was significantly longer than that in the low adherence group 39.04 ± 10.07 with a statistically significant difference $P < 0.05$. Furthermore the quality of life scores in the high adherence group were significantly higher than those in the low adherence group with a statistically significant difference $P < 0.05$ CML patients with high adherence to TKI therapy exhibit significantly better clinical outcomes. Therefore clinical interventions should be strengthened to improve patient adherence to TKI therapy thereby enhancing prognosis.

. Chronic myelogenous leukemia Tyrosine kinase inhibitors Efficacy Evaluation Survival Rate

chronic myeloid leukemia CML
 lar response MMR 2
 15%~20%
 breakpoint cluster region abelson fusion gene BCR
 ABL 1 tyrosine kinase 80 CML 42 38
 inhibitors TKIs CML 40.43±12.06 42 12 69
 TKI TKI
 TKI CML 1.2.2 Chi
 TKI TKI TKI nese version of the Morisky Medication Adherence
 Scale MMAS 8 6 0-8
 CML TKI 45 <6 6 CML
 TKI TKI 80 100%
 1 1.2.3 36 36 item short form
 1.1 health survey SF 36 7 CML
 2020 1 2023 6 8
 CML 80 0-100
 CML 80 100%
 TKI 1.3 SPSS 22.0
 5 3 t n
 % 2 Kaplan Meier
 CML progression free survival PFS
 overall survival OS Log rank
 major molecule Cox CML

P<0.05

2

2.1

1

1 - n %

Table 1 Comparison of general characteristics between the two groups - n %

	n		n %
	25	38.20 ± 11.58	12 48.0
	55	41.56 ± 12.27	30 54.5
t	1.181	0.223	8.942
P	0.242	0.643	0.003

2.2 CML

Cox

<70

TKI

P<0.05

2

2.3 TKI

TKI

P<0.05

3

3

TKI

Table 3 Comparison of TKI switch rates between the two groups

	n	TKI
	25	4 2 7.50
	55	23 21 55.00
P		5.143 0.023

2.4 MMR

MMR

P<0.05

4

2.5

Kaplan Meier

2 CML

Table 2 Multivariate regression analysis of factors associated with adherence in CML patients

	Wald	OR	95% CI	P
		=1 =0	0.421	0.201
		<70 =1 70 =0	0.782	0.249
		=1 =0	0.937	0.231
TKI		=1 =0	1.224	0.412
			4.392	1.523 1.028-2.255
			9.867	0.457 0.274-0.761
			16.451	2.553 1.614-4.038
			8.855	3.402 1.494-7.750

4 MMR

Table 4 Comparison of MMR response rates between the two groups

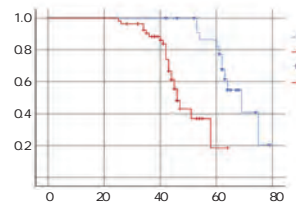
	n	MMR	MMR
	25	22	88.00
	55	30	54.55
		/	7.682
P		/	0.006

62.35±12.39

39.04±10.07

P<0.05

1



1 Kaplan Meier

Figure 1 Kaplan Meier progression free survival analysis curve

2.6

P<0.05

5

5

Table 5 Comparison of quality of life scores between the two groups

	n	SF 36
	25	85.4±8.6
	55	72.3±10.2
t		6.163
P		<0.001

3

PFS TKI

89

CML

TKI

MMR

TKI 10 1 BCR ABLT3151 J . 2023 31 5 1579 1583

11 2

12 J . 2023 31

MMR 88.00% 3 649 653.

72.73%¹³ Hughes 3

14 CML 13 112 115. 4 J . 2023 63

15 5 Lee H Seo J Shin S et al. Development and validation of sensitive BCR ABL1 fusion gene quantitation using next generation sequencing J . Cancer Cell Int 2023 23 1 106.

Jabbour CML 6 J . GMAS MMAS 8 SEAMS 2023

PFS 7 37 13 2322 2328 J .

TKI 8 2019 32 11 864 648.

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TKI TKI TFR 9 Cheng F Cui Z Li Q et al. Adherence to tyrosine kinase inhibitor and clinical outcomes in patients with chronic myeloid leukemia J . Int Immunopharmacol 2023 124 Pt A 110847.

17 <70 10 Inzoli E Aroldi A Piazza R et al. Tyrosine Kinase Inhibitor discontinuation in Chronic Myeloid Leukemia eligibility criteria and predictors of success J . Am J Hematol 2022

18 CML <70 1075 1085. \$ \$ \$ \$ " \$ \$ \$ " \$ \$ \$ " \$ \$ \$ \$ e° U•2022

19 8 ; # " : y A # \$ "#\$2" # \$ ž # °

20 MMR

AAPR

1 1 2 1 1

ALB ALP AAPR

2021 11 2023 12

126 n=35 n=91

ROC ALB ALP AAPR

ALB ALP AAPR

logistic

ALB ALP AAPR

ALB ALP AAPR

P<0.05

ALB ALP

P<0.05

ALB ALP

AUC 0.746 0.827 AAPR AUC 0.941

C CRP

CRP ALB ALP AAPR

P<0.05

P<0.05

Logistic

AAPR

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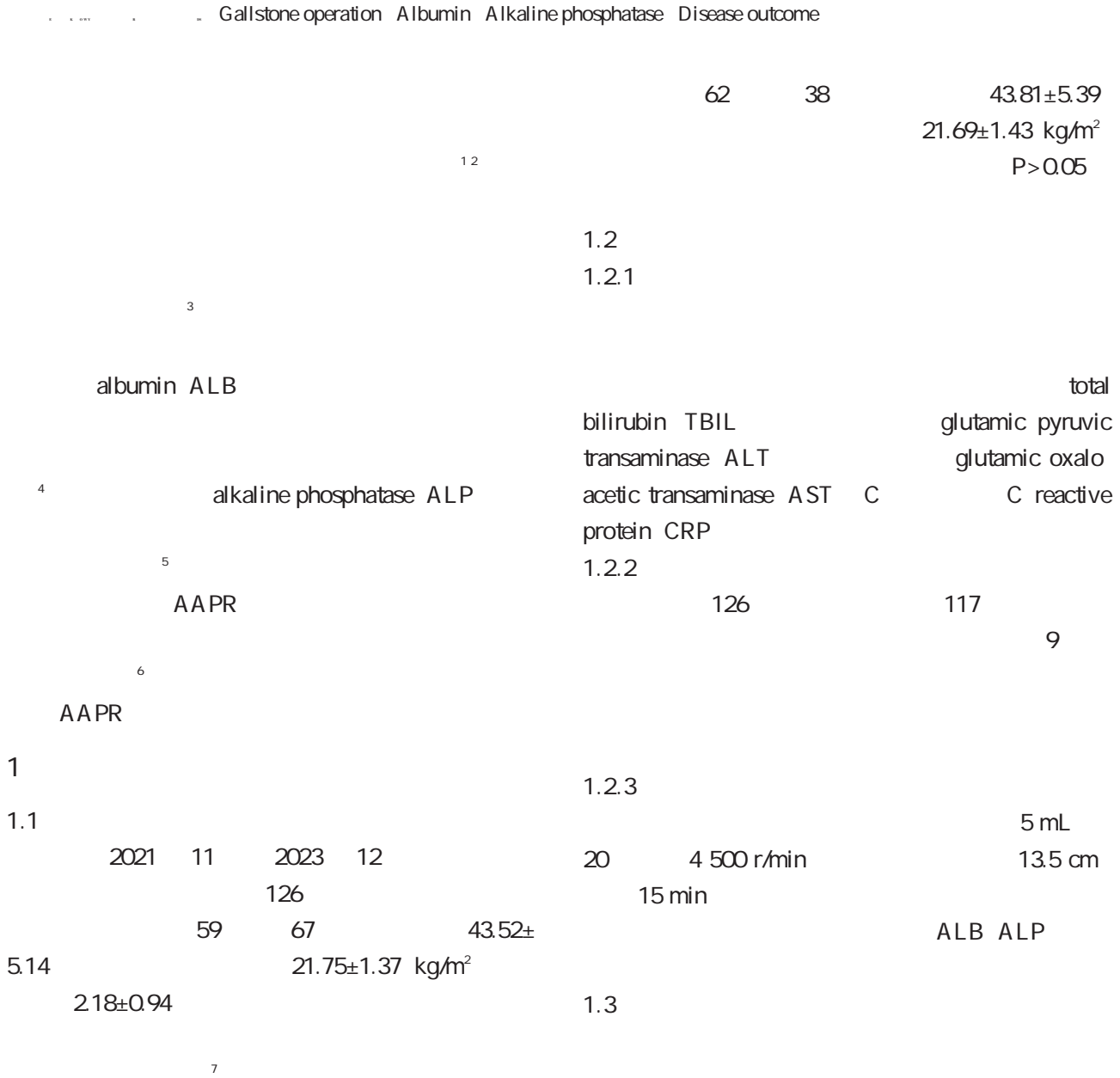
To investigate the change of serum albumin (ALB) to alkaline phosphatase (ALP) ratio (AAPR) in patients with gallstone surgery and its significance in evaluating the outcome of the disease. ... 126 patients who underwent gallstone surgery at Dezhou Hospital of Qilu Hospital Shandong University from November 2021 to December 2023 were selected as the study group. Based on the effectiveness of the surgery the study group was divided into a good prognosis group (n=91) and a poor prognosis group (n=35). Additionally 100 healthy individuals from our hospital during the same period were selected as the control group. The levels of ALB and ALP in all patients were measured and the AAPR was calculated. The receiver operating characteristic (ROC) curve was used to analyze the evaluation value of ALB ALP levels and AAPR on the disease prognosis of patients who had undergone gallstone surgery. Univariate analysis was conducted on the general data of patients with different prognoses. Statistically significant indicators were then included in the multivariate logistic regression equation to analyze the influencing factors of disease prognosis in patients who had undergone gallstone surgery. ... ALB and AAPR in the study group

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were lower compared to the control group and ALP was higher $P<0.05$. ALB and AAPR in the good prognosis group were higher than those in the poor prognosis group and ALP was lower $P<0.05$. The AUC assessed by ALB and ALP were 0.746 and 0.827 respectively and the AUC assessed by AAPR was 0.941. The proportion of laparoscopic surgery in the group with a good prognosis is significantly higher than that in the group with a poor prognosis and the C reactive protein CRP level was lower than that in the poor prognosis group $P<0.05$. CRP ALB ALP and AAPR were the influencing factors of disease outcome in patients with gallstone surgery $P<0.05$ Low expression of AAPR in patients who have undergone gallstone surgery is associated with a poor disease outcome. This suggests that AAPR could serve as a potential marker for evaluating the disease outcome in these patients.



1.4

IBM SPSS 25.0
 -
 n %
 ROC ALB ALP
 AAPR
 logistic
 P<0.05

3 ALB ALP AAPR

Table 3 Evaluation value of ALB ALP and AAPR on disease outcomes in patients undergoing gallstone surgery

	AUC	95% CI		%	%
ALB	0.746	0.701-0.796	31.92 g/L	53.27	93.62
ALP	0.827	0.782-0.877	106.16 U/L	65.36	93.62
AAPR	0.941	0.896-0.991	0.28	87.39	89.57

2

2.1 ALB ALP AAPR
 ALB AAPR
 P<0.05
 Table
 ALP
 1

4

n %

Table 2

	n=91	n=35	t/	P
	41 45.05	18 51.43	0.412	0.521
	50 54.95	17 48.57	1.013	0.313
	43.91±3.81	43.18±3.07	1.813	0.072
	2.06±0.64	2.27±0.39	1.554	0.123
kg/m ²	21.36±0.72	21.14±0.69	0.944	0.331
	39 49.37	14 60.87	1.853	0.173
	32 40.51	13 56.52	1.853	0.173
	21 23.08	11 31.43	0.931	0.335
	14 15.38	7 20.00	0.388	0.534
	17 18.68	9 25.71	0.763	0.382
	3 3.30	6 17.14	7.307	0.007
	88 96.70	29 82.86		
TBIL μmol/L	10.81±2.42	11.39±2.56	0.290	0.733
ALT U/L	35.73±0.36	1.84±0.32	1.443	0.152
AST U/L	38.94±3.19	37.14±3.47	1.567	0.120
CRP mg/L	7.74±1.26	8.39±1.51	3.583	<0.001

Table 2 Comparison of ALB ALP and AAPR between the two groups

	n	ALB g/L	ALP U/L	AAPR
	100	43.19±3.61	72.16±12.48	0.60±0.12
	126	33.92±6.12	106.16 ±15.43	0.32±0.09
t		13.405	17.876	20.040
P		<0.001	<0.001	<0.001

2.2

ALB ALP AAPR
 ALB AAPR
 ALP
 P<0.05
 2

Table 2 Comparison of ALB ALP and AAPR among patients with different disease outcomes

	n	ALB g/L	ALP U/L	AAPR
	91	36.81±5.67	97.51±13.04	0.36±0.08
	35	26.41±6.35	128.65±12.37	0.22±0.06
t		6.884	3.868	7.408
P		<0.001	<0.001	<0.001

2.3 ALB ALP AAPR

ALB ALP
 AUC 0.746 0.827 AAPR
 AUC
 0.941 3

2.4

CRP
 P<0.05 4

2.5

Logistic

=0 =1 P<0.01
 ALB ALP AAPR Logistic

CRP ALB ALP AAPR

P<0.05 5

3

				SE	Wald	P	OR	95% CI	OR	95% CI
CRP	<8.07 mg/L=0	8.07 mg/L=1	0.667	0.234	8.125	0.004	9.034	3.000	27.000	0.005
ALB	>31.92 g/L=0	31.92 g/L=1	0.873	0.229	14.533	<0.001				
ALP	<106.16 U/L=0	106.16 U/L=1	0.731	0.207	12.471	<0.001				
AAPR	>0.28=0	0.28=1	1.064	0.251	17.969	<0.001				

significant $t=3.197$ 10.144 and 2.415 $P<0.05$. The total fever cough wheezing lung rales and hospitalization time in the study group were lower than those in the control group and the differences were statistically significant $t=10.086$ 6.588 6.739 12.138 10.260 $P<0.05$. After one week of treatment the total effective rate of the study group was 94.83% which was higher than that of the control group 79.31% with statistical significance $t=6.202$ $P<0.05$. After one week of treatment TNF IL 10 CRP and PCT in both groups decreased compared with those before treatment. Compared with the control group the study group was lower and the difference was statistically significant $t=3.309$ 4.780 4.524 18.385 $P<0.05$. After one week of treatment CD4+ and CD4+/CD8+ in the two groups were higher than those before treatment and higher than those in the control group with statistical significance $t=2.903$ 2.415 $P<0.05$. CD8+ decreased compared with before treatment and the observation group was lower than the control group with statistical significance $t=2.125$ $P<0.05$ In the treatment of children s bronchopneumonia the addition and subtraction of Keli Dazao Xiefei Decoction can lead to a more significant curative effect.

. Tinglidazaoxiefei decoction Children Bronchopneumonia Immunologic function

1

2

3

1

1.1

2024 6 16 8 J Mol Diagn Ther, August 2024, Vol. 16 No. 8
y a . o a . o t & P € a A •
†

X

1.3
 1.3.1
 2 1
 1 TNF
 10 IL 10 C CRP
 PCT 2 T CD₄⁺ CD₈⁺
 CD₄⁺/CD₈⁺ 3 FVC
 1 FEV1 PEF
 1.3.2
 1 6
 2 7 1
 37
 X
 WBC
 X 50%
 3
 1.4
 SPSS 25.0
 n % 2
 P<0.05
 2
 2.1
 2 1 FVC FEV1 PEF
 0.05 2
 2.2
 P<0.05 3
 2 2

3 2 - d
 Table 3 Comparison of recovery time of symptoms and signs between two groups of children - d

	n	1	2	3	4	5
n	58	1.91±0.38	3.62±0.55	3.34±0.41	5.31±0.47	7.49±0.23
t	58	2.69±0.45	4.35±0.64	4.02±0.65	6.55±0.62	8.10±0.39
P		10.086	6.588	6.739	12.138	10.260
		<0.001	<0.001	<0.001	<0.001	<0.001

2.3
 1
 P<0.05 4
 4 2 n %

Table 4 2 Comparison of clinical efficacy between two groups of children n %

	n	1	2	3	4	5
n	58	47 81.03	8 13.79	3 5.17	55 94.83	
	58	36 62.07	10 17.24	12 20.69	46 79.31	
P					6.202	0.013

2.4
 2
 2.5
 1 TNF IL 10 CRP PCT
 P<0.05 5
 2.6
 2 1 CD₄⁺ CD₄⁺/CD₈⁺
 CD₈⁺ P<0.05 6
 3
 P<

Table 2 Comparison of pulmonary function between two groups of children -

	n	FVC L		FEV1 L		PEF L/min	
		1	2	1	2	1	2
	58	1.55±0.24	3.81±0.45 ^a	1.22±0.14	3.08±0.29 ^a	162.55±19.51	184.62±22.28 ^a
	58	1.57±0.26	3.56±0.39 ^a	1.23±0.16	2.57±0.25 ^a	161.49±22.04	175.06±20.31 ^a
t		0.430	3.197	0.358	10.144	0.274	2.415
P		0.668	0.002	0.721	<0.001	0.784	0.017

^aP<0.05

5 -
Table 5 Comparison of the changes of serum inflammatory factors between the two groups of children -

n	TNF ng/L		IL 10 ng/L		CRP mg/L		PCT µg/L	
	1	1	1	1	1	1	1	1
58	61.86±11.25	39.01±7.08 ^a	23.58±3.26	10.41±1.12 ^a	68.89±9.42	22.13±2.08 ^a	3.88±0.52	1.05±0.11 ^a
58	60.99±11.34	43.55±7.59 ^a	23.71±3.05	11.58±1.49 ^a	69.02±9.15	23.95±2.25 ^a	3.91±0.48	1.58±0.19 ^a
t	0.415	3.309	0.222	4.780	0.075	4.524	0.323	18.385
P	0.679	0.001	0.825	<0.001	0.940	<0.001	0.747	<0.001

^aP<0.05

6 2 T -
Table 6 Comparison of changes of T lymphocyte subsets in peripheral blood between two groups of children -

n	CD ₄ ⁺ %		CD ₈ ⁺ %		CD ₄ ⁺ /CD ₈ ⁺	
	1	1	1	1	1	1
58	38.81±4.67	46.79±5.75 ^a	31.41±3.85	28.22±2.69 ^a	1.24±0.23	1.66±0.39 ^a
58	38.74±4.52	43.83±5.22 ^a	31.35±3.72	29.44±2.75 ^a	1.25±0.25	1.51±0.37 ^a
t	0.082	2.903	0.085	2.415	0.224	2.125
P	0.935	0.004	0.932	0.017	0.823	0.036

^aP<0.05

8

13 14

9

15

10

1 FVC FEV1 PEF

11 12

2

1

T

TNF IL 10 CRP PCT
CD₄⁺ CD₄⁺/CD₈⁺ CD₈⁺

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Cor E NA

NA 2020 12 2021 12
 76 38 38

T₀ T₁ 1 min T₂ 3 min T₃ MAP HR

SpO₂ Cor E NA P<0.05 P>

0.05 T₀~T₃ HR MAP T₂ HR MAP
 P<0.05 T₀ T₃ Cor E NA

P<0.05

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To explore the effect of self-shaping catheter intubation on the levels of cortisol (Cor), epinephrine (E), and norepinephrine (NA) in infants with premature closure of cranial suture. According to the random number table, 76 cases of children undergoing craniostomy under general anesthesia at the Children's Hospital Affiliated to Nanjing Medical University from December 2020 to December 2021 were divided into the control group (38 cases) and the observation group (38 cases). The control group was intubated with intubation forceps, while the observation group was intubated with self-shaping catheter. The intubation related indexes (laryngoscope exposure classification) and the changes of mean arterial pressure (MAP), heart rate (HR), blood oxygen saturation (SpO₂) m after induction (T₀), glottis exposure (T₁, 1 min after intubation (T₂), and 3 min after intubation (T₃) were recorded in both groups. Changes in serum Cor, E, and NA in the two groups were also measured. The intubation time in the observation group was shorter than that in the control group, and the success rate of the first intubation was higher than that in the control group with statistical significance (P<0.05). There was no significant difference between the two groups (P>0.05). The HR and MAP of the children in T₀~T₃ groups increased and then decreased. At T₂, the HR and MAP in the observation group were lower than those in the control group with statistical significance (P<0.05). Compared to T₀, the levels of serum Cor, E, and NA in both groups increased by T₃, but the levels in the

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observation group were lower than those in the control group with statistical significance $P < 0.05$.

Compared to the intubation assisted by intubation forceps using a self-shaping catheter for nasotracheal intubation in infants with premature closure of cranial sutures can shorten the intubation time, increase the success rate of the first intubation, and result in relatively stable vital signs for the children. Additionally, this method can help lower the stress response in these infants.

Self-shaping catheter Nasopharyngeal intubation Craniostenosis Vital signs

12

3

Cortisol Cor
Norepinephrine

Epinephrine E
NA

1

1.1

12

2020 12 2021

76

38

38 4-10

7.08±0.85 American so

ciety of anesthesiologists ASA 4 18

20 3-9 kg 5.97±1.02 kg

8 30

4-11 7.11±0.87 ASA

16 22 3-10 kg 5.93±1.05

kg 9 29

ASA

P>0.05

4 5

/ ×100%

1.3.2

Cormark Lehane

8

1.3.3

1 min T₂ T₀ 3 min T₃ T₁
 Mean arterial pressure MAP
 60-100 mmHg Heart rate HR
 110-130 /min Percutaneous ar
 terial oxygen saturation SpO₂ 92%~
 99%

9

1.3.4

Cor E NA P>0.05 3
 T₀ T₃ 2 mL
 3 000 r/min
 8 r 15 min
 cobas E 602 Cor 100-
 400 ng/L E <480 ng/L NA
 200-500 ng/L

9

1.4

SPSS 26.0
 n %
 t t
 P<0.05

2

2.1

P<0.05
 P>0.05 1 2.4 Cor E NA

2.2

P>0.05 2

2.3

T₀~T₃ MAP HR SpO₂
 T2 HR MAP
 P<0.05 SpO₂

4 Cor E NA ng/L
 Table 4 comparison of serum Cor e and NA levels between the two groups ng/L

	n	Cor		E		NA	
		T ₀	T ₃	T ₀	T ₃	T ₀	T ₃
	38	157.53±26.17	187.36±28.36 ^a	238.57±23.52	302.45±41.76 ^a	208.16±25.65	363.46±37.72 ^a
	38	158.62±26.54	172.21±30.74 ^a	239.61±23.58	278.73±32.14 ^a	211.82±26.53	311.13±32.14 ^a
t		0.180	2.233	0.192	2.775	0.611	6.510
P		0.857	0.029	0.848	0.007	0.543	<0.001

T₀ ^aP<0.05

NSE IL 1 5 HT

1 2 3

IL 1 5 5 HT

NSE 2021 8 2023 8

n=39 n=30 n=21

NSE IL 1 5 HT MoCA

5 HT MoCA Pearson NSE IL 1

5 HT MoCA ROC NSE IL 1 5 HT

NSE IL 1 > >

F=76.815 34.153 P<0.05 5 HT < < F=130.092

P<0.05 NSE IL 1 t=9.397

5.274 P<0.05 5 HT t=9.703 P<

0.05 Pearson NSE IL 1 MoCA

P<0.05 5 HT MoCA P<0.05 ROC NSE IL 1

5 HT ACU 0.966 P<0.05

NSE IL 1 5 HT

NSE IL 1 5 HT

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To explore the changes of serum neuron specific enolase NSE interleukin 1

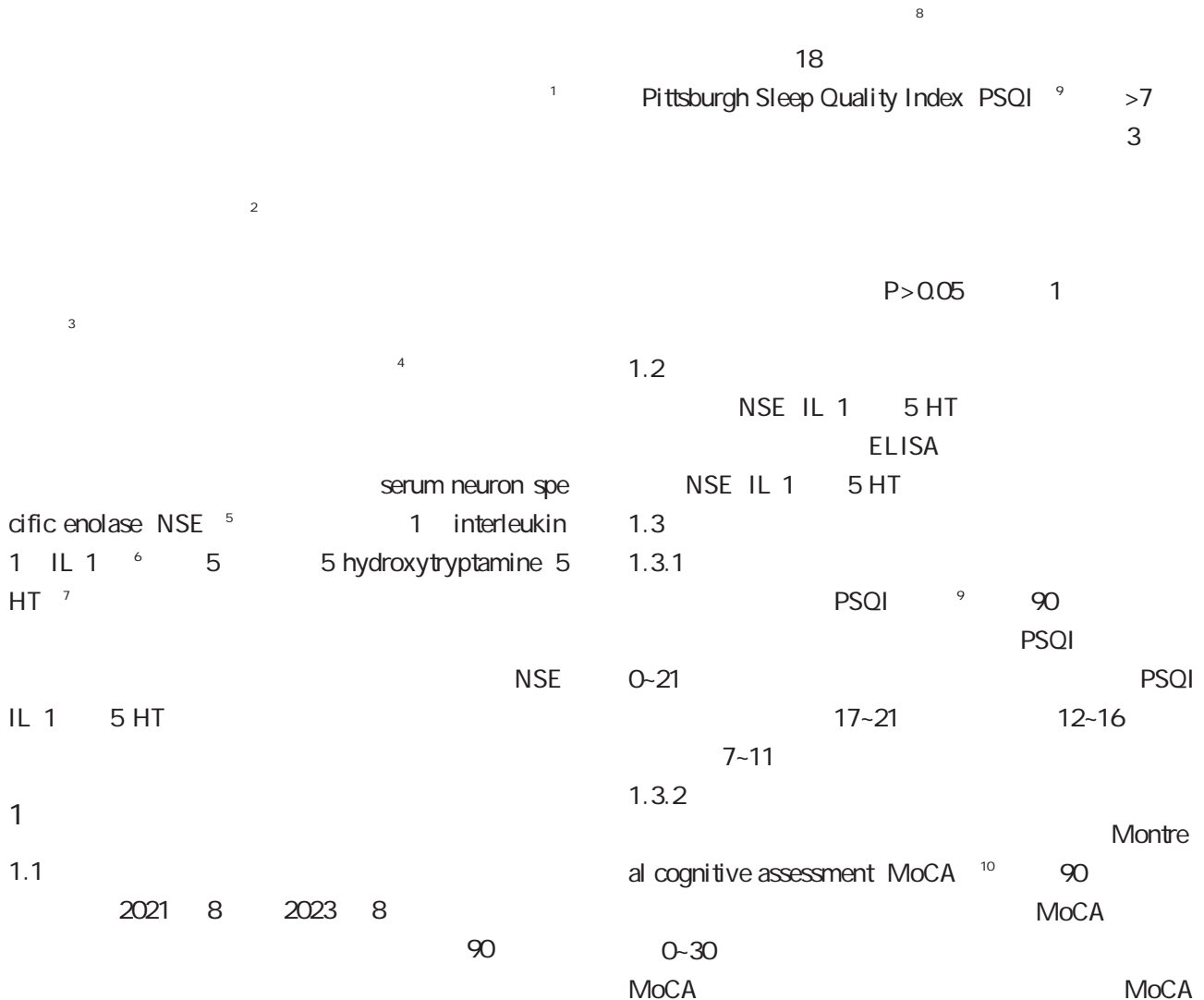
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MoCA cognitive function of patients with chronic insomnia disorder at admission the patients were further classified into two subgroups insomnia with cognitive dysfunction n=41 and simple insomnia n=49 . The relationship between serum levels of NSE IL 1 and 5 HT at admission and MoCA score was then compared between these two subgroups. Pearson correlation coefficient was used to analyze the correlation between the levels of serum NSE IL 1 and 5 HT at admission and MoCA score in patients with chronic insomnia disorder. A receiver operating characteristic ROC curve was applied to analyze the diagnostic efficiency of serum NSE IL 1 and 5 HT levels at admission for patients with insomnia and cognitive dysfunction. . . . The levels of NSE and IL 1 showed severe group > moderate group > mild group F=76.815 34.153 P<0.05 while the 5 HT level was manifested as severe group < moderate group < mild group F=130.092 P<0.05 . The NSE and IL 1 levels in insomnia with cognitive dysfunction group were significantly higher than those in simple insomnia group t=9.397 5.274 P<0.05 while the 5 HT was significantly lower than that in the simple insomnia group t=9.703 P<0.05 . Pearson correlation coefficient analysis showed that serum NSE and IL 1 levels at admission were significantly negatively correlated with MoCA score in insomnia with cognitive dysfunction group P<0.05 and 5 HT level was significantly positively correlated with MoCA score P<0.05 . ROC analysis the ACU of combined detection with serum NSE IL 1 and 5 HT was 0.966 which was better than that of single detection P<0.05 The cognitive function and sleep of patients with chronic insomnia disorder are related to increase in NSE and IL 1 and decrease in 5 HT.

. Chronic insomnia disorder Serum NSE IL 1 5 HT Cognitive function



1 n % -
Table 1 Comparison of general clinical data among the three groups n % -

	n							
	39	31 79.5	8 20.5	27.12±5.21	3.47±0.52	12.91±1.45	23 59.0	
	30	25 83.3	5 16.7	25.98±5.37	3.45±0.53	13.05±1.59	18 60.0	
	21	18 85.7	3 14.3	26.33±5.26	3.40±0.57	13.10±1.32	10 47.6	
F/			0.400	0.421	0.117	0.139	0.920	
P			0.819	0.658	0.889	0.870	0.631	

15 q LT MoCA >15
90
41
49
1.4
SPSS 21.0
t
n %
2 NL. Pearson
NSE IL 1 5 HT MoCA
P<0.05
2 . NSE

2 NSE IL 1 5 HT -
Table 2 Comparison of serum levels of NSE IL 1 and 5 HT among the three groups -

	n	NSE ng/mL	IL 1 pg/mL	5 HT ng/mL
	39	23.01±3.17 ^{ab}	79.15±7.45 ^{ab}	20.58±1.47 ^{ab}
	30	18.71±2.19 ^a	72.30±8.15 ^a	23.97±1.40 ^a
	21	14.24±2.13	62.58±6.19	26.33±1.11
F		76.815	34.153	130.092
P		<0.001	<0.001	<0.001

4 NSE IL 1 5 HT MoCA -
Table 4 Comparison of serum levels of NSE IL 1 and 5 HT and MoCA score -

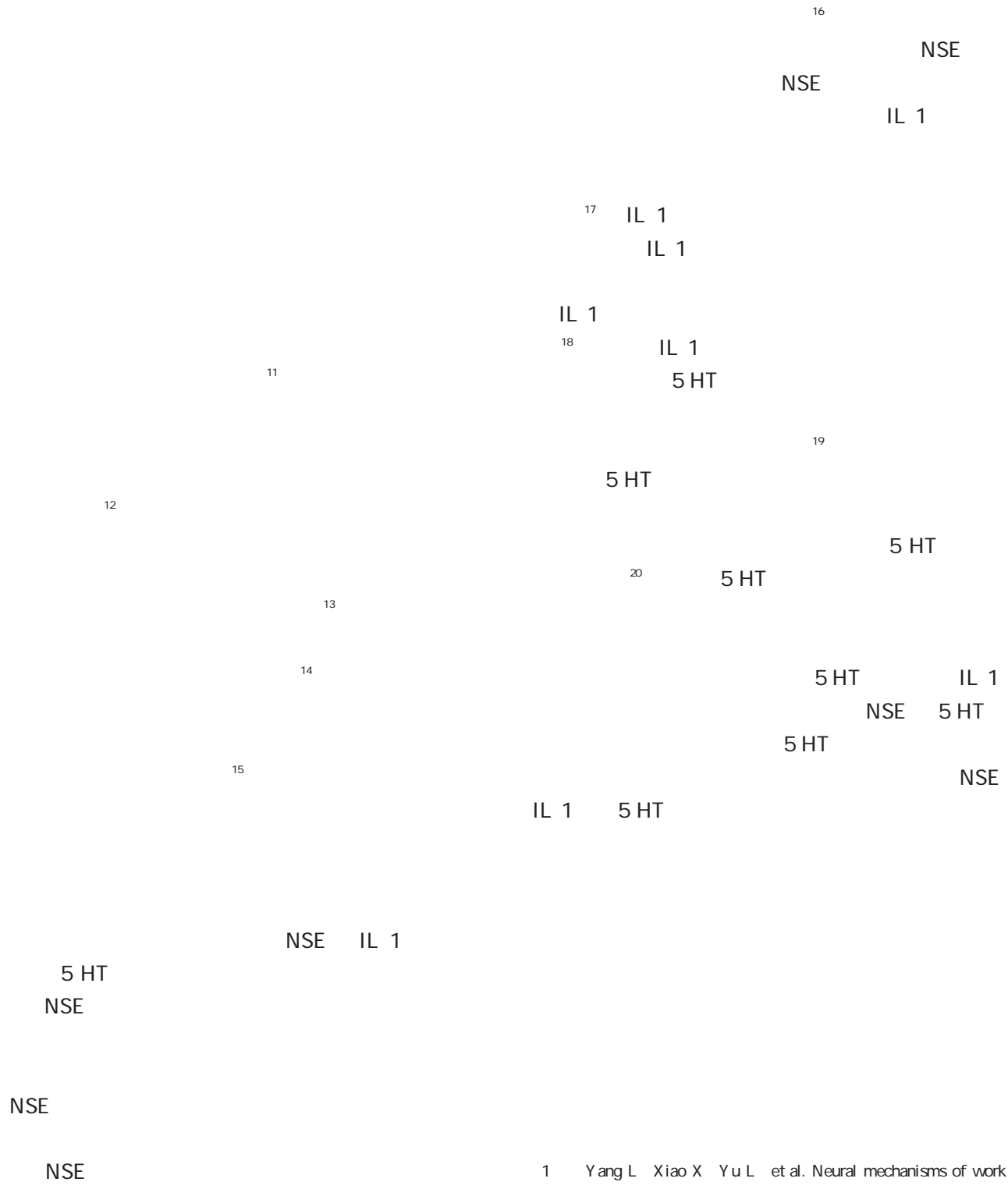
	n	MoCA	NSE ng/mL	IL 1 pg/mL	5 HT ng/mL
	41	12.78±3.51	22.89±3.14	78.22±8.38	20.58±1.47
	49	18.61±4.64	16.72±3.07	68.62±8.78	24.36±2.10
t		6.614	9.397	5.274	9.703
P		<0.001	<0.001	<0.001	<0.001

3 n % -
Table 3 Comparison of general clinical data between the two groups n % -

	n						
	41	32 78.0	9 22.0	27.56±5.44	3.53±0.57	11.98±1.33	27 65.9
	49	42 85.7	7 14.3	25.71±5.01	3.37±0.49	11.76±1.29	24 49.0
F/			0.897	1.678	1.432	0.794	2.588
P			0.343	0.097	0.156	0.429	0.108

Table 5 Predictive efficiency of s NSE IL 1 5 HT 66ot6 2 HiU H

	AUC		95% CI	%	%	Youden	P
NSE	0.918	19.33 ng/mL	0.841-0.966	89.80	75.61	0.6541	<0.001
IL 1	0.788	73.32 pg/mL	0.690-0.867	73.47	78.05	0.515	<0.001
5 HT	0.923	22.25 ng/mL	0.847-0.969	77.55	92.68	0.7023	<0.001
	0.966		0.904-0.993	83.67	97.56	0.8123	<0.001



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• •

mTOR

1 1 2 1

CHF mTOR

2021 1 2023 12

CHF n=43 n=44

3 LVEF N

NT proBNP 6 6MWD 5 > 8 8M 0 (? Né FE M E

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group $t=3.676$ 7.814 6.978 11.593 14.147 $P<0.05$ the difference was statistically significant $t=9.183$
 16.140 14.315 13.383 8.634 $P<0.05$. There were no significant differences in serum levels of Myo Cr
 ALT and AST between the two groups $t=0.673$ 0.434 0.335 0.635 $P>0.05$ Exercise
 rehabilitation can improve cardiac function activate autophagy and inhibit the PI3K/AKT/mTOR pathway in
 patients with CHF.

CHF	Exercise rehabilitation	Heart function	Autophagy	mTOR
Chronic heart failure CHF	63.68±6.59	22.59±3.23 kg/m ²		
CHF	24	17	28	
CHF	15	64.03±6.28	22.91±	
CHF	3.44 kg/m ²	22	19	P>0.05
CHF	1.2			
CHF	12			
CHF	3			
CHF	4			
CHF				2/5
target of rapamycin mTOR				3/5
CHF	mTOR	40 min	3-4	
CHF	5	3		
mTOR		1.3		
1				5 mL 30 min
1.1		10 min		3 000 r/min 10 cm
2021	1	2023	12	
CHF				
CHF			6	
New York Heart Association NYHA				
~	6			
		1.4		mTOR mRNA
				5 mL Trizol
		RNA		RNA
		cDNA cDNA		PCR
		PI3K AKT mTOR		mRNA
90		45		
		1.5		
n=43	1	2	n=44	3
			26	18

left ventricular ejection fraction 2.2
 LVEF N
 NT terminal pro brain natriuretic peptide NT proB
 NP 6 6 minute walking distance
 6MWD 30m
 6

6MWD MLHFQ
 P>0.05
 6MWD MLHFQ
 P<0.05 2
 2 -

Minnesota Living with Heart Failure
 Questionnaire MLHFQ

Table 2 Comparison of exercise endurance and quality of life between the two groups -

n	6MWD m	MLHFQ
44	301.32±14.58	347.61±22.47 ^a
43	304.18±20.76	320.47±21.85 ^a
t	0.766	7.814
P	0.452	<0.001

LC3 Bedin 1 A tg9a PI3K AKT
 mTOR mRNA

n	6MWD m	MLHFQ
44	301.32±14.58	347.61±22.47 ^a
43	304.18±20.76	320.47±21.85 ^a
t	0.766	7.814
P	0.452	<0.001

^aP<0.05

1.6
 -
 SPSS 20.0
 t P<0.05

2.3
 LC3 Bedin 1 A tg9a
 P>0.05

2
 2.1

LC3 Bedin 1 A tg9a
 P<0.05 3
 mTOR mRNA

proBNP LVEF NT
 P>0.05
 LVEF
 NT proBNP
 P<0.05 1
 1 -

2.4
 mRNA
 P<0.05 4
 PI3K AKT mTOR
 P>0.05
 PI3K
 AKT mTOR mRNA
 P<0.05

Table 1 Comparison of cardiac function indexes between the two groups -

n	LVEF %	NT proBNP pg/mL
44	53.57±4.58	59.42±4.22 ^a
43	53.12±4.91	56.14±4.10 ^a
t	0.442	3.676
P	0.659	0.001

^aP<0.05

2.5
 Myo Cr ALT AST
 P<0.05 5

3
 CHF
 CHF

Table 3 Comparison of serum autophagy markers between the two groups -

n	LC3 ng/mL	Bedin 1 pg/mL	A tg9a ng/mL
44	0.85±0.11	1.32±0.11 ^a	18.12±1.52
43	0.89±0.10	1.14±0.13 ^a	27.79±2.42 ^a
t	1.774	6.978	11.593
P	0.079	<0.001	<0.001

^aP<0.05

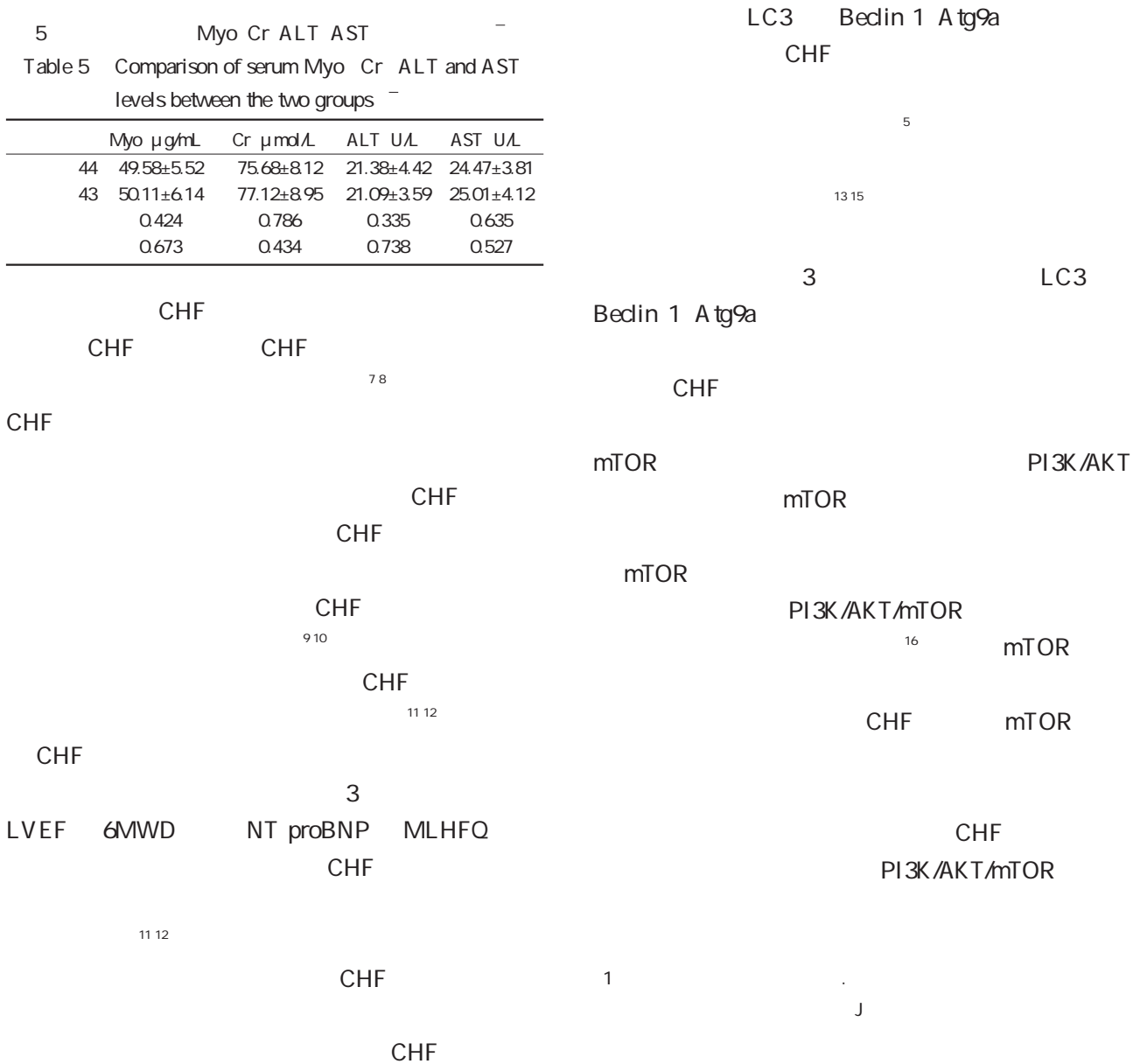
4 PI3K AKT mTOR mRNA
 Table 4 Comparison of mRNA expression levels of PI3K AKT and mTOR in peripheral blood between the two groups

	n	PI3K		AKT		mTOR	
		44	0.97±0.10	1.66±0.13 ^a	1.02±0.09	1.78±0.18 ^a	1.04±0.11
t	43	1.00±0.11	1.29±0.11 ^a	1.00±0.08	1.34±0.12 ^a	1.00±0.09	1.35±0.14 ^a
P		1.399	14.315	1.095	13.383	1.854	8.634
		0.165	<0.001	1.095	<0.001	0.067	<0.001

^aP<0.05

5 Myo Cr ALT AST
 Table 5 Comparison of serum Myo Cr ALT and AST levels between the two groups

	Myo μg/mL	Cr μmol/L	ALT U/L	AST U/L
44	49.58±5.52	75.68±8.12	21.38±4.42	24.47±3.81
43	50.11±6.14	77.12±8.95	21.09±3.59	25.01±4.12
t	0.424	0.786	0.335	0.635
P	0.673	0.434	0.738	0.527



NLRP3

2022 1 2024 1
 DN NLRP3
 40 DN 80
 3
 PBMCs NLRP3
 NLRP3
 3 FBG HbA1c
 $t=0.698$ 0.894 $P>0.05$ Scr BUN
 ACN PBMCs NLRP3 Caspase 1 mRNA 1 IL 1 18 IL 18
 Gasdermin D N GSDMD N $t=7.884$ 4.049 2.216 3.677
 5.355 7.336 6.654 7.835 $P<0.05$ 3
 $\chi^2=0.581$ $P>0.05$ DN
 NLRP3
 NLRP3

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 The First People s Hospital of Huoqiu County Lu an Anhui China 237400

To investigate the efficacy of semaglutide in the treatment of elderly diabetic nephropathy DN and its effect on the NLRP3 inflammasome pathway. . . . A total of 80 elderly patients with DN who were treated at the First People s Hospital of Huoqiu County Lu an from January 2022 to January 2024 were divided into a control group and an observation group each with 40 cases according to the random number table method. Patients in the control group received the routine treatment for clinical DN while those in the observation group received combined treatment with semaglutide based on the treatment regimen of the control group. The treatment duration for patients in both groups was 3 months. After 3 months of treatment blood glucose levels renal function indices NLRP3 inflammatory body pathway molecule expression in peripheral blood mononuclear cells PBMCs and NLRP3 inflammatory body downstream factor content in serum were compared between the two groups. Additionally differences in the occurrence of drug related adverse reactions during treatment were also assessed. . . . After 3 months of treatment there were no significant differences in fasting blood glucose FBG and glycosylated hemoglobin HbA1c levels between the two groups $t=0.698$ 0.894 $P>0.05$. The levels of blood creatinine Scr urea nitrogen BUN and

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urinary creatinine ratio ACR the mRNA expressions of NLRP3 and caspase 1 in PBMCs and the contents of serum interleukin 1 IL 1 interleukin 18 IL 18 and gasdermin D N terminal GSDMD N in the observation group were lower than those in the control group and the differences were statistically significant $t=7.884$ 4.049 2.216 3.677 5.355 7.336 6.654 7.835 $P<0.05$. During the 3 months of treatment there was no significant difference in the incidence of drug related adverse reactions between the two groups $\chi^2=0.581$ $P>0.05$ The treatment of semaglutide in elderly patients with DN is helpful in improving efficacy and ensuring treatment safety. The mechanism of its action may be related to the inhibition of the NLRP3 inflammasome pathway.

. Diabetic nephropathy Semaglutide Kidney function NLRP3 inflammasome

Diabetic nephropathy DN

12	DN	DN	DN	22	18
3		69.23±7.19			10.61±2.85
		DN 3.71±0.95			21
		19	69.18±6.85		
		10.47±2.92	DN	3.65±0.92	
2					P>
1	glucagon like peptide 1 GLP 1	0.05			
	45	1.2			
DN		DN		DN	
DN				1.5 mL	1.34
		mg/mL			0.25 mg
		1	4		0.5 mg
1					3
1.1		1.3			
		1.3.1			
2022	1	2024	1	3	
		DN	80		
				fasting blood glucose FBG	
				A1c Glycated Hemoglobin A1c HbA1c	
40		DN		Serum creatinine Scr	BUN
6	2	6		Urinary albumin creatinine ratio ACR	
60	<80			1.3.2	Peripheral blood mono
		DN		nuclear cells PBMCs	NLRP3
				mRNA	
				5.0mL	1 1

3 000 r/m 10 min P>0.05 1
 PBMCs PBMCs Trizol 2.3
 -80 mRNA Scr BUN ACR
 PBMCs RNA P>0.05 3
 actin PCR Scr BUN ACR
 PBMCs NLRP3 Caspase 1 mRNA P<0.05 2
 1.3.3 2.4 PBMCs NLRP3 mRNA
 3 5.0 mL PBMCs NLRP3 Caspase 1
 NLRP3 1 mRNA P>0.05
 interleukin 1 IL 1 18 interleukin 18 3 PBMCs NLRP3 Caspase 1
 IL 18 Gasdermin D N GSDMD N mRNA
 1.3.4 P<0.05 3
 3 2.5 NLRP3
 1.4 IL 1 IL 18 GSDMD N
 P>0.05 3
 SPSS 22.0 IL 1 IL 18 GSDMD N
 - P<0.05
 t n % 4
 2.6
 U P<0.05 3 2
 1 1
 2.1 3 1 2
 FBG HbA1c
 P>0.05 3
 FBG HbA1c $\chi^2=0.482$ P>0.05

Table 1 Comparison of blood glucose levels between the two groups

	n	FBG mmol/L		t	P	HbA1c %		t	P
		3	3			3	3		
	40	9.82±1.76	6.91±0.95	9.202	0.000	7.84±0.96	5.97±0.88	9.082	0.000
	40	9.79±1.84	6.77±0.84	9.443	0.000	7.80±0.93	5.80±0.82	10.202	0.000
t		0.075	0.698			0.189	0.894		
P		0.941	0.487			0.850	0.374		

Table 2 Comparison of renal function indexes between the two groups

	n	Scr μ mol/L		t	P	BUN mmol/L		t	P	ACR mg/mmol		t	P
		3	3			3	3			3	3		
	40	195.83±30.32	142.74±20.32	9.199	0.000	6.83±0.92	5.47±0.78	7.131	0.000	243.71±37.49	190.33±27.54	7.257	0.000
	40	194.77±8.94	108.61±18.35	26.696	0.000	6.80±0.88	4.82±0.65	11.446	0.000	241.65±36.08	178.02±21.81	9.545	0.000
t		0.212	7.884			0.149	4.049			0.250	2.216		
P		0.833	0.000			0.882	0.000			0.803	0.030		

3 PBMCs NLRP3

Table 3 Comparison of NLRP3 inflammasome pathway related molecule levels in PBMCs between the two groups

	n	NLRP3		t	P	Caspase 1		t	P
		3	3			3	3		
	40	0.84±0.13	0.76±0.09	3.200	0.002	0.76±0.09	0.66±0.08	5.252	0.000
	40	0.82±0.15	0.69±0.08	4.836	0.000	0.74±0.10	0.57±0.07	8.808	0.000
t		0.637	3.677			0.940	5.355		
P		0.526	0.000			0.350	0.000		

4 NLRP3

Table 4 Comparison of NLRP3 downstream cytokines in serum between the two groups

	n	IL 1		t	P	IL 18		t	P	GSDMD N		t	P
		3	3			3	3			3	3		
	40	54.28±7.10	40.15±5.48	9.964	0.000	26.03±4.31	19.74±3.05	7.534	0.000	17.32±2.40	13.01±1.78	9.123	0.000
	40	53.97±7.34	32.07±4.30	16.282	0.000	26.12±4.50	15.82±2.14	13.073	0.000	17.46±2.58	10.25±1.34	15.685	0.000
t		0.192	7.336			0.091	6.654			0.251	7.835		
P		0.848	0.000			0.927	0.000			0.802	0.000		

3

DN DN

DN

DN

6

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was no statistical significance in the comparison of CD3⁺ CD4⁺ CD8⁺ and CD4⁺/CD8⁺ $P>0.05$. However, the T_1 , T_2 and T_3 CD3⁺ CD4⁺ and CD4⁺/CD8⁺ levels in the observation group was higher than that in the control group and CD8⁺ was higher than that in the control group and the difference was statistically significant $P<0.05$. When comparing the incidence rate of adverse reactions between the two groups, the difference was not statistically significant $P>0.05$ Compared to sevoflurane combined anaesthesia, remifentanyl combined anaesthesia has a lesser effect on serum IL-6 and IL-2 levels in LC patients, impacting the immune combiot b e

min 10 cm 15 min P>0.05 T₁ T₂ T₃ IL 6
 IL 6 IL 2 IL 2
 P<0.05 2
 T CD3⁺
 CD4⁺ CD8⁺ CD4⁺/CD8⁺ min
 1.3.3

Table 1 Comparison of postoperative awakening time recovery time of spontaneous respiration and catheter removal time between the two groups - min

n			
59	6.85±2.14	2.36±0.68	8.34±2.38
63	7.96±2.52	5.78±1.35	10.27±3.59
t	2.613	17.488	3.475
P	0.010	<0.001	<0.001

1.4 SPSS 21.0
 - t
 t n %
 2 P<0.05

2.3 T
 T₀CD3⁺ CD4⁺ CD8⁺ CD4⁺/CD8⁺
 P>0.05 T₁ T₂ T₃
 CD3⁺ CD4⁺ CD4⁺/CD8⁺ CD8⁺
 P<0.05

2
 2.1
 P<0.05 1
 2.2 IL 6 IL 2
 T₀IL 6 IL 2
 2 IL 6 IL 2
 P>0.05 4

Table 3 Comparison of postoperative IL 6 and IL 2 levels between the two groups -

n	IL 6 pg/L				IL 2 ng/mL			
	T ₀	T ₁	T ₂	T ₃	T ₀	T ₁	T ₂	T ₃
59	16.38±2.47	26.53±2.41 ^a	31.52±3.68 ^{ab}	42.05±4.68 ^{abc}	13.53±1.10	11.46±1.35 ^a	9.53±1.26 ^{ab}	9.21±1.34 ^{ab}
63	16.45±2.64	22.51±3.47 ^a	29.86±3.45 ^{ab}	35.74±4.77 ^{abc}	13.42±1.12	12.32±1.24	10.27±1.16 ^{ab}	10.34±1.28 ^{ab}
t	0.150	7.384	2.571	7.368	0.546	3.667	3.377	4.763
P	0.880	<0.001	0.011	<0.001	0.585	<0.001	0.001	<0.001

T₀ ^aP<0.05 T₁ ^bP<0.05 T₂ ^cP<0.05

Table 4 Comparison of postoperative T lymphocyte subpopulation levels between the two groups -

n	CD3 ⁺ %				CD4 ⁺ %			
	T ₀	T ₁	T ₂	T ₃	T ₀	T ₁	T ₂	T ₃
59	60.23±9.65	56.38±8.57 ^a	54.72±6.82 ^{ab}	52.68±5.39 ^b	44.78±6.10	41.87±5.14 ^a	40.64±4.35 ^a	39.68±4.24 ^{ab}
63	61.25±8.37	59.68±8.65 ^a	57.38±5.43 ^{ab}	55.69±5.08 ^b	45.42±6.37	43.74±4.36 ^a	42.41±4.47 ^{ab}	41.53±4.32 ^{ab}
t	0.624	2.115	2.390	3.175	0.566	2.171	2.214	2.385
P	0.533	0.036	0.018	0.001	0.572	0.031	0.028	0.018

n	CD8 ⁺ %				CD4 ⁺ /CD8 ⁺			
	T ₀	T ₁	T ₂	T ₃	T ₀	T ₁	T ₂	T ₃
59	15.76±3.86	17.67±2.78 ^a	18.67±2.71 ^a	18.86±2.75 ^{ab}	2.23±0.86	2.31±0.73 ^a	2.53±0.43 ^{ab}	2.60±0.36 ^{ab}
63	14.89±3.12	15.43±2.45 ^a	16.38±2.08 ^{ab}	16.42±2.42 ^{ab}	2.26±0.75	2.66±0.69 ^a	2.73±0.35 ^a	2.81±0.42 ^a
t	1.373	4.728	5.255	5.210	0.205	2.722	2.825	2.955
P	0.172	<0.001	<0.001	<0.001	0.837	0.007	0.005	0.003

T₀ ^aP<0.05 T₁ ^bP<0.05 T₂ ^cP<0.05

4 n %
 Table 5 Comparison of the incidence of adverse reactions
 between the two groups n %

n	
59	5 8.47 1 1.69 1 1.69 3 5.08 10 16.94
63	2 3.17 4 6.34 2 3.17 1 1.58 9 14.28
P	0.164 0.685

3

LC

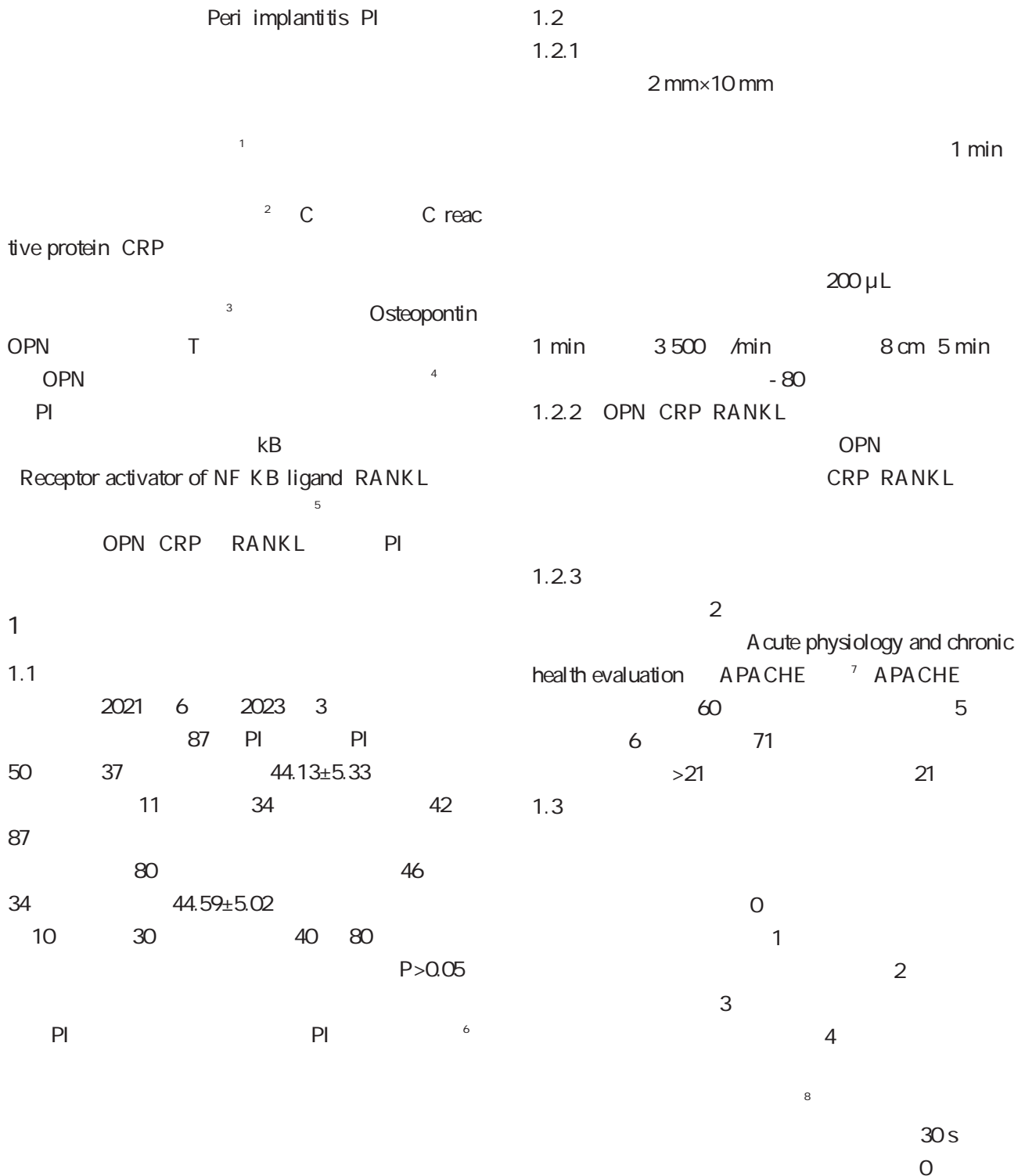
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B C C 1 1 2 1 2 1 1

2 2 2 B 1 0 C

out of 87 patients had a good prognosis and 29 had a poor prognosis. OPN, CRP, and RANKL levels were higher in the poor prognosis group than in the good prognosis group and the difference was statistically significant ($P < 0.05$). The levels of OPN, CRP, and RANKL in the gingival sulcus fluid of patients with PI were found to be abnormally high and correlated with the modified gingival index, modified bleeding index score and probing depth. Therefore, assessing the prognosis of patients with PI can be done by detecting these levels in the gingival sulcus fluid. This information can provide a basis for clinical treatment.

OPN, CRP, Nuclear factor- κ B receptor activator ligand, PI



1 2 9

3

1.4 SPSS21.0

- t

Pearson OPN CRP RANKL PI

P<0.05

2

2.1 OPN CRP RANKL

PI OPN CRP RANKL

P<0.05 1

1 OPN CRP RANKL

Table 1 Comparison of OPN CRP and RANKL levels between the two groups

	n	OPN pg/ μ L	CRP mg/L	RANKL pg/ μ L
	80	192.50 \pm 46.50	3.25 \pm 0.34	30.89 \pm 7.22
PI	87	245.48 \pm 41.15	24.32 \pm 3.10	122.31 \pm 12.94
t		7.810	60.444	55.709
P		<0.001	<0.001	<0.001

2.2

PI

P<0.05 2

2

Table 2 Comparison of periodontal clinical indicators between the two groups

	n	mm
	80	0.61 \pm 0.08
PI	87	1.46 \pm 0.33
t		22.435
P		<0.001

2.3 OPN CRP RANKL PI

OPN CRP RANKL

P<0.05

3

3 OPN CRP RANKL PI

Table 3 Correlation analysis between OPN CRP RANKL levels and PI

	OPN		CRP		RANKL	
	r	P	r	P	r	P
	0.401	<0.001	0.422	<0.001	0.374	0.007
	0.491	<0.001	0.430	<0.001	0.446	<0.001
	0.451	<0.001	0.431	<0.001	0.403	<0.001

2.4 PI OPN CRP RANKL

87 58

29 OPN

CRP RANKL

P<0.05 4

4 PI OPN CRP RANKL

Table 4 Comparison of OPN CRP and RANKL levels in patients with different prognosis in PI group

	n	OPN pg/ μ L	CRP mg/L	RANKL pg/ μ L
	58	233.71 \pm 43.59	20.62 \pm 2.69	114.67 \pm 10.23
	29	269.02 \pm 38.27	31.72 \pm 3.19	137.59 \pm 12.05
t		5.573	24.199	13.193
P		<0.001	<0.001	<0.001

3

PI

10

PI PI

PI

PI OPN CRP RANKL

PI OPN CRP

RANKL

PI

OPN

PI

CRP

PI

RANKL

PI

11 12

RANKL

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PI

CRP OPN RANKL

CRP OPN RANKL

PI

OPN

CRP

RANKL

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2

2.1

LCR HALP
LCR HALP
P<0.05 1

Table 1 Comparison of preoperative LCR and HALP scores between the two groups

	LCR	HALP
78	1.65±0.45	41.05±4.21
24	0.39±0.14	26.33±3.17
	13.475	15.786
	<0.001	<0.001

2.2

LCR HALP

LCR HALP

area under curve AUC

0.837 0.759 0.904

2

LCR HALP

Table 2 Predictive value of LCR and HALP scores for recurrence after radiofrequency ablation in cases with persistent atrial fibrillation

	AUC	95% CI	%	%
LCR	0.837	0.786-0.888	1.02	67.43
HALP	0.759	0.708-0.810	33.69	56.43
LCR+HALP	0.904	0.853-0.954		86.91

2.3

BMI

LAD

P<0.05

3

2.4

Logistic

=0 =

4

Table 4 Binary logistic stepwise regression analysis of recurrence after radiofrequency ablation in cases with persistent atrial fibrillation

	SE	Wald	OR	95% CI	P
<6.21 =0 6.21 =1	0.855	0.213	16.113	2.351 1.549-3.570	<0.001
=0 =1	0.729	0.192	14.416	2.073 1.423-3.020	<0.001
LAD <31.99 mm=0 31.99 mm=1	1.002	0.231	20.190	2.274 1.759-4.217	<0.001
LCR >1.02=0 1.02=1	1.143	0.245	23.657	3.136 1.979-4.971	<0.001
HALP >33.69=0 33.69=1	0.916	0.219	18.669	2.499 1.650-3.787	<0.001

1

Logistic

=0.05

=0.10

LAD

LCR

HALP

P<0.05

4

3

Table 3 Univariate analysis of recurrence after radiofrequency ablation in patients with persistent AF

	n=78	n=24	t/	P
<60	32 41.03	7 29.17		
60	46 58.97	17 70.83	1.093	0.296
BMI kg/m ²	22.67±3.15	25.13±3.44	3.274	<0.001
	5.24±1.12	7.18±1.63	6.618	<0.001
	21 26.92	12 50.00	4.466	0.035
	6 7.69	2 8.33	0.010	0.919
	28 35.90	8 33.33	0.053	0.818
	12 15.38	3 12.50		
	66 84.62	21 87.50	0.122	0.727
ACEI/ARB	32 41.03	11 45.83		
	46 58.97	13 54.17	0.174	0.677
LVEF %	70.69±8.23	72.64±8.55	1.006	0.317
LAD mm	35.82±3.87	40.17±4.26	4.702	<0.001
LVEDD mm	47.55±4.46	48.03±4.74	0.454	0.651

3

9

LCR

CRP

LCR AUC 0.904 LCR
LCR HALP
CRP LCR HALP

CRP LCR HALP
CRP
10 CRP

1 ACE2 Ang Ang 17 J .
2021 13 12 2008 2012

LCR J . 2022 19 10 46 49.

HALP J Li X Peng S
2022 209

HALP

13

14

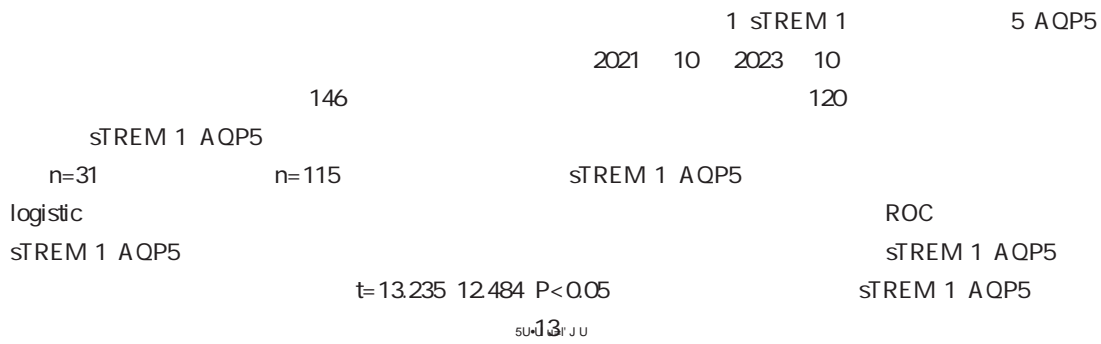
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16 HALP

LCR HALP
AUC 0.837 0.759

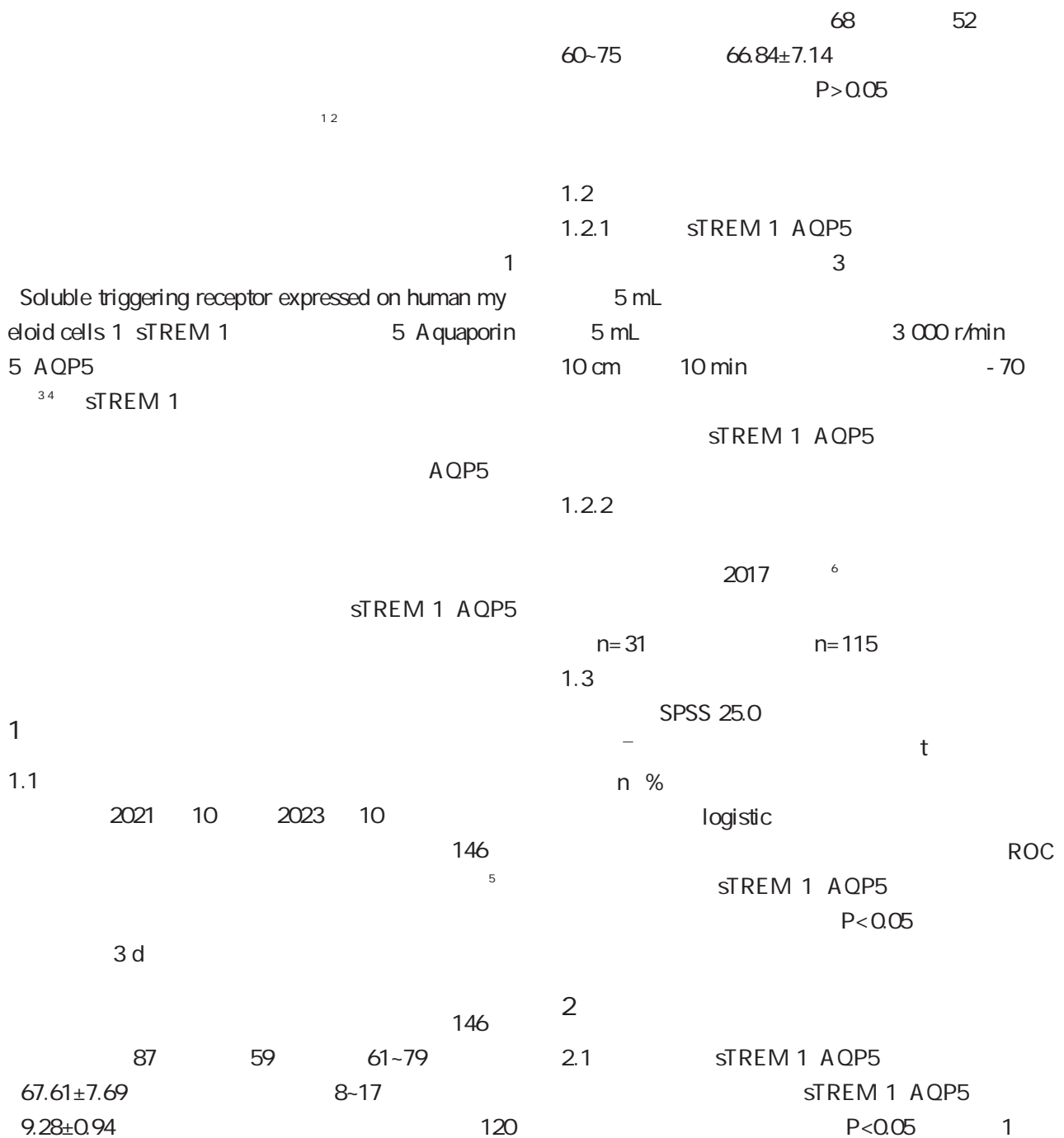
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sTREM 1 AQP5



12.484 P<0.05 . The levels of serum sTREM 1 and AQP5 as well as the proportion of diabetes mellitus operation time intraoperative blood loss and proportion of vitreous spill in the infected subgroup were higher than those in the non infected subgroup with statistical significance t=6.536 4.026 4.392 6.934 10.544 14.508 P<0.05 . History of diabetes mellitus prolonged operation time and increased levels of serum sTREM 1 and AQP5 were identified as risk factors for infectious endophthalmitis after cataract surgery P<0.05 . Serum sTREM 1 and AQP5 showed predictive value for infectious endophthalmitis after cataract surgery and the predicted areas under the curve were 0.807 and 0.905 The elevation of serum sTREM 1 and AQP5 levels is correlated with infective endophthalmitis following cataract surgery. Both indexes hold predictive significance for the development of infective endophthalmitis post cataract surgery.

... . Cataract Infectious endophthalmitis sTREM 1 Aquaporin 5



1 sTREM 1 AQP5

Table 1 Comparison of serum sTREM 1 and AQP5 levels between the observation group and the control group

	n	sTREM 1 ng/mL	AQP5 ng/mL
	146	121.22±21.46	37.18±8.14
	120	86.14±11.32	26.24±5.61
t		13.235	12.484
P		<0.001	<0.001

2 sTREM 1 AQP5
n %

Table 2 Comparison of serum sTREM 1 and AQP5 levels and clinical data between infected subgroup and non infected subgroup

	n=31	n=115	t/	P
/	16/15	71/44	1.040	0.308
	68.22±9.93	67.45±7.85	0.457	0.648
	9.36±1.01	9.26±0.94	0.517	0.606
	16 51.61	51 44.35	0.519	0.471
	14 45.16	36 31.30	4.392	0.036
	10 32.26	25 21.74	1.482	0.223
min	41.39±5.69	34.62±4.57	6.934	<0.001
mL	12.85±1.95	9.84±1.23	10.544	<0.001
	22 70.97	38 33.04	14.508	<0.001
sTREM 1 ng/mL	144.87±26.45	114.95±21.50	6.536	<0.001
AQP5 ng/mL	41.71±6.89	35.96±7.10	4.026	<0.001

78

sTREM 1 AQP5

sTREM 1 AQP5

ROC

sTREM 1 AQP5

sTREM 1 AQP5

910

sTREM 1

TREM 1

TREM 1

TREM 1

AQP5

AQP5

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sTREM 1 AQP5 æKV@'VU+4 Ð P U< Z Z

UÀ X'z'g ' F T O P # sTREM 1 AQP5 05% T Á 0% T' % U Ð T' % T M Ð T à Sa... B Q\ Do Sð B O P A # Sa... N\ % ? T Á # Ga... B D Q\ % Sa... S

3d

sTREM 1 AQP5

sTREM 1 AQP5

1516

logistic



HPV

TCT

1 2 1 1 1 1 1 1 1 1

				HPV		TCT			
				2022	7	2023	9	10124	
HPV	TCT			10124		HPV	7.34%	HPV	
				$\chi^2=29.721$		$P<0.001$		HPV 52 HPV 58	
HPV 51	HPV 16	HPV 39	TCT	2.49%	61	ASC	HSIL	21~	
LSIL				ASC	LSIL	HSIL	HPV	51.41%	
70.18%	88.89%				HPV	49	? " & D ? "	ADT1p	s» @ 1 0011 @ + C

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1 HPV n %
Table 1 HPV infection by age group n %

	n=10124	21~ n=738	31~ n=3723	41~ n=3090	51~ n=1803	61 n=770		P
	9381 92.66	658 89.16	3451 92.69	2905 94.01	1674 92.85	693 90.00	29.721	<0.001
	635 6.27	67 9.08	235 6.31	163 5.28	112 6.21	58 7.53	7.985	0.092
	108 1.07	13 1.76	37 0.99	22 0.71	17 0.94	19 2.47		

2 HPV n %
Table 2 Distribution of HPV subtypes by age n %

	n=876	21~ n=99	31~ n=315	41~ n=210	51~ n=152	61 n=100		P
HPV 16	66 7.53	5 5.05	22 6.98	22 10.48	13 8.55	4 4.00	5.642	0.228
HPV 18	28 3.20	2 2.02	9 2.86	9 4.29	6 3.95	2 2.00	1.846	0.777
HPV 31	40 4.57	3 3.03	16 5.08	10 4.76	5 3.29	6 6.00	1.785	0.775
HPV 33	24 2.74	1 1.01	6 1.90	10 4.76	6 3.95	1 1.00	6.209	0.158
HPV 35	23 2.63	3 3.03	5 1.59	7 3.33	4 2.63	4 4.00	3.097	0.536
HPV 39	61 6.96	9 9.09	26 8.25	12 5.71	7 4.61	7 7.00	3.312	0.507
HPV 45	7 0.80	1 1.01	3 0.95	0	3 1.97	0	4.584	0.223
HPV 51	85 9.70	16 16.16	33 10.48	24 11.43	8 5.26	4 4.00	12.774	0.012
HPV 52	217 24.77	14 14.14	76 24.13	57 27.14	38 25.00	32 32.00	9.515	0.049
HPV 56	60 6.85	7 7.07	25 7.94	9 4.29	13 8.55	6 6.00	3.559	0.469
HPV 58	117 13.36	12 12.12	39 12.38	22 10.48	28 18.42	16 16.00	5.868	0.209
HPV 59	41 4.68	11 11.11	15 4.76	7 3.33	3 1.97	5 5.00	10.711	0.013
HPV 66	58 6.62	8 8.08	21 6.67	14 6.67	10 6.58	5 5.00	0.768	0.943
HPV 68	37 4.22	5 5.05	14 4.44	5 2.38	6 3.95	7 7.00	4.109	0.385
HPV 82	12 1.37	2 2.02	5 1.59	2 0.95	2 1.32	1 1.00	1.040	0.930

3 TCT n %
Table 3 TCT detection in all age groups n %

	n=10124	21~ n=738	31~ n=3723	41~ n=3090	51~ n=1803	61 n=770		P
NILM	9872 97.51	712 96.48	3625 97.37	3020 97.73	1762 97.73	753 97.79	4.797	0.309
ASC	177 1.75	18 2.44	72 1.93	43 1.39	30 1.66	14 1.82	5.182	0.269
LSIL	57 0.56	8 1.08	19 0.51	20 0.65	9 0.50	1 0.13	6.866	0.143
HSIL	18 0.18	0	7 0.19	7 0.23	2 0.11	2 0.26	2.109	0.717

9 276 91.62%

1.45%

LSIL HSIL HPV

70.18% 88.89%

P<0.001

HSIL

LSIL

P<0.001

TCT

HPV

4

37.5% 6/16

77.5% 31/40

HPV 16 HPV 45 HPV

P<0.005

66

5

147

3

ASC

51.41%

HPV

HPV

DNA

E2

1 3

HPV

HPV

HPV

E6

E7

DNA

E2

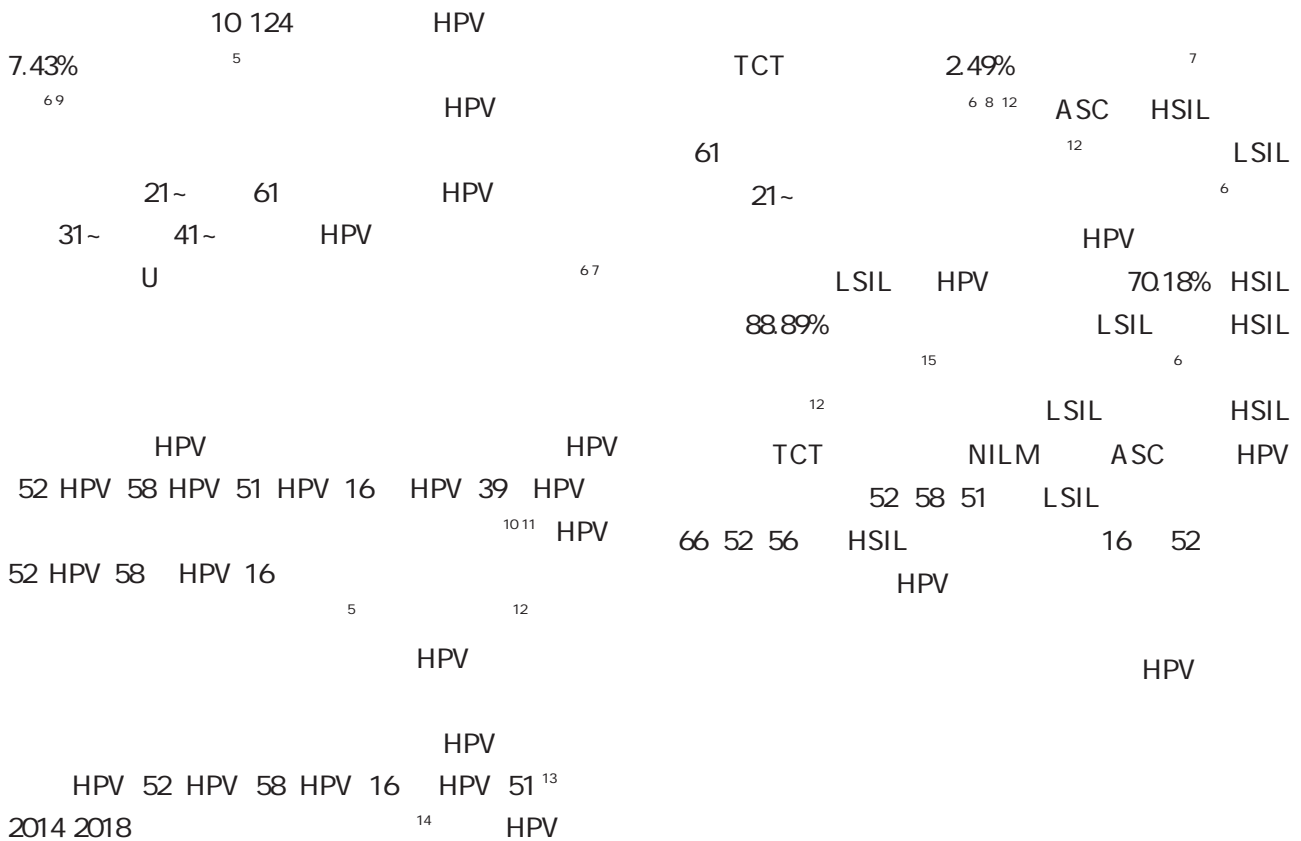
TCT

4 TCT HPV n %
Table 4 TCT results and HPV infection n %

n=10124	NILM n=9872	ASC n=177	LSIL n=57	HSIL n=18	P
9381 92.66	9276 93.96	86 48.59	17 29.82	2 11.11	485.453 <0.001
635 6.27	525 5.32	69 38.98	31 54.39	10 55.56	18.943 <0.001
108 1.07	71 0.72	22 12.43	9 15.79	6 33.33	

5 TCT HPV n %
Table 5 HPV subtype distribution for different TCT results n %

n=876	NILM n=679	ASC n=122	LSIL n=53	HSIL n=22	P	
HPV 16	66 7.53	49 7.22	7 5.74	4 7.55	6 27.27	9.317 0.02
HPV 18	28 3.20	23 3.39	3 2.46	1 1.89	1 4.55	0.776 0.829
HPV 31	40 4.57	30 4.42	4 3.28	5 9.43	1 4.55	3.336 0.288
HPV 33	24 2.74	19 2.80	4 3.28	0	1 4.55	2.009 0.477
HPV 35	23 2.63	15 2.21	6 4.92	2 3.77	0	3.541 0.246
HPV 39	61 6.96	53 7.81	7 5.74	1 1.89	0	3.773 0.267
HPV 45	7 0.80	2 0.29	4 3.28	1 1.89	0	10.534 0.011
HPV 51	85 9.70	67 9.87	12 9.84	5 9.43	1 4.55	0.696 0.897
HPV 52	217 24.77	177 26.07	27 22.13	8 15.09	5 22.73	3.781 0.286
HPV 56	60 6.85	46 6.77	6 4.92	7 13.21	1 4.55	3.782 0.260
HPV 58	117 13.36	91 13.40	19 15.57	6 11.32	1 4.55	2.185 0.535
HPV 59	41 4.68	34 5.01	7 5.74	0	0	3.399 0.284
HPV 66	58 6.62	39 5.74	8 6.56	9 16.98	2 9.09	8.781 0.024
HPV 68	37 4.22	26 3.83	7 5.74	3 5.66	1 4.55	1.932 0.561
HPV 82	12 1.37	8 1.18	1 0.82	1 1.89	2 9.09	6.800 0.053



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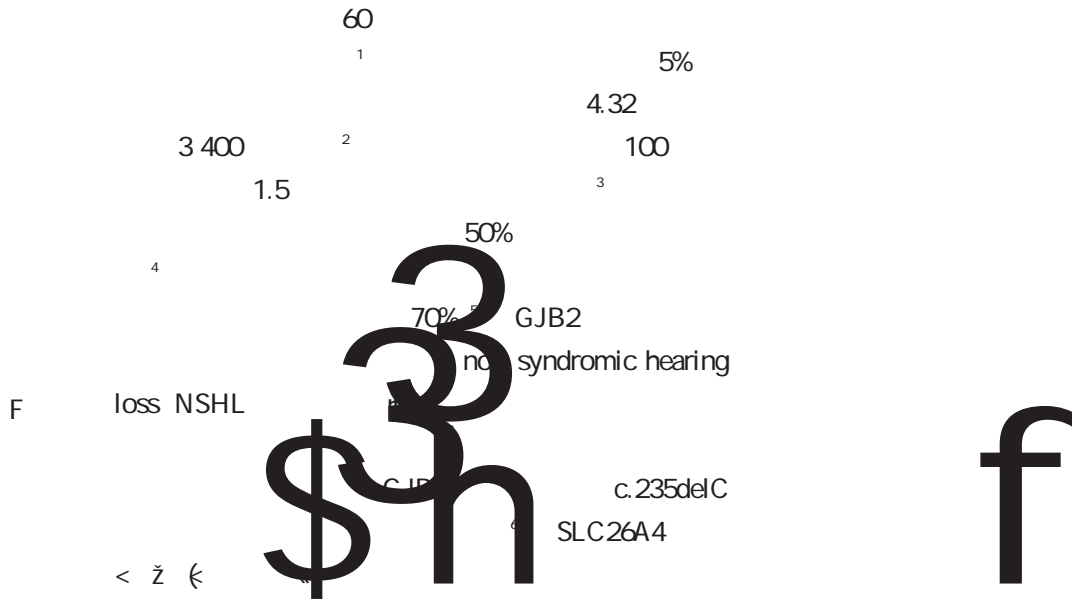
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c.235delC showed significant differences between the two nationalities. The GJB2 c.235delC and SLC26A4 c.919-2A>G mutations were most commonly found in patients with NSHL in our region. The frequency of the GJB2 gene mutation in Han patients was significantly higher than in Mongolian patients and the mutation spectra of the GJB2 and SLC26A4 genes also differed between the two groups.

NSHL Han nationality Mongolian ethnicities Gene mutations Mutation frequency



2					18		
2.1				GJB2		56.32%	49/87
	62	49.6%		SLC26A4		29.89%	26/87
		31	24.8%			22.99%	20/87
GJB2		26	20.8%	GJB2		34.21%	13/38
SLC26A4	3	2.4%		GJB3	SLC26A4	13.16%	5/38
	2	1.6%	MT CO1		2	15.79%	6/38

Table 2 Genetic mutations of NSHL patients in the Inner Mongolia region n=125 n %

		n=125	n %	n=87	n=38
GJB2	c.235delC	9	10.34	1	2.63
	c.235delC/c.299_300delAT	7	8.05	2	5.26
	c.235delC/c.176_191del16	1	1.15	1	2.63
	c.235delC/c.35delG	1	1.15	0	0.00
	c.235delC/c.427C>T	1	1.15	0	0.00
	c.235delC/c.512insAACG	1	1.15	0	0.00
SLA26A4	c.235delC	6	6.90	1	2.63
	c.919 2A>G	3	3.45	2	5.26
	c.919 2A>G/c.2168A>G	2	2.30	0	0.00
	c.919 2A>G/c.1336C>T	1	1.15	0	0.00
	c.919 2A>G/c.281C>T	2	2.30	0	0.00
	c.919 2A>G/c.2027T>A	1	1.15	0	0.00
	c.2168A>G/c.1336C>T	1	1.15	0	0.00
	c.2168A>G/c.1174A>T	1	1.15	0	0.00
	c.919 2A>G	5	5.75	2	5.26
	c.2168A>G	2	2.30	2	5.26
	c.1181_1183delTC	1	1.15	0	0.00
	c.1336C>T	1	1.15	0	0.00
GJB3	c.538C>T	2	2.30	1	2.63
MT CO1	m.7444G>A	1	1.15	1	2.63

GJB2		3	NSHL	n %
25.29%	55/250	15.6%	39/250	
GJB3	1.2%	3/250		
MT CO1	1.6%	4/250		

Table 3 Allelic mutation profiles of NSHL patients in Inner Mongolia region n %

		n=174	n=76
GJB2	c.235delC	35	20.11
	c.299_300delAT	7	4.02
	c.176_191del16	1	0.57
	c.35delG	1	0.57
	c.427C>T	1	0.57
	c.512insAACG	1	0.57
SLC26A4	c.919 2A>G	17	9.77
	c.2168A>G	6	3.45
	c.1336C>T	3	1.72
	c.281C>T	2	1.15
	c.2027T>A	1	0.57
	c.1174A>T	1	0.57
	c.1181_1183delTC	1	0.57
GJB3	c.538C>T	2	1.15
MT CO1	m.7444G>A	2	1.15

GJB2	c.235delC	SLC26A4	c.919 2A>G	
26.44%	46/174	17.82%	31/174	
GJB2		GJB2		
11.84%	9/76	SLC26A4		
		10.53%	8/76	3
2.2		GJB2		GJB2
c.235delC				
P<0.05	4			

6

6

3

NSHL

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GJB2

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34% SLC26A4

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12.6%

GJB2

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SLC26A4

5_2eY(³ ,Ub ...V2(8UbM¹ ...V5 HUb 2 .

E P M \$,!d! %œ€

LC GJB2 c. 235delC

SLC26A4 c. 919 A14 d \$ f2 delC d

6 26A4 f

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< 4

TCCD ONSD APACHE

2023 1 2023 12
n=100

TCCD ONSD
APACHE

WBC HGB
TBIL
ALT CK CK MB BNP Scr APACHE
PT SOFA GCS

1 1

1 1

1 1

1 WBC

APACHE SOFA GCS

P>0.05

SOFA GCS

$\chi^2=5.307$ P<0.05

TCCD ONSD

XIONG Shouhui YIN Wei ZHONG Jiao LIU Sun DAN Yong
Emergency EICU Changde First People's Hospital Wuling District Changde Hunan China 415003

To analyze the value of transcranial color Doppler ultrasound (TCCD) and optic nerve sheath diameter (ONSD) monitoring on blood biochemical indexes, acute physiology and chronic health evaluation (APACHE) and prognosis of cerebral resuscitation patients. Cerebral resuscitation patients with cardiac arrest who received TCCD combined with ONSD monitoring treatment at Changde First People's Hospital from January 2023 to December 2023 were selected as the study subject and categorized as the combination group (n=100). The control group (n=100) consisted of patients who received routine monitoring treatment during the same period. The blood biochemical parameters, white blood cell count (WBC), hemoglobin (HGB), creatine kinase (CK), creatine kinase isoenzyme (CK-MB), brain natriuretic peptide (BNP), serum creatinine (Scr), total bilirubin (TBIL), alanine aminotransferase (ALT) and prothrombin time (PT) of the two groups were compared on the 1st day after admission and the 1st day before discharge.

P>0.05 . However the WBC HGB CK CK MB BNP Scr TBIL ALT PT levels and APACHE SOFA GCS scores of the combined group were better than those of the control group one day before discharge and the difference was statistically significant P<0.05 . The incidence of adverse events in the combination group was lower than that in the control group and the difference was statistically significant $\chi^2=5.307$ P<0.05 TCCD combined with ONSD monitoring can improve the blood biochemical indexes and critical symptoms of patients undergoing cerebral resuscitation after cardiac arrest. This combination can also enhance the prognosis of patients.

. Cerebral resuscitation TCCD Biochemical indexes APACHE

	1	2	3	4	5	6
TCCD						
Transcranial color Doppler ultrasound						
Optic nerve sheath diameter ONSD						
TCCD						
ONS						
M9						
1-5 MHz						
7.5 MHz						
Mindray						
M9						
ONS						
3mm						
ONS						
1.1						
2023						
100						
1						
TCCD						
ONS						
12						
ONS						
1.3						
5 mL						
3 500r/min						
1						
12 cm						
1						
10min						
- 80						
55.72±8.37						
59						
41						
34						
24						
55.48±						
8.29						
23						
19						
40						
22						
60						
37						
22						
21						
20						
P>0.05						
6						
<15 min						
time						
PT						
Creatine kinase CK						
BS 800						
White blood cell count WBC						
Hemo						
globin HGB						
Total bilirubin TBIL						
Alanine aminotransferase ALT						
ACL 7000						
Prothrombin						

	Creatine kinase isoenzyme CK MB	1.6					
	Brain natriuretic peptide BNP				SPSS 22.0		
	Serum creatinine Scr					-	
				t			
1.4				t	n %	²	P<0.05
	1	1					
	APACHE	⁷		2			
	APACHE	0-71		2.1			
			Sequential		1	WBC HGB CK CK MB	
	organ failure assessment scores SOFA ⁸			BNP Scr TBIL ALT PT			
				P>0.05	1	WBC HGB CK	
		6		CK MB BNP Scr TBIL ALT PT			
	0-4	0-24			P<0.05	1	
				2.2			
			Glasgow coma score				
	GCS ⁷	GCS	3-15		1	APACHE SOFA GCS	
	12	8				P>0.05	
				1	APACHE	SOFA GCS	
1.5						P<0.05	2
	3			2.3			
						P<0.05	3
	1						

Table 1 Comparison of blood indexes in patients with cerebral resuscitation after cardiac arrest by different monitoring methods

		n=100	n=100	t	P
WBC $\times 10^9/L$	1	16.53±4.74	16.77±4.82	0.355	0.723
	1	19.84±4.97 ^a	18.19±4.93 ^a	2.357	0.019
HGB g/L	1	120.32±9.67	119.18±9.59	0.837	0.403
	1	126.48±9.81 ^a	123.09±9.72 ^a	2.454	0.015
CK U/L	1	626.79±48.37	621.71±48.19	0.744	0.457
	1	412.58±43.36 ^a	483.34±44.63 ^a	11.371	<0.001
CK MB U/L	1	93.46±7.74	92.97±7.68	0.449	0.653
	1	64.37±6.82 ^a	72.48±7.12 ^a	8.225	<0.001
BNP ng/L	1	739.46±51.47	742.61±52.13	0.430	0.667
	1	387.29±45.72 ^a	433.52±46.68 ^a	7.075	<0.001
Scr $\mu\text{mol/L}$	1	298.56±33.49	302.89±34.41	0.901	0.368
	1	172.46±26.63 ^a	207.67±29.74 ^a	8.820	<0.001
TBIL $\mu\text{mol/L}$	1	114.84±13.86	115.48±14.03	0.324	0.746
	1	69.24±11.79 ^a	79.93±12.63 ^a	6.187	<0.001
ALT U/L	1	142.29±16.36	141.44±16.21	0.369	0.712
	1	86.68±12.24 ^a	99.58±13.37 ^a	7.117	<0.001
PT s	1	18.92±3.76	18.92±3.76	0.267	0.789
	1	16.07±3.02 ^a	17.74±3.43 ^a	3.654	<0.001

^aP<0.05

2

Table 2 Comparison of symptom scores in patients with cerebral resuscitation after cardiac arrest by different monitoring methods Scores, -

	n	APACHE		SOFA		GCS	
		1	1	1	1	1	1
	100	33.42±4.97	16.73±3.98 ^a	10.49±2.42	6.18±1.57 ^a	8.83±2.16	13.86±2.92 ^a
	100	33.83±5.14	21.19±4.26 ^a	10.68±2.52	7.76±1.69 ^a	8.76±2.17	11.79±2.63 ^a
t		0.573	7.650	0.544	6.849	0.226	5.267
P		0.567	<0.001	0.587	<0.001	0.821	<0.001
	1	^a P<0.05					

3

n %

Table 3 Comparison of prognosis of patients with cerebral resuscitation after cardiac arrest by different monitoring methods

n %

	n								
	100	5 5.00	4 4.00	3 3.00	3 3.00	2 2.00	2 2.00	4 4.00	23 23.00
	100	8 8.00	6 6.00	4 4.00	4 4.00	4 4.00	3 3.00	9 9.00	38 38.00
									5.307
P									0.021

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ONSD

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percentage of peripheral blood CD8⁺ in 2 groups were lower than before treatment and the combined group was lower than the conventional group with statistical significance $P < 0.05$. The levels of serum IFN- γ , percentages of peripheral blood CD3⁺, CD4⁺ and CD4⁺/CD8⁺, forced expiratory volume (FEV1), forced vital capacity (FVC) and FEV1/FVC increased compared to before treatment with the combined group showing higher levels than the conventional group all with statistical significance $P < 0.05$. Jianpi Runfei pills could improve the levels of serum CAR, VEGF, IFN- γ and IL-17, enhance immune function and lung function in patients with pulmonary tuberculosis. They also promote the improvement of clinical symptoms and signs in patients, showing a positive therapeutic effect.

Tuberculosis Spleen invigorating lung pill CAR VEGF IFN IL 17

Group	Sample Size (n)	Age (years)	Gender (M/F)	Disease Duration (years)	Clinical Parameters				P-value
					CD3 ⁺ (%)	CD4 ⁺ (%)	CD4 ⁺ /CD8 ⁺ (%)	IFN- γ (pg/mL)	
1	30	31-69	17/13	1.1	1.1	1.1	1.1	1.1	1.3
2	30	31-69	17/13	1.1	1.1	1.1	1.1	1.1	1.3
3	30	31-69	17/13	1.1	1.1	1.1	1.1	1.1	1.3
4	30	31-69	17/13	1.1	1.1	1.1	1.1	1.1	1.3
5	30	31-69	17/13	1.1	1.1	1.1	1.1	1.1	1.3

Parameter	2020		2023		P-value
	n	Mean ± SD	n	Mean ± SD	
carotene CAR	1	51.27 ± 4.73	4	50.85 ± 4.85	P > 0.05
vascular endothelial growth factor VEGF	1	51.27 ± 4.73	4	50.85 ± 4.85	P > 0.05
interferon IFN- γ	1	51.27 ± 4.73	4	50.85 ± 4.85	P > 0.05
interleukin IL-17	1	51.27 ± 4.73	4	50.85 ± 4.85	P > 0.05

Parameter	2020		2023		P-value
	n	Mean ± SD	n	Mean ± SD	
carotene CAR	1	51.27 ± 4.73	4	50.85 ± 4.85	P > 0.05
vascular endothelial growth factor VEGF	1	51.27 ± 4.73	4	50.85 ± 4.85	P > 0.05
interferon IFN- γ	1	51.27 ± 4.73	4	50.85 ± 4.85	P > 0.05
interleukin IL-17	1	51.27 ± 4.73	4	50.85 ± 4.85	P > 0.05

Parameter	2020		2023		P-value
	n	Mean ± SD	n	Mean ± SD	
carotene CAR	1	51.27 ± 4.73	4	50.85 ± 4.85	P > 0.05
vascular endothelial growth factor VEGF	1	51.27 ± 4.73	4	50.85 ± 4.85	P > 0.05
interferon IFN- γ	1	51.27 ± 4.73	4	50.85 ± 4.85	P > 0.05
interleukin IL-17	1	51.27 ± 4.73	4	50.85 ± 4.85	P > 0.05

CAR VEGF IFN IL 17
 1.4.4 90d CytoFLEX
 CD3⁺ CD4⁺ CD8⁺ CD4⁺/
 CD8⁺ 1.4.3
 1.4.5

1 n %
 Table 1 Comparison of clinical efficacy between the two groups n %

n		n %	
60	28 46.67	24 40.00	8 13.33
60	32 53.33	25 43.33	2 3.33
			58 96.67
			3.927
P			0.048

90d
 CHESTAC 8800
 Forced expiratory volume FEV1
 forced vital capacity FVC FEV1/FVC
 1.5 SPSS 23.0 U Wj b [%H

2 - d
 Table 2 Comparison of improvement of clinical symptoms and signs between the two groups - d

n		3	
60	29.74±5.94	31.29±6.38	24.39±4.32
60	18.77±4.18	22.61±4.97	15.47±3.28
t	11.699	8.314	12.738
P	<0.001	<0.001	<0.001
			19.48±4.83
			13.29±3.72
			7.865
			<0.001
			75.94±8.38
			61.22±6.39
			10.820
			<0.001
			43.29±6.37
			34.51±5.30
			8.207
			<0.001

3 -
 Table 3 Comparison of serum cytokine levels between the two groups -

n		CAR μmol/L	VEGF pg/mL	IFN pg/mL	IL 17 pg/mL
60	1.97±0.32	388.50±16.54	5.24±0.73	43.69±7.30	
60	1.93±0.29	389.84±19.48	5.47±0.81	44.21±6.94	
t	0.717	0.406	1.634	0.400	
P	0.475	0.685	0.105	0.690	
90d	60	1.61±0.27 ^a	368.59±14.30 ^a	7.16±0.69 ^a	22.18±5.43 ^a
60	1.38±0.19 ^a	327.95±11.05 ^a	8.07±0.73 ^a	13.02±4.17 ^a	
t	5.396	17.419	7.017	10.363	
P	0.001	0.001	0.001	0.001	

^aP<0.05

4
Table 4 Comparison of immune function between two groups

	n	CD3 ⁺ %	CD4 ⁺ %	CD8 ⁺ %	CD4 ⁺ /CD8 ⁺
	60	46.84±3.52	34.02±3.62	33.20±4.58	1.02±0.35
	60	46.48±3.47	34.88±3.77	33.62±4.73	1.04±0.36
t		0.564	1.275	0.494	0.309
P		0.574	0.205	0.622	0.758
90 d	60	57.41±4.60 ^a	39.68±4.20 ^a	30.60±3.58 ^a	1.30±0.44 ^a
	60	65.02±7.93 ^a	47.51±5.72 ^a	25.17±2.84 ^a	1.89±0.61 ^a
t		6.430	8.547	9.204	6.076
P		<0.001	<0.001	<0.001	<0.001

^aP<0.05

5
Table 5 Comparison of lung function between two groups

	n	FEV1 L	FVC L	FEV1/FVC %
	60	0.94±0.24	1.31±0.32	0.72±0.27
	60	0.93±0.22	1.33±0.35	0.70±0.24
t		0.238	0.327	0.429
P		0.812	0.744	0.669
90 d	60	1.41±0.31 ^a	1.66±0.41 ^a	0.85±0.19 ^a
	60	1.75±0.42 ^a	1.88±0.44 ^a	0.93±0.22 ^a
t		5.045	2.834	2.132
P		<0.001	0.005	0.035

^aP<0.05

3

6

8

90 d

3

10

CAR

11 VEGF

12

IL 17 IFN

13

90 d

CD8⁺

CD3⁺ CD4⁺

FVC

CAR VEGF IL 17

IFN

CD4⁺/CD8⁺ FEV1 FVC FEV1/

FVC

14

15

T

CAR VEGF IFN IL 17

APBSC 38

2019 3 2023 4

G CSF

BMI

CD34+

CD34+

38

89.47% 34/38

1 1 3

100% 6/6

CD34+

CD34+

5.15 2.32 25.5 $\times 10^6/\text{kg}$ 6

7.13 3.08 25.5 $\times 10^6/\text{kg}$

BMI

P>0.05

CD34+

P<0.01

P<0.05

>4

LDL C

P>0.05

LDL C

MM

APBSC

Ac

dian number of collections of 1 1 3 . The median CD34+ cell count among successful collections was 5.15 2.32 25.5 $\times 10^6$ /kg. For the 6 patients who received plerixafor the collection success rate was 100% 6/6 with a median CD34+ cell count of 7.13 3.08 25.5 $\times 10^6$ /kg. No statistically significant differences were observed in CD34+ cell collection quantities among patients of different genders ages BMI levels primary disease remission levels number of chemotherapy cycles and disease subtypes $P > 0.05$. Pio electione leve aes sbes > 0.05. Pils liti

- 80
 CD34+ / <2×10⁶/kg
 CD34+ / 2×10⁶/kg
 5×10⁶/kg
 1.2.3
 CD34+
 Beackman navios
 1.2.4
 BMI
 CD34 +
 NIH CTCAE 4.0
 1.3
 SPSS 22
 qt (R²)
 %S
 "À%S
 L†T†HbX g

1
 Table 1 patients clinical baseline data for the effects of stem cell collection

	n	CD34+	×10 ⁶ /kg	U	P
				126	0.145
	22	8.16	0.36 25.5		
	16	5.9	0.35 11.1		
				138	0.377
<60	24	7.65	0.35 25.5		
60	14	5.8	2.32 13.3		
BMI				176	0.954
<24	17	8.1	0.35 25.5		
24	21	6.7	1.1 16.68		
				190	0.784
PR	18	6.65	0.35 19		
>PR	20	7.65	0.36 25.5		
				110	0.082
4	24	7.65	0.36 25.5		
>4	14	6.75	0.35 13.3		
				146	0.331
IgG	18	6.83	0.35 16.68		
IgG	20	8.49	0.36 25.5		

2
 Table 2 Univariate analysis affecting stem cell collection

	n=25	n=13	t/	P
			6.545	0.015
	16 64.00	6 46.15		
	9 36.00	7 53.85		
			2.455	0.117
<60	18 72.00	6 46.15		
60	7 28.00	7 53.85		
BMI			2.040	0.199
<24	12 48.00	5 38.46		
24	13 52.00	8 61.54		
			2.001	0.203
PR	11 44.00	7 53.85		
>PR	15 56.00	6 46.15		
			13.25	<0.01
4	19 64.00	5 38.46		
>4	6 36.00	8 61.54		
WBC ×10 ⁹ /L	14.02±18.55	5.96±7.38	1.901	0.054
TC mmol/L	4.81±1.25	4.75±1.07	0.158	0.896
TG mmol/L	1.51±0.81	1.45±0.88	0.196	0.877
LDL C mmol/L	1.18±0.31	1.14±0.25	0.354	0.775
HDL C mmol/L	2.82±0.90	2.12±0.79	2.301	0.027

3 Logistic
 Table 3 Binary logistic stepwise regression analysis of prognosis in stem cell harvest

		B	S.E.	Wald	Exp B	95% C.I.	P
	4 =1 >4 =2	- 2.786	1.265	4.852	0.062	0.005-0.736	0.028
LDL C	< =1 LDH C =2	2.95	1.199	6.055	19.109	1.823-200.329	0.014

2.4

11 12

2019

CD34+

G CSF

R²=0.4128 P<0.01

11

2.5

MM

G CSF

~

MM

4

4

%

Table 4 A dverse Reactions and Complications %

	42.11	16/38
	42.11	16/38
	26.31	10/38
	23.68	9/38
	7.89	3/38
	5.26	2/38
	2.63	1/38

CD34+

3

CD34+

MM

auto HSCT

MM

7

G CSF

CD34+
LDL C

8

9

1 SDF 1

SDF 1

4

CXCR4

13 15

CXCR4

10

G CSF

CXCR4

1 SDF 1

CXCR4

SDF 1 CX

CR4

SDF 1

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11 CAR IL 17 CD4- +
J . 2018 13 1 119 123.

12 J .
2023 18 1 148 152.

13 J .
2020 38 10 70 73.

14 J .
2022 31 8 1128 1131.

15 J .
2020 38 8 73 76.

PCT SAA Treg

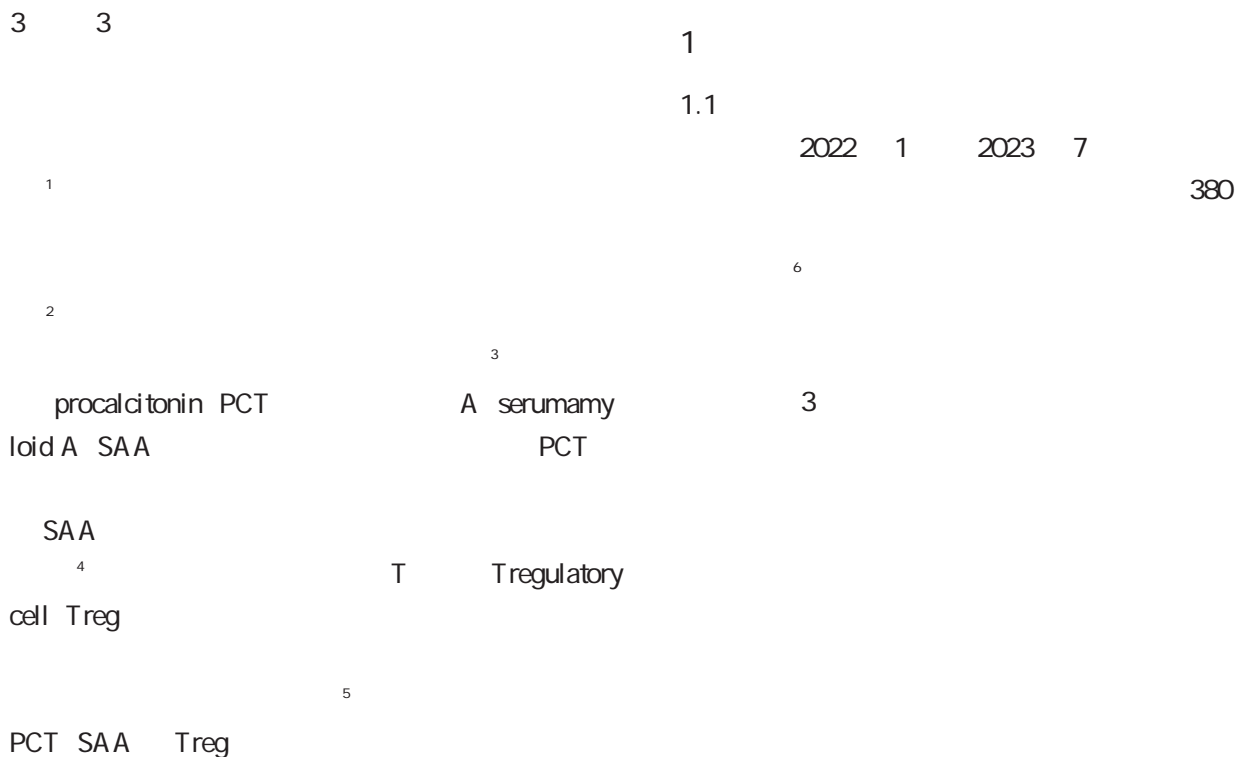
PCT SAA Treg
 2022 1 2023 7
 380
 378 PCT SAA TGF A7A
 ILA 7A Logistic
 PCT SAA TGF ILA 7A PCT SAA TGF ILA 7A
 PCT SAA ILA 7A TGF
 P<0.05 PCT SAA TGF ILA 7A
 Logistic PCT >0.5 μg/L SAA >10 mg/L
 TGF ILA 7A P<0.05 307 73
 PCT SAA TGF ILA 7A P>0.05 7 d PCT
 SAA ILA 7A TGF P<0.05 ROC PCT
 SAA TGF ILA 7A AUC 0.762
 AUC P<0.05 SAA PCT Treg
 PCT SAA T

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To investigate the value of serum procalcitonin (PCT) blood amyloid SAA and regulatory T cell (Treg) associated factor assays in guiding antimicrobial drug therapy for patients with multidrug resistant bacterial infections. 380 cases of multidrug resistant bacterial infections admitted to the First Affiliated Hospital of Hainan Medical College from January 2022 to July 2023 were selected the test group and 378 patients who underwent physical examinations in the hospital during the same period and were normal and healthy were selected as the control group. The study aimed to compare the serum PCT SAA transforming growth factor (TGF) and interleukin A7A (ILA 7A) levels between the two groups to analyze the single multifactorial factors affecting multidrug resistant infections by using multivariate logistic regression model and the serum PCT SAA TGF and ILA 7A levels of the patients in the test group with different efficacies before and after the treatment. The levels of PCT SAA TGF and ILA 7A individually and in parallel to assess the value of antibiotic treatment efficacy were analyzed. The

serum PCT SAA and ILA 7A levels in the test group were higher than those in the control group and the TGF level was lower than that in the control group and the difference was statistically significant $P < 0.05$. Univariate regression analysis showed that serum PCT SAA TGF and ILA 7A levels were single factors affecting multi resistant infections and further rows of multivariate logistic results showed that serum PCT $> 0.5 \mu\text{g/L}$ SAA $> 10 \text{ mg/L}$ reduced TGF and elevated ILA 7A were risk factors affecting multi resistant infections $P < 0.05$. There were 307 cases in the effective group and 73 cases in the ineffective group. There was no statistically significant difference in the comparison of serum PCT SAA TGF and ILA 7A between the two groups before treatment $P > 0.05$ and the levels of PCT SAA and ILA 7A in the effective group were lower than those in the ineffective group and the level of TGF was higher than those in the effective group with statistically significant differences after 7d of treatment $P < 0.05$. The ROC curve showed that the AUC of the four parallel assays of PCT SAA TGF and ILA 7A for assessing the efficacy of antibiotic therapy was 0.762 which was significantly higher than that of the individual assays $P < 0.05$. The levels of serum SAA PCT and Treg cell associated factors play a significant role in guiding the antibiotic treatment of patients with multidrug resistant infections. These indicators have the highest accuracy when tested in parallel providing a certain basis for developing a reasonable treatment plan in clinical practice.

... PCT SAA regulatory T cells Multidrug resistant infections Antibiotics



	n	PCT μg/L	SAA mg/L	Tkm	R	.Uc
	378	0.37±0.04	7.16±2.26	0.2 ± 0.03	0.71	1. ± 0.148 .28
	380	4.69±0.48	118.55±28.43			6 40.001 <
t		174.378	68.887			
P		<0.001	<0.001			

3

Logistic

Table 3 Single multifactorial multivariate logistic regression analyses affecting multi drug resistant infections

			OR	95% CI	P	OR	95% CI	P
PCT	X ₁	<0.5 μg/L=0 >0.5 μg/L=1	2.016	1.152-3.487	<0.001	1.849	1.211-2.824	0.002
SAA	X ₂	<10 mg/L=0 >10 mg/L=1	2.341	1.107-4.911	0.019	1.554	1.081-2.233	<0.001
TGF	X ₃		1.596	1.223-2.084	0.015	1.736	1.137-2.652	<0.001
ILA 7A	X ₄		4.414	1.192-14.376	0.009	1.606	1.185-2.176	<0.001

sLOX 1 SREBP 1 sST2

1 1 2 2

CHD 1 sLOX 1

2 sST2 1 SREBP 1

2020 4 2022 4 CHD 156 CHD

SAP 51 UAP 63 AMI 42

50 sLOX 1 sST2 SREBP 1 QRS

CHD sLOX 1 sST2 SREBP 1 QRS Gensini

sLOX 1 sST2 SREBP 1 QRS sLOX 1 sST2 SREBP 1

QRS Spesman CHD sLOX 1 sST2 SREBP 1

QRS SAP UAP AMI sLOX 1 sST2 SREBP 1 QRS

AMI >UAP >SAP > P<0.05 0-20 20-40 40

sLOX 1 sST2 SREBP 1 QRS 40 >20-40 >0-20

P<0.05 sLOX 1 sST2 SREBP 1 QRS

> > P<0.05 CHD sLOX 1 sST2 SREBP 1

Gensini QRS P<0.05 CHD sLOX 1 SREBP 1

sST2 CHD

1 1

2

SREBP-1

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To explore the changes of serum soluble lectin like oxidised low density lipoprotein receptor 1 sLOX 1 sterol regulatory element binding protein 1 SREBP 1 and soluble growth stimulation expressed gene 2 sST2 in patients with coronary heart disease CHD and their relationship with the severity of coronary lesions. A total of 156 patients with CHD treated at Inner Mongolia Autonomous Region People's Hospital were enrolled in the CHD group between April 2020 and April 2022. This group included 51 cases of stable angina pectoris SAP group 63 cases of unstable angina pectoris UAP and 42 cases of acute myocardial infarction AMI. Additionally 50 healthy controls were enrolled during the same period as the healthy group. The levels of sLOX 1 sST2 and SREBP 1 and QRS wave duration were detected in all subjects. These indexes were compared between healthy controls and CHD patients and among patients with

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1.

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2.

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E-mail nmgmjy2005ydy@163.com

different Gensini scores and number of lesions were compared. The relationship between sLOX 1, sST2, SREBP 1, the severity of coronary lesions, and QRS wave duration was analyzed using the Spearman correlation coefficient. There were significant differences in sLOX 1, sST2, SREBP 1 levels and QRS wave duration among the SAP group, UAP group, AMI group, and healthy group ($P < 0.05$). The levels of sLOX 1, sST2, and SREBP 1 and QRS wave duration were gradually decreased in AMI group, UAP group, SAP group, and healthy group, and the differences were statistically significant ($P < 0.05$). There were significant differences in sLOX 1, sST2, SREBP 1 levels and QRS wave duration among 0-20 point group, 20-40 point group, and >40 point group ($P < 0.05$). The levels of sLOX 1, sST2, and SREBP 1 and QRS wave duration were gradually decreased in >40 point group, 20-40 point group, and 0-20 point group, and the differences were statistically significant ($P < 0.05$). There were significant differences in sLOX 1, sST2, SREBP 1 levels and QRS wave duration among triple vessel group, double vessel group, and single vessel group ($P < 0.05$). The levels of sLOX 1, sST2, and SREBP 1 and QRS wave duration were gradually decreased in triple vessel group, double vessel group, and single vessel group, and the differences were statistically significant ($P < 0.05$). The levels of sLOX 1, SREBP 1, and sST2 in CHD patients are higher than those in healthy individuals, and they are correlated with the severity of coronary lesions. These markers can be applied as targets for the clinical prevention and treatment of CHD.

CHD, sLOX 1, SREBP 1, sST2, Coronary lesion

coronary >50%

heart disease CHD

2023 CHD 2

1 CHD 3

2 4

1 Lectin like oxidized low density lipoprotein receptor 1 LOX 1 stable angina pectoris SAP 51 Unstable angina acute myocardial infarction UAP 63

oxidized low density lipoprotein ox LDL CHD dial infarction AMI 42

sLOX 1 LOX 1 CHD 63 52

1 Sterol regulatory element binding protein 1 SREBP 1 41 50

17p11.2 $P > 0.05$ 1

2

soluble growth stimulation expressed gene 2 1.2

sST2 1.2.1 sLOX 1 sST2

33 Interleukin 33 IL 33 3 mL

sST2/IL 33 12 cm 3 500 r/min 5 min

CHD sLOX 1 SREBP 1 -80 ELISA

sST2 sLOX 1 sST2 R&D

1.2.2 SREBP 1

5 mL

1 12 cm 3 500 r/min 10 min

1.1 TRIZOL RNA

2020 4 2022 4 Takara Japan 1 μ g RNA

CHD 156 CHD cDNA cDNA PCR

CHD³ F TGCATTTTCTGACACGCTTC

Table 1 Comparison of general data between the two groups

	n	61.03±7.29	29 58.00	21 42.00	16 32.00	23 46.00	11 22.00	24.16±5.02
CHD	156	60.54±7.18	93 59.00	63 40.38	52 33.33	69 44.32	35 22.44	23.49±5.37
t/		0.418		0.041		8.830		0.780
P		0.676		0.840		0.183		0.437

R CCAAGCTGTACAGGCTCTCC PCR
 95 30s 95 10s 60 35s 70 60s
 40 SREBP 1 2^t
 1.2.3 Gensini
 CHD

AMI >UAP >SAP >
 P<0.05 2
 2 CHD sLOX 1 sST2 SREBP 1 QRS

Table 2 Comparison of sLOX 1 sST2 SREBP 1 and QRS wave duration between CHD group and healthy group

	n	sLOX 1 ng/L	SREBP 1	sST2 ng/mL	QRS ms
	50	85.18±16.27	1.12±0.15	15.36±3.01	81.91±10.53
SAP	51	146.92±27.13 ^a	1.29±0.23 ^a	20.18±4.26 ^a	91.68±9.54 ^a
UAP	63	183.44±31.65 ^{ab}	1.42±0.34 ^{ab}	32.55±6.17 ^{ab}	105.32±11.67 ^{ab}
AMI	42	221.35±36.57 ^{abc}	1.59±0.43 ^{abc}	45.29±7.08 ^{abc}	123.45±12.04 ^{abc}
F		194.155	20.228	291.500	123.697
P		<0.001	<0.001	<0.001	<0.001

1 <25% 2 26%~50% 4
 51%~75% 8 76%~90% 16
 91%~99% 100% 32 Gensini⁷
 Gensini 0~20

56 20~40 58 40 42
 1.2.4 QRS
 12 25
 mm/s QRS 3

^aP<0.05 SAP ^bP<0.05 UAP
^cP<0.05

1.3 SPSS 20.0
 n %² Pearson
 sLOX 1 SREBP 1 sST2 Gensini
 QRS Spesman
 sLOX 1 SREBP 1 sST2
 P<0.05

2.2 Gensini sLOX 1 sST2
 SREBP 1 QRS
 sLOX 1 sST2 SREBP 1 QRS
 40 >20~40 >0~20
 P<0.05 3

2
 2.1 CHD sLOX 1 sST2 SREBP 1
 QRS
 sLOX 1 sST2 SREBP 1 QRS

2.3 sLOX 1 sST2
 SREBP 1
 sLOX 1 sST2 SREBP 1 QRS
 > >
 P<0.05 4
 2.4 sLOX 1 sST2 SREBP 1 Gen
 sini
 CHD sLOX 1 sST2 SREBP 1

Table 3 Comparison of sLOX 1 sST2 SREBP 1 and QRS wave duration in patients with different Gensini scores

Gensini	n	sLOX 1 ng/L	SREBP 1	sST2 ng/mL	QRS ms
0~20	56	153.26±25.71	1.28±0.19	19.01±4.06	90.43±10.15
20~40	58	188.19±31.23 ^a	1.43±0.26 ^a	33.27±5.29 ^a	107.56±12.41 ^a
40	42	210.68±35.42 ^{ab}	1.61±0.38 ^{ab}	47.33±6.87 ^{ab}	123.65±13.09 ^{ab}
F		29.333	17.020	334.720	95.458
P		<0.001	<0.001	<0.001	<0.001

0~20 ^aP<0.05 20~40 ^bP<0.05

4 sLOX 1 sST2 SREBP 1
 Table 4 Comparison of sLOX 1 sST2 SREBP 1 and QRS wave duration in patients with different number of coronary lesions

	n	sLOX 1 ng/L	SREBP 1	sST2 ng/mL	QRS ms
	63	147.15±24.82	1.30±0.21	21.65±4.07	88.74±11.65
	52	189.27±33.19 ^a	1.41±0.32 ^a	33.18±6.13 ^a	106.25±13.49 ^a
	41	225.22±37.46 ^{ad}	1.61±0.41 ^{ab}	46.16±7.22 ^{ab}	131.22±10.53 ^{ab}
F		79.210	12.464	228.822	154.994
P		<0.001	<0.001	<0.001	<0.001

^aP<0.05 ^bP<0.05

Gensini QRS
 P<0.05 5
 5 sLOX 1 sST2 SREBP 1 Gensini
 QRS
 Table 5 Correlation between changes in sLOX 1 sST2 and SREBP 1 and Gensini score number of coronary lesions QRS wave duration

	Gensini		QRS	
sLOX 1	0.439	<0.001	0.539	<0.001
sST2	0.491	<0.001	0.618	<0.001

3

CHD
 5 LOX 1 Gensini QRS
 ox LDL
 sLOX 1 CHD 6 SREBP 1 LOX 1
 17 mRNA 4154bp
 7 sST2 IL 1 ST2 13 sST2 ST2L IL 33
 ST2L/IL 33
 CHD IL 33 ApoE
 CHD QRS 8 -/- sST2
 15 SREBP 1 A
 sLOX 1 sST2 SREBP 1 CHD SREBP 1 CHD
 9 CHD 16

IL 6 CRP Hcy ACA

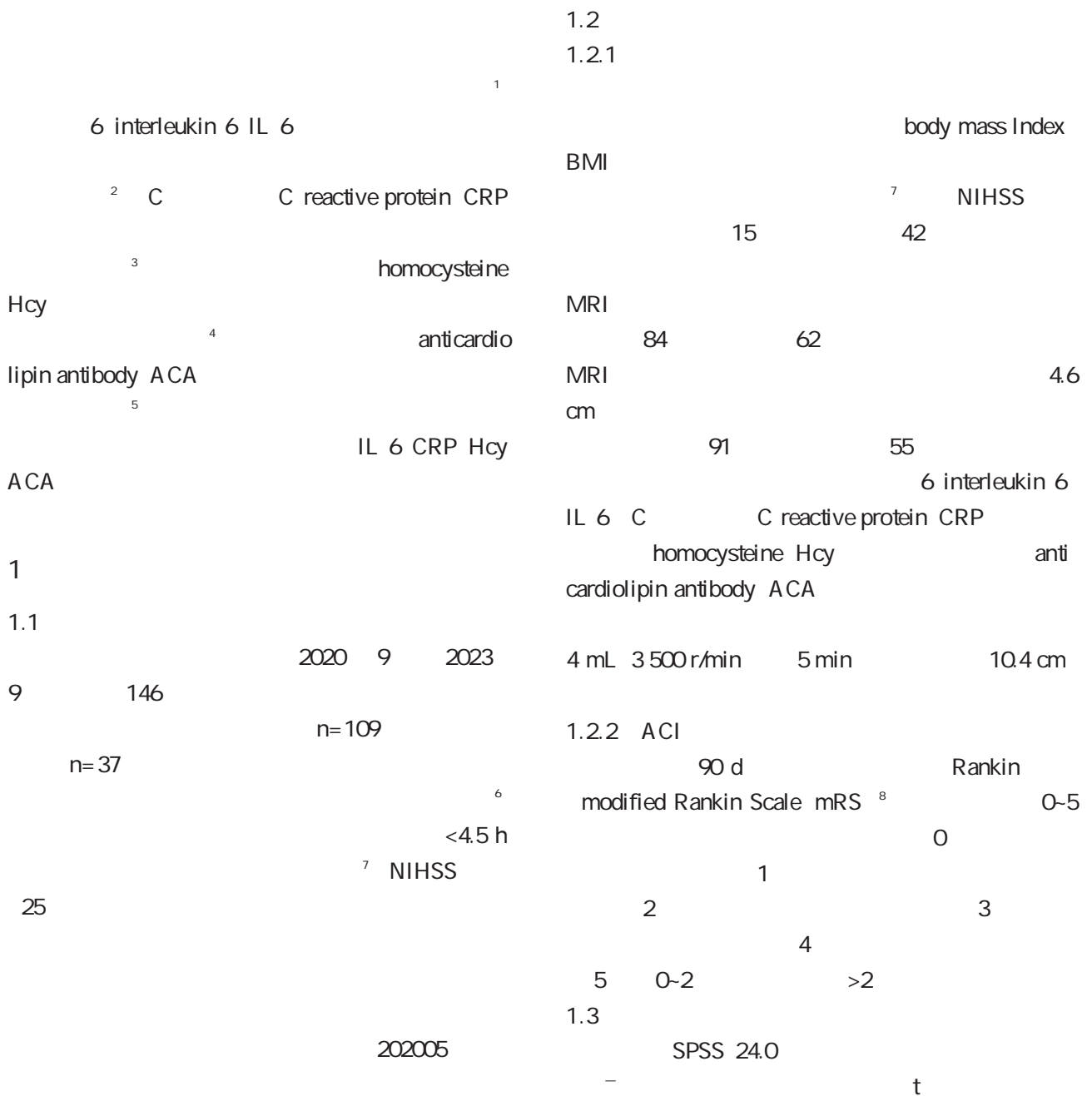
6 IL 6 C CRP Hcy
 2020 9 2023 9
 Rankin mRS
 146
 n=109 n=37
 NIHSS 6 IL 6 C CRP Hcy
 ACA Logistic Pearson
 ROC IL 6 CRP Hcy
 ACA NIHSS IL 6 CRP Hcy ACA
 t=2.651 6.507 10.217 7.946 8.571 P<0.05 NIHSS IL 6 CRP
 Hcy ACA P<0.05 IL 6 CRP Hcy ACA
 t=5.314 8.547 5.219 9.087 P<0.05 IL 6 CRP Hcy ACA
 t=10.514 21.033 15.658 11.723 P<0.05 NIHSS IL 6 CRP Hcy ACA
 mRS r=0.523 0.584 0.572 0.545 0.521 P<0.05 NIHSS IL 6 CRP
 Hcy ACA AUC 0.622 0.759 0.894 0.845 0.892
 NIHSS IL 6 CRP Hcy ACA P<0.05 IL 6
 CRP Hcy ACA
 6 C

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To analyze the relationship of interleukin 6 IL 6 C reactive protein CRP homocysteine Hcy and anticardiolipin antibody ACA with prognosis of patients with acute cerebral infarction ACI. The clinical data of 146 patients with ACI who received intravenous thrombolytic therapy at Qinhuangdao Workers Hospital from September 2020 to September 2023 were retrospectively analyzed. The modified rankin score mRS was used to evaluate the prognosis of patients who were then divided into a good prognosis group n=109 and a poor prognosis group n=37. General data neurological deficit NIHSS and laboratory indicators including creatinine Cr total bilirubin TBil albumin ALB red blood cell count RBC white blood cell count WBC platelet count PLT IL 6 CRP Hcy and ACA were compared between the two groups. A binary logistic regression model was used to analyze the influencing factors of prognosis in patients with ACI. The Pearson correlation coefficient was performed to analyze the correlation between laboratory indicators and the degree of prognosis in patients with ACI. A receiver operating characteristic ROC curve was drawn to analyze the predictive value of IL 6 CRP Hcy and ACA on the prognosis of patients. The NIHSS score IL 6 CRP Hcy and ACA levels were

lower in the poor prognosis group $t=2.651$ 6.507 10.217 7.946 8.571 $P<0.05$. Regression analysis showed that high NIHSS score and elevated levels of IL-6, CRP, Hcy, and ACA were risk factors for poor prognosis in patients with ACI $P<0.05$. The levels of IL-6, CRP, Hcy, and ACA were lower in the anterior circulation group compared to the posterior circulation group $t=5.314$ 8.547 5.219 9.087 $P<0.05$ and lower in the small area infarction group than in the large area infarction group $t=10.514$ 21.033 15.658 11.723 $P<0.05$. NIHSS score, IL-6, CRP, Hcy, and ACA were positively correlated with mRS score $r=0.523$ 0.584 0.572 0.545 0.521 $P<0.05$. The areas under the curves (AUCs) of NIHSS score, IL-6, CRP, Hcy, and ACA in predicting the prognosis of acute cerebral infarction were 0.622, 0.759, 0.894, 0.845, and 0.892 respectively. IL-6, CRP, Hcy, and ACA had higher predictive efficiency compared to NIHSS score $P<0.05$. The poor prognosis of patients with acute cerebral infarction is related to elevated levels of IL-6, CRP, Hcy, and ACA. Changes in these indicators can predict the prognosis of patients.

IL-6, CRP, Hcy, ACA Prognosis of acute cerebral infarction



n %
 2
 Logistic
 Pearson
 mRS
 ROC
 P<0.05
 2
 2.1
 P>0.05
 NIHSS
 P<0.05
 1
 n %
 1

Table 1 Comparison of general data between 2 groups

	n	n=109	n=37	t	P
				1.902	0.168
	87	63 72.71	24 27.59		
	59	46 77.97	13 22.03		
		64.84±5.77	65.47±6.33	0.560	0.576
BMI kg/m ²		23.74±4.33	22.86±3.58	1.113	0.268
				0.064	0.969
	17	11 64.71	6 35.29		
	23	14 60.87	9 39.13		
	19	12 63.16	7 36.84		
NIHSS		11.45±2.64	13.02±3.61	2.651	<0.001

2.2
 IL 6 CRP Hcy ACA
 P<0.05
 2
 2

Table 2 Comparison of laboratory indicators between 2 groups

	n=109	n=37	t	P
IL 6 ng/mL	48.74±8.45	60.29±9.18	6.507	<0.001
CRP mg/L	13.15±2.35	18.01±2.14	10.217	<0.001
Hcy μg/mL	17.37±3.37	22.87±3.31	7.946	<0.001
ACA mg/L	0.63±0.18	0.96±0.21	8.571	<0.001

2.3
 =1 =0 NIHSS >15
 =1 15 =0 IL 6 CRP Hcy ACA
 > =1 =0 Logistic
 NIHSS IL 6 CRP Hcy ACA
 P<
 0.05 3

3
 Table 3 Multivariate analysis of prognosis in patients with acute cerebral infarction

	SE	Wald	OR	95% CI	P	
NIHSS	0.974	0.325	8.982	2.649	1.401-5.008	0.003
IL 6 ng/mL	0.886	0.345	6.595	2.425	1.233-4.769	0.011
CRP mg/L	0.947	0.316	8.981	2.578	1.388-4.789	0.003
Hcy μg/mL	0.936	0.359	6.798	2.550	1.262-5.153	0.009
ACA mg/L	0.893	0.319	7.836	2.442	1.307-4.564	0.005

2.4
 IL 6 CRP Hcy ACA
 P<0.05 4

4
 Table 4 Comparison of laboratory indicators among patients with cerebral infarction at different sites of cerebral infarction

	n=84	n=62	t	P
IL 6 ng/mL	51.36±8.21	58.74±8.41	5.314	<0.001
CRP mg/L	14.02±2.14	17.15±2.25	8.547	<0.001
Hcy μg/mL	18.27±3.21	21.06±3.17	5.219	<0.001
ACA mg/L	0.67±0.16	0.92±0.17	9.087	<0.001

2.5
 IL 6 CRP Hcy ACA
 P<0.05 5

5
 Table 5 Comparison of laboratory indicators among cerebral infarction patients with different cerebral infarction areas

	n=91	n=55	t	P
IL 6 ng/mL	45.33±9.12	63.84±12.03	10.514	<0.001
CRP mg/L	11.36±2.05	20.21±3.03	21.033	<0.001
Hcy μg/mL	15.23±3.12	24.66±4.12	15.658	<0.001
ACA mg/L	0.61±0.16	0.98±0.22	11.723	<0.001

2.4 NIHSS IL 6 CRP Hcy ACA
 mRS
 NIHSS IL 6 CRP Hcy ACA
 mRS
 0.523 0.584 0.572 0.545 0.521 P <0.001

2.5 NIHSS
 ROC NIHSS IL 6 CRP
 Hcy ACA AUC

0.622 0.759 0.894 0.845 0.892
 IL 6 CRP Hcy ACA
 0.05 6 1
 6 NIHSS

NIHSS
 P<

13

Hcy
 ACA

Table 6 Predictive efficiency of NIHSS score and laboratory indicators on poor prognosis of patients with acute cerebral infarction

	AUC	95% CI		P
NIHSS	0.622	0.515-0.730	15.81	0.251 0.026
IL 6 ng/mL	0.759	0.673-0.845	50.72	0.515 <0.001
CRP mg/L	0.894	0.835-0.953	16.41	0.639 <0.001
Hcy μg/mL	0.845	0.779-0.910	17.85	0.542 <0.001
ACA mg/L	0.892	0.839-0.944	0.74	0.617 <0.001

Hcy ACA
 Hcy ACA
 Hcy ACA
 Hcy ACA
 Hcy



Figure 1 ROC curve

3
 NIHSS

9 IL 6

CRP

10
 IL 6 CRP
 NIHSS

IL 6 CRP
 U
 P<0.05

NIHSS IL 6 CRP
 NIHSS IL 6 CRP

NIHSS

11 IL 6

12 CRP

17 ROC
 NIHSS IL 6 CRP Hcy ACA
 AUC 0.622 0.759
 0.894 0.845 0.892 NIHSS IL 6
 CRP Hcy ACA IL 6
 CRP Hcy ACA
 CRP Hcy ACA
 IL 6

Sil 2R

IFN TSGF

1 1 2 1 1

Sil 2R IFN LAEC TSGF 2021 3

2023 7 LAEC 106 106

n=53 n=53

1 PD L1 1 PD 1 Sil 2R

IFN TSGF ORR DCR

P<0.05 P>0.05

PD L1 PD 1 Sil 2R TSGF IFN P<

0.05 LAEC

IFN Sil 2R TSGF

Sil 2R IFN TSGF

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To investigate the efficacy of neoadjuvant chemotherapy combined with immunotherapy in the treatment of locally advanced esophageal cancer (LAEC) and its effects on the levels of soluble interleukin 2 receptor (Sil 2R), gamma interferon (IFN), and malignant tumor specific growth factor (TSGF).

A total of 106 LAEC patients treated at Hefei Cancer Hospital, Chinese Academy of Sciences, from March 2021 to July 2023 were selected. They were divided into an experimental group (n=53) and a control group (n=53) using a random number table method. The control group received neoadjuvant chemotherapy while the experimental group received neoadjuvant chemotherapy combined with immunotherapy. The clinical efficacy, adverse reactions, serum levels of programmed death ligand 1 (PD L1), programmed death receptor 1 (PD 1), Sil 2R, IFN, and TSGF were compared before and after treatment in both groups.

The objective response rate (ORR) and disease control rate (DCR) in the experimental group were higher than those in the control group, and the differences were statistically significant (P<0.05). There was no significant difference in the grade of adverse reactions between the two groups (P>0.05). After treatment, serum levels of PD L1, PD 1, Sil 2R, and TSGF in the experimental groups were lower than those in the control

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advanced esophageal cancer LAEC
 Locally
 P>0.05 1
 20210101
 LAEC
 1
 2
 Soluble interleukin 2 receptor Sil 2R
 Interferon IFN
 Malignant tumor specific growth factor TSGF
 LAEC
 Sil 2R IFN TSGF
 1.2
 mg/m² d1 d8
 21 d/1
 75 mg/m² d2-d4
 \$
 260
 1
 1.1
 2021 3 2023 7
 LAEC 106 60.47±6.29 76
 30 24
 54 28 73
 33 23 56
 27 102 4
 2018 3
 6 KFS 70
 53 n=53 106 n=

2 n %
 Table 2 Comparison of clinical efficacy between the two groups n %

	n	CR	PR	SD	PD	ORR	DCR
	53	1 1.89	21 39.62	23 43.40	8 15.09	22 41.51	45 84.91
	53	0 0.00	12 22.64	22 41.51	19 35.85	12 22.64	34 64.15
P						4.289 0.038	5.956 0.015

3 n %
 Table 3 Comparison of clinical efficacy between the two groups n %

	n	~	~	~	~	~	~	~
	53	6 11.32	1 1.89	4 7.55	1 1.89	7 13.21	1 1.89	9 16.98
	53	7 13.21	2 3.77	5 9.43	2 3.77	8 15.09	3 5.66	10 18.87
P		0.391 0.696		0.324 0.746		0.759 0.448		0.592 0.554

4 PD L1 PD 1 n=53
 Table 4 Comparison of serum PD L1 and PD 1 levels between the two groups n=53

	n	PD L1 ng/L	PD 1 ng/L
	53	355.28±38.06	288.79±30.32 ^a
	53	352.93±38.01	311.52±34.02 ^a
t		0.318	3.631
P		0.751	0.000

^aP<0.05

2.4

Sil 2R IFN TSGF
IFN

IFN

Sil 2R_h 2R
P

Sil 2R TSGF

TSGF

2 J . 2021 18 4 137 143 157 163 IFN PTX3

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IL 1 sE cad E2

1 1 1 2 3

IL 1 sE cad E2

2020 4 2023 11

BI RADS BI RADS 4

BC 105 BC BC

BI RADS 3 PCM 95 PCM

IL 1 sE cad E2 ROC

IL 1 sE cad E2 BC IL 1 sE cad E2

V_{max} RI PI PCM $P < 0.05$

$P < 0.05$

$P > 0.05$ IL 1 sE cad E2 BC PCM

AUC 95%CI 0.933 0.863 0.966 94.26% 91.77% IL 1 sE cad

E2 $P < 0.05$ IL 1 sE cad E2 BC PCM

IL 1 E E2

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To investigate the significance of serum IL 1 sE cad and E2 combined with breast ultrasound in the differential diagnosis of plasma cell mastitis and breast cancer. . . . The patients who were initially admitted to the Department of Mammary Glands of Deyang People's Hospital from April 2020 to November 2023 and had not received treatment were selected as the research subjects. Using the BI RADS classification criteria for breast color Doppler ultrasound patients with a BI RADS score of 4 or higher were identified. Based on the postoperative pathological diagnosis of breast cancer BC 105 patients were confirmed to have BC and were categorized as the BC group. At the same time 95 patients with a breast ultrasound BI RADS score of 3 or lower diagnosed with plasma cell mastitis PCM were randomly selected and placed in the PCM group. All subjects underwent a breast ultrasound examination. The levels of serum IL 1 sE cad and E2 the ultrasonographic features and parameters of breast were compared between the two groups. An ROC curve was drawn to analyze the value of serum IL 1 sE cad E2 combined with breast

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ultrasound in the differential diagnosis between BC and PCM. . . . The levels of serum IL 1 sE cad E2 Vmax RI and PI in the BC group were significantly higher than those in the PCM group P<0.05 . There were significant differences in location size edge characteristics microcalcification posterior attenuation lateral acoustic shadow and aspect ratio between the two groups P<0.05 . There was no significant difference in boundary clarity and internal echo status between the two groups P>0.05 . The AUC 95% CI sensitivity and specificity of IL 1 sE cad E2 combined with breast ultrasound in the differential diagnosis of BC and PCM were 0.933 0.863 0.966 94.26% and 91.77% respectively significantly higher than serum IL 1 sE cad E2 breast ultrasound single diagnosis P<0.05 E2 combined with breast ultrasound can significantly improve the accuracy of differential diagnosis between BC and PCM. This is beneficial for early clinical detection and precise treatment and is crucial for optimizing patient care and enhancing treatment outcomes.

. . . . IL 1 sE cad E2 Breast ultrasound Plasma cell mastitis Breast cancer

	Plasma Cell Mastitis	PCM	68	37
Breast Cancer	BC	70	21	6
	PCM	58.15±10.56 kg	48.32±6.40	37
		30		44

IL 1 Interleukin 1 P>0.05
 E cadherin sE cad Soluble BC
 Estradiol E2 BC
 PCM

1
 1.1
 2020 4 2023 11
 3
 4
 IL 1 sE cad E2 1.2
 1.2.1

1
 1.1
 2020 4 2023 11
 BI RADS 5
 BI RADS 4 BC 105 BC Vmax
 BC BI RADS 3 Resistance Index RI
 PCM 95 PCM 1.2.2 Pulse Pressure Index PI
 BC 47.45±6.29 IL 1 sE cad E2
 57.60±10.43 kg 40 46 5 mL 2 h 3 500 r/min
 33 TNM 28 77 10cm 15min

IL 1 sE cad

E2

1.3

SPSS 23.0

Shapiro

Wilc

t

n %

ROC

IL 1 sE cad E2

BC PCM

P<0.05

2

2.1

IL 1 sE cad E2

BC IL 1 sE cad E2

PCM P<0.05 1

1 IL 1 sE cad E2

Table 1 Comparison of serum IL 1 , sE cad and E2 levels between the two groups

	n	IL 1 pg/mL	sE cad ng/mL	E2 pg/mL
BC	105	10.41±4.50	8.86±3.40	205.69±43.63
PCM	95	1.14±0.39	5.32±2.06	95.08±23.64
t		20.005	8.791	21.961
P		<0.001	<0.001	<0.001

2.2

P<0.05

P>0.05 2

2.3

BC V_{max} RI PI PCM

P<0.05 3

2.4

IL 1 sE cad E2

BC

PCM

IL 1 sE cad E2

BC

PCM

AUC 0.933

P<0.05 4 1

3

IL 1

7 IL 1

sE cad

8

9

2

Table 2 Comparison of breast ultrasound morphology between the two groups

	BC n=105	PCM n=95	t/	P
	105 100.00	3 3.16		188.304<0.001
	0 0.00	92 96.84		
cm	2.84±0.85	1.35±0.42	15.460	<0.001
	103 98.09	92 96.84	0.321	0.570
	2 1.91	3 3.16		
	1 0.95	20 21.05		
	46 43.81	0 0.00	65.026	<0.001
	58 55.24	75 78.95		
	99 94.29	90 94.74	0.019	0.888
	6 5.71	5 5.26		
	101 96.19	6 6.31	161.938	<0.001
	4 3.81	89 93.69		
	104 99.05	3 3.16	184.340	<0.001
	1 0.95	92 96.84		
	105 100.00	2 2.11	173.110	<0.001
	0 0.00	93 97.89		
	1.85±0.53	1.00±0.30	13.761	<0.001

3

Table 3 Comparison of breast ultrasound parameters between the two groups

	n	V _{max} m/s	RI	PI
BC	105	17.69±4.64	0.88±0.45	1.78±0.69
PCM	95	8.72±2.40	0.51±0.33	1.02±0.41
t		16.904	6.572	9.344
P		<0.001	<0.001	<0.001

4 IL 1 sE cad E2 BC PCM

Table 4 Value of serum IL 1 , sE cad, and E2 combined with breast ultrasound in the differential diagnosis of BC and PCM

	AUC	SE	95% CI	%	%
IL 1	0.683	0.068	0.439-0.748	70.40	69.66
sE cad	0.647	0.077	0.422-0.723	65.93	64.53
E2	0.659	0.072	0.431-0.735	67.80	65.70
V _{max}	0.711	0.064	0.705-0.794	72.39	71.56
RI	0.742	0.062	0.722-0.837	74.57	72.72
PI	0.769	0.058	0.742-0.852	76.86	73.20
IL 1 +sE cad+ E2+	0.933	0.045	0.863-0.966	94.26	91.77

sE cad

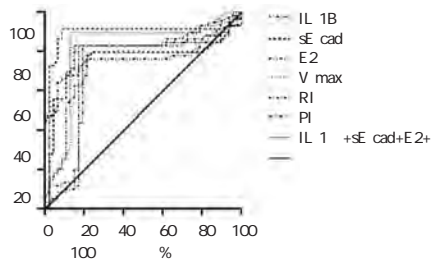
10 E2

E2

11 13

BC

BC IL 1 sE cad E2



PCM IL 1 sE cad E2
 BC
 PCM
 BC IL 1
 IL 1
 BC
 sE cad
 BC E2

#OU•#\$

SALL4 GS HSP70

HSP70 4 SALL4 GS 70
2020 7 2023 6
153 Edmondson ~ 58 ~ 95
153 >3 cm 141
SALL4 GS HSP70 SALL4 GS HSP70
SALL4 GS HSP70 > >
P<0.05 ~ SALL4 GS HSP70
~ P<0.05 ROC SALL4+GS+HSP70
95.10% 78.60% AUC=0.838 95%CI 0.755-0.922 SALL4 GS
HSP70 P<0.05 SALL4 GS HSP70
SALL4 GS HSP70

YIN Tingli JING Jianjun WANG Ying ZHANG Ying WANG Yin
Department of Oncology

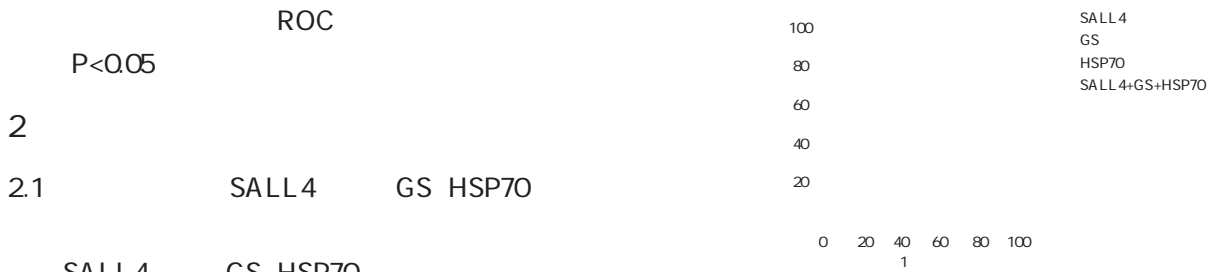


Figure 1 ROC curve of SALL4 gene GS and HSP70 for early diagnosis of liver cancer

2.2 SALL4 GS HSP70
 P < 0.05
 III IV SALL4 GS HSP70

2.3 SALL4 GS HSP70
 P < 0.05
 ROC SALL4+GS+HSP70

95.10% 78.60% AUC=0.838 95% CI 0.755-0.922
 SALL4 GS HSP70 P < 0.05

Table 3 Value of SALL4 gene GS and HSP70 in early diagnosis of liver cancer

	AUC	95% CI	%	%
SALL4	0.737	0.625-0.850	69.24	58.53
GS	0.813	0.721-0.905	71.82	61.62
HSP70	0.701	0.586-0.816	63.33	51.85
SALL4+GS+HSP70	0.838	0.755-0.922	95.10	78.60

Table 1 Comparison of expression of SALL4 gene GS and HSP70 in different tissues n %

n	SALL4		GS		HSP70							
	n	%	n	%	n	%						
153	11	7.19	142	92.81	0	0.00	19	12.42	134	87.58		
141	29	13.47	112	79.43	35	24.82	106	75.18	39	27.65	102	72.34
153	126	82.35	27	17.65	105	68.63	48	31.37	110	71.89	43	28.12
P	<0.001		<0.001		<0.001		<0.001		<0.001		<0.001	

Table 2 Comparison of expression of SALL4 gene GS and HSP70 in patients with benign liver disease and liver cancer n %

n	SALL4		GS		HSP70								
	n	%	n	%	n	%							
~	52	31	59.61	21	40.39	28	53.85	24	46.15	21	40.38	31	59.62
~	101	95	94.05	6	5.95	77	76.23	24	23.77	89	88.12	12	11.88
P	<0.001		<0.001		0.004		0.004		<0.001		<0.001		

				11	3	. SCCAg EGFR NSE
	GS			GS		J .
		12			2022 14 9 1511 1514 1518	
12	GS				4	. GPC3 HSP70 SALL4
					AFP	J .
					2020 42 8 780 782	
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						J .
					2010 24 12 1213 1214.	
HSP70					6	.
						J .
						2002 29 1 83 83.
				13	7	.
	HSP70					J .
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					8	. STZ
						J .
					2021 18 3 205 210.	
					9	. MIF sICAM 1 visfatin AFP PHC
						J .
						2020 4 2 104 107.
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						J .
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					12	. T
						Meta J .
						2018 19
					1	12 18
					13	.
						J .
						2023 54 6 1208 1218.
					14	.
						J .
						2020 19 8 898 902
1					15	Moudi B Heidari Z Mahmoudzadeh Sagheb H Alavian SM et al. Concomitant use of heat shock protein 70 glutamine synthetase and glypican 3 is useful in diagnosis of HBV related hepatocellular carcinoma with higher specificity and sensitivity J . Eur J Histochem 2018 62 1 2859.
						J .
						2022 19 06 133 136.
					2	. SALL4
						J .
						2019 35 010 2320 2323.

1532

13

2022 26 2 201 204.

15

J . 2021 36 21 5106 5108

OPN IL 1 J .

14

Logistic

2021 36 3 433 436.

J .

1 1 1 1 2

2020 5 2023 5 103

MCCB <50 MCCB 50

Logistic ROC TC TG

HDL C LDL C

103 64.08% 66/103

SAPS SANS BPRS TC TG HDL C LDL C

t/ ² =3.390 8.501 9.398 5.467 4.307 3.826 3.767 7.502 4.195 6.295 P<0.05

Logistic BPRS SANS TC TG HDL C

LDL C P<Q.05 ROC

TC TG HDL C LDL C AUC >0.65

BPRS SANS TC

TG HDL C LDL C

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To explore the risk factors of cognitive impairment in patients with schizophrenia and to analyze the predictive value of serological indexes. . . . A total of 103 patients with schizophrenia were admitted to Anqing Sixth People's Hospital between May 2020 and May 2023. They were divided into two groups the impairment group MCCB score <50 points and the non impairment group MCCB score ≥ 50 points . General data from both groups were compared. Risk factors for cognitive impairment were analyzed using multivariate logistic regression analysis. The predictive value of serum total cholesterol TC triglyceride TG high density lipoprotein cholesterol HDL C and low density lipoprotein cholesterol LDL C for cognitive impairment was analyzed using ROC curves. . . . In a study of 103 patients with schizophrenia the incidence of cognitive impairment was 64.08% 66/103 . There were significant differences in age education years hospitalization frequency scores of SAPS SANS and BPRS as well as levels of TC TG HDL C and LDL C between the impairment group and the non impairment group

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t/ ²=3.390 8.501 9.398 5.467 4.307 3.826 3.767 7.502 4.195 6.295 P<0.05 . Multivariate logistic regression analysis showed that increased age BPRS score SANS score TC and TG decreased HDL C and increased LDL C were independent risk factors for cognitive impairment in schizophrenia patients P<0.05 . The results of ROC curve analysis showed that the area under the curve AUC values of serum TC TG HDL C and LDL C for predicting cognitive impairment were all >0.65 demonstrating good predictive efficiency.

The incidence of cognitive impairment is high in schizophrenia patients. Increased age BPRS score SANS score TC and TG decreased HDL C and increased LDL C are independent risk factors for cognitive impairment. Clinically detection of blood lipid levels can evaluate the occurrence of cognitive impairment.

Schizophrenia Cognitive function Mental symptom Blood lipid

computerized cognitive remediation therapy
MCCB ⁶ 7

1
2

100
MCCB <50
MCCB 50
1.2.3

3
4

Scale for the Assessment of Positive Sym-
toms SAPS ⁷
34 0
~5
1.2.4

1
1.1

2020 5 2023 5
103
18

Scale for the
assessment of negative symptoms SANS ⁸
24 0-120
1.2.5

1.2
1.2.1

Brief Psy-
chiatric Rating Scale BPRS ⁹
5 18 29-126
1.2.6

1.2.2

MATRICES

5 mL
3 500 r/min 10 cm
10 min
Total cholesterol TC
Triglyceride TG High

density lipoprotein cholesterol HDL C
 Low density lipoprotein cholesterol
 LDL C Cobas c501

1

Table 1 Comparison of general data between impairment group and non impairment group

	n=66	n=37	t/	P
	55 83.33	29 78.38	0.387	0.534
	11 16.67	8 21.62		
	49.25±12.21	41.37±10.28	3.390	0.001
	8.85±1.05	10.88±1.34	8.501	<0.001
	45 68.18	20 54.05	0.033	0.154
	21 31.82	17 45.95		
	3.21±1.05	1.50±0.46	9.398	<0.001
SAPS	3.21±0.46	2.72±0.39	5.467	<0.001
SANS	85.76±9.34	77.68±8.75	4.307	<0.001
BPRS	40.32±5.19	35.67±6.14	3.826	<0.001
TC mmol/L	5.06±1.02	4.32±0.83	3.767	<0.001
TG mmol/L	2.81±0.53	2.04±0.44	7.502	<0.001
HDL C mmol/L	1.27±0.31	1.56±0.38	4.195	<0.001
LDL C mmol/L	3.45±0.49	2.83±0.46	6.295	<0.001

1.3
 SPSS 22.0
 Logistic
 ROC
 TC TG HDL C LDL C
 P<0.05

2
 2.1
 66 64.08%
 SAPS SANS BPRS TC TG
 HDL C LDL C
 P<0.05 1

2

Table 2 Multivariate analysis on influencing factors of cognitive impairment in patients with schizophrenia

	SE	Wald	OR	95% CI	P
	1.128 0.487	5.365	3.089	1.189 8.025	0.021
	0.874 0.484	3.261	2.396	0.928 6.188	0.072
	0.675 0.367	3.383	1.964	0.957 4.032	0.067
BPRS	1.042 0.278	9.325	2.845	1.614 4.985	<0.001
SAPS	0.685 0.379	3.267	1.984	0.944 4.170	0.071
SANS	0.786 0.338	5.408	2.195	1.131 4.257	0.021
TC	1.348 0.327	16.994	3.850	2.028 7.308	<0.001
TG	0.934 0.306	9.316	2.545	1.397 4.636	0.002
HDL C	-0.329 0.124	7.040	0.720	0.564 0.918	0.008
LDL C	0.832 0.319	6.802	2.298	1.230 4.294	0.009

2.2
 Logistic
 BPRS
 SANS TC TG HDL C
 LDL C
 P<0.05 2

2.3
 TC TG HDL C LDL C
 AUC >
 0.65
 ROC
 TC TG HDL C LDL C cut off 4.85
 mmol/L 2.27 mmol/L 1.44 mmol/L 3.17 mmol/L
 1= 0= ROC
 3 1

3

Table 3 ROC analysis of serum TC TG HDL C and LDL C for predicting cognitive impairment in patients with schizophrenia

	95%	cut off	%	%
TC	0.697 0.609-0.816	0.053 4.85 mmol/L	56.10	78.40
TG	0.803 0.760-0.916	0.040 2.27 mmol/L	78.80	70.30
HDL C	0.750 0.763-0.929	0.042 1.44 mmol/L	77.30	70.30
LDL C	0.833 0.684-0.871	0.048 3.17 mmol/L	74.20	86.50

3

10

11

64.08%

12

TNFSF 15

1	2																		
		1A	TL1A			15 TNFSF15				SNP									
						PBC													
2018	9	2020	9			120	PBC												120
rs12235514						SNP	rs55717217	rs6478108	rs4979462	rs10114470	rs1857335								
		Sanger					SNP					TL1A							
		SNP					ALT	AST	ALP	GGT	TBIL	DBIL	TBA	ALB					
							rs55717217	rs6478108	rs4979462	rs1857335									
							² =16.443	13.463	7.904	7.502	17.313	13.845	16.233	13.687	P<0.05				rs10114470
rs12235514							² =4.462	3.008	3.445	3.342	P>0.05	PBC							TL1A
							t=2.59	P<0.05		rs4979462									rs55717217
							t=2.12	2.23	3.35	3.36	P<0.05	TL1A							AST ALT
		ALT	AST				r=0.202	0.252	0.313	0.328	0.129	P<0.05	TBIL	DBIL	ALB				
		ALP	GGT	TBA															
		P>0.05	ROC					TL1A	PBC				AUC	0.838					TNFSF15
rs4979462	rs55717217	rs6478108	rs1857335	PBC									rs4979462	rs55717217					
							ALT	AST					TL1A	AST	ALT	ALP	GGT	PBC	



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- 1. 610041
- 2. 550000

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TNFSF15 SNP
 Haploview4.2 Tagger 2
 SNP 2.1 SNP
 $r^2 >0.05$ 0.8 rs4979462 rs55717217
 SNPs rs55717217 rs6478108 rs4979462 rs6478108 rs1857335
 rs10114470 rs1857335 rs12235514 Sanger P<0.05 rs10114470
 SNP rs12235514
 Chromas SNP P>0.05 SNP
 1
 1.2.2 TL1A 2.2 TL1A
 TL1A P<0.05 2
 1.2.3 2 TL1A -
 Table 2 Comparison of TL1A concentrations in peripheral blood between case group and control group -

	n	TL1A ng/L
	120	19.64±5.74
PBC	120	31.45±17.03
t		2.59
P		0.01

Ci16000
 ALT AST ALP GGT TBIL DBIL TBA ALB
 ISO15189
 1.3 SPSS 24.0 2.3 SNP
 120 PBC 8
 ALT AST ALP GGT TBIL DBIL TBA ALB
 rs4979462 rs55717217
 ALT AST P<0.05
 rs6478108 rs1857335
 P>0.05 3
 Spearman
 ROC
 P<0.05
 1 TNFSF15 6 SNP
 n %

Table 1 Comparison of the genotypes and allele frequencies of the six SNP loci of TNFSF15 n %

SNP	P		P	
rs55717217	AA	54 45.0	85 70.8	
	AG	59 49.2	31 25.8	16.443 <0.001
	GG	7 5.8	4 3.3	
rs6478108	CC	20 16.7	35 29.2	
	CT	63 52.5	63 52.5	7.904 0.019
	TT	37 30.8	22 18.3	
rs4979462	CC	51 42.5	83 69.2	
	CT	62 51.7	33 27.5	17.313 <0.001
	TT	7 5.8	4 3.3	
rs10114470	TT	22 18.3	36 30.0	
	CT	66 55.0	57 47.5	4.462 0.107
	CC	22 26.7	27 22.5	
rs1857335	GG	51 42.5	82 68.3	
	GA	61 50.8	34 28.3	16.233 <0.001
	AA	8 6.7	4 3.4	
rs12235514	GG	89 74.2	76 63.3	
	GA	29 24.2	40 33.4	3.445 0.179
	AA	2 1.6	4 3.3	

IL 12A IL 12RB2 PBC
¹⁰ TNFSF 15
 TNFSF 15
 ANCA
^{4 5 11 12}
 TNFSF 15
 9q32 TL1A TL1A
¹³ TNFSF 15
 rs4979462 PBC
¹⁴ rs4979462 rs55717217
 rs6478108 rs1857335 PBC
 rs4979462 rs55717217
 rs6478108 TNFSF 15
 rs1857335 3 UTR
 2.4 TL1A
 TL1A AST
 ALT ALP GGT TBA r=0.202
 0.252 0.313 0.328 0.129 P <0.05 TL1A
 TBIL DBIL ALB P>0.05
 2.5 ROC TL1A rs4979462 rs55717217
 PBC ALT AST
 TL1A PBC ROC PBC
 0.838 P<0.05
 1
 RNA
 PBC
^{15 16}
 TNFSF 15
 PBC
 TL1A
 Th1 Th17 TL1A
 3
 PBC IL 2 IL 4 IL 12 IL 17 IL 21 ¹³
 TL1A AST ALT ALP GGT TBA
 ROC TL1A %
 IgM #3 F F F
⁹ PBC



TNFSF15 rs4979462
 rs55717217 rs6478108 rs1857335 PBC
 rs4979462 rs55717217
 ALT AST TL1A
 AST ALT ALP GGT TBA
 PBC

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SAA HMGB1 TNF

AS A SAA B1
 HMGB1 TNF AS
 2021 1 2023 10 152
 AS Bath BASDAI
 80 C CRP SAA
 HMGB1 TNF ESR UA Spearman AS SAA HMGB1
 TNF ROC SAA HMGB1 TNF
 AS UA CRP ESR SAA HMGB1 TNF
 $t=5.827$ 50.112 50.329 35.944 46.683 67.298 $P<0.05$
 SAA HMGB1 TNF $t=10.977$ 13.302 12.705
 $P<0.05$ Spearman AS SAA HMGB1 TNF
 $r=0.743$ 0.684 0.571 $P<0.005$ ROC SAA HMGB1 TNF
 AS AUC 0.922 92.76% 90.00% $P<0.05$ AS
 SAA HMGB1 TNF SAA HMGB1 TNF AS
 AS

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To explore the correlation between the expression of serum amyloid A (SAA), high migration group protein B1 (HMGB1), tumor cell necrosis factor (TNF) and disease activity in patients with ankylosing spondylitis (AS) and analyze the value of these indicators in the diagnosis of AS and the evaluation of disease activity. 152 AS patients admitted to the Rheumatology and Immunology Department of Xinyang Central Hospital from January 2021 to October 2023 were selected as the study group. According to bath ankylosing spondylitis disease activity index (BASDAI) score, the study group was divided into a disease active group and a disease remission group. And 80 patients with rheumatoid arthritis were selected as the control group. Serum C reactive protein (CRP), SAA, HMGB1, TNF, erythrocyte sedimentation rate (ESR) and uric acid (UA) were compared between the two groups. The correlation between serum levels of SAA, HMGB1, TNF and disease activity was analyzed using the Spearman method. The value of single and combined detection of serum SAA, HMGB1 and TNF in the diagnosis of AS was

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analyzed using the receiver operating curve (ROC) The levels of serum UA CRP ESR SAA HMGB1 and TNF in the study group were significantly higher than those in the control group with statistical significance $t=5.827$ 50.112 50.329 35.944 46.683 67.298 $P<0.05$. Serum levels of SAA HMGB1 and TNF in patients with the active stage were higher than those in patients with the remission stage and the difference was statistically significant $t=10.977$ 13.302 12.705 $P<0.05$. Spearman analysis showed that the expression of SAA HMGB1 and TNF in serum of AS patients were positively correlated with disease activity $r=0.743$ 0.684 0.571 $P<0.005$. The ROC curve demonstrated that when serum SAA HMGB1 and TNF were combined the AUC for predicting AS was 0.922 with a sensitivity of 92.76% and specificity of 90.00% better than single detection $P<0.05$ The levels of SAA HMGB1 and TNF in the serum of patients with AS activity were significantly increased. SAA HMGB1 and TNF showed a significant correlation with the disease activity of AS. The combined detection of the three markers could serve as a crucial reference for diagnosing AS and the evaluating disease activity.

AS SAA HMGB1 TNF Disease activity

ankylosing spondylitis AS 1 1984 AS

6

2 18 3 *

1 AS

AS

AS Tumor Necrosis Factor TNF

2

serum amyloid A SAA

SAA SAA

3 B1 High Mobility

Group Protein B1 HMGB1

HMGB1

45 HMGB1 AS

SAA HMGB1

TNF AS

AS

1

1.1

2021 1 2023 10

152 AS

80

DAI

⁷ BASDAI

AS	9	AS	SAA	a	HMGB1	TNF
SAA				/	HMGB1	
	SAA	1 000		ROC	SAA HMGB1	
6	TNF	10	TNF	AS	AS	AUC
HMGB1			0.922		92.76%	90.00%
					SAA HMGB1	TNF
	HMGB1	Toll	AS	AS	AS	
		AS		AS	SAA HMGB1	TNF
	11	HMGB1			SAA HMGB1	TNF AS
	HMGB1			AS		
				AS		
	12 13					
HMGB1	AS		TNF			
çağan	14		AS	1	Mauro D Thomas R Guggino G et al.	Ankylosing spondy
TNF		NF B			litis an autoimmune or autoinflammatory disease	J . Nat
				2		Rev Rheumatol 2021 17 7 387 404.
			AS		miR 29a miR 146a	J .
		CRP ESR SAA			2020 12 5 587 591.	
HMGB1	TNF	AS	Hu	15		.MMP 3 SAA
		AS SAA CRP	ESR			J .
		SAA	AS		2021 33 3 361 362	
	79.49%	CRP	64.10%	ESR		HMGB1 HBP IL 10
	61.54%	SAA	AS			J .
				4	2023 33 1 31 34.	
AS				5		B1 6
					9	J .
						2022 42 12 1022 1025.
HMGB1	TNF	SAA	AS	6	van der Linden S Valkenburg HA Cats A.	Evaluation of di
			TNF		agnostic criteria for ankylosing spondylitis. A proposal for	modification of the New York criteria. Arthritis Rheum
			HMGB1	HMGB1	1984 27 4 361 368	
		B				
		AS		16	7	Abdal SJ Yesmin S Shazzad MN et al. Development of a
AS					Bangla version of the Bath Ankylosing Spondylitis Disease	Activity Index BASDAI and the Bath Ankylosing Spondyli
		BASDAI			tis Functional Index BASFI J . Int J Rheum Dis 2021 24	
AS					1 74 80.	
	SAA HMGB1	TNF		8		J .
	SAA HMGB1	TNF	BASDAI			2023 49 6 672
					675+680.	



SAO

2023 3

VEGF miR 34a HIF 1

102 SAO

2024 3

n=51

n=51

VEGF miR 34a HIF 1

Qm Vm

3.308 3.608 P<0.05

SSS

rCBF

t=5.038 3.297

GFAP

t=3.891 4.654 6.339 P<

0.05

HIF 1 miR 34a

VEGF

t=6.436 5.591 4.217 P<0.05

SAO

HIF 1 miR 34a

VEGF

miR 34a

1

$\chi^2=0.270 P>0.05$

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To investigate the efficacy of butaphthalein combined with eDaravone dextrocaphorol in the treatment of small artery occlusion type SAO stroke and the effects on serum vascular endothelial growth factor VEGF miR 34a and hypoxia inducing factor 1 HIF 1. A total of 102 SAO stroke patients diagnosed and treated at Jieshou People s Hospital from March 2023 to March 2024 were selected for the study. They were randomly divided into two groups the butylphthalein group n=51 treated with butylphthalein and the combination group n=51 treated with butylphthalein treatment and Edaravone dexcamol using a random number table method. Cerebral hemodynamics neurological function serum miR 34a VEGF HIF 1 levels and adverse reactions were compared between the two groups. After treatment the relative cerebral blood flow rCBF mean blood flow Qm and mean blood flow velocity Vm of the combination group were statistically higher than those before treatment and the butylphthalein group t=5.038 3.297 3.308 3.608 P<0.05. After treatment the Scandinavian Stroke Scale SSS score of the combination group was statistically higher than the SSS score of the butylphthalein group and the levels of glial fibrillary acidic protein GFAP in the combination group were statistically lower than those before treatment and in the butylphthalein group t=3.891 4.654 6.339 P<0.05. After treatment the expression of

HIF-1 and miR-34a in the peripheral blood of the combination group was statistically lower than before treatment and in the butylphthalatein group and the expression of VEGF was statistically higher than in the butylphthalatein group and before treatment $t=6.436$ 5.591 4.217 $P<0.05$. There was no significant difference in adverse drug reactions between the two groups $\chi^2=0.270$ $P>0.05$. The combination of butylphthalatein and Edaravone dextrocamphorol in the treatment of SAO stroke can improve cerebral hemodynamics and cerebral perfusion, enhance neurological function, reduce HIF-1 and miR-34a expression, and elevate VEGF levels.

Butylphthalatein Edaravone dextrocamphorol SAO miR-34a EGF HIF-1

Small artery occlusion SAO

2018⁶

25%

SAO

1 SAO
2 SAO

23

JSLC20230003

1.2

4

4 1

H20100041 100 mL
0.9 g 100 mL/

2 /

5

SAO

H20200007 15 mL

Vascular endothelial growth factor VEGF miR-34a Hypoxia inducible factor 1 HIF-1

100 mL 0.9%

2 /

3-14 d

1

1.3

1.3.1

1.1

CT

2022 3 2024 3

Relative cerebral blood flow rCBF

102 SAO

51

Mean blood flow

65.22±9.94

32 19
0-48 h

velocity Vm

Mean blood flow Qm

1.3.2

5 32

5 mL

34 17

3 500 r/min

61.37±10.80

0-48 h

10 min

12.5 cm

3

31

$P>0.05$

Glial fibrillary acidic protein GFAP

1.3.3 Scandinavian SSS GFAP
 Stroke Scale SSS ⁷ SSS P>0.05 GFAP
 58 SSS GFAP
 miR 34a VEGF HIF 1 RNA simple P<
 RNA 0.05 2
 RNA FastKing cDNA 2 SSS GFAP -
 cDNA 45 15 min 95 3 min Table 2 Comparison of SSS scores and GFAP levels between two groups -

miR 34a 95 3 min 95 5s
 60 15s 72 15s 40 U6
 miR 34a 5 TGCCTG
 GCAGTGTCTTAGCT 3 5 CCAGTG
 CAGGGTCCGAGGTATT 3 U6 5 CTC
 GCTTCGGCAGCACA 3 5 AAC
 GCTTCACGAATTTGCGT 3 2 ^{ct}
 miR 34a
 ABclonal VEGF HIF 1
 HIF 1 HIF 1 miR 34a
 1.3.4 VEGF P<0.05 3
 2.4
 1.4 SPSS 26.0 - P>0.05 4
 t
 t n % 3
 2 P<0.05 SAO
 2.1 rCBF Qm Vm 8
 rCBF Qm Vm
 P>0.05
 P<0.05 1 9

	n	SSS		GFAP ng/ml	
	51	27.41±2.53	48.37±4.58 ^a	10.34±2.23	5.18±1.13 ^a
	51	28.17±2.86	44.70±4.94 ^a	10.74±2.15	6.84±1.49 ^a
t		1.421	3.891	0.922	6.339
P		0.158	<0.001	0.359	<0.001

^aP<0.05

Table 1 Comparison of two groups of rCBF Qm and Vm -

	n	rCBF		Qm L/min		Vm cm/s	
	51	0.81±0.24	1.22±0.31 ^a	3.46±0.62	5.72±1.33 ^a	7.23±1.14	10.13±1.57 ^a
	51	0.79±0.20	0.93±0.27 ^a	3.36±0.71	4.89±1.21 ^a	7.38±1.20	9.14±1.45 ^a
t		0.457	5.038	0.758	3.297	0.647	3.308
P		0.648	<0.001	0.450	0.001	0.519	0.001

^aP<0.05

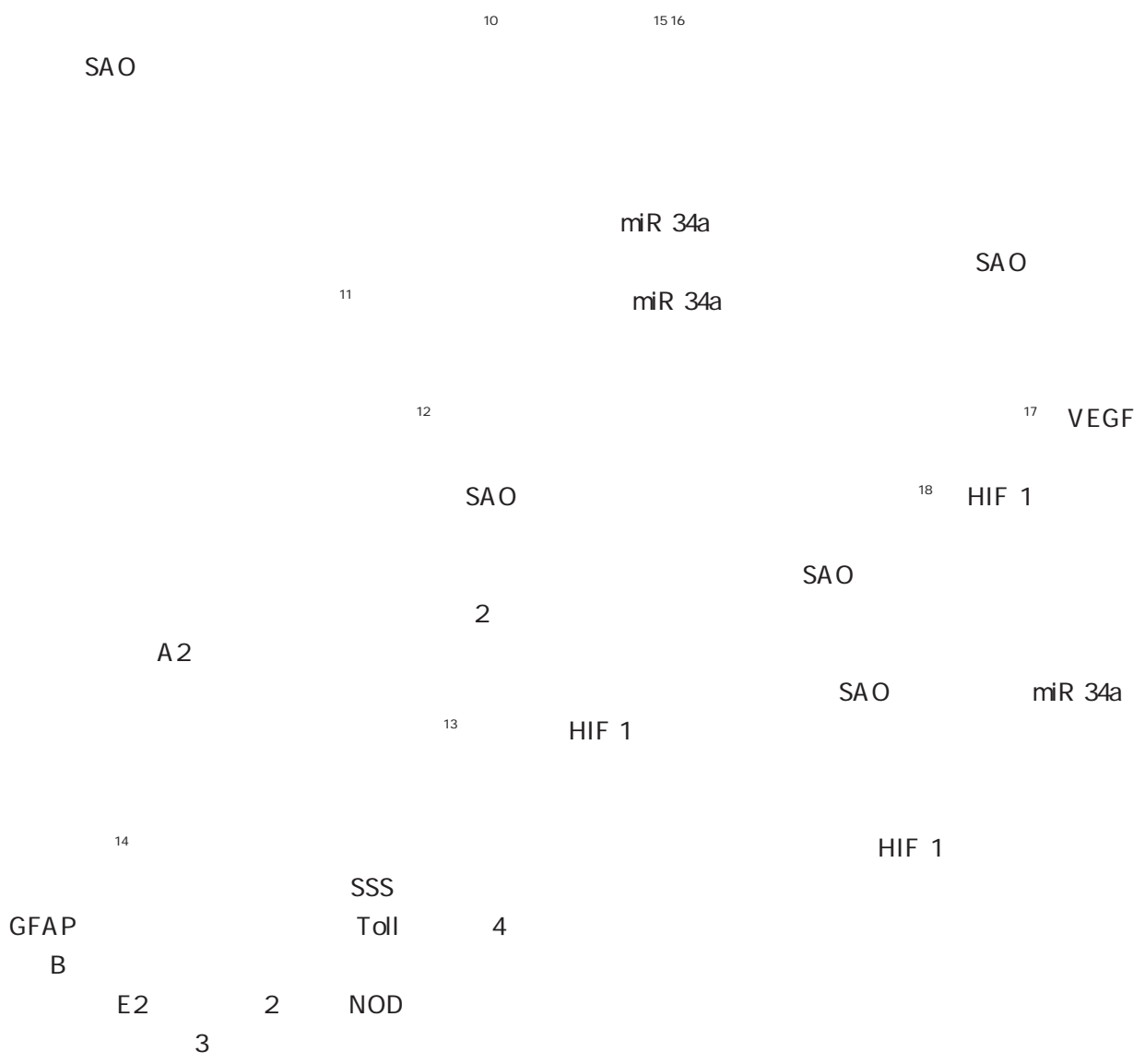
3 VEGF miR 34a HIF 1
 Table 3 Two groups of serum VEGF miR 34a HIF 1 comparison

	n	VEGF pg/mL		miR 34a		HIF 1 pg/mL	
	51	5.71±1.12	8.44±1.34 ^a	2.85±0.67	1.21±0.29 ^a	1423.02±301.24	684.59±183.75 ^a
t	51	5.82±1.27	6.83±1.56 ^a	2.91±0.71	1.67±0.42 ^a	1426.31±314.05	841.20±191.23 ^a
P		0.464	5.591	0.439	6.436	0.054	4.217
		0.644	<0.001	0.662	<0.001	0.957	<0.001

^aP<0.05

4 n %
 Table 4 Comparison of adverse drug reactions between the two groups n %

	n								
	51	0	0.00	1	1.96	2	3.92	0	0.00
	51	1	1.96	2	3.92	1	1.96	1	1.96
P									



VEGF

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12

FIB RBP NAG TRF

	NAG	TRF	FIB	HSPN	RBP	N	D
1	2023	5	114	HSPN			2021
	51			57			63
	FIB	RBP NAG TRF	FIB	RBP NAG TRF			
	t=3.160	11.001 2.882 4.168	P<0.05	FIB RBP NAG TRF			
	t=3.995	10.123 4.324 2.893	P<0.05	FIB RBP NAG HSPN			
	r=0.360	0.666 0.400 0.239	P<0.05	ROC	FIB	RBP NAG TRF	
		AUC	0.930		FIB	RBP NAG TRF	
HSPN				HSPN		N	D

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To investigate the relationship between serum fibrinogen (FIB) urinary retinol binding protein (RBP) urinary n-acetyl D-glucosaminase (NAG) transferrin (TRF) and renal pathological grade in children with purpura nephritis (HSPN). A total of 114 children with HSPN admitted to Anhui Children's Hospital from January 2021 to May 2023 were selected as the study group and divided into 63 cases of grade and 51 cases of grade according to pathological grade. In addition 57 hospitalized children with anaphylactoid purpura without renal injury were selected as the control group. The levels of serum FIB urine RBP NAG and TRF were compared between the two groups. The study group was also analyzed for the relationship between serum FIB urine RBP NAG and TRF and renal pathological grade. The predictive value of serum FIB and urine RBP was analyzed. The levels of serum FIB RBP NAG and TRF in the study group were higher than those in the control group (t value 3.160 11.001 2.882 and 4.168 respectively P<0.05). The levels of serum FIB RBP NAG and TRF in grade group were higher than those in grade group (t value 3.995 10.123 4.324 2.893 P<0.05). Serum FIB RBP and NAG were positively correlated with renal pathological grade (r value 0.360 0.666 0.400 0.239 P<0.05). ROC curve analysis showed that the AUC of serum FIB combined with urine RBP NAG and TRF to predict purpura

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Henochschonlein purpura nephri
Henoch schonlein purpu
tis HSPN
ra HSP

1 HSPN
2 HSPN
HSPN

HSPN

3 Fibrinogen FIB

4 Urinary
retinol binding protein RBP

N _FS A A u qB,, F etctB et n qB,, 4P r

s

CysC

FIB

14

18

Urea

FIB RBP NAG TRF FIB RBP NAG
ROC HSPN AUC Q.930
TRF HSPN

RBP

HSPN

RBP

RBP

RBP

FIB

RBP NAG TRF

RBP

15

RBP

HSPN

HSPN

pH

RBP

RBP

RBP

NAG

NAG

16

NAG

NAG

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2

J .

2020 36 7 535 542

3

J .

2024 27 20 2491 2497.

4

J .

2023 49 7 858 862

TRF

TRF

HSP

TRF

TRF

17

5

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2

J .

2022 50 1 83 87.

RBP NAG TRF

HSPN

FIB

6

J .

2022

FIB RBP NAG TRF

45 7 732 737.

7

HSPN

2016 J .

2017 55 9 647 651.

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2012 408

CysC

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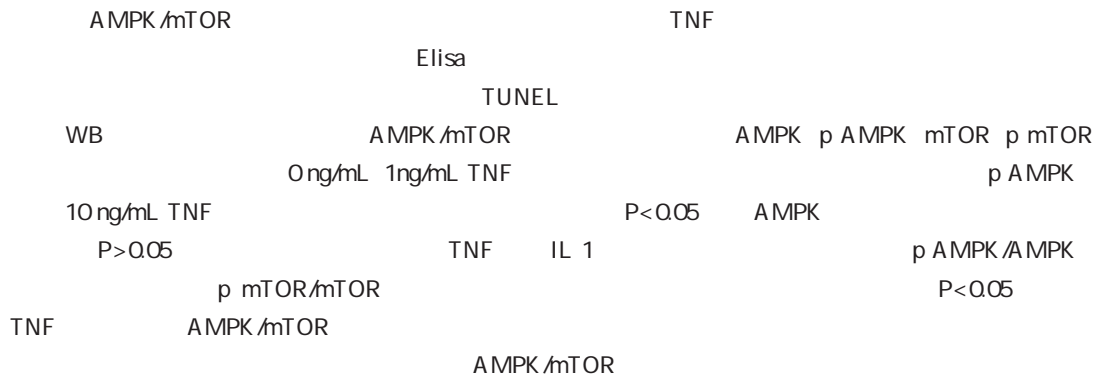
2021 39 12

895 899.

RBP NAG TRF

1563

AMPK /mTOR



CAI Canhui ZHU Zhongshou

Department of Otolaryngology Ningde Municipal Hospital Affiliated of Ningde Normal College Ningde Fujian China 352100

The relationship between inflammation and AMPK/mTOR signaling pathway in chronic nasosinusitis was explored by constructing the epithelial cell injury model of chronic nasosinusitis. Nasal mucosal epithelial cells were stimulated with different concentrations of TNF to induce rhinitis cell model. The content of inflammatory factors in normal nasopharyngeal epithelial cells (normal group) and rhinitis cells (model group) was detected by Elisa. The apoptosis of nasopharyngeal epithelial cells in normal group and model group was detected by TUNEL. The AMPK/mTOR signaling pathway related proteins (AMPK, p-AMPK, mTOR, p-mTOR) in normal group and model group were detected by WB. The relative expression of p-mTOR... Compared with 0 ng/mL and 1 ng/mL TNF treated rhinitis epithelial cells, p-AMPK protein was up-regulated in 10 ng/mL TNF treated cells, the difference was statistically significant (P < 0.05), but the changes of AMPK protein at different concentrations were not statistically significant (P > 0.05). Compared with the normal group, the contents of TNF and IL-1 in the

injury by activating AMPK /mTOR pathway.

Chronic rhinosinusitis epithelial cell injury AMPK/mTOR channel Inflammatory factors Apoptosis

12 1

2

3

4

5

1

1.1

1.2

1.2.1

1.2.2

1.2.3

1.2.4

1.2.5

GAPDH mTOR Monoclonal Phospho mTOR Ser2448 Monoclonal AMPK Alpha Polyclonal Phospho AMPK Thr172

mTOR Monoclonal Phospho AMPK ECL

Thermo

PBS

HNEpC RPMI 1640 +10% FBS+1% PBS CO₂ 70%~80% 0.25%

AMP /

AMP activated protein kinase/mammalian target of rapamycin AMPK/mTOR

AMPK

mTOR

TNF

TNF

WB AMPK p AMPK TNF

0 1 10 100ng/mL 24 h

enzyme linked immunosorbent assay ELISA

20 cm

3 000 rpm 15 min 450 nm OD OD TNF IL 1

dUTP Terminal deoxynucleotidyl Transferase Mediated Nick End Labeling TUNEL

4% 20 min 3 H₂O₂ Triton 100

10min TUNEL 1 9

37 1 h DAPI 1 500 5 min

HNEpC RPMI 1640 ELISA

1 ELISA Tunel BCA

50T FBS P/S DAPI

Gibco

Western Blot WB

20 cm

4 3 000 rpm 15 min
 BCA loading buffer 98 5 min
 SDS PAGE 4
 GAPDH 1 10 000 AMPK 1 2 000 p AMPK 1
 1 000 mTOR 1 5 000 p mTOR 1 2 000
 TBST 3 2 h TBST 3

1.3

Prism 8.0 GraphPad USA

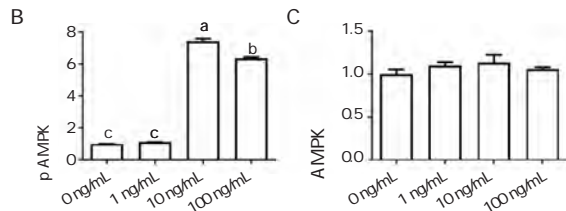
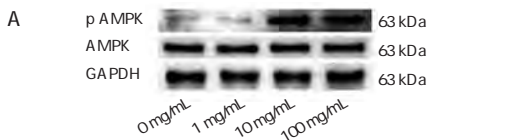
t P<0.05

2

2.1

TNF AMPK

TNF HNEpC 0ng/mL
 1 ng/mL 10 ng/mL 100 ng/mL AMPK
 P>0.05 10ng/mL
 p AMPK 0ng/mL 1 ng/mL
 100 ng/mL P<0.05
 100 ng/mL p AMPK 0 ng/mL
 1 ng/mL 10ng/mL
 P<0.05 0ng/mL 1 ng/mL p AMPK
 P<0.05 1



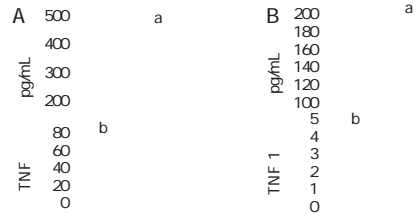
A AMPK p AMPK GAPDH B p AMPK
 C AMPK
 a 0 1 100 ng/mL P<0.05 b 0 1 10 ng/mL
 P<0.05 c 0 1 ng/mL P<0.05
 1 TNF AMPK
 p AMPK

Figure 1 Comparison of AMPK and p AMPK protein expression in nasal epithelial cells with different concentrations of TNF

2.2

TNF IL 1
 P<0.05

2



A TNF B IL 1
 P<0.05 a P<0.05 b

2 ELisa

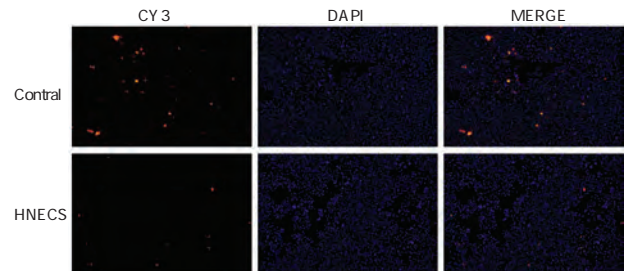
TNF IL 1

Figure 2 The levels of TNF and IL 1 were detected by ELisa

2.3

0.05 3

P<



3 TUNEL staining ×100
 Figure 3 TUNEL detection of apoptosis fluorescent TUNEL staining ×100

2.4 AMPK/mTOR

AMPK mTOR
 P>0.05

p AMPK /AMPK

P<0.05

p mTOR/mTOR

P<0.05

4

3

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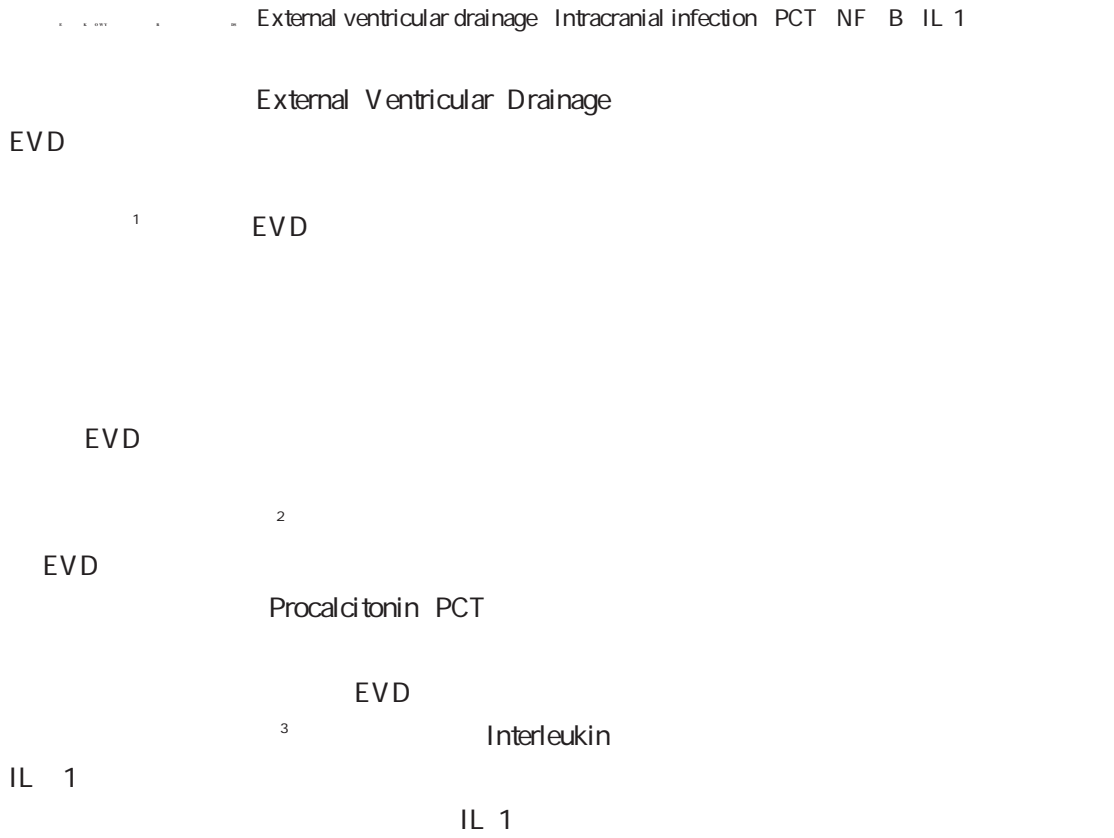
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Multivariate logistic regression analysis showed that hypoproteinemia external ventricular drainage duration >7 days and increased expression levels of NF B PCT and IL 1 in cerebrospinal fluid were independent risk factors for intracranial infection after EVD OR=2.298 2.197 2.179 1.893 1.978 P<0.05 . The ROC areas for NF B PCT IL 1 and their combined detection were 0.718 0.753 0.726 and 0.870 respectively P<0.05 The disturbance in the expression levels of NF B PCT and IL 1 in cerebrospinal fluid is a significant risk factor for intracranial infection following EVD. The combined detection of these three factors has a high predictive value for intracranial infection after EVD.



1.3

SPSS 22.0

- t

n %
Logistic EVD

ROC NF B PCT

IL 1 EVD P<

0.05

E dJk R R40EF UCE UCE UCE UCE UCE UCE

2

2.1

ICU d

ICU 4š1 FQ0 % % -

NF B

PCT IL 1
P<0.05 1

2.2 EVD
ICU

NF B

PCT IL 1 EVD

>7 d

NF B PCT IL 1 EVD
P<0.05

2

2.3 NF B PCT IL 1 EVD

ROC NF B PCT IL 1

ROC 0.718 0.753 0.726

0.870 P<

0.05 3 1

3

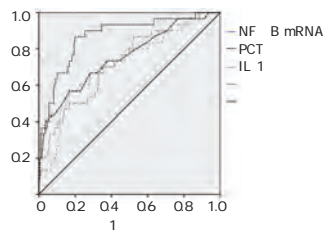
8

9

EVD

2%~24%

EVD



1 EVD ROC
Figure 1 ROC curve analysis of intracranial infection after EVD

10 EVD

11 NF B

12 NF B I B NF B EVD IL 1

13 14 IL 1

15 Liu IL 1 IL 1 IL 1 IL 6 IL 1 PCT

16 PCT

17 EVD >7d

18 NF B PCT IL 1 EVD

19 NF B PCT IL 1 EVD >7d

20 NF B PCT IL 1 EVD

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TLR4 NF B

4 TLR4 B NF B 2019 1 MHD Toll
 118 MHD 2022 11
 n=52 n=66
 + 0.8g/ 3 /d + + 15g/
 2 /d 2 2 24 h
 UPE SCr BUN IL 6 /
 NLR TNF TLR4 NF B mRNA 2
 $\chi^2=5.449 P<0.05$ UPE SCr BUN
 P>0.05 UPE SCr BUN
 P<0.05 IL 6 NLR TNF P>0.05 IL 6
 NLR TNF P<0.05 TLR4 NF B mRNA
 P>0.05 TLR4 NF B mRNA
 P<0.05 P=0.903
 MHD TLR4 NF B
 Toll 4 B

FEI Chengqiu JIANG Ming WANG Xin WANG Yicheng
 Department of Nephrology Lu an Shili Hospital Lu an Anhui China 237000

To investigate the effect of Huangqi Yishen granule combined with sevelamer on the efficacy and Toll like receptor 4 (TLR4) and nuclear transcription factor B (NF B) in patients with maintenance hemodialysis (MHD). The clinical data of 118 MHD patients admitted to Lu an City Lu an World Hospital from January 2019 to November 2022 were retrospectively analyzed. They were divided into a control group (n=52) and a combined group (n=66) according to different treatment methods. The control group received routine treatment + sevelamer (0.8 g/time 3 times/d). The combined group received routine treatment + sevelamer + Huangqi Yishen Granules (15 g/time 2 times/d) both groups were treated for 2 months. The clinical efficacy renal function (24 h urinary protein excretion rate (UPE) serum creatinine (SCr) blood urea nitrogen (BUN) microinflammatory state (interleukin (IL) 6 neutrophil / lymphocyte ratio (NLR) tumor necrosis factor a (TNF a) TLR4 NF B mRNA levels and incidence of adverse reactions were statistically analyzed before and 2 months after treatment in the two groups. After 2 months of treatment the total effective rate of the combined group was significantly higher than that of the control group and the difference was statistically significant ($\chi^2=5.449 P<0.05$). After treatment UPE

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SCr and BUN in the two groups decreased and those in the combined group were lower the differences were statistically significant $t=5.953$ 2.900 8.309 $P<0.05$. After treatment IL 6 NLR and TNF in both groups decreased and those in the combined group were lower the differences were statistically significant $t=4.956$ 3.841 5.098 $P<0.05$. There was no significant difference in TLR4 and NF B mRNA levels between the two groups before treatment $t=1.526$ 0.622 $P>0.05$. After treatment TLR4 and NF B mRNA in both groups decreased and those in the combined group were lower the differences were statistically significant $t=4.714$ 4.494 $P<0.05$. There was no significant difference in the incidence of adverse reactions between the two groups $\chi^2=0.015$ $P=0.903$ Huangqi Y ishen Granule combined with sevelamer can improve the therapeutic effect on MHD patients. This combination has been shown to enhance renal function and reduce micro inflammatory states. These improvements may be linked to the suppression of TLR4 and NF B expression.

. MHD Huangqi Y ishen Granules Micro inflammatory state TLR4 NF B



1.3.2

2.4 TLR4 NF B
 TLR4 NF B mRNA
 P>0.05 TLR4 NF B
 mRNA
 P<0.05 4

Table 4 Comparison of TLR4 and NF B levels between the two groups

	n	TLR4 mRNA		NF B mRNA	
	66	1.21±0.24	0.69±0.12 ^a	1.10±0.19	0.74±0.13 ^a
	52	1.15±0.17	0.83±0.20 ^a	1.08±0.15	0.86±0.16 ^a
t		1.526	4.714	0.622	4.494
P		0.130	<0.001	0.536	<0.001

2.5
 4/66 6.06%
 3.85% 2/52
 P>

Table 5 Comparison of adverse reactions between two groups

	n	n %		n %	
	66	1 1.51	1 1.51	2 3.03	4 6.06
	52	1 1.92	0 0.00	1 1.92	2 3.85
²					0.015
P					0.903

3

MHD

11 12

MHD

13

MHD

TLR4 NF B

MHD

1 Zhao S, Chen X, Wan Z, et al. Associations of serum 25 hydroxyvitamin D and vitamin D receptor polymorphisms with

MHD

15

MHD

MHD

IL 6 TNF
¹⁶ NLR

MHD

MHD

17 18

TLR4 NF B

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19

21 1 ALI PLR

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2. 230022

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also increased successively while the ALI value decreased successively the difference was statistically significant $F=83.871$ 224.621 189.263 $P<0.05$. The progressive group had a higher proportion of patients with a smoking e žgr i l s e l p H r t

2023 LC 13

1.2
1.2.1

TNM

23 25 10 36

19 21 1 cytokeratin 19 10

fragment antigen 21 1 CYFRA 21 1 min 10cm 3500r/min BK 200

4 5 / CYFRA 21 1

platelet to lymphocyte ratio PLR advanced lung cancer inflammatory index BC 3000

ALI

PLR ALI= $\frac{\text{kg/m}^2 \times \text{g/dL}}{\text{kg} / \text{m}^2 \text{ PLR} = \text{g/dL}}$ / =

CYFRA 21 1 ALI PLR

1.3

IMB SPSS 25.0

1.1

2020 1 2023 10

2

150 6

2.1 TNM CYFRA 21 1 ALI

18 PLR

150 CYFRA 21 1

2 97 PLR > > > ALI

53 35-88 < < < $P<0.05$ 1

1 TNM CYFRA21 1 ALI PLR

Table 1 Expression of CYFRA21 1 ALI and PLR in patients with different TNM stages

TNM	n	CYFRA21 1 ng/mL	ALI	PLR
	25	4.04±1.04	59.44±4.02	113.82±12.10
	10	5.06±0.73 ^a	55.73±4.45 ^a	132.97±5.69 ^a
	36	7.48±2.76 ^{ab}	46.27±2.68 ^{ab}	156.66±21.44 ^{ab}
	79	11.36±2.40 ^{abc}	37.75±4.93 ^{abc}	186.13±7.70 ^{abc}
F		83.871	189.263	224.621
P		<0.001	<0.001	<0.001

^aP<0.05 ^bP<0.05 ^cP<0.05

2.2

50% 4 <50% 96 54

P>0.05 CYFRA21 1 PLR

ALI

P<0.05 2

2.3

2 P<0.05

Logistic

CYFRA21 1 PLR ALI

P<0.05 3

2.4 PLR ALI CYFRA21 1

ROC

CYFRA21 1 PLR ALI

3

Logistic

Table 3 Multivariate Logistic regression analysis of poor prognosis in patients with lung cancer

	SE	Wald	OR	95% CI	P
=1 =0	0.578	0.242	5.705	1.782 1.109 2.864	0.017
=1 =0	0.549	0.289	3.609	1.732 0.983 3.051	0.057
~ =1 ~ =0	0.571	0.239	5.708	1.770 1.108 2.828	0.017
PLR	0.603	0.249	5.865	1.828 1.122 2.977	0.015
ALI	-0.544	0.216	6.343	0.580 0.380 0.886	0.012
CYFRA21 1	0.559	0.217	6.636	1.749 1.143 2.676	0.010

4 PLR ALI CYFRA21 1

ROC

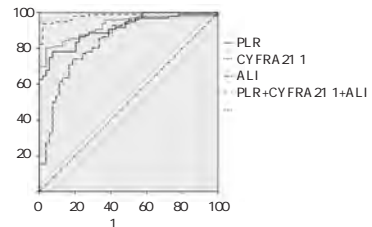
Table 4 ROC characteristics of PLR and ALI combined with CYFRA21 1 to evaluate poor prognosis in patients with lung cancer

	%	%	95%CI	AUC	P
PLR	163.36	0.707	78.1 92.6	0.858-0.951	0.904 <0.001
ALI	44.01	0.555	74.0 81.5	0.779-0.913	0.846 <0.001
CYFRA21 1	7.49 ng/mL	0.765	80.2 96.3	0.884-0.967	0.926 <0.001
LDH+SAA +IL 17A		0.919	93.8 98.1	0.966-0.999	0.982 <0.001

2 n % Table 2 Single factor analysis affecting prognosis of patients with lung cancer n %

	n=54	n=96	t/	P
	54.21±10.37	56.89±11.29	1.436	0.153
	27 50.00	35 36.46	2.614	0.106
	27 50.00	61 63.54		
	5 9.26	16 16.67	2.272	0.132
	49 90.74	70 72.92		
	35 64.81	62 64.58	0.001	0.978
	19 35.19	34 35.42		
	16 29.63	53 55.21	9.103	0.003
	38 70.37	43 44.79		
	15 27.78	46 47.92	5.809	0.016
	39 72.22	50 52.08		
TNM	~ 21 38.89	14 14.58	11.413	<0.001
	~ 33 61.11	82 85.42		
PLR	135.55±21.33	179.16±26.77	10.272	<0.001
ALI	51.66±8.66	40.35±6.62	8.966	<0.001
CYFRA21 1 ng/mL	5.55±1.45	10.67±3.21	11.090	<0.001

P<0.05 0.982 4 1



1 PLR ALI CYFRA21 1 ROC

Figure 1 ROC curve of PLR and ALI combined with CYFRA21 1 to evaluate poor prognosis in patients with lung cancer

3

18

8 CYFRA21 1

ALI PLR
ALI

/

ROC

CYFRA21 1 PLR ALI

9

0.982

CYFRA21 1

10

CYFRA21 1

PLR

~ ALI ~
ALI CYFRA21 1

ALI

PLR

5 K \$ ~~BC~~ I B > 5 ;

CYFRA21 1

11

ALI PLR

12

~ CYFRA21 1 PLR ALI

13 14

CYFRA21 1 PLR ALI
ALI

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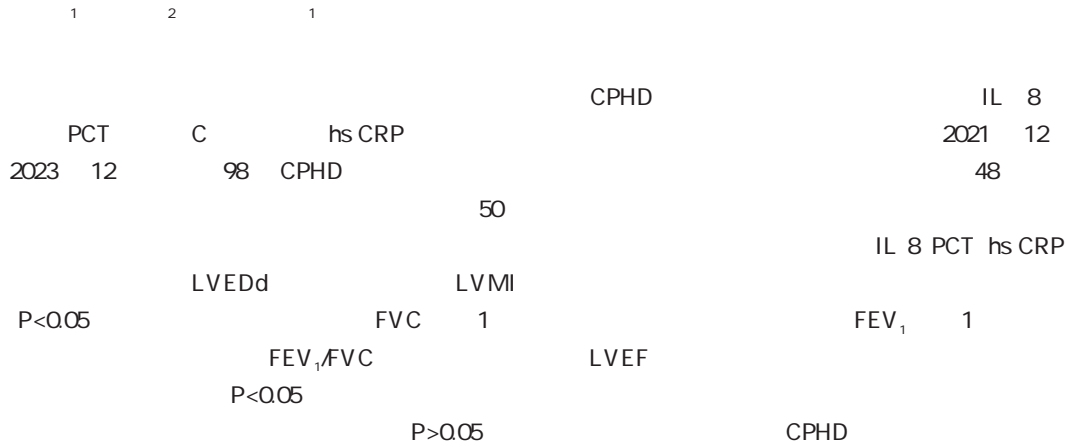
16

T

17

IL 8 PCT

hs CRP



SHI Dekun¹ NIE Wei² HUO Yongqiang¹

1. Department of Medicine Huaibei Hospital of Traditional Chinese Medicine Huaibei Anhui China 235000 2. Department of Critical Care Medicine Huaibei Hospital of Traditional Chinese Medicine Huaibei Anhui China 235000

To explore the influence of Zhenwu decoction on cardiopulmonary function and levels of interleukin IL 8 procalcitonin PCT and high sensitivity C reactive protein hs CRP in patients with chronic pulmonary heart disease CPHD... The medical records of 98 patients with CPHD at Huaibei Hospital of Traditional Chinese Medicine were retrospectively analyzed from December 2021 to December 2023. 48 patients treated with conventional Western medicine were included in the control group and 50 patients who received conventional Western medicine combined with Zhenwu decoction were enrolled in the study group. The TCM syndrome scores cardiopulmonary function inflammatory cytokines before and after treatment and adverse reactions during treatment were observed in the both groups... After treatment the scores of TCM syndromes IL 8 PCT hs CRP left ventricular end diastolic diameter LVEDd and left ventricular mass index LVMI in the two groups decreased significantly P<0.05. These indicators were lower in the study group compared to the control group P<0.05. The forced vital capacity FVC forced expiratory volume in the first second FEV1 ratio of forced expiratory volume in the first second to forced vital capacity FEV1/FVC and left ventricular ejection fraction LVEF in the two groups

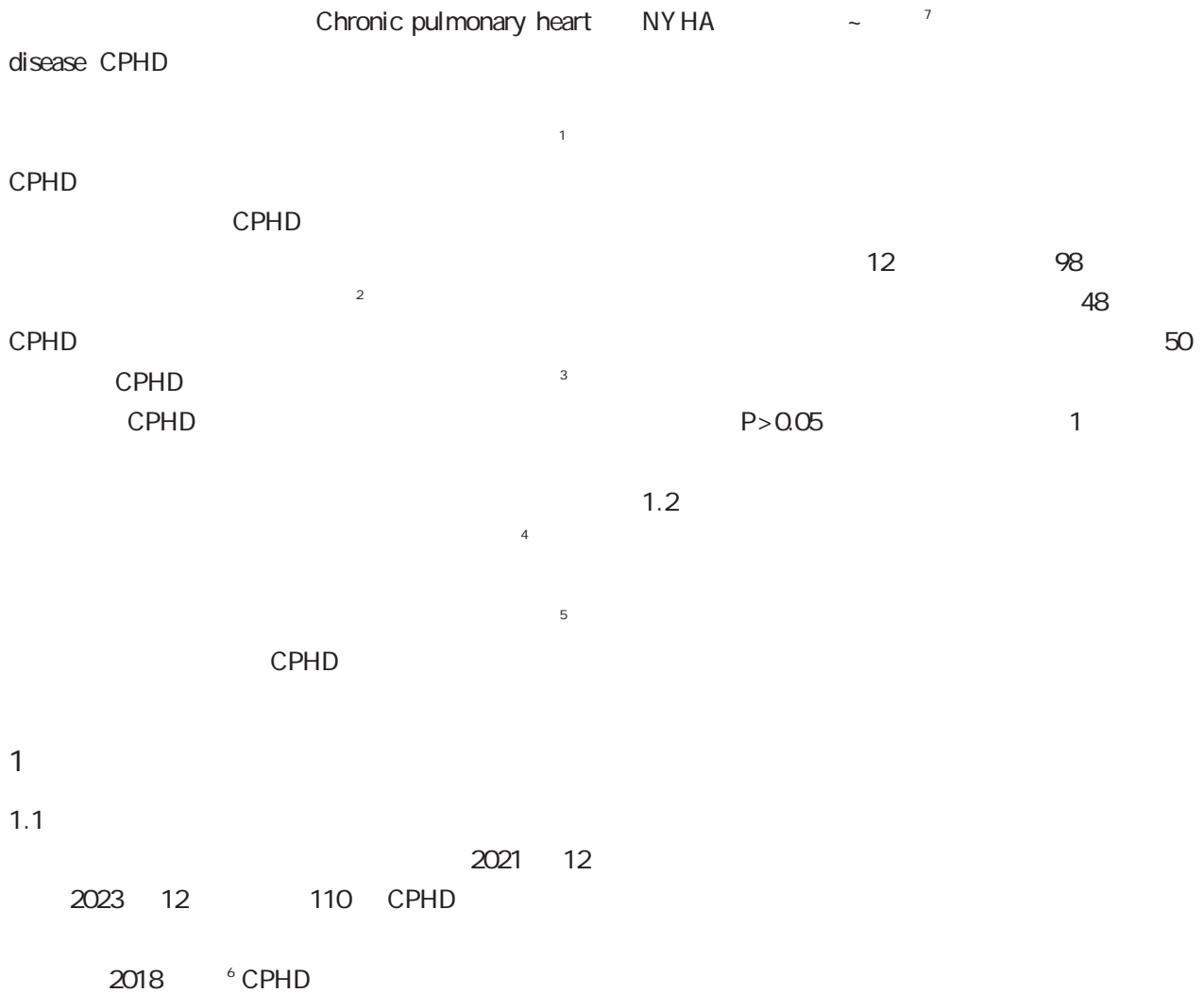
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after treatment significantly increased $P < 0.05$. The indicators in the study group were higher than those in the control group $P < 0.05$. There were mild adverse reactions in both groups during treatment which were relieved with symptomatic treatment. There was no statistical significance in the total incidence rate of adverse reactions $P > 0.05$. The application of Zhenwu decoction in treating patients with CPHD can enhance cardiopulmonary function recovery effectively lower the levels of inflammatory factors alleviate the clinical symptoms and demonstrate good safety.

Zhenwu decoction CPHD Cardiopulmonary function Inflammatory cytokines



new york heart association

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9 . NP
SCC CYFRA21 1
J . 2020 28 13 2254 2259.

10 . LDH LMR ALI
J . 2023 15
12 2189 2193.

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12 . PLR NLR
J .
2023 33 3 13 18.

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15 Hwang M Canzonio JV Rosners et al. Peripheral b# Oo a #m e 3711dra
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days of treatment the wound infection rate of the combined group was lower than that of the control group and the difference was statistically significant $P < 0.05$. After 14 days of treatment the CRP value and wound volume value of the combined group were lower than those of the control group and the difference was statistically significant $P < 0.05$. The wound healing rate in the combined group was 86.67% which was hi_f R h 8

2.3 MMP2 TMP1 VEGF P>0.05
 P<0.05 VEGF 3 n % -
 MMP2 TMP1 NPWT VEGF P<0.05 4
 TMP1 MMP2

Table 3 Comparison of wound healing rate toe amputation rate and wound healing time between the two groups n % -

	14 d				d		
NPWT	15	4	9	2	13 86.67 ^a	1 6.67 ^a	65.53±24.36 ^a
/	15	0	6	9	6 40.00	7 46.77	97.47±17.06 ^a
					5.167	4.261	13.954
					0.023	0.039	<0.001

Table 4 Simple effect analysis of MMP2 TMP1 and VEGF levels at different time points between the two groups -

	n	0d	7 d	14 d	F	P
MMP2	15	2.29±0.18	2.04±0.19	1.89±0.29	27.590	<0.001
	15	2.32±0.23	2.33±0.28	2.15±0.19	4.488	0.043
t		0.234	11.883	8.779		
P		0.632	0.002	0.006		
TMP1	15	827.79±80.03	849.89±79.70	872.18±81.38	17.862	<0.001
	15	838.69±89.74	720.18±73.42	632.53±87.40	42.846	<0.001
t		0.123	21.491	60.410		
P		0.728	<0.001	<0.001		
VEGF	15	348.77±47.50	365.65±41.17	382.61±38.67	1.808	0.190
	15	349.35±38.74	330.58±46.40	290.26±39.40	24.480	<0.001
t		0.01	4.796	41.983		
P		0.971	0.037	<0.001		

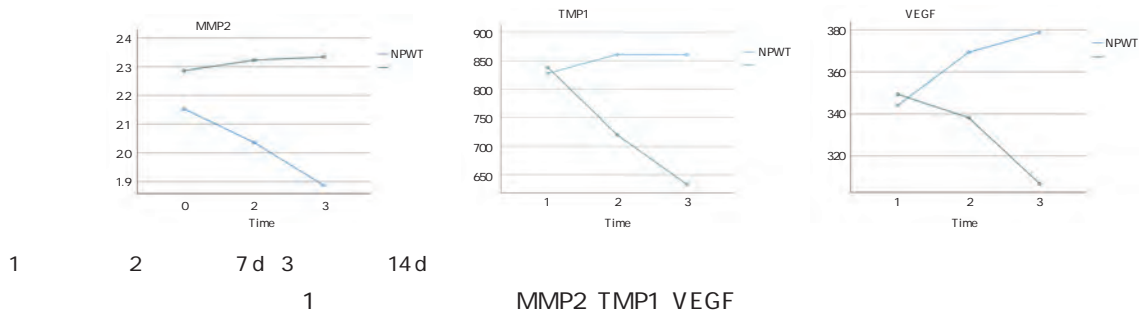


Figure 1 The interaction and levels of MMP2 TMP1 and VEGF at different time points between the two groups

2.4 3 6
 3 6
 P<0.05 P>
 0.05 6
 3
 7 d
 46.7% DFO

Table 6 Comparison of follow up between the two groups n %

	3		6	
n	3	6	3	6
	15 0 0.00 ^a	0 0.00	0 0.00	3 20.00
	15 3 20.00	2 20.00	1 6.67	4 26.67
	4.493	4.493	<0.001	0.536
P	0.034	0.034	1.000	0.464
	0.000 ^a	1 6.67	0 0.00	3 20.00
	0 0.00	0 0.00	0 0.00	0 0.00
	0.000 ^a	1 6.67	0.000 ^a	1 6.67
	0.000 ^a	0.000 ^a	0.000 ^a	0.000 ^a

^aP<0.05

MMP2

TMP1 VEGF

11

14 d CRP

1 J . 2019 21 7 636 640

2 J . 2022 36 6 604 607.

12 NPWT

3 Garrett M Gray S et al. Audit of diabetes related lower ex-
tremity amputations in the Northern Region of New Zealand
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NPWT DFO

4 J . 2019
20 4 207 229

5 J .
2022 30 2 189 192

7 d 14 d MMP2

6 J . 2021 50 8 155
157+161.

MMP 2

7 J . 2021 26
9 718 719.

13 TIMP 1 MMPs

8 . VSD rb bFGF Wagner2
J . 2024 44 7 39 41.

6 VEGF

9 J .
2020 22 10 787 790

10 . SPSS J .
2021 35 9 900 905.

MMP2

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betic Foot Disease Outcomes of a Multidisciplinary Clinical
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VEGF TMP1

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nitrile based fibres with chelated Ag ions for antibacterial ap-
plications. J . R Soc Open Sci 2020 7 7 310 324.

14 15 VEGF

13 2 2 Smad3
J . 2021 41 2 5 7.

6 NPWT

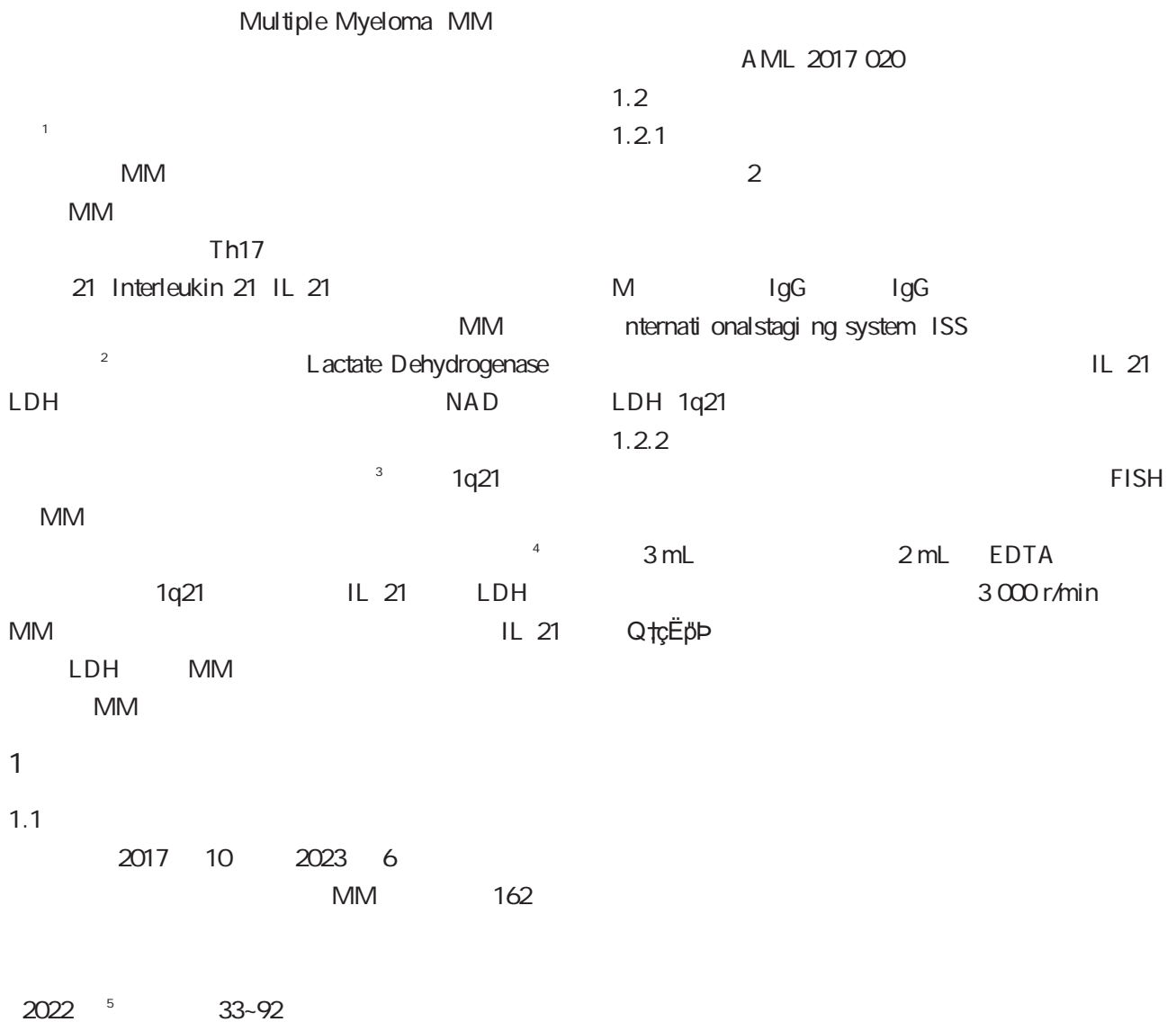
14 Tallapaneni V Kalaivani C Pamu D et al. Acellular Scaf-
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of Diabetic Wounds J . Tissue Eng Regen Med 2021 18
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DFO

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proteinases on the healing of diabetic foot ulcer A systematic
review. J Tissue Viability 2023 32 1 51 58.

tive group were significantly higher than those in the effective group with statistical significance $\chi^2=11.953$ 11.116 39.329 all $P<0.05$. The efficacy of MM patients was negatively correlated with serum IL 21 LDH levels and 1q21 amplification positive rate $r=-0.613$ -0.294 -0.572 all $P<0.05$. Multivariate regression analysis showed that age >60 combined amyloidosis 1q21 amplification elevated serum IL 21 and LDH expression levels were independent risk factors for the prognosis of MM patients $P<0.05$. ROC curve showed that the ROC areas of serum IL 21 and LDH and their combined detection were 0.785 0.781 and 0.849 respectively all $P<0.05$. The amplification of 1q21 and the high expression of serum IL 21 and LDH may affect the efficacy of MM patients and the combined detection of serum IL 21 and LDH has a high value in the prognosis assessment of MM patients.

Multiple myeloma 1q21 amplification Lactate dehydrogenase Interleukin 21



113 49 3 2

1 n % -

1.3 n=64 n=98

SPSS 22.0

t n %

2 Spearman kendall

IL 21 LDH 1q21 MM

Logistic MM

IL 21 LDH MM

ROC

P<0.05

2

2.1 IL 21 LDH

1q21

IL 21 LDH 1q21

P<

0.05 1

1 IL 21 LDH 1q21

n % -

Table 1 Comparison of serum IL 21 LDH levels and 1q21 amplification positive rate in different therapeutic groups

	n	IL 21 pg/mL	LDH U/L	1q21
	113	118.46±7.35	246.48±68.36	30 26.55
	49	142.77±18.58	393.17±94.54	39 79.59
t		11.953	11.116	39.329
P		0.000	0.000	0.000

2.2 IL 21 LDH 1q21 MM

MM IL 21 r=-0.613 LDH

r=-0.294 1q21 r=-0.572

P<0.05

2.3

ISS

1q21 IL 21

LDH P<

0.05 2

Table 2 Comparison of clinical data between good prognosis group and poor prognosis group

	n=162	n=64	n=98	t	P
>60	99	31 48.44	68 69.39	7.150	0.008
60	63	33 51.56	30 30.61	0.141	0.708
	89	34 53.13	55 56.12		
	73	30 46.87	43 43.88		
ISS				3.960	0.047
~	89	29 45.31	60 61.22		
	73	35 54.69	38 38.78		
M				0.515	0.473
IgG	56	20 31.25	36 36.73		
IgG	106	44 68.75	62 63.27	0.207	0.649
	18	8 12.50	10 10.20		
	144	56 87.50	88 89.80	5.486	0.019
	23	4 6.25	19 19.39		
	139	60 93.75	79 80.61		
1q21				15.879	0.000
	69	15 23.44	54 55.10		
	93	49 76.56	44 44.90		
IL 21 pg/mL		118.24±10.62	130.76±11.35	7.038	0.000
LDH U/L		239.86±80.31	324.15±92.37	5.972	0.000

2.4 MM

MM

>60 1q21

IL 21 LDH MM

P<0.05 3

2.5 IL 21 LDH MM

ROC IL 21 LDH

ROC 0.785 0.781

0.849

P<0.05 4 1

3

MM

MM

7 MM

3 MM
 Table 3 Multivariate regression analysis of independent risk factors influencing the prognosis of MM patients

			S.E	Wald	OR	95% CI	P		
	=0	=1	0.664	0.235	7.954	1.943	1.226-3.079	0.005	
	=0	=1	0.721	0.283	6.491	2.056	1.181-3.581	0.011	
ISS	~	=0	=1	0.742	0.483	2.360	2.100	0.815-5.412	0.124
1q21	=0	=1	0.531	0.201	6.979	1.701	1.147-2.522	0.008	
IL 21 pg/mL			0.683	0.218	9.816	1.980	1.291-3.035	0.002	
LDH U/L			0.730	0.228	10.251	2.076	1.327-3.244	0.001	

IL 21
 *

• •

lncRNA PVT1 HIF 1

Q19039

1. 610011
2. 518000

E mail xiackangli_cpu@126.com

1 PCR

Table 1 PCR primer sequence

	5 3	5 3
IncRNA PVT1	TTGGCACATACAGCCATCAT	GCAGTAAAAGGGGAACACCA
GAPDH	GAAGGTGAAGGTCGGAGTC	GAAGATGGTGATGGGATTTTC

Spearman
PVT1 HIF 1
ROC
EMs
PVT1 HIF 1
ROC
P<0.05

n %
2
EMs
R AFS
IncRNA PVT1 HIF 1
Z
IncRNA
EMs

Table 4 Comparison of serum IncRNA PVT1 and HIF 1 levels in patients with different R AFS stages

	n	IncRNA PVT1	HIF 1 pg/mL
	16	1.30±0.24	34.60±9.46
	25	1.69±0.36 ^a	74.25±16.83 ^a
	38	2.15±0.54 ^{ab}	106.03±28.54 ^{ab}
	13	2.71±0.68 ^{abc}	131.95±32.78 ^{abc}
F		25.236	50.317
P		<0.001	<0.001

2
2.1
EMs
P<0.05
BMI

^aP<0.05 ^bP<0.05
^cP<0.05
2.4 IncRNA PVT1 HIF 1 R AFS

P>0.05
2
2.2 IncRNA PVT1 HIF 1
EMs
IncRNA PVT1
P<
0.05
3

Pearson
HIF 1
r=0.419 P<0.05
Spearman
EMs
IncRNA
PVT1 HIF 1
R AFS
r₁=0.467 r₂=0.699 P<0.05

3
IncRNA PVT1 HIF 1
Table 3 Comparison of serum IncRNA PVT1 and HIF 1 expression levels between the two groups

	n	IncRNA PVT1	HIF 1 pg/mL
EMs	92	1.96±0.46	88.63±22.64
	92	1.09±0.31	41.33±11.36
t		15.044	17.911
P		<0.001	<0.001

2.5 IncRNA PVT1 HIF 1 EMs
ROC
EMs
AUC
0.985
87.0%
78.3%

2.3 R AFS EMs IncRNA
PVT1 HIF 1
R AFS
IncRNA PVT1 HIF 1
P<0.05
R AFS
EMs
IncRNA PVT1 HIF 1
P<0.05
4

AUC Z=2.015 P=0.044
HIF 1
AUC Z=2.836 P=0.005
1
2.6 IncRNA PVT1 HIF 1 EMs
IncRNA PVT1=1.35 HIF 1 =66.99 pg/mL
IncRNA PVT1 1.35
81 <1.35 11

2
Table 2 Comparison of general data between the two groups

	n	BMI kg/m ²	n %	n %
EMs	92	34.86±9.32	21.23±4.31	42 45.65
	92	35.73±9.47	20.94±4.26	54 58.70
t		0.628	0.459	43 46.74
P		0.531	0.647	23 25.00
				36 39.13
				46 50.00
				1.087
				1.778
				<0.001
				0.297
				0.182

PDK 1

1 2 2

CHF 3 3 1 PDK 1

2022 3 2024 3 180 CHF

CHF NYHA PDK1 mRNA C

110 suPAR 6 IL 6 8 IL 8

CRP Pearson ROC CHF CHF

PDK1 mRNA t=12.356 P<0.05 CRP suPAR IL 6 IL 8

t=12.193 19.347 18.742 13.044 P<0.05 CHF

PDK1 mRNA t=9.925 P<0.05 CRP suPAR

IL 6 IL 8 t=5.444 5.647 6.312 8.015 P<0.05 CHF

PDK1 mRNA CRP suPAR IL 6 IL 8 P<0.05 CHF

PDK1

3 1

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1. Department of Cardiology Chengdu Lansheng Brain Hospital Sichuan Chengdu China 610030

2. Internal Medicine Department of Chengdu Jinsha Hospital Sichuan Chengdu China 610000

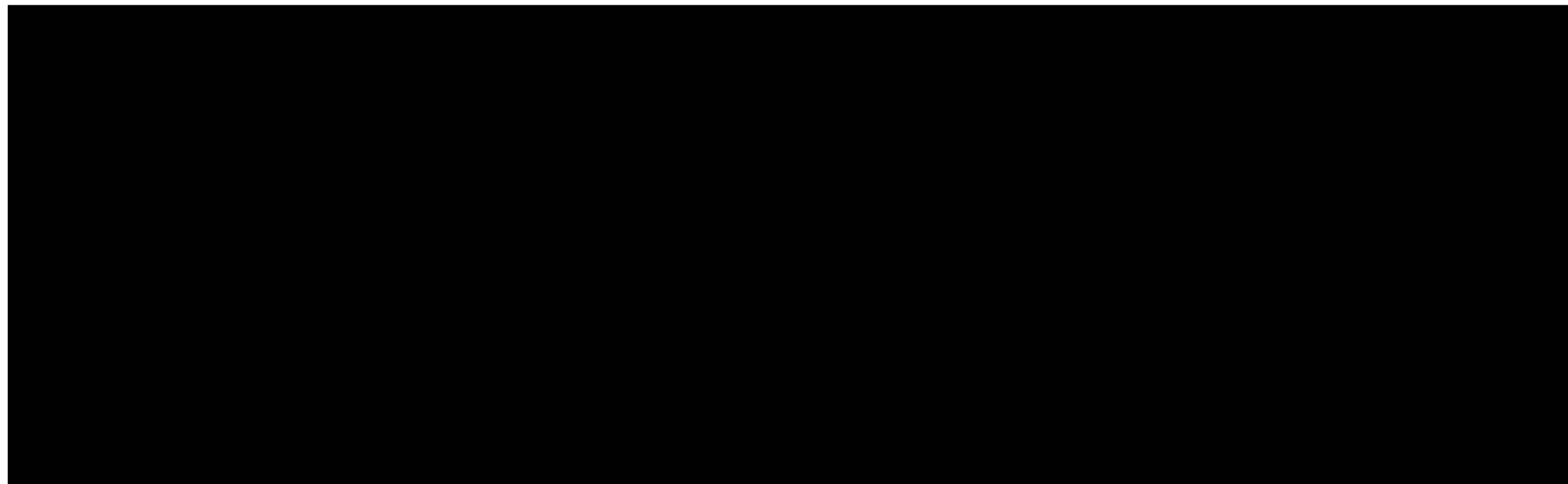
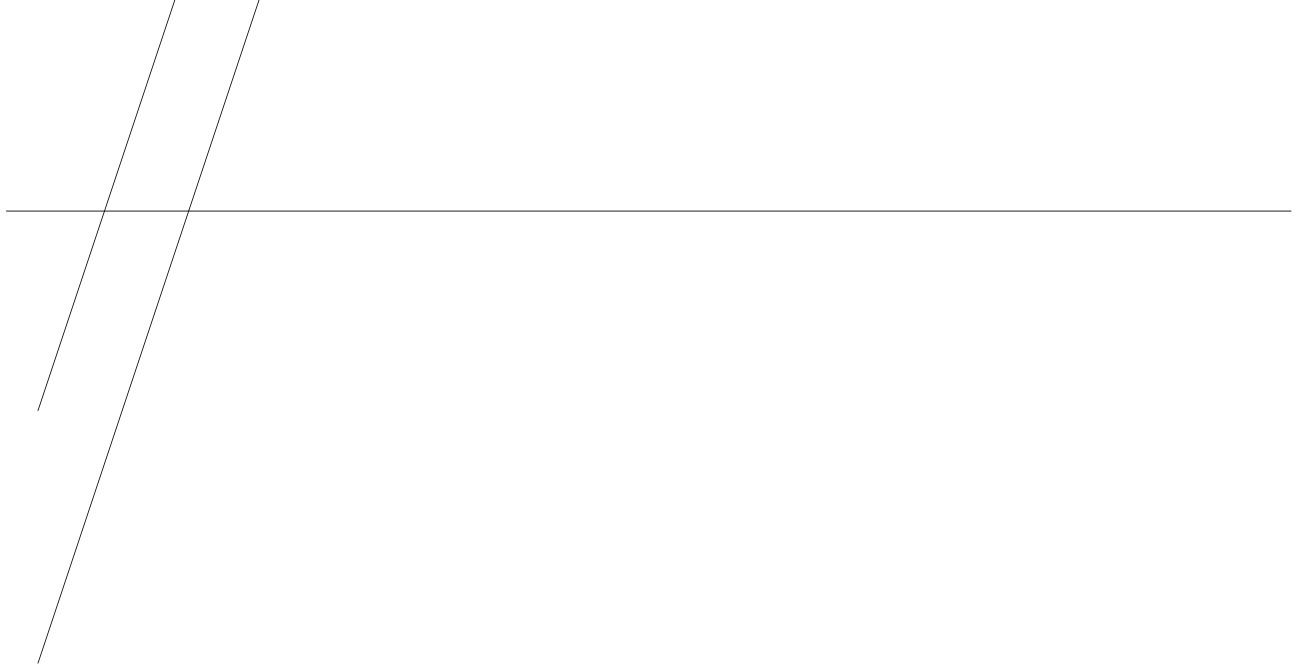
To investigate the correlation between the expression of 3-phosphoinositide dependent protein kinase 1 (PDK1) in peripheral blood and inflammatory response in patients with chronic heart failure (CHF) and its clinical significance. A total of 180 patients with CHF admitted to Lansheng Brain Hospital in Chengdu from March 2022 to March 2024 were selected for the CHF group. This group was further divided into mild heart failure with New York Heart Association (NYHA) grade I and severe heart failure with grade III-IV. Additionally, 110 healthy individuals who underwent physical examinations at our hospital during the same period were included as the control group. The mRNA expression level of PDK1 in peripheral blood and the levels of serum C-reactive protein (CRP), soluble urokinase-type plasminogen activator receptor (suPAR), interleukin 6 (IL-6), and interleukin 8 (IL-8) in serum were measured. Statistical analysis using t-test was conducted to compare the differences in PDK1, CRP, suPAR, IL-6, and IL-8 among all groups. The Pearson test was used to analyze correlations, and ROC curves were generated to assess the diagnostic value of each indicator in determining the condition of CHF. The mRNA expression level of PDK1 in the peripheral blood of the CHF group was lower than that of the control group (t=12.356, P<0.05), and the levels of

25ND018

1. 610030

2. 610000

E-mail: Xiangchunhua001@163.com



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 Reads
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2021 44 9 799 807.

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3 J .
2020 38 11 681 689.

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1181 1195.

6 Goddacre N et al. a reference viral database RVDB to enhance bioinformatics analysis of high throughput sequencing for novel virus detection J . mSphere 2018 3 2 69 78.

ER

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HSD17B6

17 HS

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3 HSD17B6

3.1 HSD17B6

ovarian syndrome PCOS

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HSD17B6 ²⁷

1 Programmed death re

ceptor 1 PD 1

PD 1 HSD17B6 HSD3B1 CYP19A1

1 3 N 3

²³ HSD17B6 LUAD LUAD ²⁸

5

4.3 HSD17B6 PCa HSD17B6 PCa DHT HSD17B6

DHT HSD17B

6 HSD17B10 DHT HSD17B6

PCa ^{7 24} PCa

PCa PCa HSD17B6

PCa HSD17B6

3 /3 diol DHT PCa

HSD17B6

2 HSD17B6

HSD17B6

HSD17B6 PCa ²⁵ PCa

3 / DHT

DHT

4.4 HSD17B6 HSD17B6

Non small

cell lung cancer NSCLC

NSCLC

HSD17B6 NSCLC ²⁶

HSD17B6 HSD17B6

- 1 Penning TM. Human hydroxysteroid dehydrogenases and pre-receptor regulation insights into inhibitor design and evaluation J . J Steroid Biochem Mol Biol 2011 125 1 2 46-56.
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